EPA United States Environmental Protection Agency

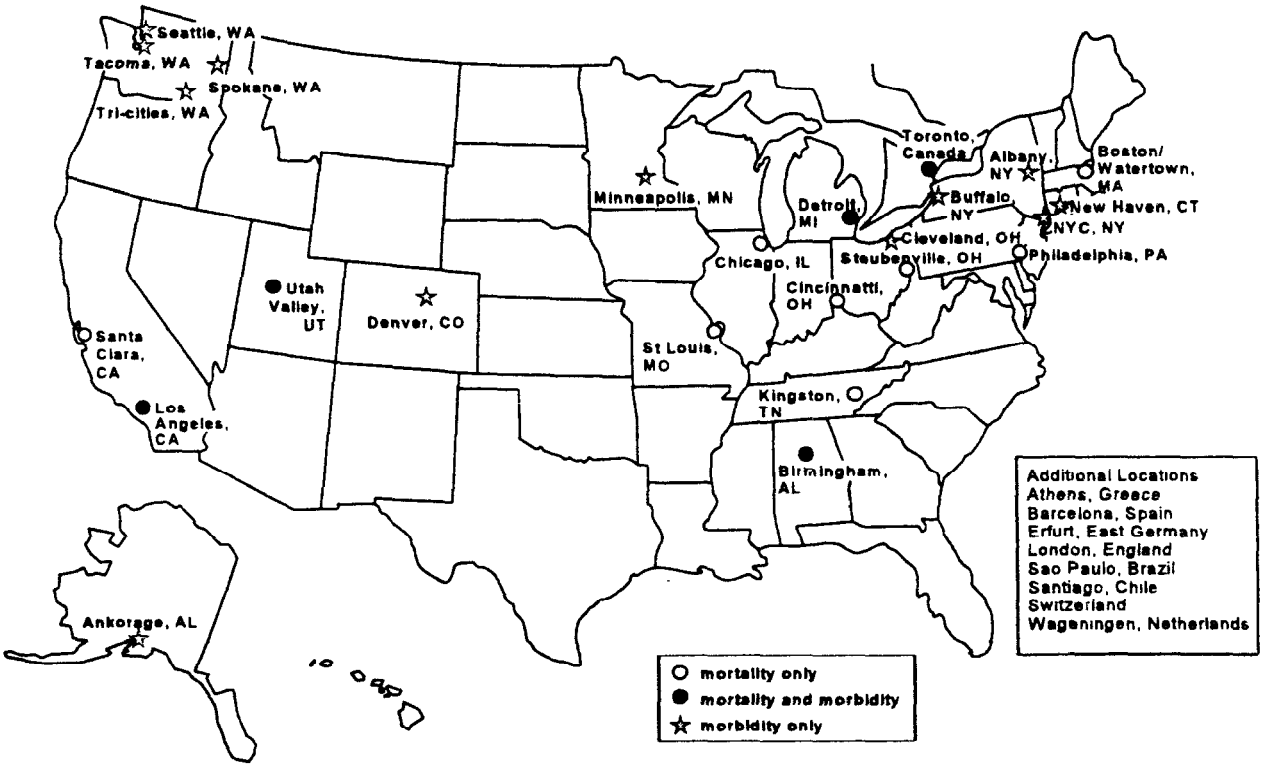
Office of Air Quality Planning and Standards
Research Triangle Park
North Carolina 27711

EPA-452\R-96-013
July 1996

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

Policy Assessment of Scientific and Technical Information

OAQPS Staff Paper



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Cover illustration: Locations of recently published community epidemiology studies finding statistically significant associations between short-term concentrations of particulate matter and health effects (CD, Tables 12-2 through 12-5). Studies conducted on three continents have found both increased morbidity and mortality to be associated with a variety of particle measurement devices, including mass measurements of TSP, PM10, PM2.5, sulfates, and acids, and optical based approaches including BS, KM, and COH. Although the highest PM-10 concentrations in the U.S. are in the West, most of the results in North America are from eastern communities, at PM-10 concentrations that are generally below those permitted by the current standards.

DISCLAIMER

This report has been reviewed by the Office of Air Quality Planning and Standards (OAQPS), U. S. Environmental Protection Agency (EPA), and approved for publication. This OAQPS Staff Paper contains the findings and conclusions of the staff of the OAQPS and does not necessarily represent those of the EPA. Mention of trade names or commercial products is not intended to constitute endorsement or recommendation for use.

ACKNOWLEDGEMENTS

This Staff Paper is the product of the Office of Air Quality Planning and Standards (OAQPS). For the primary standards, the principal authors include John Bachmann, Dr. Jane C. Caldwell, Patricia D. Koman, Harvey M. Richmond, Eric G. Smith of OAQPS, Dr. Tracey J. Woodruff of the Office of Policy, Planning and Evaluation (OPPE), and Dr. Karen M. Martin of OAQPS who also managed the project. For the secondary standards, principal authors include Rich Damberg, Chebryll Edwards, and Bruce Polkowsky of OAQPS. The authors would like to acknowledge John Haines for his guidance and expertise, and Terence Fitz-Simons, David Mintz, and Miki Wayland for providing expertise and analysis. Finally, the authors wish to acknowledge Tricia Crabtree for providing substantial wordprocessing and general support for the Staff Paper. The Staff Paper includes comments from OAQPS, the Office of Research and Development, OPPE, Regional Offices, and the Office of General Counsel within EPA.

On two different occasions, draft chapters of this document were formally reviewed by the Clean Air Scientific Advisory Committee (CASAC). Helpful comments and suggestions were also submitted by a number of independent scientists, by officials from several State and local air pollution organizations, by environmental groups, by industrial groups, and by individual companies.

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REVIEW OF THE NATIONAL AMBIENT AIR QUALITY STANDARDS FOR PARTICULATE MATTER:

POLICY ASSESSMENT OF SCIENTIFIC AND TECHNICAL INFORMATION

I. PURPOSE

The purpose of this Office of Air Quality Planning and Standards (OAQPS) Staff Paper is to evaluate the policy implications of the key studies and scientific information contained in the EPA document, "Air Quality Criteria for Particulate Matter" (U.S. EPA, 1996, henceforth referred to as the CD), and to identify the critical elements that EPA staff believes should be considered in review of the national ambient air quality standards (NAAQS) for particulate matter (PM). This assessment is intended to help bridge the gap between the scientific review contained in the CD and the judgments required of the Administrator in setting ambient standards for PM. Thus, emphasis is placed on identifying those conclusions and uncertainties in the available scientific literature that the staff believes should be considered in selecting particulate pollutant indicators, forms, averaging times, and levels for the primary (health) and secondary (welfare) standards. These specifications must be considered collectively in evaluating the health and welfare protection afforded by PM standards.

While this Staff Paper should be of use to all parties interested in the standards review, it is written for those decision makers, scientists, and staff who have some familiarity with the technical discussions contained in the CD. This Staff Paper presents factors relevant to the evaluation of current primary and secondary NAAQS, as well as staff conclusions and recommendations of suggested options for the Administrator to consider.

II. BACKGROUND

A. Legislative Requirements

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

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

The U.S. Court of Appeals for the District of Columbia Circuit has held that the requirement for an adequate margin of safety for primary standards was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting. It was also intended to provide a reasonable degree of protection

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

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

In selecting a margin of safety, the EPA considers such factors as the nature and severity of the health effects involved, the size of the sensitive population(s) at risk, and the kind and degree of the uncertainties that must be addressed. Given that the "margin of safety" requirement by definition only comes into play where no conclusive showing of adverse effects exists, such factors which involve unknown or only partially quantified risks have their inherent limits as guides to action. The selection of any particular approach to providing an adequate margin of safety is a policy choice left specifically to the Administrator's judgment (*Lead Industries Association v. EPA*, supra, 647 F.2d at 1161-62).

Section 109(d)(1) of the Act requires that "not later than December 31, 1980, and at 5-year intervals thereafter, the Administrator shall complete a thorough review of the criteria published under section 108 and the national ambient air quality standards ... and shall make such revisions in such criteria and standards ... as may be appropriate ...." Section 109(d)(2) requires that an independent scientific review committee be appointed and provides that the committee "shall complete a review of the criteria ... and the national primary and secondary ambient air quality standards ... and shall recommend to the Administrator any ... revisions of existing criteria and standards as may be appropriate ...." Since the early 1980's, this independent review function has been performed by the Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board.

B. History of PM NAAQS Reviews

1. Establishment of the NAAQS for Particulate Matter

National ambient air quality standards for PM were first established in 1971, based on the original criteria document (DHEW, 1969). Particulate matter is the generic term for a broad class of chemically and physically diverse substances that exist as discrete particles (liquid droplets or solids) over a wide range of sizes. Particles originate from a variety of anthropogenic stationary and mobile sources as well as natural sources. Particles may be emitted directly or formed in the atmosphere by transformations of gaseous emissions such as sulfur oxides, nitrogen oxides, and volatile organic substances. The chemical and physical properties of PM vary greatly with time, region, meteorology, and source category, thus complicating the assessment of health and welfare effects.

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

2. First Review of NAAQS for Particulate Matter

In October 1979 (44 FR 56731), EPA announced the first review of the criteria document and NAAQS for PM and, after a lengthy and elaborate process, promulgated significant revisions of the original standards in 1987 (52 FR 24854, July 1, 1987).[2] In that decision, EPA changed the indicator for particles from TSP to PM_{10}, the latter referring to particles with a mean aerodynamic diameter less than or equal to

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[2]The revised standards were based on a revised Criteria Document (U.S. EPA, 1982a), a corresponding Staff Paper (U.S. EPA, 1982b), and subsequent addenda to those documents (U.S. EPA, 1986a; U.A. EPA, 1986b). A detailed description of the process followed in reviewing and revising the original Criteria Document and NAAQS appears in the notice of final rulemaking (52 FR at 24636-37).

10 μm.[3] 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 $\mu g/m^3$, expected annual arithmetic mean. The secondary standard was revised by replacing it with 24-hour and annual standards identical in all respects to the primary standards. The revisions also included a new reference method for the measurement of PM_{10} in the ambient air and rules for determining attainment of the new standards. On judicial review, the revised standards were upheld in all respects (Natural Resources Defense Council v. Administrator, 902 F. 2d 962 (D.C. Cir. 1990), cert. denied, 111 S. Ct. 952 (1991)).

3. Recent Litigation

The American Lung Association filed suit in February 1994 to compel EPA to complete the present review of the PM NAAQS by December 1995. The U.S. District Court for the District of Arizona subsequently ordered EPA to complete its review and any revision of the PM NAAQS by publishing a final decision in the Federal Register by January 31, 1997, with publication of a proposed decision required by June 30, 1996 (American Lung Association v. Browner, CIV-93-643-TUC-ACM (D. Ariz., October 6, 1994)). As subsequently modified, the court-ordered schedule requires publication of the proposed and final decisions by November 29, 1996, and June 28, 1997, respectively.

4. Current Review of the Particulate Matter NAAQS

In December 1994, EPA presented its plans for completing review of the criteria document and NAAQS for PM under the court order to the CASAC. In addition, EPA's OAQPS completed a PM NAAQS Development Project Plan in January 1995, which incorporated CASAC comments, identifying key issues to be addressed in this Staff Paper

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[3]The more precise term is 50 percent cut point or 50 percent diameter (D_{50}). This is the aerodynamic particle diameter for which the efficiency of particle collection is 50 percent. Larger particles are not excluded altogether, but are collected with substantially decreasing efficiency and smaller particles are collected with increasing (up to 100 percent) efficiency. Ambient samplers with this cut point provide a reliable estimate of the total mass of suspended particulate matter of aerodynamic size less than or equal to 10 μm.

as well as the basis for the additional scientific and technical assessments needed to address the policy issues.

EPA desires to incorporate as much peer review and public input into the review as is possible under the court-ordered schedule. Accordingly, as part of the development of the CD, EPA hosted a public PM-Mortality Workshop in November 1994, at which seminal new studies on particles and health effects were presented and discussed. In January 1995, the EPA's National Center for Environmental Assessment (NCEA) hosted three public peer-review workshops on drafts of key chapters of a revised CD.

Successive external review drafts of the revised CD were reviewed by CASAC and the public at public meetings held on August 3-4, 1995 and December 15-16, 1995. The first external review draft of this Staff Paper was also reviewed by CASAC and the public at the December 16, 1995 meeting. Based on CASAC and public comment, NCEA revised the CD and submitted chapters the committee had requested for additional review (namely CD chapters 1, 5, 6, and 13) to CASAC and the public for review at a public meeting held February 29, 1996. At this meeting, CASAC also discussed the plan and methodologies for the risk assessment presented in this Staff Paper. On March 15, 1996, CASAC sent a letter to the EPA Administrator indicating the committee's satisfaction with the CD (Wolff, 1996b). NCEA made additional revisions to the document to respond to comments from CASAC and the public and completed the CD on April 12, 1996. At a public meeting held on May 15-16, 1996, CASAC and the public reviewed the revised Staff Paper, provided additional comments, and came to closure on the document. On June 13, 1996, CASAC sent a closure letter on the Staff Paper to the EPA Administrator (Wolff, 1996c). Both CASAC closure letters are reproduced in Appendix G of this Staff Paper.

III. APPROACH

This Staff Paper is based on the scientific evidence reviewed in the CD and takes into consideration CASAC and public comments received on the previous drafts. The staff has also considered comparative air quality and quantitative risk analyses in evaluating the appropriateness of retaining or revising the current primary NAAQS and in assessing potential alternative NAAQS. Technical and economic analyses examining visibility impairment and soiling and materials damage have also been considered in evaluating the appropriateness of retaining or revising the current secondary NAAQS and in assessing potential alternative NAAQS.

The approach taken in this Staff Paper is to assess and integrate the above information in the context of those critical elements that the staff believes should be considered in reviewing the primary and secondary standards. Attention is drawn to judgments that must be made based on careful interpretation of incomplete or uncertain evidence. In such instances, the Staff Paper provides the staff's evaluation, sets forth alternatives the staff believes should be considered, and recommends a course of action.

A. Bases for Initial Analytical Assessments

The staff identified several possible policy alternatives to provide a basis for commencing initial analytical assessments of air quality, human exposure, and health risks.

1. Primary Standards

As in the 1987 review of the NAAQS, selecting the most appropriate indicator for PM is a major issue for this review. Thus, the staff planned for initial analytic assessments of the assumption that this PM NAAQS review might result in setting or retaining one or more primary standards from the following possibilities:

- Short-term Standard: A 24-hour standard using a fine particle indicator, a PM_{10} indicator, or both; and
- Long-term Standard: An annual standard using a fine particle indicator, a PM_{10} indicator, or both.

The staff also recognized that other indicators of PM pollution (e.g., sulfates and acids) may be important in relating effects to PM pollution.

2. Secondary Standards

In revising the secondary standards, the staff has focused primarily on two types of effects: 1) visibility impairment and 2) soiling and materials damage. In the case of visibility, this Staff Paper briefly assesses available scientific information in order to determine an appropriate regulatory approach for addressing regional haze. A key consideration in this assessment is that a number of factors that influence visibility impairment vary significantly between the eastern and western parts of the U.S. Thus, this Staff Paper examines the advisability of a uniformly implemented and attained secondary NAAQS as contrasted to the establishment of a regional haze program under section 169A of the Clean Air Act. This Staff Paper also examines the available literature on material damage and soiling to ascertain whether such information provides a basis for establishing a separate national secondary NAAQS to protect against such effects.

B. Organization of Document

The remainder of this Staff Paper is organized as outlined below. Chapter IV summarizes differences among the various fractions of PM_{10}, air quality trends for both PM_{10} and fine particles, characterizations of average "background" concentrations, information on relationships between PM and population exposures, and the air quality implications of ongoing PM control programs designed to attain the current PM NAAQS.

Chapter V discusses available information on PM dosimetry and hypotheses regarding mechanisms of toxicity, the nature of health effects associated with PM, sensitive subpopulations, and integrated evaluations of the scientific evidence. Chapter V also presents alternative interpretations of the evidence and uncertainties surrounding reported health effects associations and specific agents of concern which are important for the Administrator to consider in selecting appropriate primary standards.

Chapter VI summarizes health risk assessments conducted for two urban areas to provide quantitative estimates of the risks to public health associated with 1) existing PM air quality levels, 2) projected air quality levels that would occur upon attainment of the current PM_{10} standards, and 3) projected air quality levels associated with attainment of alternative $PM_{2.5}$ standards.

Drawing on these factors and on information contained in the previous chapters, Chapter VII presents staff conclusions and recommendations for the Administrator to consider in reaching decisions on the retention and/or revision of the primary NAAQS. The chapter addresses alternative pollutant indicators, averaging times, forms, and levels, with summary sections highlighting both key uncertainties and related staff research recommendations as well as staff's overall recommendations for a suite of primary standards.

With respect to review of the secondary standards, Chapter VIII presents information on visibility impairment and soiling and materials damage, discusses pertinent scientific, technical, and policy considerations, and offers staff conclusions and recommendations for the Administrator to consider in reaching a decision on retention and/or revision of the secondary NAAQS.

IV. AIR QUALITY: CHARACTERIZATION AND IMPLICATIONS

This chapter defines the various subclasses of particulate matter (PM) and then briefly discusses the chemical and physical properties of PM in the atmosphere, recent PM concentrations and trends, the relationships between PM and population exposures, and the air quality implications of PM_{10} controls. This information is important both in interpreting the available health effects and welfare information and in making recommendations for appropriate indicators for PM.

A. Characterization of U.S. Ambient Particulate Matter

PM represents a broad class of chemically and physically diverse substances. The principal common feature of PM is existence as discrete particles in the condensed (liquid or solid) phase spanning several orders of magnitude in size, from molecular clusters of 0.005 μm in diameter to coarse particles on the order of 100 μm.[1] In addition to characterizations by size, particles can be described by their formation mechanism or origin, chemical composition, physical properties, and in terms of what is measured by a particular sampling technique.

In most locations, a variety of diverse activities contribute significantly to PM concentrations, including fuel combustion (from vehicles, power generation, and industrial facilities), residential fireplaces, agricultural and silvicultural burning, and atmospheric formation from gaseous precursors (largely produced from fuel combustion). Other sources include construction and demolition activities, wind blown dust, and road dust. From these diverse sources come the mix of substances that comprise PM. The major chemical constituents of PM_{10} are sulfates, nitrates, carbonaceous compounds (both elemental and organic carbon compounds), acids, ammonium ions, metal compounds, water, and crustal materials. The amounts of these components vary from place to place and over time.

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[1] In this Staff Paper, particle size or diameter refers to aerodynamic diameter, which is defined as the diameter of a spherical particle with equal settling velocity but a material density of 1 g/cm^3, normalizing particles of different shapes and densities (CD, page 3-8).

1. Multi-modal Size Distributions

The health and environmental effects of PM are strongly related to the size of the particles. The aerodynamic size and associated composition of particles determines their behavior in the respiratory system (i.e., how far the particles are able to penetrate, where particles are deposited, and how effective the body's clearance mechanisms are in removing them as discussed in Chapter V). Furthermore, particle size is one of the most important parameters in determining atmospheric lifetime of particles, which is a key consideration in assessing health effects information because of its relationship to exposure. The total surface area and number of particles, chemical composition, water solubility, formation process, and emission sources all vary with particle size. Particle size is also a determinant of visibility impairment, a welfare consideration linked to fine particle concentrations. Thus, size is an important parameter in characterizing PM, and particle diameter has been used to define the present standards.

The multi-modal distribution of particles based on diameter has long been recognized (Whitby et al., 1972; Whitby et al., 1975; Willeke and Whitby, 1975; National Research Council, 1979; U.S. EPA, 1982a; U.S. EPA, 1982b; U.S. EPA, 1986b; CD Section 3.1.3.2). Although particles display a consistent multi-modal distribution over several physical metrics such as volume and mass, specific distributions may vary over place, conditions, and time because of different sources, atmospheric conditions, and topography. Based on particle size and formation mechanism, particles can be classified into two fundamental modes: fine and coarse modes. Figure IV-1 illustrates an idealized mass distribution of the fine and coarse modes. A depiction of typical number, surface area, and volume distribution of ambient particles is shown in Figure IV-2. This latter figure illustrates that fine particles can be further subdivided into nuclei or ultrafine, and accumulation modes.[2] As illustrated in the figure,

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[2] Typically, the accumulation mode can be characterized by mass median aerodynamic diameter (MMAD) of 0.3 to 0.7 μm and a geometric standard deviation (sigma-g) of 1.5 - 1.8 (CD, page 13-5). The CD defines ultrafine particles as $\leq$ 0.1 μm in diameter (CD, Sections 3.1.3 and 13.2.1). Nuclei or ultrafine particles tend to exist as disaggregated particles for very short periods of time (minutes) and rapidly coagulate into accumulation mode particles (CD page 3-10). Accumulation mode particles, however, do not grow further into the coarse particle mode.

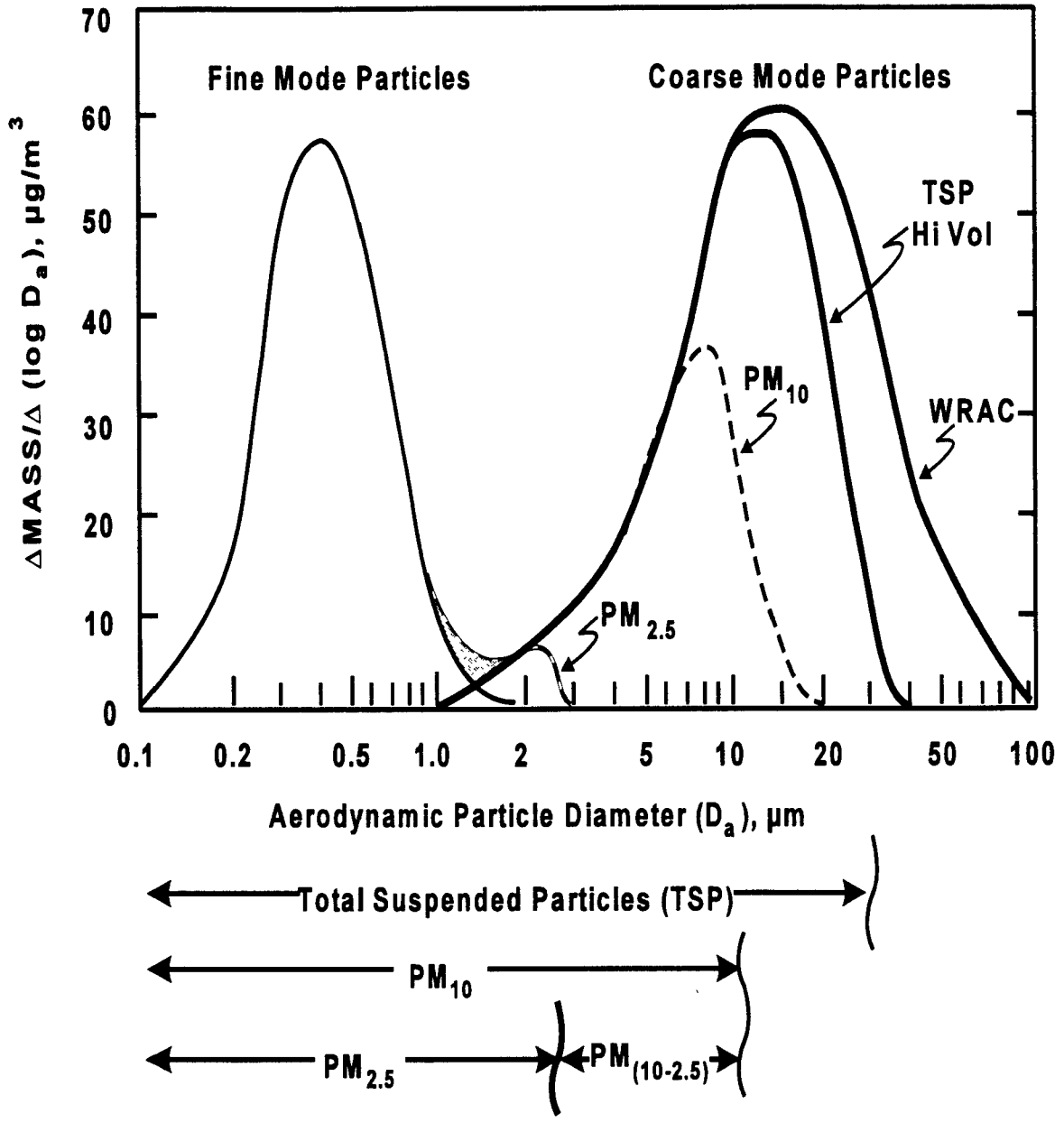

Figure IV-1. Idealized Distribution of Ambient Particulate Matter

Distribution shows fine and coarse mode particles and fractions collected by size-selective samplers such as the wide range aerosol collector (WRAC) and the TSP high volume sampler. (Adapted from Wilson and Suh (1996); CD Figure 3-3).

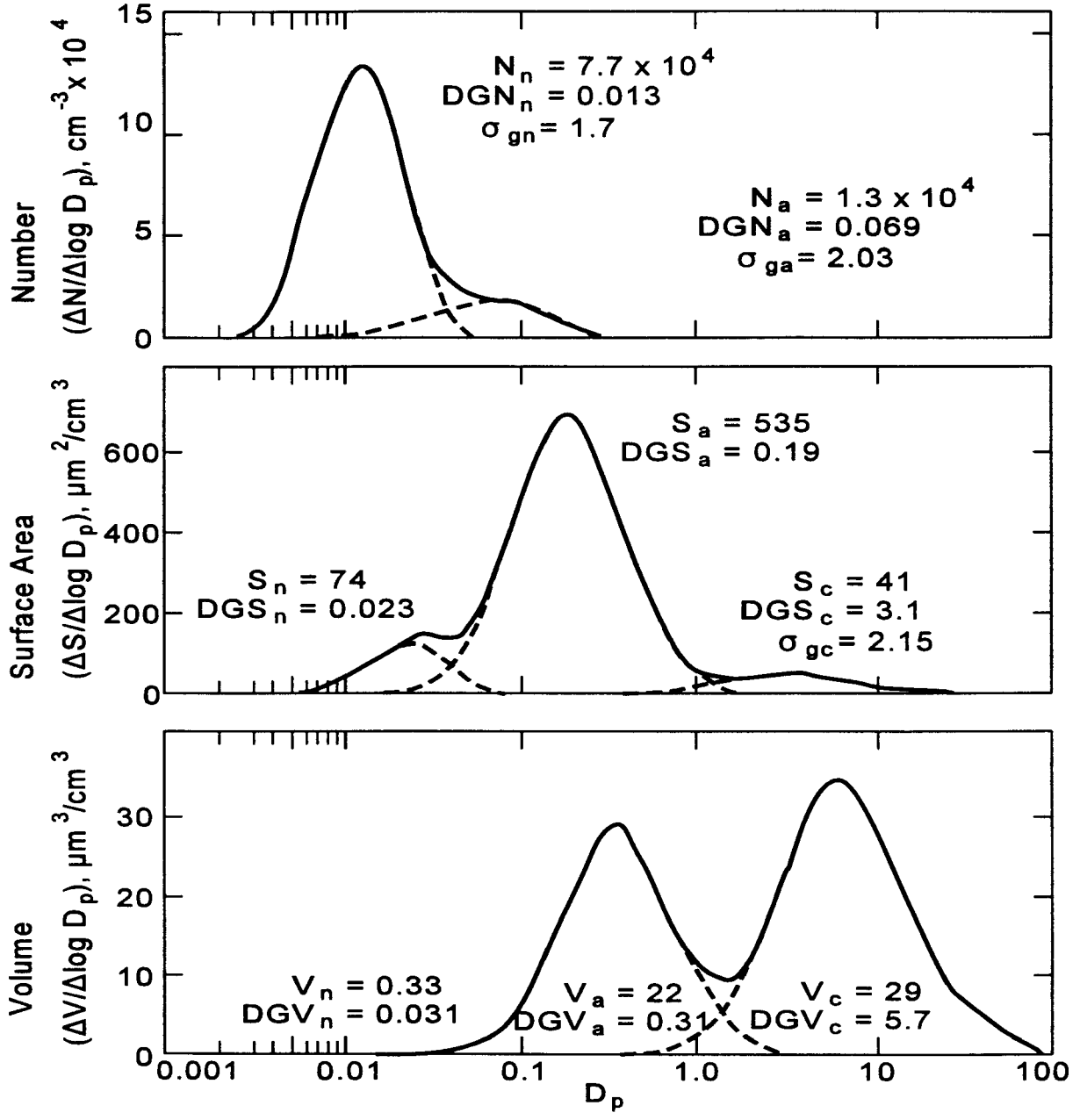

FIGURE IV-2. DISTRIBUTION OF PARTICLES BY NUMBER, SURFACE AREA, AND VOLUME. Distribution of coarse (c), accumulation or fine (a), and nucleation or ultrafine (n) mode particles by three characteristics. DVG = geometric mean diameter by volume; DSG = geometric mean diameter by surface area; DGN = geometric mean diameter by number. Source: Whitby (1978); CD, Figure 13-4.

even when the fine mode contains about 40 percent of the volume or mass of PM_{10}, it accounts for most of the surface area and number of particles.

The CD concludes that an appropriate cut point[3] for distinguishing between the fine and coarse modes lies in the range of 1.0 μm to 3.0 μm where the minimum mass occurs between the two modes (CD, Section 3.1.2; Miller et al., 1979). The CD states that the data do not provide a clear choice of cut point given the overlap that occurs between the modes. Most ambient measurements of fine particle mass in the U.S. have used instruments with cut points of 2.5 or 2.1 μm. Appendix A outlines the policy considerations involved in making the staff recommendation for using 2.5 μm as the cut point for measuring fine particles.

Table IV-1 introduces some of the size-related terminology used in this Staff Paper. For the purposes of this document, PM_X (e.g., X = 1, 2.5, 10, 15, 10-2.5) is used to refer to gravimetric measurements with a 50 percent cut point of X μm diameter while the terms *fine* or *coarse particles* will be used more generally to refer to the fine and coarse modes of the particle distribution. The distinction highlights the role of formation mechanism and chemistry in addition to size in defining fine and coarse mode particles. Any specific measurement (e.g., $PM_{2.5}$) is only an approximation for fine particles.[4]

In addition to gravimetric fine particle measurements, PM has been characterized in the U.S. and abroad using a variety of filter-based optical techniques including British or black smoke (BS), coefficient of haze (COH), and carbonaceous material (KM), as well as estimates derived from visibility measurements (CD, Chapter 4 and 12; see Appendix B of Staff Paper for limitations in determining mass). In locations where they are calibrated to standard mass units (e.g. London), these measurements can be useful as surrogates for fine particle mass (CD, Chapter 4).

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[3] When used in the context of sampling, *cut point* is a term used to describe the separation efficiency curve for samplers. The cut point is typically described by the aerodynamic diameter at which the sampler achieves 50 percent collection efficiency.

[4] Monitor design, measurement temperature, and inlet efficiency can also affect which particles are included in the definitions of the various size fractions (CD, Chapter 4). Sampling protocols may also affect the amount of semivolatile organics and nitrates and particle-bound water included in a measurement.

TABLE IV-1. PARTICLE SIZE FRACTION TERMINOLOGY USED IN THIS STAFF PAPER

Term	Description
Size Distribution Modes	
Fine particles	Fine mode particles which are generally formed through chemical reaction, nucleation, condensation of gases, and coagulation of smaller particles; contains most numerous particles and represents most surface area.
Coarse Particles	Coarse mode particles which are mostly generated from mechanical processes through crushing or grinding.
Sampling Measurements	
Total Suspended Particles	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[1] 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 to have an effective cut point of 10 μm aerodynamic diameter. This measurement includes the fine mode and part of the general coarse mode and is an indicator for thoracic particles (i.e., particles that penetrate to the tracheo-bronchial and the gas-exchange regions of the lung).
$PM_{2.5}$	Particles measured by a sampler that contains a size fractionator (classifier) designed to have an effective cut point of 2.5 μm. The collected particles include most of the fine mode. Some small portion of the coarse mode may be included depending on the sharpness of the sampler efficiency curve and the size of coarse mode particles present.
Coarse fraction of PM_{10} $PM_{(10-2.5)}$	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.

[1]When discussing samplers, cut point is a term used to describe the separation efficiency curve for particle collection devices. The cut point is typically described by the particular aerodynamic diameter at which the sampler achieves 50% collection efficiency. Aerodynamic diameter is defined as the diameter of a spherical particle with equal settling velocity but a material density of 1 g/cm^3. This normalizes particles of different shapes and densities.

[2]PM_X indicates an 50 percent cut point of X μm diameter. Because samplers have a collection efficiency that varies around the cut point, not all particles less than X μm diameter will be collected and some particles greater than X μm diameter will be collected.

The distinction between any specific measurement of fine particles and fine mode (or a measurement of coarse particles and coarse mode) is important because in the subsequent chapters of this Staff Paper, the staff draws public health conclusions regarding fine and coarse mode particles and in doing so the staff relies on the available measurements. Examples of fine particle measurements include $PM_{2.5}$, BS, COH, and concentrations of specific chemical classes predominantly in the fine fraction such as sulfates and acids all judged to be surrogates for fine mode particles. Measurements of coarse particles include $PM_{10-2.5}$, $PM_{15-2.5}$, and TSP minus PM_{10}.

2. Properties of Fine and Coarse Fraction Particles

As summarized in Table IV-2, fine and coarse particles can be differentiated by their sources and formation processes, chemical composition, solubility, acidity, atmospheric lifetime and behavior, and transport distances (CD Chapter 3). The key properties of fine and coarse particles are described below.

a. Sources and Formation Processes

Fine and coarse particles generally have distinct sources and formation mechanisms although there may be some overlap. Primary fine particles are formed from condensation of high temperature vapors during combustion (CD, page 3-2). Fine particles are usually formed from gases in three ways: (1) nucleation (i.e., gas molecules coming together to form a new particle), (2) condensation of gases onto existing particles, and (3) by reaction in the liquid phase (CD, page 13-7). Particles formed from nucleation also coagulate to form relatively larger particles, although such particles normally do not grow into the coarse mode (CD, Section 3.1.3.2). Particles formed as a result of chemical reaction of gases in the atmosphere are termed secondary particles because the direct emission from a source is a gas that is subsequently converted to a product that either has a low enough vapor pressure to form a particle or reacts further to form a low vapor pressure substance. Some examples include the conversion of sulfur dioxide (SO_2) to sulfuric acid droplets that further react with ammonium to form particulate sulfate, or the conversion of nitrogen dioxide (NO_2) to nitric acid which reacts further with ammonia to form particulate ammonium nitrate (NH_4NO_3) (CD, Section 3.2.2). Although directly emitted particles are found in the fine fraction (the most common

TABLE IV-2. COMPARISON OF AMBIENT FINE AND COARSE MODE PARTICLES

	Fine Mode	Coarse Mode
Formed from:	Gases	Large solids/droplets
Formed by:	Chemical reaction; Nucleation; Condensation; Coagulation; Evaporation of fog and cloud droplets in which gases have dissolved and reacted.	Mechanical disruption (e.g., crushing, grinding, abrasion of surfaces); Evaporation of sprays; Suspension of dusts.
Composed of:	Sulfate, $SO_4^=$; Nitrate, NO_3^-; Ammonium, NH_4^+; Hydrogen ion, H^+; Elemental carbon Organic compounds (e.g., PAHs, PNAs); Metals (e.g., Pb, Cd, V, Ni, Cu, Zn, Mn, Fe); Particle-bound water.	Resuspended dusts (e.g., soil dust, street dust); Coal and oil fly ash; Metal oxides of crustal elements (Si, Al, Ti, Fe); $CaCO_3$, NaCl, sea salt; Pollen, mold spores; Plant/animal fragments; Tire wear debris.
Solubility:	Largely soluble, hygroscopic and deliquescent.	Largely insoluble and non-hygroscopic.
Sources:	Combustion of coal, oil, gasoline, diesel, wood; Atmospheric transformation products of NO_x, SO_2, and organic compounds including biogenic species (e.g., terpenes); High temperature processes, smelters, steel mills, etc.	Resuspension of industrial dust and soil tracked onto roads; Suspension from disturbed soil (e.g., farming, mining, unpaved roads); Biological sources; Construction and demolition; Coal and oil combustion; Ocean spray.
Lifetimes:	Days to weeks	Minutes to hours
Travel Distance:	100s to 1000s of kilometers	< 1 to 10s of kilometers

Source: Adapted from Wilson and Suh (1996); CD (Table 3-15, p. 3-145)

being particles less than 1.0 μm in diameter from combustion sources), particles formed secondarily from gases dominate the fine fraction.

By contrast, most of the coarse fraction particles are emitted directly as particles and result from mechanical disruption such as crushing, grinding, evaporation of sprays, or suspensions of dust from construction and agricultural operations. Simply put, most coarse particles are formed by breaking up bigger particles into smaller ones. Energy considerations normally limit coarse particle sizes to greater than 1.0 μm in diameter (CD, Chapter 3). Some combustion-generated particles such as fly ash are also found in the coarse fraction.

b. Chemical Composition, Solubility, and Acidity

Fine and coarse mode particles generally have distinct chemical composition, solubility, and acidity. Fine mode PM is mainly composed of varying proportions of several major components: sulfates, nitrates, acids, ammonium, elemental carbon, organic carbon compounds, trace elements such as metals, and water. By contrast, coarse fraction constituents are primarily crustal, consisting of Si, Al, Fe, and K (note that small amounts of Fe and K are also found among the fine mode particles but stem from different sources). Biological material such as bacteria, pollen, and spores may also be found in the coarse mode. As a result of the fundamentally different chemical compositions and sources of fine and coarse fraction particles, the chemical composition of the sum of these two fractions, PM_{10}, is more heterogenous than either mode alone.

Figure IV-3 presents a synthesis of the available published data on the chemical composition of $PM_{2.5}$ and coarse fraction particles in U.S. cities by region described in Chapter 6 of the CD. The CD concludes that the fine and coarse fraction are composed of different chemical constituents and that each fraction also has regional patterns resulting from the differences in sources and atmospheric conditions (CD, Section 6.6). Differences across the country in sources and atmospheric conditions contribute to the variability. In addition to the larger relative shares of crustal materials in the West, total concentrations of coarse fraction particles are generally higher in the arid areas of the Western and Southwestern U.S.

In general, fine and coarse particles exhibit different degrees of solubility and acidity. With the exception of carbon and some organic compounds, fine particle mass is largely

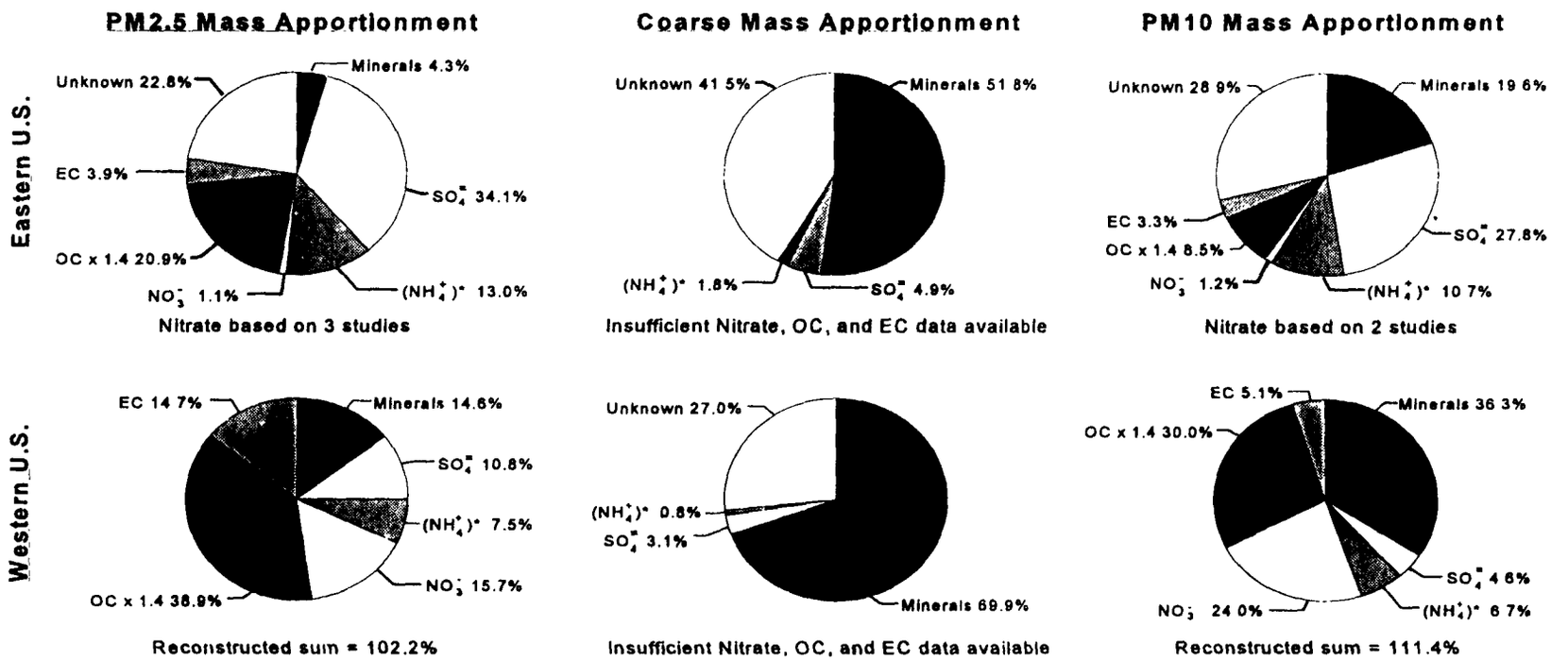

Figure IV-3. Major constituents of $PM_{2.5}$, Coarse Fraction, and PM_{10} (CD, Figures 6-85a-c).* Sulfate ions, ammonium ions, and organic carbon account for most of the $PM_{2.5}$ mass. Eastern $PM_{2.5}$ has more sulfate; whereas, many western sites have a larger nitrate contribution and twice the proportion of organic carbon compounds of eastern sites. In contrast, minerals dominate the coarse fraction, ranging from over 50 percent in the Eastern U.S. to 70 percent in the Western U.S. of coarse fraction mass. Total concentrations of coarse fraction particles are generally higher in the arid areas of the Western and Southwestern U.S. than in the Eastern U.S.

The analysis focuses on data from the Harvard Six-City Study and the Inhalable Particle Network (IPN) as well as other published data shown in CD Tables A-2a-c. (NH_4^+) represents the concentration of NH_4^+ that would be required if all the sulfate ion were present as ammonium sulfate and all the NO_3^- as NH_4NO_3. Therefore, (NH_4^+)* represents an upper limit to the true concentration of (NH_4^+)*. The unknown fraction of fine mass is assumed to be mainly water .

soluble in water and hygroscopic (i.e., fine particles readily take up and retain water). The fine particle mode also contains the acidic fraction (CD, Section 3.3.1). By contrast, coarse particles are mostly insoluble, non-hygroscopic, and generally basic.

c. Atmospheric Behavior

Fine and coarse particles typically exhibit different behavior in the atmosphere. These differences affect several exposure considerations including the representativeness of central-site monitored values and the behavior of particles formed outdoors once inside homes and buildings where people spend most of their time (as discussed below in Section C).

Fine accumulation mode particles typically have longer atmospheric lifetimes (i.e., days to weeks) than coarse particles and tend to be more uniformly dispersed across an urban area or large geographic region, especially in the Eastern U.S. (CD Sections 3.7, 6.3, and 6.4; Wilson et al., 1995; Eldred and Cahill, 1994; Wolff et al., 1985; Shaw and Paur 1983; Altshuller 1982; Leaderer et al., 1982). As noted above, secondary fine particles are formed by atmospheric transformation of gases to particles. Such atmospheric transformation can take place locally during atmospheric stagnation or during transport over long distances. For example, the formation of sulfates from SO_2 emitted by power plants with tall stacks can occur over distances exceeding 300 kilometers and 12 hours of transport time; therefore, the resulting particles are well mixed in the air shed (CD, Sections 3.4.2.1, and 6.4.1) Once formed, the very low dry deposition velocities of fine particles contribute to their persistence and uniformity throughout an air mass (CD, Sections 6.4 and page 7.2; Suh et al., 1995; Burton et al., 1996).

Larger particles generally deposit more rapidly than small particles; as a result, total coarse particle mass will be less uniform in concentration across an urban area than are fine particles (CD, Sections 3.7, and 13.2.4). Because coarse particles may vary in size from about 1 μm to over 100 μm, it is important to note their wide range of atmospheric behavior characteristics. For example, the larger coarse particles (> 10 μm) tend to rapidly fall out of the air and have atmospheric lifetimes of only minutes to hours depending on their size and other factors (Wilson and Suh, 1995; Chow et al., 1991; CD, Section 3.2.4). Their spatial impact is typically limited by a tendency to fallout in the proximate area downwind of their

emission point. Such large 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 impaction on surfaces (DRI, 1995; CD, Sections 7.2.2 and 13.2.4). The atmospheric behavior of smaller "coarse fraction" particles ($PM_{10-2.5}$) is intermediate between that of the larger coarse particles and smaller fine particles. Thus, coarse fraction particles may have lifetimes on the order of days and travel distances of up to 100 km or more.[5] While it may be reasonable to expect that coarse fraction particles would be less homogeneously distributed across an urban area than fine particles in areas with regionally high fine particle concentrations (e.g. the eastern U.S.), this is not consistently true in a variety of locations (DRI, 1995). In some locations, source distribution and meteorology affects the relative homogeneity of fine and coarse particles, and in some cases, the greater measurement error in estimating coarse fraction mass (Rodes and Evans, 1985) precludes clear conclusions about relative homogeneity.

Nevertheless, because fine particles remain suspended for longer times (typically on the order of days to weeks as opposed to days for coarse fraction particles) and travel much farther (i.e., hundreds to thousands of kilometers) than coarse fraction particles (i.e., tens to hundreds of kilometers), all else being equal, fine particles are theoretically likely to be more uniformly dispersed across urban and regional scales than coarse fraction particles. In contrast, coarse particles tend to be less evenly dispersed around urban areas and exhibit more localized elevated concentrations near sources (CD, Section 13.2.7; DRI, 1995).

d. Correlations between $PM_{2.5}$ and Coarse Fraction Mass

As might be expected from the differences in origin, composition, and behavior, ambient daily fine and coarse fraction mass concentrations generally are not well correlated. An analysis (SAI, 1996) of several data sets conducted for this review reported the R-squared statistic between daily $PM_{2.5}$ and $PM_{10-2.5}$ mass to be 0.13 for all non-rural sites and 0.21

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[5] In extreme cases, dust storms occasionally cause very long-range transport of the smaller size coarse particles.

when rural sites were included.[6] The results indicate a poor correlation between daily averages of the fine and the coarse fractions. In some specific instances, however, fine and coarse fractions may be correlated. For example, a vehicle moving on a dusty road would emit fine particles from the exhaust and produce coarse particle emissions from the road dust. In locations with poorly controlled industrial emissions of both fine and coarse particles, R^2 as high as 0.7 have been reported (Schwartz et al., 1996a).

e. Summary

In summary, the fine and coarse mode particles are distinct entities with differing sources and formation processes, chemical composition, atmospheric lifetimes and behaviors, and transport distances. The CD concludes that these profound differences alone justify consideration of fine and coarse fraction particles as separate pollutants for measurement and development of control strategies. The fundamental differences between fine and coarse particles are also important considerations in assessing the available health effects and exposure information.

B. PM Air Quality Patterns

This section outlines geographic distributions of PM as well as ambient concentration trends and background levels for PM_{10} and fine particles.

1. PM Concentrations and Trends

a. PM_{10} Concentrations and Trends

State and local air pollution control agencies have been collecting PM_{10} mass concentration data using EPA-approved reference samplers and reporting these data to EPA's publicly available AIRS database since mid-1987. Figure IV-4 shows geographic distribution of the 83 areas that are listed as not attaining the current PM_{10} standards as of September

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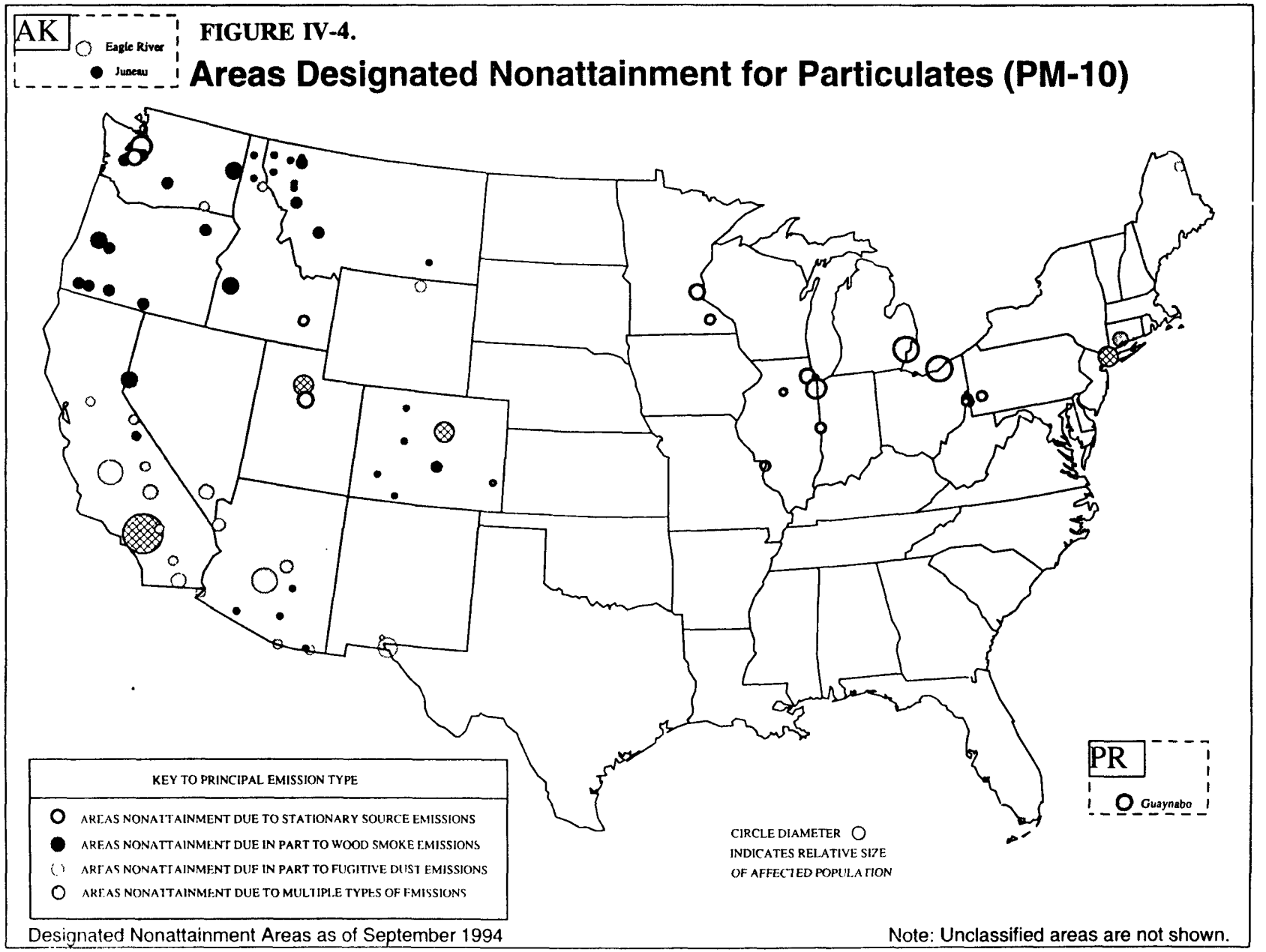
[6] SAI (1996) reported the following:
(1) R^2 = 0.13 of daily $PM_{2.5}$ with daily coarse fraction mass concentrations (n = 8,676) between 1988 and 1993 using the Aerometric Information Retrieval System (AIRS), Interagency Monitoring of Protected Visual Environments (IMPROVE), California Air Resources Board (CARB) Dichotomous Network (1990-1993 data), with rural sites removed.
(2) R^2 = 0.21 of daily $PM_{2.5}$ with daily coarse fraction mass concentrations (n = 31,510; 57% rural data) between 1985 and 1993 using AIRS, IMPROVE, CARB Dichot Network (1990-1993 data), and South Coast Air Basin (SCAB) Intensive Monitoring Network (IMN) (1985-1986).


AK
Eagle River
Juneau
FIGURE IV-4.
Areas Designated Nonattainment for Particulates (PM-10)
KEY TO PRINCIPAL EMISSION TYPE
AREAS NONATTAINMENT DUE TO STATIONARY SOURCE EMISSIONS
AREAS NONATTAINMENT DUE IN PART TO WOOD SMOKE EMISSIONS
AREAS NONATTAINMENT DUE IN PART TO FUGITIVE DUST EMISSIONS
AREAS NONATTAINMENT DUE TO MULTIPLE TYPES OF EMISSIONS
CIRCLE DIAMETER
INDICATES RELATIVE SIZE
OF AFFECTED POPULATION
PR
Guaynabo
Designated Nonattainment Areas as of September 1994
Note: Unclassified areas are not shown.

1994; the figure also summarizes the prevalent contributing sources and size of population residing in nonattainment areas. Most of the non-attainment areas are in the Western U.S. with fewer in heavily populated or industrialized eastern areas. Many of the highest values occur in western areas with fugitive dust sources and in mountain valleys impacted by wood smoke during winter inversions (CD, Section 6.5).

National trends may readily be examined for the 6-year period from 1988 to 1993 as illustrated in Figures IV-5a and IV-5b. The figures represent 799 trend sites, mostly from urban and suburban locations as well as a few remote locations; monitoring sites with data in at least five of the six years are included. The figures show the trend and site-to-site variability in the composite annual mean and the ninetieth percentile of 24-hour PM_{10} concentrations.[7] The trend for the composite annual mean shows a steady decline totaling 20 percent over the six-year period from 1988 to 1993. The ninetieth percentile similarly decreases 19 percent over the same period (U.S. EPA, 1994a). Annual average PM_{10} concentrations ranged from 25 to 35 $\mu g/m^3$ for most U.S. regions by 1994. Additional information about current PM_{10} concentrations are presented in Appendix C.

b. Fine Particle Concentrations and Trends

The $PM_{2.5}$ concentration data are considerably more limited than for PM_{10}. From 1983 to 1993, fewer than 50 sites reported data to AIRS in any given year.[8] Figure IV-6 displays a quarterly smoothed geographic distribution of the IMPROVE and Northeast States Coordinated Air Use Management (NESCAUM) networks' $PM_{2.5}$ data. These data generally do not include urban concentrations but represent the regional non-urban concentrations. The figure shows both the regional character of elevated fine particle levels in the Eastern U.S. and

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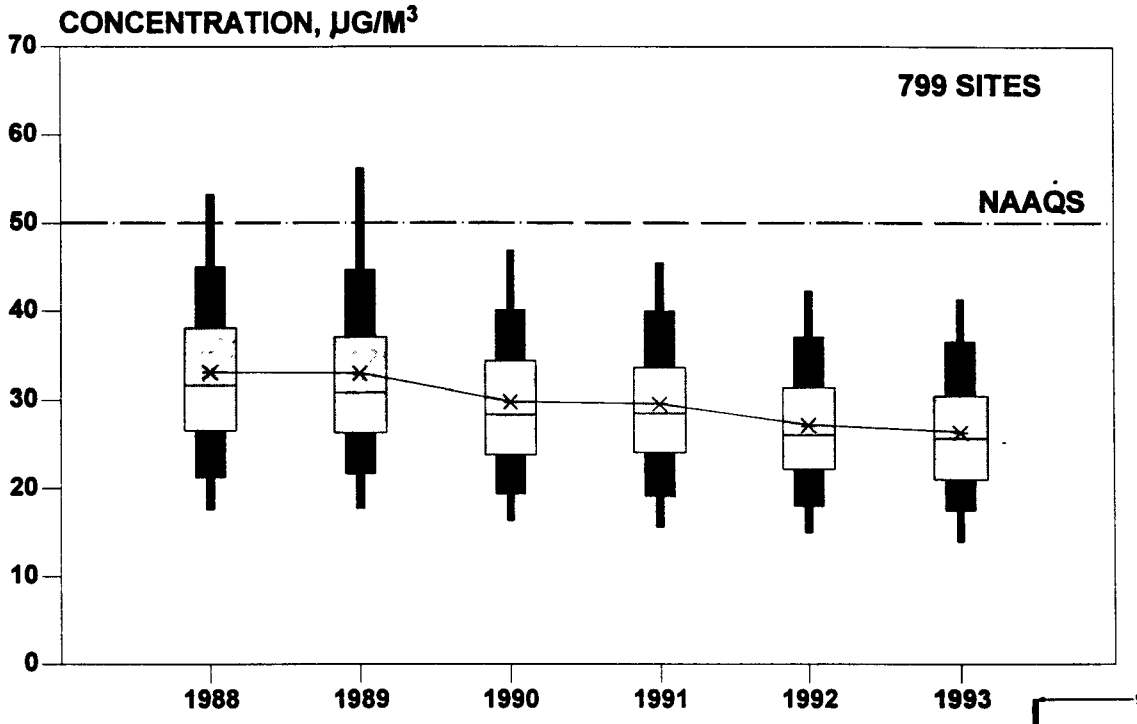
[7] The ninetieth percentile statistic is used because PM_{10} sampling frequency varies among sites and may change from one year to the next at some sites. This statistic is less sensitive to changes in sampling frequency than are the maximum or second maximum peak values. Most PM_{10} sites sample on a once every six day schedule.

[8] Additional special studies have also monitored PM2.5, but these data are not reported in AIRS. For this review, EPA assembled other available data sets for analysis (see CD, Section 6.10 and SAI, 1996). The databases assembled to support this Staff Paper include AIRS, Inhalable Particle Network (IPN) (1982-1984), IMPROVE (1987-1995), CARB Dichotomous Network (1990-1993), and SCAB IMN (1985-1986). Figure C-4 in Appendix C provides a summary of the available data for fine particles.

FIGURE IV-5a. **PM-10 TREND, 1988-1993**
(ANNUAL ARITHMETIC MEAN)



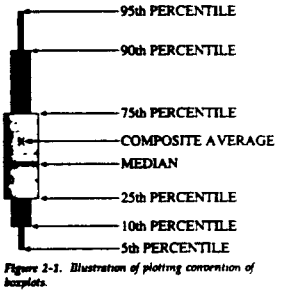
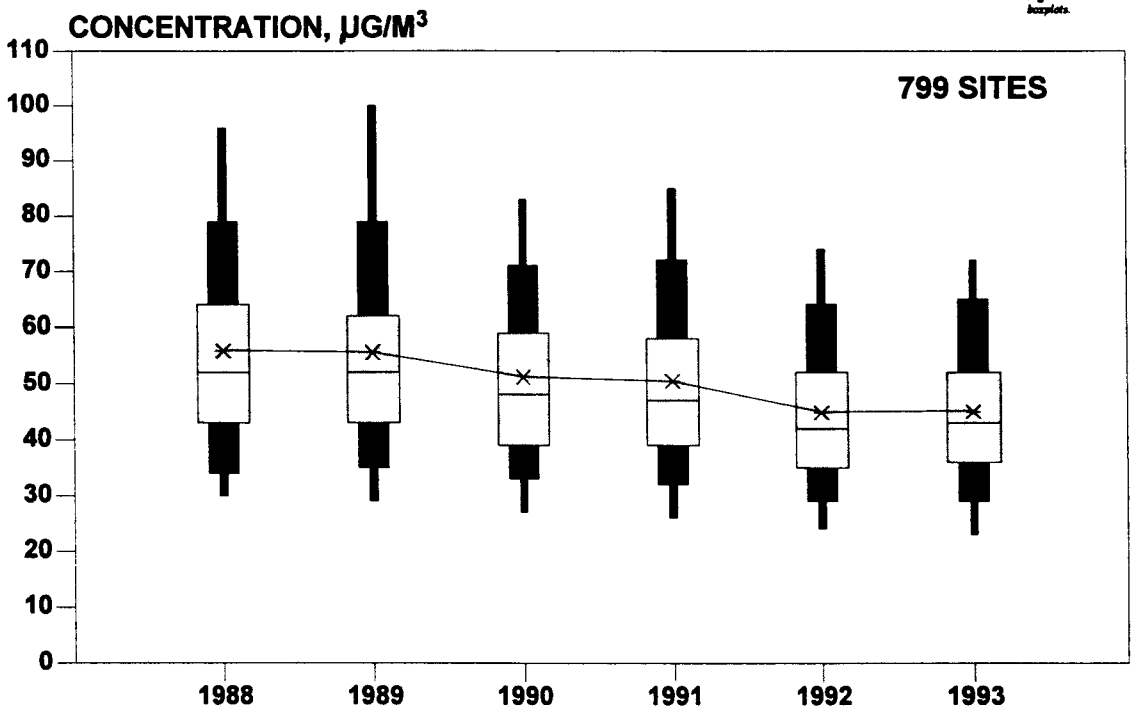


Figure 2-1. Illustration of plotting convention of boxplots.

FIGURE IV-5b. **PM-10 TREND, 1988-1993**
(90th PERCENTILE)



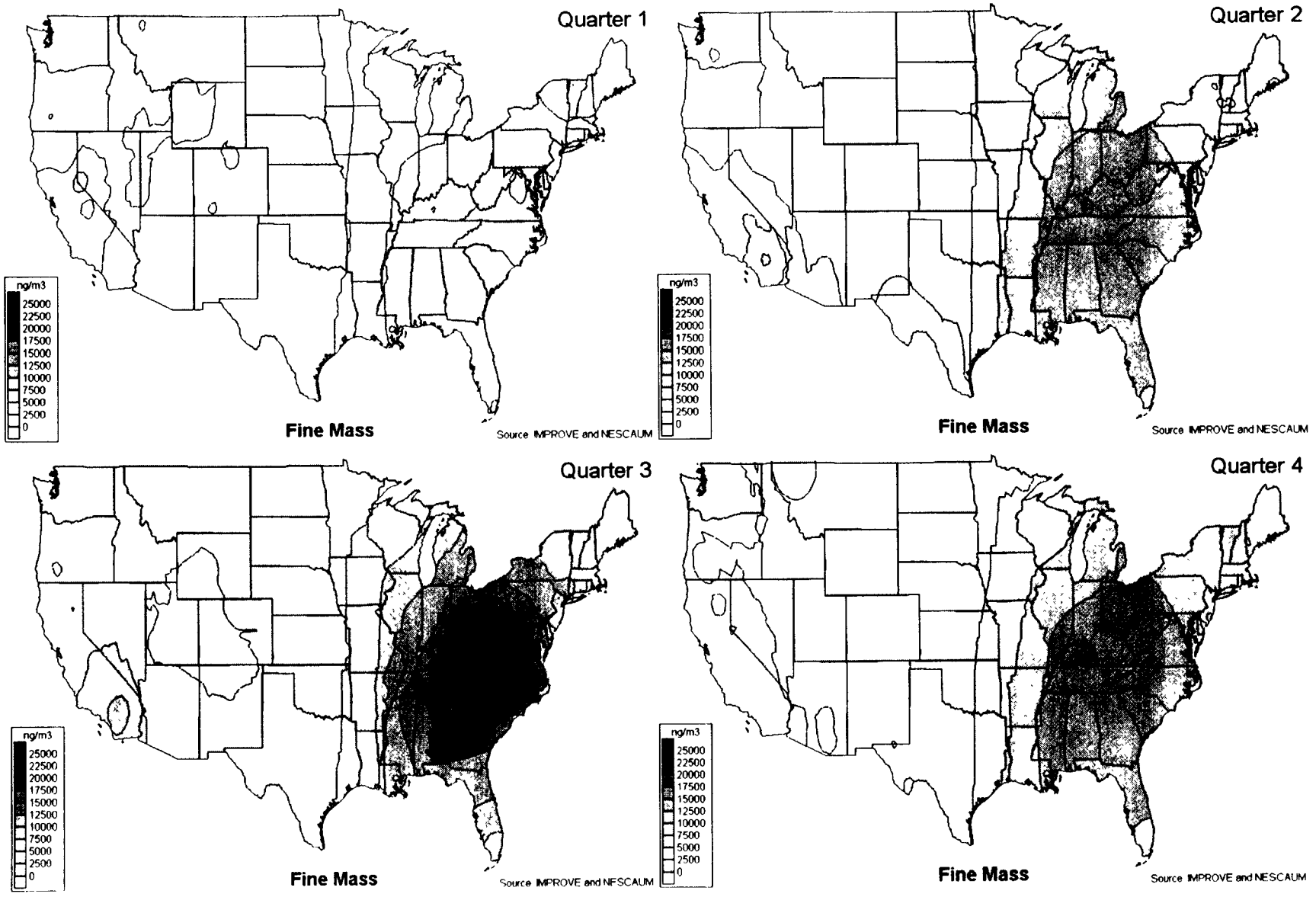


FIGURE IV-6. FINE MASS CONCENTRATION DERIVED FROM NONURBAN IMPROVE/NESCAUM NETWORKS. (CD, Figure 6-8).

California as well as a strong seasonality. In the Eastern U.S. high fine particle levels dominated by sulfates occur in the summer often in conjunction with elevated ozone levels.

National $PM_{2.5}$ trends are not available because of the limited number of sites measuring $PM_{2.5}$ and the sampling period at most sites is restricted to a few years. The development of national trends is further hindered because $PM_{2.5}$ is measured using a variety of sampling frequencies and a variety of non-standard sampling equipment (because there is currently no federal reference and equivalency program for $PM_{2.5}$).

However, visibility data can be used as a reasonable surrogate to estimate fine particle trends because the extinction coefficient (B_{ext}) is directly related to fine particle mass (CD, page 6-216). Sufficient visibility data are available to produce national trends from 137 U.S. sites (principally airports) since 1948 (CD, Section 6.10.2; NAPAP, 1991). The location of these sites reflects suburban and urban locations with airports. Figure IV-7 depicts trends maps for the 75th percentile extinction coefficient for summer and winter quarters. The figures show significant regional and seasonal trends. In the northeastern states, winter haze shows a 25 percent decrease while in the southeastern states, there is a 40 percent increase in winter haze (NAPAP, 1991).[9] The summer haziness in the Northeast shows an increase up to the mid-1970s followed by a decline. In the Southeast, there was an 80 percent increase in summer haziness, mainly occurring in the 1950s and 1960s (NAPAP, 1991). During the summer months, haziness (extinction coefficient) in the East can be dominated by sulfate (with associated water and ammonium). In this situation, visibility trends may be a better surrogate for sulfate than for non-sulfate related fine particle components (see subsection c below).

Visibility and fine particles have been monitored with more precision by the IMPROVE network from 1987 to present. In eastern remote locations, air quality data from 1982 to 1992 showed roughly a 3 percent annual increase in sulfate mass concentration during the summer and a smaller negative (although not statistically significant) trend in the winter (Eldred and

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[9] For the NAPAP analyses, the Northeast was defined as Indiana, Ohio, Pennsylvania, New York, Kentucky, West Virginia and New England states, and the Southeast was defined as states south of the Ohio River and east of the Mississippi (NAPAP, 1991).

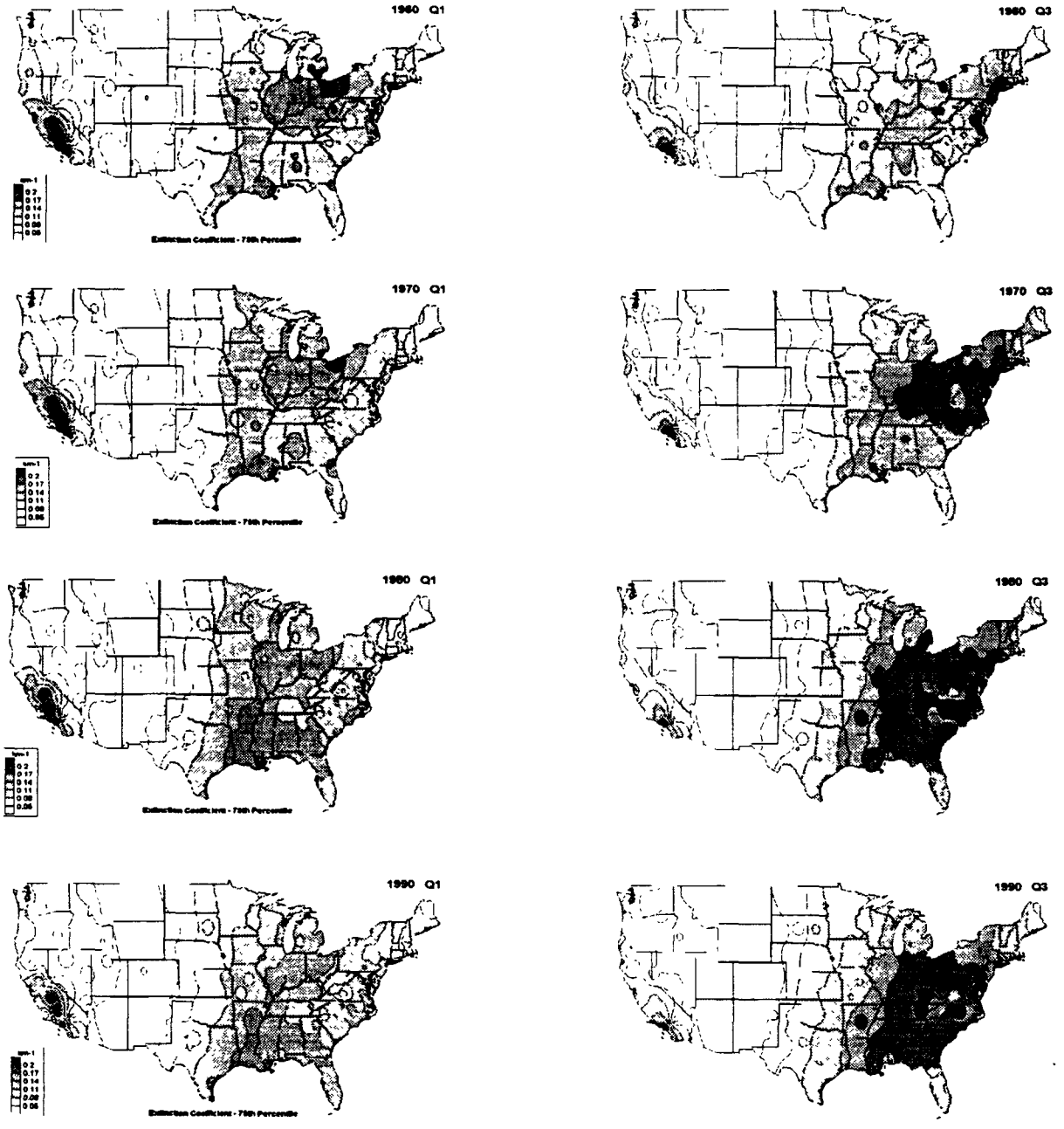


Figure IV-7. U.S. trend maps for the 75th percentile extinction coefficient, B_{ext} for winter (Q1) and summer (Q3) (after CD, Figure 6-112). B_{ext} (km^{-1}) is derived from visual range (VR) data by B_{ext}=3.9/VR. Data obtained under natural impairment conditions (i.e. rain, snow, fog) were eliminated. Because of the reationship between extinction and fine particle mass, these trends can be used to make some inferences about regional fine particle trends. As noted in the text and Figure IV-8, summertime visibility trends in the eastern U.S. are greatly influenced by the sulfate fraction of fine particles.

Cahill 1994). Western visibility monitoring through the IMPROVE network has not shown any trends for the period.

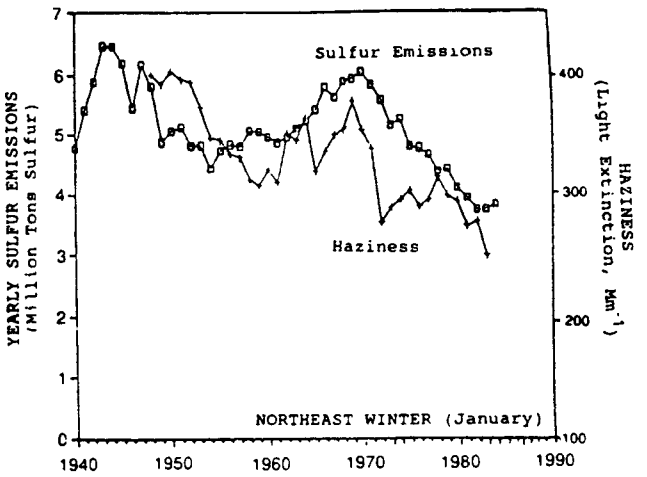
c. Trends in Emissions of Fine Particle Precursor Gases

SO_2, nitrogen oxides (NO_x), which encompasses NO and NO_2, and certain organic compounds are major precursors of secondarily formed fine particles, as described above. The relationship between precursor emission reductions and ambient $PM_{2.5}$ is nonlinear in many aspects; thus, it is difficult to project the impact on $PM_{2.5}$ arising from expected changes in PM precursor emissions without air quality simulation models that incorporate treatment of complex chemical transformation processes. In general terms, one would expect that emission reductions of SO_2 should lead to reductions in sulfate aerosol, but reductions will vary by season, depending on both emission fluctuations and changes in prevailing meteorology and photochemistry.

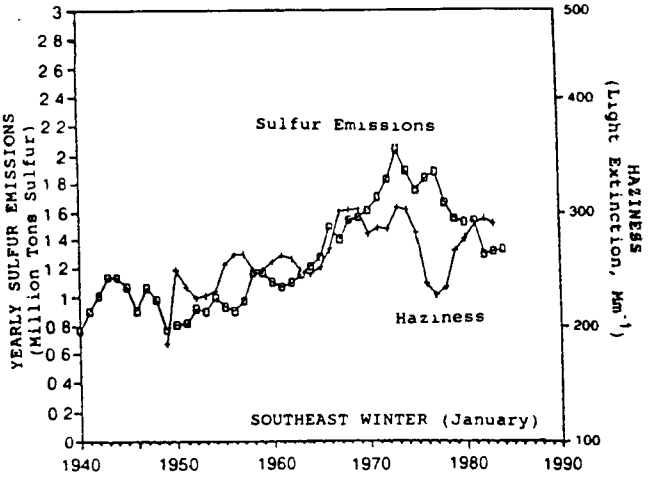
Figure VI-8 shows comparisons of sulfur emissions for summer and winter with extinction measurements derived from airport visibility data over the Northeast and Southeast in the winter and summer seasons where sulfates are currently the major contributor to light extinction (NAPAP, 1991). The correspondence between sulfur emissions and extinction coefficient is fairly close, particularly in the summer, but not an absolute match. For some years there are increases or decreases in extinction coefficient without corresponding changes in sulfur emissions, which likely reflect changes in non-sulfate particles as well as changes in meteorology and errors in emissions and visibility data. Overall, these data point to a strong relationship between sulfur emissions and regionally occurring fine particle concentrations in the Eastern U.S. (NAPAP, 1991).

It is noteworthy that major reductions in precursor emissions have occurred in the past, such as the large SO_2 reductions that were achieved in the 1970s and 1980s in some locations because of other CAA programs such as the SO_2 NAAQS implementation, prevention of significant deterioration (PSD) program, and later from the new source performance standards (NSPS) program. Similarly, NO_x emissions increases have been limited due to PSD, NSPS, and mobile source control programs. Future reductions in SO_2 of slightly less than 1 percent per year for the next 9 years are projected for the Eastern U.S., primarily from electric

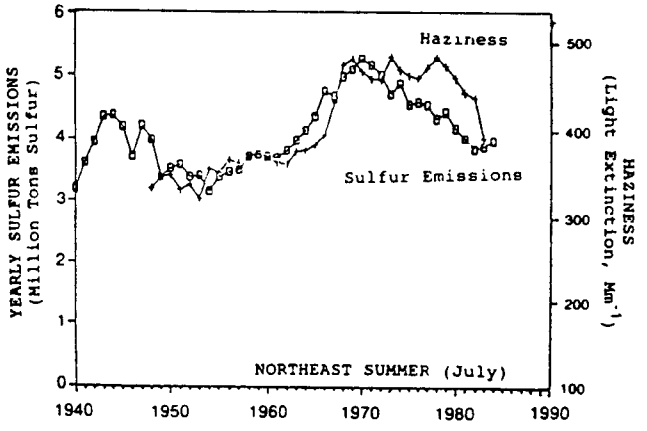
FIGURE IV-8. TRENDS IN VISIBILITY AND SULFUR EMISSIONS IN THE EASTERN U.S.



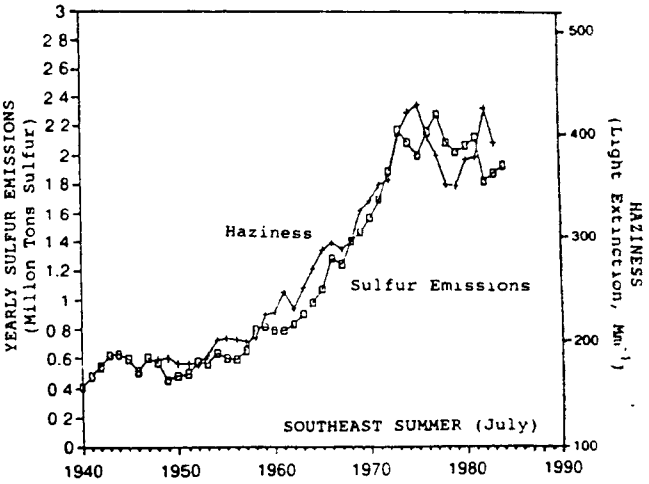
Comparison of sulfur emission trends (◊) and extinction coefficient (+) for the northeastern region during the winter months.



Comparison of sulfur emission trends (□) and extinction coefficient (+) for the southeastern region during the winter months.



Comparison of sulfur emission trends (◊) and extinction coefficient (+) for the northeastern region during the summer months.



Comparison of sulfur emission trends (□) and extinction coefficient (+) for the southeastern region during the summer months.

Source: NAPAP, 1991

utilities (U.S. EPA 1995b). These projected reductions are due to the Acid Deposition Program, as required under Title IV of the 1990 CAA Amendments. Substantial NO_x controls are also required for motor vehicles and utilities under the CAA Amendments.

2. Background Levels

Natural sources contribute to both fine and coarse particles in the atmosphere. 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 emissions of PM and precursor emissions of VOC, NO_x, and SO_x in North America. Estimating background concentrations is important for the health risk analyses presented in Chapter VI and the assessment of fine particle concentrations and visibility effects in Chapter VIII.

Background levels of PM vary by geographic location and season. The natural component of the background arises from physical processes of the atmosphere that entrain small particles of crustal material (i.e., soil) as well as emissions of organic particles and nitrate precursors resulting from natural combustion sources such as wildfire. In addition, certain vegetation can emit fine organic aerosols as well as vapor phase precursors or organic particles. Biogenic sources and volcanos also emit sulfate precursors. The exact magnitude of this natural portion of PM for a given geographic location can not be precisely determined because it is difficult to distinguish from the long-range transport of anthropogenic particles and precursors. Based on published reports that attempt to construct a representation of total PM mass from the sum of estimated natural contributions for the PM components noted above, the criteria document provides broad estimates of background PM levels for longer averaging times as shown in Table IV-3.

TABLE IV-3. PM_{10} AND $PM_{2.5}$ REGIONAL BACKGROUND LEVELS

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

Source: CD, page 6-44. The lower bounds of the above ranges are based on compilations of natural versus human-made emission levels, ambient measurements in remote areas, and regression studies using human-made and/or natural tracers (NAPAP, 1991; Trijonis, 1982). The upper bounds are derived from the multi-year annual averages of the "clean" remote monitoring sites in the IMPROVE network (Malm et al., 1994). It is important to note, however, that the IMPROVE data used here reflect the effects of background and anthropogenic emissions from within North America and therefore provide conservative estimates of the upper bounds.

As noted in the estimates, there is a definite geographic trend to these levels with the lower values applicable to the Western U.S. and the higher values applicable to 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 West.

The range of expected background concentrations on a short-term basis is much broader. Specific natural events such as wildfires, volcanic eruptions, and dust storms can lead to very high levels of PM comparable to or greater than those observed in polluted urban atmospheres. Because such excursions are essentially uncontrollable, EPA has developed an "natural events" policy that removes consideration of them from attainment decisions.[10] Disregarding such large and unique events, some estimate of the range of "typical" background on a daily basis can be obtained from reviewing various multi-year data as well as special field studies. On very clean days, IMPROVE daily measurements are less than 1 $\mu g/m^3$ of $PM_{2.5}$. On some days atmospheric conditions are more conducive to accumulation and formation of PM from both natural and anthropogenic emissions sources. Upper bound estimates of daily

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

background as high as 12 $\mu g/m^3$ PM_{10} have been made based on short-duration studies in remote "clean" areas of the Eastern U.S. (Wolff et al., 1983). Observed peak to mean ratios in natural areas over much longer time periods can provide a rough guide to the highest 24 hour levels arising from "routine" natural emissions and meteorology conducive to maximum particle accumulation. Because such meteorology appears prevalent in the Southeastern US, staff developed 24-hour peak to annual mean ratios for $PM_{2.5}$ data taken from the four Southeastern IMPROVE sites (Bachmann, 1996). If one assumes that the broad regional distribution of anthropogenic and natural sources of PM are somewhat similar, present day observed peak to mean ratios of 2 to 4 can be assumed to apply to the background annual values in Table IV-3. This estimation approach suggests that the highest background 24 hour $PM_{2.5}$ levels over the course of a year could be on the order of 15 to 20 $\mu g/m^3$.

C. Air Quality Implications for Interpreting Epidemiological Studies

Based on the examination of the substantial body of data, the CD concludes that the differences in exposure relationships alone of fine and coarse fraction particles are sufficient to justify the consideration of fine and coarse particles as separate classes of pollutants (CD page 13-94). The CD notes that the likelihood of ambient fine mode particles being significant contributors to PM-related health effects in sensitive populations (discussed in Chapter V of this Staff Paper) is related to the linkages between fluctations in outdoor concentrations of PM and personal exposure to outdoor PM, particularly in indoor environments where people spend most of their time and where many chronically ill elderly can be expected to spend all their time (U.S. EPA 1989a; Spengler et al., 1981). In this regard, while both fine and coarse fraction particles can penetrate indoors with similar efficiency (CD, Sections 7.2, 7.7, and 13.2.7; Wallace, 1996; Koutrakis et al., 1992; Lioy et al., 1990), once inside, the longer residence time of fine particles compared to coarse fraction particles enhances the probability of a linkage between fluctuations in outdoor concentrations and day-to-day population exposures for fine mode particles of outdoor origin, as compared to coarse fraction particles of outdoor origin (DRI, 1995; CD, Sections 7.6 and 13.2.7; Wallace, 1996; Anuszewski et al., 1992). In addition, the more uniform distribution of fine particles expected across many urban areas with regionally elevated concentrations and their well-correlated variation from

site to site within a given city mean that fine particle measurements at central monitors may provide a better indicator of day-to-day variations in potential exposure to outdoor particles (CD, Section 13.2.7; Burton et al., 1996; Wallace, 1996; Wilson and Suh, 1996).

1. Representativeness of Central Monitor Measurements of PM Exposures

The CD concludes that central monitoring can be a useful, if imprecise, index for representing the average exposure of people in a community to PM of outdoor origin (CD, Chapter 7; Tamura et al., 1996; Wallace, 1996; Tamura and Ando, 1994; Suh et al., 1993). Thus, for both the prospective cohort and time series epidemiological studies, it appears reasonable to use a representative central monitor or spatially averaged group of monitors to represent the mean community exposure to outdoor PM.

In addition, the CD concludes that fixed-station ambient PM measurements (e.g., PM_{10}, TSP) generally approximate total ambient fine particle exposure more closely than coarse fraction PM exposure (CD Chapter 13.4.3). Within the fine fraction, fixed-station measurements of ambient sulfates likely approximate total exposure to sulfates better than similar measurements of H^+ characterize total exposure to acidity because a higher proportion of $SO_4^=$ persists indoors (whereas, H^+ is neutralized by indoor ammonia). Thus, the CD concludes that on balance, available health effects estimates from community studies, whatever their magnitude and direction, are subject to more uncertainty for the coarse fraction than the fine mode, and for H^+ than for $SO_4^=$ (CD, page 13-52).

Individual personal exposures to PM can vary considerably from the concentrations measured at a monitoring station. Typically, in the U.S. PM personal exposure measurements are higher than the ambient PM concentrations due to indoor sources of particles such as cooking, smoking, and cleaning. Because of relative day-to-day consistency within any given residence of indoor sources and sinks of PM, the longitudinal (time series) correlation of personal exposure of a specific individual to total indoor PM_{10} (from both outdoor and indoor sources) and ambient PM_{10} can be very high. In homes with minimal indoor sources of PM_{10}, the R^2 values can range above 0.9 when these sources are consistent from day-to-day (CD, page 7-164).

The CD reports similar high correlations between personal and ambient values of sulfate in a cross-sectional exposure study (R^2 = 0.92 reported in Suh et al. (1993); CD, page 7-105). Similar high correlations for total sulfur were found by Ozkaynak et al. (1996) in the PTEAM study. These results are noteworthy because unlike PM_{10}, which has both indoor and outdoor sources, sulfate is virtually all of outdoor origin. Consequently, only the traits of the indoor environment, such as air conditioning, modify personal exposures to sulfates while indoors (CD, page 7-105). By contrast, the strength of cross sectional comparisons between total PM_{10} or $PM_{2.5}$ personal exposures and ambient concentrations can vary greatly depending upon the presence of smoking, cooking, or other strong indoor/personal sources (Wallace, 1996).

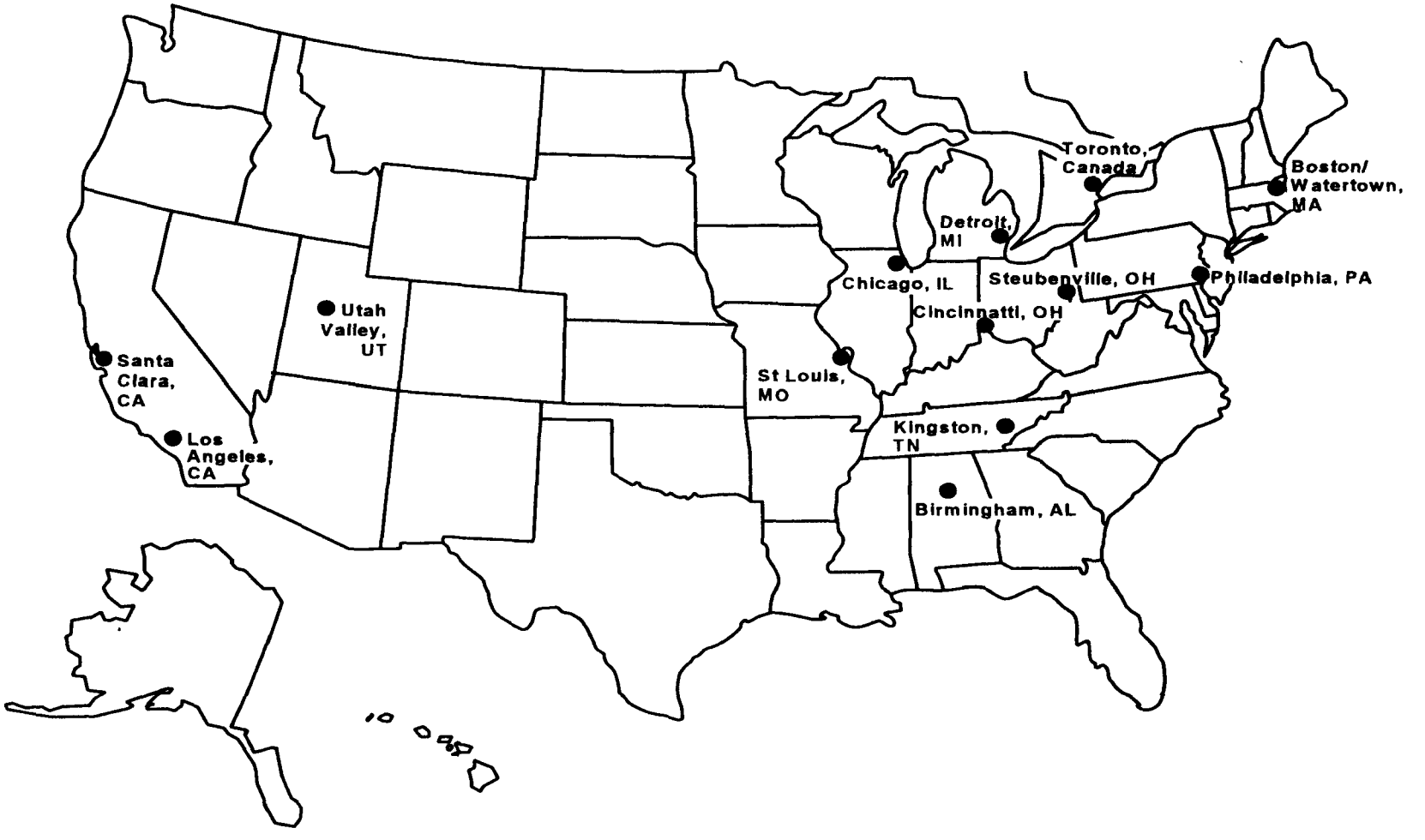
The day-to-day relationship between PM concentrations monitored at a central station and measurements of personal exposure is important to interpreting the time series community health studies. The CD notes that longitudinal exposure studies are more relevant to interpreting the time series epidemiologic studies than the cross-sectional exposure analyses because the cross-sectional studies often are more influenced by the variations in indoor sources (e.g., one household with a smoker and a smoke-free household) and sinks between subjects (CD, Section 7.4.2; Wallace, 1996). Cross-sectional regression analyses of indoor on outdoor $PM_{2.5}$ and PM_{10} concentrations generally explain less than half of the variance ($R^2 <$ 0.50); however, longitudinal regressions (for a single home measured over a series of days) often have much better indoor-outdoor relationships (R^2 ranging up to 0.9) (CD, Section 7.8).

Thus, the CD concludes that measurements of daily variations of ambient PM concentrations, as used in the time series epidemiologic studies presented in Chapter V, have a plausible linkage to the daily variations of human exposures to PM from ambient sources for the populations represented by the ambient monitoring stations (CD, Chapter 7). The CD concludes that this linkage will be better for indicators of fine particles than for indicators of fine plus coarse particles (i.e., PM_{10} or TSP).

2. $PM_{2.5}$ and PM_{10} Comparisons in Areas Relevant to the Health Studies

Figure IV-9 shows the locations of selected community health studies which reported positive, statistically significant associations between short-term exposure to PM and excess

FIGURE IV-9 Locations where community epidemiology studies associating short-term PM exposure with mortality were conducted in North America.



[1] Locations of PM studies using a variety of PM indicators (e.g. PM_{10} , $PM_{2.5}$, $SO_4^=$, TSP) and reporting statistically significant results (See CD tables 12-2 through 12-5)

mortality, which are discussed in Chapter V. Significantly, despite the fact that most of the PM_{10} non-attainment areas are mainly in the Western U.S. (see Figure IV-4), the mortality studies were conducted mainly in Eastern U.S. cities, many of which attain the current standards. The eastern sites where studies were conducted have a higher level of regional fine particles (as shown in Figures IV-6 and IV-7). Table IV-4 presents available information about fine particle concentrations in selected cities relevant to the health studies.

By contrast, the coarse fraction in the eastern U.S. is lower, on both an absolute concentration and relative fraction of PM_{10} basis than in the Western U.S. In the Eastern U.S., less than half of the daily PM_{10} mass concentration is coarse fraction material. The seasonal coarse fraction to PM_{10} ratios in the Northeast, for instance, range from 0.36 to 0.38, with an average of all seasons of 0.37 (SAI, 1996).

The Western U.S. has a more complicated pattern of fine and coarse particles because of its more complex mix of sources, topography, and seasonal variability. In some western urban areas, fine particle levels can be equal to or greater than those observed in the Eastern U.S. (see Table IV-4). Urban areas such as Los Angeles, CA, Utah Valley, UT, and Denver, CO, have relatively high contributions of local precursor emissions that may contribute to the formation of fine particles.

D. Air Quality Implications for Risk Management Strategies

Through the state implementation plan process, State and local agencies are responsible for adopting strategies to control PM in areas with violations of the PM NAAQS.[11] Conversely, areas that currently meet the PM_{10} NAAQS are not required to implement any controls. In non-attainment areas, the implementing agency typically selects control strategies based on its evaluation of which strategies are most effective at reducing PM_{10} concentrations contributing to an exceedance, considering the ability of the area or source to implement the controls and cost. Accordingly, implementing agencies take into account financial costs,

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[11] In moderate non-attainment areas, the CAA requires the application of reasonably available control measures (RACM) and the attainment of the NAAQS as expeditiously as practicable. The expeditiousness test requires the application of reasonably available control technology (RACT). EPA provides guidance on RACM/RACT. Under the guidance, States have flexibility in choosing the mix of controls used to attain the NAAQS.

TABLE IV-4. $PM_{2.5}$ CONCENTRATIONS IN SELECTED CITIES

LOCATION	MONITOR TYPES (Number)	YEARS OF DATA COLLECTION	TOTAL NUMBER OF OBSERVATIONS AT SELECTED MONITOR*	AVERAGE $PM_{2.5}$ ANNUAL MEAN ($\mu g/m^3$)	2nd HIGHEST $PM_{2.5}$ VALUE ($\mu g/m^3$)	YEAR OF 2nd HIGHEST VALUE
Boston, MA	SPM (1)	1986-88	193	19.2	55	1986
Detroit, MI	SPM (4)	1988-92	149	22.4	73	1989
Harriman, TN	SPM**	1980-87	1,481	20.8	-	-
Los Angeles-Long Beach, CA	SPM (2)	1988-89	90	32.0	88	1988
Minneapolis-St. Paul, MN	Unknown (1)	1986-87	98	13.0	38	1986
New York, NY	SPM (4)	1986-93	309	39.5	91	1988
Philadelphia, PA	SLAMS (1)	1986-91	249	20.9	47	1987
Portage, WI	SPM**	1979-87	1,436	11.2	-	-
Riverside-San Bernardino, CA	SLAMS (1) SPM (1)	1988-89	111	42.8	114	1989
Salt Lake City-Ogden, UT	Unknown (1) SLAMS (2)	1986-88	121	29.3	91	1988
St. Louis, MO	SPM (5) Unknown (1)	1985-93	44	16.0	49	1987
Steubenville-Weirton, OH-WV	SPM (4)	1990-91	51	25.7	81	1990
Topeka, KS	SPM**	1979-88	1,432	12.2	-	-

Key: SPM - Special Purpose Monitor
SPM** - Data from dichotomous virtual impactors reported in Schwartz et al. (1996a).
SLAMS - State + Local Air Monitoring System
*With multiple monitors in an area, monitor with highest in annual mean selected.

availability of technology, suitability of the measure to the specific problem, legal authority of the implementing agency over the emission source (e.g., local sources within a jurisdiction are normally controlled rather than sources of long range transport), and other factors. Because the current standards use a PM_{10} indicator, the extent to which any strategy controls fine or coarse particles is not currently a consideration. As long as the strategies adopted can be reliably demonstrated to provide for expeditious attainment of the standards, EPA does not require one specific measure over another in moderate non-attainment areas. Coarse fraction particles may be preferentially controlled because of their larger contribution to PM_{10} mass concentration in some areas, their local impact, and the relatively lower cost per ton removed.

Of the 83 PM_{10} nonattainment areas shown in Figure IV-4, 37 are eligible for redesignation to attainment, based on air quality data for 1992 to 1994, and an additional seven have preliminary data which suggest they may also be meeting the current standards. The implementation of the PM_{10} NAAQS encompasses diverse sources and solutions. The major sources contributing to PM non-attainment areas include fugitive dust, woodsmoke, stationary sources (e.g., including stacks and materials processing fugitive emissions from steel mills), and mixed areas (that may include the above sources plus additional sources such as regional transport or motor vehicles).

Table IV-5 presents additional information on the non-attainment areas and the progress towards attainment based on air quality data. Areas dominated by residential woodsmoke and stationary sources have made the most improvement to meet the PM NAAQS, as measured by the number of areas with improved air quality data. Areas with fugitive dust problems and mixed sources (most of which have a fugitive dust problem from activities such as construction and road dust as well as primary and secondary motor vehicle contributions and other sources) have made less progress because local areas with large mobile source contributions have difficulty reducing these emissions and areas with windblown fugitive dust problems are often unable or have limited ability to control the major sources of their problems from soil erosion.

TABLE IV-5. SUMMARY OF PM_{10} NON-ATTAINMENT AREAS BY SOURCE TYPE

Dominant Source Type	Number of PM_{10} Non-attainment Areas	Areas eligible for redesignation based on air quality data*	Difference
Fugitive Dust	23	5	18
Woodsmoke	32	20	12
Stationary Sources	23	12	11
Mixed Sources	5	0	5
Total	83	37	46

* Areas with complete data shown only. Implementing agencies must complete other requirements to be redesignated.

Although implementing agencies have no requirement to consider the relative contributions of fine and coarse particles to the control strategies adopted, national emission inventories and special studies provide some limited information about the relative contributions of fine and coarse fraction particles. Generally, fugitive dust sources tend to produce predominantly coarse fraction particles; residential woodsmoke is predominantly composed of fine particles; and stationary sources typically emit a mixture of fine and coarse fraction particles from a facility (U.S. EPA, 1995b).

Because of the heterogenous nature of the sources of PM_{10}, several different types of complex situations confront implementing agencies. Table IV-6 summarizes the relative contributions of PM_{10} sources and solutions in five areas typical of how successful implementing agencies have dealt with the PM_{10} NAAQS in each of the broader categories described above (Blais, 1996). The additional details in this table make apparent that even in a typical community affected mostly by fine particle residential woodsmoke such as Klamath Falls, OR, as much as 17 percent of the PM_{10} can be attributed to coarse fraction geological material prompting the implementing agency to take appropriate steps to curb these coarse PM_{10} emissions. Some mixed source areas may be able to meet the NAAQS by preferentially controlling the locally emitted coarse fraction particles without controlling fine particles.

The PM NAAQS program has not historically focused on the reduction of PM precursors to reduce PM concentrations except in a few special situations (e.g., Los Angeles,

TABLE IV-6. PM10 NAAQS IMPLEMENTATION CASE STUDIES SUMMARY

casesum2.wk4

Type	Location	Percent of Annual PM10 Concentration	Sources	Control Strategies	Predominant Fraction
Fugitive Dust	**Coachcella Valley, CA**	30	Windblown dust from erosion	No controls	C
		20	Windblown dust from human activities such as resuspension by vehicle traffic and suspension by construction, agricultural and recreational activities	Pave or chemically stabilize parking lots and unpaved roads and shoulders, limit vehicle speeds on unpaved roads, erection of windbreaks, street sweeping watering, revegetation, & restrictions on construction, demolition,& agricultural activities	C
	Highest daily average PM10 712 ug/m3 (in 1989)	13	Vegetative burning	Transferring waste to energy-conversion plant	F & C
	Annual Average PM10 90.2 ug/m3 (in 1989)	8	Motor vehicle emissions	Conversion of county's diesel bus fleet to natural gas	F
		7	Ammonium nitrate (transported from LA Basin)	No controls	F
		6	Ammonium sulfate (transported from LA Basin)	No controls	F
Woodsmoke	**Klamath Falls, OR**	73	Residential wood combustion	Woodstove replacement and burning bans	F
		17	Geological material	Replace highway sanding with liquid deicing, street sweeping, control of track out from unpaved roads and construction sites	C
	Highest daily average PM10 792 ug/m3 (in 1988)	2	Secondary aerosols	No controls	F & C
		2	*Vegetative burning*	*No controls*	F
		1	Industrial	No controls	F
Stationary Sources	**Steubenville, OH**	56*	Steel mills (stack and fugitive process emissions, fugitive dust from paved and unpaved roads, storage piles, and parking lots)	Increased chemical wet suppression of unpaved roadways; parking areas; raw material, scrap and slag separation, processing, & storage piles; enclosure of rail and truck unloading station; switch boiler fuel; add control equipment to blast furnace; vent blast furnace bleeder to boilers	F & C
	Highest daily average PM10 176 ug/m3 (in 1989)	32*	Electric utilities	No additional controls beyond Acid Rain program	F
	Annual Average PM10 45.4 ug/m3 (in 1989)	6*	Mobile sources	No additional controls	F
		6*	Road dust	No controls	C
		* based on Emissions Inventory estimates			
Western U.S. Mixed Sources	**Denver, CO**	35*	Utilities and industrial boilers (Ammonium nitrate & sulfate)	Restrictions on oil use, limits for NOx and SOx emissions	F
	Highest daily average PM10 189 ug/m3 (in 1987)	33*	Reentrained road dust	Switching to alternative materials; enhanced street sweeping	C
	Annual Average PM10 49 ug/m3 (in 1987)	7*	Residential wood combusion	Restriction on burning, conversion to cleaner heating technologies	F
		* apportionment from high concentration day			
Typical Eastern U.S. Attainment Area	**Philadelphia, PA**		Sources not characterized because area attains PM10 Standards	No additional controls	F & C
	Highest daily average PM10 97 ug/m3 (in 1989)				
	Annual Average PM10 40.3 ug/m3 (in 1989)				
	"Hot spot" PGW	--	Sorting scrap metal; processing slag from casting	Control of emissions from slag piles	C
		--	Melting, smelting, and refining; fugitive emissions from furnace	Enclose blast furnace (enforced via consent order, not SIP)	F
	Highest daily average PM10 567 ug/m3 (in 1993)				
	Annual Average PM10 110 ug/m3 (in 1994)		Source: Blais, 1996		

Key
F = Fine Mode C = Coarse Fraction

CA, and Provo, UT). Although the CAA requires consideration of secondary PM, implementing agencies are not required to control sources which are not within their non-attainment area or if source-receptor relationships are not established. Many non-attainment areas explicitly do not consider the control of secondary fine PM transported into their area from other sources (e.g., regional background from Ohio River Valley affecting Steubenville, OH, and secondary fine particles from LA Basin affecting Coachella Valley, CA). Instead, implementing agencies preferentially control locally generated coarse and fine fraction sources.

V. CRITICAL ELEMENTS IN THE REVIEW OF THE PRIMARY STANDARDS

A. Introduction

This chapter summarizes key information relevant to assessing the known and potential health effects associated with airborne PM, alone and in combination with other pollutants that are routinely present in the ambient air. A more comprehensive discussion of this information can be found in Chapters 10 - 13 of the Criteria Document (EPA, 1996). The presentation here organizes the key health effects information into those critical elements essential for the evaluation of current and alternative standards for PM. Specifically, this chapter summarizes: 1) key dosimetry information and hypotheses regarding mechanisms by which particles that penetrate to and deposit in various regions of the respiratory tract may potentially exert effects; 2) the nature of effects that have been reported to be associated with PM in community air, largely drawn from the more recent epidemiologic information, 3) the identification of sensitive populations and subgroups that appear to be at greater risk to the effects of community air containing PM; 4) issues raised in assessing community epidemiologic evidence on PM, including alternative interpretations of the evidence; and 5) evidence and alternative interpretations of the effects associated with the two major components of ambient PM_{10}, fine and coarse fraction particles.

The discussions of hypothesized mechanisms, effects, sensitive populations, and epidemiology include consideration of the full range of particle sizes and composition commonly found in urban and regional air. The PM epidemiological data base has greatly expanded since the last review, and suggests a variety of health effects are associated with ambient PM at concentrations extending from those found in the London episodes down to levels currently experienced in a number of U.S. cities (CD, p 13-1). Although a number of measures of PM have been used in such studies, based on an integrated assessment of the full range of laboratory and observational data, the revised CD and this staff assessment conclude that the ambient particles of greatest concern to health remain those smaller than 10 μm diameter. Accordingly, the discussion of effects, sensitive populations, and epidemiology highlights quantitative information on PM_{10}, but also includes some quantitative and qualitative information derived from studies of physical and chemical components of PM_{10}. Based on atmospheric considerations summarized here in Chapter IV and supporting health

evidence, the CD recommends separate consideration of the fine and coarse fractions of PM_{10}. The final section of this Chapter evaluates the extent to which the available quantitative and qualitative evidence might be used to support separate standards for the fine and coarse fractions of PM_{10}.

B. Mechanisms

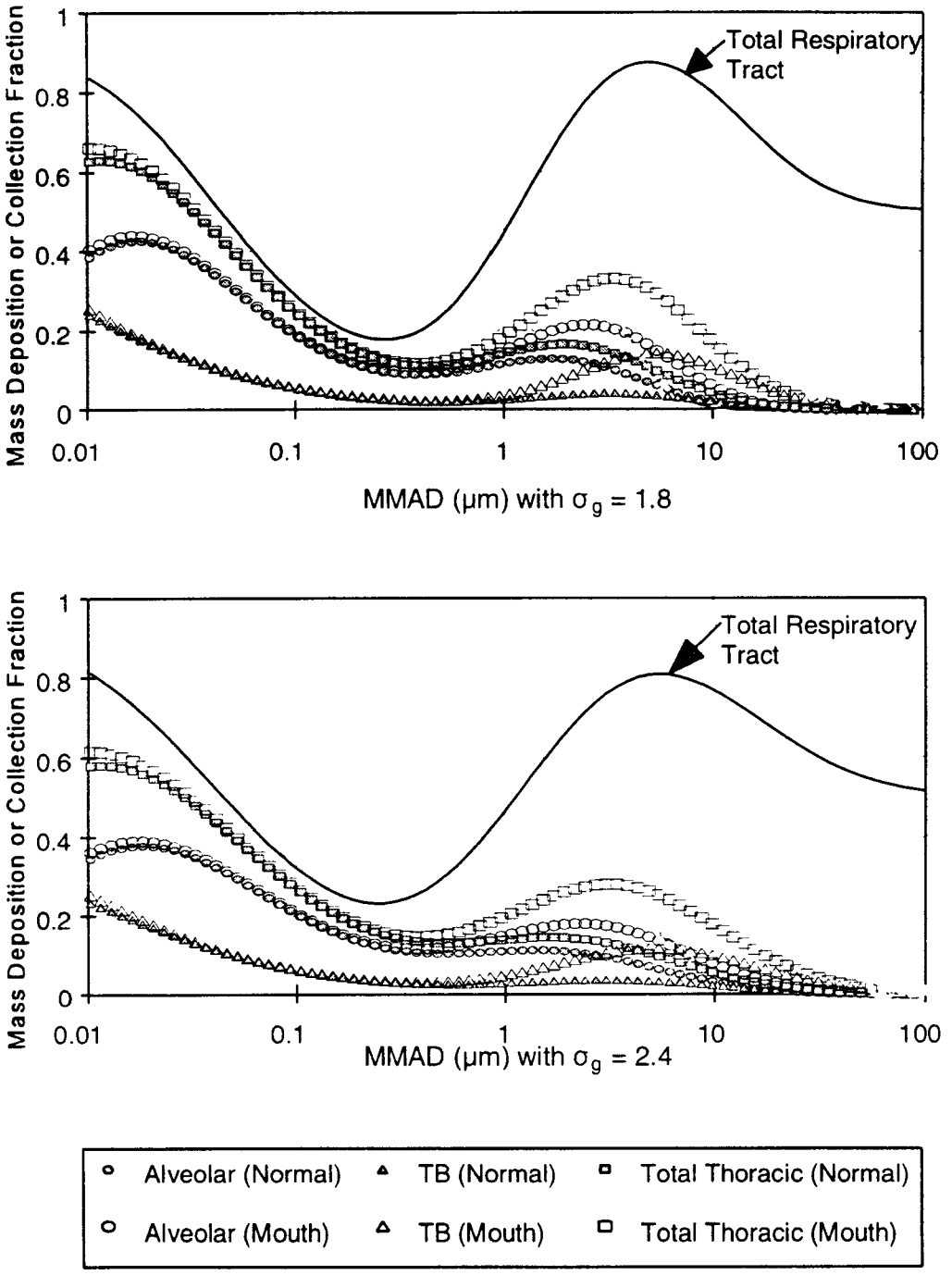
This section briefly summarizes available information concerning the penetration and deposition of particles in the respiratory tract and outlines hypothesized physiological and pathological responses to PM. It is important to emphasize that, at present, available toxicological and clinical information yields no demonstrated biological mechanism(s) that can explain the associations between ambient PM exposure and mortality and morbidity reported in community epidemiologic studies. Thus, any discussion of possible mechanisms linking ambient PM exposures to mortality and morbidity effects is necessarily limited to hypotheses derived from animal or human studies conducted at exposure levels of PM constituents far higher than found in ambient air. The major purposes of the discussion presented here is to identify available information of greatest relevance that helps identify those fractions of PM that are most likely to be of concern to health, to examine possible links between ambient particles deposited in various regions of the respiratory tract and reported effects in humans, and to focus attention on the kinds of mechanistic research needed to provide a biological basis for elucidating mechanisms that may provide support for a causal link between ambient PM exposures and reported health effects. An expanded treatment of key particle dosimetry considerations, potential mechanisms by which PM exposure is hypothesized to produce effects in humans at ambient exposure levels, and the limitations of the current human clinical and toxicological database can be found in Appendix D and in Chapters 10, 11, and 13 of the CD.

An evaluation of the ways by which inhaled particles might ultimately affect human health must take account of patterns of deposition and clearance in the respiratory tract. The human respiratory tract can be divided into three main regions: (1) extra-thoracic, (2) tracheobronchial, and (3) alveolar regions (CD, Table 10-1, Figure 10-5). The regions differ markedly in structure, function, size, mechanisms of deposition, and sensitivity or reactivity to deposited particles (U.S. EPA, 1982b, CD, Figure 10-6). The junction of conducting and

respiratory airways appears to be a key anatomic focus; many inhaled particles of critical size are deposited in the respiratory bronchioles that lie just distal to this junction, and many of the changes characteristic of emphysema involve respiratory bronchioles and alveolar ducts (Hogg et al., 1968). Retention of deposited particles depends on clearance and translocation mechanisms that vary with each of the three regions (See Appendix D). Coughing, mucociliary transport, endocytosis by macrophages or epithelial cells, and dissolution and absorption into the blood or lymph are important mechanisms of clearance in the tracheobronchial region. Endocytosis by macrophage or epithelial cells and dissolution and absorption into the blood or lymph are the dominant mechanisms of clearance in the alveolar region (CD, pp. 10-55, 56).

Figure V-1 illustrates the regional deposition of particle distributions of varying aerodynamic diameter. In essence, regional deposition of ambient particles in the respiratory tract does not occur at divisions clearly corresponding to the atmospheric aerosol distributions shown in Chapter IV. The CD provides simulations of deposition of ambient particle distributions that indicate fine and coarse particles are deposited in both the tracheobronchial and alveolar regions (CD, Chapter 10). Table V-1 provides estimated deposition patterns in the human lung for typical particle size distributions found in Philadelphia and Phoenix; these simulations are for adult males with normal breathing. The CD shows that as mouth-breathing or workload increases so does deposition in the bronchial and alveolar regions. For those individuals considered to be mouth breathers, deposition increases for coarse particles in the tracheobronchial region (CD, pp. 166-168).

Evidence from epidemiological studies of occupational and historical community exposures and laboratory studies of animal and human responses to simulated ambient particle components suggests that at exposures well above current standards, particles may produce physiological and ultimately pathological effects by a variety of mechanisms. The previous criteria and standards review included an integrated extensive examination of available literature on the potential mechanisms, consequences, and observed responses to particle deposition organized according to major regions of the respiratory tract (EPA, 1982b). Based on this assessment and the composition of typical urban PM, staff concluded, with CASAC concurrence



FigureV-1. Human respiratory tract PM deposition fraction versus mass median aerodynamic diameter (MMAD) with two different geometric standard distributions (σ_g = 1.8 or σ_g = 2.4). Alveolar, tracheobronchial, or total thoracic deposition fractions predicted for normal augmenter versus mouth breather adult male using a general population (ICRP66) minute volume activity pattern and the 1994 ICRP66 model. After CD, Figure 13-3.

TABLE V-1. MODELED 24-HR REGIONAL DEPOSITION FOR MEASURED AMBIENT PARTICLE SIZE DISTRIBUTIONS (After CD Tables 10-21, 23)*

City	Particle Fraction	Mode Size (MMAD)	Total Mass Deposition	Tracheobronchial Deposition	Alveolar Deposition
Philadelphia	Fine	0.436 μm	84 μg	9 μg	37 μg
	Coarse	28.8 μm	270 - 330 μg**	3 - 7 μg**	1-12 μg**
Phoenix	Fine	0.188 μm	42 μg	8 μg	26 μg
	Coarse	16.4 μm	440 - 530 μg**	10 - 15 μg**	12 - 29 μg**

*Results for normal breathing for adult males. Particle size distribution from impactor data. Total mass assumed 50 $\mu g/m^3$.

**Separate estimated deposition of "intermodal" peak of 2.3 to 2.6 μm in the original table is excluded for clarity, and because this peak may be an artifact of the sampling. Because it is possible that much of this mass (intermode) may be the "tail" of the coarse mode fraction, a range is given for coarse mode mass. The lower bound is the original estimate for the coarse mode. The upper bound is the sum of the estimates for the coarse model plus the intermode. This may tend to overstate coarse mode deposition relative to fine, which also contributes to the intermode.

(Friedlander, 1982), that particles that deposit in the thoracic region (tracheobronchial and alveolar regions), i.e. particles smaller than 10 μm diameter, were of greatest concern for standard setting. The staff identified a number of potential mechanisms and supporting observations by which common components of ambient particles that deposit in the thoracic region, alone or in combination with pollutant gases, might produce health effects (Table 5-2, EPA, 1982b). While there has been little doubt in the scientific community that the historical London air pollution episodes had profound effects on daily mortality and morbidity, no combination of the mechanisms/observations advanced in the last review has been sufficiently tested or generally accepted as explaining the historical community results. Moreover, as noted above, the potential mechanisms cited in the last review were based on insights developed from laboratory and occupational/community epidemiological studies that involved concentrations that are substantially higher than those observed in current U.S. atmospheres, and in many cases using laboratory generated particles that may be of limited relevance to community exposures.

As discussed in the CD, the significant body of new epidemiologic evidence that has accumulated since the last review of PM criteria and standards provides "evidence that serious health effects (mortality, exacerbation of chronic disease, increased hospital admissions, etc.) are associated with exposures to ambient levels of PM found in contemporary U.S. urban airsheds even at concentrations below current U.S. PM standards" (CD, p. 13-1). This increasing evidence has prompted renewed interest in generating testable hypotheses regarding potential mechanisms that might ultimately provide support for a causal link between health effects and particle exposure at these much lower levels. Table V-2 provides a very general summary of recent thinking concerning how particles may affect sensitive subpopulations as more fully discussed in the Criteria Document (CD, pp. 13-67 to 72, CD, pp. 11-179 to 185) and in Appendix D of this paper.

Because Table V-2 condenses and groups a number of hypotheses that have appeared in the literature and the CD in a summary fashion, several points should be noted. A complete definition of mechanisms of action for PM would involve description of the pathogenesis or origin and development of any related diseases or processes resulting in premature mortality; this is not currently possible. Some of the entries in the Table, on the other hand, may be more accurately described as intermediate responses potentially caused by PM exposure rather than complete mechanisms. The descriptions provide some rationale as to how such responses might conceivably contribute to the types of clinically relevant health endpoints reported in the literature, although evidence for action at low concentrations is presently lacking. It appears unlikely that the complex mixes of particles that are present in community air pollution would act alone though any single pathway of response. Accordingly, it is plausible that several responses might occur in concert to produce reported health endpoints. Some of the hypotheses in the Table may be more likely to be associated with effects from short-term rather than long-term exposure to PM, while others may relate to both. It is also important to note that a number of recent investigations have begun to examine promising new approaches involving new animal models, methods of concentrating ambient particles, and examination of the possibly more toxic constituents of PM such as ultra-fine particles and transition metals. This work, as well as future research, should provide important insights on mechanisms for the next standards review.

Table V-2. Hypothesized Mechanisms of PM Toxicity*

Response	Description
Increased Airflow Obstruction	PM exposure may aggravate existing respiratory symptoms which feature airway obstruction. PM-induced airway narrowing or airway obstruction from increased mucous secretion may increase abnormal ventilation/perfusion ratios in the lung and create hypoxia. Hypoxia may lead to cardiac arrhythmias and other cardiac electrophysiologic responses that in turn may lead to ventricular fibrillation and ultimately cardiac arrest. For those experiencing airflow obstruction, increased airflow into non-obstructed areas of the lung may lead to increased particle deposition and subsequent deleterious effects on remaining lung tissue, further exacerbating existing disease processes. More frequent and severe symptoms may be present or more rapid loss of function.
Impaired Clearance	PM exposure may impair clearance by promoting hypersecretion of mucus which in turn results in plugging of airways. Alterations in clearance may also extend the time that particles or potentially harmful biogenic aerosols reside in the tracheobronchial region of the lung. Consequently alterations in clearance from either disturbance of the mucociliary escalator or of macrophage function may increase susceptibility to infection, produce an inflammatory response, or amplify the response to increased burdens of PM. Acid aerosols impair mucociliary clearance.
Altered Host Defense	Responses to an immunological challenge (e.g., infection), may enhance the subsequent response to inhalation of nonspecific material (e.g., PM). PM exposure may also act directly on macrophage function which may not only affect clearance of particles but also increase susceptibility and severity of infection by altering their immunological function. Therefore, depression or over-activation of the immune system, caused by exposure to PM, may be involved in the pathogenesis of lung disease. Decreased respiratory defense may result in increased risk of mortality from pneumonia and increased morbidity (e.g., infection).

Cardiovascular Perturbation	Pulmonary responses to PM exposure may include hypoxia, bronchoconstriction, apnea, impaired diffusion, and production of inflammatory mediators that can contribute to cardiovascular perturbation. Inhaled particles could act at the level of the pulmonary vasculature by increasing pulmonary vascular resistance and further increase ventilation/perfusion abnormalities and hypoxia. Generalized hypoxia could result in pulmonary hypertension and interstitial edema that would impose further workload on the heart. In addition, mediators released during an inflammatory response could cause release of factors in the clotting cascade that may lead to increased risk of thrombus formation in the vascular system. Finally, direct stimulation by PM of respiratory receptors found throughout the respiratory tract may have direct cardiovascular effects (e.g., bradycardia, hypertension, arrythmia, apnea and cardiac arrest).
Epithelial Lining Changes	PM or its pathophysiological reaction products may act at the alveolar capillary membrane by increasing the diffusion distances across the respiratory membrane (by increasing its thickness) and causing abnormal ventilation/perfusion ratios. Inflammation caused by PM may increase "leakiness" in pulmonary capillaries leading eventually to increased fluid transudation and possibly to interstitial edema in susceptible individuals. PM induced changes in the surfactant layer leading to increased surface tension would have the same effect.
Inflammatory Response	Diseases which increase susceptibility to PM toxicity involve inflammatory response (e.g., asthma, COPD, and infection). PM may induce or enhance inflammatory responses in the lung which may lead to increased permeability, diffusion abnormality, or increased risk of thrombus formation in vascular system. Inflammation from PM exposure may also decrease phagocytosis by alveolar macrophages and therefore reduce particle clearance. (See discussions above for other inflammatory effects from PM exposure.)

*Summarization from the CD (p. 13-67 to 72; p. 11-179 to 185) and Appendix D of this document.

In conclusion, dosimetric information shows that both fine and coarse fraction particles smaller than 10 μm can penetrate and deposit in the tracheobronchial and alveolar regions of the lung. Particles also may carry other harmful substances with them to these regions with the smaller particles having the greatest surface area available for such transport (see section IV). While a variety of responses to constituents of ambient PM have been hypothesized to contribute to the reported health effects, there is no currently accepted mechanism(s) as to how relatively low concentrations of ambient PM may cause the health effects that have been reported in the epidemiologic literature. Therefore, there is an urgent need to expand ongoing research on the mechanisms by which PM, alone and in combination with other air pollutants, may cause adverse health effects.

C. Nature of Effects

The evidence for the kinds of health effects associated with exposures to PM comes from a large body of literature dating back more than 40 years. This section reviews and discusses the findings and conclusions concerning the principal health effects associated with PM exposure contained in the CD (CD, Chapters 11,12,13). Evidence for such conclusions and findings as well as for associations drawn from epidemiological studies, controlled human exposures, and animal toxicology is discussed and evaluated in the CD (CD, Chapters 11, 12, and 13), Appendix D of this document, and below. For reasons presented in the previous section, it is more likely that such effects are primarily related to particles smaller than 10 μm in diameter. Evidence with respect to the fine and coarse fractions of PM_{10} is discussed in Section V.F.

The scientific information discussed and evaluated in the CD and in this staff paper suggests that the key health effects categories associated with PM include:

- Increased Mortality
- Indices of Morbidity associated with Respiratory and Cardiovascular Disease
    - Hospital Admissions and Emergency Department Visits
    - School Absences
    - Work Loss Days
    - Restricted Activity Days

- Effects on Lung Function and Symptoms
- Morphological Changes
- Altered Host Defense Mechanisms

Most of the effects categories listed above have been consistently associated with PM exposure from a number of community epidemiological studies, with supporting insights from animal toxicology and controlled human exposures of various constituents of PM conducted at higher-than-ambient levels. Primary evidence of PM-related morbidity comes from indicators of aggravation of existing disease. In addition, while mechanisms of lung injury by particles have not been elucidated, there is agreement that the cardio-respiratory system is the major target.

Before discussing the effects, it is important to note some key characteristics and limitations of the kinds of studies used to identify them. The strengths and weakness of epidemiological studies in general are discussed in some detail in the CD throughout Chapters 12 and 13. While epidemiological studies alone cannot be used to demonstrate mechanisms of action, they can provide evidence useful in making inferences with regard to causal relationships, as in the case of cardiovascular disease and cigarette smoking (CD, Chapter 12). The CD discusses criteria for the use of epidemiological studies as an aid to inferring cause-effect relationships rather than merely establishing associations (CD, Section 12.1.2). It then reviews the criteria used to assess the scientific quality of epidemiological studies of community air pollution containing PM[1]. Particularly important issues and uncertainties for evaluation of the PM epidemiology studies are related to model specification, control for potential confounders, exposure misclassification, and consistency and coherence. These issues are discussed in detail in the CD and summarized here in Section 5.E.

Based on a comprehensive evaluation of the extensive published community data, the CD concludes that "the weight of epidemiologic evidence indicates that ambient PM exposure has affected the public health of U.S. populations" (CD, p. 13-27). As the CD points out,

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[1] Community air pollution refers to the mix of outdoor ambient PM and other pollutants that occur in typical urban/suburban atmospheres.

however, "little non-epidemiologic evidence is presently available to either support or refute a causal relationship (i.e., to construct an exposure-dose-response continuum) between low ambient concentrations of PM and increased morbidity and mortality risks" (CD, p. 13-27 to 28).

Under ideal circumstances, animal toxicology and controlled human exposure studies can provide qualitative and quantitative support for environmental epidemiology. In the case of PM, however, the lack of published experimental human and laboratory animal studies involving relevant exposure levels and experimental subjects representative of sensitive subpopulations identified in the epidemiological studies presents problems in providing an integrated assessment (CD, p 13-2). Epidemiological studies describe relationships between regionally and temporally variable mixtures of particles and gases in community air pollution and mortality and morbidity in sensitive populations -- most notably the elderly and individuals with cardiopulmonary disease, which includes adults and children with asthma. In contrast, experimental studies of PM effects in humans tend to use healthy young adult humans (or those with only mild disease) and examine mainly reversible physiologic and biochemical effects from exposure to laboratory-generated acidic aerosols, sulfates or nitrates. Similarly, experimental studies on laboratory animals have tended to use genetically homogenous healthy animals to examine a broader range of effects from individual components of the PM mix. In both animal and human studies, the limited number of individuals exposed greatly limits the ability to detect effects at concentrations close to ambient levels. In addition, extrapolation of quantitative and qualitative results from animal studies to human is encumbered by methodologic difficulties from differences in dosimetry. The various species used in inhalation toxicological studies do not receive identical doses in comparable respiratory tract regions when exposed to identical aerosols (see Appendix D). Consequently few laboratory experiments have used appropriate models of susceptibility to PM which limits evaluation of possible mechanisms and potential quantitative effects comparisons.

However, at least qualitative support for some of the epidemiologic observations has been reported for specific components of the ambient particle mix in controlled clinical studies of humans as well as studies in animals. For such studies, the biological responses

occurring in the respiratory tract following PM inhalation encompass a range of effects including: respiratory symptoms such as wheeze and coughing, changes in pulmonary function, altered mucocilary clearance, inflammation, changes in lung morphology and tumor formation (CD, p. 13-70, p. 11-1). In the vast majority of studies, however, results were observed only at concentrations of specific substances or simple mixtures that are significantly higher than those found in contemporary atmospheres. Because the health effects produced by PM exposure are dependent on the chemical composition, size, and concentration of particles, as well as species tested, these aspects of experimental paradigms used to characterize PM toxicity are noted in the following discussion. However, in this discussion, the emphasis is placed on reported effects of PM in general, rather than a specific emphasis on particle size or composition.

Key evidence illustrating each of the major effects categories listed above is outlined below, with an emphasis on the more recent information.

1. Mortality

a. Mortality From Short-Term Exposures to PM

i. Historical Findings From Community Epidemiology

The most notable reports of the health effects from community air pollution containing high PM have come from the dramatic pollution episodes of Belgium's industrial Meuse Valley (Firket, 1931); Donora, Pennsylvania (Schrenk et al., 1949); and London, England (Ministry of Health, 1954). In these cases, winter weather inversions led to very high particle concentrations in ambient air, which were associated with large simultaneous increases in mortality and morbidity (especially among individuals with preexisting cardio-pulmonary conditions). In a ten year follow-up study, survivors of the Donora, Pennsylvania pollution episode with either chronic disease prior to the episode, or those who became acutely ill during the episode, were found to have higher subsequent rates of mortality and illness (Ciocco and Thompson, 1961).

Analyses of a series of episodes in London indicated an excess of mortality (mostly from cardiopulmonary causes) occurred with abrupt increases in particles (including sulfuric acid) accompanied by simultaneously high levels of SO_2 (Martin, 1964; Martin and Bradley, 1960). Although the London studies measured PM as British Smoke (BS), gravimetric mass

calibrations permitted development of quantitative mass-concentration relationships. There was general acceptance in the 1982 CD (EPA, 1982a) and in critical reviews of PM-associated health effects (Ware et al, 1981; Holland et al, 1979) that London air pollution at high levels (at or above 500 - 1000 $\mu g/m^3$ of both pollutants) was causally related to increased mortality.

During the previous review of the PM standards, the London mortality studies were augmented by several more extensive time-series analyses examining the PM pollution/mortality relationship across 14 London winters (e.g, Mazumdar et al, 1982; Schwartz and Marcus, 1986; Ostro, 1984). These studies used more sophisticated statistical techniques to examine relationships between routine variations in PM and sulfur dioxide levels and mortality. Such analyses showed a continuum of response across the full range of PM levels in London and suggested that effects from exposure to PM occurred at levels more similar to those observed in the U.S.. Some of these studies suggested, although not conclusively, that particles were more likely to be responsible for the associations of health effects with air pollution than SO_2 (e.g., Mazumdar et al 1982). These studies and analysis of associations of health effects with the lower levels of PM measured in the 14 London winters (150 $\mu g/m^3$ as BS) was influential in the selection of the level of the current 24-hour PM_{10} standard (EPA, 1982b; 1986).

ii. Recent Findings

Beginning in 1987, two important developments took place. Investigators began to use more sophisticated statistical techniques, originally based on econometric techniques, to further evaluate the association between short-term variations in PM and mortality (CD, p 12-32). In addition the expansion of particle monitoring, related to the revision of the standard, increased the information concerning size-specific PM levels in cities throughout the U.S.. From 1987 to present, numerous epidemiological studies have reported statistically significant positive associations[2] between short-term exposures to PM and mortality. In these studies, investigators have observed statistically significant associations between

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

increased daily or several-day average concentrations of PM (as measured by a variety of indices: TSP, PM_{10}, $PM_{2.5}$, COH, KM, and BS) and excess mortality in communities across the U.S. as well as in Europe and South America. Of 38 studies published between 1988 and 1996, most found statistically significant associations between increases in ambient PM concentration and excess mortality (CD, Table 12-2). These studies are consistent with the earlier analyses of the London winters, but extend the association to lower concentrations for a large number of areas with differing climate, aerosol composition, and amounts of co-occurring gaseous pollutants such as SO_2 and O_3.

Table V-3 presents a comparison of relative risk estimates reported for PM-related mortality expressed in terms of a PM_{10} increment. A generally consistent association is found between changes in PM_{10} levels and mortality in most of these studies, with a range of 2 percent to 8 percent increase in daily mortality for a 50 $\mu g/m^3$ increase in PM_{10} for those with statistically significant results. In the studies with statistically significant results, mean PM_{10} concentrations ranged from 18 to 58 $\mu g/m^3$ and maximum daily concentrations from 80 to 365 $\mu g/m^3$. These studies were conducted in a number of different geographic locations in North America. Each of these locations differ significantly in pollution and weather patterns. Yet most of these studies finds a statistically significant association between increased mortality and PM_{10} that is relatively consistent across the studies. It is of note that a rough estimate of the relative risk for a 50 $\mu g/m^3$ increase in PM (as PM_{10}) for the 1952 episode in London (1.06) is in the range of those reported for the recent studies (Schwartz et. al., 1994).

iii. Specific Causes of Mortality Associated with PM

Table V-4 summarizes the relative risks for total mortality, respiratory and cardiovascular causes of death, and mortality among the elderly for the community studies evaluating cause of death. Reported cases of "respiratory related" deaths were assigned to individuals who had been diagnosed with acute respiratory illness (e.g., symptoms involving the upper respiratory tract and pneumonia), as well as COPD and pneumoconioses when they died. In general, these studies reported stronger significant relationships between short-term PM concentrations and deaths in those with respiratory and cardiovascular disease than for

Cover illustration: Locations of recently published community epidemiology studies finding statistically significant associations between short-term concentrations of particulate matter and health effects (CD, Tables 12-2 through 12-5). Studies conducted on three continents have found both increased morbidity and mortality to be associated with a variety of particle measurement devices, including mass measurements of TSP, PM10, PM2.5, sulfates, and acids, and optical based approaches including BS, KM, and COH. Although the highest PM-10 concentrations in the U.S. are in the West, most of the results in North America are from eastern communities, at PM-10 concentrations that are generally below those permitted by the current standards.

TABLE V-3. ESTIMATED MORTALITY INCREASE PER 50 $\mu g/m^3$ INCREASE IN 24-h PM_{10} CONCENTRATIONS FROM U.S. STUDIES (After CD, Table 13-3)

Study Location	RR (± CI) Only PM in Model	RR (± CI) Other Pollutants in Model	Reported PM_{10} Levels Mean (Min/Max)[†]
Increased Total Acute Mortality			
Six Cities[a]		—	
Portage, WI	1.04 (0.98, 1.09)	—	18 (±11.7)
Boston, MA	1.06 (1.04, 1.09)	—	24 (±12.8)
Topeka, KS	0.98 (0.90, 1.05)	—	27 (±16.1)
St. Louis, MO	1.03 (1.00, 1.05)	—	31 (±16.2)
Kingston/Knoxville, TN	1.05 (1.00, 1.09)	—	32 (±14.5)
Steubenville, OH	1.05 (1.00, 1.08)	—	46 (±32.3)
St. Louis, MO[c]	1.08 (1.01, 1.12)	1.06 (0.98, 1.15)	28 (1/97)
Kingston, TN[c]	1.09 (0.94, 1.25)	1.09 (0.94, 1.26	30 (4/67)
Chicago, IL[h]	1.04 (1.00, 1.08)	—	37 (4/365)
Chicago, IL[g]	1.03 (1.02, 1.04)	1.02 (1.01, 1.04)	38 (NR/128)
Utah Valley, UT[b]	1.08 (1.05, 1.11)	1.19 (0.96, 1.47)	47 (11/297)
Birmingham, AL[d]	1.05 (1.01, 1.10)	—	48 (21, 80)
Los Angeles, CA[f]	1.03 (1.00, 1.055)	1.02 (0.99, 1.036)	58(15/177)

References:

[a]Schwartz et al. (1996a).
[b]Pope et al. (1992, 1994)/O_3.
[c]Dockery et al. (1992)/O_3.
[d]Schwartz (1993).
[e]Ito and Thurston (1996)/O_3.
[f]Kinney et al. (1995)/O_3, CO.
[g]Styer et al. (1995).

[†]Min/Max 24-h PM_{10} in parentheses unless noted otherwise as standard deviation (± S.D), 10 and 90 percentile (10, 90). NR = not reported.
[*]Means of several cities.

TABLE V-4. COMPARISON OF TOTAL MORTALITY WITH AGE- AND CAUSE-SPECIFIC MORTALITY FOR SHORT-TERM EXPOSURE STUDIES

Study	Total Mortality, Relative Risk per 50 $\mu g/m^3$ PM10	Age- and Cause-specific Mortality per 50 $\mu g/m^3$ PM10
Respiratory Related		
Utah Valley, Pope et al. (1992)	1.08 (1.05 - 1.11)	1.20 (1.11 - 1.29)
Chicago, Styer et al. (1995)	1.04 (1.00 - 1.08)	1.12 (0.99 - 1.26)
Chicago, Ito and Thurston (1996)	1.03 (1.01, 1.04)	1.07 (1.02, 1.12)
Birmingham, Schwartz (1993)*	1.05 (1.01 - 1.10)	1.08 (0.88 - 1.32)
Santiago, Chile, Ostro et al. (1995a)	1.04 (1.035 - 1.06)	1.06 (1.03 - 1.10)
Elderly		
Chicago, Styer et al. (1995)	1.04 (1.00 - 1.08)	1.08 (1.03 - 1.13)
Santiago, Chile, Ostro et al. (1995a)	1.04 (1.035 - 1.06)	1.05 (1.03 - 1.06)
Cardiovascular Related		
Utah Valley, Pope et al. (1992)	1.08 (1.05 - 1.11)	1.09 (1.02 - 1.17)
Chicago, Styer et al. (1995)	1.04 (1.00 - 1.08)	1.03 (0.98 - 1.09)
Chicago, Ito and Thurston (1996)	1.03 (1.01 - 1.04)	1.02 (1.00 - 1.03)
Birmingham, Schwartz (1993)	1.05 (1.01 - 1.10)	1.08 (1.02 - 1.14)
Santiago, Chile, Ostro et al. (1995a)	1.04 (1.035 - 1.06)	1.04 (1.02 - 1.06)

* The Schwartz (1993) study was of COPD.

other conditions, as well as a larger effect in the elderly (>65) than in the general population (CD, Chapter 12; Styer et al., 1995; Ostro, 1995a; Schwartz, 1994a; Pope et al., 1992). The CD notes that the relative risk for respiratory-related mortality was up to 4.3 times as large as that for total mortality (CD, p. 12-77). As noted in the CD, such results are supportive of the biological plausibility of a PM/air pollution effect on mortality.

iv. Experimental Animal Studies

The vast majority of studies examining short-term exposures to animals of components of PM have found mortality only at concentrations well above ambient levels of PM, even in sensitive species (e.g., guinea pig). Such studies appear to be of little relevance to the effects observed in humans at ambient levels (CD, Table 11-18, p. 11-42,43).

b. Mortality From Long-Term Exposures to PM

Prior to 1990, cross sectional studies were generally used to evaluate the relationship between mortality and long-term exposure to PM. These, as well as more recent cross-sectional studies, are summarized in Tables 12-14 and 12-15 in the CD. These studies have reported, for at least one of the experimental designs used in each study, statistically significant positive associations linking higher long-term concentrations of various indices of PM with higher mortality rates across communities. However, absent other supporting evidence, the unaddressed confounders and methodological problems inherent in these studies have limited their usefulness. The previous staff paper concluded that such studies provided only suggestive evidence of long-term mortality associated with PM exposure (EPA, 1982b). In the recent literature, however, new prospective cohort studies have reported results that may lend additional support to the earlier results. These studies use subject-specific information and appear to provide more reliable findings (CD, section 13.4.1.1), although the uncertainties in controlling for a number of factors such as smoking, lifestyle, and exposure patterns are improved by the design of cohort studies, they remain greater than for short-term studies conducted in single communities. The results of three recent studies (Abbey et al., 1991; Dockery et al., 1993; Pope et al., 1995) are summarized in Table V-5 and described briefly below.

Dockery et al., (1993) analyzed survival of 8,111 adults followed for 14 years in six cities in the eastern U.S. (Six City Study). Extensive information was obtained regarding

TABLE V-5. RELATIVE RISK BETWEEN THE MOST POLLUTED AND LEAST POLLUTED CITIES FOR TOTAL POPULATION AND FORMER AND CURRENT SMOKERS IN THE PROSPECTIVE COHORT STUDIES

A) Harvard Six City Study, Dockery et al. (1993)

Endpoint	Total Population RR*	Non-Smokers RR*	Former Smokers RR*	Current Smokers RR*	No Occupational Exposure RR*
Total Mortality	1.26 (1.08 - 1.47)	1.19 (0.90 - 1.57)	1.35 (1.02 - 1.77)	1.32 (1.04 - 1.68)	1.17 (0.93 - 1.47)
Cardiopulmonary Disease	1.37 (1.11 - 1.68)	---	---	---	---
Lung Cancer	1.37 (0.81 - 2.31)	---	---	---	---

The results (and 95 percent confidence intervals) were reported in the paper between the city with the highest level of $PM_{2.5}$ (Steubenville, OH, average 29.6 $\mu g/m^3$) and the lowest level of $PM_{2.5}$ (Portage, WI, 11.0 $\mu g/m^3$).

B) American Cancer Society Study, Pope et al. (1995)

Endpoint	Total Population RR**	Non-Smokers RR**	Current and Former Smokers RR**
Total Mortality	1.17 (1.09 - 1.26)	1.22 (1.07 - 1.39)	1.15 (1.05 - 1.26)
Cardiopulmonary	1.31 (1.17 - 1.46)	1.43 (1.18 - 1.72)	1.24 (1.08 - 1.42)
Lung Cancer	1.03 (0.80 - 1.33)	0.59 (0.23 - 1.52)	1.07 (0.82 - 1.39)

The results (and 95 percent confidence intervals) were reported in the paper between the city with the highest and the lowest level of $PM_{2.5}$ of the 47 cities examined.

* Per 18.6 $\mu g/m^3$ increase in $PM_{2.5}$.

**Per 24.5 $\mu g/m^3$ increase in $PM_{2.5}$.

potential confounders for each individual, including, smoking, education level, and occupation. After adjustment for these co-variates, the authors found elevations in several measures of long-term PM concentration ($PM_{15/10}$, $PM_{2.5}$ and sulfates) were significantly associated with increases of total mortality. The adjusted increase in risk (26 percent, CI of 8-47 percent) from PM exposure was nearly equal for $PM_{15/10}$, $PM_{2.5}$ and sulfates between the cities with highest and lowest levels of air pollution.

A second prospective cohort study was conducted by Pope et al. (1995) which used 7-year survival data, between 1982 and 1989, for over half a million adults in 151 U.S. cities [American Cancer Society (ACS) study]. This study was designed to follow-up on the suggestion made from the Six City study that long-term exposure to fine particles is associated with increased mortality. To test this hypothesis, the association between multi-year concentrations of two fine particle indicators, sulfates and $PM_{2.5}$, and mortality was evaluated. As in the Six City study, information for each individual was used to adjust for important risk factors, such as age, sex, race, smoking, passive smoking, and occupation. After adjustment for the other risk factors, $PM_{2.5}$ concentrations were found to be associated with a 17 percent (CI of 9-26 percent) increase in total mortality, with sulfate concentrations associated with a 15 percent (CI of 5-26 percent) increase in total mortality, between cities with the least and most polluted air.

The Six City study found somewhat higher RR estimates for mortality than the ACS study. The sensitivity of the RR estimates to important confounders can be assessed by evaluating the effects estimates for different subgroups of the populations (Table V-5). Two subgroups in this population with high potential for confounding are smokers and those with occupational exposures to PM. With regard to smokers, both the Six City and ACS studies evaluated the association between fine particle levels and total and cause-specific mortality by smoking status. The ACS study compared the risk of mortality associated with PM separately for those who never smoked and those who have at one time smoked. The Six City study compared risk of mortality associated with exposure to fine particles for the total population, former smokers, current smokers, and nonsmokers. All categories showed elevated risk; only the non-smoking category failed to achieve statistical significance. The ACS study, which had a much larger population and consequently greater statistical power,

found a statistically significant association with total mortality and nonsmokers as well as for the total population and current and former smokers. It is possible that the RR estimates are sensitive to specification of smoking and occupational exposure, and as such adjusting for these variables in the Six City study may have been inadequate to fully capture the potential confounding from these variables.

The Six City study also evaluated the RR of mortality for the population non-occupationally exposed, defined as those who report no exposure to gases, fumes or dust. The RR for non-occupationally exposed individuals similar to that for non-smokers, but also did not achieve statistical significance. The ACS study did not evaluate the occupational subgroup separately. However, the authors note that the RR was not sensitive to the inclusion of occupational exposure variables after adjusting for cigarette smoking.

Some reviewers have raised concerns regarding the adequacy of the adjustment for confounders in the prospective cohort studies, maintaining that other uncontrolled factors may well be responsible for the observed mortality rates (Lipfert, 1995; Moolgavkar and Luebeck, 1996; Moolgavkar, 1994). In particular, these authors have suggested that the Six City Study did not control adequately for smoking and other factors. However, both the Six City Study and the ACS study evaluated the association between PM and mortality among never smokers and found relative risks that were similar in magnitude, and for the much larger population in the ACS study, statistically significant. Lipfert (1995) evaluated the Six Cities using State average sedentary lifestyle data. Based on this evaluation, he suggested that much of the mortality associations in the Six Cities might be explained by this additional factor, if it had been included in the original study. Aside from the fact that such State average data suffers from the same problems that have plagued past cross-sectional analyses, both the Six City Study and the ACS study adjusted for body mass index as well as other factors using individual specific data that should provide adjustments that are related to sedentary lifestyle. The CD notes that it is unlikely that these studies overlooked plausible confounders, although the addition of unaccounted factors might well alter the magnitude of the association (CD, 12-180).

Both the Six City and the ACS studies evaluated specific causes of mortality associated with PM (Table V-5). As with the short-term studies, the increase in risk of

mortality associated with PM was mostly attributed to increases in mortality from cardiopulmonary causes. The Six City study reported a 37 percent (CI of 11-68 percent) increase in mortality from cardiopulmonary causes associated with $PM_{2.5}$ levels, after adjusting for covariates, between the most polluted and least polluted city. Similarly, the ACS study reported a 31 percent (CI of 17-46 percent) increase in such mortality associated with $PM_{2.5}$ levels, after adjusting for covariates, between the most polluted and least polluted city. Taken together, the ACS study and the Six City study did not find any other statistically significant associations between PM levels and specific causes of mortality other than from cardiopulmonary causes.

Neither study showed any statistically significant increase in risk for lung cancer associated with undifferentiated fine PM exposure, although the ACS study found a significant association with sulfates. While earlier studies provided some evidence suggestive of an association of increased cancer at high PM exposure levels, the 1982 CD could not draw any conclusions with regard to such an association. Thus, there continues to be little epidemiological evidence for an effect of ambient PM on cancer rates. Evidence of potential cancer risk from specific particulate matter components comes from laboratory studies. Polycyclic aromatic hydrocarbons (PAHs), commonly found as combustion products, are perhaps the best studied class of potential carcinogens in PM. Extracts of organic material from particle emissions have been shown to induce tumors in a variety of studies (CD, p. 11-123). Extrapolation to human risk from such studies are difficult because of different species and age, route of exposure (e.g., not inhalation assays in animals), physico-chemical properties of the material, and exposure concentration. In any event, no clear evidence of sulfates acting as a carcinogen have been reported in the toxicological literature in the CD.

A third prospective cohort study of about 6,000 white, nonhispanic, non-smoking long-term residents of California (Abbey et al., 1991, California Seventh Day Adventist Study), did not find a significant association between total mortality and TSP. However, this study has more limited statistical power than one of the other two studies because of the smaller number of deaths (4 percent of deaths reported in the ACS study). More importantly, the PM indicator (days of high TSP) is of questionable usefulness as an indicator of levels of exposure to PM_{10} or $PM_{2.5}$, particularly for cohorts residing in various

locations in California. Cohorts classified with equivalent TSP exposure could experience varying exposures to fine and coarse fraction particles. For example, frequently high TSP exposures to cohorts near the South Coast could have less days of exposure to fine particle smog, while other cohorts could have similar high TSP exposures from dust storms.

The CD concludes that the Six City study and the ACS study, taken together with the earlier cross-sectional studies, suggest possible increases in mortality for specific disease categories that are consistent with long-term exposure to airborne particles. Moreover, as discussed in Chapter 13 of the CD and below, at least some fraction of these deaths likely reflect cumulative PM impacts above and beyond those seen from acute exposures (CD, p. 13-34). To the extent that this is true, additional caution must be used in interpreting these studies because some of the effects may be due to historical exposures that are significantly higher than those used as an index of population exposures in these studies.

c. Extent of Life Shortening

An important consideration in evaluating mortality effects in a public health context is the potential shortening of lifespan ("mortality displacement" or "prematurity of death") associated with PM exposure in these studies. Epidemiological findings suggest ambient PM exposure affects mortality both in the short and long term, and promotes potentially life-shortening chronic illness in the long term (CD, p. 13-44). The relative risk estimates from the PM mortality cohort studies are considerably larger (Dockery et al, 1993) to somewhat larger (Pope et al, 1995) than those from the daily mortality studies, suggesting that a substantial portion of the deaths associated with long-term PM exposure may be independent of the daily deaths associated with short-term exposure (CD, p. 13-44).

Information concerning life shortening of only a few days comes from the daily time-series studies. These studies indicate greater incidence and severity of effects are associated with PM exposure in vulnerable individuals, primarily the elderly (i.e., 65 years of age or older) and individuals with preexisting respiratory disease. Thus, it is reasonable to expect that some of the mortality associated with short-term pollution is occurring in the weakest individuals who might have died within days even without PM exposure ("harvesting effect"). Such a pattern is often seen for some other environmental insults, such as high

temperature (Kalkstein, 1991). However, direct evidence from short-term PM exposure studies concerning the degree of mortality displacement observed is limited (CD, p. 13-44).

The CD cites only two studies, Spix et al. (1993) and Cifuentes and Lave (1996), that have attempted to quantitatively test this hypothesis. Their analyses are based on the premise that if short-term "harvesting" is occurring, an observed increase in mortality on a day with high pollution should result in a corresponding decrease in mortality in subsequent days. The analysis by Spix et al. suggests a small portion of the PM-associated mortality occurs in individuals who would have died anyway. The authors speculate, on the other hand, that exposure to PM may also lead to the extra stress that causes the death of a seriously ill person who may have otherwise recovered.

Cifuentes and Lave used two different methods to evaluate the potential for a "harvesting effect" from exposure to PM. In the first method, they examined a series of correlations to test the hypothesis that an increase in mortality in one day leads to a decrease in mortality in subsequent days (as evidenced by negative correlations). They report a negative correlation for a 2 day lag for all deaths, but it was not significant. While this result indicates some portion of deaths may be from those who would have died anyway, it is not an adequate test since it does not consider the effect of previous days of pollution. They extended the analyses by considering "episodes" of pollution, which are defined as multi-day periods of relatively high air pollution that are preceded and followed by periods of relatively low air pollution. Their result suggests that there is some mortality displacement of a few days occurring in a portion of the population. However, the Cifuentes and Lave estimates are for those deaths which occur in addition to deaths estimated from the regression model. The authors conclude "more research is needed to estimate which fraction, if any of the total deaths estimated ... is due to mortality displacement of a few days only".

An alternative explanation of the observed daily mortality results is that the sensitive subpopulations for PM effects could be continually changing as people contract disease and recover (Schwartz, 1994b; Samet et al., 1995; and Bates, 1992). Thus, it is possible that death might be substantially premature if a person becomes seriously ill and without the extra stress of PM would otherwise have recovered. This hypothesis can be explored by evaluating deaths that occur outside the hospital, based on the premise that patients with

current life-threatening symptoms of disease would be more likely to be in a hospital. Schwartz (1994c) has reported an increase in sudden deaths for individuals who were not hospitalized on days with high PM levels in Philadelphia.

The CD suggests that a portion of deaths associated with long-term exposure to PM are independent of the short-term exposures and could be on the order of years (CD, p. 13-45). Quantification of the degree of life shortening observed in the long-term cohort mortality studies (Dockery et al., 1993; Pope et al., 1995) is difficult and requires assumptions about life expectancies given other risk factors besides PM exposure, the ages at which PM-attributable deaths occur, and the general levels of medical care available in an area to sensitive subpopulations. Because of the uncertainties discussed above, the CD concludes that it is not possible to confidently estimate quantitatively the number of years lost (CD, p. 13-45).

2. Indices of Morbidity Associated with Respiratory and Cardiovascular Disease

Given the statistically significant positive associations between community PM concentrations and mortality outlined above, it is reasonable to anticipate that the same kinds of community-based observational studies should find increased morbidity with elevated levels of PM. This is indeed the case where morbidity effects are measured through increased hospital admissions indicating aggravation of existing disease in the elderly (Table V-6). There is coherence across these morbidity studies, the mortality studies discussed above, and discussions of sensitive subpopulations presented in section C below. The majority of such studies find effects associated with PM exposure to be linked to subpopulations with respiratory or cardiovascular disease (CD, section 13.4.3.5). Numerous studies have observed positive associations between exposure to PM and responses ranging from severe effects (e.g., increased hospitalization for respiratory and cardiovascular conditions) to moderate exacerbation of respiratory conditions. The key evidence for associations of PM exposure with such effects is summarized below.

a. Hospital Admissions and Emergency Department Visits

A number of epidemiological studies report statistically significant positive associations between short-term exposures to PM and hospital admissions for respiratory-related and cardiac diseases. Hospital admissions and emergency room visits for these

TABLE V-6. ESTIMATED INCREASED HOSPITAL ADMISSIONS FOR THE ELDERLY PER 50 $\mu g/m^3$ INCREASE IN 24-h PM_{10} CONCENTRATIONS FROM U.S. AND CANADIAN STUDIES

(After CD, Table 13-3)

Study Location	RR (± CI) Only PM in Model	RR (± CI) Other Pollutants in Model	Reported PM_{10} Levels Mean (Min/Max)[†]
Respiratory Disease			
Toronto, CAN[I]	1.23 (1.02, 1.43)[‡]	1.12 (0.88, 1.36)[‡]	30-39*
Tacoma, WA[J]	1.10 (1.03, 1.17)	1.11 (1.02, 1.20)	37 (14, 67)
New Haven, CT[J]	1.06 (1.00, 1.13)	1.07 (1.01, 1.14)	41 (19, 67)
Cleveland, OH[K]	1.06 (1.00, 1.11)	—	43 (19, 72)
Spokane, WA[L]	1.08 (1.04, 1.14)	—	46 (16, 83)
COPD			
Minneapolis, MN[N]	1.25 (1.10, 1.44)	—	36 (18, 58)
Birmingham, AL[M]	1.13 (1.04, 1.22)	—	45 (19, 77)
Spokane, WA[L]	1.17 (1.08, 1.27)	—	46 (16, 83)
Detroit, MI[O]	1.10 (1.02, 1.17)	—	48 (22, 82)
Pneumonia			
Minneapolis, MN[N]	1.08 (1.01, 1.15)	—	36 (18,58)
Birmingham, AL[M]	1.09 (1.03, 1.15)	—	45 (19, 77)
Spokane, WA[L]	1.06 (0.98, 1.13)	—	46 (16, 83)
Detroit, MI[O]	—	1.06 (1.02, 1.10)	48 (22, 82)
Ischemic HD			
Detroit, MI[P]	1.02 (1.01, 1.03)	1.02 (1.00, 1.03)	48 (22, 82)

References:

[I]Thurston et al. (1994)/O_3.
[J]Schwartz (1995)/SO_2.
[K]Schwartz et al. (1996b).
[L]Schwartz (1996).
[M]Schwartz (1994e)
[N]Schwartz (1994f).
[O]Schwartz (1994d).
[P]Schwartz and Morris (1995)/O_3, CO, SO_2 .

[†]Min/Max 24-h PM_{10} in parentheses unless noted otherwise as standard deviation (± S.D), 10 and 90 percentile (10, 90). NR = not reported. *Means of several cities.*
[‡]RR refers to total population, not just >65 years

diseases reflect prevalence, severity, and patterns of health care utilization. Table V-6 summarizes the results for admissions for all respiratory disease and specific respiratory or cardiovascular diseases such as COPD (emphysema, chronic bronchitis, bronchiectasis, asthma, etc,), pneumonia, and heart disease (see also CD, Tables 12-8 to 12-11). Of the 13 studies included in the CD tables, 12 found statistically significant associations between increases in PM level and increased risk of admission to the hospital, including evaluation of cause-specific admissions for respiratory diseases when only PM was in the model. As with the mortality studies, associations between PM exposure and hospital admissions (Table V-6) have been observed in communities throughout North America (Birmingham, Detroit, Spokane, Tacoma, New Haven, Utah Valley, New York State, Ontario, Canada). These studies reported 6 to 25 percent increases in hospital admissions for respiratory disease associated with a 50 $\mu g/m^3$ increase in PM_{10}. Specifically, studies reported 6 to 9 percent increases in admissions for pneumonia, and 10 to 25 percent increases for COPD for the elderly, associated with a 50 $\mu g/m^3$ increase in PM_{10}. A recent study of hospital admissions for cardiovascular illness (Schwartz and Morris, 1995) reported that PM_{10} was positively and significantly associated with daily admissions for ischemic heart disease, with SO_2, CO, and O_3 making no independent contribution to the effect. In the same study PM_{10} and CO were both independently associated with congestive heart failure admissions.

When viewed together, these studies demonstrate an association between hospital admissions for respiratory and cardiac causes and PM exposure (CD, Chapter 13). These results also suggest a greater effect on admissions for COPD that for other causes from exposure to PM, and are consistent with those of the mortality studies which also found a stronger association between respiratory-related mortality and PM exposure than for all causes of mortality.

b. School Absences, Work Loss Days and Restricted Activity Days

School absences, restricted activity days, and work loss days can also be used as indicators of acute respiratory conditions, though these are indirect measures compared to actual diagnosis and measurement of respiratory conditions. However, it is not clear whether the effects reported in this way result from aggravation of chronic disease (e.g., COPD), acute infection, or non-specific symptomatic effects. Nevertheless, the results of these

studies show consistent statistically significant associations between such measures of morbidity and increased short-term levels of indicators of PM. Ransom and Pope (1992) have reported a statistically significant association between PM levels and school absences; this is consistent with an effect from PM exposure, since respiratory conditions are the most frequent cause of school absences (CD, Chapter 12). In addition, three other studies reported statistically significant associations between community air pollution, as indicated by PM, and work loss days and restricted activity days (Ostro, 1983; Ostro and Rothschild, 1989; Ostro, 1987). More specifically, a study by Ostro and Rothschild (1989) reported significant associations between PM exposure and respiratory-related restricted activity days. All of these studies used two- to four- week lag times between elevations in PM levels and school absences, work loss days, and restricted activity days. This suggests that not only are there immediate effects after elevations of PM exposure (e.g., increased hospital admissions), but PM may elicit effects which are exhibited at a later time. These results are consistent with a hypothesis of increased susceptibility to respiratory infection resulting from exposure to PM.

3. Altered Lung Function and Symptoms

Community epidemiology studies of ambient PM levels, and studies of exposure of humans (clinical studies) and laboratory animals to PM components, show that PM exposure is also associated with altered lung function and increased respiratory symptoms. Effects on respiratory mechanics can range from mild transient changes with little direct health consequence to incapacitating impairment of breathing. Symptomatic effects also vary in severity, but at minimum suggest a biological response that is often more sensitive than lung function measurements.

a. Effects Related to Short-Term Exposures To PM

i. Community Air Pollution Studies

Table V-7 lists a number of community studies highlighted in the CD from U.S. communities that show associations between PM exposure and both respiratory symptoms and immediate pulmonary function changes [e.g., forced expiratory capacity for one second (FEV_1) and peak expiratory flow rate (PEFR)]. Studies reporting symptoms have found associations between short-term exposures of PM and upper respiratory symptoms (e.g.,

TABLE V-7. ESTIMATED LUNG FUNCTION CHANGES AND RESPIRATORY SYMPTOMS PER 50 $\mu g/m^3$ INCREASE IN 24-h PM_{10} CONCENTRATIONS FROM U.S. AND CANADIAN STUDIES (After CD, Table 13-3)

Study Location	RR (± CI) Only PM in Model	RR (± CI) Other Pollutants in Model	Reported PM_{10} Levels Mean (Min/Max)[†]
Increased Respiratory Symptoms			
Lower Respiratory			
Six Cities[Q]	2.03 (1.36, 3.04)	Similar RR	30 (13,53)
Utah Valley, UT[R]	1.28 (1.06, 1.56)[r]	—	46 (11/195)
	1.01 (0.81, 1.27)[r*]		
Utah Valley, UT[S]	1.27 (1.08, 1.49)	—	76 (7/251)
Cough			
Denver, CO[X]	1.09 (0.57, 2.10)	—	22 (0.5/73)
Six Cities[Q]	1.51 (1.12, 2.05)	Similar RR	30 (13, 53)
Utah Valley, UT[S]	1.29 (1.12, 1.48)	—	76 (7/251)
Decrease in Lung Function			
Utah Valley, UT[R]	55 (24, 86)[**]	—	46 (11/195)
Utah Valley, UT[S]	30 (10, 50)[**]	—	76 (7/251)
Utah Valley, UT[W]	29 (7,51)[***]	—	55 (1,181)

References:

[Q]Schwartz et al. (1994).
[R]Pope et al. (1991).
[S]Pope and Dockery (1992).
[T]Schwartz (1994g)
[W]Pope and Kanner (1993)
[X]Ostro et al. (1991)

[†]Min/Max 24-h PM_{10} in parentheses unless noted otherwise as standard deviation (± S.D), 10 and 90 percentile (10, 90). NR = not reported
[r]Children.
[r*]Asthmatic children and adults.
Means of several cities.
[**]PEFR decrease in ml/sec.
[***]FEV_1 decrease.

hoarseness, sore throat), lower respiratory symptoms (chest pain, phlegm, and wheeze), fever, cough, and acute respiratory illness. Additional studies of European communities are reported in Table 12-12 of the CD. Four studies from Table 12-12 evaluated respiratory symptoms in all children (Schwartz et al., 1994; Hoek and Brunekreef, 1993; Hoek and Brunekreef,1995; Schwartz et al., 1991), and all but one found positive statistically significant associations with exposure to PM with one or more symptoms. Two studies evaluated respiratory symptoms in asthmatic children (Pope et al., 1991, Ostro, 1995) and found statically significant positive associations with exposure to PM, although in the Ostro (1995) study, the effect could not be separated from O_3. A study of non-asthmatic symptomatic and asymptomatic children in Utah Valley found statistically significant positive associations between increased PM levels and all symptoms in the symptomatic children. For asymptomatic children, statistically significant positive and consistent associations were found between PM exposure and cough, although no statically significant associations were found for lower respiratory symptoms and inconsistent results for upper respiratory symptoms (Pope and Dockery, 1992). The four studies in adults were inconsistent. Taken together, these studies suggest that sensitive individuals, such as children (especially those with asthma or pre-existing respiratory symptoms) may have increased or aggravation of symptoms associated with PM exposure, with or without reduced lung function.

ii. Controlled Exposures to Laboratory Aerosols

The 1982 CD (EPA, 1982a) and staff paper summarized earlier literature on controlled human and occupational exposures to a variety of particulate substances. This summary (Table 5-2, EPA 1982) highlights studies which report that broncho-constriction and associated symptoms may be induced by chemical or mechanical irritation by high concentrations of inert dusts (e.g. Andersen et al., 1979; Constantine et al., 1959), re-suspended urban dust (Toyama, 1964), coarse organic dusts (e.g. Dosman, 1980), fine acid aerosols (e.g. Utell et al. 1981), and fine particles in combination with pollutant gases (Koenig et al, 1981; McJilton et al., 1976).

Measurements of pulmonary function and symptoms resulting from acid sulfate aerosols have been a primary focus of PM research in short-term (<24 hours) controlled human clinical and animal studies (CD, Table 11.2). Short exposures to fine H_2SO_4

aerosols in environmental chambers, with short periods of exercise, have been reported to cause a slight concentration-related increase in lower respiratory symptoms (cough, sputum, dyspnea, wheeze, chest tightness, substernal irritation) (Avol et. al.,1988a,b).

Asthmatic subjects appear to be more sensitive than healthy subjects to the effects of acid aerosols on lung function (Utell et al., 1982), but the reported effective concentration differs widely among studies (CD, Table 11-2). Adolescent asthmatics may be more sensitive than adult asthmatics and may experience small decrements in lung function in response to H_2SO_4 at exposure levels less than 100 $\mu g/m^3$ (Koenig et al., 1989; CD, p. 11-24). A more recent study of H_2SO_4 ($< 1\mu m$ diameter) on subjects with asthma and COPD (emphysema or chronic bronchitis) found pulmonary function decrements at acid levels as low as 90 $\mu g/m^3$ (Morrow et al., 1994). Even in studies reporting an overall absence of effects on lung function, some individual asthmatic subjects appear to demonstrate clinically important effects (CD, p. 11-31).

Relevant to considerations of the characteristics of acid aerosols that may elicit effects in asthmatic subjects, lung function effects in asthmatic subjects have been correlated with hydrogen ion content of the sulfate aerosol (CD, p. 11-17) and affected by neutralization by oral ammonia (Utell et al., 1983; 1989) and buffering capacity of the aerosol (Fine et al., 1987b). Recent studies also suggest that submicrometer size aerosols may alter lung function to a greater degree than larger sized aerosols in asthmatic subjects (CD, p. 11-31; Avol et al., 1988a,b,) albeit at larger concentrations than found to affect adolescent asthmatics (Koenig et al., 1983, 1989).

Changes in clinical status of human subjects are often accompanied by changes in airway responsiveness as measured by the sensitivity to challenge by a broncho-constrictive agent. Airway responsiveness may be a predictor of responsiveness to acid aerosol exposure in asthmatic subjects (Utell et al, 1983b; Hanley et al., 1992). Accordingly, effects from exposures to pollutants which increase airway responsiveness may be clinically significant even in the absence of direct effects on lung function (Godfrey, 1993; Wiess et al., 1993). Despite the absence of effects on lung function in healthy subjects, Utell et al. (1983a) observed in healthy nonsmokers an increase in airway responsiveness to carbachol challenge 24 hours (but not immediately) following exposure to 450 $\mu g/m^3$ H_2SO_4 (0.8 μ m diameter),

which suggests the possibility of delayed effects. Other studies which have attempted to measure airway responsiveness immediately after acid aerosol exposure have reported little if any effect from low levels of acid aerosol exposure (CD, p. 11-33,34).

Studies in humans have suggested an increase in airway responsiveness to O_3 following low concentrations of H_2SO_4 aerosol exposure in both healthy and asthmatic subjects (Linn et al., 1994; Frampton et al., 1995; CD). Synergistic or interactive effects between sulfates and SO_2 exposure have not been demonstrated (CD, p. 11-37). Indeed, given the low solubility of SO_2 in acid aerosol, it is unlikely that fine acid particles could facilitate an interaction through transport of SO_2 to the deeper regions of the lungs, to which SO_2 alone has difficulty penetrating (U.S. EPA, 1994c). Reflex broncho-constriction by high levels of SO_2 could, however, increase the deposition of particles in the tracheobronchial region by narrowing the conductive airways.

As described in the CD, controlled human studies of PM are limited as they tend to use pulmonary function and symptoms from exposure to acid aerosols as the endpoint of response, and few have examined airway inflammation or other more sensitive indicators related to pulmonary function changes. No studies have examined effects of particles or acid aerosol exposure on airway inflammation in asthmatic subjects (CD, p. 11-30).

Many laboratory animal studies have also been conducted using acid aerosol exposures with the most recent studies on effects on pulmonary function presented in Table 11-5 of the CD. In general, exposure to H_2SO_4 at levels ranging above ambient but < 1000 $\mu g/m^3$ does not produce direct changes in pulmonary function in healthy animals except in guinea pigs (CD, Table 11-5). Airway hyper-responsiveness (alteration in the degree of reactivity to exogenous or endogenous bronchoactive agents resulting in increased airway resistance at levels of these agents that would not affect airways of normal individuals) from exposure to ($< 1\mu m$ diameter) H_2SO_4 particles has been reported in several studies (Chen et al., 1992b; Gearhart and Schlesinger, 1986; and El-Fawal and Schlesinger, 1994). Hyper-responsiveness has also been observed to be increased in guinea pigs exposed to acid-coated particles in comparison to pure H_2SO_4 aerosols of the same size (Amdur and Chen, 1989; Chen et al., 1992b). Whatever the underlying mechanism, the results of pulmonary function

studies indicate that H_2SO_4 is a broncho-active agent and can therefore alter lung function of exposed animals via contraction of smooth muscle (CD, p. 11-47).

b. Effects Related to Long-Term Exposures

Table V-8 summarizes effects estimates reported from studies highlighted in the CD which assess the association between long-term exposure to PM and pulmonary function changes and symptoms of respiratory disease. Two initial studies conducted in the Harvard six cities (Ware et al., 1986, Dockery et al., 1989) demonstrated that there is a statistically significant association of particulate pollution with respiratory symptoms in children, with no significant changes in lung function. As noted in the CD, the absence of significant findings in lung function effects in the Six City comparison may be due to the inherent variability of the measure. To follow-up on the suggestions that respiratory symptoms and probably lung function were associated mostly with fine particle levels and acidity, a more comprehensive study of 24 cities across North America using the same questionnaire was conducted (Raizenne et al., 1996; Dockery et al., 1996). The cities were chosen to provide a gradient in aerosol acidity exposures. Air monitoring data was collected for one year. This study reported statistically significant positive associations between bronchitis and sulfate concentration and acidity as well as between changes in lung function (FVC) and PM_{10}, $PM_{2.5}$, sulfate particle concentration, and particle acidity indicators.

Abbey et al. (1995a,b,c) in California reported elevated but marginally non-significant associations, which were in the range of the results of the other studies, between sulfate concentration and bronchitis well as acute obstructive disease, as defined in the studies. Two other long-term pulmonary function studies (presented in Table 12-22 of the CD) reported decreases in lung function in children (with no confidence level given) (Spector et al., 1991) and statistically significant decreases in lung function in adults (Ackermann-Liebrich et al., 1996) associated with long-term PM exposure.

The results from the long-term respiratory symptom studies are consistent and supportive of those reported for short-term studies. The CD concludes that the results are consistent with a PM gradient (CD, p. 12-372), and that while the evidence is suggestive for long-term exposure to PM being associated with pulmonary lung function decrements, it is more limited (CD, p. 12-202).

TABLE V-8. MORBIDITY EFFECTS ESTIMATES PER INCREMENTS[a] IN ANNUAL MEAN LEVELS OF FINE/THORACIC PARTICLE INDICATORS FROM U.S. AND CANADIAN STUDIES (After CD, Table 13-5).

Type of Health Effect & Location	Indicator	Change in Health Indicator per Increment in PM[a]	Range of City PM Levels Means ($\mu g/m^3$)
Increased bronchitis in children		Odds Ratio (95% CI)	
Six City[d]	$PM_{15/10}$	3.26 (1.13, 10.28)	20-59
Six City[e]	TSP	2.80 (1.17, 7.03)	39-114
24 City[f]	H^+	2.65 (1.22, 5.74)	6.2-41.0
24 City[f]	$SO_4^=$	3.02 (1.28, 7.03)	18.1-67.3
24 City[f]	$PM_{2.1}$	1.97 (0.85, 4.51)	9.1-17.3
24 City[f]	PM_{10}	3.29 (0.81, 13.62)	22.0-28.6
Southern California[g]	$SO_4^=$	1.39 (0.99, 1.92)	—
Decreased lung function in children			
Six City[d,h]	$PM_{15/10}$	NS Changes	20-59
Six City[e]	TSP	NS Changes	39-114
24 City[i,j]	H^+ (52 nmoles/m^3)	−3.45% (-4.87, -2.01) FVC	—
24 City[i]	$PM_{2.1}$ (15 $\mu g/m^3$)	−3.21% (-4.98, -1.41) FVC	—
24 City[i]	$SO_4^=$ (7 $\mu g/m^3$)	−3.06% (-4.50, -1.60) FVC	—
24 City[i]	PM_{10} (17 $\mu g/m^3$)	−2.42% (-4.30, -.0.51) FVC	—

[a]Estimates calculated annual-average PM increments assume: a 100 $\mu g/m^3$ increase for TSP; a 50 $\mu g/m^3$ increase for PM_{10} and PM_{15}; a 25 $\mu g/m^3$ increase for $PM_{2.5}$; and a 15 $\mu g/m^3$ increase for $SO_4^=$, except where noted otherwise; a 100 nmole/m^3 increase for H^+.
[d]Dockery et al. (1989)
[e]Ware et al. (1986)
[f]Dockery et al. (1996)
[g]Abbey et al. (1995a,b,c)
[h]NS Changes = No significant changes.
[i]Raizenne et al. (1996)
[j]Pollutant data same as for Dockery et al. (1996)

The CD points out that the increased risk for respiratory symptoms and related respiratory morbidity reported in the above studies is important not only because of the immediate and longer-term symptoms produced, but also because of the longer-term potential for increases in the development of chronic lung disease. Specifically, recurrent childhood respiratory illness has been suggested to be a risk factor for later susceptibility to lung damage (Glezen, 1989; Samet, 1983; Gold et al., 1989).

4. Morphological Damage

Traditional epidemiology has not been used to evaluate the extent to which PM directly alters lung tissues and components, although some autopsy studies have found qualitative evidence of a community air pollution effect on the lung (e.g., Ishikawa et al. 1969). Evidence of morphological damage from PM exposure has come from animal and occupational studies for acid aerosols and other PM components.

a. Acid Aerosols

Morphological alterations associated with exposure to acid aerosols have been most extensively studied and are outlined in Table 11-6 of the CD. Single or multiple exposures to H_2SO_4 at fairly high levels (> 1 mg/m^3) produce a number of characteristic morphological responses (e.g., alveolitis, bronchial and/or bronchiolar epithelial desquamation and edema) (CD, p. 11-52). Chronic exposure to H_2SO_4 at concentrations ≤ 1 mg/m^3 produces a response characterized by hypertrophy and hyperplasia of epithelial secretory cells. Gearhart and Schlesinger (1988), however, show that chronic exposure of H_2SO_4 (250 μg/m^3, 0.3μm) also produces an increase in the relative number of smaller airways in rabbits which can be an early change relevant to clinical small airway disease (CD, p. 11-52). Long-term (68 months exposure) studies of combinations of SO_2 (1.1 mg/m3) and submicrometer sulfuric acid (90 μg/m3) exposure of dogs found no pronounced effects at the end of exposure, but a number of morphological changes, including an increase in interalveolar pores (incipient emphysema), was found to increase for up to 3 years following exposure (Hyde et al., 1978; Gillespe, 1980).

Morphologic and cellular damage to the respiratory tract following exposure to acid aerosols may be determined by methods other than direct microscopic observation (CD, p. 11-53). Animal studies of exposure to fine (0.3 μm) diameter and ultrafine (0.04 μm)

diameter H_2SO_4 aerosols (300 $\mu g/m^3$) have reported lavage fluid to contain increases in lactate dehydrogenase and protein (markers of cytotoxicity and increased cellular permeability) following a single exposure to guinea pigs (Chen et al., 1992a).

In addition, modulation of biological mediators of inflammatory responses (e.g. eicosanoids) as well as smooth muscle tone (e.g. prostaglandins and leukotrienes) could be involved in damage to the respiratory tract after particle exposure. Changes in prostaglandins (Schlesinger et al; 1990b) have also been observed in lung perfusate after exposure to H_2SO_4 and lavage. Since some of the prostaglandins are involved in regulation of muscle tone, changes in these mediators may be involved in the development of airway responsiveness found with exposure to acid sulfates (CD, p. 11-54).

b. Silica, Crustal Dusts, and other PM Components

Silica has long been considered to be a major occupational health hazard, with exposure to crystalline silica being associated with pulmonary inflammation and fibrosis (CD, p. 11-127). The differing forms of silica (amorphous versus crystalline) are thought to have differential potential for toxicity, but data on amorphous forms is limited (CD, p. 11-128). There are limited data on ambient concentrations of silica, which is generally found in the coarse fraction. Based on analyses of the silica content of resuspended crustal material collected from several U.S. cities as part of the last review, staff concluded that the risk of silicosis at levels permitted by the current long-term PM_{10} NAAQS was low. This earlier conclusion is supported by the CD based on the integration of occupational and autopsy findings with ambient silica concentrations (CD, p. 13-79).

The 1982 staff paper (U.S. EPA, 1982b) reported that some risk of long-term exposure to crustal dusts is suggested by autopsy studies of farm workers and residents in the Southwest (Sherwin et al., 1979), desert dwellers (Bar-Ziv and Goldberg, 1974), and zoo animals and humans exposed to various crustal dusts near or slightly above current ambient levels in the Southwest (Brambilla et al, 1979). These studies found evidence of a silicate pneumonoconiosis, which was related to local crustal materials. Responses ranged from the buildup of particles in macrophages with no clinical significance to possible pathological fibrotic lesions. No inferences regarding quantitative exposures of concern could be drawn from these studies (U.S. EPA 1982b).

Kleinman et al. (1995) have reported increases in alveolar wall thickness as well as alveolar chord length and cross sectional area from exposure of rats to road dust (900 $\mu g/m^3$, 4 μm diameter), ammonium sulfate (70 $\mu g/m^3$, 0.2 μm diameter), and ammonium nitrate (350 $\mu g/m^3$, 0.6μm diameter). The authors suggest such morphometric changes could lead to a decrease in compliance or a "stiffening" of the lung.

Coating the surface of particles with certain transition metals, such as iron, may have the potential to enhance pulmonary injury to a variety of environmental particles (CD, p. 11-92; Costa et al., 1994a,b; Tepper et al., 1994). These metals can catalyze the oxidative deterioration of biological macromolecules and thus could potentially cause oxidative injury to the respiratory tract (CD, p. 11-92). Silica particles have been reported to be rendered more toxic when complexed with iron. Rats fed with iron depleted diets (and thus having less iron available from body stores to complex intratracheally instilled silica particles and to decrease antioxidant molecules in lung tissue) exhibited less inflammation and fibrotic injury after such exposures (Ghio et al., 1994; 1992; Ghio and Hatch, 1993). However, there is difficulty in extrapolating the results of experimental paradigms used in these studies (intratracheally instillation) to ambient exposure situations.

5. Effects on Host Defense Mechanisms

Responses to air pollutants often depend upon their interaction with respiratory tract defenses such as clearance and antigenic stimulation of the immune system. Furthermore, either depression or over-activation of these systems may be involved in the pathogenesis of lung diseases (CD, p. 11-55). Acid aerosols (H_2SO_4) alter mucociliary clearance in healthy human subjects at levels as low as 100 $\mu g/m^3$ with effects being dependent on the concentration and duration of the acid aerosol exposure, the size and distribution of the acid particles, and the region of the airways being examined (CD, p. 11-56 to 60, Leikauf et al., 1984). In addition, the acidity of the aerosol has been reported to affect mucociliary clearance in animals (CD, p. 11-60). Acid aerosols have been shown to elicit a slowing in clearance that lasts several months following multiple exposures (Lippmann et al., 1981). Persistent impairment of clearance may lead to the inception or progression of acute or chronic respiratory disease, and may be a plausible link between acid aerosol exposure and respiratory disease (CD, p. 11-61).

Little is known about the effects of particles on humoral (antibody) or cell-mediated immunity. Since numerous bioaerosols (potential antigens) are present in inhaled air, the possibility exists that acid sulfates may enhance immunologic reaction and thus produce a more severe response with greater pulmonary pathogenic potential (CD, p. 11-67). There is evidence that H_2SO_4 exposure may be a factor in promoting lung inflammation by acting as a vehicle to increase antigenicity (Pinto et al., 1979; CD, p. 11-69). Guinea pigs have been reported to show increased sensitivity to inhaled antigen (ovalbumin) with concurrent H_2SO_4 exposure (1,910 $\mu g/m^3$ <1 μm diameter) as demonstrated by hyper-responsive airways (Osebold et al., 1980). In addition, Fujimaki et al. (1992) have demonstrated that guinea pigs have altered mast cell function after exposure to high concentrations of H_2SO_4 (1000 and 3000 $\mu g/m^3$). These cells are involved in allergic responses including broncho-constriction (CD, p. 11-69).

Alveolar macrophages not only play a major role in defense against bacteria, but are involved in the induction and expression of immune reactions, and are capable of release of pro-inflammatory cytokines (CD, p. 11-56). In order to maintain the function of clearance, macrophages must be competent in a number of other functions including phagocytosis, mobility, and attachment to a surface (CD, p. 11-63).

Macrophages also produce a number of biologically active chemicals which are involved in host defense [tumor necrosis factor (TNF) release activity and production of superoxide radical] (CD, p. 11-66). Exposure to H_2SO_4 (50 to 500 $\mu g/m^3$, 0.3μm diameter) in rabbits produced reductions in TNF cytotoxic activity as well as reduction in superoxide radical in alveolar macrophages recovered by lavage (Zelikoff and Schlesinger, 1992). However, exposure to H_2SO_4 (300 $\mu g/m^3$, 0.3 and 0.04μm diameter) in guinea pigs enhanced TNF and hydrogen peroxide from alveolar macrophages (Chen et al., 1992a). Such differences in response may reflect either interspecies differences or differences in experimental conditions. Kleinman et al. (1995) have reported in their study of cellular and immunological injury by PM that respiratory burst activity by macrophages was depressed by exposure to fine ammonium sulfate (70 $\mu g/m^3$, 0.2 μm diameter), ammonium nitrate (350 $\mu g/m^3$, 0.6μm diameter) particles, and road dust (900 $\mu g/m^3$, 4 μm diameter)

Animal infectivity models have been used to examine effects of H_2SO_4 exposure on susceptibility to bacterial infection. Exposures of up to 1 mg/m^3 of submicrometer H_2SO_4 aerosols for 30 days alone have not resulted in enhanced susceptibility to bacterially-mediated respiratory disease in mice (See Table 11-8 in the CD). However, Zelikoff et al. (1994) demonstrated an effect of high concentrations of acid alone in rabbits exposed for 2 h/day for 4 days to 500 to 1000 μg/m^3 H_2SO_4 and demonstrated reduction of intracellular killing and uptake of the bacterium Staphylococcus aureus by alveolar macrophages.

Multi-pollutant exposures have been shown to elicit changes in infectivity in mice after short-term exposure. For example, Gardiner et al. (1977) reported increased susceptibility to infection by exposing mice to O_3 (0.1 ppm) followed by H_2SO_4 (0.9 mg/m^3). Neither pollutant produced any effect alone. Although conducted using high acid levels, the results of this study are of particular interest given the co-occurrence of O_3 and acid sulfates in summertime episodes over broad regions of North America.

D. Sensitive Subpopulations

The recent epidemiologic information summarized in the CD provides evidence that several subgroups are apparently more sensitive (susceptible) to the effects of community air pollution containing PM. As discussed above, observed effects in these groups range from the decreases in pulmonary function reported in children to increased mortality reported in the elderly and in individuals with cardiopulmonary disease. Furthermore, the same individual characteristics which can be described in those who succumbed to air pollution during the more extreme historical episodes are also present in those most susceptible to effects during routine fluctuations in PM level. Table V-9 is a qualitative assessment of the short-term and long-term PM epidemiologic evidence with regard to subgroups that appear to be at greatest risk with respect to particular health endpoints. It is a condensation of results presented in Tables 13-6 and 13-7 of the CD. The table summarizes the findings for the indicated health indices in the specified subpopulations.

TABLE V-9. QUALITATIVE SUMMARY OF RECENT PM COMMUNITY EPIDEMIOLOGIC RESULTS FOR SHORT- AND LONG - TERM EXPOSURE***

Age Class	Subpopulation	Mortality		Morbidity**		Lung Function Change	
		Acute (Exposure to PM)	Chronic (Exposure to PM)	Acute (Exposure to PM)	Chronic (Exposure to PM)	Acute (Exposure to PM)	Chronic (Exposure to PM)
Adults	Elderly	+	0	+	0	0	0
	Pre-existing Respiratory Disease*	+	+	+	0	0	0
	Pre-existing Cardiovascular Disease	+	+	+	0	0	0
Children	General	ID	+\-	+	+	+	+\-
	Pre-existing Respiratory Disease	0	0	+	0	+	0
Adults and Children	Asthmatics	0	0	+	+	+	0

* Note, this includes those with pneumonia, acute bronchitis and COPD.
** Note, morbidity includes hospitalization and emergency room visits and community morbidity and symptoms reported in table 13-6 of the CD.
*** Note; + indicates positive associations have been reported for this group with PM exposure; +\- means few pertinent studies identified, weight of evidence of PM related effect is somewhat positive but uncertain; 0 means that no pertinent studies have been identified; ID means insufficient data, at least 1 pertinent study identified but inference as to weight of evidence is not warranted.

The following section expands upon individual risk factors (including age, asthma, COPD, and cardiovascular disease), characteristics of those factors which may increase inherent susceptibility to PM effects, and incidence of such risk factors (as well as overall mortality associated with such factors) to provide some perspective on the scope of subpopulations at risk from PM exposure. Table 13-9 of the CD presents more detailed information concerning the incidence of selected cardiorespiratory disorders by age and by geographic region. In addition, Table 12-1 of the CD shows age-specific and age-adjusted U.S. death rates for selected causes in 1991 and selected components in 1979, 1990, and

1991. Information from these tables is incorporated in the discussion below, and gives some indication of the relative sizes of sensitive subpopulations. Such subpopulations may experience effects at lower levels of PM than the general population, and thus, the subsequent magnitude of effects may be greater.

1. Individuals with Respiratory and Cardiovascular Disease

Both the early London episode studies and the most recent community studies in North America have found air pollution with elevated particle concentrations to be associated with increased mortality, hospital admissions, and symptoms in individuals with respiratory and cardiovascular disease (CD, Chapter 13). Because smoking is associated with the same types of cardiopulmonary diseases which characterize individuals also susceptible to PM exposures, smoking is an important variable to be controlled in epidemiologic studies attempting to investigate the effects of PM (see CD, p.13-86 for further discussion).

COPD is the most common pulmonary cause of death, the fourth leading cause of death overall (84,000 deaths in 1989, U.S. Bureau of the Census 1992), and a major cause of disability. COPD incidence increases with age of the population (e.g., excluding asthma, the incidence rate for those over 75 is approximately twice that as for those under 45 years of age) (CD, Table 13-9). Patients with COPD have a larger relative risk of mortality from PM exposure than the general population (CD, Chapter 12, see Section C of this document). COPD is a broad disease category used to cover patients with varying degrees of chronic bronchitis, emphysema and asthma, etc. COPD is characterized by airway obstruction in which there is increased resistance to airflow during forced expiration. According to the International Classification of Disease definitions and classification codes, COPD includes chronic bronchitis, emphysema, asthma, and pneumonitis. Many epidemiology studies use these codes and therefore reported effects such as hospital admissions for COPD include asthma admissions. The American Thoracic Society only includes emphysema and chronic bronchitis in their definition of COPD and, when referring to COPD, the CD uses this definition. Subcategories of COPD, emphysema, and chronic bronchitis may result in chronic inflammation of distal airways, destruction of the lung parenchyma, and loss of supportive elastic tissue leading to airway closure during expiration (CD, p. 13-84).

Recent community studies summarized in the previous section also found increased risk from death and morbidity (increased hospital admissions) due to cardiovascular causes associated with exposure to increased PM concentration (Tables V-4, V-6). As with COPD, the preexisting condition of heart disease occurs at high frequency in the general population and contributes significantly to total mortality (represents 1/3 of all causes of mortality for all ages) (CD, Table 12-1). The pathophysiology of many lung diseases is related to cardiac function, and plausible, but undemonstrated mechanisms have been advanced that suggest possible links between effects of air pollution exposure and the presence of cardiovascular disease [Table V-2, Appendix D, Bates (1992)].

2. Individuals with Infections

Individuals with respiratory symptoms are at increased risk of morbidity and mortality from PM exposure and are often those with respiratory infection. Exposure to PM may exacerbate illness from infectious agents and increase risk of severe outcomes. In general, increased mortality associated with PM exposure from pneumonia and influenza has been reported for the elderly. Mortality rates from pneumonia and influenza combined are just somewhat lower than those for COPD and allied conditions (i.e, asthma) (CD, Table 12-1). As with COPD, there is also an increased rate of mortality from pneumonia and influenza with increasing age. An increase in respiratory symptoms in children has also been reported to be associated with PM exposure (see Section C of this Chapter).

3. The Elderly

Although recent epidemiology studies suggest higher relative risks for people over 65 years of age, currently little information suggests how aging in the absence of pathology might make the elderly more susceptible to the effects of ambient particles (Cooper et al., 1991). Length of exposure increases the cumulative lung burden (dose equals concentration times time) which may be related to susceptibility to particle effects. The elderly may be more sensitive to respiratory insult from PM because such exposure may have effects on pulmonary and cardiovascular function which augment decreases seen with increasing age. In addition, cardiorespiratory disease and infection (e.g., pneumonia and influenza) are more prevalent in the elderly which may predispose such individual to effects of PM exposure. In

people over 75 years of age, 40% have some form of heart disease, 35% have hypertension, and approximately 10% have COPD (CD, p. 13-84).

4. Children

Increased community morbidity, decreased lung function, and increased respiratory symptoms have been reported to be associated with PM exposure in children, both as a general group and in individuals with respiratory illness (CD, Table 13-6). Children have the potential to be inherently more susceptible to the effects of PM as they show a greater incidence of respiratory and other illness, suggesting decreased immunological protection, and higher deposition of particles than adults (CD, p.10-77). Children may spend more time outdoors and may have higher ventilation rates due to increased activity and thus have increased inhalation of outdoor pollutants (CD, Chapter 10). Infants in particular have been hypothesized to be a sensitive subpopulation for PM effects as exposure may increase the incidence or severity of acute respiratory infection including bronchitis, bronchiolitis, and pneumonia (Samet et al., 1995). However, recent studies in North America have not found clear evidence of increased mortality or morbidity associated with exposure to PM in infants or children (CD, Chapter 12). The rate of mortality from pneumonia and influenza is relatively high for children under 1 year of age (11 times that for children 1 to 4 years, twice that of adults 45-54 years of age) (CD, Table 12-1).

5. Asthmatic Individuals

Asthma is a lung disease characterized by (1) airways obstruction that is reversible, but only partially in some patients, either spontaneously or with treatment, (2) airways inflammation, and (3) increased airway responsiveness to a variety of stimuli. The airways of asthmatics may be hyper-responsive to a variety of stimuli including exercise, cigarette smoke, odors, irritating fumes, changes in temperature, humidity, allergens, pollen, dust, as well as viral infection (CD, p. 13-86). [A more complete discussion of the characteristics of asthma may be found in the SO_2 Staff Paper (U.S. EPA, 1994c)]. The heightened responsiveness of the airways of asthmatics to such substances and conditions raises the possibility of exacerbation of this pulmonary disease by PM.

Increases in PM have been associated with increased hospital admissions for asthma, worsening of symptoms, decrements in lung function and increased medication use (CD,

Chapter 12, Tables V-6, V-7). There are approximately 13 million people in the U.S. with asthma and that number is increasing (National Center for Health Statistics, 1994). Incidence of asthma is higher among children and young adults, with asthma being the leading cause of non-infectious respiratory mortality below age 55. Approximately 70% of all asthma-related deaths occur after age 55 (National Center for Health Statistics, 1993). The available studies of PM and mortality do not, however, single out asthma from the larger category of respiratory-related mortality. Thus, from the available evidence a direct association between PM exposure and asthma mortality has not been demonstrated.

E. Evaluation of the Epidemiological Evidence

The majority of the evidence concerning health effects of PM exposure comes from epidemiological studies. While severe effects at the high concentrations of air pollution in the historical episodes are widely accepted as being causally related, there is less consensus as to the most appropriate interpretation of studies finding associations of health effects with ambient levels of PM below the current NAAQS (e.g., Schwartz, 1994b; Dockery et al., 1995; Moolgolvkar, 1995b; Moolgolvkar and Luebeck, 1996; Li and Roth, 1995; Samet et al., 1996a; Wyzga and Lipfert, 1995). Thus, evaluation and interpretation of the epidemiological studies is key to assessing the weight of the evidence for causal relationships between health effects and PM exposures at ambient levels below the NAAQS. Evaluation of the epidemiological evidence for these purposes requires both assessing the individual studies as well as the body of evidence as a whole for drawing appropriate conclusions.

The CD summary of perspectives on the epidemiology studies is pertinent here:

> "By far the strongest evidence for ambient PM exposure health risks is derived from epidemiologic studies. Many epidemiologic studies have shown statistically significant associations of ambient PM levels with a variety of human health endpoints, including mortality, hospital admissions and emergency room visits, respiratory illness and symptoms measured in community surveys, and physiologic changes in mechanical pulmonary function. Associations of both short-term and long-term PM exposure with most of these endpoints have been consistently observed. The general internal consistency of the epidemiologic data base and available findings have led to increasing public health concern, due to the severity of several studied endpoints and the frequent demonstration of associations of health and physiologic effects with ambient PM levels at or below the current U.S. NAAQS for PM_{10}. The weight of epidemiologic evidence suggests that ambient PM exposure has affected the public health of U.S. populations. However, there remains much uncertainty in the

published data base regarding the shapes of PM exposure-response relationships, the magnitudes and variabilities of risk estimates for PM, the ability to attribute observed health effects to specific PM constituents, the time intervals over which PM health effects are manifested, the extent to which findings in one location can be generalized to other locations, and the nature and magnitude of the overall public health risk imposed by ambient PM exposure.

The etiology of most air pollution-related health outcomes is highly multifactorial, and the effect of ambient air pollution exposure on these outcomes is often small in comparison to that of other etiologic factors (e.g., smoking). Also, ambient PM exposure in the U.S. is usually accompanied by exposure to many other pollutants, and PM itself is composed of numerous physical and chemical components. Assessment of the health effects attributable to PM and its constituents within an already-subtle total air pollution effect is difficult even with well-designed studies. Indeed, statistical partitioning of separate pollutant effects may somewhat artificially describe the etiology of effects which actually depend on simultaneous exposure to multiple air pollutants. Furthermore, identification of anatomic sites at which particles trigger end-effects and elucidation of biological mechanisms through which these effects may be expressed are still at an early stage. Thus, it remains difficult to form incisive a priori hypotheses to guide epidemiologic and experimental research. Lack of clear mechanistic understanding also increases the difficulty with which available findings can be integrated in assessing the coherence of PM-related evidence.

In this regard, several viewpoints currently exist on how best to interpret the epidemiology data: one sees PM exposure indicators as surrogate measures of complex ambient air pollution mixtures and reported PM-related effects represent those of the overall mixture; another holds that reported PM-related effects are attributable to PM components (per se) of the air pollution mixture and reflect independent PM effects; or PM can be viewed both as a surrogate indicator as well as a specific cause of health effects. In any case, reduction of PM exposure would lead to reductions in the frequency and severity of the PM-associated health effects (CD, pp. 13-31)."

The CD also outlines major criteria useful in evaluating the adequacy and strength of the epidemiological studies and in interpreting them. These criteria include quality of the aerometric data, clear definition of study populations and health endpoints, appropriate statistical analysis, adequate control of confounders, and evaluation of the consistency and coherence of the findings with other known facts (CD, Chapter 12). The CD addresses each of these issues, including both the strengths and inherent limitations of such studies. The discussion below in Section V.E.1 focuses on several key factors identified in evaluating the

individual studies and outlines observations on sensitivity to model specification, exposure error, and potential confounding by weather and other pollutants. Individual studies can not be used by themselves to determining whether attributable health effects are occurring from current levels of PM because of inherent limitations in any single study. Thus, to evaluate the potential for PM to effect public health, the collective weight of evidence from studies must be evaluated together. Accordingly, the interpretation of individual studies is followed by a discussion of the consistency and coherence of the epidemiological evidence across studies.

1. Interpretation of Individual PM Study Results

a. Model Selection and Specification

The recent epidemiological literature contains extensive discussion of model selection and specification for short-term mortality studies (CD. Section 12.6.2.1). The discussion has focussed on a number of issues including distributional assumptions, assumptions about temporal structure or correlation, assumptions about random and systematic components of variability, assumptions about the shape of the relationship between response and covariate, and assumptions about additivity and interactions of covariates (CD, 13.4.2.3). Sensitivity of the effects estimates to model specification has been explored by many authors, and an in-depth discussion of model specification for short-term mortality studies is presented in Section 12.6.2 of the CD, where PM_{10} studies of mortality are reviewed and analyzed (Pope et al. 1992a; Ostro et al., 1996; Dockery et al., 1992; Thurston and Kinney, 1995; Kinney et al., 1995; Ito et al., 1995; Styer et al., 1995). Also, importantly, alternative TSP mortality analyses for the same city, Philadelphia (Moolgavkar et al. 1995b; Li and Roth, 1995; Wyzga and Lipfert, 1995; Cifuentes and Lave, 1996; Samet et al., 1995; Schwartz and Dockery, 1992b) are reviewed and analyzed. Based on these assessments, the models appear to be most sensitive to the following specifications: adjustments for seasonality and for long-term time trends; adjustments for co-pollutants; and adjustments for weather (CD, p. 13-53).

While the CD finds that model specification is important and can influence the health effect estimates from PM exposure, it also notes that appropriate modelling strategies have been adopted by most investigators (CD, section 13.4.3.2), that have resulted in consistent PM effects estimates reported across the studies. These strategies include use of several

standard models (e.g. GLM, LOESS) and a number of particular specifications. For example, it is important to remove long-term trends in the data before evaluating the association between short-term changes in PM and health effects. As the CD points out, a several different methods used by the various authors are adequate for carrying out this adjustment, including nonparameteric detrending, use of indicator variables for season and year, and filtering (CD, section 13.1.3.2). The CD concludes that, "the largely consistent specific results, indicative of significant positive associations of ambient PM exposures and human mortality/morbidity effects, are not model specific, nor are they artifactually derived due to misspecification of any specific model. The robustness of the results of different modelling strategies and approaches increases our confidence in their validity" (CD, p. 13-54).

b. Measurement Error

A difficulty in interpretation of the epidemiological studies, particularly for quantitative purposes, is the determination of uncertainties and possible biases introduced by measurement error in the outdoor monitors. In the ecological context of the daily mortality/morbidity studies, investigators estimate a population-level index of pollution exposure for those at risk of dying or experiencing illness. The variation in mortality/morbidity is modeled implicitly as a function of the variation in this index. Measurement error includes both the error in the measurements themselves and the error introduced by using a central monitor to estimate such population-level exposures. It is important to examine the possible effect measurement error may have on the reported associations in the studies, as it may bias the results in either direction. Unfortunately, most studies provide only qualitative assessments of this issue, as opposed to their more formal treatment of weather and some other confounders. The discussion that follows is drawn from the CD assessment of the relationship between the monitored pollutant levels (using TSP, PM-10, and fine particles as indicators) and exposure and on how the error in the measurements might bias the reported associations.

The CD points out that, although generally useful for qualitative epidemiologic demonstration of PM effects, TSP measurements can include large coarse-mode particles do not penetrate to the thoracic region. Thus, TSP can reasonably be expected to provide

"noisy" estimates of exposure-effect relationships if such relationships are due to thoracic particle fractions of the measured TSP mass. By definition, PM_{10} is a better index of thoracic particles than is TSP, and PM_{10} may be a better index of ambient fine particle exposure than TSP because the smaller particulate fraction contained in PM_{10} is more uniformly distributed in an urban area or region than are larger coarse particles also indexed by TSP. As discussed in Section 13.2.6, $PM_{2.5}$ particles are generally likely to be more uniformly distributed than coarse particles within an urban airshed. For example, measurements of the coarse fraction of PM_{10} appear to be more variable from site to site, while $PM_{2.5}$ levels have been shown to be particularly well correlated across at least one eastern metropolitan region, i.e., Philadelphia (Burton et al., 1996; Wilson and Suh, 1996), as well as in more limited data from Riverside, CA (Wallace, 1996). The use of a spatial average of multiple TSP or PM_{10} monitors in some studies (e.g., Philadelphia, Minneapolis) can reduce exposure uncertainties for these less uniform pollutant indicators.

Even if outdoor levels near population centers are well represented by monitors, the extent to which outdoor concentration fluctuations are found to affect indoor concentrations and personal exposures to outdoor-origin particles is still an issue of particular importance. Some of the sensitive populations in the short-term mortality and hospital admissions studies (i.e., the elderly and those with pre-existing disease) can be expected to spend more time indoors than the general population. Some commentors have expressed concerns regarding the lack of correlation shown in some cross sectional studies of outdoor and indoor or personal exposures, and suggest that confounding by indoor sources of PM might bias the effects/outdoor PM response function towards a linear relationship when a threshold model may be more appropriate.[3] The CD assessment of this issue, however, found longitudinal correlations of personal exposure to PM_{10} can be well correlated with outdoor measurements. The CD assessment concluded that "the exposure to indoor-generated particles will not be

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[3]Implicit in this suggestion is the hypothesis that indoor- and outdoor-generated particles are essentially the same with respect to those characteristics important to producing particular health effects of concern. While some indoor-generated particles may have composition similar to outdoor PM, there may be significant differences in the adsorbed components, acidity, and other physico-chemical properties of potential importance that are more unique to particles that originate in a complex urban atmosphere. The relative importance of such factors is critical to testing the above hypothesis.

correlated with the concentration of ambient (outdoor-generated) particles, and time-series epidemiology based on ambient measurements will not identify health effects of indoor-generated particles" (CD, p. 1-10). Furthermore, the CD assessment of the literature found that "the measurements of daily variations of ambient PM concentrations, as used in the time-series epidemiology studies of Chapter 12, have a plausible linkage to the daily variations of human exposures to PM from ambient sources, for the populations represented by the ambient monitoring stations. This linkage should be better for indicators of fine particles ($PM_{2.5}$) than for indicators of fine plus coarse particles (PM_{10} or TSP), which, in turn, should be better than indicators of coarse particles ($PM_{10-2.5}$)" (CD, p 1-10). The strength of the correspondence between outdoor concentrations and personal exposure levels on a day-to-day basis serves to reduce, but not eliminate, the potential error introduced by using outside monitors as a surrogate for personal exposure.

The effect of instrument and "representativeness" components of measurement error of PM and other covariates on the association between PM and effects can vary with modeling approach. Measurement error in the exposure variable, PM, in a univariate regression can bias the association toward the null. However, in multivariate regressions, which are used in the PM literature, the association is also influenced by the relationship between PM and the other covariates which can bias the association in either direction. This issue has been discussed in two recent analyses, one of cardiovascular hospital admissions in Detroit, (Schwartz and Morris, 1995) and the other of mortality in the six cities of the Six City Study, (Schwartz et al, 1996). In the cardiovascular hospital admission study, Schwartz and Morris discuss the potential influence of measurement error from the other covariates, CO and weather on the PM/cardiovascular hospital admissions relationship. High correlation between the covariates and the exposure of interest represents potential influence of error in the covariates on the exposure of interest. They evaluated the correlation between the covariates and found the correlations between CO levels and the weather variables, and between CO and PM levels, were small. In addition, the correlation between PM levels and weather variables was also small. They conclude that such low correlations may imply it is likely significant portions of bias do not come from the covariates, but from the error in measuring PM, which would decrease the association between PM levels with hospital

admissions. The authors point out, however, that this does not mean that the estimated magnitude of the associations was unbiased.

This issue is explored further in the short-term mortality study in the six cities of the Six City Study (Schwartz et al., 1996). The authors examine the potential influence of measurement error on the association between excess mortality and $PM_{2.5}$ levels. They note that the correlations between $PM_{2.5}$ level and the other covariates, (e.g., weather) are not large, and thus not likely to influence the measurement error in the level of $PM_{2.5}$ itself. They examine this by leaving weather terms out of the regression model, which is similar to a large measurement error in these terms, and find a slight decrease in the effects estimate for exposure to $PM_{2.5}$. They further test the effects of measurement error in the city of Boston by creating 10 new $PM_{2.5}$ exposure variables each based on the original $PM_{2.5}$ measurement with additional random error. They then repeat the multivariate regression 10 times using each of the 10 new $PM_{2.5}$ variables. They find the mean coefficient for PM effects with the added measurement error was reduced by 13% compared to the original effects coefficient. These two results suggest that the net effect of random measurement error in the multiple regression is to bias toward underestimating the particle effect.

Schwartz et al., 1996 did not, however, assess either the effect of differential measurement error among the various particulate components, or the effect of other co-pollutants. Because coarse fraction particles occurring at the lower concentrations found in most of the six-cities are likely measured with less precision than are fine particles (Rodes and Evans, 1985), any effects of coarse particles would tend to be underestimated relative to fine particles (CD, p. 13-52). This does not diminish the significance of the findings for fine particles or PM_{10}, particularly in view of the fact that the association remained highly significant even when limited to days with $PM_{2.5}$ concentrations under 25 $\mu g/m^3$. Measurement error would be expected to be greater for fine particles at these lower concentrations than for the full data set.

Although the issue of confounding by other pollutants (e.g., SO_2, CO, O_3, NO_x NO_2) is addressed in a subsequent section, measurement error clearly has implications for separating the effects of individual pollutants from a complex urban mixture. When collinear pollutants having different degrees of exposure error are entered into a regression jointly, the

variable with the least exposure error will tend to be assigned higher significance, all else being equal (Lipfert and Wyzga, 1995a).

While the magnitude of measurement error and its effect on the PM/health effect associations is unknown, it is possible to test potential influences of measurement error in the PM measure or the influence of other covariates. Some aspects of these issues have been discussed in two recent studies, suggesting -- although not conclusively -- that the influence of measurement error is to bias the estimate downward. Nevertheless, a comprehensive, formal treatment of exposure misclassification studies of PM and other community air pollutants is an important research need. As discussed below, however, the consistency of the PM/effects relationship in multiple locations with widely varying indoor/outdoor conditions and a variety of monitoring approaches makes it less likely that the observed findings are an artifact of exposure misclassification.

c. Potential Influence of other Covariates in Short-Term Studies

Other factors that vary temporally with PM may influence the estimated relationship between PM and health effects, either independently or through interaction with PM. Independent risk factors related to both PM concentrations and the health effect of interest which could potentially confound the apparent associations between PM exposure and health effects. Inadequate control for confounding can result in incorrect interpretations, e.g., regarding the reported effect as being the result of an observed risk factor, when a third variable (the confounder) is really responsible. The estimated relationship between PM and health effects can also be biased up or down by potential interactions between PM and other risk factors, particularly other pollutants.

Significant attention has been focused on addressing potential confounders in the short-term studies. The CD points out that it is preferable to control confounding by designing a study in such a way that potential confounders are avoided (CD, Section 12.6.3.4). However, in many studies this is not a feasible option because it is not possible to avoid some potential confounders, such as weather, and in some cases, the levels of PM and the confounders are highly correlated. This can also be a problem for areas in which co-pollutants are derived from a common mixture of sources, such as combustion.

The CD discusses the difficulty in conducting studies in enough cities to make the appropriate number of comparisons. As discussed more fully in section V.E.2 below, however, the observed similarities in relative risk of health effects from PM exposure across study areas with large differences in the potential for confounding from copollutants adds credibility to the conclusion that the PM mortality effects are real (CD, p. 12-331).

Covariates associated with daily changes in health effects, such as weather, season and levels of other pollutants (e.g., SO_2) potentially associated with PM levels need to be considered. Most of the epidemiology studies of PM have considered at least some of the potential confounders in their analysis. These studies have used a number of methods to address or reduce confounding, with varying degrees of success. Less attention has been given to effects modification from the interaction between co-occurring pollutants and PM. A summary of the major issues discussed in the CD regarding the potential influence of other potential risk factors on PM and the most relevant PM studies is presented below.

i. Weather

Weather is an important confounder in short-term PM studies because fluctuations in weather are associated with both changes in PM and other pollutant levels and health effects reported in the studies[4]. Individual studies have used a variety of approaches to separate the effects of PM exposure and weather with most treatments appearing to be adequate (CD, p. 13-54). Most studies include temperature and dewpoint as covariates in their studies (CD, p. 13-54). In addition, many investigators use statistical methods to adjust for weather and season on an annual basis when modeling the PM and health effect relationship. In several of these studies (Schwartz, 1993a, 1994a, 1994d, 1994e, 1994f) nonlinear functions have been used that can reflect the complex relationship between weather and health effects [e.g., the effect of temperature in Birmingham, Alabama (Schwartz, 1993a)]. In other studies, linear and categorical variables were used (e.g., for very high temperature days) to adjust for routine fluctuations in weather and extreme conditions (Kinney et al., 1995; Pope et al., 1992). In an examination of the sensitivity of the associations of exposure to PM_{10} with

[4]The relationship between temperature and health effects over the course of a year tends to be "U" shaped, with increasing effects on days with very hot or cold temperatures (Moolgavkar and Luebeck, 1996).

health effects to control for weather, several studies reported distinct effects of weather on mortality that were largely separable from the effects of PM exposure in the areas studied. Moreover, elimination of all weather variables from the PM-mortality models did not substantially affect the size of the observed associations between PM exposure and excess mortality (Schwartz et al., 1996; Schwartz and Dockery, 1992a, 1992b).

Because of the limitations in using temperature and humidity alone to examine the much more complex changes that accompany various weather patterns, two recent studies of pollution and mortality associations in Utah Valley (Pope and Kalkstein, 1996) and Philadelphia (Samet et al., 1996b) further examined confounding by weather through the use of synoptic weather categories. In these studies the synoptic weather categories were defined independently of the health effects information, in an approach first recommended by Kalkstein (1994). Both studies show that the reported association between PM exposure and excess mortality was relatively insensitive to the changes in weather. All of the studies of daily PM levels and mortality use some method to adjust for weather, and report consistent associations between PM exposure and health effects.

The CD concludes that the PM coefficient is relatively insensitive to different methods of weather adjustment, as recently demonstrated in the recent studies and the reanalysis by HEI (CD, p. 13-54). Recent studies have adequately addressed the role of weather-related variables. (CD, p. 13-54). Clearly, weather affects human health; however, it is highly unlikely that weather can explain a substantially greater portion of the PM attributable health effects than has already been accounted for in the models (CD, p. 13-54).

ii. Confounding By Other Pollutants

One of the concerns raised by a number of authors conducting reanalyses of the mortality studies is whether the observed PM effects are confounded or modified by other pollutants commonly occurring in community air such as SO_2, O_3, NO_2, and CO (Samet et al., 1995, 1996a; Moolgavkar et al., 1995b; Moolgavkar and Luebeck, 1996; Li and Roth, 1995). Based on successive reanalyses, Moolgavkar has advanced the contention that PM is serving as a surrogate for the general ambient air pollution mixture and that the reported health effects are more appropriately attributed to the mixture rather than to PM alone (Moolgavkar 1995b; Moolgavkar and Luebeck, 1996). Much of the support for this interpretation comes from the recent reanalyses of the Philadelphia data where it has proven

to be difficult to separate individual effects of multiple pollutants (Samet et al., 1995, 1996a; Moolgavkar et al., 1995b; Moolgavkar and Luebeck, 1996; Li and Roth, 1995). The HEI investigators concluded that "...a single pollutant of the group TSP, SO_2, NO_2, and CO cannot be readily identified as the best predictor of mortality" based only on analyses of the Philadelphia data (Samet et al., 1996a).

The CD examined the evidence for confounding in these and other studies in some detail in Section 12.6. It concludes that other pollutants can play a role in modifying the relationship between PM and health effects. The CD also notes that some studies have found little change in the PM relative risk (RR) after inclusion of other copollutants in the model and in analyses where the PM RR estimate diminished, the RR typically remained statistically significant (CD 13-57). Based on an evaluation of the existing studies and its assessment of confounding within and across a number of areas with differing combinations of pollutants, the CD concludes that the PM health effects associations are valid and, in a number of studies, not seriously confounded by co-pollutants (CD, p. 13-57). The role of co-pollutants in modifying the apparent RR associated with PM is less clear. The following discussion summarizes evidence regarding PM confounding and effects modification for each of several criteria pollutants.

Sulfur Dioxide (SO_2). SO_2, which was present at high concentrations with PM during the historical episodes, has long been seen as a potential confounder of the PM effect. Reanalyses of the extensive London data (Schwartz and Marcus, 1986) provided some support for the suggestion of Mazumdar et al., (1981) that at lower SO_2 values in London, mortality effects may be associated with PM alone. The more recent studies, in particular short-term exposure mortality studies, have applied several approaches to address SO_2 confounding, including restriction (studies in areas with low SO_2 levels) and more direct means. The discussion below highlights key findings from the recent epidemiological studies together with other pertinent information from SO_2 and PM air quality relationships and from studies of the penetration of SO_2, alone and in combination with particles, to the respiratory tract described below.

In areas where the potential for confounding from SO_2 is relatively high, investigators have adjusted for SO_2 in the model (Ostro et al., 1995a; Toulomi et al., 1994; Schwartz and

Dockery, 1992a). These studies have also conducted sensitivity analysis of the association between PM and health effects, by evaluating the association before and after adding SO_2 to the model. These analyses produced inconsistent results. Studies conducted in Santiago Chile, Philadelphia, PA and Sao Paulo, Brazil, found that the association between PM and mortality remained positive and significant after the addition of SO_2; whereas, the association between SO_2 and mortality became insignificant (Ostro et al. 1996; Schwartz 1992a; Saldiva et al., 1995). A similar analysis in Athens, Greece found that after modeling both SO_2 and PM, the association with SO_2 remained significant and positive (Touloumi et al., 1994). The estimates of associations with health effects for both pollutants were reduced, however.

The PM/SO_2 confounding issue has been thoroughly explored in Philadelphia through extensive analysis by several investigators, where SO_2 and PM are highly correlated (Schwartz, 1992a; Moolgavkar, 1995b; Li and Roth, 1995; Samet et al., 1995, 1996a). In these studies, investigators have been concerned about the potential for confounding from SO_2 in the observed TSP/mortality association. The original analysis by Schwartz and Dockery (1992a) evaluated the association between TSP and mortality in Philadelphia between 1973-1980. They found the association between TSP and mortality remained significant after adding SO_2 to their model; whereas, the relationship between SO_2 and mortality became insignificant. Moolgavkar et al. (1995b) evaluated the association between TSP and mortality in Philadelphia between 1973-1988. In this study, they attempted to account better for modification of the effect of air pollution on mortality by factors that vary with season (e.g., weather, pollutant mix, activity patterns). The Philadelphia daily air pollution/mortality data set is one of those large enough to conduct such seasonal analyses without undue loss of statistical power. Modeled individually, both pollutants were found to be significantly associated with mortality in each season. In models where TSP and SO_2 where included simultaneously, they concluded that TSP was positively associated with mortality in the summer and fall, and SO_2 was positively associated in all four seasons[5].

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[5]In a seasonal analysis of the later years of the Philadelphia data (1983-88), Cifuentes and Lave (1996) found somewhat different results. In their analysis, SO_2 was only significant in the winter, and only without TSP in the model, while TSP was significant in spring and summer and the coefficient was stable across all seasons (CD, p. 12-53).

HEI evaluated both of the Philadelphia data sets discussed above (Samet et al., 1995; Samet et al., 1996d) and conducted their own analysis on data collected directly from the National Center for Health Statistics and EPA's AIRS database. Although the overall results of the reanalyses were similar to those of the original authors, the new HEI analyses used techniques that revealed a more complex, non-linear set of relationships among pollutants, season, and mortality. The authors concluded that the Philadelphia data showed a relationship between air pollution and mortality, but that it would be difficult to use the results of this single study to attribute such effects solely to particles. The combined pollutant mortality relationships are of some interest. The first HEI analysis explored the relationship between SO_2 and TSP in depth. The relationship between TSP and mortality indicates a monotonically increasing response occurs only at particle levels above 100 $\mu g/m^3$ TSP. This result is consistent with either a no-observed- effects level for TSP at 100 $\mu g/m^3$ or a reduced association caused by a correlation with SO_2 at lower concentrations. Conversely, SO_2 displays a monotonically increasing concentration response function from the lowest levels to about 40-60 ppb, where the curve flattens out. It is difficult to find a plausible mechanism for such a concentration-response relationship for a single pollutant, suggesting confounding is likely.

Dockery et al. (1995) commented on the HEI analysis, suggesting that TSP and SO_2 are indicators of a more appropriate risk factor, such as fine particles. The facts that fine particle sulfates and SO_2 share a common source in Philadelphia and that the coarse fraction of TSP is poorly correlated with the fine fraction (CD, Table 6-15) indicate that either or both pollutants could reasonably serve as a surrogate for fine particles. In this event, SO_2 itself might play no direct role in causing effects, with only a fraction of TSP participating. Resolution of the merit of the original investigator's suggested hypothesis, however, must await the results of subsequent studies that use fine particle indicators in lieu of TSP.

In evaluating the findings in Philadelphia, an important consideration is the evidence on the penetration and deposition of particles in the respiratory system as compared to SO_2. Although quantitative support is lacking, the discussion of controlled human and animal studies of particles indicates that smaller particles can more effectively penetrate to the portions of the lung where irritation or other interactions with lung tissues might produce

effects. (See section V.A above). Beyond reflex broncho-constriction observed only at very high peak levels, however, deep lung effects of SO_2 are minimal because gas-phase SO_2 is generally efficiently removed in the extrathoracic region in humans (U.S. EPA, 1994c). This lack of penetration in the lung greatly reduces the likelihood that SO_2 alone could produce significant cardio-pulmonary effects, particularly for sensitive individuals spending more of their time indoors where SO_2 concentrations are low due to rapid removal by indoor surfaces. However, one mechanism by which SO_2 can be transported deeper into the lung is absorption or dissolution onto the surfaces of atmospheric particles (See Section V.F). In this case, the complex results reported by HEI in regard to effects associated with SO_2 exposure might be partially reflecting varying atmospheric interactions of the two pollutants, rather than a direct SO_2 effect.

Given the difficulty in ascribing effects to a single pollutant in Philadelphia or similar cities where elevated particles are associated with SO_2, confounding by SO_2 can be addressed by assessing the PM/mortality relationship in areas with low levels of SO_2. Dockery et al., (1993) found no association between SO_2 and mortality in Kingston and St. Louis, areas with considerably lower SO_2 levels. While consistent associations between PM and health effects are observed across the different studies, the reported association between health effects and SO_2 can vary widely. In Steubenville, the association between SO_2 and mortality was ten-fold greater than in Philadelphia (i.e., coefficients of 0.0104 versus 0.00132 per ppb) (Schwartz and Dockery, 1992a,b) although the two areas have comparable SO_2 levels.

In a single city such as Philadelphia, where SO_2 and PM levels are highly correlated, it is more difficult to ascribe the observed mortality effects to a single pollutant. In such cases, consideration of the observed relationships and relevant information on air quality, indoor exposures, dosimetry, and mechanisms suggest that it is unlikely that an independent effect of SO_2 is occurring that does not involve PM. Moreover, given the number of studies using different methods to correct for potential confounding in areas of high and low SO_2 that find an association between PM and mortality, it is unlikely that SO_2 is responsible for all of the observed associations between PM and mortality. Similarly, when the more severe morbidity endpoints such as respiratory-related hospital admissions are considered, the

presence or absence of SO_2 is also seen to have little effect on observed PM associations (see Table V-11, Schwartz, 1995a) in most cases.

Ozone. The co-occurrence of episodes involving high temperatures with elevated levels of O_3 and PM raised the potential for confounding, particularly during the O_3 season in large regions of eastern North America, Los Angeles, and some other cities). In such cases, covariate adjustment has often been used to try to distinguish the effects of multiple pollutants. A number of studies using such methods have found PM to be a stronger predictor of mortality than O_3 (Dockery et al. 1992b; Saldiva et al., 1995; Kinney et al., 1995; Ostro et al., 1996). Adjusting for the presence of O_3 did not significantly affect the associations with PM and mortality. For example, in Los Angeles, which has the highest concentrations of O_3 studied, investigators found a significant association between both PM and O_3 mortality when each pollutant was entered into the model separately, but found no significant association between O_3 and mortality in models that included PM (Kinney, 1995). On the other hand, the coefficient for PM remained stable when O_3 was in the model along with PM, but the uncertainty in the PM association increased, making it marginally significant; this finding suggests that the PM-mortality association was not completely independent of O_3 (CD, p. 13-55). In Santiago, where a negative correlation exists between O_3 and PM levels, no association was observed between O_3 and mortality across a full year even without PM in the model; this was despite summertime values of O_3 that were twice the U.S. standard (Ostro et al., 1996). In the Utah Valley, O_3 and PM were also negatively correlated, and the inclusion of O_3 as a covariate strengthened the estimated PM effect (Pope et al. 1995a, Table V-3). Furthermore, the relative risk estimates for PM were relatively unchanged and there was little increase in the width of the confidence interval after inclusion of O_3 in the model, and indicating little evidence of confounding of the PM effect (CD, p. 13-52).

Samet et al., (1996a) extended their analysis of the Philadelphia mortality data by examining combinations of multiple pollutants (TSP, O_3, NO_2, SO_2, and CO). This analysis found a low correlation between PM and O_3, indicating independence between the two pollutants. Ozone had a stable and significant association with mortality that appeared to be independent of the other pollutants. The effect estimate for TSP was lowered, but remained

significant when O_3 was added to the model. The CD reanalysis of the HEI results suggests that O_3 may be a potential confounder of TSP in the summer, but not in other seasons (CD, p. 12-297).

In some locations, the potential for O_3 to confound the effects caused by PM is minimized by the low concentrations of O_3 observed during seasons which show a robust PM effect. Examples include Utah Valley and Santa Clara, where O_3 levels are minimal in the winter when the PM levels are high (Pope et al., 1992a; Fairley, 1990). The discussion above of confounding by weather notes a number of cities with cooler climates, where particles are associated with mortality, which would have low O_3 levels.

There is a higher potential for O_3 confounding for the risk of respiratory-related morbidity, because multiple studies have demonstrated apparent separable associations between respiratory effects and PM and O_3 concentrations. Moreover, the recent review of the O_3 criteria found that the biological basis for O_3 aggravation of respiratory symptoms was supported by controlled human and animal studies (EPA, 1986c). The respiratory-related hospital admission studies often find O_3 and PM are each singularly associated with respiratory-related admissions (Schwartz, 1994d; Schwartz, 1996; Burnett et al., 1994). When both pollutants are modeled together, the association between PM and respiratory-related admissions in general remains relatively unchanged, indicating a separable effect independent of O_3. The potential for O_3 confounding for cardiac-related hospital admissions appears to be much lower. Two studies have reported that PM is associated with cardiac hospital admissions but O_3 is not (Burnett et al., 1995; Schwartz and Morris, 1995).

Carbon Monoxide (CO). The lethality of high concentrations of CO is well documented; as such, it must be considered as a potential confounder in community studies (U.S. EPA, 1991). Three of the short-term PM exposure studies examined the effect of CO on the PM/mortality relationship. A study in Athens found a significant association between mortality and CO and PM when each pollutant was considered separately (Touloumi et al., 1994). When considered together, only PM remained significantly associated with mortality. However, there was a high correlation between CO and PM making such separation difficult. Similarly in Los Angeles, where CO and PM were also correlated, positive associations between each pollutant and mortality were reported when both were

evaluated simultaneously (Kinney et al., 1995). However, in Chicago, insignificant associations were reported between CO and mortality (Ito et al., 1995). The recent analysis by HEI of Philadelphia also evaluated the role of CO in mortality (Samet et al., 1996a). Similar to the other studies they found a moderate correlation between TSP and CO concentrations, and they considered CO, along with SO_2 and NO_2 to be interrelated with TSP because of their common sources. Their results show that the average CO concentration on current and previous day was never significantly associated with mortality, whereas CO lagged by three and four days, was significantly associated with mortality. The authors note that this finding was not expected given the mechanism of CO toxicity and the half-life of carboxyhemoglobin. With TSP and lagged CO in the model, they find both TSP and lagged CO level are each significantly associated with mortality. Based on an extended analysis of these results, the CD finds that TSP effects can be reasonably distinguished from CO in all seasons (CD, p. 12-297).

The results from these studies are inconsistent with respect to CO. Because of the nature of urban sources of CO as well as indoor sources, exposure misclassification may introduce significant problems, which reduces the ability of community studies to detect a CO effect. In addition, while cardiovascular effects are plausibly linked to CO, controlled studies do not suggest CO is a respiratory irritant (U.S. EPA, 1991). It is therefore unlikely to confound studies reporting respiratory related mortality, hospital admissions, or aggravation of conditions such as asthma, all of which are linked to PM.

The potential relationship of CO and PM to cardiovascular effects was examined in the Schwartz and Morris (1995) study of hospital admissions for cardiovascular diseases in Detroit. They found an association between CO and PM and ischemic heart disease and congestive heart failure admissions when evaluating each pollutant separately. When evaluated together, CO was no longer associated with ischemic heart disease admissions, but the association with admissions for congestive heart failure for both pollutants remained relatively unchanged, suggesting each pollutant had a separable, independent association with congestive heart failure. While significant exposure to CO in microenvironments characterized by high CO levels may render a hypoxic effect on patients with cardiopulmonary disease, which may aggravate heart disease (see section B above and

Appendix D), it is unlikely that most patients would be exposed to such a level of CO. In addition, once taken to the hospital or to other places with low CO the carboxy hemoglobin levels of such patients would rapidly decline.

Nitrogen Dioxide (NO_2). By comparison, fewer of the mortality studies have directly assessed NO_2 as a potential confounder of PM_{10} effects. Several such studies have reported high correlations between NO_2 and PM in Los Angeles, CA; Toronto, Canada; and Santiago, Chile (Kinney, 1991, Ostro et al., 1996, Özkaynak et al., 1994). Mixed results were reported concerning the association between NO_2 and mortality. Kinney and Özkaynak (1991) found a statistically significant relationship with NO_2 and mortality in Los Angeles, but reported that these results were interchangeable with CO and PM, since the correlations were so high between these pollutants. In Los Angeles and some other Western U.S. cities, nitrogen oxide emissions are themselves a major source of fine particles and nitric acid. The Santiago study found, however, that NO_2 was not associated with mortality when included in the model of PM and mortality (Ostro et al., 1996). Furthermore, the association between PM and mortality remained relatively unchanged after addition of NO_2 to the model. Similar results were found in the Sao Paulo study, where NO_2 was not associated with mortality in adults after including PM_{10} in the model (Saldiva et al., 1995). All these studies were conducted in areas of relatively high NO_2 levels; Santiago had the lowest mean level of 0.0556 ppm. A study in St. Louis, with a lower mean level of 0.02 ppm, found no significant association between mortality and NO_2 (Dockery et al., 1992b). While the association between NO_2 and health effects in these studies is inconsistent, the association between PM and health effects remains positive and consistent, both across study areas with varying levels of NO_2 and after controlling for NO_2 in the model (Ostro et al., 1996; Saldiva et al., 1995; Schwartz et al., 1994).

NO_2 was also included in the multi-pollutant analyses of mortality in Philadelphia. Moolgavkar and Luebeck (1996) found that, when all co-pollutants were entered simultaneously into their model, NO_2 appeared to emerge as the most important pollutant. By contrast, the recent HEI multi-pollutant analysis (Samet et al., 1996a) of mortality in Philadelphia found that with both TSP and NO_2 in the model, the coefficient and the t-value for TSP increased. NO_2, on the other hand, was not significantly associated with mortality

when modeled alone, and when TSP or all pollutants combined were included in the model, the coefficient for NO_2 became significantly negative. In essence, the more limited results for NO_2 and mortality to date do not show a consistent association.

2. Consistency and Coherence of the Epidemiological Studies

While individual studies indicate health effects are associated with PM, a more comprehensive synthesis of the available evidence is needed to evaluate fully the likelihood of PM causing effects at levels below the current NAAQS. Because individual studies in themselves are inherently limited as a basis for addressing causality, the consistency and coherence of the effects across the studies must be considered. As noted above, it is too difficult to resolve the question of confounding using these results from any single city because of the correlation among all the pollutants (Samet et al, 1996a). The HEI investigators conclude that "insights into the effects of individual criteria pollutants can be best gained by assessing effects across locations having differing pollutant mixtures and not from the results of regression models based on data from single locations" (Samet et al., 1996a). The consistency of the association is evidenced by its repeated observation by different investigators, in different places, circumstances and time; and by the consistency of the associations with other known facts (CD, Chapter 13; Bates, 1992). A complement to consistent associations found for individual endpoints is coherence, which is the logical or systematic interrelationship among different health indices, which should be demonstrated across the studies of different endpoints. As the CD notes, the discussion of the consistency and coherence of the epidemiological studies must be largely qualitative because it relies on a series of judgments concerning the reliability of the individual studies (CD, p. 13-58). The consistency and the coherence of the PM epidemiological evidence is discussed and evaluated below.

a. Consistency

The CD summarizes over 80 community epidemiological studies evaluating associations between short-term PM levels and mortality and morbidity endpoints in tables 12-2 and 12-8 to 12-13. Over 60 of these have found consistent, positive, significant associations between short-term PM levels and mortality and morbidity endpoints. These studies have been conducted in a number of geographic locations throughout the world,

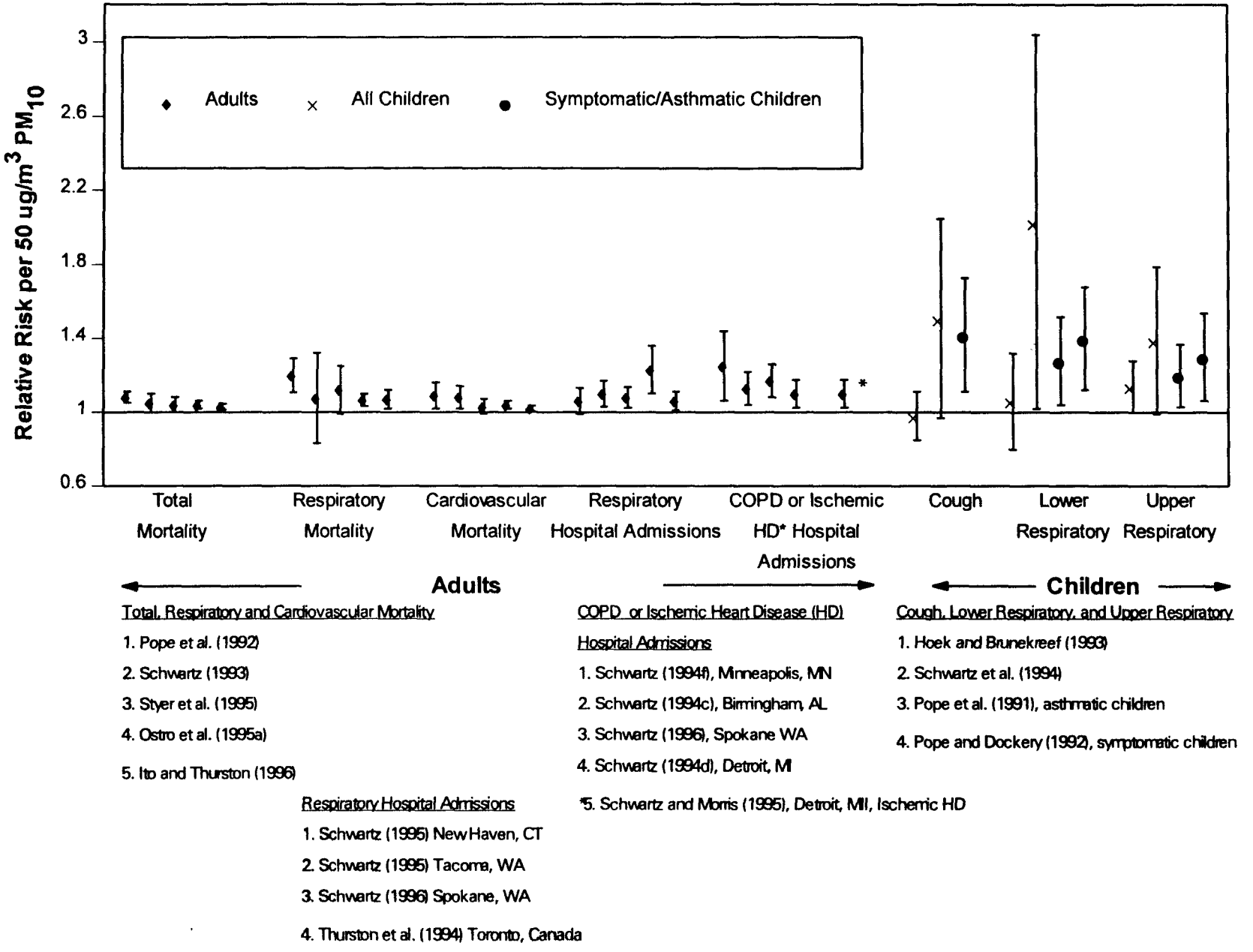
including the US, Canada, Europe and Latin America, using a variety of statistical techniques, and with varying temporal relationships. Despite the variations in the approaches, the effects estimate for each health endpoint is relatively consistent among the studies. Figure V-2 displays the estimated relative risk per 50 $\mu g/m^3$ PM_{10} increase derived from the U.S. and Canadian short-term studies of mortality and morbidity effects presented in Tables V-4, V-6, and V-7.

Clearly, the relative risk estimates exhibit some variation for particular endpoints. For example, the relative risk estimates for mortality associated with a 50 $\mu g/m3$ increase in PM10 range from 1.02 to 1.08. The CD observes that this kind of variation in the RR estimates would be expected for the following reasons: 1) the relative toxicity of PM varies from region to region; 2) the demographic and socioeconomic characteristics of the population vary regionally; 3) the health status, and thus the distribution of the sensitive population vary regionally; and 4) ambient PM levels vary regionally. Thus, the CD concludes that some variation in the RR estimates is not inconsistent with a real effect of PM exposure on daily mortality (CD, Section 13-4.1.1). Similarly, some variation in the RR estimates for morbidity endpoints would be expected, as is observed in Figure V-2.

The large number of studies in a number of different geographic areas, provides an opportunity to evaluate the consistency and sensitivity of the PM estimates to different levels of potential influence by weather and copollutants. Such an evaluation allows consideration of both the potential for confounding from these factors and interpretation of whether the observed health effects are attributable to PM or to the complex air pollution mixture. As for confounding, the CD notes generally similar RR estimates for acute mortality in different studies with different levels of potential confounding copollutants lend credibility to the conclusion that the PM mortality effects are real (CD, p. 12-33).

If PM is acting independently, then a consistent association should be observed in a variety of locations of differing relative proportions of particles and potential gaseous pollution confounders. If, instead, the observed PM effect results from influence from another pollutant, either through confounding or synergistic interaction, the associations with PM would be expected to be consistently high in areas with high concentrations of the pollutant, and consistently low in areas with lower concentrations of the pollutant. In

FIGURE V-2. **Relationship Between Relative Risk per 50 µg/m3 PM_{10} and Specific Causes of Mortality and Morbidity in Adults and Children.**



addition, consistent PM effects across a range of pollutants indicates would indicate that it is more likely that there is an independent effect from PM, that is not confounded by other components of the air pollution mix. Figure V-3 shows the reported relative risk of PM_{10} effects and associated levels of SO_2, NO_2, O_3, and CO from studies conducted in the U.S. as reported in Table V-3. The relative risks are those reported in each of the studies, unadjusted for the other pollutants. The figure indicates that the association with PM_{10} remains reasonably consistent through a wide range of concentrations of these potentially influential pollutants. While it is possible that different pollutants may serve to confound or otherwise influence particles in different areas[6], it seems unlikely that this would lead to such similar associations and relative risk numbers for particles. Within the observed range of relative risk, however, it is certainly possible that other pollutants might modify the apparent effects of particles by atmospheric interactions (e.g., through dissolution/adsorption or aerosol formation reactions) or by independent effects on sensitive populations (e.g. respiratory function changes from O_3 or SO_2) as described in the previous section. Moreover, the possibility of exposure misclassification for primary gaseous pollutants (e.g., CO, SO_2) could diminish their apparent significance. Nevertheless, epidemiological studies have been conducted in a broad range of areas across the U.S. and Canada, where meteorological and pollution patterns vary distinctly. These studies find a consistent, positive association between PM and mortality and morbidity effects. The CD has concluded that the effects are unlikely to be explained by weather (CD, p. 13-54), that the PM effects are not sensitive to other pollutants and the "findings regarding the PM effects are valid" (CD, p. 13-57).

b. Coherence

In addition to the consistently observed associations for each effect, this collection of studies shows coherence in the kinds of health effects associated with PM exposure. The CD

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[6]In this interpretation of the results advanced by Moolgavkar and Luebeck (1996), CO, for example, would lead to a false association with particles in Utah Valley where SO_2 was low, and SO_2 would lead to a false particle signal in Philadelphia, where CO levels were more modest. Such a serendipitous combination of variable confounding would make the more ubiquitous pollutant, particles, appear to be consistently associated with the effect. In this event, at least two other pollutants, or an unidentified substance(s) correlated with them, would be associated with mortality and other effects.

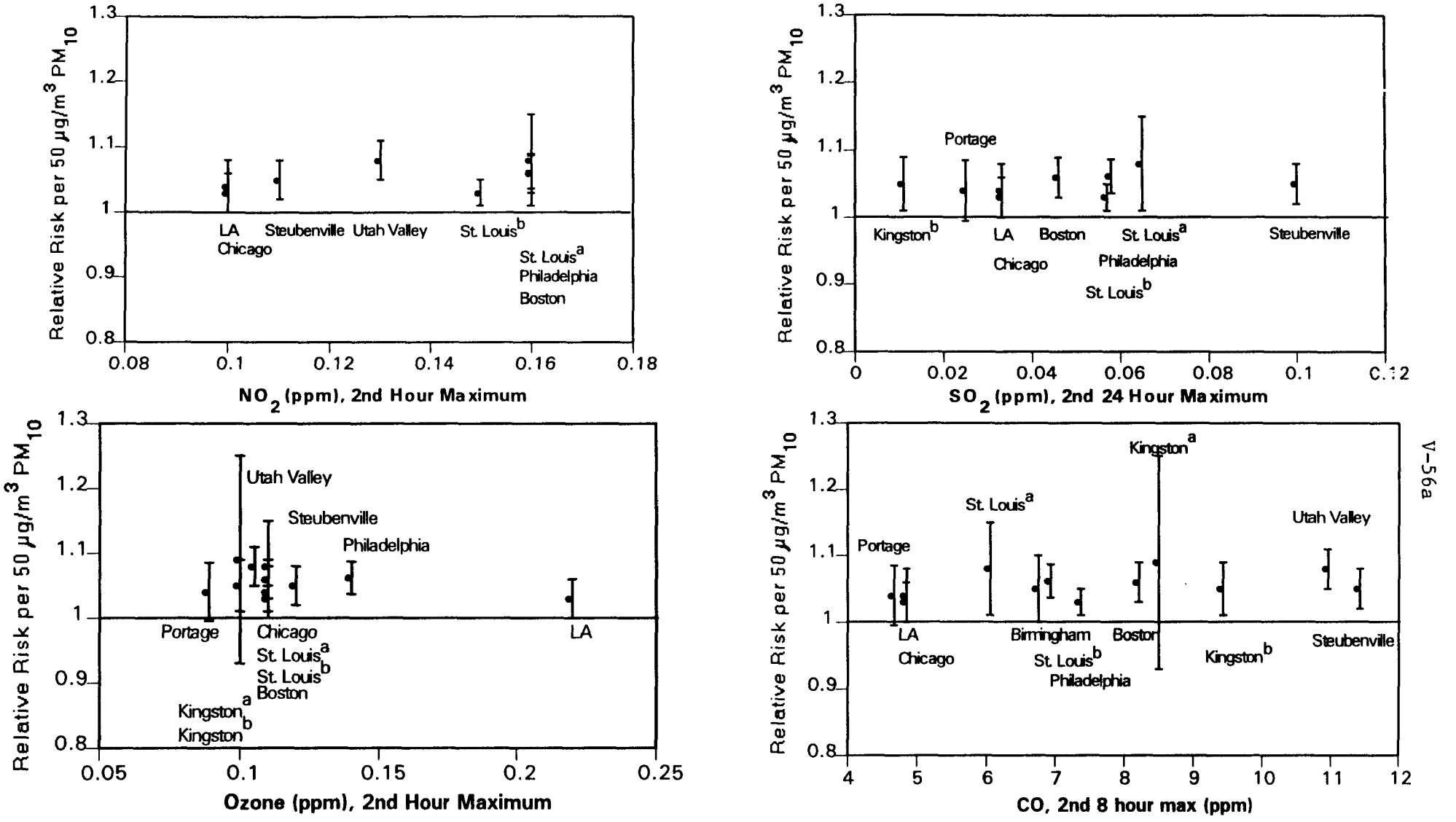

FIGURE V-3a. Relationship Between Relative Risk of Mortality Associated with PM_{10} and Maximum Levels of SO_2, CO, NO_2 and Ozone

Data on SO_2, CO, NO_2 and O_3 are from EPA's AIRs Database. Pollution concentration value for each city is the mean of the 2nd maximum value observed at all monitors in the study area over the study time period, which is designed to represent typical high daily values in each city. Cities without recorded levels of a pollutant are not included. The RRs are from the cities referenced in Table V-3. Superscripts a and b on St. Louis and Kingston indicate the RR from the Dockery et al. 1992 study and the Schwartz et al. 1996a study respectively. Chicago RR is from Styer et al. 1995.

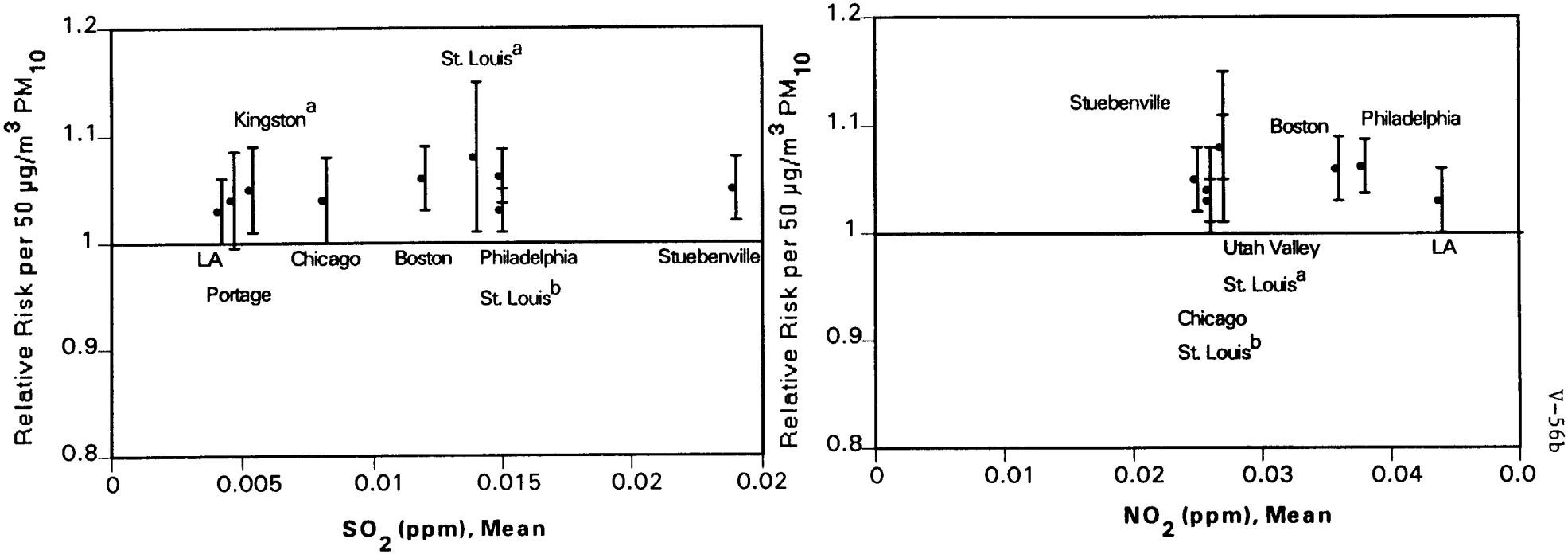

FIGURE V-3b. Relationship Between Relative Risk of Mortality Associated with PM_{10} and Mean Values of SO_2 and NO_2.

Data on SO_2, and NO_2 are from the EPA AIRs Database. The concentration value for each city is the mean of all recroded values observed at all monitors in the study area over the study time period. Cities without recorded levels of the pollutant are not included. The RRs are from the cities referenced in Table V-3. Superscripts a and b on St. Louis and Kingston indicate the RR from the Dockery et al. 1992 study and the Schwartz et al. 1996a study respectively. Chicago RR is from Styer et al. 1995.

provides a qualitative review of the coherence of the health effects associated with both short-term and long-term exposure to PM (CD, Tables 13-6 and 13-7). Short-term exposure to PM is related to a number of effects ranging from mortality to morbidity and changes in lung function and respiratory symptoms. The association of PM with mortality is mainly linked to respiratory and cardiovascular causes, which is consistent with the range of observed morbidity effects, from respiratory and cardiovascular-related hospital admissions to changes in lung function. In addition, the CD tables show a number of similar health effects are associated with both long-term and short-term exposure to PM.

This qualitative coherence is further supported by quantitative coherence across several endpoints as demonstrated in Figure V-2 and Table V-10 which also provides some perspective on the baseline incidence for effects of concern. Observations of increases in cardiovascular and respiratory mortality associated with PM should be accompanied by more frequently occurring increases in hospital admissions for the same causes. Table V-10 shows this to be the case. Using the RR estimates developed in Chapter 12, the CD finds about 0.3 respiratory deaths expected per day per million for all age groups attributable to a 50 $\mu g/m^3$ increase in PM. The CD notes a higher expected increase in respiratory-related hospital admissions of 2.0 per day per million in the total population. Similar results are found for cardiovascular deaths, with 0.9 cardiovascular deaths and 2.3 cardiovascular hospital admissions per million per day associated with a 50 $\mu g/m^3$ increase in PM. There are some numerical inconsistencies in Table V-10, but, given the diversity of the studies and analytical methods used to derive the estimates, the coherence between the mortality and morbidity endpoints is consistent with expectations (CD, p. 13-64).

The coherence is further strengthened by multiple studies demonstrating associations with a range of effects in the same population. Studies in Detroit, Birmingham, Philadelphia and Utah Valley show increased frequency of a variety respiratory and cardiovascular related health effects associated with PM exposure in the same population (CD, Section 13.4.3.5). For example, studies in Utah Valley have shown a number of closely related outcomes associated with PM exposures, including decrements in lung function, increased respiratory symptoms, increased medication use in asthmatics, and increased elementary school absences (frequently due to upper respiratory illness). Finally, there is coherence in the sense that the

TABLE V-10. QUANTITATIVE COHERENCE OF ACUTE MORTALITY AND HOSPITALIZATION STUDIES (CD, Table 13-8)

Age Group	Health Endpoint	Population Annual Baseline Per Million Total Population	Population Daily Baseline Per Million Total Population	PM_{10} Lag Time	Excess Risk per 50 $\mu g/m^3$ PM_{10} Incr.	Possible Number of PM-Related Events Per Day Per 1 Mil. Pop. for 50 $\mu g/m^3$ PM_{10} Increment
Whole Population						
All	Total mortality	8,603[1]	23.6	< 2d	0.03[2]	0.7
				3-5d	0.06[2]	1.5
	Total hospit.	124,110[3]	340.0	-	-	-
All	Resp. mortality	676[1]	1.85	3-5d	0.19[4]	0.3
	Total resp. hospitalization	12,180[3]	33.4	< 2d	0.06[5]	2.0
All	Cardiovascular mortality	3,635[1]	10.0	3-5d	0.09[4]	0.9
	Heart disease hospitalization	21,310[3]	58.4	< 2d	0.04[6]	2.3
Elderly						
65+	Total mortality	6,201[7]	17.0	2d	0.06[8]	1.0[8]
	Total hospit.	42,845[9]	117.4	-	-	-
65+	Total resp. hospitalization	5,101[9]	14.0	≤ 1d	0.08[5]	1.1
	Pneumonia hospit.	2,335[9]	6.4	≤ 1d	0.08[10]	0.5
	COPD hospit.	2,560[11]	7.0	≤ 1d	0.16[5]	1.1
	Heart disease hospitalization	13,502[9]	37.0	≤ 1d	0.06[6]	2.2

[1]From National Center for Health Statistics (1993).
[2]From EPA meta-analyses, Table 12-30, models without copollutants.
[3]From Table 12-6, based on first-listed diagnoses for discharges.
[4]From Pope et al. (1991), Schwartz (1993) for Utah Valley and Birmingham, variance-weighted average, Table 12-4.*
[5]From Table 12-8, average.*
[6]From Table 12-11.*
[7]Assuming elderly as 12.6% of 1991 U.S. population.
[8]Based on different set of studies than for above whole population (ALL), i.e., 65+ PM mortality risk from Saldiva et al. (1994) and Ostro et al. (1996) variance-weighted average; Section 12.3.*
[9]From Table 12-6,* assuming 12.6%, age 65+.
[10]From Table 12-10,* average.
[11]From 1992 detailed tables; excludes asthma (ICD 493).
*All Table references to Chapter 12 of the CD.

observed health effects, which are related to respiratory and cardiovascular causes, are those that would most likely to be associated with the inhalation route.

The CD concludes there is evidence for increased health effects risks associated with PM exposure ranging in severity from asymptomatic pulmonary function decrements, to respiratory and cardiopulmonary illness requiring hospitalization, and finally to excess mortality from respiratory and cardiovascular causes (especially in those older than 65 years of age) (CD, p. 13-67). Such a coherence of effect greatly adds to the strength and plausibility of the association (Bates, 1992).

F. Health Effects Associated with Fine and Coarse Fraction Particles

The health effects information summarized in previous sections of this chapter and in the criteria document provides substantial evidence that ambient PM, alone or in combination with commonly occurring pollutant gases, is associated with small but significant increases in mortality and morbidity in some sensitive populations at concentrations below the levels of the current ambient standards for PM. An examination of potential confounders and other methodologic issues associated with these studies suggests that these associations are valid (Section V.E). Taken together, the extensive body of recent epidemiologic studies show both qualitative and quantitative consistency suggestive of causality, although supporting evidence for plausible mechanisms of action that have been hypothesized is lacking in the published literature. The purpose of this section is to examine the health effects evidence most useful in determining which PM measure(s) are the most appropriate surrogate(s) or indicators for those components of PM that are most likely to be associated with the array of health effects discussed in the previous sections of this chapter.

A substantial body of quantitative effects information exists for PM_{10}, which is the indicator most frequently used in recent community studies (CD, Tables 13-3, 13-5). Particle dosimetry and mechanistic considerations continue to suggest that typically occurring ambient particles capable of penetrating to the thoracic regions of the respiratory tract (i.e. $< 10\mu m$ diameter) are of greatest concern to health (Section V-B). As discussed in Chapter IV, PM_{10} occurring in ambient atmospheres is composed of two distinct mass fractions (fine mode and coarse mode fractions). Based on atmospheric chemistry, exposure, and mechanistic considerations, the CD concludes it would be most appropriate to "consider fine

and coarse mode particles as separate subclasses of pollutants" (CD, p. 13-94) and to measure them separately in order to plan effective control strategies.

Accordingly, this section summarizes evidence on the health effects associated with fine and coarse fraction particles[7], with an emphasis on epidemiologic results the criteria document judges as most useful in making quantitative conclusions. While the epidemiological data providing a direct comparison of the health effects of fine and coarse particles are quite limited in comparison to that of PM_{10} (which contains both coarse and fine mode fractions), multiple indicators of fine mass and/or its constituents ($PM_{2.5}$, SO_4, COH, KM, BS) have been associated with short term effects in over 15 different cities on three continents. In addition, in community studies where PM_{10} is known to be dominated by fine (e.g. Philadelphia) or coarse (e.g. Anchorage) particles, some qualitative inferences can be made about the dominant fraction. The following sections review the epidemiologic evidence presented in the CD for health effects associated with fine and coarse mode particles and discusses their implications. The discussion addresses 1) community studies using fine particle indicators, 2) community studies directly comparing fine and coarse fractions, 3) studies of PM_{10} effects in communities with high coarse particle levels, and 4) insights from air quality, toxicology, and controlled human studies on particle characteristics as they relate to the potential toxicity of the two fractions.

The focus of this examination is on evidence that permits a quantitative evaluation of the extent to which fine and coarse fractions of PM_{10} are most likely to be associated with the key health effects categories of mortality, morbidity, symptoms, and functional changes in sensitive populations. This is a more meaningful and tractable comparison than that between PM_{10} and the fine fraction of PM_{10}, which is inherently confounded. Given the profound physicochemical differences between the two subclasses of PM_{10}, it is reasonable to expect some differences may exist in both the nature of potential effects and in the relative concentrations required to produce similar responses. In this regard, components within both pollutant classes could be implicated in causing effects, but the level and nature of risk posed

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[7]Tables 13-6 and 13-7 of the CD provide a qualitative summary of the strength of the epidemiologic evidence for several alternative indicators of PM, including thoracic, fine, coarse, and individual components of fine particles (sulfate and acids).

may vary between the two. In that event, the most appropriate protection from the effects of particles smaller than 10 μm would be provided by consideration of more than one indicator in developing control strategies. (CD, p. 13-94).

1. Epidemiological Studies using Fine Particle Indicators

This section briefly summarizes the epidemiological evidence on the health effects associated with fine particles as measured by a variety of indicators. As noted in the CD (Tables 13-6, 13-7), community studies have shown fine particles to be associated with a range of health outcomes, including mortality in sensitive population groups, increased hospitalization, respiratory symptoms, and decreased lung function. While a number of the studies used an indicator of fine particle mass, such as sulfates, many of them employed $PM_{2.5}$ or $PM_{2.1}$ instruments. These studies are listed in Tables V-11, V-12 and V-13, with key aspects summarized below.

a. Short-Term Studies

Tables V-11 and V-12 lists 18 studies identified in the CD as evaluating short-term associations between mortality and morbidity and a number of different measures of fine particles. Table V-11 lists studies that used filter based optical techniques (BS, KM, COH, see Appendix B), which provide mainly qualitative support for an association of mortality and fine particles, while Table V-12 lists quantitative results from studies reporting gravimetrically measured components that serve as indicators of particles in the fine fraction (i.e. sulfates and acids), and direct measures of $PM_{2.5}$ or $PM_{2.1}$. These tables indicate that statistically significant associations have been found between fine particles and mortality in a number of cities. Six of these studies found statistically significant associations with mortality and fine particles as measured with filter-based optical techniques (BS, KM and COH), while two others could not separate effects of particles from potential confounding by other pollutants (Kinney and Özkaynak, 1991) or the effects of a heat wave (Katsoyanni et al., 1993). More quantitative results on fine particles ($PM_{2.1}$) and mortality are provided by Schwartz et al (1996a), which includes 6 cities (Table V-12). This study is reviewed in detail in the subsection V.F.2 below, along with other studies that provide direct comparison of effects associated with fine and coarse particles.

TABLE V-11. SHORT-TERM EXPOSURE EPIDEMIOLOGICAL STUDIES OF MORTALITY USING OPTICAL FINE PARTICLE INDICATORS*

City	Study Years	Indicator	Reference
Acute Mortality			
London	1963-1972,winters 1965-1972, winters	BS	Thurston et al., 1989 Ito et al., 1993
Athens	1975-1987 July, 1987 1984-1988	BS	Katsouyanni et al., 1990 Katsouyanni et al., 1993 Touloumi et al., 1994
Los Angeles	1970-1979 1970-1979	KM	Shumway et al., 1988 Kinney and Özkaynak, 1991
Santa Clara	1980-1986, winters	COH	Fairley, 1990

*BS, KM, COH are optical measurements that are most directly related to elemental carbon concentrations, but only indirectly to mass (See Appendix B). Site specific calibrations and/or comparisons of such optical measurements with gravimetric mass measurements in the same time and city are needed to make inferences about particle mass. Both the nature of the monitor inlet and the fact that elemental carbon particles are found in the fine fraction mean such measurements reflect variations in fine particle mass (if calibrated) or in that portion of fine particles indexed by elemental carbon (largely primary combustion particles). Comparisons between the respective optical measurements and mass measurements were made for the historical London winters (EPA, 1982a), the Athens studies (Katsouyanni et al., 1995), and Santa Clara (Fairly, 1990). Such comparisons were not reported for the Los Angeles study using KM, but the same investigators also reported significant associations between mortality and PM gravimetric mass in Los Angeles (Kinney et al., 1995).

TABLE V-12. FINE PARTICLE INDICATOR ($PM_{2.5}$, $SO_4^=$, H^+) EFFECTS STUDIES FROM THE U.S. AND CANADA (CD, Tables 13-4, 12-2, 12-13)

	Indicator	RR (± CI) per 25 $\mu g/m^3$ PM Increase	PM Levels Mean (Min/Max)[†]
Acute Mortality			
Six City[A]			
Portage, WI	$PM_{2.5}$	1.030 (0.993, 1.071)	11.2 (±7.8)
Topeka, KS	$PM_{2.5}$	1.020 (0.951, 1.092)	12.2 (±7.4)
Boston, MA	$PM_{2.5}$	1.056 (1.038, 1.0711)	15.7 (±9.2)
St. Louis, MO	$PM_{2.5}$	1.028 (1.010, 1.043)	18.7 (±10.5)
Kingston/Knoxville, TN	$PM_{2.5}$	1.035 (1.005, 1.066)	20.8 (±9.6)
Steubenville, OH	$PM_{2.5}$	1.025 (0.998, 1.053)	29.6 (±21.9)
Increased Hospitalization			
Ontario, CAN[B]	$SO_4^=$	1.03 (1.02, 1.04)	Min/Max = 3.1-8.2
Ontario, CAN[C]	$SO_4^=$	1.03 (1.02, 1.04)	Min/Max = 2.0-7.7
	O_3	1.03 (1.02, 1.05)	
NYC/Buffalo, NY[D]	$SO_4^=$	1.05 (1.01, 1.10)	NR
Toronto, CAN[D]	H^+ (Nmol/m^3)	1.16 (1.03, 1.30)*	28.8 (NR/391)
	$SO_4^=$	1.12 (1.00, 1.24)	7.6 (NR, 48.7)
	$PM_{2.5}$	1.15 (1.02, 1.78)	18.6 (NR, 66.0)
Increased Respiratory Symptoms			
Southern California[F]	$SO_4^=$	1.48 (1.14, 1.91)	R = 2-37
Six Cities[G]	$PM_{2.5}$	1.19 (1.01, 1.42)**	18.0 (7.2, 37)***
(Cough)	$PM_{2.5}$ Sulfur	1.23 (0.95, 1.59)**	2.5 (3.1, 61)***
	H^+	1.06 (0.87, 1.29)**	18.1 (0.8, 5 9)***
Six Cities[G]	$PM_{2.5}$	1.44 (1.15-1.82)**	18.0 (7.2, 37)***
(Lower Resp. Symp.)	$PM_{2.5}$ Sulfur	1.82 (1.28-2.59)**	2.5 (0.8, 5.9)***
	H^+	1.05 (0.25-1.30)**	18.1 (3.1, 61)***
Denver, CO[P]	$PM_{2.5}$	0.0012 (0.0043)***	0.41 - 73
(Cough, adult asthmatics)	$SO_4^=$	0.0042 (.00035)***	0.12 - 12
	H^+	0.0076 (0.0038)***	2.0 - 41
Decreased Lung Function			
Uniontown, PA[E]	$PM_{2.5}$	PEFR 23.1 (-0.3, 36.9) (per 25 $\mu g/m^3$)	25/88 (NR/88)
Seattle, WA[Q]	b_{ext}	FEV1 42 ml (12,73)	5/45
Asthmatics	calibrated by $PM_{2.5}$	FVC 45 ml (20,70)	

References:

[A]Schwartz et al. (1996a)
[B]Burnett et al. (1994)
[C]Burnett et al. (1995) O_3
[D]Thurston et al. (1992, 1994)
[E]Neas et al. (1995)
[F]Ostro et al. (1993)
[G]Schwartz et al. (1994)
[P]Ostro et al. (1991)
[Q]Koenig et al. (1993)

[†]Min/Max 24-h PM indicator level shown in parentheses unless otherwise noted as (± S.D.), 10 and 90 percentile (10,90).
[*]Change per 100 nmoles/m^3.
[**]Change per 20 $\mu g/m^3$ for $PM_{2.5}$; per 5 $\mu g/m^3$ for $PM_{2.5}$ sulfur; per 25 nmoles/m^3 for H^+.
[***]50th percentile value (10,90 percentile).
[****]Coefficient and SE in parenthesis.

Nine studies in the U.S. and Canada have found positive associations between short-term exposure to gravimetrically measured fine particles or components (including sulfates and acids) and indicators of morbidity, including increased hospital admissions, increased respiratory symptoms and decreased lung function (Table V-12). All the studies found a positive association between $PM_{2.5}$ and measured health effects; in eight of the studies the associations were significant. A particularly informative study was conducted by Thurston et al. (1994b) in Toronto, which evaluated the associations of respiratory-related hospital admissions with a range of particle indicators. This study is discussed below in subsection V.F.2.

b. Long-Term Studies

Table V-13 lists the studies the CD finds most useful for presenting quantitative estimates of effects associated with long-term exposure to PM (CD, Table 13-5). Two recent prospective studies, the Six City Study and the ACS study, reflect significant methodological advances over earlier cross-sectional studies and provide the best evidence of the association between long-term PM exposure and mortality. The relative strength of the results for fine and coarse indicators is discussed below in subsection V.F.2.

The designs and approaches of the Six City and ACS studies are complementary in nature (See Section V-13). The Six City study provided a more complete consideration of co-occurring pollutants that might confound the results (O_3, SO_2, NO_2), but lacked some power due to the limited number of cities and the size of the total population included. The ACS study was designed to test the major hypothesis derived from the Six City study, namely that long-term exposure to fine particles (as $PM_{2.5}$ or sulfates) was associated with increased mortality. The ACS design improved upon the Six City study by evaluating a larger population in many more cities across the U.S. (151) but, based on the earlier findings, did not include multiple pollutants. The ACS study found a significant association between mortality and both $PM_{2.5}$ and sulfates (Table V-13). For reasons discussed in Section V.C., the staff concludes the somewhat smaller effects estimates from the ACS study are likely more useful for risk assessment of long-term mortality than those from the Six City study. In addition, consideration must be given to the role of earlier exposures to higher concentrations with respect to the applicability of these estimates based on a few years of

TABLE V-13. EFFECT ESTIMATES PER INCREMENTS[a] IN ANNUAL MEAN LEVELS OF FINE/THORACIC PARTICLE INDICATORS FROM U.S. AND CANADIAN STUDIES (CD, Table 13-5).

Type of Health Effect & Location	Indicator	Change in Health Indicator per Increment in PM[a]	Range of City PM Levels Means ($\mu g/m^3$)
Increased total chronic mortality in adults		Relative Risk (95% CI)	
Six City[b]	$PM_{15/10}$	1.42 (1.16-2.01)	18-47
	$PM_{2.5}$	1.31 (1.11-1.68)	11-30
	$SO_4^=$	1.46 (1.16-2.16)	5-13
ACS Study[c] (151 U.S. SMSA)	$PM_{2.5}$	1.17 (1.09-1.26)	9-34
	$SO_4^=$	1.10 (1.06-1.16)	4-24
Increased bronchitis in children		Odds Ratio (95% CI)	
Six City[d]	$PM_{15/10}$	3.26 (1.13, 10.28)	20-59
Six City[e]	TSP	2.80 (1.17, 7.03)	39-114
24 City[f]	H^+	2.65 (1.22, 5.74)	6.2-41.0
24 City[f]	$SO_4^=$	3.02 (1.28, 7.03)	18.1-67.3
24 City[f]	$PM_{2.1}$	1.97 (0.85, 4.51)	9.1-17.3
24 City[f]	PM_{10}	3.29 (0.81, 13.62)	22.0-28.6
Southern California[g]	$SO_4^=$	1.39 (0.99, 1.92)	—
Decreased lung function in children			
Six City[d,h]	$PM_{15/10}$	NS Changes	20-59
Six City[e]	TSP	NS Changes	39-114
24 City[i,j]	H^+ (52 nmoles/m^3)	-3.45% (-4.87, -2.01) FVC	—
24 City[i]	$PM_{2.1}$ (15 $\mu g/m^3$)	-3.21% (-4.98, -1.41) FVC	—
24 City[i]	$SO_4^=$ (7 $\mu g/m^3$)	-3.06% (-4.50, -1.60) FVC	—
24 City[i]	PM_{10} (17 $\mu g/m^3$)	-2.42% (-4.30, -.0.51) FVC	—

[a]Estimates calculated annual-average PM increments assume: a 100 $\mu g/m^3$ increase for TSP; a 50 $\mu g/m^3$ increase for PM_{10} and PM_{15}; a 25 $\mu g/m^3$ increase for $PM_{2.5}$; and a 15 $\mu g/m^3$ increase for $SO_4^=$, except where noted otherwise; a 100 nmole/m^3 increase for H^+.
[b]Dockery et al. (1993)
[c]Pope et al. (1995)
[d]Dockery et al. (1989)
[e]Ware et al. (1986)
[f]Dockery et al. (1996)
[g]Abbey et al. (1995a,b,c)
[h]NS Changes = No significant changes.
[i]Raizenne et al. (1996)
[j]Pollutant data same as for Dockery et al. (1996)

monitoring (CD, P 12-366). If the effects are the result of long-term exposures, as opposed to the sum of episodic or daily effects, then the reported relative risk estimate are apt to be high.

Cross-sectional studies conducted by Özkaynak and Thurston (1987, 1989) and Lipfert (1988) provide some additional insights into the relationship between long-term exposure to fine particle indicators and mortality. Özkaynak and Thurston's cross-sectional analysis of various particle measures and 1980 total mortality across US cities found the most consistent and significant associations with fine particles and sulfates. In their analysis, TSP and PM_{15} were often found to be nonsignificant predictors of mortality. Lipfert also analyzed 1980 total mortality across US cities in relation to different particle measures (CD, p. 12-15). In general, when evaluating single site TSP or PM_{15} and sulfates or $PM_{2.5}$ in models with the same covariates, the effects estimates for sulfates and fine particles were generally larger than those for TSP or PM_{15}. Some model specifications also show significant associations between mortality and multi-station TSP. A supplemental analyses of the Lipfert 1980 data in the CD found that the introduction of numerous potentially confounding variables (e.g. water hardness, sedentary lifestyle) reduced but did not eliminate the $PM_{2.5}$ effect on mortality (CD, Fig 12-7)[8]. Clearly there are inherent methodological issues with these ecological approaches, but they show evidence of associations between long term measures of fine particles, including sulfates, and mortality that are quantitatively more consistent with the lower risk estimates found in the ACS study (CD, p 12-177).

Several studies have evaluated the association between long-term fine particle exposure and increased respiratory symptoms and decreased lung function most which have been conducted in children (Table V-13). The 24 city studies are of particular interest. These studies evaluated the association between different measures of long term PM (PM_{10}, $PM_{2.5}$, SO_4 and H^+) and respiratory symptoms and pulmonary function in children (Raizenne 1996; Dockery et al. 1996). The one year surveys found a significant increase in bronchitis

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[8]In this example, the $PM_{2.5}$ effect was reduced from 0.045 to 0.02 deaths per $\mu g/m^3$. While it is likely, that addition of some of these variables to the Six Cities and ACS cohort studies would reduce the effects estimates for these two studies as well, the relevance and independence of including all of their variables (e.g., sedentary lifestyle and overweight) can be questioned.

in children (one episode or more) associated with particle strong acidity and fine particulate sulfates. Elevated, but nonsignificant associations were observed between reporting a bronchitis and $PM_{2.5}$ and PM_{10}. No other respiratory symptoms, including asthma symptoms, were significantly associated with any of the pollutants.

In contrast to the earlier 6 city results, annual mean particle strong acidity, total sulfates, $PM_{2.5}$ and PM_{10} were all significantly associated with FVC and FEV1 deficits (Table V-13). A slightly larger FVC decrement was found for children who were lifelong residents of their communities, though it was not significantly different. For the 24 cities, there was a strong correlation between particle strong acidity and sulfates ($r=0.90$) and PM2.1 ($r=0.82$), but not with PM_{10} ($r=0.47$). Thus, it is difficult to ascribe the association to any one of the 3 fine particle indicators.

2. Community Studies Comparing Effects of Fine and Coarse Fraction PM

Several studies provide quantitative information directly comparing the association between health effects and fine and coarse particles. They include an examination of short-term PM exposure mortality in the Harvard six cities (Schwartz et al., 1996), a short-term exposure hospital admission study (Thurston et al., 1994b), and the long-term exposure mortality Six City Study (Dockery et al., 1993). Supporting information on long term effects can also be found in the data from the ACS study (Pope et al., 1995b) and the 24 city study reports (Spengler et al, 1996; Dockery et al., 1996; Razienne et al, 1996).

a. Short-Term Comparisons

A recent analysis of mortality in six cities by Schwartz et al (1996) evaluated the association between mortality and 5 different particle measures: coarse fraction particles ($PM_{15/10}$ minus $PM_{2.5}$); thoracic particles ($PM_{15/10}$), $PM_{2.5}$, Sulfates, and H+. Table V-14 highlights the results for coarse fraction, thoracic, and $PM_{2.5}$ particles. The estimated increase in mortality associated with $PM_{10/15}$ was positive in all the cities except for Topeka, where there was no association (Table V-3). In all of the other cities, the observed increases ranged from 3.0 to 6% for a 50 μg/m increase in PM_{10} (Table V-3), consistent with the range reported for previous PM_{10} studies (2 to 9%). A graphical display of the results for the components of PM_{10} suggests, however, that most, if not all, of the PM_{10} effect in these cities appears to be due to fine particles (Figure V-4). The estimated increase in daily

TABLE V-14. ESTIMATED INCREASE IN DAILY MORTALITY, 95% C1, AND t STATISTIC BY CITY AND COMBINED ESTIMATE ASSOCIATED WITH A 10 $\mu g/m^3$ INCREASE IN PARTICULATE MASS CONCENTRATIONS. EFFECT OF EACH PARTICLE MASS MEASURE ASSOCIATIONS ESTIMATED SEPARATELY, CONTROLLED FOR LONG-TERM TRENDS AND WEATHER.

Study City	Correlation $PM_{2.5}$/CM	$PM_{2.5}$	CM	PM_{10}
Boston	0.23	2.2% (1.5%, 2.9%) t=6.31	0.2% (-0.6%, 1.2%) t=0.58	1.2% (0.7%, 1.7%) t=4.86
Knoxville	0.44	1.4% (0.2%, 2.6%) t=2.26	1.0% (-0.6%, 2.6%) t=1.20	0.9% (0.1%, 1.8%) t=2.21
St. Louis	0.45	1.1% (0.4%, 1.7%) t=3.17	0.2% (-0.7%, 1.1%) t=0.45	0.6% (0.1%, 1.0%) t=2.42
Steubenville	0.69	1.0% (-0.1%, 2.1%) t=1.79	2.4% (0.5%, 4.3%) t=2.43	0.9% (0.1%, 1.6%) t=2.17
Portage	0.32	1.2% (-0.3%, 2.8%) t=1.64	0.5% (-1.2%, 2.3%) t=0.57	0.7% (-0.4%, 1.7%) t=1.22
Topeka	0.29	0.8% (-2.0%, 3.6%) t=0.53	=1.3% (-3.3%, 0.6%) t=1.32	-0.5% (-2%, 0.9%) t=0.67
All Cities Combined				
Total Mortality		1.5% (1.1%, 1.9%) t=7.13	0.4% (-0.1%, 1.0%) t=1.48	0.8% (0.5%, 1.1%) t=5.84
Ischemic Heart Disease		2.1% (1.5%, 2.7%) t=7.12		
Chronic Obstructive Pulmonary Disease		3.3% (1.0%, 5.7%) t=2.79		

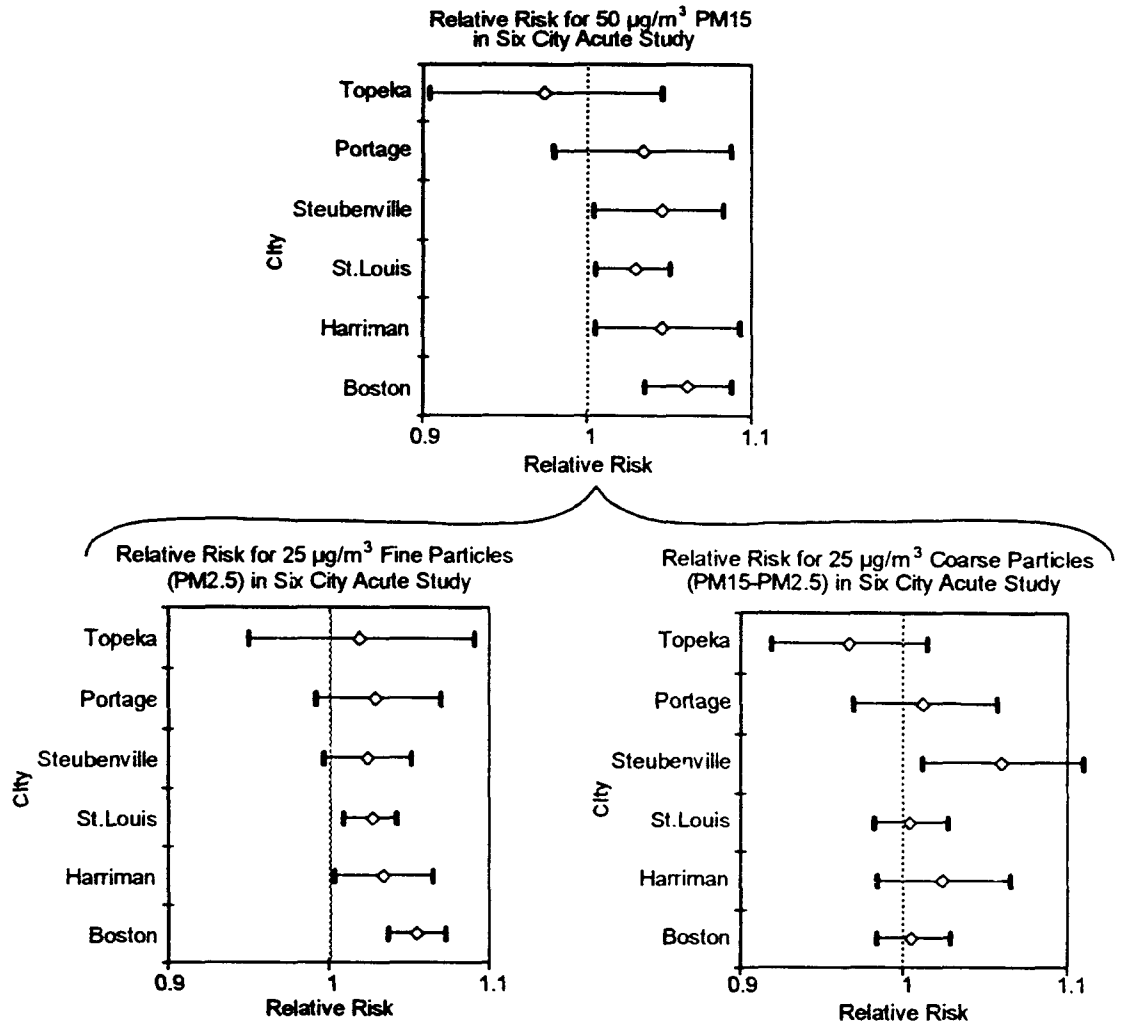

Figure V-4. Relative risks of acute mortality in the Six City Study, for thoracic particles (PM_{10}, PM_{15}), fine particles ($PM_{2.5}$) and coarse fraction particles (PM_{15}-$PM_{2.5}$). The coarse fraction effects are small and insignificant, except in Steubenville, where there is a high correlation between fine and coarse particles (R2=0.69). In Topeka, which has the second highest level of coarse fraction particles, the association is negative and nearly significant.

Source: CD, Figure 12-33. U.S. EPA graphical depiction of results from Schwartz et al. (1996).

mortality associated with $PM_{2.5}$ was consistently positive in all 6 cities (0.8 to 2.2% for a 10 $\mu g/m^3$ $PM_{2.5}$ increase) and statistically significant in 3 cities. In contrast, the relative risks for mortality associated with coarse particles was inconsistent across the 6 cities (-1.3% to 2.4% for a 10 $\mu g/m^3$ increase in coarse particles) (Table V-14). The association with coarse particles was significant only in Steubenville, but it is difficult to interpret these results given the high correlation between fine and coarse particles (r=0.69) in this city. All of the other cities have r of 0.45 or less. The negative but non-significant association between PM_{10} and mortality in Topeka noted above appears to be driven by the coarse fraction. Although Topeka has the highest percentage of crustal particles and the second highest average coarse mass, coarse particles have a nearly significant negative association with mortality, while fine particles have a positive but non-significant association. While greater measurement error for the coarse fraction (see Section V.E above) could depress a potential coarse particle effect, this would not explain the results in Topeka relative to other cities. Even considering relative measurement error, these results provide no clear evidence implicating coarse particles in the reported effects.

In a combined analysis across the 6 cities, $PM_{2.5}$ was significantly associated with an increase in mortality of 2.1% (CI 1.5% to 2.6% for a 25th to 75 percentile increase in $PM_{2.5}$). In contrast, the coarse particles were associated with a small but insignificant increase in mortality, 0.4% (CI -0.1% to 1.0%, for a 25th to 75th percentile increase in coarse particles). To determine whether coarse particles were independently associated with mortality, both fine and coarse particles were considered simultaneously in the regression across all six cities. The estimated effect for $PM_{2.5}$ across the interquartile range remained unchanged with a significant association with mortality (2.1%, CI 1.5% to 2.6%). Conversely, the coarse particle estimate was substantially lowered (-0.2%, CI -0.8% to 0.4% for the interquartile range). This study provides clear evidence that fine particles are more likely to be responsible for the numerous observed associations between PM_{10} and mortality. The study also evaluated the association with fine particles by age and cause of death. Similar to studies of PM_{10} and mortality, a higher RR estimates for deaths from ischemic heart disease and deaths from chronic obstructive pulmonary disease was found in their

analysis (Table V-14). The authors note that this is a similar pattern to that seen in London during the 1952 dramatic pollution episode.

Thurston et al. (1994b) evaluated the association between summertime respiratory and asthma related hospital admissions and 5 different particle measures: acids, sulfates, fine particles, coarse particles and PM_{10}. Without adjusting for the risk associated with concurrent O_3 levels, the investigators found a significant association between respiratory-related hospital admissions and all measures of particles except the coarse fraction. Only fine acids and sulfates were significantly associated with asthma admissions in the univariate models. When O_3 was included in the model, only acids and sulfates remained significantly associated. The authors note the high correlations between the other particle measures and O_3 concentration make it difficult to select a best indicator, but these results provide no evidence of a coarse particle association with respiratory admissions in an area meeting the PM_{10} standards. The authors conclude that, based on the relative strengths of hospital admissions associations, the particle indicator, could be ranked as $H+ > \text{sulfates} > PM_{2.5} > PM_{10}$.

b. Long-Term Comparisons

The Six City study evaluated the relationship between mortality and long-term exposure to particles using several indicators; total particles, inhalable particles, fine particles, coarse particles, sulfate fine particles and non-sulfate fine particles (Dockery et al., 1993). Figure V-5 plots the relationship between mortality risk and each of the particle indicators. Although such comparisons involving only 6 cities should be viewed with caution, there is a trend toward increasing associated of relative risk of mortality with the particle indicator as the size of the particle indicator decreases (CD, Chapter 13). Although some association is apparent for TSP alone, the "super-coarse" fraction of particles larger than 10-15 μm does not appear to be clearly linked with mortality, particularly in areas other than Steubenville. This further supports the notion that extrathoracic particles present a lower risk than thoracic PM. The distinction between $PM_{2.1}$ and coarse fraction ($PM_{10-2.1}$) particles is less clear, although -- as was the case in the short term mortality results above -- the relative risk for the city with the highest proportion of crustal materials (Topeka) appears

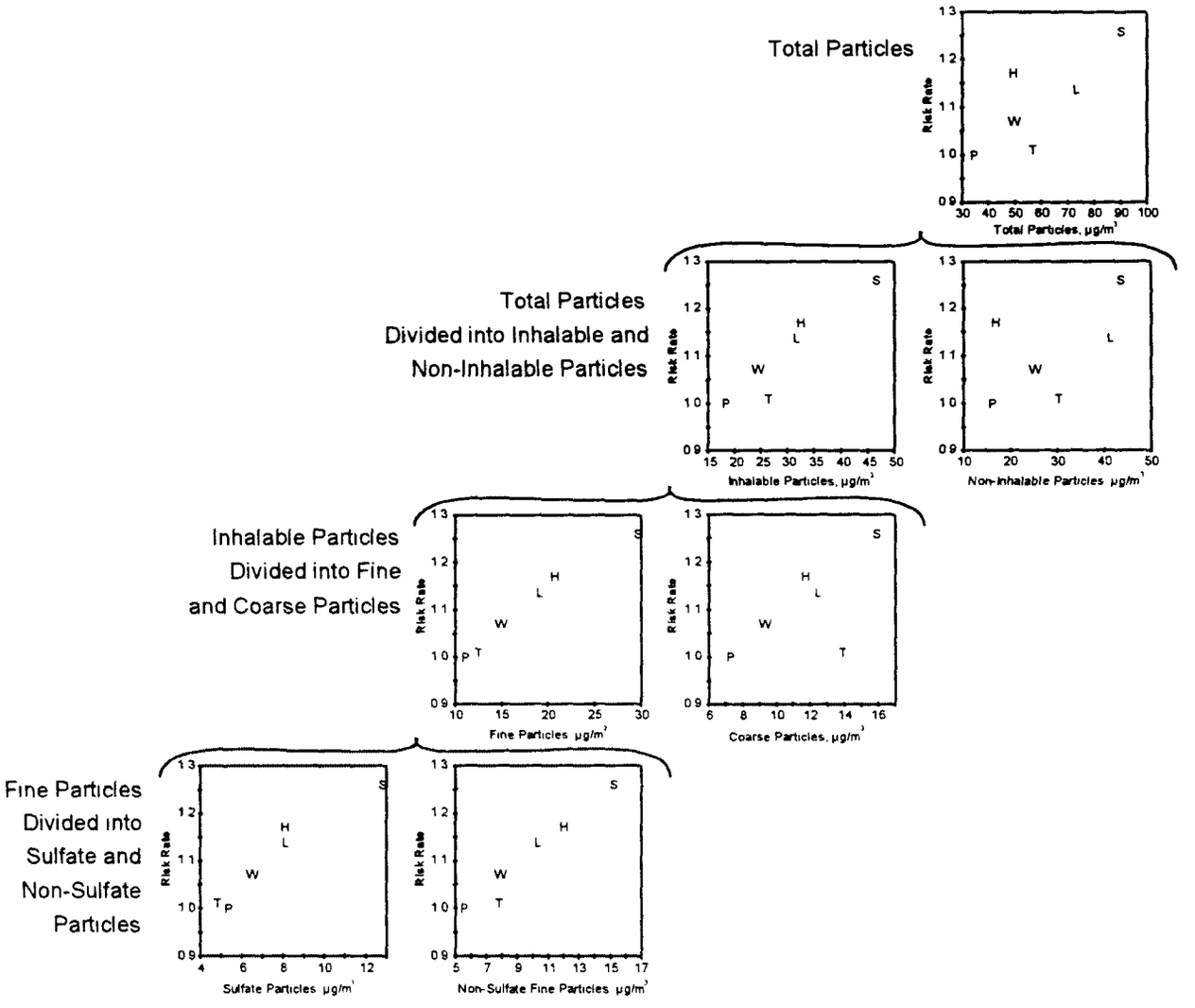

Figure V-5. Adjusted relative risks for mortality are plotted against each of seven long-term average particle indices in the Six City Study, from largest range (total suspended particles, upper right) through sulfate and nonsulfate fine particle concentrations (lower left). Note that a relatively strong linear relationship is seen for fine particles, and for its sulfate and non-sulfate components. Topeka, which has a substantial coarse particle component of inhalable (thoracic) particle mass, stands apart from the linear relationship between relative risk and inhalable (thoracic) particle concentration. Some gradient exists for all indicators with respect to Steubenville and Portage..

Source: CD, Figure 12-8. U.S. EPA replotting of results from Dockery et al. (1993).

to be more consistent with a fine particle effect. For the other cities, there is less difference between fine and coarse rankings.

Some additional insight into the Six City results is found in an ecological analysis of data from the ACS study (Pope et al., 1995b). Figure V-6 shows scatterplots of adjusted mortality and PM as indicated by sulfate and TSP taken from the ACS study. These figures show a pattern consistent with a sulfate mortality effect across a large number of cities, but no clear relationship for TSP. The relative position of the six cities in these figures shows that, consistent with the original study design (Ferris et al, 1986), which selected cities to show gradients in both TSP and sulfur oxides, the mortality risk in the six cities shows an apparent relationship with both sulfates and TSP. The similarity in gradients for mortality for both fine particles (sulfates) and TSP in the six cities is not typical of the full set of 151 cities in the ACS study. Given the strong significant association between fine particles and mortality in the full ACS and Six City cohort studies and the lack of significant association with TSP in the ACS data (Pope et al., 1995b), the evidence for chronic mortality effects appears to be stronger for fine particles than for coarse.

Both the ACS study and the Six City study found the increase in risk of mortality associated with fine particle matter was mostly attributed to increases in cardiopulmonary mortality. As noted in Section 5.C, the Harvard Six City study reported a 37 percent increase in cardiopulmonary mortality associated with $PM_{2.5}$, and the ACS study reported a 31 percent increase in cardiopulmonary mortality associated with $PM_{2.5}$.

The negative results of the third prospective cohort study (Abbey et al, 1991) do not diminish the above conclusions. As noted in section V-C, despite the theoretically improved approach to exposure classification in this study (CD, p. 12-162), the choice of PM indicator (days $>200\ \mu g/m^3$ as TSP) for a large number of California sites limits the inferences that can be made about smaller particles sizes. Peak TSP in various times and places in California may be associated with coarse agricultural or road dust or high photochemically derived fine particles. Unlike other national cross sectional comparisons that use mean TSP from multiple monitors in metropolitan areas spanning the East and Midwest U.S. (e.g. Lipfert, 1993), peak TSP in California is less likely to be a useful surrogate for fine or thoracic particles. Thus, while neither this study nor the ACS study finds a significant

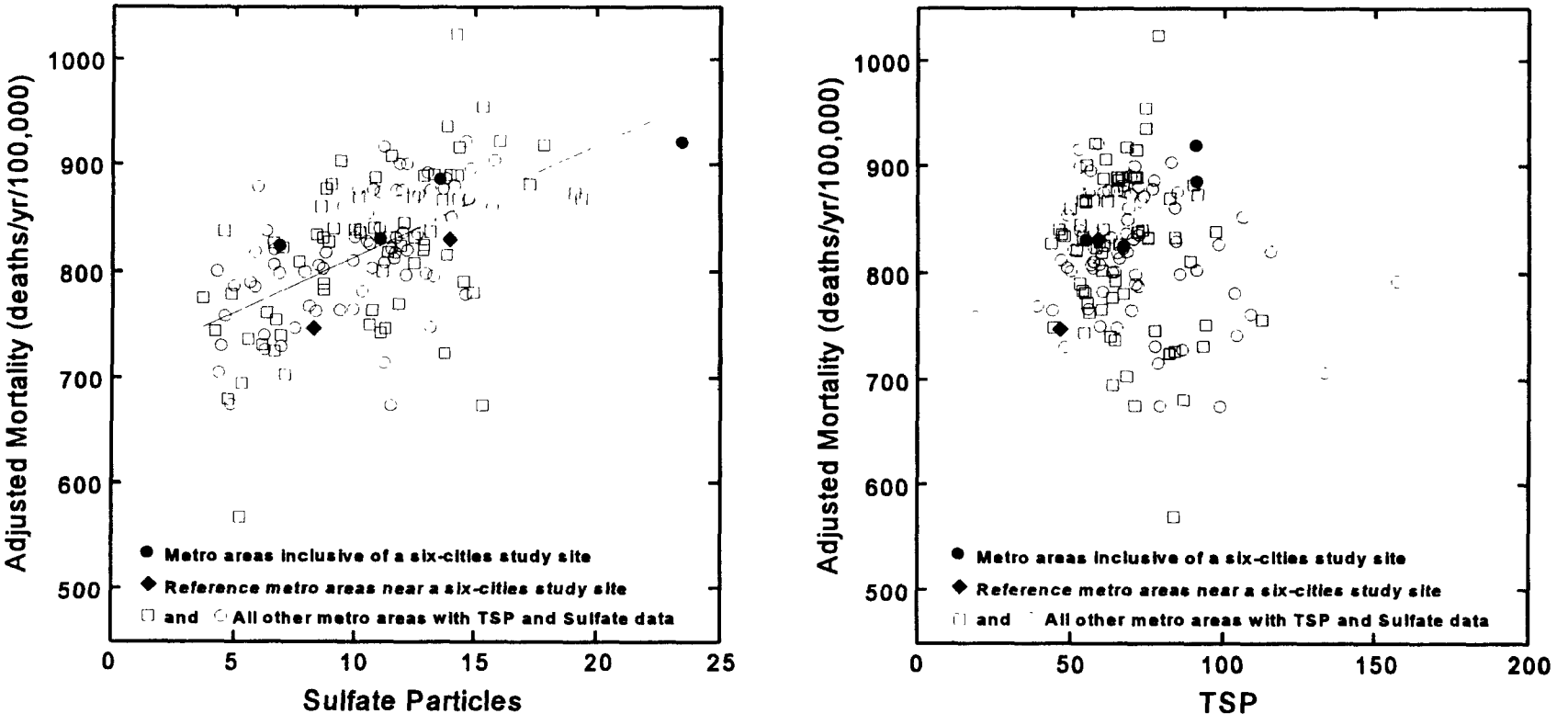

Figure V-6. . Age-sex-race adjusted mortality rates from ACS study plotted against mean sulfate and TSP levels for U.S. metropolitan areas (Pope, 1995). This ecologic comparison shows a clear association betwen mortality and sulfates but not for TSP. Metro-areas in or near the Harvard six cities, however, show a gradient for both sulfate and TSP, reflecting the original city selection criteria..

mortality effect of long term exposures to TSP, only the ACS study tested this hypothesis with respect to fine particles using appropriate measurements.

Staff also further examined the data in the 24 city studies of the effects of PM on lung function in children (Raizenne et al., 1996). As noted above, the authors report significant associations between lung function and strong acids, sulfates, $PM_{2.1}$, and PM_{10}, but did not report on any analyses for coarse fraction particles. Figure V-7 plots the lung function results for the 22 cities where such data were taken against both $PM_{2.1}$ and coarse fraction ($PM_{10-2.1}$). The lack of any significant association of coarse particles is apparent. The careful selection of the cities and study participants was intended to provide a clear gradient across regions with elevated fine acid aerosols and areas with lower levels, and to provide for a separation of potential O_3 and PM effects. Multiple pollutants and indoor conditions were considered. The use of children of similar socioeconomic status and race reduces much of the confounding. This study provides clear evidence of an effect of fine particles that is independent of coarse fraction particles.

A longitudinal study by Johnson et al. (1990) in five Montana cities evaluated the association between lung function and TSP, fine and coarse particles in school children over one school year. They found significant decrements in FEV1 for TSP and significant decrements in FVC for fine particles, but at best, results were insignificant and inconsistent in effects for coarse particles.

3. Epidemiological Studies of Areas Dominated by Coarse Particles

The studies discussed in Section V.F.2 above are the only ones cited in the CD to have evaluated the association between directly measured coarse particles and health effects. In general, such studies have found equivocal results, suggesting an inconsistent or insignificant association between coarse particles and mortality and morbidity. However, with the possible exceptions of Steubenville and Topeka, the concentrations of coarse particles were relatively low and below those of fine particles, and measurement error could have influenced the results. The CD identifies only two additional studies as suggesting morbidity effects associated with short-term episodes of coarse particles (p. 13-47). In these cases, coarse particles were not measured, but ancillary evidence indicates that measured

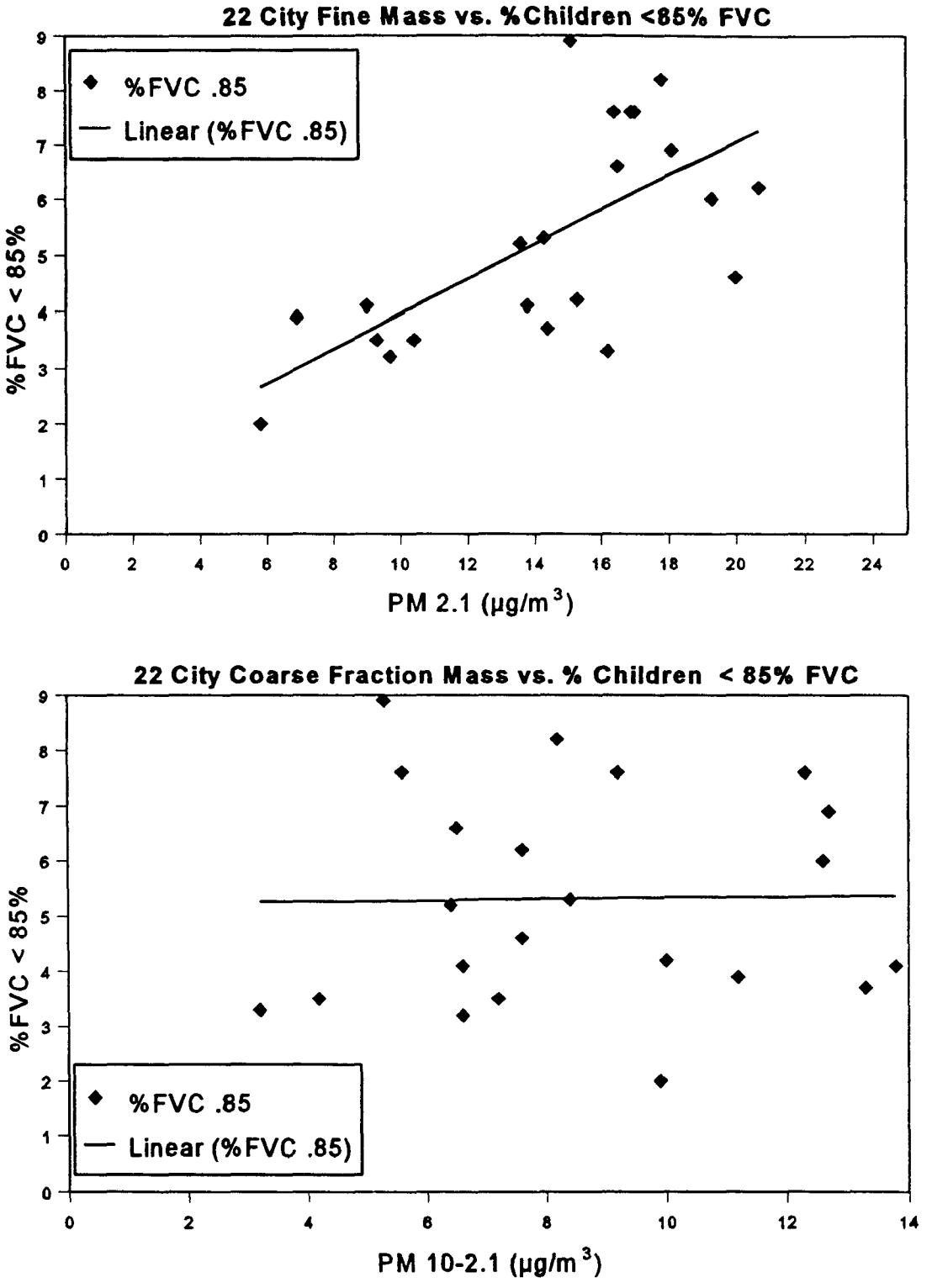

Figure V- 7. % of Children with <85% Normal FVC vs. Annual Fine and Coarse Fraction Mass in 24 City Study. (EPA graphical depiction of results from Raizenne et al., 1996; Spengler et al, 1996). The relationship between fine mass and lung function decrement is significant. No clear relation is shown for coarse fraction particles, which are generally at low concentrations in these cities.

PM_{10} is likely to be dominated by coarse particles, at least during significant episodes or seasons..

A study in Anchorage, Alaska evaluated the association between PM_{10} and daily outpatient visits taken from insurance claims for employees for the State of Alaska and the Municipality of Anchorage (Gordian et al, 1996). They collected data on asthma, bronchitis, COPD, congestive heart failure, diarrhea and upper respiratory illness (defined as upper respiratory problems such as sore throat, sinusitis, earaches, rhinitis, and other nonspecific upper airway problems). They were not able to evaluate COPD and congestive heart failure because of insufficient number of cases. The investigators report that there are no industrial sources of the fine portion of PM_{10} in Anchorage, and the scanning electron microscopy of 10 random samples found over 80% of the PM_{10} mass was between 2.5 to 10 μm in diameter. Daily PM_{10} values ranged from 5 to 565 $\mu g/m^3$ (corresponding to a volcanic eruption), with an average over the 22-month study period of 45.5 $\mu g/m^3$. Gordian et al., report a 3-6% increase in visits for asthma and a 1-3% increase in visits for upper respiratory illness associated with 10 $\mu g/m^3$ increase in PM_{10}. They found no association with visits for bronchitis. They also found a nonsignificant association with PM_{10} in the period immediately after a volcanic eruption, and significant associations in the period excluding the volcanic eruption. The authors suggest that personal intervention minimized exposure after the eruption.

Hefflin et al., (1994) evaluated the potential influence of dust storms on emergency room visits for respiratory disorders in three Southeast Washington State communities. The investigators report that particle exposure is mostly from windblown soil and related natural crustal materials (the majority volcanic in origin). Thus, PM is likely dominated by coarse particles. This area also had high levels of PM_{10}, with peak 24-hour values ranging from 1 to 1,689 $\mu g/m^3$ with an average of 40 $\mu g/m^3$. Aside from the periodic dust storms, the authors provide no additional evidence regarding the size composition of PM_{10} (e.g. extent of wood stoves, other sources). In contrast to Gordian, Hefflin et al. found a significant 0.35% increase in emergency room visits for bronchitis associated with a 10 $\mu g/m^3$ increase in PM_{10}. They also found a significant 0.45% increase in emergency room visits for sinusitis for a 10 $\mu g/m^3$ increase in PM_{10} levels over 150 $\mu g/m^3$. There was no association with

asthma. They found a slight association between emergency room visits and two high dust storms days where particle concentrations were over 1,035 and 1,689 $\mu g/m^3$, but suggested that the reduced unit risk could have been related to mitigating behavior in these severe conditions.

These studies are suggestive of potential associations between high concentrations of coarse particles and health effects, but with some inconsistencies. The effects estimates for the Hefflin et al. study are much smaller than the Gordian et al. study. In addition, the Gordian et al. study found an association between PM-10 and asthma but not with bronchitis, and the Hefflin study found the opposite. This contrast should be interpreted cautiously due to possible difference in disease classifications int he two study areas. Hefflin et al. (1994) have found overall asthma incidences in the region to be lower than expected, reducing the power of the study to detect effects. Both studies report multiple exceedences of the PM_{10} standard. The apparent diminished response of the very highest days suggests that mitigative measures such as staying indoors on days of perceived dust episodes offered some protection against the effects of coarse particles on asthma and upper respiratory illness. Based on the Gordian results and the potential for significant deposition of coarse particles in the tracheobronchial regions of the lung where they may irritate sensitive receptors in asthmatics, the CD concludes that particles in the coarse fraction appear to be associated with the exacerbation of asthma via ambient exposure (CD, p. 13-51).

4. Relevant Physicochemical Differences between Fine and Coarse Fraction Particles

Current understanding of the toxicology of ambient PM suggests that fine and coarse particles may have different biological effects (CD, p. 13-91). The discussion below summarizes information the CD presents regarding differences in potential toxicity between the two fractions based on composition and size related properties.

a. Comparisons of fine and coarse component toxicity in laboratory studies

A comparison of the major components of typical ambient particles (Table IV-2) and the size and composition of particles studied in the recent toxicologic literature (CD, Chapter 11) suggests that, while substantial work has been conducted on simulated constituents of fine particles such as acid aerosols, trace elements, and components of diesel particles, very little attention has been focused on health effects from exposure to ambient coarse particles or

their significant components. The only study in humans of a coarse aerosol (10 μm diameter NaCl, see Table IV-2) cited in Chapter 11 (CD, Table 11-1) was considered to be a control for an acid fog exposure. Furthermore, because of size limitations of particles that can appreciably deposit in the tracheobronchial and alveolar region in small laboratory animals, most experimental animals studies involve fine particle exposures (CD, p. 13-44). The most clear and relevant comparison between the different constituents typically found in the fine and coarse fractions of PM was that of Kleinman et al (1995), who found that the relative cellular and immunological toxicity of fine particle components, sulfate (70 $\mu g/m^3$, 0.2μm diameter $(NH_4)_2\ SO_4$) and nitrate (350 $\mu g/m^3$, 0.6μm diameter NH_4NO_3) were greater than that of a typical resuspended coarse fraction component - road dust (900 $\mu g/m^3$, 4μm diameter), in the rat. While it is clear from the results of the study that the road dust elicited effects and was present in some concentration in thoracic region of the rat, the extent of deposition was not given in the study and it is possible that some of the differential toxicity shown between fine and coarse particle constituents in this study are due to differential penetration efficiencies of the particles.

Chapter 11 of the CD highlights the results of a volcanic ash study (Raub et al, 1985) as a comparison of fine and coarse mode particles. This study used intratracheal instillation of large amounts of 12.2 μm and 2.2 μm diameter volcanic ash into rats. The authors report finding a number effects at the higher concentration used, but essentially no difference in several measures of toxicity. While these result are of interest, the 2.2 μm particles should not be characterized as fine mode, but rather as the "tail" of the coarse mode. Thus, this study suggests little or no difference in the toxicity of coarse mode particles of different sizes, but even this conclusion is limited by the artificial nature by which the particles were deposited in the animals.

Raub et al. (1985) also found no differences in toxic responses between normal and emphysemic animals inhaling 9600 $\mu g/m^3$ submicrometer sized volcanic ash for short durations. Mauderly (1990) found that emphysematous rats had less effects than normal animals because of the sparing effects of emphysema to high levels of diesel particles. However, Raabe et al. (1994) exposed rats with induced emphysema to two fine particle mixtures intended to simulate a London aerosol (ammonium sulfates, coal fly ash, lamp black

carbon) and a California aerosol (ammonium sulfates and nitrate, graphitic carbon, clay, and trace metal sulfates). Even at the lowest levels tested (550 -800 $\mu g/m^3$), 3 to 30 day exposures resulted in significant responses that were greater than those seen in normal animals (CD, p 11-176).

b. Toxicity of Fine and Coarse Mode Chemical Components

Table IV-2 lists the key differences in chemical composition of fine and coarse particles. The CD review highlights a number of specific components of PM that could be of concern to health, including typically fine components (e.g., acids, certain metals, diesel particles, and ultrafines), and typically coarse components (e.g., silica and bioaerosols). It is clear that components of both modes can produce responses, although in general, the fine mode appears to contain more of the irritant substances potentially linked to the kinds of effects observed in the epidemiological studies. The following is a brief summary of the potential toxicity associated with fine and coarse substances.

Most of the aerosol acidity is contained in the fine fraction. Section V-C details a variety of effects associated with acids in community epidemiology and at high levels in laboratory studies. Acids may produce effects as liquid droplets or surface coatings in mixtures. For example, Chen et al. (1990) exposed guinea pigs to fly ash derived from either low or high sulfur coal. The acidity of the resulting particles was proportional to sulfur content with the greatest pulmonary functional response noted for the high sulfur fly ash.

Acid aerosol exposure has been associated with changes in airway morphology as well as airway responsiveness (Gearhart and Schlesinger, 1988; Kleinman et al., 1995; Chen et al., 1992b; Gearhart and Schlesinger 1986; and El-Fawal and Schlesinger, 1994) in experimental animals. Markers of cytotoxicity and increased cellular permeability, following a single exposure to fine or ultrafine H_2SO_4 aerosols, have also been reported (Chen et al., 1992a). Levels of biological mediators of inflammatory responses, as well as smooth muscle tone, have been shown to be altered after exposure to fine acid aerosols (0.3 μm diameter) and lavage. Fine acid aerosol exposure has been shown to alter macrophage function, production of tumor necrosis factor cytotoxic activity, and superoxide radical production, all of which are related to host defense mechanisms. Fine aerosols of ammonium sulfate and

nitrate at relatively low levels have also been shown to alter antigen binding and respiratory burst activity by macrophages (Kleinman et al., 1995).

As noted in the 1982 Staff Paper, extractable organic matter from particles with potential carcinogenic activity is also preferentially derived from the fine fraction. The CD (p. 5-10) notes that the majority of diesel exhaust particles is in the fine mode and both short and long term inhalations of diesel particles are associated with respiratory effects at higher than ambient levels in experimental animals. Occupational studies report (at levels higher than ambient concentrations) bronchitis, impaired respiratory function, cough, and wheezing (CD, Table 11-11), all of which have been reported in community air pollution studies of PM.

Ultrafine aerosols (<0.1 μm) are a class of fine particles that have the potential to cause toxic injury to the respiratory tract as seen in studies conducted both *in vivo* and *in vitro* (CD, p. 13-76). An important aspect of their potential toxicity is their relatively low solubility (CD, p. 13-77). Studies on a number of relatively insoluble ultrafine particles (diesel, carbon black), present in the ambient air as aggregated ultrafines, indicate that inhalation exposure to these as well as TiO_2 to rats are associated with epithelial cell proliferation, chronic pulmonary inflammation, pulmonary fibrosis, and induction of lung tumors at high concentrations (CD, p. 13-77). Ultrafine particle have also been shown to evade macrophage phagocytosis and penetrate the interstitium more easily than larger sized particles (Takenaka et al., 1986; Ferin et al., 1990, CD, p. 13-77). There is also evidence that some aggregated insoluble ultrafine particles dissociate into singlet ultrafine particles in the lung which would facilitate transport across the epithelium (Takenaka et al., 1986; Ferin et al., 1990; Oberdörster et al, 1994; CD, p. 13-77). Because of their short lifetime, it is unclear that unaggregated ultrafine particles make up any significant fraction of the mass of fine particles or of PM_{10}, other than in the vicinity of significant sources of ultrafine particles. The relationship between ultrafine numbers (or mass) and the mass of fine or thoracic particles found in typical community air pollution has not been established. Although the CD provides little direct information, it might be expected that penetration and persistence of unaggregated ultrafine particles to indoor environments would be limited. For these reasons, it is questionable whether ultrafine aerosols could be playing a major role in

the reported epidemiologic associations between the measured mass of fine or PM_{10} particles and health effects in sensitive populations. Because of the potential toxicity suggested by the available literature, however, this an area where significant additional research is needed.

The only major coarse particle components highlighted in the CD summary are silica and bioaerosols. The majority of silica particle mass is found in the coarse fraction (CD, p. 11-127). Occupational, but not community exposures to crystalline silica has been associated with pulmonary inflammation and silicosis (pulmonary fibrosis from silica) (Spencer 1977; Morgan et al 1980; Bowden, 1987). Although some evidence of long term accumulation of silicate material at near ambient levels has been noted (Section V-C), the CD provides no evidence of any significant short term effects of ambient silica. Thus, there is no evidence suggesting that this class contributes to the observed daily mortality and morbidity effects.

Bioaerosols (which includes fungal spores, pollen, bacteria, viruses, endotoxins, and animal and plant debris) can be distributed in both fine and coarse fractions and are capable of producing serious health effects. Strong sources (e.g., grain elevators) of these materials may have obvious effects on allergic individuals. However, as the CD points out, the annual variability, relative mass, and distribution of such materials suggests that they too "appear to be unlikely to account for observed ambient (outdoor) PM effects on human mortality and morbidity demonstrated by epidemiology studies reviewed in Chapter 12" (CD, p. 11-136).

c. Physical Aspects of Fine and Coarse Particles

Figure IV-2 and Table IV-2 show key differences between fine and coarse particles. The fine fraction contains by far the largest number of particles and a much larger aggregate surface area than the coarse fraction. As noted above, the size range of particles containing the largest number of particles (<0.02 μm) is not that with most of the mass of the aerosol (fine or coarse). However, most of the aggregate surface area of the entire size distribution of typical urban particles is contained in the fine size range of 0.1 to 1.0 μm diameter (CD, Figure 13-4; Figure IV-2). Unlike the case with particle number, therefore, it is clear that the aggregate surface area of PM_{10} is likely to be strongly related to the mass of fine particles (see Figure IV-). This relationship should be a common property of PM in a variety of different urban settings.

The greater surface area of the fine fraction means this fraction has a substantially greater potential for absorption of other potentially toxic components of PM (e.g. metals, acids, organic materials), as well as for dissolution or absorption of pollutant gases. It is the surface of a particle that is primarily in contact with respiratory cells and surfaces (CD, p. 13-68). The total surface area of a particle may be important in the presentation of active groups on the surface of the particle to cell surfaces (CD, p. 13-26). Biological effects on epithelial cells or macrophages may depend on the number of cell surface receptors stimulated or occupied by particles. Consequently, numbers of particles may be relevant to their toxic effect (CD, 13-27). Therefore, in comparison to coarse mode particles, fine mode particles will have the greatest probability of interactions with potential respiratory targets of toxicity through increased numbers of particles as well as surface area (see Appendix D).

The CD notes that the presence of surface coatings can increase the toxicity of particles. Such considerations may be important when trying to ascertain the appropriate dose metric for evaluation of lower respiratory tract health outcomes (CD, p. 13-24). For example, retardation of alveolar macrophage phagocytosis due to particle overload appears to be better correlated with particle surface area than particle mass (Morrow, 1988; Oberdörster eta al 1995a,b, CD, p. 13-24). Various biological responses (e.g., reduction in lung volumes and diffusion capacity, alteration in biochemical markers, and changes in lung tissue morphology) in guinea pigs have been reported after exposure to ultrafine zinc coated with a surface layer of H_2SO_4 (CD, Chapter 11, Chen et al., 1992b,1995). These responses were much greater than those following exposure to larger size H_2SO_4 in pure droplet form yet having similar mass concentration of acid. A possible mechanism for the differential toxicity of the two aerosols is the difference in particle numbers deposited at target sites. At an equal total sulfate mass concentration, H_2SO_4 existed on many more particles when layered on the ZnO carrier particles than when dissolved into aqueous droplets. In addition, a recent study by Chen et al., (1995) confirmed that the number of particles in the exposure atmosphere, not just total mass concentration of acid, is an important factor in biological responses following acidic sulfate particle inhalation when aerosols having the same size distribution were compared (CD, Chapter 11).

Coating the surface of insoluble particles with certain transition metals (e.g. iron) has been shown to enhance pulmonary toxicity (Costa et al., 1994a,b,; Tepper et al., 1994). Accordingly, fine particles may serve as an efficient carrier of more toxic material to respiratory tract targets. Coating of micrometer-sized particles with formaldehyde has been shown to increase the delivery of formaldehyde and consequently increase irritant responses in human subjects (CD,13-76). Jakab and Hemenway (1993) suggest that reaction products on particle surfaces may be more toxic than the primary material. Exposure to O_3 was shown to increase the toxicity of carbon black particles in mice. The authors hypothesized that this result was due to a "reaction of O_3 on the surface of the carbon black particles in the presence of adsorbed water, producing surface bound, highly toxicologically reactive oxygen species" (CD, p. 11-161).

Increased surface coating of water or the presence of hygroscopic sulfates, nitrates, and organic compounds found as droplets in the fine fraction may also increase the potential for delivery of irritant species such as SO_2, hydrogen peroxide, and aldehydes to more sensitive regions of lung, which, when in the gas phase, would normally be removed in the extrathoracic region (CD, p 13-9). The potential for increasing delivery of pollutant gases provides some basis for expecting some interaction among PM as a pollutant and gases observed in community studies.

d. Deposition in Sensitive Individuals

As shown in Table V-1, both fine and coarse particles penetrate to and deposit in the tracheobronchial and alveolar region. Based on the epidemiological results and deposition considerations, it is reasonable to expect that high levels of coarse particles alone could aggravate asthmatics through tracheobronchial deposition. However acids and fine particles have also been associated with hospital admissions for asthma in areas with relatively low coarse mass (Thurston et al., 1992). Receptors that have been linked to an asthmatic response have been demonstrated to be in areas of the lung where both coarse and fine particles deposit (see Appendix D). Moreover, certain insoluble coarse particles can deposit and remain for extended periods in the alveolar region, although the relation to the chronic effects observed in epidemiologic studies is unclear..

The epidemiological studies suggest greater mortality and morbidity effects in individuals with cardiopulmonary disease. In this regard, it is of note that fine particles have been shown to have a greater deposition in the lungs of individual with chronic respiratory disease than in normal subjects (CD, Chapter 13). Such individuals also have reduced clearance for these particles (see Appendix D). Thus, the potential for greater target tissue dose in susceptible patients is present (CD, Chapter 11). Simulations discussed in Chapter 10 of the CD, suggest that adolescent children (14-18 yrs of age) are predicted to have greater respiratory tract daily mass deposition of submicron particles than adults.

5. Summary and Conclusions

The staff assessment of the evidence finds substantial quantitative and qualitative information on the effects of fine particles and its constituents. Because of the remarkable volume of pertinent literature produced in the last 9 years, far more quantitative epidemiologic data exist today for relating fine particles to mortality, morbidity, and lung function changes in sensitive populations on a short- and long-term basis than was the case for PM_{10} at the conclusion of the last review.[9] Like the PM_{10} studies, the fine particle studies consistently find positive, significant associations between fine particle levels and mortality and morbidity endpoints, with over 20 studies conducted in a number of geographic locations throughout the world, including the US, Canada, and Europe. This collection of studies shows qualitative coherence in the types of health effects associated with fine particle exposure including mortality, morbidity, symptoms, and changes in lung function (Tables V-11 to V-13). The association with mortality is mainly attributable to respiratory and cardiovascular causes, which is consistent with the range of observed respiratory and cardiovascular-related morbidity effects, from respiratory and cardiovascular-related hospital admissions, respiratory symptoms to changes in lung function.

By contrast, the CD and this staff assessment find much less direct evidence in the recent epidemiologic and toxicologic literature regarding the potential effects of coarse particles. The previous staff assessment of occupational and toxicologic literature (EPA

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[9]The 1986 staff assessment of the quantitative basis for the standard cited studies conducted in essentially 3 locations for the 24-hour standard and 4 studies involving a total of 10 cities for the annual standard; none measured PM_{10} (EPA, 1986).

1982a,b) as well as the present review have found ample qualitative reasons to be concerned about elevated levels of coarse particles smaller than 10 μm. These effects (e.g., asthma) are consistent with enhanced deposition of coarse particles in the tracheobronchial region (CD, p. 13-51). However, unlike the case for fine particles, the clearest community evidence regarding coarse particles finds such effects only in areas with numerous marked exceedences of the current PM_{10} standard (CD, p. 13-51). In this regard, it appears that the weight of the available evidence allowing direct comparisons suggests that ambient coarse particles are either less potent or a poorer surrogate for community effects of air pollution than are fine particles.

It is clear, however, that still more quantitative evidence exists today for PM_{10}, which includes both fine and coarse particles. The above assessment does not conclusively demonstrate that coarse particles play no role in the effects associated with PM_{10} at levels below the standard. The potential role of coarse particles in producing such effects could be masked in community studies by potential differences in measurement error and exposure patterns between fine and coarse particles. As noted in the CD, fine particles tend to be more uniformly distributed than coarse mode particles within (and among) urban areas. Moreover, the apparent greater infiltration ratio (penetration and settling) of fine particles indoors means that variations in both short- and long-term personal exposures to outdoor PM will be more influenced by fine than coarse particles.

It is also important to note that some of the more important components of ambient fine particles (e.g. acid sulfates) have no notable indoor sources, while a substantial fraction of indoor coarse particles comes from indoor resuspension of local crustal (e.g. deposited or tracked in on footwear) and other coarse materials (Wallace, 1996). This means that any effects that are potentially produced by coarse particles (from outdoor air and indoor resuspension) are more likely to be decoupled from outdoor concentrations. The less even urban distribution of coarse particles and stronger indoor sources would tend to diminish the power of community studies of outdoor air to detect the effects of such crustally derived materials as compared to fine particles (CD, p. 1-9). Viewed from another perspective, this also suggests that efforts to reduce any such effects by controlling outdoor coarse particles would be less successful than a program to reduce outdoor fine particle effects. Thus, while

the epidemiologic data are not conclusive with regard to the potential effects of coarse particles, they more strongly support the notion that fine particles are a better surrogate for that fraction of ambient PM that is most clearly associated with the health effects observed in community air pollution studies at levels below the current standards. This view is also supported by qualitative considerations derived from a consideration of the toxicologic implications of the profound physical and chemical differences associated with components of these fractions.

VI. RISK ASSESSMENT

The objective of this PM health risk assessment is to provide quantitative estimates of the risks to public health associated with 1) existing air quality levels, 2) projected air quality levels that would occur upon attainment of the current PM_{10} standards, and 3) projected air quality levels that would occur upon attainment of alternative $PM_{2.5}$ standards. As an integral part of this assessment, qualitative and, where possible, quantitative characterizations of the uncertainties in the resulting risk estimates have been developed, as well as information on baseline incidence rates for the health effects considered. This assessment provides information most relevant to evaluating alternative levels of PM standards, rather than to selecting the most appropriate indicator of PM. This risk information is intended as a tool that may, together with other information presented in this Staff Paper, assist the Administrator in selecting primary PM standards that, in her judgment, would reduce risks to public health sufficiently to protect public health with an adequate margin of safety, recognizing that such standards will not be risk-free.

As discussed in section V.E above, the CD concludes that the overall consistency and coherence of the epidemiologic evidence suggests a likely causal role of ambient PM in contributing to adverse health effects (CD, p. 13-1). Also discussed in section V.E. is an alternative interpretation, suggested by some researchers, that PM may be serving as an index for the complex mixture of pollutants in urban air. The risk assessment described here is premised on the assumption that PM (measured as PM_{10} and $PM_{2.5}$) is causally related to the health effects observed in the epidemiological studies and/or that PM is a useful index for the mixture of pollutants that is related to these effects.

In presenting this risk assessment, the staff cautions that despite the consistency and coherence of the epidemiological evidence with respect to the existence of effects, quantitative relative risk results derived from these studies include significant uncertainty. Due to the uncertainties in the concentration-response study results, as well as the many sources of uncertainty inherent in the analyses presented in this chapter, the risk estimates developed in this assessment should not be interpreted as precise measures of risk. The major uncertainties

and assumptions associated with these analyses are highlighted in the following discussion and presentation of results. In addition, some key uncertainties are addressed quantitatively through individual sensitivity analyses as well as integrated uncertainty analyses which assess the combined effects of several key uncertainties.

The following sections summarize the scope of the analyses, key components of the risk model, and results of baseline risk and sensitivity analyses. A detailed discussion of the risk assessment methodology and results is presented in technical support documents (Abt Associates, 1996a,b).

A. General Scope

The PM risk analyses focus on selected health effects endpoints such as increased daily mortality, increased hospital admissions for respiratory and cardiopulmonary causes, and increased respiratory symptoms for children. Although the risk analyses could not address all of the various health effects for which there is some evidence of association with exposure to PM, all such effects are identified and considered above in section V.C. All concentration-response functions used in these analyses are based on findings from human epidemiological studies, which rely on fixed-site, population-oriented, ambient monitors as a surrogate for actual integrated PM exposures. Measurements of daily variations of ambient PM concentrations, as used in the time series epidemiological studies that provide the concentration-response relationships for these analyses, have a plausible linkage to the daily variations of exposure from ambient sources for the populations represented by ambient monitoring stations, as discussed in Chapter IV. The CD concludes that this linkage should be better for indicators of fine particles (e.g., $PM_{2.5}$) than for indicators of fine plus coarse particles (e.g., PM_{10}, TSP), and in turn, should be better than indicators of inhalable coarse fraction particles (PM_{10} - $PM_{2.5}$) (CD, p. 1-10). A more detailed discussion of the possible impact of exposure misclassification on the estimated concentration-response relationships derived from the community epidemiological studies is presented above in section V.E.

These PM risk analyses feature:

- analyses of risks under a recent 12-month period of air quality (labeled "as is" air quality) and under a situation where air quality just attains various alternative standards being considered;
- estimates of risks for the urban centers of two example cities, one eastern (Philadelphia County) and one western (Southeast Los Angeles County), rather than national estimates;
- estimates of risks only for concentrations exceeding an estimated background level; and
- qualitative and quantitative consideration of uncertainty, including sensitivity analyses of key individual uncertainties and integrated uncertainty analyses combining key uncertainties.

More specifically, consistent with the recommendations to the Agency provided in the January 5, 1996 CASAC letter to the Administrator (Wolff, 1996b), alternative 24-hr and annual $PM_{2.5}$ standards are examined alone and in combination with the current PM_{10} standards. This focus also reflects the conclusions drawn in the CD (CD, Chapter 13) that it is appropriate to consider fine and coarse fraction particles separately, and that for mortality and some measures of morbidity, the most consistent associations are seen with fine and thoracic particles (e.g., $PM_{2.5}$, PM_{10}) as compared to coarse fraction particles (CD, Chapter 13; section V.F above). The scope of these analyses initially focuses on developing risk estimates for portions of two selected urban areas: Philadelphia County and a portion (roughly the southeastern third) of Los Angeles County (hereafter referred to as "Los Angeles County"). These areas were chosen based on availability of PM_{10} and $PM_{2.5}$ air quality data, and the desire to include areas from the eastern and western parts of the United States to reflect regional differences in the makeup of PM. Finally, estimates of risks above background PM concentrations are judged to be more relevant to policy decisions about the level of ambient air quality standards than estimates that include risks potentially attributable to uncontrollable background PM concentrations.

B. Components of the Risk Model

In order to estimate the change in health effects incidence corresponding to the difference in PM levels between "as is" conditions and just attaining alternative standard scenarios, the following three key components are required for a given health endpoint and selected city: 1) air quality information, 2) concentration-response relationships, and 3) baseline health incidence rates. Figure VI-1 is a broad schematic depicting the role of these components in the risk analyses. The general health risk model which combines changes in PM air quality concentrations (Δx), the concentration-response relationships for a given health endpoint (reflected by β, the PM coefficient derived from epidemiology studies), and the baseline health effects incidence rate (y) for a given health endpoint is represented by equation 1:

Equation 1 $$\Delta y = y[e^{\beta \Delta x} - 1]$$

Estimates of risk (i.e., health effects incidences attributable to PM) are quantified for PM concentrations above background except for those studies in which the range of observed PM concentrations did not go down to estimated background (e.g., the prospective cohort mortality studies). For these studies effects are quantified down to the lowest concentrations observed in the study. As indicated in Figure VI-1, sensitivity analyses on various key inputs to the PM health risk model are conducted as part of this assessment, as well as an integrated uncertainty analysis that examines the potential impact of combining several key uncertainties. Each of these key components is briefly discussed below.

1. Air Quality Information

The air quality information required to conduct the PM risk analyses includes: 1) "as is" air quality data for both PM_{10} and $PM_{2.5}$ from population-oriented monitors for the selected cities, 2) estimates of background PM concentrations appropriate to that location, and 3) a method for adjusting the "as is" data to reflect patterns of air quality change estimated to occur when each city attains various alternative standards. Table VI-1 provides a summary of the

Figure VI-1 Major Components of Particulate Matter Health Risk Analysis

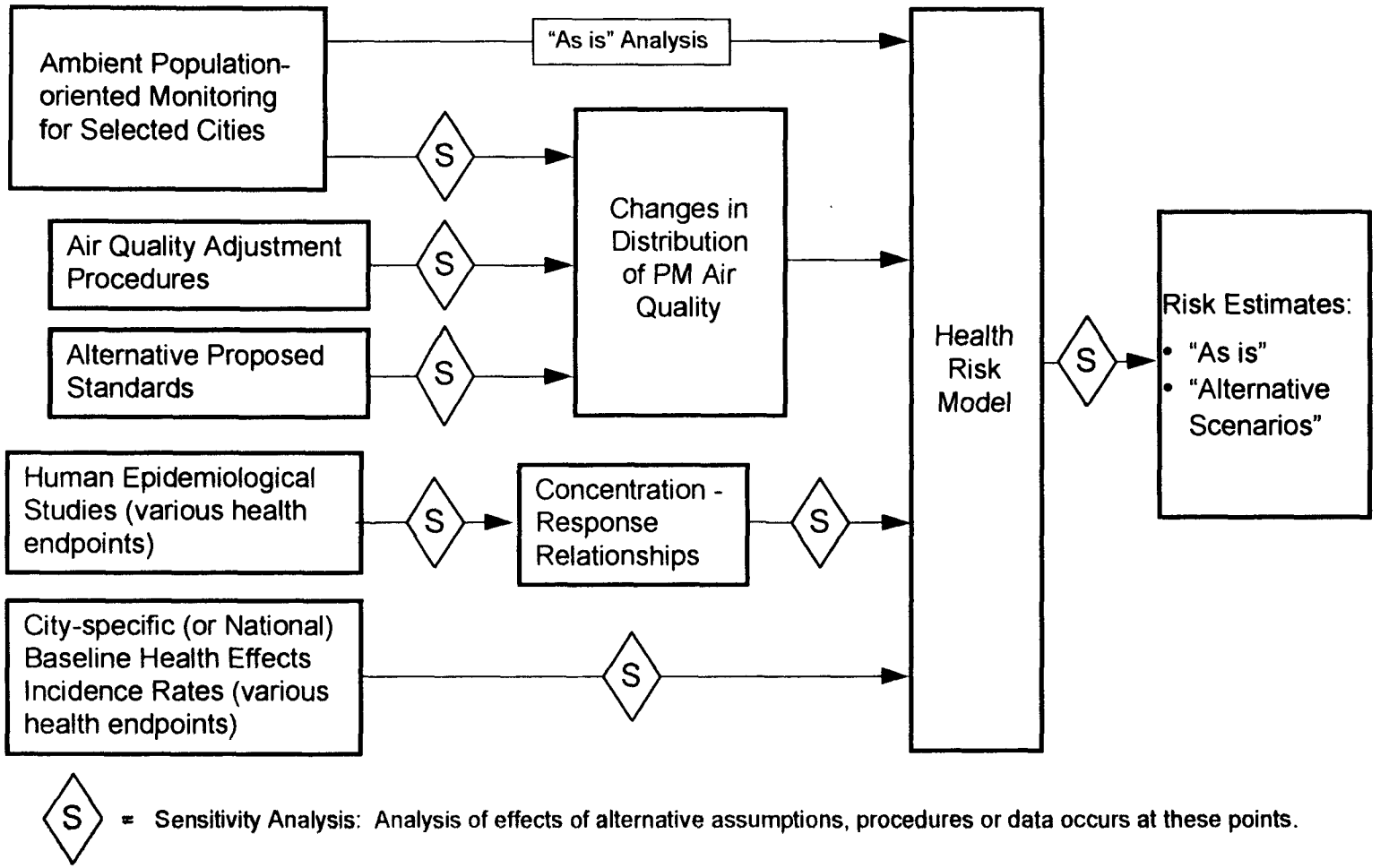

TABLE VI-1. CITIES EXAMINED IN PM RISK ANALYSIS

City	Population[a] (millions)	Year	% of Days on Which Air Quality Data are Available		PM_{10}[b]		$PM_{2.5}$[b]	
			PM_{10}	$PM_{2.5}$	Annual Average ($\mu g/m^3$)	Second Max, 24-hr Avg. ($\mu g/m^3$)	Annual Average ($\mu g/m^3$)	Second Max, 24-hr Avg. ($\mu g/m^3$)
Philadelphia County, PA	1.6	1992-93	99	96	25	77	17	72
Los Angeles County, CA	3.6	1995	59	59	52	195	30	129

[a]Based on 1990 U.S. Census data.

[b]Concentrations are reported for the monitor with the highest value.

Note: More detailed information about the air quality data in these cities is presented in Section 4 of Abt Associates (1996b).

PM_{10} and $PM_{2.5}$ air quality data for the two areas included in these analyses. The PM_{10} and $PM_{2.5}$ monitoring information for Philadelphia County are from three monitors used in the Acid Aerosol Characterization Study during 1992-1993 (network sites described in Suh et al., 1995). The monitoring information for southeast Los Angeles County comes from two dichotomous samplers operated during 1995 by the South Coast Air Quality Management District. Figure VI-2 presents frequency distributions of the daily PM_{10} and $PM_{2.5}$ concentrations in Philadelphia County based on spatially averaging the reported concentrations available from the different monitors for each day. Figures VI-3 and VI-4 show the frequency distributions of the daily PM_{10} and $PM_{2.5}$ concentrations by quarter in southeast Los Angeles County based on spatially averaging the reported concentrations available from the different monitors for each day.

As discussed above, these ambient concentrations are used as a surrogate for population exposures in these analyses, a procedure consistent with the health literature but which adds uncertainty to the risk estimates. In an effort to limit uncertainties that would result in combining data across different monitoring methods, only information from these monitors was used directly in the risk analysis.[1]

Background PM concentrations used in these analyses are defined in Chapter IV as the distribution of PM concentrations that would be observed in the U.S. in the absence of anthropogenic emissions of PM and its precursors in North America. For these analyses, an estimate of the annual average background level is desired, rather than a daily average (e.g., the maximum 24-hour level), since estimated risks are aggregated for each day throughout the year. The staff have chosen to use the midpoint of the appropriate ranges of annual average estimates for PM background presented in Table IV-3 for the base case risk estimates (i.e.,

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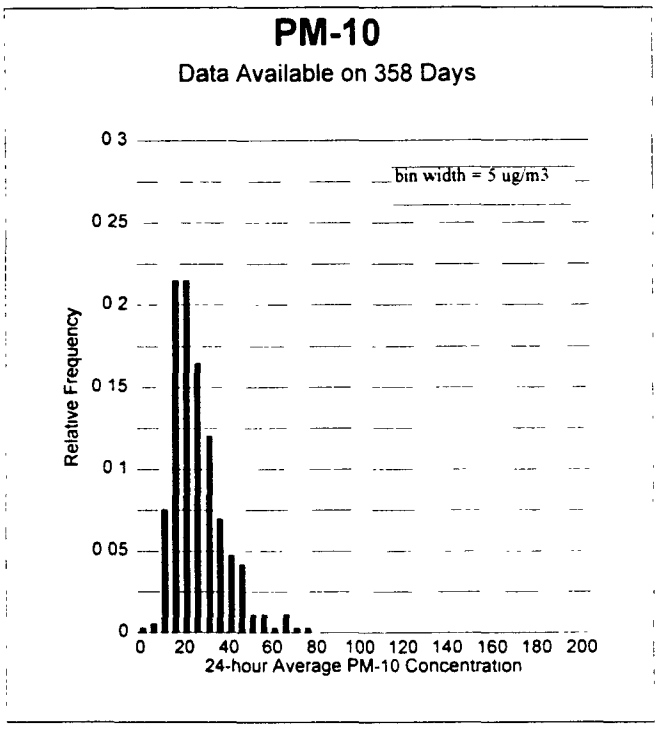
[1]Although not directly used in the risk analyses, information from the AIRS database for sites in Los Angeles county was used to help define the region of Los Angeles County included in this analysis (see Abt Associates, 1996b).

Figure VI-2. Daily Average PM Concentration Frequencies
Philadelphia County, September 1992 - August 1993



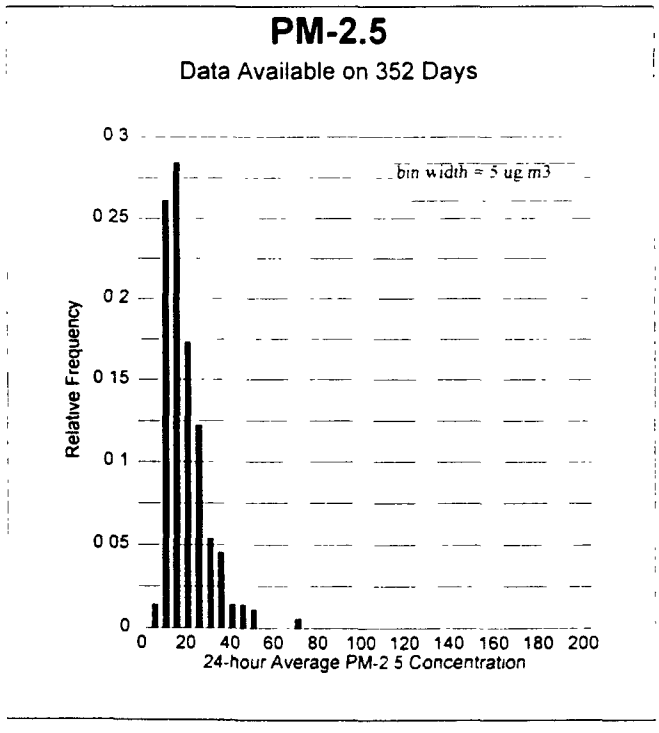
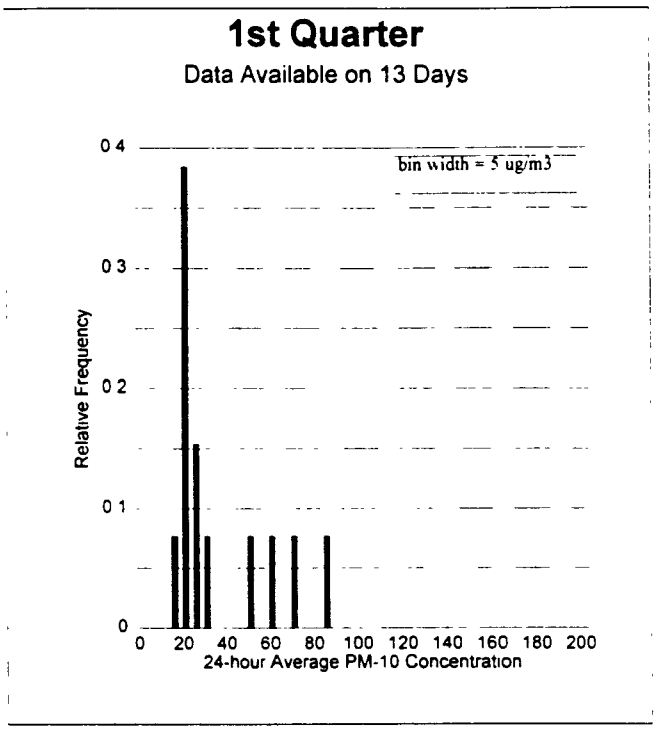
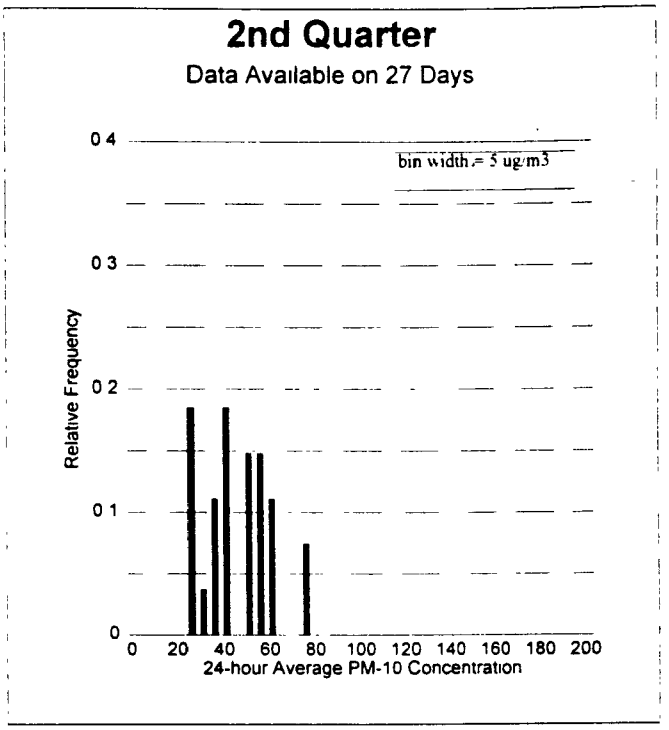
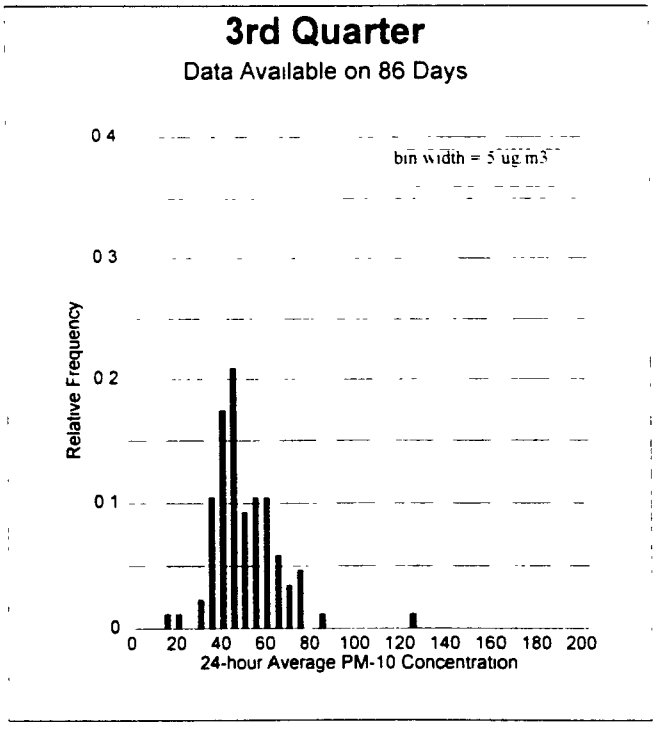


Figure VI-3. Daily Average PM-10 Concentrations for Southeast Los Angeles County, 1995







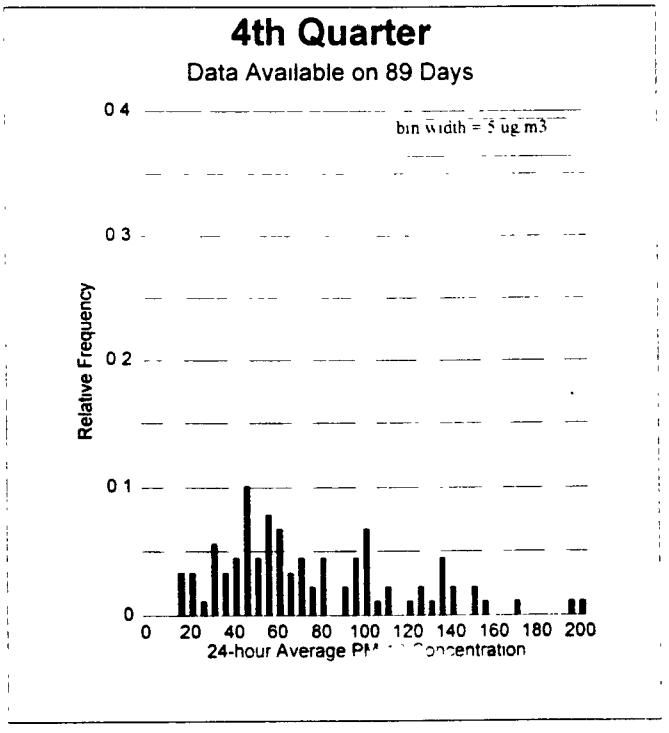
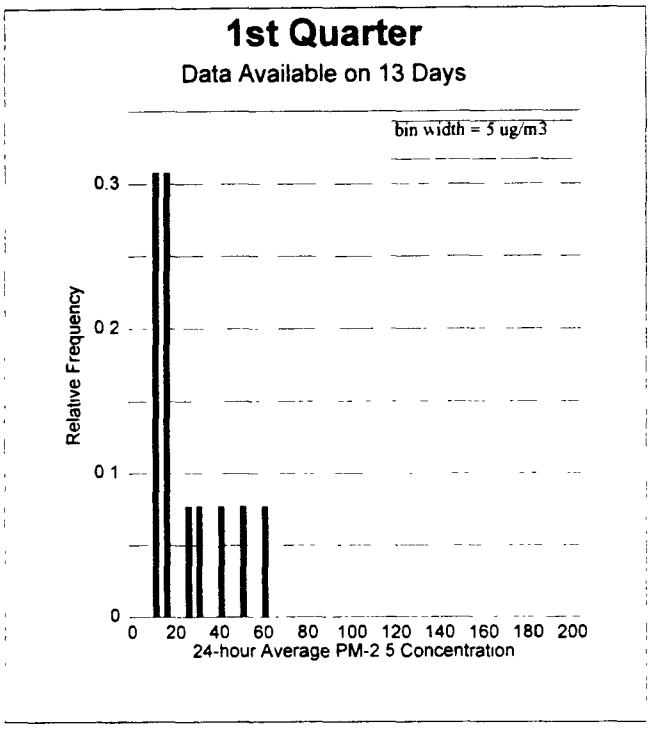
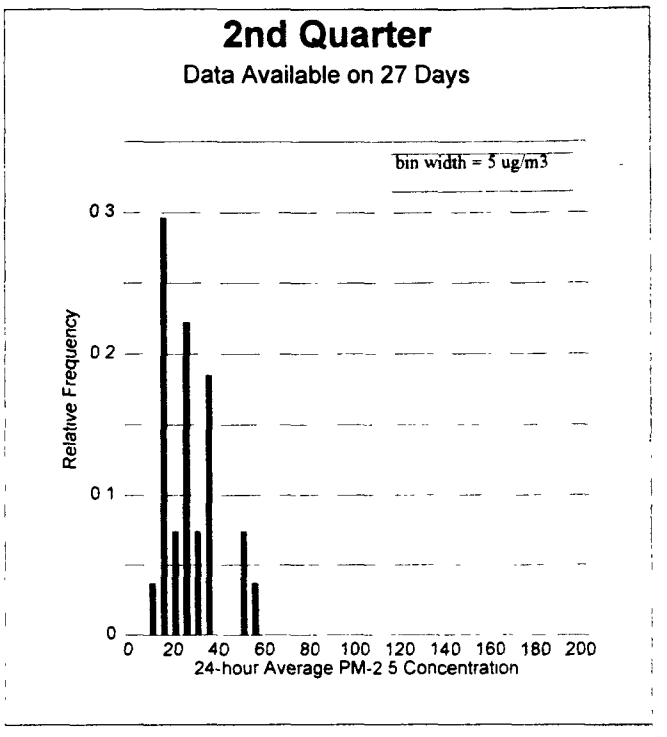
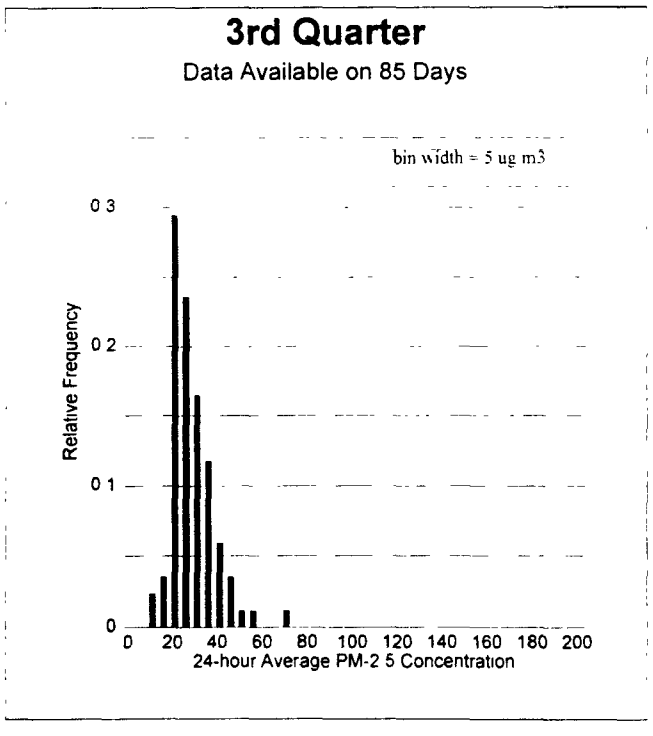
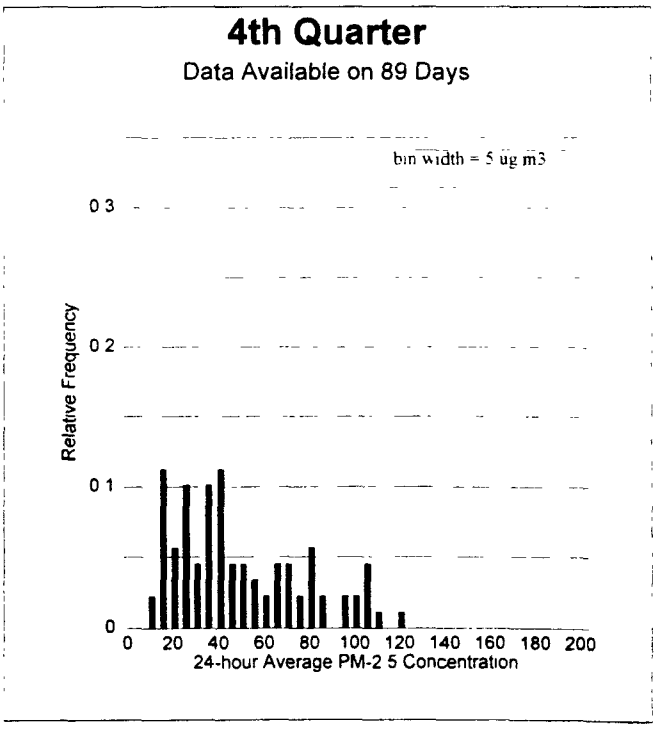


Figure VI-4. Daily Average PM-2.5 Concentration Frequencies For Southeast Los Angeles County, 1995









eastern values are used for Philadelphia and western values for Los Angeles):

- For PM_{10}: 5 - 11 $\mu g/m^3$ for Philadelphia, and 4 - 8 $\mu g/m^3$ for Los Angeles
- For $PM_{2.5}$: 2 - 5 $\mu g/m^3$ for Philadelphia, and 1 - 4 $\mu g/m^3$ for Los Angeles.

Sensitivity analyses have been done using the appropriate lower and upper ends of the above ranges to characterize the impact of this model input choice on the risk estimates.

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

2. Concentration-Response Functions

The second key component in the risk model is the set of concentration-response relationships which provide estimates of the relationship between each health endpoint of interest and ambient PM concentrations. Table VI-2 summarizes the selected epidemiological studies which are judged adequate by the CD to provide estimated concentration-response relationships for a variety of health endpoints associated with elevated PM_{10} and/or $PM_{2.5}$ exposures (CD, Tables 13-3, 13-5). Only studies based on either PM_{10} and/or $PM_{2.5}$ as a measure of PM have been used in these analyses. Each study provides an estimate of relative

Table VI-2. Selected Epidemiological Studies and Associated Relative Risk Estimates Used in Risk Analyses

Health Effect	PM Indicator	Study Location	Reported PM Levels ($\mu g/m^3$) Mean (Range)[1]	Estimated Relative Risk[2] (95% Confidence Interval)	Pooled Relative Risk[3]
TOTAL MORTALITY					
Short-term Exposures	PM_{10}	Six Cities[a]			
		Portage, WI	18 (+11.7)	1.04 (0.98, 1.09)	
		Boston, MA	24 (+12.8)	1.06 (1.04, 1.09)	
		Topeka, KS	27 (+16.1)	0.98 (0.90, 1.05)	
		St. Louis, MO	31 (+16.2)	1.03 (1.00, 1.05)	
		Kingston/Knoxville, TN	32 (+14.5)	1.05 (1.00, 1.09)	1.04 (0.99, 1.09)
		Steubenville, OH	46 (+32.3)	1.05 (1.00, 1.08)	
		Chicago, IL[b]	38 (NR/128)	1.03 (1.02, 1.04)	
		Utah Valley, UT[c]	47 (11/297)	1.08 (1.05, 1.11)	
		Birmingham, AL[d]	48 (21,80)	1.05 (1.01, 1.10)	
		Los Angeles, CA[e]	58 (15/177)	1.03 (1.00, 1.06)	
	$PM_{2.5}$	Six Cities[a]			
		Portage, WI	11.2 (+7.8)	1.03 (0.99, 1.07)	
		Topeka, KS	12.2 (+7.4)	1.02 (0.95, 1.09)	
		Boston, MA	15.7 (+9.2)	1.06 (1.04, 1.07)	1.04 (1.00, 1.07)
		St. Louis, MO	18.7 (+10.5)	1.03 (1.01, 1.04)	
		Kingston/Knoxville, TN	20.8 (+9.6)	1.04 (1.01, 1.07)	
		Steubenville, OH	29.6 (+21.9)	1.03 (1.00, 1.05)	
Long-term Exposures	$PM_{2.5}$	ACS Study[f] (50 U.S. SMSA)	9-34[4]	1.17 (1.09, 1.26)	---
HOSPITAL ADMISSIONS -- Short-term Exposures					
All Respiratory Causes (for Elderly > 64 years)	PM_{10}	Tacoma, WA[g]	37 (14, 67)	1.10 (1.03, 1.17)	
		New Haven, CT[g]	41 (19, 67)	1.06 (1.00, 1.13)	1.09 (1.02, 1.19)
		Cleveland, OH[h]	43 (19, 72)	1.06 (1.00, 1.11)	
		Spokane, WA[i]	46 (16, 83)	1.08 (1.04, 1.14)	

Health Effect	PM Indicator	Study Location	Reported PM Levels ($\mu g/m^3$) Mean (Range)[1]	Estimated Relative Risk[2] (95% Confidence Interval)	Pooled Relative Risk[3]
	$PM_{2.5}$	Toronto[l]	18.6 (NR/66.0)	1.15 (1.02, 1.28)	
HOSPITAL ADMISSIONS -- Short-term Exposures					
COPD (for Elderly > 64 years)	PM_{10}	Minneapolis, MN[k] Birmingham, AL[l] Spokane, WA[l] Detroit, MI[m]	36 (18,58) 45 (19,77) 46 (16,83) 48 (22,82)	1.25 (1.10, 1.44) 1.13 (1.04, 1.22) 1.17 (1.08, 1.27) 1.10 (1.02, 1.17)[5]	1.14 (1.05, 1.31)
Ischemic Heart Disease (for Elderly > 64 years)	PM_{10}	Detroit, MI[n]	48 (22,82)	1.02 (1.01, 1.03)	---
Congestive Heart Failure (for Elderly > 64 years)	PM_{10}	Detroit, MI[n]	48 (22,82)	1.03 (1.01, 1.05)	---
Pneumonia (for Elderly > 64 years)	PM_{10}	Minneapolis, MN[k] Birmingham, AL[l] Spokane, WA[l] Detroit, MI[m]	36 (18,58) 45 (19,77) 46 (16,83) 48 (22,82)	1.08 (1.01, 1.15)[5] 1.09 (1.03, 1.15) 1.06 (0.98, 1.13) 1.06 (1.02, 1.10)[5]	1.07 (1.01, 1.14)
RESPIRATORY SYMPTOMS					
Lower Respiratory Symptoms in Children: Short-term Exposures	PM_{10}	Six Cities[o] Utah Valley, UT[p]	30 (13,53) 46 (11/195)	2.03 (1.36, 3.04)[6] 1.28 (1.06, 1.56)	---
	$PM_{2.5}$	Six Cities[o]	18.0 (7.2-37)	1.44 (1.15-1.82)[6]	---
Bronchitis in Children: Long-term Exposures	$PM_{15/10}$	Six Cities[g]	20-59[4]	3.26 (1.13, 10.28)[6]	---

References:

[a]Schwartz et al. (1996a)
[b]Ito and Thurston (1996)
[c]Pope et al. (1992)
[d]Schwartz (1993a)
[e]Kinney et al. (1995)
[f]Pope et al. (1995)
[g]Schwartz (1995)
[h]Schwartz et al. (1996b)
[i]Schwartz (1996)
[l]Thurston et al. (1994b)
[k]Schwartz (1994f)
[l]Schwartz (1994e)
[m]Schwartz (1994d)
[n]Schwartz and Morris (1995)
[o]Schwartz et al. (1994)
[p]Pope et al. (1991)
[q]Dockery et al. (1989)

Endnotes:

1. Range of 24-hour PM indicator level shown in parentheses is typically either the standard deviation (+ S.D.) or 10th and 90th percentiles.
2. Based on a 50 $\mu g/m^3$ increase for PM_{10} studies, and a 25 $\mu g/m^3$ increase in $PM_{2.5}$ studies.
3. See Abt Associates (1996b) for calculation method.
4. Range of city means of PM levels.
5. Only RR reported includes other pollutants in model.
6. Odds ratio.

risk (β), along with a measure of the uncertainty (95% confidence interval) of the estimate, associated with specific changes in PM levels (i.e., a 50 $\mu g/m^3$ increase in PM_{10} or a 25 $\mu g/m^3$ increase in $PM_{2.5}$).

As indicated in the CD, the most credible approach to risk analysis would be to use site-specific relative risk (RR) estimates for PM (CD, p.13-87). For Los Angeles County, site-specific RRs are available from two studies (Kinney et al, 1995; Ostro et al., 1995). Philadelphia County has been the location of several studies reporting associations between PM and mortality and hospital admissions, but none of the published reports have used PM_{10} or $PM_{2.5}$. Since site-specific relative risks are not available for all endpoints in both locations (and in the absence of more information concerning which individual studies might most appropriately characterize the health risk in a risk analysis location), an approach was employed which combined available information from all the key studies for a health endpoint. A form of meta analysis (referred to as a "pooled analysis" in this Staff Paper) was conducted which combined the results of the various studies. For comparison purposes, Table VI-2 lists the mean estimate of RR from the pooled analysis along with the RRs for the individual studies comprising the pooled analysis.

Given differences in population, particle size distribution, and other environmental stressors (e.g., weather variables, co-pollutants), RRs may be expected to vary from location to location. The CD notes such variation appears to be observed in coefficients for mortality associated with short-term exposures, and cautions against the application of a single "best estimate" relative risk value across various locations (CD, p.13-87). The pooled analyses in this risk analysis have utilized an "empirical Bayes" approach in an effort to more fully reflect the range of relative risk estimates, and accompanying statistical uncertainty, seen from location to location. Standard meta analysis techniques, such as a random effects meta analysis, estimate a mean relative risk and the statistical uncertainty around that mean estimate. The empirical Bayes approach estimates the underlying distribution of RRs observed across areas and the likelihood that any relative risk estimate from that distribution will be applicable to an uninvestigated location. The empirical Bayes approach uses the random effects model framework, in which the relative risks from different locations can be genuinely different,

while adjusting the relative risk and statistical uncertainty observed in individual locations to some degree to reflect the information available from the entire set of studies (see Abt Associated, 1996b, for further details). However, the distribution of RRs from the empirical Bayes approach provides uncertainty estimates ("credible intervals") which are intended to represent the range of reported RRs (and not simply the uncertainty around a mean estimate) and is not restricted to assuming a normal distribution (see Abt Associates, 1996b, Exhibit 5.12). As a result, credible intervals from the empirical Bayes approach are typically wider than confidence intervals from random effects meta analysis[2] and are expected to more fully convey information on both statistical uncertainty and potential inherent differences (due to different population characteristics, PM size distributions, etc.) in the RRs for different geographic locations.[2]

In the risk analyses, the 5th and 95th percentile values from the distributions of RRs estimated by the empirical Bayes approach are provided as a 90% "credible interval" to characterize uncertainty in the risk estimates for each endpoint. (In Table VI-2, the 95% credible interval around the pooled relative risk estimate is provided instead, to facilitate comparison with the reported RRs from the original studies). In the risk analyses the mean of the distribution based on the empirical Bayes approach is also reported as an estimate of the central tendency of the distribution. Because a random effects framework was used for the empirical Bayes approach, this mean estimate is identical to what would be estimated by a random effects meta analysis. A more detailed description of the techniques used to develop the pooled estimates and the application of the empirical Bayes approach is provided in the technical support document (Abt Associates, 1996b).

In the absence of site-specific RRs for all the endpoints of interest (a product of data limitations that preclude constraining the assessment solely to those areas where both adequate air quality and concentration-response information are available), pooled analyses using this

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[2] Exhibit 5.10 of Abt Associates (1996b) shows that the credible intervals resulting from the empirical Bayes approach are wider for cases in which a number (6-10) of location-specific concentration-response relationships are available (e.g., mortality associated with short-term exposures of PM_{10} or $PM_{2.5}$), but not substantially different for hospital admissions endpoints for which fewer studies (3-4) were pooled.

empirical Bayes approach is one method employed to allow potential differences in RR from location to location to be reflected in the risk estimates. As an additional approach, sensitivity analyses have been performed evaluating the effects of including alternative studies or excluding studies or groups of studies from the pooled analyses (Appendix F, Table F-4; Abt Associates, 1996b).

The CD identifies the interpretation of specific concentration-response relationships as the most problematic issue for risk assessment purposes at this time due to the absence of clear evidence regarding mechanisms of action for the various health effects of interest (CD, p.13-87). The reported study results used in these analyses are based on linear models extending over the range of air quality within the study, as illustrated in Figure VI-5 (CD, Figure 13-5) by Line A. This model implies a possible linear, no-threshold underlying relationship potentially extending to zero PM concentrations (illustrated by Line B). Alternatively, the existing data do not rule out the possible existence of an underlying non-linear, threshold relationship (illustrated by Line C). Although these alternative interpretations of study results could significantly affect estimated risks, only very limited information is available to aid in resolving this issue (CD, pp. 13-87-91). Thus, the approach taken in this risk assessment is to address alternative models through sensitivity and integrated uncertainty analyses to develop ranges of estimated risks, rather than characterizing any of the sets of risk estimates as representing best estimates.

To frame the sensitivity analyses of concentration-response models, the results from various studies have been examined through a number of alternative approaches to identify appropriate PM concentration "cutpoints"[3] which define the lower end of the range over which the concentration-response functions would be applied. Table VI-3 summarizes the cutpoints examined in the sensitivity and integrated uncertainty analyses. A more detailed discussion of the basis for selecting these particular cutpoints is presented in Appendix E.

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[3] "Cutpoint" as used in Chapter VI refers to concentrations determined to be of interest for evaluating the sensitivity of risk estimates to assumptions about the shape of concentration-response relationships. This is in contrast to the use of the term "cutpoint" in Chapter IV, which refers to the aerodynamic diameter of particles being sampled by a monitor.

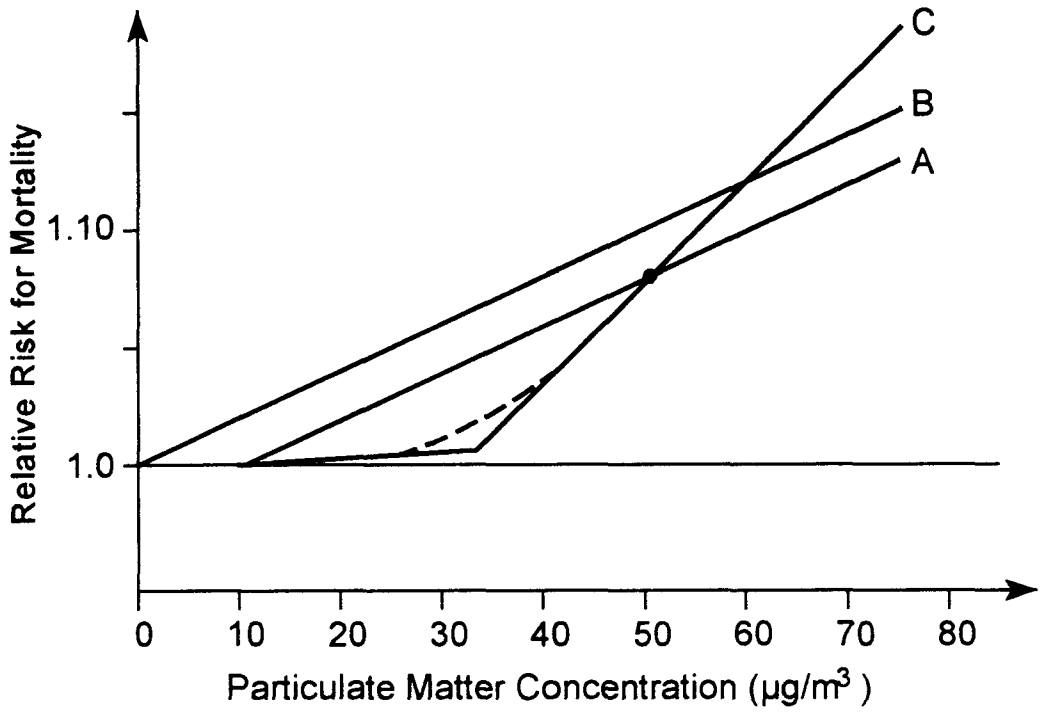


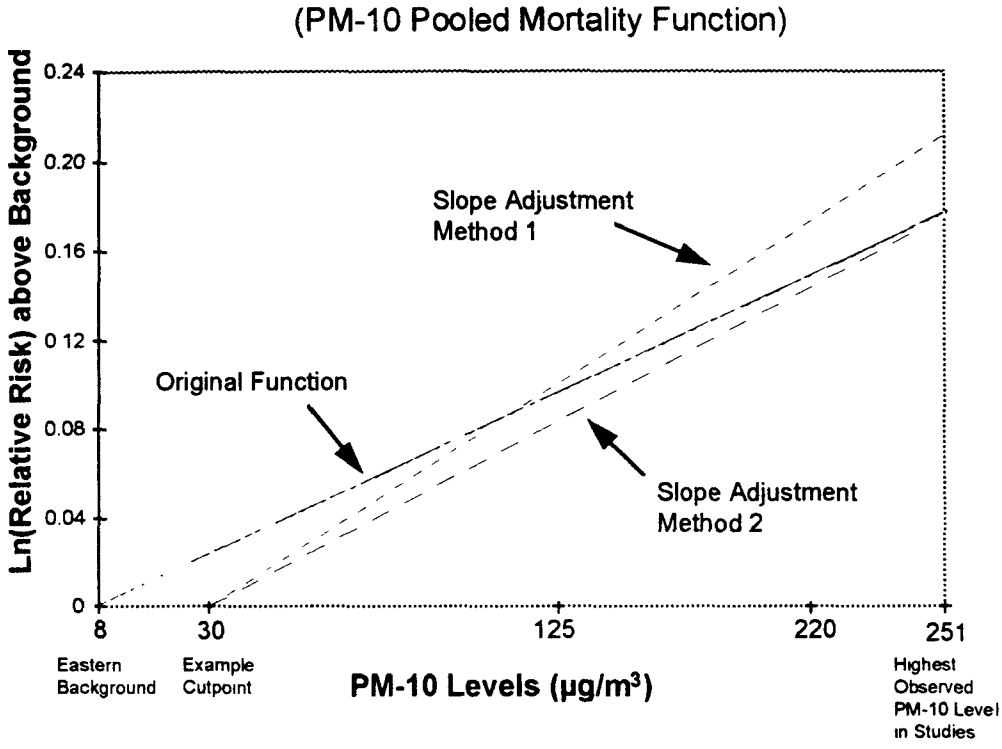
Figure VI-5. Schematic Representation of Alternative Interpretations of Reported Epidemiologic Relative Risk (RR) Findings with Regard to Possible Underlying PM Mortality Concentration-Response Functions (CD, Figure 13-5). Published studies typically only report results from linear models that estimate RR over a range of observed PM concentrations as represented by Line A (specific PM values shown are for illustrative purposes only), compared against baseline risk (RR = 1.0) at the lowest observed PM level. One alternative interpretation is that the RR actually represents an underlying linear, no-threshold PM-mortality relationship (Line B) with the same slope as Line A but extending below the lowest observed PM level essentially to 0 $\mu g/m^3$. Another possibility is that the underlying functional relationship may have a threshold (illustrated by Curve C), with an initially relatively flat segment, not statistically distinguishable from the baseline risk (1.0) until some PM concentration where it sharply increases (or more likely somewhat less sharply ascends in the vicinity of the breakpoint as shown by the dashed lines).

Table VI-3. Concentration-Response "Cutpoints" Examined in Uncertainty Analyses

Pollutant	Health Effects	Cutpoints Examined ($\mu g/m^3$)		
PM_{10}	Effects Associated with Short-Term Exposure	20	30	40
$PM_{2.5}$	Effects Associated with Short-Term Exposure	10	18	30
$PM_{2.5}$	Effects Associated with Long-Term Exposure	12.5	15	18

In conjunction with defining such concentration cutpoints, the slopes of the concentration-response functions have been increased to reflect the effect of potential thresholds at the selected levels. This concept that the slope above a cutpoint would be expected to increase somewhat in a threshold model is illustrated by the comparison of linear and nonlinear models applied, for example, to the TSP data set from Philadelphia presented in the CD (CD, Table 13-6; Appendix F, Figure F-1). Figure VI-6 illustrates the two methods used to adjust slopes when nonlinear models with cutpoints were applied in the risk analyses. The first method adjusts the slope of the relationship from the cutpoint to the maximum concentration observed in the health effects studies so that the area under this line is the same as the area under the original concentration-response relationship that went down to estimated background. To compensate for fewer PM-associated health effects at low concentrations (and no effects below the cutpoint level), the adjusted function must rise more rapidly than the original function. The second slope adjustment method assumes that the RR associated with the maximum concentration observed in the studies is the same as in the original function and, therefore, the concentration-response relationship extends from the cutpoint to the RR observed at the maximum concentration in the original study. This second method increases the slope less than the first method. It is important to recognize that the two adjustment

Figure VI-6. Slope Adjustment Methods Used in Sensitivity and Uncertainty Analyses



Relative Risks shown are the risks associated with elevated PM-10 levels relative to the risks associated with the background PM level (8 µg/m^3) for Philadelphia County.

methods are illustrative and intended to roughly bound the potential impact on concentration-response relationships if cutpoints or thresholds above background exist.

Based on this examination of study results, presented in Appendix E, the cutpoints identified in Table VI-3 have been selected as a basis for a series of sensitivity and uncertainty analyses. Results of sensitivity and uncertainty analyses involving cutpoint and other important uncertainties are presented in section VI.C below.

An additional issue concerning the appropriate interpretation of ambient PM concentration-response relationships is whether they may represent effects resulting from the combined exposure to ambient and indoor particles (or some subset of ambient and indoor exposures, such as the combined exposure to ambient and indoor combustion source particles). While total personal exposure to ambient and indoor particles can be substantially higher than exposure to ambient particles alone[4], the CD concludes that additional exposure to particles indoors from sources independent of ambient sources (which individuals can be exposed to when either outdoors or indoors, since particles penetrate residential indoor microenvironments (CD, p. 1-9)) would not be expected to systematically affect coefficients of ambient concentration-response relationships (CD, p. 1-10).

3. Baseline Health Effects Incidence Rates

The third key component required in the PM risk analyses is an estimate of the baseline health effects incidence rate corresponding to "as is" PM levels. Incidence rates express the occurrence of a disease or event (e.g., asthma episode, hospital admission, death) in a specified time period, usually per year. Health effects incidence rates vary among geographic

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[4]For example, the PTEAM study found that for a study population in Riverside, CA, during a period in which daytime ambient PM_{10} concentrations measured at a central monitor averaged 91 $\mu g/m^3$ and ranged from 37 - 158 $\mu g/m^3$ (10th -90th percentile of daytime concentration distribution), daytime total personal exposure averaged approximately 60% higher (150 $\mu g/m^3$, ranging from 60 - 263 $\mu g/m^3$ (10th -90th percentile) (Clayton et al, 1993). However, nighttime ambient and personal exposures were highly similar [mean concentrations were identical (77 $\mu g/m^3$) with ambient PM_{10} values ranging slightly above and below personal exposure values across the group (10th-90th percentile range 30 -156 $\mu g/m^3$ ambient; 37 - 135 $\mu g/m^3$ personal)].

areas due to differences in population characteristics (e.g., age distribution) and factors affecting illness or response (e.g., smoking, occupation, income levels, air pollution levels).

Tables VI-4 and VI-5 provide a summary of population estimates and baseline mortality and morbidity incidence rates used in these analyses for Philadelphia and Los Angeles Counties. Mortality rates are based on county-specific data from the National Center for Health Statistics. Morbidity rates for hospital admissions in Philadelphia are based on Philadelphia County admissions data obtained from the Delaware Valley Hospital Council, and for Los Angeles County from California's Office of Statewide Health Planning and Development. For respiratory symptoms, baseline incidence information on symptoms is not routinely reported, so for these endpoints the incidence rates from the studies themselves were used. This would be expected to introduce considerable uncertainty, since baseline symptoms incidence would be expected to vary across locations, and because many diary studies (e.g., Schwartz et al., 1994; Pope et al., 1991) do not record symptoms incidence across an entire year. Thus, incidence estimates for respiratory symptoms are particularly uncertain and are primarily included to provide perspective on the number of effects estimated relative to other health effects.

Uncertainty in baseline incidence rates primarily affects estimates of numerical incidence (e.g., counts of number of hospital admissions, symptoms). Percent of incidence estimates can be obtained without the use of baseline incidence health information, since almost all of the key studies used in the risk analysis report results in the form of RR versus air quality (the exception being Thurston et al., 1994) which generate the same percent of incidence estimates regardless of the baseline incidence rates. Baseline incidence rates are only involved in estimating the implication of the estimates of percentage incidence in terms of numbers of health effects.

Table VI-4. Relevant Population Sizes for Philadelphia County and Southeast Los Angeles County

Population	Philadelphia County	Southeast Los Angeles County
Total	1,590,000	3,640,000
Ages ≥ 65	241,000 (15.2%)	322,000 (8.9%)
Children, ages 8-12	103,000 (6.5%)	282,000 (7.8%)
Children, ages 10-12	62,000 (3.9%)	166,000 (4.6%)
Asthmatic Children, ages 9-11	3,900* (0.3%)	10,700* (0.3%)
Asthmatic African-American Children, ages 7-12	--	1,800* (0.05%)

*Incidences for asthmatic children were obtained using the national asthma prevalence among children (6.3%). The incidence of asthmatic African-American children ages 7-12 in Southeast L.A. County, for example, is 3,640,000 multiplied by {0.0937 (the proportion of the population that is ages 7-12) x 0.085 (the proportion of the population that is African-American) x 0.063 (the proportion of the national population of children that are asthmatic)}.

Table VI-5. Baseline Health Effects Incidence Rates

Health Effect	Philadelphia County	Southeast Los Angeles County	National Average[a]
Mortality[b] (per 100,000 general population/year)	1280	667	830
Morbidity:			
A. Hospital Admissions (per 100,000 general population/year)			
Total respiratory hospital admissions[c] (all ages): ICD codes 466, 480-482, 485, 490-493	816	427	--
Total respiratory hospital admissions (65 and older): ICD codes 460-519	650	428	504
COPD admissions (65 and older): ICD codes 490-496	202	116	103
Pneumonia admissions (65 and older): ICD codes 480-487	257	205	229
Ischemic heart failure (65 and older): ICD codes 410-414	614	307	450
Congestive Heart Disease (65 and older): ICD code 428	487	197	231
B. Respiratory Symptoms (percent of relevant population)			
Lower Respiratory Symptoms (LRS) in children, ages 8-12 (number of cases of symptoms per day)	0.15%*	0.15%*	--
Lower Respiratory Symptoms (LRS) in asthmatic children, ages 9-11 (number of days of symptoms)	16%*	16%*	--
(Doctor diagnosed) acute bronchitis in children ages 10-12 per year	6.5%*	6.5%*	--

All incidence rates are rounded to the nearest unit.

a. National rates for hospital admissions for patients over 64 years of age were obtained from Vital and Health Statistics, Detailed Diagnoses and Procedures, National Hospital Discharge Survey, 1990. June, 1992. CDC. Hyattsville, Md. Each rate is based on the number of discharges divided by the 1990 population of 248,709,873 .

b. Mortality figures exclude suicide, homicide, and accidental death, which corresponds to the measures used in the epidemiological studies employed in this analysis.

c. Although a baseline incidence rate is not needed for calculating the incidence of total respiratory hospital admissions associated with PM (because the concentration-response function is linear), it is needed for calculating the percent change in incidence associated with PM.

*Baseline incidence rates for respiratory symptoms were taken from the original studies: Schwartz et al. (1994): percent of all child-days on which there were respiratory symptoms, as defined in the study; Pope et al. (1991): for number of days of LRS in asthmatic children ages 10-12; and Dockery et al. (1989), for acute bronchitis in white children ages 10-12.

4. Limitations and Uncertainties

This PM health risk assessment involves substantial uncertainties given the nature of the pollutant, limited data on population exposures, and the nature of the epidemiological evidence of effects. The major uncertainties include:

- Limited information on air quality and on human activity patterns (e.g., how they vary over time and location compared to the original studies) add uncertainty to the analyses. Errors in measurement of relevant air quality, both instrument error in monitored concentrations and errors resulting from using averages of population-oriented monitors to represent population exposure, are potentially important sources of uncertainty.
- Modeled air quality simulations of attainment of alternative PM standards introduce potentially significant uncertainties, particularly in assessing the impact of alternative standards with regard to the pattern of reductions that would be observed across the distribution of air quality values.
- The use of uncertain estimates of annual average background PM concentration for each location results in uncertainties with regard to estimates that are representative of risks in excess of those potentially attributable to uncontrollable background PM levels.
- Insufficient information exists to fully assess the extent to which PM concentration-responses functions reflect the best estimates of risk associated with PM, as well as whether such functions are transferable across cities due to (1) variations in PM composition across cities, (2) the possible role of associated copollutants in influencing PM risk, and (3) variations in the relation of total exposure to ambient monitoring in different locations. There also is the additional uncertainty concerning the transferability of health functions to future PM aerosol mixes.
- The use of pooled concentration-response functions from studies in several locations to represent the overall effect of particles on a particular health endpoint in any one location introduces uncertainty.
- The impact of historical air quality on estimates of health risk from long-term PM exposures is not well understood, nor is the duration of time that a reduction in particle

concentrations must be maintained in a given location in order to experience the predicted reduction in health risk.

- Normalizing the health risk experienced or reduced in different locations due to differences in the completeness of the air quality data sets introduces uncertainty.
- Additional uncertainty is related to baseline health effects incidence information, particularly where location specific information is not available and must be estimated either by scaling national incidence rates or using reported rates from the original studies. Uncertainties in baseline health information would be expected to affect numerical estimates of total incidence more than estimates of the percentage of incidence.

Sensitivity and uncertainty analyses addressing many of these uncertainties are presented along with the PM risk estimates in the following section and in Appendix F.

C. Risk Estimates for Philadelphia and Los Angeles Counties

In the sections below risk estimates are first presented for the two locations analyzed using base case assumptions associated with "as is" PM levels. Risk estimates are then presented for Los Angeles County with PM levels adjusted to just attain the current PM_{10} standards using base case assumptions. Finally, risk estimates are presented associated with attainment of alternative $PM_{2.5}$ standards. For each of these cases, the potential impacts of alternative assumptions and uncertainties inherent in the risk assessment are examined in sensitivity analyses of individual key uncertainties and in an integrated uncertainty analysis that looks at the combined effect of several uncertainties.

1. Base Case Risk Estimates Associated with "As Is" PM Levels

The estimated health risks associated with exposure to short- and long-term ambient particle concentrations in Philadelphia County and Los Angeles County have been estimated using base case assumptions, as discussed in Section VI-B, for recent 12 month periods. Estimates for health risks posed by ambient particles measured both as PM_{10} and $PM_{2.5}$ are provided. The risk estimates for PM_{10} and $PM_{2.5}$ should be viewed as providing alternative estimates of the total health impacts of particles for the health endpoints listed in the Tables. The risk estimates for the two different measures of PM should not be summed. The estimates

are for annual health risks from particle concentrations above estimates of annual background concentrations (8 $\mu g/m^3$ PM_{10} and 3.5 $\mu g/m^3$ $PM_{2.5}$ in Philadelphia County, 6 $\mu g/m^3$ PM_{10} and 2.5 $\mu g/m^3$ $PM_{2.5}$ for Los Angeles County).

These risk estimates of effects associated with particles have been restricted to those endpoints where associations between particles and health endpoint have been demonstrated in U.S. and Canadian cities (CD, p.13-36). Risk estimates for other health endpoints reported to be associated with short-term PM_{10} concentrations, such as emergency room visits for asthma (Schwartz et al., 1993), respiratory hospitalization in children (Pope, 1991), school absences (Ransom and Pope, 1992), symptoms of cough (Schwartz et al., 1994; Ostro et al., 1991; Pope and Dockery, 1992), and asthma medication usage (Pope et al., 1991), or associated with short-term $PM_{2.5}$ concentrations, such as respiratory-related restricted activity days and work loss days in adults (Ostro and Rothschild, 1989) have not been developed. Risk estimates also have not been developed for some health endpoints reported to be associated with long-term PM concentrations, such as chronic bronchitis in adults (Abbey et al., 1995a) and decreased lung function in children (Raizenne et al., 1996) In addition, risk estimates have not been extended to different age groups from those in the original study, even though this means often estimating risks for only narrow age groups of children.[5]

a. Philadelphia County

Base case risk estimates presented in Table VI-6 suggest that PM is associated with between 1.1-1.8% (90% credible intervals (CrI) = 0.8-1.4% to 1.1-2.5%) of total mortality for short-term exposures and with about 4.6% (CrI 2.8-6.2%) of total mortality for long-term exposures in Philadelphia County. The risk estimates associated with long-term exposure are likely to reflect both a component of mortality from short-term exposures as well as mortality not tightly linked to daily changes in PM concentrations. Expressed in terms of number of deaths, the mortality incidence in Philadelphia County estimated to be associated with PM

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[5]However, for studies of respiratory symptoms in Caucasian children which were restricted to exclude racial differences for analytical purposes (Schwartz et al., 1994; Pope et al., 1991; Dockery et al., 1989) the resulting concentration-response relationships were applied to the whole population of children in the pertinent age group (children 8-12, 0-11, and 10-12 years old, respectively) in the two cities examined for the risk analysis.

ranges from 220 deaths (CrI 160-290) associated with short-term exposures to 920 deaths (CrI 580-1260) associated with long-term exposures.

Base case morbidity risk estimates associated with "as is" PM levels in Philadelphia county are approximately 2.4% (CrI 1.5-3.3%) of total respiratory hospital admissions for individuals over 64 based on a pooled analysis of studies using PM_{10} as the pollutant indicator. This compares to an estimated risk of 2.0% (CrI 0.5-3.5%) of total respiratory hospital admissions for all ages in Philadelphia County based on a single study using $PM_{2.5}$ as the pollutant indicator. Risks associated with PM exposure range from 0.7-1.4%(CrI 0.3-1.2 to 0.7-2.1%) of cardiac hospital admissions among individuals over 64 years of age for ischemic heart disease and congestive heart failure.

Risks associated with short-term exposures to PM range from 6.8% (CrI 2.4-10.9%) to 20.1% (CrI 10.3-28.3%) of the lower respiratory symptoms reported in children 8-12 years in age, depending on PM indicator and the exact ages and asthma status of the children. Long-term exposure to PM over the course of the year was estimated to be associated with a 0.3% (CrI 0-0.6%) increase in incidence of doctor diagnosed acute bronchitis among 10-12 year olds.

b. Los Angeles County

Base case risk estimates associated with "as is" PM levels in Los Angeles County are presented in Table VI-7. The PM_{10} and $PM_{2.5}$ annual concentrations are approximately double the PM concentrations in Philadelphia (annual mean concentration of approximately 52 $\mu g/m^3$ PM_{10} and 30 $\mu g/m^3$ $PM_{2.5}$ in Los Angeles County versus 25 $\mu g/m^3$ PM_{10} and 17 $\mu g/m^3$ $PM_{2.5}$ for Philadelphia). Risks associated with "as is" particle levels in Los Angeles County are estimated to range from 1.6-3.7% (CrI 0.2-3.1% to 0.8-6.3%) of total mortality for short-term exposure and to be approximately 11.9% (CrI 7.5-16.0%) of total mortality for long-term exposure. The estimate of 1.6% of total mortality is based on a study of mortality in Los Angeles County (Kinney et al., 1995). This lower estimate of mortality incidence may be due in part to the fact that this study employed the shortest averaging time (1 day) of those included in the pooled estimate (CD, p.12-72).

Table VI-6. Estimated Annual Health Risks Associated with "As Is" PM Concentrations in Philadelphia County, September 1992- August 1993 (for base case assumptions)

Health Effects*			Health Effects Associated with PM-10 Above Background**		Health Effects Associated with PM-2 5 Above Background**	
			Incidence	Percent of Total Incidence	Incidence	Percent of Total Incidence
Mortality (all ages)	(A) Associated with short-term exposure		220 (160 - 290)	1 1% (0 8 - 1 4)	370 (220 - 510)	1 8% (1 1 - 2 5)
	(B) Assoc with long-term exposure (51 locations)		-- -- -- -- -- --	-- -- -- -- -- --	920 (580 - 1260)	4 6% (2 8 - 6 2)
Hospital Admissions Respiratory	(C) Total Respiratory (all ages)		-- -- -- -- -- --	-- -- -- -- -- --	260 (70 - 450)	2 0% (0 5 - 3 5)
	(D) Total respiratory (>64 years old)		250 (150 - 340)	2 4% (1 5 - 3 3)	-- -- -- -- -- --	-- -- -- -- -- --
		(E) COPD (>64 years old)	120 (80 - 150)	3 7% (2 5 - 4 7)	-- -- -- -- -- --	-- -- -- -- -- --
		(F) Pneumonia (>64 years old)	80 (50 - 100)	1 9% (1 3 - 2 6)	-- -- -- -- -- --	-- -- -- -- -- --
Hospital Admissions Cardiac	(G) Ischemic Heart Disease *** (>64 years old)		80 (30 - 120)	0 8% (0 3 - 1 3)	70 (30 - 120)	0 7% (0 3 - 1 2)
	(H) Congestive Heart Failure *** (>64 years old)		110 (50 - 160)	1 4% (0 7 - 2 1)	100 (50 - 150)	1 3% (0 6 - 2 0)
Lower Respiratory Symptoms in Children****	(I) Lower Respiratory Symptoms (# of cases) (8-12 year olds)		< 10000 > (8000 - 11000)	17 5% (15 3 - 19 6)	< 11000 > (6000 - 15000)	20 1% (10 3 - 28 3)
	(J) Lower Respiratory Symptoms (# of days) (9-11 year old asthmatics)		< 16000 > (6000 - 25000)	6 8% (2 4 - 10 9)	-- -- -- -- -- --	-- -- -- -- -- --
	(K) Doctor-diagnosed Acute Bronchitis associated with long-term exposure (10-12 year olds)		< 190 > (20 - 370)	0 3% (0 0 - 0 6)	-- -- -- -- -- --	-- -- -- -- -- --

* Health effects are associated with short-term exposure to PM, unless otherwise specified

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level Background PM-10 is assumed to be 8 ug/m3, background PM-2 5 is assumed to be 3 5 ug/m3

*** PM-2 5 results based on using PM-2 5 mass as PM-10 mass in the PM-10 functions

****Angle brackets <> indicate incidence calculated using baseline incidence rates reported in studies, with no adjustment for location-specific incidence rates This increases the uncertainty in the incidence estimates

The numbers in parentheses for pooled functions are NOT standard confidence intervals
All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability

Sources of Concentration-Response (C-R) Functions

(A) PM-10 C-R function based on pooled results from studies in 10 locations, PM-2 5 C-R function based on pooled results from studies in six locations

(B) Pope et al , 1995

(C) Thurston, et al , 1994

(D) PM-10 C-R based on pooled results from 4 functions

(E) PM-10 C-R based on pooled results from 4 functions

(F) PM-10 C-R based on pooled results from 4 functions

(G) Schwartz & Morris, 1995

(H) Schwartz & Morris, 1995

(I) Schwartz, et al , 1994

(J) Pope et al , 1991

(K) Dockery et al , 1989

The estimated mortality risks in Los Angeles County based on the pooled, short-term mortality functions and the long-term mortality functions expressed in either percentage terms or as number of deaths are roughly two to three times the risks estimated applying the same functions in Philadelphia County. The population of the Los Angeles County area used in the analysis is more than twice as large as Philadelphia County (3.6 million versus 1.6 million), however, the death rate is half of that observed in Philadelphia (667 versus 1280 per 100,000). The differences in population size and death rate between the two study areas are largely off-setting in terms of the risk calculations, but Los Angeles County PM annual levels are nearly double those observed in Philadelphia county. Thus, the differences in risk estimates between the two study areas appears to be largely due to differences in PM levels.

With respect to morbidity health endpoints, short-term exposures to PM concentrations in Los Angeles County are estimated to be associated with approximately 6.9% (CrI 4.2-9.4%) to 7.7% (CrI 2.1-13.4%) of total respiratory hospital admissions (all ages and individuals over 64, respectively). PM also is estimated to be associated with between 1.4% (CrI 0.6-2.3%) to 4.1%(CrI 2.0-6.1%) of cardiac hospital admissions among individuals over 64 years of age for ischemic heart disease and congestive heart failure.

Short-term exposure to PM in Los Angeles County is estimated to be associated with between 18.4% (CrI 6.9-28.0%) and 41.4% (CrI 37.2-45.2) of the lower respiratory symptoms reported in children 8-12 years in age, depending on PM indicator and the ages, races, and asthma status of the children. These incidences seem high, and EPA staff notes that questions can be raised about the transferability of concentration-response functions derived in eastern U.S. locations to Los Angeles. Therefore, risk estimates based on a recent study of asthmatic symptoms among African-American children in central Los Angeles are provided for comparison (Ostro et al., 1995). Estimates based on this study indicate that daily variations in PM concentrations are associated with 19.3% (CrI 6.4-29.2%) of the reported incidence of shortness of breath, which is similar to that derived from the other studies. Long-term exposure to PM over the course of the year is estimated to be associated with a 3.1% increase (CrI 0.4-4.7%) in incidence of doctor diagnosed acute bronchitis among 10-12 year olds.

Table VI-7. Estimated Annual Health Risks Associated with "As Is" PM Concentrations in Southeast Los Angeles County, 1995* (for base case assumptions)

Health Effects**		Health Effects Associated with PM-10 Above Background***		Health Effects Associated with PM-2 5 Above Background***	
		Incidence	Percent of Total Incidence	Incidence	Percent of Total Incidence
Mortality (all ages)	(A) Associated with short-term exposure	800 (570 - 1020)	3 3% (2 3 - 4 1)	900 (200 - 1560)	3 7% (0 8 - 6 3)
	(B) Associated with short-term exposure (study done in Los Angeles)	400 (40 - 750)	1 6% (0 2 - 3 1)	– – – – – –	– – – – – –
	(C) Associated with long-term exposure (51 locations)	– – – – – –	– – – – – –	2,920 (1850 - 3930)	11 9% (7 5 - 16 0)
Hospital Admissions Respiratory	(D) Total Respiratory (all ages)	– – – – – –	– – – – – –	1,200 (330 - 2080)	7 7% (2 1 - 13 4)
	(E) Total Respiratory (>64 years old)	1,070 (660 - 1460)	6 9% (4 2 - 9 4)	– – – – – –	– – – – – –
	(F) COPD (>64 years old)	440 (310 - 560)	10 3% (7 3 - 13 1)	– – – – – –	– – – – – –
	(G) Pneumonia (>64 years old)	420 (290 - 550)	5 6% (3 9 - 7 3)	– – – – – –	– – – – – –
Hospital Admissions Cardiac	(H) Ischemic Heart Disease**** (>64 years old)	260 (100 - 420)	2 3% (0 9 - 3 7)	160 (60 - 260)	1 4% (0 6 - 2 3)
	(I) Congestive Heart Failure**** (>64 years old)	290 (140 - 430)	4 1% (2 0 - 6 1)	180 (90 - 270)	2 5% (1 2 - 3 8)
Lower Respiratory Symptoms in Children *****	(J) Lower Respiratory Symptoms (# of cases) (8-12 year olds)	< 62000 > (56000 - 68000)	41 4% (37 2 - 45 2)	< 51000 > (28000 - 68000)	34 4% (19 1 - 45 7)
	(K) Lower Respiratory Symptoms (# of days) (9-11 year old asthmatics)	< 115000 > (43000 - 175000)	18 4% (6 9 - 28 0)	– – – – – –	– – – – – –
	(L) Days of shortness of breath (7-12 year old African American asthmatics in Los Angeles)	< 7200 > (2400 - 10900)	19 3% (6 4 - 29 2)	– – – – – –	– – – – – –
	(L) Doctor-diagnosed Acute Bronchitis associated with long-term exposure (10-12 year olds)	< 5090 > (680 - 7750)	3 1% (0 4 - 4 7)	– – – – – –	– – – – – –

* Southeast Los Angeles County was not in attainment of current PM-10 standards (50 ug/m3 annual average standard and 150 ug/m3 daily standard) in 1995 Figures shown use the actual reported concentrations

** Health effects are associated with short-term exposure to PM, unless otherwise specified

*** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level Background PM-10 is assumed to be 6 0 ug/m3 and background PM-2 5 is assumed to be 2 5 ug/m3

**** PM-2 5 results based on using PM-2 5 mass as PM-10 mass in the PM-10 functions

*****Angle brackets <> indicate incidence calculated using baseline incidence rates reported in studies, with no adjustment for location-specific incidence rates This increases the uncertainty in the incidence estimates

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability.

Sources of Concentration-Response (C-R) Functions

(A) PM-10 C-R function based on pooled results from studies in 10 locations, PM-2 5 C-R function based on pooled results from studies in six locations

(B) Kinney et al ,1995

(C) Pope et al , 1995

(D) Thurston, et al , 1994

(E) PM-10 C-R based on pooled results from 4 functions

(F) PM-10 C-R based on pooled results from 4 functions

(G) PM-10 C-R based on pooled results from 4 functions

(H) Schwartz & Morris, 1995

(I) Schwartz & Morris, 1995

(J) Schwartz, et al , 1994

(K) Pope et al , 1991

(L) Dockery et al , 1989

c. Key Uncertainties

There are additional uncertainties about the risk estimates for both locations beyond those reflected in the credible intervals. These additional uncertainties include but are not limited to the degree of transferability of concentration-response functions and measurement error in air quality values for each location. Because national or community gathering of respiratory symptoms information is not routinely performed, the numbers of days or cases of symptoms is estimated by applying the percentage of incidence associated with PM to the baseline incidence rates reported in the health studies, which are from locations different than those being analyzed, with the exception of the Ostro et al. (1995) study. Baseline incidence may be considerably different from that observed in the cities analyzed, resulting in additional uncertainty pertaining to the numerical estimates of incidence reported in Tables VI-6 and VI-7. The estimates of percent incidence are less uncertain than the estimates of incidence counts for respiratory symptoms risk estimates in both Philadelphia and Los Angeles.

2. Base Case Risk Estimates Upon Attainment of Current Standards

For comparisons with alternative standards it is desirable to estimate health risks associated with PM air quality that does not include the effects of concentrations in excess of those allowed by the current national PM standards. For Philadelphia county, Table VI-6 also represents the estimated health risks associated with PM at or below the current PM_{10} standards, since the monitors used in estimating Philadelphia's air quality are already in attainment of the current PM_{10} standards. For Los Angeles County, however, the estimates given in Table VI-7 include contributions from concentrations in excess of those allowed by the current PM_{10} standards. The PM_{10} concentrations for the monitors used in the risk analysis in Los Angeles County have an annual mean controlling value of 52 $\mu g/m^3$ and a 2nd-daily max controlling value of 195 $\mu g/m^3$, versus the current PM_{10} standards of 50 $\mu g/m^3$ annual mean and 150 $\mu g/m^3$, 24-hr average. Adjusting PM air quality for Los Angeles County to simulate attainment of the current PM_{10} standards introduces additional uncertainty into the risk estimates, but is required in order to compare risks associated with attaining the current PM_{10} standards with risks associated with meeting alternative $PM_{2.5}$ standards.

The method chosen to simulate attainment of the current PM_{10} standards is to apply a proportional rollback to both PM_{10} and $PM_{2.5}$ concentrations (preserving the $PM_{2.5}/PM_{10}$ ratio) to air quality concentrations that "just attain" current standards (under current interpretation, this means reducing annual mean concentrations to 50.4 $\mu g/m^3$, and the second daily max concentration[6] to 154 $\mu g/m^3$, to reflect rounding conventions used to judge attainment). This modeling of attainment in Los Angeles County through proportional rollback contains two analytic assumptions. First, it assumes that the general shape of the distribution of PM air quality concentrations in Los Angeles County will remain the same as observed under the "as is" situation and that PM levels will be reduced proportionately based on the controlling standard. For Los Angeles County the 24-hr second daily max concentration of 195 $\mu g/m^3$ is the controlling value and needs to be reduced 21% to bring it into attaintment. Thus, the amount of each PM concentration above estimated background for the 1995 year in Los Angeles County was reduced by 21%. The second assumption is that the relationship between $PM_{2.5}$ and PM_{10} ($PM_{2.5}/PM_{10}$ ratio = 0.58) would be preserved as PM_{10} concentrations are reduced. If control strategies are used to reach attainment that preferentially controls coarse particles relative to fine particles (as has been observed in some areas, see Chapter IV), or that preferentially controls fine particles relative to coarse particles, this simplifying assumption introduces some inaccuracy. If the error is in the direction of not adequately reflecting a preferential control of coarse particles, then $PM_{2.5}$ concentrations in the "just attain PM_{10} standards case" would be expected to be higher than those estimated in this analysis. In this case, larger reductions in PM health risks would be expected than those reported later in the alternative standards risk analysis.

The results for Los Angeles County based on simulating attainment of the current PM_{10} standards are shown in Table VI-8. The reduction in PM concentrations results in an approximately 18-28% reduction in the risk estimates associated with short-term PM exposures compared to "as is" levels. This provides an example of how the estimated change in health

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[6] The current 24-hr standards are applied to the 4th highest daily concentration in a three year period. Since we are only examining a year of air quality concentrations in the risk analysis, the second daily max concentration was chosen as an approximate surrogate for the 4th highest concentration in three years value.

Table VI-8. Estimated Annual Health Risks Associated with Attainment of Current Standards in Southeast Los Angeles County, 1995* (for base case assumptions)

Health Effects**		Health Effects Associated with PM-10 Above Background***		Health Effects Associated with PM-2 5 Above Background***	
		Incidence	Percent of Total Incidence	Incidence	Percent of Total Incidence
Mortality (all ages)	(A) Associated with short-term exposure	630 (450 - 800)	2 6% (1 8-3 3)	710 (430 - 970)	2 9% (1 7 - 3 9)
	(B) Associated with short-term exposure (study done in Los Angeles)	290 (30 - 550)	1 2% (0 1 - 2 2)	-- -- -- -- -- --	-- -- -- -- -- --
	(C) Associated with long-term exposure (51 locations)	-- -- -- -- -- --	-- -- -- -- -- --	2,110 (1330 - 2860)	8 6% (5 4 - 11 7)
Hospital Admissions Respiratory	(D) Total Respiratory (all ages)	-- -- -- -- -- --	-- -- -- -- -- --	940 (250 - 1630)	6 1% (1 6 - 10 5)
	(E) Total Respiratory (>64 years old)	840 (520 - 1160)	5 4% (3 3 - 7 4)	-- -- -- -- -- --	-- -- -- -- -- --
	(F) COPD (>64 years old)	350 (240 - 440)	8 2% (5 8 - 10 5)	-- -- -- -- -- --	-- -- -- -- -- --
	(G) Pneumonia (>64 years old)	330 (230 - 430)	4 4% (3 1 - 5 8)	-- -- -- -- -- --	-- -- -- -- -- --
Hospital Admissions Cardiac	(H) Ischemic Heart Disease**** (>64 years old)	200 (80 - 330)	1 8% (0 7 - 2 9)	130 (50 - 200)	1 1% (0 4 - 1 8)
	(I) Congestive Heart Failure**** (>64 years old)	230 (110 - 340)	3 2% (1 5 - 4 8)	140 (70 - 210)	2 0% (1 0 - 3 0)
Lower Respiratory Symptoms in Children *****	(J) Lower Respiratory Symptoms (# of cases) (8-12 year olds)	< 52000 > (46000 - 57000)	34 8% (31 0 - 38 4)	< 43000 > (23000 - 58000)	28 7% (15 4 - 39 0)
	(K) Lower Respiratory Symptoms (# of days) (9-11 year old asthmatics)	< 93000 > (34000 - 143000)	14 9% (5 5 - 23 0)	-- -- -- -- -- --	-- -- -- -- -- --
	(L) Days of shortness of breath (7-12 year old African American asthmatics in Los Angeles)	< 5200 > (1700 - 8100)	14 1% (4 6 - 21 8)	-- -- -- -- -- --	-- -- -- -- -- --
	(L) Doctor-diagnosed Acute Bronchitis associated with long-term exposure (10-12 year olds)	< 3760 > (470 - 6190)	2 3% (0 3 - 3 7)	-- -- -- -- -- --	-- -- -- -- -- --

* Southeast Los Angeles County was not in attainment of current PM-10 standards (50 ug/m3 annual average standard and 150 ug/m3 daily standard) in 1995 "As is" daily PM-10 concentrations were first rolled back to simulate attainment of these standards "As is" daily PM-2 5 concentrations were rolled back by the same percent as daily PM-10 concentrations See text in Chapter VI for details

** Health effects are associated with short-term exposure to PM, unless otherwise specified

*** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level Background PM-10 is assumed to be 6 0 ug/m3 and background PM-2 5 is assumed to be 2 5 ug/m3

**** PM-2 5 results based on using PM-2 5 mass as PM-10 mass in the PM-10 functions

*****Angle brackets <> indicate incidence calculated using baseline incidence rates reported in studies, with no adjustment for location-specific incidence rates This increases the uncertainty in the incidence estimates

The numbers in parentheses for pooled functions are NOT standard confidence intervals
All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability
See text in Chapter VI for details

Sources of Concentration-Response (C-R) Functions

(A) PM-10 C-R function based on pooled results from studies in 10 locations, PM-2 5 C-R function based on pooled results from studies in six locations
(B) Kinney et al ,1995
(C) Pope et al , 1995
(D) Thurston, et al , 1994
(E) PM-10 C-R based on pooled results from 4 functions
(F) PM-10 C-R based on pooled results from 4 functions
(G) PM-10 C-R based on pooled results from 4 functions
(H) Schwartz & Morris, 1995
(I) Schwartz & Morris, 1995
(J) Schwartz, et al , 1994
(K) Pope et al , 1991
(L) Dockery et al , 1989

risks associated with PM is approximately equal to the amount of proportional air quality reduction required (for Los Angeles County, a reduction of 21% in air quality concentrations results in a 18-28% reduction in health risks associated with short-term exposures). This correspondence results from the shape of the concentration-response relationships reported in the literature and in the base case analysis, which are essentially linear over most of the range of concentrations considered here. For risks associated with long-term exposures, the reduction is greater than the relative change in PM levels because estimated health risks associated with long-term exposures are quantified relative to lowest observed annual mean concentrations in the health studies used in the risk analysis which are considerably in excess of background.

Although there are substantial uncertainties in predicting annual health risks associated with attainment of the current standards in Los Angeles County, the estimates in Table VI-8 suggest that short-term exposure to PM could be associated with approximately 1.2% (CrI 0.1-2.2%) to 2.9% (CrI 1.7-3.9%) of mortality, 5.4% (CrI 3.3-7.4%) of respiratory hospital admissions for those over 65, 1.1% (CrI 0.4-1.8%) to 3.2% (CrI 1.5-4.8%) of cardiac hospital admissions for ischemic heart disease and congestive heart failure, and from 14.9% (CrI 5.5-23.0%) to 34.8 (CrI 31.0-38.4%) of respiratory symptoms in children upon attainment of the current PM_{10} standards. Estimated mortality associated with long-term exposure is about 8.6% (CrI 5.4-11.7%) and doctor-diagnosed acute bronchitis associated with long-term exposure is about 2.3% (CrI 0.3-3.7%) upon attainment of the current NAAQS. However, in considering such estimates it is important to consider the substantial uncertainties that may affect these estimates. The next section summarizes the results of several sensitivity analyses to provide some insight into the magnitude of the uncertainties associated with the PM risk estimates. Additional uncertainties, not captured by the sensitivity analyses, were discussed previously in Section VI.B and VI.C.1.c.

3. Uncertainty Analyses of Estimated Risks Associated with "As Is" PM Levels in Philadelphia County and Attaining Current PM_{10} Standards in Los Angeles County

a. Sensitivity Analyses of Individual Key Uncertainties

A number of sensitivity analyses of the health risk model have been conducted to provide some perspective on the impact of various uncertainties and assumptions on the health risk estimates presented in this Staff Paper. These sensitivity analyses are presented in Appendix F and in the technical support document (Abt Associates, 1996b). Table VI-9 summarizes the results of a number of these sensitivity analysis indicating the effects of alternative specifications for several important air quality and concentration-response parameters (background, cutpoint concentrations, averaging time for mortality functions, and the effects of reduced slopes for long-term mortality functions resulting from the potential effects of inadequately considered confounders or previous air quality). The results are presented as a range of estimates of the percent of mortality and respiratory hospital admissions incidence associated with PM under "as is" air quality in Philadelphia County.

From Table VI-9 it can be seen that the estimates of health risks show particular sensitivity to assumptions concerning the use of appropriate cutpoint concentrations for quantifying risk.[7] The cutpoints used in the analysis can be used to inform judgments concerning the potential effects of nonlinear concentration-response relationships resulting from potential biological considerations, copollutant effects, or exposure misclassification associated with the use of ambient monitors as a measure of population exposures.

Disaggregating the pooled PM_{10} mortality analysis into subsets of studies with effects estimates based on more homogenous averaging times also can make substantial differences in the estimates of PM_{10} mortality health risk; for example, when studies with the shortest (1-day) and longest (3-5 day) averaging times are contrasted. As would be expected, assuming lower than reported coefficients for long-term mortality risk from PM exposures reduces risk

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[7]To quantify risks above various cutpoints, two alternative slope adjustment methods have been used to examine the potential impact of a concentration-response function having a steeper slope (i.e., larger RRs per μg) above specified cutpoints. See Figure VI-6 and discussion in Appendix F for further details.

estimates by an amount equal to the reduction in the coefficient. The estimates of health risks associated with PM also show some degree of sensitivity to alternative specifications of background concentrations.

One important uncertainty that is not included in Table VI-9 concerns the effect of copollutants on the estimated risks associated with PM. The base case estimates risk resulting from concentration-response relationships developed without inclusion of copollutants. Since not all of the studies included in the base case analysis controlled for copollutants by simultaneously incorporating them in the analysis, it is not possible to directly estimate the sensitivity of the base case results by taking into account the effect of simultaneous inclusion of all copollutants in all studies. However, an examination of the sensitivity of risk estimates from individual studies that did include copollutants is provided in Appendix F, Table F-5b. The results for most, but not all, of the studies are consistent with the assessment in the CD that the magnitude of PM effects and their statistical uncertainty in many studies showed little sensitivity to the adjustment for copollutants (CD, p.13-55). As discussed in Section V.E., however, reanalyses of Philadelphia using TSP data by the HEI (Samet et al., 1996a) and Mooglavkar et al. (1995a,b) have reported a potential for more significant interaction by copollutants when multiple pollutants are entered into the concentration-response model. The implications of the perspective that PM may be serving as an index reflecting the effects of several pollutants in combination is discussed below in section VI.C.4 and is an area of uncertainty that needs to be investigated further.

Similar sensitivity analyses to the ones summarized above for Philadelphia County were performed for Los Angeles County. A primary point of interest is that the Los Angeles County risk estimates show less sensitivity to the choice of cutpoint than the Philadelphia County results, since a larger proportion of days in Los Angeles County have PM concentrations above some or all of the cutpoints analyzed (see exhibits 7.17 - 7.20 in Abt Associates, 1996b).

Table VI-9. Summary of Selected Sensitivity Analyses on Estimates of Risk Associated with PM in Philadelphia County

HEALTH ENDPOINT	PM Indicator	BASE CASE Central Estimate	SENSITIVITY ANALYSES Central Estimates				
			BACKGROUND[1] (Low-High Concentration)	CUTPOINT[2] Method I (Low-High)	CUTPOINT[2] Method II (Low- High)	AVG TIME[3] (5 day-1 day)	SLOPE REDUCTION[4] Long-Term Study
MORTALITY Short-Term Exposure	PM_{10}	**1.1%**	1.3 - 0 9%	0.4 - 0.1%	0.4 - 0.1%	1.8 -0.4%	---
	$PM_{2.5}$	**1.8%**	2.0 - 1.6%	1.1 - 0.1%	1.0 - 0.1.%	---	---
MORTALITY Long-Term Exposure	$PM_{2.5}$	**4.6%**	No change[5]	2.4 - 0%[6]		---	3.4 - 2.3%
HOSPITAL ADMISSIONS Total Respiratory[7]	PM_{10}	**2.4%**	2 9 - 1.9%	1 3 - 0.4%	1.0 - 0.2%	---	---
	$PM_{2.5}$	**2.0%**	2.3 - 1 8%	1 4 - 0 4%	1.2 - 0.2%	---	---

[1] Low = 5 $\mu g/m^3$ PM_{10}, 2 $\mu g/m^3$ $PM_{2.5}$; High = 11 $\mu g/m^3$ PM_{10}, 5 $\mu g/m^3$ $PM_{2.5}$; Base Case = 8 $\mu g/ m^3$ PM_{10}, 3.5 $\mu g/m^3$ $PM_{2.5}$

[2] Low = 20 $\mu g/m^3$ PM_{10}, 10 $\mu g/m^3$ $PM_{2.5}$; High = 40 ug/m^3 PM_{10}, 30 $\mu g/m^3$ $PM_{2.5}$; Base Case = linear relationship above background. Method I and Method II refer to methods of adjusting the slope of the concentration-response relationship above the cutpoint upwards to different extents to reflect the anticipated effect of a "hockey stick"-style threshold concentration response function. See Appendix F for further details..

[3] 5 day = results using 3-5 day averaging time studies; 1 day = result using single day averaging time study; Base Case used 2 day averaging time.

[4] First number represents effect of 33% reduction in slope; second number represents effect of 50% reduction in slope; Base Case used relative risk as reported in study (i.e., no adjustment). Slope Reduction intended to roughly model potential effects of previous air quality or uncontrolled confounding.

[5] Background concentration sensitivity analyses make no difference in the risk estimates for mortality from long-term exposure since the lowest observed concentrations in this studies (the limit to which the concentration-response function was applied) was well above background.

[6] Low = 12.5 $\mu g/m^3$ $PM_{2.5}$; High = 18 $\mu g/m^3$ $PM_{2.5}$; Base Case = linear relationship above the lowest observed concentration in study (9 $\mu g/m3$). No slope adjustment was made to the long-term mortality concentration-response relationship when applying the cutpoints.

[7] Total Respiratory Hospital Admissions for those > 64 yrs of age for PM_{10}; for all ages for $PM_{2.5}$

In general, these sensitivity analyses indicate that alternative analytic choices within the range of those considered in this analysis may lead to sizable differences in risk estimates. However, these are also primarily intended as bounding exercises to characterize the magnitude of potential uncertainty, and as such do not reflect judgments concerning the likelihood of specific alternative cases tested.

b. Integrated Uncertainty Analysis

In addition to individual sensitivity analyses discussed above, an integrated uncertainty analysis has been conducted for mortality associated with short-term exposures to $PM_{2.5}$ to assess the potential combined effects of several key uncertainties simultaneously. Through Monte Carlo sampling approaches, a distribution of values for several key parameters in the model has been estimated or specified, and 90 percent credible intervals have been generated representing the probability that the risk estimates fall within a particular range once the combined effect of these uncertainties have been considered. An advantage of this approach is that it allows the combined effect of several uncertainties to be quantitatively estimated. A major difficulty of the approach, however, is that the method inherently requires an estimate of the distribution of values for each uncertainty included, even if little empirical evidence is available to inform what is an appropriate choice for each distribution. Since there is little information on which to base some of the distributions and/or weightings chosen to represent certain key parameters in the integrated uncertainty analyses, the results of this analysis should be viewed as illustrative in character. The purpose of the analysis is to show the potential sensitivity of the risk estimates when several uncertainties, rather than just a single uncertainty, are considered simultaneously.

As discussed earlier in this Chapter, there are a number of uncertainties encountered as one attempts to estimate health risks associated with PM levels for a given city or location. Given the availability of specific data for baseline health effects incidence and daily PM air quality data for the two locations examined (i.e., Philadelphia and Los Angeles Counties), staff judges that the uncertainties associated with these two inputs to the risk model are relatively small compared to the uncertainties associated with what is the appropriate concentration-response function for these locations. Therefore, the integrated uncertainty

analysis is primarily focused on the concentration-response uncertainties, since this is judged to be the largest source of uncertainty in the health risk model. In addition, uncertainty about background levels and uncertainty about how PM air quality distributions might change upon attainment of alternative standards also is included in the analysis.

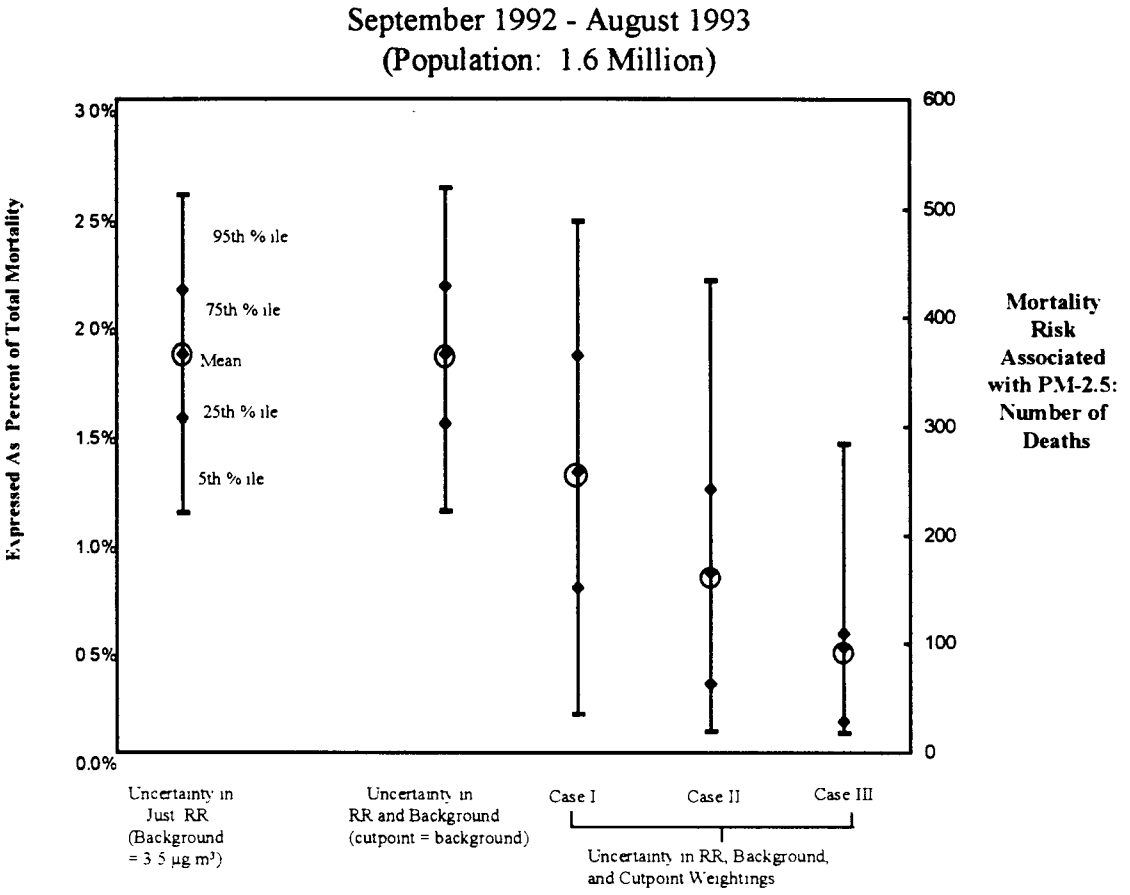
Table VI-10 below summarizes how each of the uncertainties incorporated into the integrated uncertainty analysis is treated. As outlined in Appendix E, there is substantial uncertainty concerning whether cutpoint concentrations above background exist based on a review of the available data. As discussed previously in this Chapter and in Appendix E, various approaches have been used to derive cutpoints of interest from the available data. The current data does not provide strong evidence concerning where a cutpoint concentration might exist (CD). To account for this state of uncertainty, the integrated uncertainty analysis use several illustrative weightings to assess the possible effects of this important uncertainty in combination with other key uncertainties (i.e., estimated background levels, air quality rollback approach). Each of the key uncertainties were incorporated sequentially into the analysis to illustrate the impact of each uncertainty on the risk estimates.

Figure VI-7 displays the results of the integrated uncertainty analysis for mortality associated with short-term exposure to $PM_{2.5}$ for Philadelphia County under the "as is" scenario. The risk estimates are expressed in terms of both number of deaths over a 1-year period and as a percent of total mortality. Each vertical bar represents a set of risk estimates that includes the uncertainties identified below the bars. The mean estimate is given, as well as the 5th, 25th, 75th, and 95th percentiles. The first vertical bar includes only uncertainty in the RR and assumes that background equals 3.5 $\mu g/m^3$. The second vertical bar incorporates uncertainty in RR and in the $PM_{2.5}$ background concentration for Philadelphia, with the cutpoint set equal to the background concentration. The final three vertical bars incorporate uncertainties about RR, background, and three weighting schemes differentially weighting the likelihood that various cutpoint (or threshold) concentrations exist. The three weighting schemes are indicated in the box below Figure VI-7. Case I represents a judgment that concentration-response functions are more likely to exist down to background or 10 $\mu g/m^3$; Case III represents a judgment that concentration-response functions are more likely to have a

Table VI-10. Summary of Uncertainties Incorporated Into Integrated Uncertainty Analysis

Uncertainty	Distribution
Coefficient (β) in concentration-response function	Based on distribution of β's obtained from pooled results of $PM_{2.5}$ mortality studies in six locations
Cutpoints in concentration-response function	Four cutpoints (background, 10, 18, 30 $\mu g/m^3$) with three discrete weighting schemes and two slope adjustment methods
Background $PM_{2.5}$ concentration	Uniform distribution on the intervals [2,5] and [1,4] ($\mu g/m^3$) for Philadelphia County and Los Angeles County, respectively, based on the estimated ranges identified in the CD for the Eastern and Western sections of the United States
Shape of $PM_{2.5}$ air quality distribution upon attainment of alternative standards	Based on distribution of regression slope of linear rollback over background to ratio of second high 24-hr $PM_{2.5}$ values for 129 pairs of site-years of data (see Section 8.2 in Abt Associates (1996b))

Figure VI-7. Effect of Several Uncertainties on Mortality Risk Associated With Short-Term Exposure to PM-2.5 in Philadelphia County



Uncertainty in background concentration enters into these calculations only when the cutpoint is set equal to background. The other cutpoints are greater than the highest background concentration considered.

Cutpoint Weighting Schemes

	Case I	Case II	Case III
Background	0.5	0.2	0.05
10 μg/m^3	0.3	0.3	0.15
18 μg/m^3	0.15	0.3	0.5
30 μg/m^3	0.05	0.2	0.3

cutpoint at 18 or 30 $\mu g/m^3$; and Case II represents a judgment that concentration-response functions are somewhat more likely to have cutpoints in the 10-18 $\mu g/m^3$ range.[8] Figure VI-8 shows a similar figure for Los Angeles County where attainment of the current PM_{10} standards is simulated.

The results of the integrated uncertainty analysis illustrate the impact on the mortality risk estimates of whether or not one judges there to be a likely cutpoint or threshold above estimated background levels. If one assumes no cutpoint above background, mortality associated with short-term exposure in Philadelphia County under the "as is" scenario is estimated to be about 1.8 (CrI 1.2-2.7) percent of total mortality or 375 (CrI 225-525) excess deaths. Allowing for the possibility of a cutpoint above estimated background levels, three alternative cutpoint weighting schemes reduce the mean risk estimates to about 1.3, 0.8, and 0.5 percent of total mortality for Cases I, II, and III, respectively. For Cases I and II the 90 percent credible intervals also become considerably wider than the risk estimates incorporating only uncertainty in the RR slope and estimated background concentration and all three cutpoint weighting schemes indicate a lower bound of the 90 percent credible interval of about 0.2-0.3 percent of total mortality. For Los Angeles County under the just attaining the current PM_{10} standards, the mean mortality risk estimates assuming no cutpoint is about 2.8 percent (CrI 1.7-3.8). The alternative cutpoint weighting schemes reduce the mean mortality risk estimates to about 2.2, 1.6, and 1.2 percent for Cases I, II, and III, respectively. The higher risk estimates in Los Angeles County are due mainly to the higher $PM_{2.5}$ levels, since Philadelphia County air quality is lower (i.e., better) than the current PM_{10} standards.

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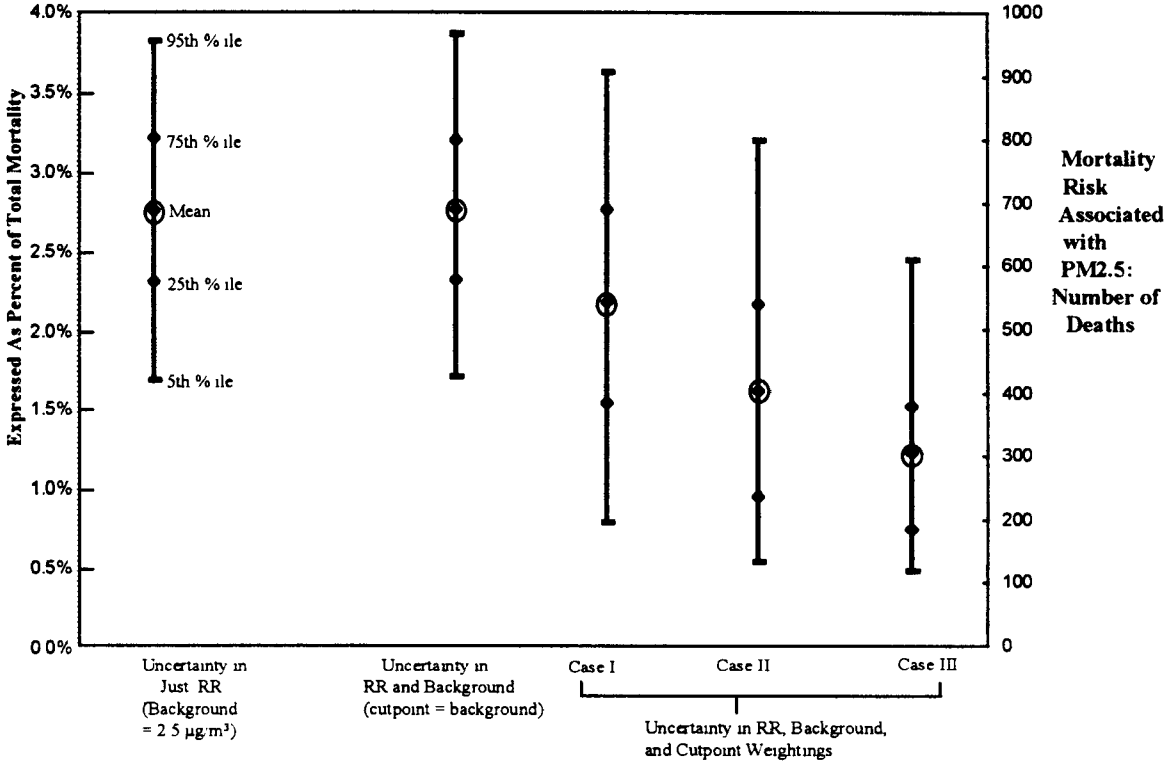
[8]In the sensitivity analysis described previously in the Chapter two different methods for adjusting the slope of the concentration-response function were examined when various cutpoints (or thresholds) were analyzed. In the integrated uncertainty analysis, the two slope adjustment methods were given equal weight.

Figure VI-8. Effect of Several Uncertainties on Mortality Risk Associated With Short-Term Exposure to PM-2.5 After Meeting Current PM-10 Standards in Los Angeles County

(Population: 3.6 Million)



Uncertainty in background concentration enters into these calculations only when the cutpoint is set equal to background. The other cutpoints are greater than the highest background concentration considered.

Cutpoint Weighting Schemes

	Case I	Case II	Case III
Background	0.5	0.2	0.05
10 $\mu g/m^3$	0.3	0.3	0.15
18 $\mu g/m^3$	0.15	0.3	0.5
30 $\mu g/m^3$	0.05	0.2	0.3

4. Risk Estimates Associated with Alternative $PM_{2.5}$ Standards

This section presents risk estimates associated with just attaining several alternative $PM_{2.5}$ standards for the Philadelphia and Los Angeles County study areas. In addition to risk estimates using base case assumptions, individual sensitivity analyses and integrated uncertainty analyses also are presented, analogous to the approach used for the "as is" risk estimates. The additional uncertainty introduced primarily by adjusting air quality to reflect future attainment of alternative standards also is discussed.

a. Base Case Risk Estimates

Table VI-11a summarizes the air quality information indicating which monitor in each location has the "controlling value" for a rollback to attain 24-hr or annual mean alternative standards.[9] Table VI-11b shows the amount of reduction in air quality required to attain the alternative $PM_{2.5}$ standard, and which standard of the combination, daily or annual, is "controlling" (i.e., requires the larger reduction in concentration). To model attainment of alternative $PM_{2.5}$ standards, a proportional rollback approach is used as the base case. Although it is extremely difficult to predict what patterns of air quality would be observed in these two locations upon attaining alternative $PM_{2.5}$ standards, a preliminary investigation of changes in $PM_{2.5}$ air quality observed over the past 15 years of limited monitoring reported to the AIRS database finds that the general pattern of air quality changes observed is a proportional change in both daily and annual mean concentrations (Abt Associates, 1996b). The estimated effects of alternative assumptions concerning patterns of air quality rollback are presented in Table VI-14.

Tables VI-12a and VI-12b show the risk estimates for just attaining alternative $PM_{2.5}$ standards in Philadelphia County, and Tables VI-13a and VI-13b show the risk estimates for just attaining alternative $PM_{2.5}$ standards in Los Angeles County using base case assumptions. Similar to the approach used to model attainment of the current PM_{10} standards in Los Angeles

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[9] The terminology of "controlling value" and "controlling monitor" are used here as synonyms for the well-known terms "design value" and "design value monitors". The monitors used in the risk analysis are not genuine design value monitors established for particular air sheds, and thus the alternative terminology is used to avoid confusion.

Table VI-11a. **Controlling Monitors for Rollbacks to Attain Alternative PM-2.5 Standards**

Monitor Site	Weighted Annual Average $PM_{2.5}$ Concentration*	Second Daily Maximum 24-Hour $PM_{2.5}$ Concentration*	Controlling Monitor
Philadelphia County			
N/E	16	65	
PBY	17	72	For daily standard
TEM	17	70	For annual standard
Southeast Los Angeles County			
Central LA	24	91	For annual standard
Diamond Bar	22	102	For daily standard

All concentrations are given in $\mu g/m^3$.

*Both weighted annual averages and second daily maximum concentrations at the two monitors in Southeast Los Angeles County were adjusted to reflect attainment of the current PM_{10} annual standard of 50 $\mu g/m^3$ and the current PM_{10} daily standard of 150 $\mu g/m^3$. These standards are currently attained in Philadelphia County.

Table VI-11b. Controlling Standards and Percent Rollbacks Necessary to Attain Alternative $PM_{2.5}$ Standards

Alternative PM-2.5 Standards		**Philadelphia County**	**Southeast Los Angeles County**
Annual Avg. Standard	24-Hour Standard	Controlling Standard and Percent Rollback*	Controlling Standard and Percent Rollback**
20 alone		----	Annual -- 18.8%
20	65	Daily -- 10.4%	Daily -- 37.0%
20	50	Daily -- 32.3%	Daily -- 52.1%
20	25	Daily -- 68.7%	Daily -- 77.3%
15 alone		Annual -- 15.5%	Annual -- 42.0%
15	65	Annual -- 15.5%	Annual -- 42.0%
15	50	Daily -- 32.3%	Daily -- 52.1%
15	25	Daily -- 68.7%	Daily -- 77.3%

All concentrations are given in $\mu g/m^3$.

*Based on controlling values for Philadelphia County of 17 $\mu g/m^3$ for the annual standard and 72 $\mu g/m^3$ for the daily standard.

** Based on controlling values for Southeast Los Angeles County of 24 $\mu g/m^3$ for the annual standard and 102 $\mu g/m^3$ for the daily standard.

Table VI-12a. Estimated Changes in Health Risks Associated with Meeting Alternative PM-2.5 Standards in Philadelphia County, September 1992 - August 1993 (for base case assumptions)

Health Effects*		PM-2 5-Associated Incidence associated with current standards**	Incidence Associated with Meeting Alternative Standards			
			20 ug/m3 annual	20 ug/m3 annual and 65 ug/m3 daily	20 ug/m3 annual and 50 ug/m3 daily	20 ug/m3 annual and 25 ug/m3 daily
Mortality (all ages)	(A) Associated with short-term exposure	370 (220 - 510)	370 (220 - 510)	330 (200 - 460)	250 (150 - 340)	110 (70 - 160)
	Percent Reduction in PM-Associated Incidence:* **		0.0%	10.8%	32.4%	70 3%
	Percent Reduction in Total Incidence:**		0 0%	0.2%	0 6%	1.3%
	(B) Associated with long-term exposure	920 (580 - 1260)	920 (580 - 1260)	750 (440 - 960)	390 (230 - 490)	0 (0 - 0)
	Percent Reduction in PM-Associated Incidence:		0 0%	18 5%	57.6%	100 0%
	Percent Reduction in Total Incidence:		0 0%	0.8%	2.6%	4 6%
Hospital Admissions Respiratory	(C) Total Respiratory (all ages)	260 (70 - 450)	260 (70 - 450)	230 (60 - 400)	180 (50 - 300)	80 (20 - 140)
	Percent Reduction in PM-Associated Incidence:		0 0%	11 5%	30.8%	69 2%
	Percent Reduction in Total Incidence:		0.0%	0.2%	0 6%	1 4%
Hospital Admissions Cardiac	(D) Ischemic Heart Disease***** (>64 years old)	70 (30 - 120)	70 (30 - 120)	60 (30 - 110)	50 (20 - 80)	20 (10 - 40)
	(E) Congestive Heart Failure***** (>64 years old)	100 (50 - 150)	100 (50 - 150)	90 (40 - 130)	70 (30 - 100)	30 (20 - 40)
	Range of Percent Reductions in PM-Associated Incidence:		0.0% - 0 0%	10.0% - 14.3%	28.6% - 30.0%	70.0% - 71.4%
	Range of Percent Reductions in Total Incidence:		0.0% - 0.0%	0.1% - 0.1%	0.2% - 0.4%	0.5% - 0.9%
(F) Lower Respiratory Symptoms (8-12 yr olds) ******		< 11000 > (6000 - 15000)	< 11000 > (6000 - 15000)	< 10000 > (5000 - 13000)	< 7000 > (4000 - 9000)	< 3000 > (2000 - 4000)
	Percent Reduction in PM-Associated Incidence:		0.0%	9.1%	36.4%	72 7%
	Percent Reduction in Total Incidence:		0.0%	1.6%	7.3%	14.6%

* Health effects are associated with short-term exposure to PM, unless otherwise specified

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background PM-2 5 level Background PM-2 5 is assumed to be 3 5 ug/m3 in Philadelphia County

*** The percent reduction in PM-associated incidence achieved by attaining alternative standards as opposed to the current standards is the reduction in incidence divided by the incidence associated with current standards For example, the percent reduction in PM-associated incidence of mortalit associated with short-term exposure to PM-2 5 achieved by meeting both a 15 ug/m3 annual and a 65 ug/m3 daily standard is (370-330)/370=10 8%

**** The percent reduction in total incidence achieved by attaining current or alternative standards is the reduction in incidence achieved by attaining the standard divided by the total (not only PM-associated) incidence.

***** PM-2 5 results based on using PM-2 5 mass as PM-10 mass in the PM-10 functions

******Angle brackets <> indicate incidence calculated using baseline incidence rates reported in studies, with no adjustment for location-specific incidence rates *This increases the uncertainty in the incidence estimates*

The numbers in parentheses for pooled functions are NOT standard confidence intervals All the numbers in parentheses are interpreted as 90% credible intervals based on Monte Carlo analysis that takes into account both statistical uncertainty and possible geographic variability See text in Chapter VI for details

Sources of Concentration-Response (C-R) Functions
(A) C-R function based on pooled results from studies in six locations
(B) Pope et al , 1995
(C) Thurston, et al , 1994
(D) Schwartz & Morris, 1995
(E) Schwartz & Morris, 1995
(F) Schwartz, et al , 1994

Table VI-12b. Estimated Changes in Health Risks Associated with Meeting Alternative PM-2.5 Standards in Philadelphia County, September 1992 - August 1993 (for base case assumptions)

Health Effects*		PM-2.5-associated Incidence associated with current standards**	Incidence Associated with Meeting Alternative Standards			
			15 ug/m3 annual	15 ug/m3 annual and 65 ug/m3 daily	15 ug/m3 annual and 50 ug/m3 daily	15 ug/m3 annual and 25 ug/m3 daily
Mortality (all ages)	(A) Associated with short-term exposure	370 (220 - 510)	310 (190 - 430)	310 (190 - 430)	250 (150 - 340)	110 (70 - 160)
	Percent Reduction in PM-Associated Incidence:* **		16.2%	16.2%	32.4%	70.3%
	Percent Reduction in Total Incidence:**		0.3%	0.3%	0.6%	1.3%
Mortality (all ages)	(B) Associated with long-term exposure	920 (580 - 1260)	660 (390 - 850)	660 (390 - 850)	390 (230 - 490)	0 (0 - 0)
	Percent Reduction in PM-Associated Incidence:		28.3%	28.3%	57.6%	100.0%
	Percent Reduction in Total Incidence:		1.3%	1.3%	2.6%	4.6%
Hospital Admissions Respiratory	(C) Total Respiratory (all ages)	260 (70 - 450)	220 (60 - 380)	220 (60 - 380)	160 (50 - 300)	80 (20 - 140)
	Percent Reduction in PM-Associated Incidence:		15.4%	15.4%	30.8%	69.2%
	Percent Reduction in Total Incidence:		0.3%	0.3%	0.6%	1.4%
Hospital Admissions Cardiac	(D) Ischemic Heart Disease***** (>64 years old)	70 (30 - 120)	60 (30 - 100)	60 (30 - 100)	50 (20 - 80)	20 (10 - 40)
	(E) Congestive Heart Failure***** (>64 years old)	100 (50 - 150)	80 (40 - 130)	80 (40 - 130)	70 (30 - 100)	30 (20 - 40)
	Range of Percent Reductions in PM-Associated Incidence:		14.3% - 20.0%	14.3% - 20.0%	28.6% - 30.0%	70.0% - 71.4%
	Range of Percent Reductions in Total Incidence:		0.1% - 0.3%	0.1% - 0.3%	0.2% - 0.4%	0.5% - 0.9%
(F) Lower Respiratory Symptoms (8-12 yr olds) ******		< 11000 > (6000 - 15000)	< 9000 > (5000 - 12000)	< 9000 > (5000 - 12000)	< 7000 > (4000 - 9000)	< 3000 > (2000 - 4000)
	Percent Reduction in PM-Associated Incidence:		18.2%	18.2%	36.4%	72.7%
	Percent Reduction in Total Incidence:		3.6%	3.6%	7.3%	14.6%

* Health effects are associated with short-term exposure to PM, unless otherwise specified

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background PM-2.5 level. Background PM-2.5 is assumed to be 3.5 ug/m3 in Philadelphia County

*** The percent reduction in PM-associated incidence achieved by attaining alternative standards as opposed to the current standards is the reduction in incidence divided by the incidence associated with current standards. For example, the percent reduction in PM-associated incidence of mortality associated with short-term exposure to PM-2.5 achieved by meeting both a 15 ug/m3 annual and a 65 ug/m3 daily standard is (370 - 310)/370 = 16.2%

**** The percent reduction in total incidence achieved by attaining current or alternative standards is the reduction in incidence achieved by attaining the standard divided by the total (not only PM-associated) incidence

***** PM-2.5 results based on using PM-2.5 mass as PM-10 mass in the PM-10 functions

******Angle brackets <> indicate incidence calculated using baseline incidence rates reported in studies, with no adjustment for location-specific incidence rates. This increases the uncertainty in the incidence estimates

Sources of Concentration-Response (C-R) Functions

(A) C-R function based on pooled results from studies in six locations.

(B) Pope et al., 1995

(C) Thurston, et al., 1994

(D) Schwartz & Morris, 1995

(E) Schwartz & Morris, 1995

(F) Schwartz, et al., 1994

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All the numbers in parentheses are interpreted as 90% credible intervals based on Monte Carlo analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter VI for details.

Table VI-13a. Estimated Changes in Health Risks Associated with Meeting Alternative PM-2.5 Standards in Southeast Los Angeles County, 1995* (for base case assumptions)

Health Effects		PM-2.5-Related Incidence associated with current standards**	Incidence Associated with Meeting Alternative Standards			
			20 ug/m3 annual	*20 ug/m3 annual and 65 ug/m3 daily*	*20 ug/m3 annual and 50 ug/m3 daily*	*20 ug/m3 annual and 25 ug/m3 daily*
Mortality (all ages)	(A) Associated with short-term exposure	710 (430 - 970)	560 (350 - 780)	430 (270 - 600)	310 (210 - 460)	120 (100 - 220)
	Percent Reduction in PM-Associated Incidence:* **		21.1%	39.4%	56.3%	83.1%
	Percent Reduction in Total Incidence:***		0.6%	1.1%	1.6%	2.4%
	(B) Associated with long-term exposure	2110 (1330 - 2860)	1540 (980 - 2080)	940 (600 - 1260)	480 (310 - 640)	0 (0 - 0)
	Percent Reduction in PM-Associated Incidence:		27.0%	55.5%	77.3%	100.0%
	Percent Reduction in Total Incidence:		2.3%	4.8%	6.6%	8.6%
Hospital Admissions Respiratory	(C) Total Respiratory (all ages)	940 (250 - 1630)	750 (200 - 1320)	570 (160 - 1030)	410 (120 - 780)	160 (50 - 370)
	Percent Reduction in PM-Associated Incidence:		20.2%	39.4%	56.4%	83.0%
	Percent Reduction in Total Incidence:		1.2%	2.4%	3.4%	5.0%
Hospital Admissions Cardiac	(D) Ischemic Heart Disease ***** (>64 years old)	130 (50 - 200)	100 (40 - 160)	80 (30 - 120)	60 (20 - 90)	20 (10 - 40)
	(E) Congestive Heart Failure ***** (>64 years old)	140 (70 - 210)	110 (60 - 170)	80 (40 - 130)	60 (30 - 100)	20 (20 - 40)
	Range of Percent Reductions in PM-Associated Incidence:		21.4% - 23.1%	38.5% - 42.9%	53.8% - 57.1%	84.6% - 85.7%
	Range of Percent Reductions in Total Incidence:		0.3% - 0.4%	0.4% - 0.8%	0.6% - 1.1%	1.0% - 1.7%
(F) Lower Respiratory Symptoms (8-12 yr olds)******		< 43000 > (23000 - 58000)	< 32000 > (18000 - 43000)	< 23000 > (14000 - 31000)	< 16000 > (10000 - 22000)	< 6000 > (5000 - 9000)
	Percent Reduction in PM-Associated Incidence:		25.6%	46.5%	62.8%	86.0%
	Percent Reduction in Total Incidence:		7.3%	13.3%	18.0%	24.7%

Health effects are associated with short-term exposure to PM, unless otherwise specified

* Los Angeles County was not in attainment of current PM-10 standards in 1995. Figures shown assume actual PM-10 concentrations are first rolled back to simulate attainment of these standards, and that actual PM-2.5 concentrations are rolled back by the same percent as PM-10. See text in Chapter VI for details.

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background PM-2.5 level. Background PM-2.5 is assumed to be 2.5 ug/m3 in Southeast Los Angeles County.

*** The percent reduction in PM-associated incidence achieved by attaining alternative standards as opposed to the current standards is the reduction in incidence divided by the incidence associated with current standards. For example, the percent reduction in PM-associated incidence of mortality associated with short-term exposure to PM-2.5 achieved by *meeting both a 20 ug/m3 annual and a 65 ug/m3* daily standard is (710 - 420)/710 = 40.8%.

**** The percent reduction in total incidence achieved by attaining current or alternative standards is the reduction in incidence achieved by attaining *the standard divided by the total (not only PM-associated) incidence*.

***** PM-2.5 results based on using PM-2.5 mass as PM-10 mass in the PM-10 functions.

******Angle brackets <> indicate incidence calculated using baseline incidence rates reported in studies, with no adjustment for location-specific *incidence rates. This increases the uncertainty in the incidence estimates.*

Sources of Concentration-Response (C-R) Functions:
(A) C-R function based on pooled results from studies in 6 locations
(B) Pope et al., 1995
(C) Thurston, et al., 1994
(D) Schwartz & Morris, 1995
(E) Schwartz & Morris, 1995
(F) Schwartz, et al., 1994

The numbers in parentheses for pooled studies are NOT standard confidence intervals. All the numbers in parentheses are interpreted as 90% credible intervals based on Monte Carlo analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter VI for details.

Table VI-13b. Estimated Changes in Health Risks Associated with Meeting Alternative PM-2.5 Standards in Southeast Los Angeles County, 1995* (for base case assumptions)

Health Effects		PM-2.5-Related Incidence associated with current standards**	Incidence Associated with Meeting Alternative Standards			
			15 ug/m3 annual	15 ug/m3 annual and 65 ug/m3 daily	15 ug/m3 annual and 50 ug/m3 daily	15 ug/m3 annual and 25 ug/m3 daily
Mortality (all ages)	(A) Associated with short-term exposure	710 (430 - 970)	390 (250 - 560)	390 (250 - 560)	310 (210 - 460)	120 (100 - 220)
	Percent Reduction in PM-Associated Incidence:* **		45.1%	45.1%	56.3%	83.1%
	Percent Reduction in Total Incidence:** **		1.3%	1.3%	1.6%	2.4%
	(B) Associated with long-term exposure	2110 (1330 - 2860)	810 (520 - 1090)	810 (520 - 1090)	480 (310 - 640)	0 (0 - 0)
	Percent Reduction in PM-Associated Incidence:		61.6%	61.6%	77.3%	100.0%
	Percent Reduction in Total Incidence:		5.3%	5.3%	6.6%	8.6%
Hospital Admissions Respiratory	(C) Total Respiratory (all ages)	940 (250 - 1630)	520 (140 - 950)	520 (140 - 950)	410 (120 - 780)	160 (50 - 370)
	Percent Reduction in PM-Associated Incidence:		44.7%	44.7%	56.4%	83.0%
	Percent Reduction in Total Incidence:		2.7%	2.7%	3.4%	5.0%
Hospital Admissions Cardiac	(D) Ischemic Heart Disease ***** (>64 years old)	130 (50 - 200)	70 (30 - 110)	70 (30 - 110)	60 (20 - 90)	20 (10 - 40)
	(E) Congestive Heart Failure ***** (>64 years old)	140 (70 - 210)	80 (40 - 120)	80 (40 - 120)	60 (30 - 100)	20 (20 - 40)
	Range of Percent Reductions in PM-Associated Incidence:		42.9% - 46.2%	42.9% - 46.2%	53.8% - 57.1%	84.6% - 85.7%
	Range of Percent Reductions in Total Incidence:		0.5% - 0.8%	0.5% - 0.8%	0.6% - 1.1%	1.0% - 1.7%
(F) Lower Respiratory Symptoms (8-12 yr. olds)******		< 43000 > (23000 - 58000)	< 21000 > (13000 - 28000)	< 21000 > (13000 - 28000)	< 16000 > (10000 - 22000)	< 6000 > (5000 - 9000)
	Percent Reduction in PM-Associated Incidence:		51.2%	51.2%	62.8%	86.0%
	Percent Reduction in Total Incidence:		14.7%	14.7%	18.0%	24.7%

Health effects are associated with short-term exposure to PM, unless otherwise specified

* Los Angeles County was not in attainment of current PM-10 standards in 1995. Figures shown assume actual PM-10 concentrations are first rolled back to simulate attainment of these standards, and that actual PM-2.5 concentrations are rolled back by the same percent as PM-10. See text in Chapter VI for details.

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background PM-2.5 level. Background PM-2.5 is assumed to be 2.5 ug/m3 in Southeast Los Angeles County.

*** The percent reduction in PM-associated incidence achieved by attaining alternative standards as opposed to the current standards is the reduction in incidence divided by the incidence associated with current standards. For example, the percent reduction in PM-associated incidence of mortality associated with short-term exposure to PM-2.5 achieved by meeting both a 15 ug/m3 annual and a 65 ug/m3 daily standard is (710-390)/710 = 45.1%.

**** The percent reduction in total incidence achieved by attaining current or alternative standards is the reduction in incidence achieved by attaining the standard divided by the total (not only PM-associated) incidence.

***** PM-2.5 results based on using PM-2.5 mass as PM-10 mass in the PM-10 functions.

****** Angle brackets <> indicate incidence calculated using baseline incidence rates reported in studies, with no adjustment for location-specific incidence rates. This increases the uncertainty in the incidence estimates.

Sources of Concentration-Response (C-R) Functions

(A) C-R function based on pooled results from studies in 6 locations

(B) Pope et al., 1995

(C) Thurston, et al., 1994

(D) Schwartz & Morris, 1995

(E) Schwartz & Morris, 1995

(F) Schwartz, et al., 1994

The numbers in parentheses for pooled studies are NOT standard confidence intervals. All the numbers in parentheses are interpreted as 90% credible intervals based on Monte Carlo analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter VI for details.

Table VI-14. Sensitivity Analysis: Effect of Alternative Rollback Methods on Mortality Estimates Short-term Exposure (Pooled Function) and Long-term Exposure PM-2.5 Mortality Functions Philadelphia County, September 1992 - August 1993

Initial Air Quality: 16.3 ug/m3 annual average, 69.3 ug/m3 2nd daily maximum

		Percent Change in PM-Associated Incidence		Portion of Proportional Rollback Incidence Reduction Achieved by Alternative Rollback
	Alternative Standard	All PM concentrations rolled back equally	Higher PM concentrations reduced more	
(A) Mortality associated with short-term exposure	15 ug/m3 annual	10.6%	9.2%	86.4%
	50 ug/m3 daily	29.7%	18.6%	62.6%
(B) Mortality associated with long-term exposure	15 ug/m3 annual	19.4%	19.4%	100.0%
	50 ug/m3 daily	54.1%	39.3%	72.6%

* Health effects incidence was quantified across the range of PM concentrations observed in each study, but not below background PM-2.5 level, which is assumed to be 3.5 ug/m3.

(A) C-R function based on studies in 6 cities
(B) Pope et al., 1995

County, alternative $PM_{2.5}$ standards have been modeled based on the amount of air quality reduction required to meet the numerical value of the controlling standard. Rounding conventions to be applied to any $PM_{2.5}$ standards have not been determined yet, and so the effect of rounding conventions has not been incorporated into this analysis of alternative standards. Several points from these Tables are of particular interest:

- Daily standards control the air quality reduction, and thus the estimated health risk reductions observed, for almost all of the alternative standards scenarios (Table VI-11b). In Philadelphia, which has an "as-is" annual mean concentration close to 15 $\mu g/m^3$, an annual standard of 20 $\mu g/m^3$ has no effect on reducing estimated incidence of health effects (Table VI-12a). Attaining an annual standard of 15 $\mu g/m^3$ without a daily standard is estimated to result in reductions in air quality concentrations and health risks (about 14-20% reduction for effects associated with short-term exposures and about 28% reduction for mortality associated with long-term exposure). However, the estimated reductions in health risks associated with attaining the 50 $\mu g/m^3$ 24-hr standard are significantly higher (e.g., about 29-36% reduction in mortality and other health effects associated with short-term exposures and about 58% reduction in mortality associated with long-term exposure upon attaining a 50 $\mu g/m^3$ 24-hr standard). Attaining a 25 $\mu g/m^3$ 24-hr standard in Philadelphia County is estimated to result in the largest risk reductions (e.g., about 69-73% reduction in mortality and other health effects associated with short-term exposures and 100% reduction in mortality associated with long-term exposures to PM).
- In Los Angeles County, an annual standard of 20 $\mu g/m^3$ is estimated to reduce air quality concentrations about 19%, with all three of the 24-hr alternative standards (65 $\mu g/m^3$, 50 $\mu g/m^3$, and 25 $\mu g/m^3$) requiring considerably greater reductions. A 15 $\mu g/m^3$ annual standard controls the amount of air quality reduction and estimated health risk reduced for the case involving a 65$\mu g/m^3$ alternative 24-hr standard, but not for cases involving a 50 $\mu g/m^3$ or 25 $\mu g/m^3$ alternative 24-hr standard. An annual standard of 15 $\mu g/m^3$ alone reduces estimated health risks associated with PM about 43-51% for

mortality and other health effects associated with short-term exposure and about 62% for mortality associated with long-term exposure relative to just attaining the current PM_{10} standards in Los Angeles County. Attaining a 50 $\mu g/m^3$ 24-hr standard reduces estimated health risks associated with PM about 54-63% for mortality and other health effects associated with short-term exposure and about 77% for mortality associated with long-term exposure. Attaining a 25 $\mu g/m^3$ 24-hr standard is estimated to further reduce health risks relative to the current PM_{10} standards, with about a 83-86% reduction in mortality and other health effects associated with short-term exposure and a 100% reduction in mortality associated with long-term exposure. As expected, the estimated health risk reductions are larger for Los Angeles County than Philadelphia County due to the higher PM air quality levels associated with meeting the current PM_{10} standards (i.e., baseline air quality in Philadelphia is below the level required to meet the current standards).

- The proportion of estimated risk associated with reductions in $PM_{2.5}$ under alternative standard scenarios can be considered either as a percentage in the PM-associated incidence reduced or as a percentage of total incidence of that health endpoint due to PM and all other causes. As an example, standards of 15 $\mu g/m^3$ and 50 $\mu g/m^3$ 24-hr in Philadelphia County lead to an estimated 32% reduction in mortality associated with short-term exposures to PM and a 29-36% reduction in morbidity (hospital admissions and respiratory symptoms) associated with short-term exposures to PM. These changes result in reductions in the overall incidence rates of these endpoints that are considerably smaller. For example, a 32% reduction in mortality associated with short-term PM exposures leads to an estimated 0.6% reduction in the total mortality incidence.
- Estimates of the reduction in total annual incidence of mortality upon attainment of alternative standards are more uncertain than estimates of the reduction in total annual incidence of other health effects, as a consequence of uncertainties in the extent of mortality displacement (shortening of life) that may be associated with PM (see Section V C.1.c; CD, pp. 13-44-45). These uncertainties concerning the degree of mortality

displacement are not as salient for estimates of reductions in annual mortality incidence associated with long-term PM exposures compared to short-term PM exposures, since the type of study design that produced the long-term exposure concentration-response functions provides findings that indicate effects on annual mortality rates (Utell and Frampton, 1995). However, depending on assumptions concerning the biological lags and cumulative effects of air pollution involved in these long-term exposure studies, uncertainty is involved concerning how long an area would need to be in attainment of an alternative standard in order for the full measure of estimated mortality rate reduction to be realized.

- Greater percent reduction of PM-associated risks is estimated for mortality associated with long-term exposures to PM than from short-term exposures. This is the consequence of quantifying increases in mortality associated with long-term exposures only at concentrations considerably above background ($PM_{2.5}$ concentrations > 9 $\mu g/m^3$ based on Pope et al. (1995)).

b. Individual Sensitivity Analysis Concerning Air Quality Rollbacks

The estimates of risk reductions in Tables VI-12 and VI-13 particularly depend on what inherently must be assumptions about the pattern of air quality reductions that will be observed in the future in attaining the alternative standard cases. While the base model used assumes a proportional reduction would be observed in all $PM_{2.5}$ concentrations above background as a consequence of control strategies intended to meet a controlling annual mean or 24-hr standard, it is quite possible that substantial differences in $PM_{2.5}$ air quality reductions could occur across the $PM_{2.5}$ distribution.[10] An attempt to bound the potential effects of these possible alternative rollbacks has been examined in a sensitivity analysis of PM-associated

[10]Information on past reductions of $PM_{2.5}$ concentrations as a direct result of NAAQS is not available, given that prior and current ambient standards for particles regulated larger particle indicators (TSP, PM_{10}). Existing monitoring information can be examined instead, although it is uncertain how much of the variation observed will reflect actual control strategies versus more general year-to-year variability. In a preliminary examination of changes in the distribution of $PM_{2.5}$ concentrations from sites with multiple years of data (from AIRS and CARB data sets), Abt Associates found that while a proportional rollback was a reasonable approximation of the central tendency of variation observed, considerable variation in this relationship was observed (see Abt Associates, 1996b for more information).

mortality risks by choosing alternative assumptions for modeling $PM_{2.5}$ rollbacks. The results of this sensitivity analysis are presented in Table VI-14. The alternative reduction approach provided for illustration decreases the upper 10% of $PM_{2.5}$ 24-hr air quality concentrations by a larger amount (a ratio of 1.6) than the reductions in the remaining 90% of the distribution of PM air quality concentrations and is intended to model a control strategy that preferentially targets peak PM levels.

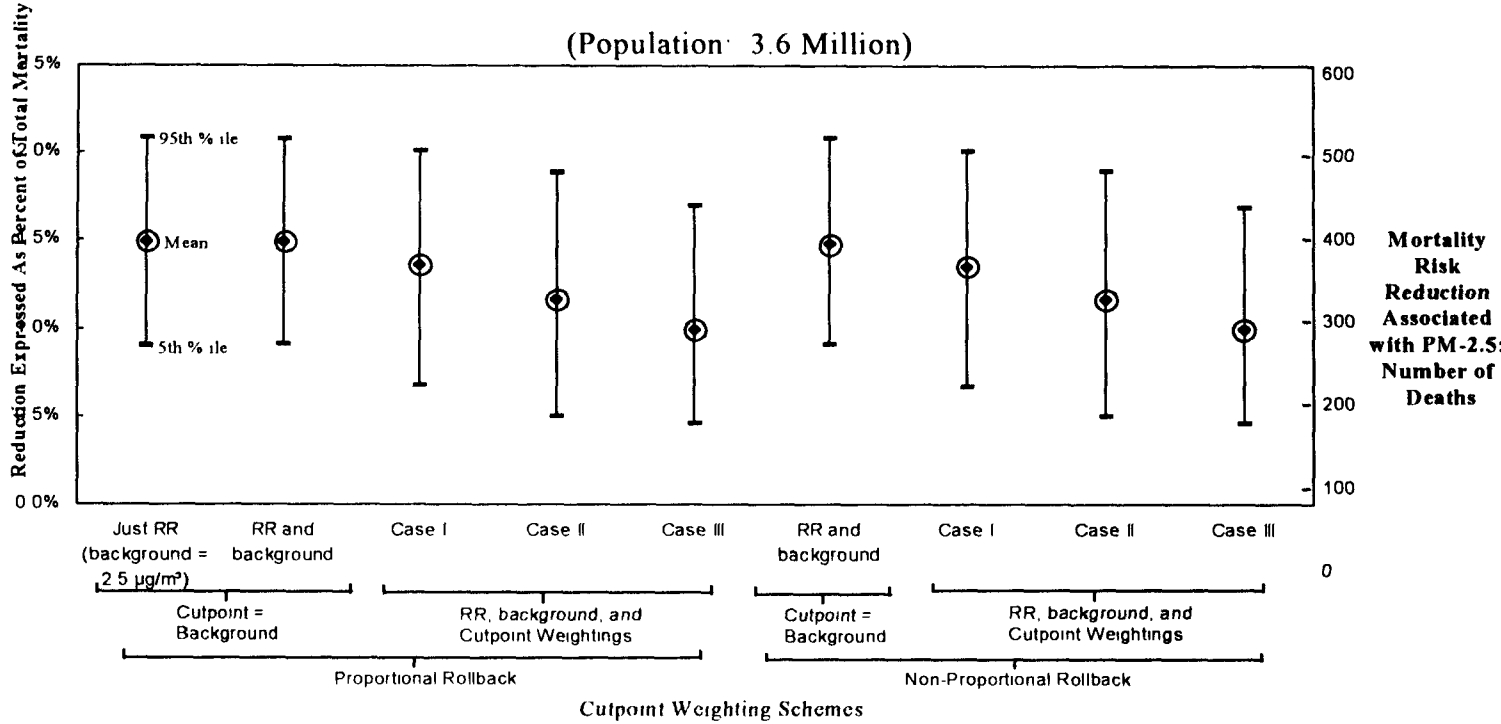
The results of the sensitivity analysis in Table VI-14 indicate that estimated mortality risks reduced by annual $PM_{2.5}$ standards are largely insensitive to the pattern of rollbacks in $PM_{2.5}$ concentrations, whereas estimates of risk associated with alternative 24-hr $PM_{2.5}$ standards are somewhat more sensitive to the choice of rollback methodology.

c. Integrated Uncertainty Analysis

Using the same approach described previously in Section VI.C.3.b, an illustrative integrated uncertainty analysis was prepared for estimating the reduction in mortality risk associated with short-term exposures upon attainment of example alternative $PM_{2.5}$ standards in Los Angeles County. These risk reductions were calculated relative to the scenario where Los Angeles County just attains the current PM_{10} standards. Figure VI-9 displays the results of the integrated uncertainty analysis for attaining example $PM_{2.5}$ standards of 15 $\mu g/m^3$, annual average and 50 $\mu g/m^3$, 24-hour average in Los Angeles County. Several sources of uncertainty were progressively included from left to right in the figure. The first vertical line reflects only uncertainty in the RR. The second vertical line includes uncertainty in RR and estimated background concentration, but no cutpoints are included. The next three vertical lines incorporate uncertainty about cutpoints, using the same three cutpoint weighting schemes discussed previously in Section VI.C.3.b and employs a proportional rollback method to simulate attainment of the $PM_{2.5}$ standards. The last three vertical lines also incorporate uncertainty about cutpoints, but use a non-proportional rollback approach to simulate attainment of the $PM_{2.5}$ standards.

As was observed in the earlier integrated uncertainty analysis, uncertainty about cutpoints has the largest impact on the estimated risk reduction associated with alternative standards. In contrast, the use of a proportional or non-proportional rollback method appears

Figure VI-9. Effect of Several Uncertainties on Reductions in Mortality Risk Associated With Short-Term Exposure to PM-2.5 Upon Attaining PM-2.5 Standards of 15 μg/m³ Annual and 50 μg/m³ Daily in Los Angeles County



Cutpoint Weighting Schemes

	Case I	Case II	Case III
Background	0 5	0 2	0.05
10 μg/m³	0.3	0.3	0.15
18 μg/m³	0 15	0 3	0.5
30 μg/m³	0.05	0.2	0 3

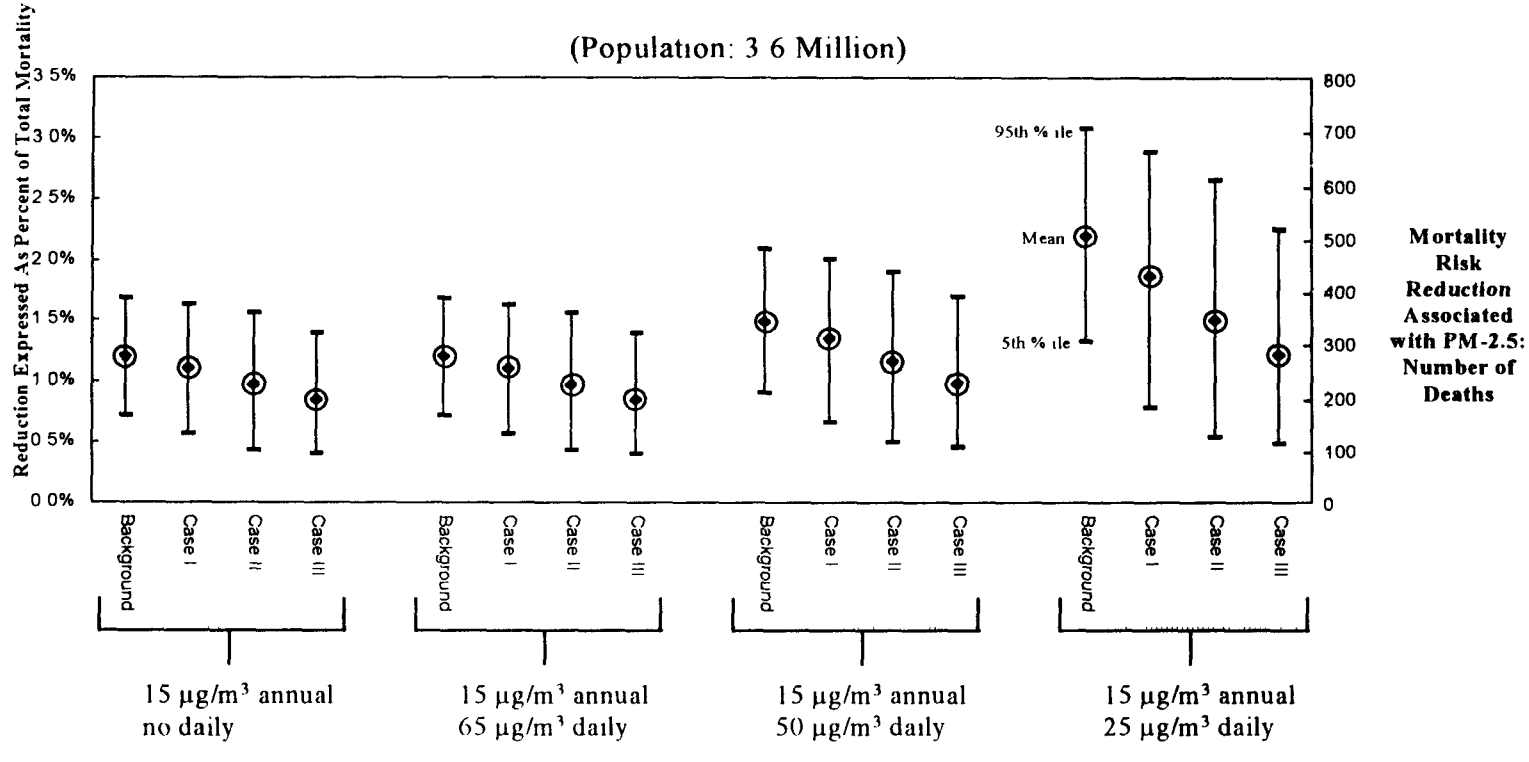
to have only a slight impact on the estimated risk reduction for mortality associated with short-term exposure to $PM_{2.5}$ when placed in the context of the other uncertainties that also affect our ability to predict risk reductions from alternative $PM_{2.5}$ standards.

In addition to the uncertainties inherent in estimating risks for the as is scenarios, such as the relative risk, background, and cutpoint uncertainties assessed in the integrated uncertainty analyses, estimates of reductions in risk resulting from attainment of alternative $PM_{2.5}$ standards are subject to uncertainties related to the projection of air quality that would occur when alternative standards are attained. These uncertainties relate in part to the potential that $PM_{2.5}$ may be serving in varying degrees as an index for air pollution (either by indexing the effects of other gaseous copollutants in addition to $PM_{2.5}$, or by indexing relatively more harmful constituents within $PM_{2.5}$). Such uncertainties may serve to alter estimates of risk reduction associated with attainment of alternative $PM_{2.5}$ standards, and the anticipated effects of potential strategies used to reduce PM concentrations.

Figure VI-10 displays the results of the integrated uncertainty analysis for Los Angeles County associated with attainment of several alternative PM2.5 standards. Four sets of standards are included: an annual standard alone set at 15 $\mu g/m^3$, and three pairs of standards with an annual standard set at 15 $\mu g/m^3$ accompanied by a 24-hour standards set at 65, 50, or 25 $\mu g/m^3$. In this figure, each set of four vertical lines represents the estimated risk reduction where uncertainties about background, RR, and cutpoint, and form of rollback have been included. The first vertical line in each group, labeled "background", assumes a cutpoint set equal to background, while the next three lines represent the three different cutpoint weighting schemes described previously and listed in the table at the bottom of the figure.

The estimated risk reduction associated with the 15 $\mu g/m^3$ annual standard alone is the same as that associated with this annual standard coupled with a 65 $\mu g/m^3$ daily standard, because the annual standard is the controlling standard. The greatest risk reduction is associated with the 15 $\mu g/m^3$ annual, 25 $\mu g/m^3$ daily standards pair. For this standard combination, the estimated mean risk reduction is about 2.2% (CrI 1.3-3.0) of total mortality or about 500 (CrI 300-700) excess deaths avoided when the cutpoint is set equal to the estimated background concentration level. Under the alternative cutpoint weighting schemes,

Figure VI-10. Effect of Several Uncertainties on Reductions in Mortality Risk Associated With Short-Term Exposure to PM-2.5 Upon Meeting Alternative PM-2.5 Standards in Los Angeles County



Cutpoint Weighting Schemes

	Case I	Case II	Case III
Background	0 5	0 2	0.05
10 µg/m³	0 3	0.3	0.15
18 µg/m³	0.15	0 3	0.5
30 µg/m³	0 05	0 2	0.3

the estimated mean risk reduction for this same suite of standards is reduced to about 1.2 to 1.8% of total mortality (or about 290-430 excess deaths avoided) depending on the weighting scheme used. As discussed previously, the percent reduction in total mortality can be expressed as either a percentage of total mortality due to all causes as shown on Figures VI-9 and VI-10 or as a percent reduction in the PM-associated mortality. For example, a reduction of 1.5% in total mortality (or 400 deaths) corresponds to a 56% reduction in PM-associated excess mortality and a 1.0% decrease in total mortality (or 300 deaths) corresponds to a 42% reduction in PM-associated mortality.

5. Key Observations from the Risk Analyses

This Chapter has presented a summary of a PM health risk assessment that quantifies health risks associated with 1) existing air quality levels, 2) projected air quality levels that would occur upon attainment of the current PM_{10} standards, and 3) projected air quality that would occur upon attainment of several alternative $PM_{2.5}$ standards in two urban areas. Summarized below are key observations resulting from the risk analyses, as well as several important caveats and limitations:

1) Fairly wide ranges of risk estimates result for mortality and morbidity health effects in the two locations analyzed when the effects of key uncertainties and alternative assumptions are considered

2) In the staff's judgment, estimates of mortality and morbidity risks remain significant from a public health perspective when the current PM_{10} standards are attained.

These points are illustrated below for mortality risks using base case and alternative assumptions as well as for morbidity risks using base case assumptions. For example, risk of mortality from short-term $PM_{2.5}$ exposures upon attainment of the current standards was estimated to range from approximately 400 to 1,000 deaths a year in Los Angeles County (population = 3 6 million) under base case assumptions, and from approximately 100 to 1,000 deaths across alternative assumptions considered in the integrated uncertainty analysis For Philadelphia County (population = 1 6 million), a city with more moderate air quality already well

below the current standards, mortality risk associated with short-term $PM_{2.5}$ exposures ranged from approximately 200 to 500 deaths under base case assumptions, and from approximately 20 to 500 deaths under alternative assumptions. In addition, risks of morbidity effects associated with exposures to $PM_{2.5}$ are estimated to center around approximately a thousand hospital admissions and many thousands of cases of respiratory symptoms in children per year for Los Angeles, with several hundred hospital admissions and thousands of cases of respiratory symptoms estimated for Philadelphia (mean estimates of base case assumptions).

3) Attainment of the range of alternative $PM_{2.5}$ standards considered was estimated to lead to essentially no changes in PM-associated risk to very substantial changes, depending on the city and the levels of the standards

Mortality and morbidity risks associated with short-term PM exposures in Los Angeles County are estimated to be reduced by roughly 20-25% upon attainment of an annual $PM_{2.5}$ standard of 20 $\mu g/m^3$ and 45-50% for an annual standard of 15 $\mu g/m^3$ beyond the risks associated with attainment of the current PM_{10} standards when base case assumptions are used. Under alternative assumptions, a greater proportion of PM-associated risk would be expected to be reduced (although reductions in the absolute incidence of health effects may be less). Daily standards ranging from 65 $\mu g/m^3$ to 25 $\mu g/m^3$ would reduce PM-associated risks from roughly 40% to 85% beyond those associated with attainment of the current PM_{10} standards when base case assumptions are used For an area already within attainment of the current standards (Philadelphia County), risk reductions are estimated upon attainment of an annual standard of 15 $\mu g/m^3$ (of roughly 15-20%) and attainment of 24-hr standards of 65 to 25 $\mu g/m^3$ (ranging from 10-70%, respectively), for base case assumptions.

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

Alternative cutpoint concentrations considered for these analyses could result in as much as a 3 to 4-fold difference in estimated risk associated with PM exposures in Los Angeles County

(Figure VI-8, see also Exhibits 7.19 and 7.20, Abt Associates, 1996b) depending on the degree of confidence one imputed to the likelihood that a $PM_{2.5}$ cutpoint concentration existed at the highest concentrations evaluated relative to the base case assumptions. In an area with PM concentrations well below the current PM standards (e.g., Philadelphia County), differences in "as is" risk for alternative cutpoint assumptions may be even greater, since these locations would be expected to have a greater proportion of air quality values below the cutpoint concentration.

5) Based on results from the sensitivity analysis of key uncertainties and/or the integrated uncertainty analyses, quantitative consideration of the following uncertainties have a much more modest impact on the risk estimates: inclusion of individual copollutant species when estimating PM effect sizes; the choice of approach to adjusting the slope in analyzing alternative cutpoints; the value chosen to represent average annual background PM concentrations; and the choice of rollback adjustment approaches for simulating attainment of alternative PM standards.

6) Risk analyses of alternative standard scenarios incorporate several additional sources of uncertainty, including: uncertainty in the pattern of air quality concentration reductions that would be observed across the distribution of PM concentrations in areas attaining the standards ("rollback uncertainty") and uncertainty concerning the degree to which current PM risk coefficients may reflect contributions from other pollutants, or the particular contribution of certain constituents of $PM_{2.5}$, and whether such constituents would be reduced in similar proportion to the reduction in $PM_{2.5}$ as a whole.

To the extent concentrations of other combustion source copollutants are reduced more or less than $PM_{2.5}$ concentrations in attaining alternative $PM_{2.5}$ standards, estimates of health risk reduced by alternative $PM_{2.5}$ standards would be expected to vary in proportion to the degree to which such copollutants have a genuine role in producing, or modifying the ability of PM to produce, some of the health effects associated with PM in current concentration-response relationships. Similarly, if specific constituents of $PM_{2.5}$ mass have differing potencies in producing health effects relative to other $PM_{2.5}$ constituents, estimates of risk reduced would be expected to vary if these constituent concentration are reduced to different degrees by control strategies designed to attain alternative $PM_{2.5}$ standards.

VII. STAFF CONCLUSIONS AND RECOMMENDATIONS ON PRIMARY NAAQS

This chapter presents staff conclusions and recommendations for the Administrator to consider in deciding whether to retain, revise, and/or supplement the current primary PM NAAQS. Drawing from the synthesis of information and analyses contained in both the Criteria Document (CD, Chapter 13) and in Chapters IV, V, and VI herein, this chapter begins with staff findings on the overall adequacy of the current primary standards for PM, going on to address each of the major components needed to specify ambient standards: pollutant indicator, averaging time, form, and level. Staff conclusions and recommendations on each of these interrelated components for the current and alternative primary standards are based on considering how both the components of an individual standard and a suite of standards operate together to protect public health with an adequate margin of safety.

In recommending a range of options for the Administrator to consider, the staff notes that the final decision is largely a public health policy judgment. A final decision must draw upon scientific information about health effects and risks, as well as judgments about how to deal with the range of uncertainties that are inherent in the scientific evidence and analyses. The staff's approach to informing these judgments is based on a recognition that the available health effects evidence generally reflects a continuum consisting of levels at which scientists generally agree that health effects are likely through lower levels at which the likelihood and magnitude of the response become increasingly uncertain. This approach is consistent with the requirements of the NAAQS provisions of the Clean Air Act and with how EPA and the courts have historically interpreted the Act. These provisions do not require the Administrator to establish a NAAQS at a zero-risk level but rather at a level that avoids unacceptable risks and, thus, protects public health with an adequate margin of safety.

In addition, the staff notes that especially where considerable uncertainty exists with regard to appropriate policy choices based on the scientific information and analyses, it is appropriate to consider the risk management implications of alternative approaches that represent scientifically sound options. For example, if the Administrator concludes that the current standards should be revised to provide greater health protection, it is appropriate to consider whether it would be more effective and efficient to do so by tightening the current PM_{10} standards

or by establishing new $PM_{2.5}$ standards. Thus, staff has considered risk management implications together with the scientific evidence in assessing whether alternative approaches to establishing PM standards would provide both the requisite level of protection and an effective and efficient basis for pollution control strategies that will result in the attainment and maintenance of adequate public health protection.

A. Adequacy of the Current Primary Standards for Particulate Matter

As discussed in Chapter II, the Clean Air Act calls for periodic review of the criteria and the NAAQS. The overarching issue in such reviews is whether revision of the existing standards is appropriate to reflect advances in scientific knowledge. The information presented in the Criteria Document and this Staff Paper is intended to provide a scientifically sound and policy-relevant basis, in accordance with sections 108 and 109 of the Clean Air Act, for the Administrator to reach conclusions with respect to whether the existing standards should be revised and, if so, what revised or new standards, are appropriate. The concluding section of the integrative summary of health effects information in the PM Criteria Document provides the following cogent summary of the science with respect to this issue for the current review of the PM standards:

> "The evidence for PM-related effects from epidemiologic studies is fairly strong, with most studies showing increases in mortality, hospital admissions, respiratory symptoms, and pulmonary function decrements associated with several PM indices. These epidemiologic findings cannot be wholly attributed to inappropriate or incorrect statistical methods, misspecification of concentration-effect models, biases in study design or implementation, measurement errors in health endpoint, pollution exposure, weather, or other variables, nor confounding of PM effects with effects of other factors. While the results of the epidemiology studies should be interpreted cautiously, they nonetheless provide ample reason to be concerned that there are detectable health effects attributable to PM at levels below the current NAAQS" (CD, p 13-92).

This finding from the review of the scientific criteria clearly calls into question the adequacy of the current NAAQS. The extensive PM epidemiologic database provides evidence of serious health effects (e.g., mortality, exacerbation of chronic disease, increased hospital admissions) in susceptible population groups (e.g., the elderly older adults with chronic cardiopulmonary disease). Although the increase in individual relative risk is small for the most

serious outcomes, it is likely significant from an overall public health perspective, because of the large number of individuals in susceptible population groups that are exposed to ambient PM (CD, p 1-21). While the lack of demonstrated mechanisms that explain the range of epidemiologic findings is an important caution which limits conclusions as to causality, qualitative information from laboratory studies of the effects of particle components at high concentrations and dosimetry considerations suggest that the kinds of effects observed in community studies (e.g., respiratory- and cardiovascular-related responses) are at least plausibly related to particulate matter. Indeed, the CD points to the consistency of the results of the epidemiologic studies from a large number of different locations and the coherent nature of the observed effects as being suggestive of a likely causal role of ambient PM in contributing to the reported effects. Given the evidence that such effects may occur at levels below the current standards, as well as the nature and potential magnitude of the public health risks involved, the staff believes that revision of the current standards is clearly appropriate. Thus, the principal recommendation of this staff assessment is that the current standards should be revised.

The remainder of this chapter focuses on developing a range of alternative standards for the Administrator to consider in determining what revised or new standards are appropriate to protect public health. In formulating alternative approaches to establishing adequately protective, effective, and efficient PM standards, staff concurs with the important conclusion from the CD that fine and coarse fractions of PM_{10} should be considered as two separate pollutants (CD, p 13-93). As discussed in Section V.F., the staff assessment finds sufficient evidence to support establishment of separate standards relating to these two fractions of PM_{10}. On the other hand, the staff also notes the larger body of epidemiologic evidence and air quality information related to undifferentiated PM_{10}.

Therefore, staff concludes that it is reasonable to consider two alternative approaches for revising the standards: 1) adopt more protective standards using PM_{10} as the sole indicator combining fine and coarse fractions; and 2) develop separate standards for fine and coarse fractions of PM_{10} using appropriate indicators for each fraction. Conceptually, the first approach is precautionary and gives significant weight to recent findings using PM_{10} as a surrogate for both fine and coarse fraction particles, with less consideration of the evidence that suggests that the

current standards provide adequate protection for coarse fraction particles. Because the PM_{10} monitoring network is in place, it also would result in more immediate implementation of revised standards. The second approach is based on the view that in the long run, more effective and efficient protection can be provided by separately targeting appropriate levels of controls to fine and coarse particles. Because of the need to develop and install additional monitors, this approach would provide additional time to consider significant new scientific information before any such standards were actually implemented.

The relative merit of these two alternative approaches are considered in the next section, which also summarize staff conclusions and recommendations regarding indicators for thoracic particles, fine particles, and coarse fraction particles. Subsequent sections focus on identifying alternative averaging times, forms, and levels for the recommended approach.

B. Alternative PM Indicators and Risk Management Implications

1. PM_{10} as Surrogate Indicator for Fine and Coarse Fraction Particles

The most recent summary of scientific information in the CD and outlined in Chapters IV and V continues to support past staff and CASAC recommendations regarding selecting size specific-indicators for PM standards. More specifically, the staff believes that the following conclusions reached in the 1987 assessment remain valid:

1) Health risks posed by inhaled particles are influenced both by the penetration and deposition of particles in the various regions of the respiratory tract and by the biological responses to these deposited materials.

2) The risks of adverse health effects associated with deposition of ambient fine and coarse fraction particles in the thorax (tracheobronchial and alveolar regions of the respiratory tract) are markedly greater than for deposition in the extrathoracic (head) region. Maximum particle penetration to the thoracic region occurs during oronasal or mouth breathing.

3) The risks of adverse health effects from extrathoracic deposition of general ambient PM are sufficiently low that particles which deposit only in that region can safely be excluded from the standard indicator.

4) The size specific indicator(s) should represent those particles capable of penetrating to the thoracic region, including both the tracheobronchial and alveolar regions.

Based upon the above considerations as well as the available information on human dosimetry of particles, in the previous review the staff and CASAC recommended a size specific indicator that included particles less than or equal to a nominal 10 μm cut point, termed PM_{10}. The recent information on human particle dosimetry contained in the CD provides no basis for changing 10 μm as the appropriate dividing line for particles capable of penetrating to the thoracic regions. The recent epidemiologic literature also provides some evidence that thoracic particles can be somewhat more closely linked to effects than can the "super coarse" (>10 μm) fraction of TSP (e.g. Dockery et al., 1993). The CD concludes that "recent analyses have substantiated the previous selection of PM_{10} as an indicator of particle-related health effects" (CD, p. 13-93).

In selecting the most appropriate indicator(s) for the PM standards, the staff believes that consideration should be given to protecting public health through the use of standards that are as effective and efficient as possible. An effective set of standards would capture all of the most harmful constituents of PM_{10} and target them such that an appropriate level of control occurs for the harmful components. Conceptually, a broad based PM indicator such as TSP set at a stringent enough level can provide effective protection for the most harmful components. However, because such a standard would set unnecessarily stringent controls on extrathoracic constituents unlikely to be most harmful, it would not be an efficient standard. As staff concluded in the previous review, a PM_{10} indicator provides more efficient as well as more effective health protection than would TSP (U.S. EPA, 1982b). In the present review, it is important to make use of the current state of knowledge to select an indicator(s) that not only captures all of the most harmful components (i.e., an effective indicator), but also places greater emphasis for control on those constituents or fractions that are most likely to result in the largest risk reduction (i.e., an efficient indicator).

Therefore, consideration of the available evidence regarding the components of PM_{10} most likely responsible for the observed health effects categories at various levels is critical to maximizing the effectiveness and efficiency of health protection strategies. The indicator is used

to target and monitor health protection strategies, and the choice is key to overall health protection provided by the PM NAAQS. Given these concerns and the expanded information, the staff believes it is appropriate to reexamine the question of whether the PM_{10} indicator should undergo additional refinement to reflect new scientific understandings of fine and coarse fraction particles as separate pollutants.

The staff assessment of the progress made through implementing the current PM_{10} standards is instructive in this regard (Section IV.D). Figure IV-4 and Table IV-5 summarize how the States and EPA characterize the major sources of PM_{10} and the extent of progress to date. In essence, the lessons learned from past TSP and PM_{10} programs can be summarized as follows:

- PM_{10} is generally viewed as a local rather than a regional problem. This is clearly appropriate in most Western areas with the highest PM_{10} levels. However, even in the eastern U.S., where high regional levels of transported fine particles make significant, but not dominant, contributions to PM_{10} mass, programs tend to focus on control of local sources, in part because of the difficulty in developing multi-jurisdictional strategies. This means that abatement programs will generally focus on the most readily available local sources of primary particles, leaving secondary or regional options as a last resort.

- In areas where local fine particle sources are overwhelmingly dominant, for example in areas with high woodsmoke contributions (e.g., Klamath Falls, OR), PM_{10} controls have led to significant reductions in fine particles. Historically, TSP-based local programs have also resulted in significant reductions in local primary fine particle emissions from coal combustion and industrial sources (e.g., New York City, Pittsburgh, PA).

- In areas where fugitive sources of crustal materials are clearly dominant (e.g., Coachcella Valley, CA), PM_{10} programs focus on measures that reduce road dust, construction, and related sources. These programs have had limited success to date. Local sources of precursor gases contributing to fine particles generally are not addressed.

- In areas dominated by local point source complexes (industrial emissions), both coarse and fine controls are applied, and sources sometimes may trade reductions between the two on a mass basis. Where source complexes are located in a zone of high transported fine particles, the transported component is treated as background, increasing the need for local controls; this likely results in greater relative control for coarse particles than fine.

- In Western areas having "mixed" contributions, including significant local secondary particle formation, three areas (SCAB[1]; Provo, UT; Denver, CO) have begun to require controls of gaseous precursors (SO_x, NO_x) in addition to fugitive dust and other controls.

- Any reductions in fine particles related to regional sulfur oxides emissions that have taken place to date are not related to implementation of the PM_{10} or TSP standards, but the SO_2 NAAQS and other mandated requirements of the CAA, such as the acid rain program.

This experience is a useful guide for a qualitative examination of the potential effectiveness and efficiency of alternative revised protective standards using PM_{10} as the sole surrogate for the harmful components of PM. To provide a basis for such examination, Table VII-1 presents a set of increasingly more protective alternative PM_{10} standards drawn from the staff analysis of potential PM_{10} effects "cutpoints" developed in Appendix E for the risk assessment. These alternatives do not reflect staff recommendations, but are examples presented for the purpose of the present assessment of the PM_{10} indicator. The table indicates the regional distribution of the percentage of counties (meeting a 50% data completeness criteria) that would not attain the listed alternatives. The table also notes the characteristic regional contribution of coarse fraction particles to PM_{10} mass, which, like total mass, is generally highest in the West.

Looking first at annual PM_{10} standards alone, the table suggests that a moderate reduction from the current level (to 40 $\mu g/m^3$) would result in few controls in eastern areas, but would approach the combined effect of the current 24-hour and annual standards in the West. A more substantial reduction in an annual standard to 30 $\mu g/m^3$ would affect about half of the Western areas and also begin to prompt additional controls in the East. By comparison, a revised 24-hour PM_{10} standard of 100 $\mu g/m^3$ (alone or in combination with a 40 $\mu g/m^3$ annual) would have effects similar to a 30 $\mu g/m^3$ annual standard alone in the East, but affect still more (approximately 55 to over 75%) Western areas. Based on the implementation experience outlined above, the eastern areas would likely develop control programs to achieve such standards with an initial focus on local sources of PM_{10}, which would tend to result in a proportionally greater reduction for coarse

[1] South Coast Air Basin of California.

TABLE VII-1. PERCENTAGE OF COUNTIES NOT MEETING ALTERNATIVE PM_{10} STANDARDS*

	Level of Alternative Standards**	All	SW	NW	CE	SE	NE
County Total		482	60	80	68	99	175
Annual	50	2.3	13.	3.8	0	0	0
	40	7.3	22	15	7.4	1.0	2.3
	30	29	45	48	26	16	23
24-hr	150	12	27	34	8.8	2.0	3.4
	100	35	55	76	32	25	16
	50	97	97	98	90	100	98
Combined Standards	50/150	12	26	34	8.8	2.0	3.4
	40/100	35	55	76	32	25	16
	30/50	97	97	98	90	100	98
Coarse/PM_{10}* **	--	0.44	0.55	0.60	0.37	0.44	0.37

* Based on 1991-1993 data, using 50% data completeness criteria and the Appendix K missing data adjustment to account for less than every day sampling frequencies. See staff analyses (Fitz-Simons et al., 1996).

** Based on current 1-expected-exceedance form of the 24-hour PM_{10} NAAQS and current expected annual average of annual PM_{10} NAAQS, at the highest monitor for each standard.

*** Regional median ratio of coarse fraction mass to PM_{10} mass all seasons, based on available data from few sites (SAI, 1996).

fraction particles than fine. Without a more detailed assessment beyond the scope of this paper, it is not clear whether or how much PM_{10} standards set at these levels would also prompt more balanced reductions in fine and coarse fraction particles in the East. In the West, however, widespread nonattainment resulting from such PM_{10} standards would clearly prompt much more coarse particle control, based on the prevailing high coarse fraction content of PM_{10}.

This analysis suggests that, nationwide, progressively reducing the level of the PM_{10} standards alone to the middle levels in the table would place relatively more emphasis on additional controls for coarse fraction particles than for fine. On a regional basis, relatively less impetus for additional control would be placed on the East, which has the highest regional concentrations of fine particles, than on the West, which has the highest localized concentrations of coarse fraction particles. Clearly, PM_{10} standard levels somewhere in the range below the middle levels shown in Table VII-1 would also result in relatively more control of fine particles in the East. Such standards would inevitably increase the number of areas needing to address coarse fraction particles in the West.

One view of the risk management implications of the recent epidemiology holds that a single PM_{10} indicator is most appropriate because more studies have used PM_{10} and it would therefore be more prudent to prompt proportional reductions in the major components of PM_{10}. Even accepting such a view, however, our analysis indicates that reduced PM_{10} standards would not result in proportional reductions in fine and coarse fraction particles in the very areas from which most of the epidemiological results are derived (see cover figure). Selecting levels that would achieve such proportional reductions in the East through a PM_{10} indicator alone would still result in significantly disproportional coarse particle control in the West[2]. In essence, the above analysis is consistent with the admonition in the CD that more effective PM_{10} programs can be achieved by establishing separate targets for fine and coarse fraction particles (CD, 13-94).

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[2]The acid rain program should result in some additional regional SO_x reductions in the East. However, much of the improvement has already been realized with more gradual reductions over the next 15 years due to the banking and trading components. The existence of such a program, however, provides no justification for establishing inappropriate PM NAAQS targets, nor for the potential over control of coarse fraction particles, particularly in the West.

From this analysis, then, a decision to provide increased health protection through standards indexed by undifferentiated PM_{10} alone would have to be based on two additional premises: 1) fine and coarse fraction particles are likely to produce similar health effects at equivalent concentrations, i.e. be of relatively comparable toxicity; and 2) control strategies for fine and coarse fraction particles would produce roughly equivalent reductions in exposure in sensitive populations shown to be at increased risk of PM effects. Yet, the staff analyses of the available information as summarized in Section V.F provides little support for either premise. While the relative toxicity of fine and coarse fraction particles is not clearly established, both physical and chemical toxicologic considerations suggest that fine particles are likely to be more toxic for several, although not necessarily all, of the relevant effects categories than are coarse fraction particles (Section V.F). Based on the direct comparisons in epidemiological studies and on exposure considerations, the staff further concludes that - - whatever the relative toxicity of fine and coarse fraction particles - - control of sources of ambient fine particles is likely to be more effective in reducing exposure to sensitive subpopulations than is control of sources of ambient coarse fraction particles.

Given the available evidence, a uniform reduction in the levels of the PM_{10} standards could provide effective health protection from the effects of the most harmful components of PM_{10}, but only at concentrations that appear to be unnecessarily stringent with respect to coarse fraction particles. Limited, but important epidemiological evidence as well as mechanistic considerations suggest that coarse fraction particles are linked to effects in areas that exceed the current PM_{10} standards (CD, p. 13-51). Given the lack of evidence with respect to coarse particle effects at concentrations at or below the level of the current PM_{10} standards, however, little justification exists for proportional, much less disproportional, reductions in coarse fraction particles beyond those afforded by the current standards. By contrast, a number of epidemiological studies have used fine particles as an indicator. The available evidence comparing the two fractions suggests that fine particles are a better surrogate for those components of PM_{10} that are associated with adverse effects at levels below the current standard (sectionV.F). For these reasons, staff concludes that a single PM_{10} indicator would not provide the most effective and efficient protection from the health effects of particulate matter. Instead, the data available in this review

suggest that the most effective and efficient approach would be to control PM_{10} through separate standards for fine and coarse fraction particles.

2. Alternative Surrogate Indicators for Fine and Coarse Fraction Particles

The large number of recent community epidemiologic studies and improvements in human exposure and air quality presented in the CD and outlined in Chapters IV and V above have greatly expanded the information regarding associations between contemporary community air pollution containing particles and morbidity and mortality in sensitive subpopulations as compared to the previous review. Even with the presence of other pollutants in the communities studied, PM is independently associated with the observed health effects. While earlier studies mainly relied on BS, TSP, and sulfates as particle indicators, the recent work has added a much larger body of quantitative and qualitative information on PM_{10}, with a lesser but still substantial number of community studies that provide specific information on fine particles, including sulfate and acid aerosol components, and to a still lesser extent, coarse fraction particles (CD, p 1-21).

The CD concludes that the indices most consistently associated with health endpoints are thoracic (PM_{10} or PM_{15}) and fine particle indicators. Less consistent relationships have been observed for TSP and the coarse fraction of PM_{10} (CD, p 1-21). Based on an examination of relevant information in the CD on fine and coarse fraction particles (Section V.F), the staff concludes that the weight of the available evidence allowing direct comparisons suggests that ambient coarse fraction particles are either less potent or a poorer surrogate for community effects of air pollution than are fine particles. This assessment finds that the limited evidence suggestive of independent coarse particle effects was found in areas that significantly exceed the current standards, while reported associations with fine particles frequently occur at levels well below the current standards.

The staff concurs with the CD recommendation that "it would be appropriate to consider fine and coarse mode particles as separate subclasses of pollutants" (CD, p 13-94). The staff also concludes that sufficient information exists to do so. The analysis in the preceding section indicates that establishing distinct targets for fine and coarse fraction particles would provide more effective and efficient health protection strategies for PM. Therefore, the staff recommends

that separate standards be established for the fine and coarse fractions of PM_{10}. The discussion below outlines staff conclusions and recommendations for selecting indicators for such standards.

a. Surrogate Indicators for the Fine Fraction of PM_{10}

Although fine mode particles consist of several distinct chemical classes (Table IV-2), they share a number of important characteristics related to size and formation mechanisms. The CD concludes that none of these subclasses can be specifically implicated as the sole or even primary cause of specific morbidity and mortality effects (CD, p. 13-93). In essence, fine particle mass is a surrogate for whatever components appear to be causing the mortality and morbidity effects in community air pollution.

In examining the potential effectiveness of fine particles as a surrogate, it is useful to consider the results of various analyses of air pollution and mortality in Philadelphia as discussed in Section V.E (Moolgavkar et al., 1995; Wyzga and Lipfert, 1995; Samet et al., 1995, 1996a; Cifuentes and Lave, 1996). The CD evaluation of these multiple investigations concludes that for this single city example, it appears most difficult to separate independent effects of PM (as TSP) and SO_2, concluding that the relationship between these pollutants and mortality may be inherently non-linear (CD, p 13-57). Several clearly hypothetical explanations have been advanced to explain these results. The following qualitative assessment of several speculative, but plausible hypotheses (in italics), outlines the potential implications of these alternatives for the effectiveness of fine particle control as a surrogate:

- *The complex relationship is a statistical artifact and only one of the pollutants is causally related. If the pollutant is PM,* then fine particle control would clearly be beneficial. *If the pollutant is SO_2,* which occurs at moderate levels in Philadelphia, reductions in local and transported SO_2 precursor control prompted by a fine particle standard would reduce health risk.[3]

- *The relationship is real and due to increased penetration of an SO_2 complex carried on carbonaceous or other non-acidic particles.* Then local controls of primary fine particle combustion sources would likely reduce risks, because reducing the aggregate particle surface area (by reducing fine mass) is more likely to reduce dose than SO_2 reductions.

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[3] As noted in section V.E, the evidence across multiple areas shows that PM is consistently associated with mortality in areas with high and low SO_2, making the second explanation unlikely.

- *The relationship is due to the association between SO_2 and acidic sulfates, which are the active agent.* In this case, fine particle controls are clearly beneficial.

- *The relationship is due to the combined interactions of SO_2 and particles in different regions of the respiratory tract.* Again, control of fine particles would be beneficial.

The staff does not have to accept any one of these hypothesized explanations as more likely to conclude that control of fine particles as a class appears to be a reasonable approach to reducing health risks in this particular example of potential confounding. It is also useful to note that, because of their relatively low surface area and origin, such a conclusion would not be as applicable to control of coarse fraction particles.

Although the above examples of alternative consequences of the use of fine particles as a surrogate are limited to PM and SO_2 interactions, some of these outcomes would extend to PM interactions with other pollutants as well. Given the large surface area of aqueous droplet and/or dry fine particles, as well as the multiplicity of similar effects caused by common gaseous pollutants such as ozone and related photochemical products and precursors, and NO_2 in addition to SO_2, direct or indirect interactions among these pollutants would not be unexpected (Section V.F.; CD, p 13-9.). Because ozone precursors, including NO_2 and volatile organic compounds, are also secondary particle precursors, it is reasonable to expect that the control of fine particles could also prompt control of local and regional sources of some of these precursors as well as SO_2. On the other hand, beyond the possibility of effects modifications in the body, the potential for gas/particle interactions between PM and CO is limited. It is also less clear that fine particle control would prompt significant additional CO control, the major contributors of which, mobile sources, are already subject to significant national reduction requirements. The rationale for concluding that the existence of PM effects is unlikely to be due to confounding by other pollutants is discussed in Section V.E.

The above examples also illustrate why, based on current information, it is more appropriate to control fine particles as a group, as opposed to singling out particular classes. The qualitative literature has found various effects of high concentrations of fine sulfuric acid, ammonium sulfates and nitrate, carbonaceous materials, and transition metals, alone or in some

cases, in combination with gases (CD, Chapter 11; Section V.C). Community studies have found significant associations between fine particles or PM_{10} and health in areas with significant mass contribution of these fine components, including sulfates (6 cities), wood smoke (Santa Clara), nitrates (Los Angeles and Utah Valley), secondary organics (Los Angeles), and acid sulfate aerosols (24 City Study). As noted above, it is not possible to rule out any one of these components as contributing to fine particle effects.

The most substantial laboratory and epidemiologic data for any single class of fine particles exists for sulfates and associated acids. The data for acids, which are more difficult to measure, is less consistent than for sulfates. For example, the recent 24 City Study data suggest that regionally high exposures to acids in modest sized communities in the "sulfate belt" are associated with bronchitis and decreased lung function in children (Dockery et al., 1996; Raizenne et al., 1996). Yet relatively strong correlations exist between acids, sulfates, and fine particles, making it difficult to single out any factor with confidence (CD, p 13-93). Indeed, the staff considers sulfates useful as an indicator of fine particles for assessing the health effects literature. This literature suggests that reductions of regional sulfates as part of a fine particle standard control program would likely reduce mortality and morbidity risks for the large segments of the sensitive population who reside in the East. It would be inappropriate, however, to extend this finding to establishing a separate sulfate standard, alone or in combination with fine particle standards. A sulfate standard, even if understood as an indicator of all fine particles as suggested by Lippmann and Thurston (1996), would be less likely to lead to controls of the other potentially harmful components of fine particles.

A number of monitoring approaches have been used as indicators for fine particles (Appendix B). All of them have inherent strengths and weakness (CD, pp. 1-6 to 7). In selecting an indicator for a fine particle NAAQS, the staff places great weight on providing consistency with the largest segment of the epidemiologic data, and to a lesser extent, on making use of the existing fine particle data in the U.S. Staff have submitted their recommendations regarding the most appropriate monitoring approach for a fine particle standard to the CASAC Technical

Subcommittee for Fine Particle Monitoring[4]. The staff rejected the use of filter based optical approaches because they are more sensitive to variations in carbon and require mass calibration (CD, p 1-6). Although direct optical (e.g., nephelometry) and other continuous methods can offer significant advantages and are often well correlated with gravimetric mass measurements, under some circumstances they are less well linked, in part because of losses of semi-volatile components (CD, p 1-6). Further development of such approaches for routine use is an important need. Because most of the quantitative epidemiological data for fine particles and PM_{10} were based on gravimetrically determined mass, staff recommends that this measurement principle be adopted for fine particle standards. Although some loss of nitrate and other semi-volatile mass can occur with such methods, gravimetric approaches are most directly related to the available epidemiology, and they can be used to provide composition information helpful for developing control strategies. Again, improved continuous approaches that could be used as equivalent methods for fine particles are an important development need.

Staff also recommend the use of a sharp 2.5 μm cutpoint for a fine particle indicator. As discussed in Chapter IV and Appendix A, the minimum particle diameter between the fine and coarse modes lies between 1 and 3 μm, and the scientific data support a cut point to delineate fine particles in this range. Because of the potential overlap of fine and coarse particle mass in this intermodal region, specific cut points are only an approximation of fine mode particles. Thus, the decision within this size range is largely a policy judgement. The staff recommendation for a 2.5 μm cutpoint is based on considerations of consistency with health data, the limited potential for intrusion of coarse fraction particles into the fine fraction, and availability of monitoring technology. Therefore, the staff recommends using $PM_{2.5}$ as the fine particle indicator. The definition will be further specified in the Federal Reference Method and equivalency program.

$PM_{2.5}$ encompasses all of the potential agents of concern in the fine fraction, including most sulfates, acids, fine particle metals, organics, and ultrafine particles and includes most of the aggregate surface area and particle number in the entire PM distribution. $PM_{2.5}$ has been used

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[4]The Subcommittee met to review these recommendations as well as specifications for a possible Federal Reference Method and Monitoring Guidance at a public meeting on March 1, 1996.

directly in health studies as described in the CD and Chapter V. Although a number of studies have used $PM_{2.1}$, in most locations there should be little difference in mass. The more widespread use of $PM_{2.5}$ measurement technologies since the 1970s has resulted in the generation of relatively more data for this cutpoint than for other cutpoints for fine fraction particles.

$PM_{2.5}$ does have some potential for intrusion of the "tail" of the coarse mode during episodes of fugitive dust concentrations (See Appendix A). Staff recommends a sharp inlet for the FRM to minimize this potential intrusion of coarse mode particles. Such intrusions into $PM_{2.5}$ measurements is not anticipated to be significant in most situations; nevertheless, if subsequent data reveal problems in this regard, this issue can and should be addressed on a case-by-case basis in the monitoring and implementation programs. Because the purpose of a $PM_{2.5}$ standard is to direct controls toward sources of fine mode particles, it would be appropriate to develop analytical procedures for identifying those cases where a $PM_{2.5}$ standard violation would not have occurred in the absence of coarse mode particle intrusion.[5] Consideration should be given to a policy similar to the natural events policy (See Chapter IV) for addressing such cases.

Some commentors have recommended use of a smaller cutpoint at 1 μm (PM_1) to further reduce coarse particle intrusion. PM_1 has not been used in health studies, although in most cases mass should be similar as for cutpoints of 2.1 or 2.5. While this indicator could reduce intrusion of fugitive dust, it might also omit portions of hygroscopic acid sulfates in high humidity episodes. PM_1 sampling technologies have been developed; however, the PM_1 samplers have not been widely field-tested to date. Of some concern is the theoretical possibility that different flow velocities for the smaller cut might increase the loss of semivolatile materials relative to a larger cut. Thus, the staff recommends the use of $PM_{2.5}$ as the fine particle cutpoint.

b. Surrogate Indicators for the Coarse Fraction of PM_{10}

The CD and staff assessment finds that epidemiologic information, dosimetry and toxicology support the need for a particle indicator that addresses the health effects of coarse fraction particles smaller than nominal 10 μm. Coarse fraction particles deposit in both the

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[5] Analytical procedures could involve measurements of chemical components related to local coarse mode particles as a basis for developing a coarse mode intrusion estimate. Lundgren et al. (1996) have submitted a paper suggesting one such approach

tracheobronchial and alveolar region. Although the role of coarse fraction particles in much of the recent epidemiological results is unclear, studies where coarse fraction particles are the dominant fraction of PM_{10} suggest that the major short-term effects include aggravation of asthma and increased upper respiratory illness. Such effects are supported by dosimetric considerations (CD, p 13-51). Children, who spend more time in outdoor activities, may encounter higher exposures and doses of coarse fraction particles than other potentially sensitive populations. Long-term deposition of insoluble coarse fraction particles in the alveolar region may have the potential for enhanced toxicity, in part because clearance from this region of the lung is significantly slower than from the tracheobronchial region. Qualitative support for this concern is found in autopsy studies of animals and humans exposed to various ambient crustal dusts at or slightly above ambient levels typical in the Western U.S. (Section V.C).

In selecting an indicator for coarse fraction particles, it is important to note that the existing ambient data base for coarse fraction particles ($PM_{10-2.5}$) is smaller than that for fine particles, and that the only studies of clear quantitative relevance have used undifferentiated PM_{10}. However, it is possible to consider PM_{10} itself as a useful surrogate for coarse fraction particles, when used in conjunction with $PM_{2.5}$ standards. As noted above, in many areas with high fugitive dust, this is already the case with respect to control strategies. Because coarse fraction particles in such areas contribute significantly more mass than smaller particles, risk managers have incentives to focus reduction measures on particle sources that contribute the most by mass. The monitoring network already in place for PM_{10} is large. Therefore, if a fine particle indicator were chosen, the staff would recommend retention of PM_{10} as the indicator to protect against the risks of coarse fraction particles.

3. Staff Conclusions and Recommendations for Particle Indicators

Based on the above assessments and the scientific information in the CD, the staff draws the following conclusions and recommendations:

1) Ambient particles capable of penetrating to the thoracic region represent the greatest risk to health. Previous staff and CASAC recommendations for 10 μm as the appropriate cut point for such particles remain valid. In examining alternative approaches to increasing the protection afforded by PM_{10} standards, the staff finds that reducing the levels of the

current standards would not provide the most effective and efficient protection from the health effects of particulate matter.

2) The recent health evidence, the fundamental differences between fine and coarse fraction particles, and implementation experience with PM_{10} have, however, prompted the staff to consider separate standards for the fine and coarse fractions of PM_{10}.

3) The staff finds that the available information is sufficient to support separate indicators for these pollutant classes. While it is difficult to distinguish the effects of fine or coarse fraction particles from those of PM_{10}, consideration of comparisons between fine and coarse fraction particles suggests that fine particles are a better surrogate for those particle components linked to mortality and morbidity effects at levels below the current standards. Coarse fraction particles are most clearly linked with certain effects at levels above those allowed by the current standards.

4) In selecting an indicator for fine particles, staff recommends use of a 2.5 μm cut point for fine particle mass. Adoption of sulfate or other chemical class indicators is not advisable during this review. In selecting an indicator for coarse fraction particles, the staff recommends use of PM_{10}.

C. Alternative $PM_{2.5}$ Standards for Control of Fine Fraction Particles

1. Averaging Time

The current primary PM NAAQS include both a 24-hour standard, with no more than one expected exceedance, and an annual standard with an expected arithmetic mean form. These standards were intended jointly to protect the public against the health effects associated with both short-term and long-term exposures to PM based on epidemiological and other health studies available at the conclusion of the last review. The recent health effects information includes reported associations with both short-term (from less than 1 day to up to 5 days) and long-term (from generally a year to several years) measures to PM. This information, summarized in Chapter V, provides increased support for consideration of both short-term and long-term standards, as discussed below.

a. Short-term $PM_{2.5}$ Standard

The current 24-hour averaging time is consistent with the majority of the results from community epidemiological studies, which have reported associations of 24-hour concentrations of PM_{10}, fine particles, and TSP with an array of health effects. Nevertheless, because some such studies have found a stronger association with a multiple day average (Pope et al., 1992; Ostro et al., 1995; Pope and Dockery et al., 1992), the staff considered whether a multiple day averaging time would be more appropriate. The above results are also consistent with the existence of a lagged single exposure effect of PM, which may not be due to multiple day exposures. Moreover, some studies have found health effects to be associated with same day or previous day PM concentrations. For example, such associations are shown by mortality studies in Los Angeles, CA; Birmingham, AL; St. Louis, MO; Toronto, Canada; Santiago, Chile; Athens, Greece; and London, England. Further, most hospital admissions studies show associations with same day concentrations. In any case, a 24-hour standard can effectively protect against episodes lasting for several days while also protecting sensitive individuals who may experience effects after a single day of exposure. Thus, the staff concludes that the complexity in adopting a multiple day averaging time, e.g. 3 to 5 days, would not provide more effective protection than a 24-hour average.

The staff has also considered the evidence regarding effects associated with PM exposures of durations less than 24 hours. Some investigators prior to the 1987 review (Lawther et al., 1970) speculated that the observed health effects might be largely due to short-term peaks on the order of an hour. Controlled human and animal exposures to specific components of fine particles, such as acid aerosols, also suggest that some effects, such as bronchoconstriction, can occur after exposures of minutes to hours. Some epidemiological studies of exposures to acid aerosols have also found changes in respiratory symptoms in children using averaging times less than a 24-hour period (e.g., 12 hours). However, the majority of effects have been associated with daily or longer exposure to PM. Moreover, limitations in current mass monitoring devices make shorter durations less practical at present. A 24-hour average can be expected to provide significant protection from potential effects associated with short duration peaks in most urban atmospheres. Thus, although some study results may be suggestive of short duration effects, the

staff does not believe that the reported results provide a satisfactory quantitative basis for setting a general particle standard with an averaging time of less than 24 hours. The staff believes that additional research is needed to examine short duration exposures.

The staff recommends that consideration be given to retaining the current 24-hour averaging time as a means of controlling short-term ambient $PM_{2.5}$ concentrations, especially peak concentrations, and thus providing protection from health effects associated with short-term (from less than 1-day to up to 5-day) exposures to $PM_{2.5}$.

b. Long-term $PM_{2.5}$ Standard

As summarized in Chapter V, community epidemiological studies have reported associations of annual concentrations of $PM_{2.5}$, sulfates, PM_{10}, and TSP with an array of health effects, notably increased mortality (Dockery et al., 1993, Pope et al., 1995), respiratory symptoms and illness (e.g., bronchitis and cough in children), and reduced lung function. The relative risks associated with such exposures, although highly uncertain, appear to be larger than those associated with short-term exposures. Based on the available epidemiology and consideration of relevant toxicologic and dosimetric information, staff concludes that significant, and potentially independent, health consequences are associated with long-term PM exposures (CD, p 13-34)[6].

The staff notes that some health endpoints may better reflect the cumulative effects of PM exposures over a number of years (CD, p. 1-13). In such cases, an expected annual average standard would provide effective protection against long-term exposures to PM that exceed several years. Requiring a much longer averaging time would complicate and unnecessarily delay control strategies and attainment decisions.

In addition, an annual standard would have the effect of controlling air quality across the entire yearly distribution of 24-hour $PM_{2.5}$ concentrations to varying degrees, although such a standard would not as effectively limit peak 24-hour concentrations as would a 24-hour standard.

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[6]The seasonality of wintertime smoke and summertime regional acid sulfate and ozone suggest that an intermediate averaging time might also be appropriate in future reviews. Annual effects associated with acids, such as those observed by Dockery et al (1996) and Raizenne et al (1996) might be interpreted as the result of repeated seasonally high exposures.

Thus, an annual standard could also provide protection from health effects associated with short-term exposures to $PM_{2.5}$.

Based on the above considerations, the staff recommends consideration be given to retaining an annual averaging time as a means of controlling both long- and short-term ambient $PM_{2.5}$ concentrations, and thus providing protection from health effects associated with both long- and short-term exposures to $PM_{2.5}$.

2. Form -- General Approaches

a. 24-Hour $PM_{2.5}$ Standard

As part of the last review, the 24-hour standard was changed from a deterministic form, in which the standard was not to be exceeded more than once per year, to a statistical form. The statistical form selected permits no more than one expected-exceedance, averaged over 3 years. The basis for this change in the form of the standard was that a statistical form can offer a more stable target for control programs and, with reasonably complete data, is less sensitive to truly unusual meteorological conditions than the deterministic form (U.S. EPA, 1982b). The staff continues to believe that this rationale is sound, but could be extended to consider alternatives that have been developed in conjunction with the ongoing review of the ozone standard. These general approaches to defining the form of a 24-hour standard include multiple exceedances and concentration percentile forms, as discussed more specifically in the next section in conjunction with the level of alternative standards.

One additional approach that is also being considered for the ozone standard is some form of averaging across multiple monitors. In a previous review of the PM NAAQS, staff recommended consideration of a multiple monitor spatial average form in its earlier recommendations for a secondary fine particle standard (EPA, 1982b). Such a form would better focus risk management activities on reductions in area or regionwide fine particle concentrations. Because the health effects information (as well as the risk assessment in Chapter VI) is keyed to fluctuations in areawide fine particle concentrations, such a form would also be more directly related to reduction in population risk. Such an approach would not have to require multiple monitors in all areas, assuming location criteria specified sites representative of areawide population exposures. If such an approach were adopted, consideration should be given to the

extent to which peak localized exposures might result in unacceptable individual risk. Limits on localized peak exposures might be provided through the 24-hour PM_{10} NAAQS, if retained, which is applied at each monitor individually. Appropriately located PM_{10} monitors would likely limit not only coarse fraction particle levels but also fine particle levels that result from highly localized emission sources.

b. Annual $PM_{2.5}$ Standard

As part of the last review, the annual standard was changed from a geometric mean to an expected arithmetic mean of the daily measurements. This change in the form of the standard was based on an arithmetic mean being 1) more directly related to dose, which is associated with observed health effects, 2) more sensitive to repeated short-term peaks, and 3) more consistent with other annual NAAQS (U.S. EPA, 1982b). The staff continues to believe that this rationale is sound and, thus, recommends that an expected arithmetic average form be adopted for an annual $PM_{2.5}$ standard. Further, as discussed above for a 24-hour standard, staff recommends consideration be given to adopting a spatial averaging approach for an annual $PM_{2.5}$ standard.

3. Level and Specific Forms

In developing an approach to formulating recommendations on appropriate ranges of levels and specific forms for 24-hour and annual $PM_{2.5}$ standards, staff has taken into account the following considerations:

1) Recent new epidemiological studies are noteworthy in their scope and efforts to account for potential confounding and other uncertainties (e.g., characterization of exposure). However, each individual study has inherent and methodological limitations and interpretation of these findings is the subject of ongoing debate within the scientific community. Thus, the staff views its assessment of each individual study in the context of the overall body of epidemiological evidence (with mechanistic support from toxicological and dosimetry studies) and the consistency and coherence of results across studies and effects.

2) As noted in the last review, it continues to be the case that even the best epidemiological studies have inherent limitations. Further, the available studies do not provide clear evidence of population thresholds of response. Thus, the staff recognizes that attempting

to identify "lowest observed effects levels" and adding margins of safety below such levels is not an appropriate approach in this case. Instead, the staff has attempted to assess the nature of health effects and risks, and the associated uncertainties, along a continuum of exposures using the full range of available health and exposure data from studies identified in the CD as being appropriate for quantitative assessments.

3) Relative to other single pollutants for which NAAQS have been set, establishing appropriate ranges of levels for $PM_{2.5}$ standards involves unusually large uncertainties. While recent studies help to reduce the uncertainties that were present in the last review, they do not change this basic observation relative to other NAAQS. To better address these uncertainties over time, the staff believes that research should continue into the more difficult problem of identifying and assessing potential health effects that may be associated with specific chemical and physical characteristics within the fine and coarse fractions of thoracic particles. However, even without any additional chemical-specific evidence, the staff believes that the large uncertainties inherent in setting $PM_{2.5}$ standards do not preclude our identifying appropriate ranges of policy alternatives from which specific standards can be selected to effectively and efficiently protect public health with an adequate margin of safety.

Taking these considerations into account, the staff's approach to formulating recommendations on appropriate ranges of standard levels and forms for the recommended $PM_{2.5}$ indicator and averaging times is based on: 1) quantitative results from studies showing statistically significant associations between ambient concentrations of fine fraction particles and health effects; 2) information on U.S. air quality distributions and estimated background levels of $PM_{2.5}$; 3) examinations of the quantitative concentration-response relationships suggested by specific epidemiological studies identified in the CD as appropriate for quantitative assessment purposes; 4) quantitative risk analyses that provide estimates of risk associated with air quality under "as is" conditions and attainment of current and alternative new $PM_{2.5}$ standards; and 5) quantitative and qualitative consideration of the sensitivity of the risk estimates to key assumptions and inherent uncertainties in these analyses that affect the margins of safety associated with ranges of standard levels. This approach recognizes that final decisions about appropriate PM standard levels and

forms must draw not only on scientific information about health effects and risks, but also on policy judgments about avoiding unacceptable risk from a public health perspective, addressing the uncertainties inherent in the evidence and assessments, and establishing health protective standards that serve as a meaningful guide to action in developing strategies to reduce unacceptable health risks associated with anthropogenic contributions to ambient $PM_{2.5}$ levels.

These staff assessments and considerations are discussed below for both 24-hour and annual $PM_{2.5}$ standards. The following discussions are based on information in the CD and in Chapters IV, V, and VI, and associated appendices, of this Staff Paper.

a. 24-Hour $PM_{2.5}$ Standard

Several key observations discussed below frame the staff's thinking in defining a range of 24-hour $PM_{2.5}$ levels and specific forms for the Administrator to consider in selecting an appropriate standard that protects public health with an adequate margin of safety from adverse health effects associated with ambient levels of $PM_{2.5}$.

- Staff notes, based on consideration of the body of evidence as a whole as discussed throughout this Staff Paper, that $PM_{2.5}$ concentrations occurring in areas that attain the current PM_{10} standards are likely to be associated with increased risks of mortality, hospital admissions, and respiratory symptoms in various sensitive subgroups.

As a result, staff concludes that an appropriate range of 24-hour $PM_{2.5}$ levels should result in reductions in health risks relative to the risks associated with the current PM_{10} standards. Results estimated for the highest 24-hour $PM_{2.5}$ level considered in the quantitative risk assessment done for two example cities, 65 $\mu g/m^3$, suggest that this level would result in some reductions in risks relative to the current standard, with the amount of reductions likely to vary from city to city.

As would be expected from these risk results, a $PM_{2.5}$ level of 65 $\mu g/m^3$ is below the $PM_{2.5}$ level that corresponds, based on a national average ratio, to the current PM_{10} standard level of 150 $\mu g/m^3$ (i.e., a $PM_{2.5}$ level of approximately 75 $\mu g/m^3$). Staff notes that the use of a national average ratio does not take into account the highly regional nature of the ratio between $PM_{2.5}$ and PM_{10}. In some Eastern areas, a $PM_{2.5}$ level as high as about 100 $\mu g/m^3$ could correspond to the current 24-hour PM_{10} standard level, whereas in some Western areas the corresponding $PM_{2.5}$

level could be as low as about 50 $\mu g/m^3$. Thus, there is no "equivalent" level that applies nationally based on information on ratios between $PM_{2.5}$ and PM_{10}. Alternatively, "equivalence" with the current NAAQS could be considered on the basis of determining the $PM_{2.5}$ standard level that would result in approximately the same number of counties that would not be in attainment. Consistent with the information provided in Table VII-1 for alternative PM_{10} standards, Table VII-2 presents the predicted total and regional distribution of the percentage of counties that would not attain the listed alternative $PM_{2.5}$ standards defined in terms of the current forms.[7] By comparison with Table VII-1, it can be seen that, based on the 1991-1993 PM_{10} data used to develop the two tables, a $PM_{2.5}$ level of greater than 75 $\mu g/m^3$ but well less than 100 $\mu g/m^3$ is predicted to result in approximately the same number of nonattainment counties as for the current 24-hour and annual NAAQS combined.

Based on the above discussion, although there is no clear point at which "equivalence" with the current NAAQS would be achieved, in staff's judgment consideration should be given to a $PM_{2.5}$ standard set below a level reflecting any type of approximate equivalence with the current NAAQS. Thus, staff recommends consideration be given to bounding the upper end of the range below 75 $\mu g/m^3$, at approximately 65 $\mu g/m^3$.

- Epidemiological studies reporting statistically significant associations were conducted in areas in which the mean 24-hour $PM_{2.5}$ concentrations ranged from approximately 16 to 30 $\mu g/m^3$ for mortality studies, with hospital admissions and respiratory symptoms studies falling within this range (Table VI-2).

Staff notes that these concentrations are relevant to considering a range of a standard, in that these studies are generally interpreted as providing risk estimates for which there is greatest confidence around the mean of the air quality data. However, as discussed in section V.E, there are significant uncertainties in any given study due to model specification, exposure misclassification, confounding, and other issues. Thus, staff believes that no one $PM_{2.5}$

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[7] The predicted comparison of counties not meeting alternative $PM_{2.5}$ standards in Table VII-2 is derived from an analysis that estimates $PM_{2.5}$ air quality from the much larger PM_{10} data base in AIRS (Fitz-Simons et al., 1996). As such, these estimates are highly uncertain and are presented here for rough comparative purposes only.

TABLE VII-2. PREDICTED PERCENTAGE OF COUNTIES NOT MEETING ALTERNATIVE $PM_{2.5}$ STANDARDS*

	Level of Alternative Standards**	All	SW	NW	CE	SE	NE
County Total		482	60	80	68	99	175
Annual	25	2.5	5.0	3.8	4.4	0	1.7
	20	8.7	15	8.8	15	4.0	6.9
	15	36	27	28	48	26	43
	10	84	52	65	93	95	94
24-hr	100	6.8	13	24	4.4	1.0	1.1
	75	15	28	41	15	2.0	6.3
	65	23	38	59	21	8.1	10
	50	42	58	78	35	38	25
	25	98	97	98	96	100	98
Combined Standards	25/75	15	28	41	16	2.0	6.3
	20/65	24	38	59	24	10	11
	15/50	56.	58	78	56	50	50

* These estimates are based on a methodology that uses the PM_{10} data in AIRS, together with more limited information on $PM_{2.5}/PM_{10}$ relationships, to predict which monitors might exceed a given $PM_{2.5}$ alternative standard. Such estimates are highly uncertain and should be interpreted with caution. More speifically, the estimates are based on 1991-1993 data, using a 50% data completeness criteria, and applying the Appendix K missing data adjustment to account for less than every day sampling frequenciew. See staff analyses (Fitz-Simons et al., 1996) which discusses methodology for calculating estimated $PM_{2.5}$ values.

** Based on current 1-expected-exceedance form of the 24-hour PM_{10} NAAQS and current expected annual average of annual PM_{10} NAAQS. at the highest monitor for each standard.

concentration derived from any particular study should appropriately serve as the basis for the level of a standard.

- Results from the quantitative risk assessment presented in section VI.C suggest a pattern of a continuum of decreasing risk with lower levels of alternative $PM_{2.5}$ standards, extending over and likely below the range of 65 to 25 $\mu g/m^3$ $PM_{2.5}$ included in the risk analyses.

Based on the limited risk analyses for two example cities, using base case assumptions, a 24-hour $PM_{2.5}$ standard of 25 $\mu g/m^3$ is estimated to reduce PM-related risks associated with short-term exposures for the effects considered by roughly 70% - 85%, relative to risks associated with attaining the current standards. Alternatively, at a 24-hour $PM_{2.5}$ level of 65 $\mu g/m^3$, risks are estimated to be reduced by roughly 10% and 40% for the Philadelphia and Los Angeles study areas, respectively. Putting these risk estimates into a broader perspective, these PM-related risk reductions translate into much smaller reductions relative to the total incidence of such effects from any cause. Relative to total incidence, a $PM_{2.5}$ standard of 25 $\mu g/m^3$ may reduce total mortality risk by roughly 1% to 2%, total hospital admissions by roughly 1% to 5%, and respiratory symptoms in children by roughly 15% - 25%. Alternatively, at a level of 65 $\mu g/m^3$, total mortality risk may be reduced by roughly 1% or less, total hospital admissions by roughly 2% to less than 1%, and respiratory symptoms in children by roughly 2% to 13%.

In terms of total incidence of effects upon attainment of alternative $PM_{2.5}$ standards, mortality incidence associated with short-term PM exposures is estimated to range from roughly 300 to 400 events per year for the Philadelphia (population 1.6 million) and Los Angeles (population 3.6 million) study areas, respectively, with a $PM_{2.5}$ standard of 65 $\mu g/m^3$. At a level of 25 $\mu g/m^3$, mortality incidence is estimated to be roughly on the order of 100 events per year in each study area. Estimated incidences of hospital admissions for respiratory and cardiac causes are up to 70% greater than those of mortality events. Respiratory symptom incidence is judged to be considerably more uncertain than estimates for the other effects, with roughly 10 to over 20 thousand events per year in the Philadelphia and Los Angeles study areas, respectively, at a level of 65 $\mu g/m^3$, and from roughly 3 to 6 thousand events per year, respectively, at a level of 25

$\mu g/m^3$. Thus, under base case assumptions, rough estimates of incidences are appreciably lower, but not eliminated in going from a $PM_{2.5}$ standard of 65 to 25 $\mu g/m^3$.

Staff emphasizes that these estimates are based on only two cities, include significant uncertainties, and are sensitive to a number of assumptions that have been considered in the integrated uncertainty analyses discussed in Chapter VI. Thus, policy judgments that are based in part on a consideration of such results should also take into account these uncertainties, critical assumptions, and the public health implications of the estimated incidence rates.

- Sensitivity analyses designed to address alternative assumptions in the risk analyses presented in section VI.C. suggest that estimated risks are sensitive to a number of assumptions, including in particular assumptions about the shape of concentration-response relationships and the ranges of air quality to which they are applied. The examination of concentration-response relationships that helped to frame the sensitivity and integrated uncertainty analyses provides information useful in identifying an appropriate $PM_{2.5}$ range for consideration.

For several alternative assumptions examined in the sensitivity and integrated uncertainty analyses, relatively small to moderately large differences in estimated risks were predicted across the range of alternative assumptions considered. In examining relevant concentration-response relationships using a variety of approaches, staff identified alternative cutpoints for the lower end of the range of air quality over which it may be appropriate to calculate increased risk from the studies. From the short-term $PM_{2.5}$ studies, staff identified concentrations of 10, 18, and 30 $\mu g/m^3$ as potential cutpoints reflecting increased uncertainties in this lower range of observed concentrations and inherent limitations in the data to detect any potential effects thresholds that may be present within that range. Relative to base case risk estimates, which do not assume any effects threshold or cutpoint within the range of the data, mortality risks estimated from the integrated uncertainty analysis are lower by as much as a factor of 2 across the range of alternative assumptions considered. Thus, alternative assumptions, most notably about the shape of the concentration-response relationship, can have significant impacts in lowering the estimated total PM-related risk for "as is" air quality as well as for attainment of the current NAAQS and alternative $PM_{2.5}$ standard cases.

- Several epidemiological studies reporting statistically significant effects include ranges of air quality that may approach estimates of background levels in some locations.

To serve as a meaningful guide to action in developing strategies to reduce unacceptable health risks associated with anthropogenic contributions to ambient $PM_{2.5}$ levels, staff believes that a standard should be set at a level sufficiently above estimated background levels. As discussed in Chapter IV, while estimated annual average $PM_{2.5}$ background levels range from approximately 2 to 5 $\mu g/m^3$ in the East and 1 to 4 $\mu g/m^3$ in the West, maximum annual 24-hour fine particle concentrations of 15 to 20 $\mu g/m^3$ are possible from background sources particularly in Eastern areas. Further, staff notes that on a daily basis exceptional natural events such as forest fires can result in even higher background concentrations, but such excursions are dealt with through the natural events policy in implementing the standards.

In taking into account the above observations, staff believes that the lower end of a range of $PM_{2.5}$ levels for the Administrator to consider in selecting an appropriate standard level should be less than 25 $\mu g/m^3$ but greater than 15 to 20 $\mu g/m^3$. While at 25 $\mu g/m^3$ significant reductions in risk may result, mortality studies show significant associations even when the observed means of 24-hour $PM_{2.5}$ concentrations in each of the study locations are approximately at or below 20 $\mu g/m^3$. Further, an assessment of concentration-response relationships below these levels suggested consideration of possible thresholds at concentrations of 18 and 10 $\mu g/m^3$. On the other hand, staff believes an appropriate standard should be sufficiently above estimated background levels so as to meaningfully facilitate the design and implementation of realistic air quality management strategies. Further, staff is mindful that the Act does not require that NAAQS be set at a zero-risk level, but rather at a level that avoids unacceptable risks and, thus, protects public health with an adequate margin of safety.

- With regard to specific alternative forms of 24-hour $PM_{2.5}$ standards, staff analyses of predicted $PM_{2.5}$ concentrations provide an illustrative comparison of the impact in terms of the number of counties that would not attain alternative forms for an example standard level (Table VII-3).

Table VII-3 compares the predicted impact of alternative exceedance-based forms (ranging from 1 to 5 exceedances per year) and concentration percentile forms (including the average n^{th} concentration percentile, with n ranging from the 95^{th} to the 99^{th} percentile) for an example 24-hour $PM_{2.5}$ standard level held constant at 50 $\mu g/m^3$ (in conjunction with an annual

$PM_{2.5}$ standard set at 15 $\mu g/m^3$).[8] As can be seen from the table, the form of the standard can result in significant differences in the number of areas that would not attain a given standard, such that the degree of health protection provided by a standard is a function of both the level and form of the standard.

TABLE VII-3. PREDICTED COMPARISON OF ALTERNATIVE FORMS FOR A 24-HOUR $PM_{2.5}$ STANDARD
(For counties meeting a 15 $\mu g/m^3$ annual $PM_{2.5}$ standard)

Alternative Forms of Standard	Number of Counties Projected to Meet 24-hour Standard of 50$\mu g/m^3$	Number of Counties Not Projected to Meet 24-hour Standard of 50$\mu g/m^3$
1 Exceedance	210	99
2 Exceedance	229	80
3 Exceedance	268	41
4 Exceedance	274	35
5 Exceedance	280	29
Avg 99th percentile	277	32
Avg 98th percentile	292	17
Avg 95th percentile	303	6

NOTE: Of the 482 counties with at least 50% data completeness per quarter 1991-93, 309 meet the PM2.5 annual standard, and 173 do not. Exceedance forms include the Appendix K missing data adjustment to account for less than every day sampling frequencies. See staff analyses in Fitz-Simons et al. (1996).

[8] As for Table VII-2, these staff estimates are based on predicting $PM_{2.5}$ concentrations based on the available PM_{10} data base, and are highly uncertain. See staff analyses in Fitz-Simons et al. (1996).

In weighing all these factors and considerations outlined above, staff offers the following conclusions and recommendations:

1) The lower end of the range of consideration for a new 24-hour $PM_{2.5}$ standard should be 20 $\mu g/m^3$. Considering a standard at this level would place significant weight on the consistency and coherence of the body of evidence as a whole, and on the results of quantitative analyses of concentration-response information and risks, even in light of inherent uncertainties in the analyses and alternative interpretations possible for each study considered independently. The staff believes that a 24-hour $PM_{2.5}$ standard set at this level, while not likely to be risk-free, would be precautionary in nature in protecting against a full range of short-term effects associated with the identified sensitive subgroups of the population. A standard set at this level would give less weight to concerns that the relied-upon studies may not have completely controlled for all potential confounding variables nor fully accounted for all limitations in the exposure data. Staff notes that this level is at the upper end of the range of uncertainty for peak 24-hour $PM_{2.5}$ background concentrations.

2) The upper end of the range of consideration for a new 24-hour $PM_{2.5}$ standard should be approximately 65 $\mu g/m^3$. A standard set at or near this level would give significant weight to both the qualitative and quantitative uncertainties inherent in the most recent epidemiological studies, and, conversely, little weight to the quantitative assessments of the evidence and associated risks. Such a standard would likely provide increased protection relative to the current standard.

3) In selecting a level for a 24-hour $PM_{2.5}$ standard within this range, the staff suggests that the Administrator also take into account the degree and nature of protection that would be afforded by a new annual $PM_{2.5}$ standard. The joint protection provided by a suite of standards that includes both 24-hour and annual $PM_{2.5}$ standards may be an important consideration in selecting the levels for each standard. One possible policy approach would be to view an annual $PM_{2.5}$ standard, as discussed below, as serving as the target for control programs designed to effectively lower the entire distribution of $PM_{2.5}$ concentrations, thus protecting not only against long-term effects but also short-term

effects as well. With this approach, the 24-hour $PM_{2.5}$ standard could be set so as to protect against the occurrence of peak 24-hour concentrations that would likely not be controlled in areas attaining a new annual $PM_{2.5}$ standard. Thus, in conjunction with an annual $PM_{2.5}$ standard, the Administrator may judge that the 24-hour standard should be set so as to limit only those peak 24-hour concentrations that are likely to persist upon attainment of the annual standard.

4) In selecting a form for a 24-hour $PM_{2.5}$ standard within the range of alternative forms analyzed, the staff suggests that the Administrator give primary consideration to a concentration percentile form. Concentration percentile forms are more stable and better take into account differences in sampling frequencies than the single (i.e., the current form) and multiple exceedance forms. Further, consideration should be given to the relative health protection provided by alternative forms at a given level, considering the relative impact of alternative forms on the number of counties affected by a particular form, and, thus, the number of areas likely to experience reduced risks to public health as a result of attaining a given standard level and form.

b. Annual $PM_{2.5}$ Standard

Similar to the approach outlined above for a 24-hour standard, the following observations frame the staff's thinking in defining a range of annual $PM_{2.5}$ levels:

- Staff notes that annual $PM_{2.5}$ concentrations occurring in some areas that attain the current PM_{10} standards are likely to be associated with increased risk of mortality beyond that associated with short-term mortality effects, as well as possibly increases in doctor-diagnosed cases of acute bronchitis in children.

- Further, as discussed above in the section on averaging times, an annual standard would have the effect of controlling air quality across the entire yearly distribution of 24-hour $PM_{2.5}$ concentrations to varying degrees, such that an annual standard set an appropriate level could also provide protection from health effects associated with short-term exposures to $PM_{2.5}$.

Based on the above considerations, the staff recommends consideration be given to use of an annual averaging time as a means of controlling both long- and short-term ambient $PM_{2.5}$

concentrations, and thus providing protection from health effects associated with both long- and short-term exposures to $PM_{2.5}$.

By comparing information in Tables VII-1 and VII-2, it can be seen that for the 1991-1993 data presented in the two tables, an annual $PM_{2.5}$ level of 25 $\mu g/m^3$ is estimated to result in approximately the same number of nonattainment counties as the current PM_{10} NAAQS. In staff's judgment consideration should be given to an annual $PM_{2.5}$ standard set below a level reflecting approximate equivalence with the current annual NAAQS. Thus, staff recommends consideration be given to bounding the upper end of the range below 25 $\mu g/m^3$, at approximately 20 $\mu g/m^3$.

Alternatively, in viewing an annual standard as creating a target for control programs designed to effectively lower the entire distribution of $PM_{2.5}$ concentrations, staff concludes that an appropriate range of annual $PM_{2.5}$ levels for such a standard should result in reductions in health risks relative to the risks associated with the combination of current 24-hour and annual PM_{10} standards. Under this approach, a comparison of Tables VII-1 and VII-2 suggests that an annual $PM_{2.5}$ standard level of less than 20 $\mu g/m^3$ would be needed to result in the same number of predicted nonattainment counties as for the combination of current 24-hour and annual PM_{10} NAAQS.

- Based on the long-term mortality study used in the quantitative risk assessment (Pope et al., 1995), a statistically significant association was observed across 151 cities in which the annual $PM_{2.5}$ concentrations ranged from approximately 9 to 34 $\mu g/m^3$ (Table VI-2); a somewhat similar range is estimated from the long-term studies of lung function decrements and doctor-diagnosed bronchitis in children (Table V-13).

Staff notes that these concentrations are relevant to considering a range for an annual standard, although, as discussed in Chapter VI and Appendix E, staff recognizes that uncertainty in the concentration-response relationships increase at the lower end of the range of data due in part to inherent limitations in discerning any potential effects threshold that may actually be present. In examining the concentration-response relationships for long-term mortality from the Pope et al. (1995) study, as well as from the more uncertain Dockery et al. (1993) study, possible concentration cutpoints at which effects threshold may potentially exist were identified (Chapter VI and Appendix E). The lowest such cutpoint was 12.5 $\mu g/m^3$, based on inherent limitations of

the data for discerning effects thresholds, and a cutpoint of 15 $\mu g/m^3$ was identified based on visual inspection of the data. The minimum mean concentrations in these two studies were 18 $\mu g/m^3$.

- The body of evidence from long-term exposure studies, together with results from the quantitative risk assessment presented in section VI.C, suggests a pattern of a continuum of decreasing risk with lower levels of alternative annual $PM_{2.5}$ standards, likely extending below the range of concentrations included in the analyses, 15 and 20 $\mu g/m^3$ $PM_{2.5}$ annual average.

Based on these limited analyses for two example cities, and applying only base case assumptions, the analyses estimate that an annual $PM_{2.5}$ standard of 15 $\mu g/m^3$ may reduce PM-related risks for mortality associated with long-term exposures by roughly 30 and 60% relative to risks associated with attaining the current NAAQS for Philadelphia and Los Angeles study areas, respectively. Alternatively, at a $PM_{2.5}$ level of 20 $\mu g/m^3$, reduction in risks associated with long-term exposure in Los Angeles county are estimated at 30%; staff notes that this level does not result in any estimated risk reduction in Philadelphia county because the current annual mean in Philadelphia is below this level. Putting these risk estimates into a broader perspective, these PM-related risk reductions translate into much smaller reductions relative to the total incidence of such effects from any cause. Relative to total incidence, an annual $PM_{2.5}$ standard of 15 $\mu g/m^3$ may reduce total mortality risk associated with long-term exposures by roughly 1and 5% for the Philadelphia and Los Angeles study areas, respectively. Alternatively, at a level of 20 $\mu g/m^3$, total mortality risk for Los Angeles county may be reduced by roughly 2%.

In terms of total incidence of effects upon attainment of alternative annual $PM_{2.5}$ standards, mortality incidence associated with long-term exposures to PM is estimated to range from roughly less than 1000 to about 1500 events per year for the Philadelphia and Los Angeles study areas, respectively, with an annual $PM_{2.5}$ standard of 20 $\mu g/m^3$, to roughly on the order of half as many events per year for each study location at a level of 15 $\mu g/m^3$. Thus, under base case assumptions, rough estimates of incidences are appreciably lower, but not eliminated, in going from an annual $PM_{2.5}$ standard of 20 to 15 $\mu g/m^3$.

Staff again emphasizes that these estimates are based on only two cities, include significant uncertainties, and are sensitive to a number of assumptions that can not be fully addressed by

additional analysis of available data. Thus, policy judgments that are based in part on a consideration of such results should also take into account these uncertainties, inherent limitations in available data and analyses, and the public health implications of the estimated incidence rates.

- Sensitivity analyses designed to address alternative assumptions in the risk analyses presented in section VI.C. suggest that estimated long-term risks are sensitive to a number of assumptions, including in particular assumptions about the shape of concentration-response relationships and the ranges of air quality to which they are applied and historical air quality information used in the analysis. The examination of concentration-response relationships and historical air quality that helped to frame these particular sensitivity analyses provides information useful in identifying an appropriate $PM_{2.5}$ range for consideration.

Based on an analysis of long-term mortality using the alternative cutpoints discussed in Chapter VI, staff notes that estimated risk for Philadelphia County is roughly 50% lower than the base case estimate if a 12.5 $\mu g/m^3$ cutpoint is applied. Similarly, applying a cutpoint of 15 $\mu g/m^3$ reduces estimated long-term mortality risk by over 75%, while applying a cutpoint of 18 $\mu g/m^3$ results in an estimate of no long-term mortality risk for "as is" air quality in Philadelphia County. Further, by assuming higher historical $PM_{2.5}$ concentrations than were reported in the Pope et al. (1995) study, estimated risk would be significantly lower than the base case estimate (Appendix F). Thus, alternative assumptions about the shape of the long-term PM concentration-response relationships and historical air quality can have very significant impacts on the estimated risk reductions associated with attaining alternative $PM_{2.5}$ standards.

In taking into account the above observations, staff believes that the lower end of a range of $PM_{2.5}$ levels for the Administrator to consider in selecting an appropriate annual standard level should be consistent with the lowest cutpoint for a possible threshold derived from an examination of the long-term mortality concentration-response relationships, 12.5 $\mu g/m^3$. Staff believes that such an annual level is sufficiently above estimated annual $PM_{2.5}$ background levels as to serve as a meaningful standard to facilitate the design and implementation of realistic air quality management strategies. Further, as noted above, staff is mindful that the Act does not require that NAAQS be set at a zero-risk level, but rather at a level that avoids unacceptable risks and, thus, protects public health with an adequate margin of safety.

In weighing all these factors and considerations outlined above, staff offers the following conclusions and recommendations:

1) The lower end of the range of consideration for a new annual $PM_{2.5}$ standard should be 12.5 $\mu g/m^3$. Considering a standard at this level would place significant weight on the consistency and coherence of the body of evidence as a whole, and on the results of quantitative analyses of concentration-response information and risks, even in light of inherent uncertainties in the analyses and alternative interpretations possible for the relevant studies. The staff believes that an annual $PM_{2.5}$ standard set at this level, while not likely to be risk-free, would be precautionary in nature in protecting against long-term mortality effects and other long-term morbidity effects such as lung function decrements and doctor-diagnosed bronchitis in children. A standard set at this level would give less weight to concerns that the relied-upon studies may not have completely controlled for all potential confounding variables nor fully accounted for all limitations in the exposure data.

2) The upper end of the range of consideration for a new annual $PM_{2.5}$ standard should be 20 $\mu g/m^3$. A standard set at or near this level would give significant weight to both the qualitative and quantitative uncertainties inherent in the long-term epidemiological studies, and, conversely, little weight to the quantitative assessments of the evidence and associated risks. Such a standard would likely provide some increased protection relative to the current annual standard.

3) As discussed above, in selecting a level for an annual $PM_{2.5}$ standard within this range, in conjunction with a 24-hour $PM_{2.5}$ standard, staff suggests that the Administrator take into account the joint protection likely to be afforded by both standards. In an approach that viewed the annual $PM_{2.5}$ standard as the primary target for control programs designed to effectively lower the entire distribution of $PM_{2.5}$ concentrations, the Administrator may choose to consider an annual standard from the lower end of this range. Correspondingly a 24-hour $PM_{2.5}$ standard could be set so as to protect against the occurrence of peak 24-hour concentrations that would likely not be controlled by areas attaining a new annual $PM_{2.5}$ standard. For example, an annual $PM_{2.5}$ standard at 15 $\mu g/m^3$ may be expected to result in substantially reduced 24-hour levels, potentially limiting the second highest 24-

hour levels to less than about 50 $\mu g/m^3$ in approximately 90% of the areas, thus adding to the protection against short-term effects afforded by a 24-hour standard (SAI, 1996).

D. Alternative PM_{10} Standards for Control of Coarse Fraction Particles

1. Averaging Time

If fine particle standards are adopted, the major function of the PM_{10} standard would be to protect against the known and anticipated effects associated with coarse fraction particles in the size range of 2.5 to 10 μm. As noted above, coarse fraction particles are plausibly associated with certain effects from both long and short-term exposures. Some epidemiologic evidence suggests increased asthma and upper respiratory infections may be associated with daily increases in PM_{10} that was dominated by coarse fraction particles (Gordian et al, 1996), while another study suggests smaller relative risks of bronchitis symptoms after daily episodes of very high fugitive dust (Hefflin et al, 1994). Both studies reported multiple exceedences of the current 24-hour NAAQS with PM_{10} peaks exceeding 900 $\mu g/m^3$. The potential build up of insoluble coarse fraction particles in the lung after long-term exposures to high levels should also be considered.

These studies show an important characteristic of significant coarse particle events. In a number of Western areas, multiple exceedences occur in relation to high winds increasing emissions from naturally occurring or human-disturbed surfaces. In the Gordian et al. (1996) study, the worst levels occurred in relation to a volcanic eruption. In a number of cases, such excursions are exempted from control by the natural events policy. In some areas, variations in annual rainfall or windspeed cause year-to-year changes in dust emissions, making implementation and assessment of control strategies more difficult. It is therefore appropriate to consider which combination of averaging time and form might provide a more robust target for practical coarse particle controls. In this regard, basing control on an annual standard alone or in combination with a 24-hour standard with multiple exceedences may provide adequate protection from potential long- and short-term effects of coarse fraction particles.

2. Level and form for alternative averaging times

a. Annual PM_{10} Standard

The nature of the more limited information for coarse fraction particles means the approach for selecting a level of the standard should be less like the recommended approach for fine particles, and more related to the approach taken in the last review for PM_{10}. In that approach, evidence from limited quantitative studies was used to select a range, with support from the qualitative literature used to support decisions within the range (EPA, 1982b, 1986).

The major quantitative basis for the level of the current annual PM_{10} standard was a study of children by Ware et al. (1986), conducted as part of the Harvard Six City series. This study has been supplemented in the recent literature by a follow-up long-term cohort study of acute bronchitis in children (Dockery et al., 1989). This study found somewhat better associations with PM_{15} than with $PM_{2.5}$ over the entire cohort, but a direct comparison with coarse fraction particles was not presented. However, still more recent studies found bronchitis symptoms in a larger cross sectional comparison to be unrelated to somewhat lower coarse particle concentrations than found in some of the six cities (Dockery et al, 1996). It is possible, but not conclusive, that coarse fraction particles, in combination with fine particles, may have influenced the observed effects, at least at the levels in the three most polluted cities in the study. From an exposure/deposition perspective, it is possible that cumulative deposition of coarse fraction particles could be elevated in children, who are more prone to be active outdoors than sensitive adult populations. Based on the original study by Ware et al. (1986), in the last review, staff recommended consideration that the lower bound of the range for the annual standard be set at 40 $\mu g/m^3$ (EPA, 1986).

Qualitative evidence of other long-term coarse particle effects, most notably from long-term buildup of silica containing materials, supports the need for a long-term standard, but does not provide evidence of effects below this range (CD, p 13-79). Staff concludes that the qualitative evidence with respect to biological aerosols (13-79) also supports the need to limit coarse materials, but should not form the major basis for a national standard. The nature and distribution of such materials, which vary from endemic fungi (e.g. valley fever) to pollens larger than 10 μm are not appropriately addressed by traditional air pollution control programs.

A PM_{10} standard in the range of 40 to 50 $\mu g/m^3$ (current level) would also provide substantial protection against the effects of 24-hour exposures associated with asthma and upper respiratory infections. The national mean ratio for the second highest 24-hour concentration in a year to the annual mean is 2.41 (SAI, 1996). This indicates that the mean second highest 24-hour concentrations associated with such a range (about 95 to 120 $\mu g/m^3$) would be well below the current standard. Peak levels at the worst sites could still exceed the level of the current 24-hour standard. Additional information on the relative short-term protection afforded by the current annual standard is summarized in the discussion below.

Staff recommends that consideration be given to adopting an annual PM_{10} standard in the range of 40 to 50 $\mu g/m^3$ to protect against the long- and short-term effects of coarse fraction particles. Such a standard would provide a more robust target for coarse particle controls that would be less sensitive to episodic natural events.

b. 24-Hour PM_{10} Standard

Consideration should also be given to a 24-hour standard for coarse fraction particles as measured by PM_{10}. The level of the current 24-hour PM_{10} standard (150 $\mu g/m^3$) was based in large measure on the London mortality and morbidity studies (EPA, 1982b). As noted above, staff believes that fine particles are a better surrogate for such effects. The main quantitative basis for a short-term standard is provided by the two fugitive dust studies referenced above. Because these studies reported multiple large exceedences of the current 24-hour standard they suggest no need to lower the level of the standard below 150 $\mu g/m^3$.

If a 24-hour PM_{10} standard is retained in conjunction with a fine particle standard, consideration should be given to maintaining the current level and revising the PM_{10} standard to a more robust form. Such forms would be less sensitive to naturally occurring episodes. Staff have conducted analyses of several alternative forms for a PM_{10} standard, similar to the analyses for alternative forms for a $PM_{2.5}$ standard as discussed above. Table VII-4 compares the impact of alternative exceedance-based forms (ranging from 1 to 5 exceedances per year) and concentration percentile forms (including the average n^{th} concentration percentile, with n ranging from the 95^{th} to the 99^{th} percentile) for an example 24-hour PM_{10} standard level held constant at 150 $\mu g/m^3$ (in conjunction with an annual PM_{10} standard set at 50 $\mu g/m^3$). As can be seen from the table, the

analysis suggests that a 50 $\mu g/m^3$ annual standard would limit 24-hour exceedences in all but nine of the sites to 5 or less (i.e., only nine sites would not attain a standard with a 5-exceedance form). Staff is examining alternative analytical approaches to provide additional insight into the relative protection afforded by these forms.

Because of the episodic nature of coarse particle excursions, the staff recommends that if a 24-hour standard is adopted, consideration should be given to one of the alternative more robust forms presented in Table VII-4, with or without an accompanying annual PM_{10} standard.

3. Summary of Coarse Fraction (PM_{10}) Standard Conclusions and Recommendations

Staff conclusions and recommendations are as follows:

1) As an indicator for coarse fraction particles, in conjunction with a $PM_{2.5}$ standard, the basis and purpose for the PM_{10} standards have been altered.

2) Staff recommends consideration of an annual PM_{10} standard in the range of 40 to 50 $\mu g/m^3$ to protect against both the short- and long-term effects of coarse fraction particles. An annual standard would provide a robust target for effective coarse particle control and monitoring strategies.

3) Consideration should also be given to a 24-hour PM_{10} standard of 150 $\mu g/m^3$ with a revised, more robust form selected from the range of alternatives presented in Table VII-4. Additional analyses of these forms are needed before more definitive recommendations can be made.

TABLE VII-4. COMPARISON OF ALTERNATIVE FORMS FOR A 24-HOUR PM_{10} STANDARD
(For counties meeting a 50 $\mu g/m^3$ annual PM_{10} standard)

Alternative Forms of Standard	Number of Counties Projected to Meet 24-hour Standard of 150$\mu g/m^3$	Number of Counties Not Projected to Meet 24-hour Standard of 150$\mu g/m^3$
1 Exceedance	425	46
2 Exceedance	433	38
3 Exceedance	451	20
4 Exceedance	455	16
5 Exceedance	462	9
Avg 99th percentile	455	16
Avg 98th percentile	467	4
Avg 95th percentile	471	0

NOTE: Of the 482 counties with at least 50% data completeness per quarter 1991-93, 471 meet the PM_{10} annual standard, and 11 do not. Exceedance forms include the Appendix K missing data adjustment to account for less than every day sampling frequencies. See staff analyses in Fitz-Simons et al. (1996).

E. Summary of Key Uncertainties and Research Recommendations

Staff believes it is important to emphasize the unusually large uncertainties associated with establishing standards for PM relative to other single component pollutants for which NAAQS have been set. The CD and this Staff Paper note throughout a number of unanswered questions and uncertainties that remain in the scientific evidence and analyses as well as the importance of ongoing research to address these issues. Prior to summarizing staff recommendations on the primary PM NAAQS in the next section, this section summarizes key uncertainties and related staff research recommendations.

1) One of the most notable aspects of the available information on PM is the lack of demonstrated mechanisms that would explain the mortality and morbidity effects associated with PM at ambient levels reported in the epidemiological literature. The absence of such mechanistic information limits judgments about causality of effects and appropriate concentration-response models to apply in quantitatively estimating risks. Building on promising preliminary findings from ongoing research involving more representative animal models and particle mixes and levels, staff believes there is an urgent need to expand ongoing research on the mechanisms by which PM, alone and in combination with other air pollutants, may cause health effects at levels below the current NAAQS.

2) Uncertainties and possible biases introduced by measurement error in the outdoor monitors, including both the error in the measurements themselves and the error introduced by using central monitors to estimate population exposure, contributes to difficulties in interpreting the epidemiological evidence. To address these concerns, additional research into improved continuous sampling and analyses methods, together with the use of a research-oriented ambient monitoring network and personal monitors to better characterize relationships between personal exposure and outdoor/indoor air quality, is needed for PM components as well as for other criteria pollutants. For example, monitoring techniques that allow new epidemiological studies to address not only size fractionation and improved measurements of semi-volatile particles but also particle number and surface area will be important to isolate key components of fine and coarse fraction particles. Further, examination of potential exposure to ultrafine particles near highways and other possible sources, for example, is important to determine the extent to which these materials persist long enough to present significant exposure to sensitive population groups.

3) Inherent in epidemiological studies such as those cited in this review is the question as to whether or to what extent the observed effects attributed to PM exposures are confounded by other pollutants commonly occurring in community air, such as SO_2, ozone, NO_2, and CO. In particular, a number of authors conducting reanalyses of

mortality studies within a given city, most notably for Philadelphia, have demonstrated that it may not be possible to separate individual effects of multiple pollutants when those pollutants are highly correlated within a given area. Based on its assessment of available information regarding potential confounding within and across a number of areas with differing combinations of pollutants, as recommended in the HEI reanalysis report, the CD concludes that in general the reported PM effects associations are valid and not likely to be seriously confounded by copollutants. Nevertheless, additional research and analyses are important to better characterize the extent to which PM-related effects may be modified by the presence of other copollutants in the ambient air.

4) Although staff has concluded that it is more likely than not that fine fraction particles play a significant role in the reported health effects associations, identification of specific components and/or physical properties of fine particles which are associated with the reported effects is very important for both future reviews of the standards and in development of efficient and effective control strategies for reducing health risks. Epidemiological and toxicological research is needed to isolate key components (e.g., nitrates, sulfates, organics, metals, ultra fine particles) and/or characteristics of fine particles, as well as to identify the nature and extent of subpopulations most susceptible to the adverse effects associated with such components and/or characteristics. Such research is critical in addressing uncertainties in estimating risk reductions likely to be achieved by alternative fine particle standards and new implementation strategies.

5) Uncertainties in the shape of concentration-response relationships, most specifically whether linear or threshold models are more appropriate, significantly affects the confidence with which risks and risk reductions can be estimated. Mechanistic and epidemiological research highlighted above would likely help reduce such uncertainties.

6) Unaddressed confounders and methodological uncertainties inherent in epidemiological studies of long-term PM exposures limit interpretations and conclusions that can be drawn with regard to associations between PM and chronic health effects. Additional research and analysis are needed to reduce the uncertainties related to the appropriate exposure

periods and historical air quality to consider in evaluating such studies, and to better address life-style and other potentially important cofactors.

7) An important aspect in characterizing the nature of the mortality risk associated with short- and long-term exposures to PM, from a public health perspective, is the extent to which lifespans are being shortened. Available epidemiological evidence provides a very limited basis for testing hypotheses as to whether and to what extent lifespans are shortened by only a few days or by years. More research is needed to quantitatively characterize the degree of prematurity of deaths associated with exposures to PM.

8) The characterization of annual and daily background concentrations likely to occur across the U.S. contains significant uncertainties. Additional air quality monitoring and analyses that improve these background characterizations would help to reduce the uncertainties in estimating health risks relevant to standard setting, i.e., those risks associated with exposures to PM in excess of background levels.

9) Despite long-standing staff recommendations for a comprehensive examination of the effects associated with exposures to coarse fraction particles, there continues to be a lack of animal, clinical, and community studies in this area. Such research would potentially provide both qualitative and quantitative information that could allow for the establishment of a coarse fraction particle standard rather than continued reliance on a PM_{10} standard as the means to control exposures to coarse fraction particles.

F. Summary of Staff Recommendations on Primary PM NAAQS

The major staff recommendations and supporting conclusions from sections VII.A-D are briefly summarized below:

1) The current PM standards should be revised. As the Criteria Document concludes, current evidence provides ample reason to be concerned that there are detectable health effects attributable to PM at levels below the current NAAQS. Given the nature and potential magnitude of the public health risks involved, staff believes revision of the current standards is clearly appropriate. The health effects reported, ranging from premature mortality to various measures of morbidity, including increased hospital

admissions, aggravation of existing respiratory disease, including asthma, and decreased lung function, include effects that are clearly adverse to public health.

2) Ambient particles capable of penetrating to the thoracic region, including both the fine and coarse fractions of PM_{10}, should continue to be the focus of PM standards. Staff concludes that these thoracic particles represent the greatest risk to health, and that the previous recommendations for 10 μm as the appropriate cutpoint for such particles remain valid.

3) The fine and coarse fractions of PM_{10} should be considered as two separate pollutants based on the recent health evidence, the fundamental differences between fine and coarse fraction particles, and implementation experience with PM_{10}. The staff concludes that the available information is sufficient to support separate indicators for these separate pollutants. Further, while it is difficult to distinguish the effects of fine or coarse fraction particles from those of PM_{10}, consideration of comparisons between fine and coarse fractions suggests that fine fraction particles are a better surrogate for those particle components linked to mortality and morbidity effects at levels below the current standards. In contrast, coarse fraction particles are more likely linked with certain effects at levels above those allowed by the current PM_{10} standards. In examining alternative approaches to increasing the protection afforded by PM_{10} standards, the staff concludes that reducing the levels of the current PM_{10} standards would not provide the most effective and efficient protection from these health effects.

4) A 2.5 μm cutpoint (i.e., $PM_{2.5}$) should be used as the indicator for fine fraction particles, and the current PM_{10} indicator should now be used as the indicator for the coarse fraction particles. A $PM_{2.5}$ indicator for fine fraction particles is specifically recommended based primarily on consistency with the health effects literature and the suitability and availability of ambient monitors. The recommendation for PM_{10} as the indicator for coarse fraction particles is based on the very limited data base and monitoring capabilities directly for coarse fraction particles, as well as the applicability of the existing PM_{10} monitoring network. Further, staff concludes that use of sulfate or other chemical class indicators is not advisable on the basis of this review.

5) Staff recommends that new $PM_{2.5}$ standards be established for two averaging times.

a) Annual and 24-hour $PM_{2.5}$ standards should be established as the most appropriate standards to address health effects associated with both short-term (from less than 1 day up to 5 days) and long-term (from months to years) exposures to fine fraction particles.

b) Staff recommends consideration of more robust forms for a 24-hour standards (especially concentration percentile forms), averaged over three years. In addition, staff recommends consideration be given to using the average of multiple monitors representative of population exposure as part of the form of the annual and/or 24-hour standards. Staff also recommends the retention of the current expected arithmetic average form of the annual standard.

c) Staff recommends that the Administrator consider selecting the level of a new 24-hour $PM_{2.5}$ standard from the range of 20 $\mu g/m^3$ to approximately 65 $\mu g/m^3$, and the level of a new annual $PM_{2.5}$ standard from the range of 12.5 $\mu g/m^3$ to approximately 20 $\mu g/m^3$. These recommended ranges are based primarily on quantitative results from epidemiological studies, examinations of concentration-response relationships suggested by these studies, quantitative risk assessment, including consideration of the sensitivity of the risk estimates to key assumptions and inherent uncertainties in the underlying data and analytic approaches, and relevant policy considerations based on air quality analyses. In recommending these ranges, staff is mindful that the Clean Air Act does not require that NAAQS be set at zero-risk levels, but rather at level that avoid unacceptable risks to public health, thus protecting public health with an adequate margin of safety. Further, in selecting specific levels for $PM_{2.5}$ standards, staff recommends that the Administrator consider the joint protection afforded by both the 24-hour and annual standards. The recommended approach is to view an annual $PM_{2.5}$ standard as the primary target for control programs designed to effectively lower the entire distribution of $PM_{2.5}$ concentrations, with a corresponding 24-hour $PM_{2.5}$ standard

set so as to protect against the occurrence of peak 24-hour concentrations that would likely not be controlled by areas attaining such a new annual $PM_{2.5}$ standard.

6) Staff recommends that an annual PM_{10} standard be retained, alone or in combination with a 24-hour PM_{10} standard.

   a) Staff recommends that the Administrator consider selecting the level of an annual PM_{10} standard from the range of 40 $\mu g/m^3$ to 50 $\mu g/m^3$, with an expected arithmetic mean form. Such a standard would reflect the range considered in the last review, and would protect against the principal effects of concern, including effects associated with both short- and long-term exposures to PM such as aggravation of asthma, upper respiratory infections, and bronchitis in children, as well as the long-term build-up of insoluble coarse fraction particles in the lung.

   b) Further, if a 24-hour PM_{10} standard is retained, staff recommends retention of the current level of 150 $\mu g/m^3$, but with a revised, more robust form to better address the episodic nature of coarse particle excursions.

VIII. CRITICAL ELEMENTS IN THE REVIEW OF THE SECONDARY STANDARD FOR PARTICULATE MATTER

A. Introduction

This chapter presents critical information for the review of the secondary NAAQS for particulate matter drawing upon the most relevant information contained in the CD and other significant reports. The welfare effects of most concern for this review are visibility impairment, soiling, damage to man-made materials, and damage to and deterioration of property. For each category of effects, the chapter presents (1) a brief summary of the relevant scientific information and (2) a staff assessment of whether the available information suggests consideration of secondary standards different than the recommended primary standards. Staff conclusions and recommendations related to the secondary standard for PM are presented at the end of the chapter.

It is important to note that the discussion of fine particle effects on visibility in chapter 8 of the CD is intended to only include information complementary to several other significant reviews of the science of visibility. These reports include the 1991 report of the National Acid Precipitation Assessment Program, the National Research Council's *Protecting Visibility in National Parks and Wilderness Areas* (1993), and EPA's 1995 *Interim Findings on the Status of Visibility Research*. Where appropriate, this chapter of the staff paper will cite the above reports directly.

The chapter does not address the effects of particles on climate change. As discussed in the criteria document, particles (in the submicrometer size range) can result in perturbations of the radiation field that are generally expressed as radiative forcing. Radiative forcing due to aerosols has a cooling effect on climate through the reflection of solar energy. This is in contrast to "greenhouse gas" that produces a positive long wave radiative forcing which has a warming effect. Given the complex interaction of these two phenomena and the present state of the science, it is the staff's judgment that these effects should not be addressed in this paper, but should instead be considered in the broader context of global climate change.

B. Effects of PM on Visibility

1. Definition of Visibility and Characterization of Visibility Impairment

Visibility can be defined as the degree to which the atmosphere is transparent to visible light (NRC, 1993; CD, 8-3). Visibility effects are manifested in two principal ways: (1) as local impairment (e.g., localized hazes and plumes) and (2) as regional haze. These distinctions are significant both to the ways in which visibility goals may be set and air quality management strategies may be devised.

Local-scale visibility degradation has been generally defined as impairment that is "reasonably attributable" to a single source or group of sources. A localized haze may be seen as a band or layer of discoloration appearing well above the terrain, and may result from complex local meteorological conditions. "Reasonably attributable" impairment may include contributions to local hazes by individual or several identified sources. Plumes are comprised of smoke, dust, or colored gas that obscure the sky or horizon relatively near sources. Sources of locally visible plumes, such as the plume from an industrial facility or a burning field, are often easy to identify. Overall, visible plumes appear to be minor contributors to visibility impairment in Class I areas (i.e., certain national parks, wilderness areas, and international parks as described in section 162(a) of the Clean Air Act) (NRC, 1993).

The second type of impairment, regional haze, is produced from a multitude of sources and impairs visibility in every direction over a large area, possibly over several states. Regional haze masks objects on the horizon and reduces the contrast of nearby objects. The formation, extent, and intensity of regional haze is a function of meteorological and chemical processes, which sometimes cause fine particle loadings to remain suspended in the atmosphere for several days and to be transported hundreds of kilometers from their sources (NRC, 1993). It is this second type of visibility degradation that is principally responsible for impairment in national parks and wilderness areas across the country (NRC, 1993). Visibility in urban areas may be dominated by local sources, but may be significantly affected by long-range transport of haze as well. Fine particles transported from urban areas in turn may be significant contributors to regional-scale impairment in Class I areas.

2. Significance of Visibility to Public Welfare

Visibility is an air quality-related value having direct significance to people's enjoyment of daily activities in all parts of the country. Survey research on public awareness of visual air quality using direct questioning typically reveals that 80% or more of the respondents are aware of poor visual air quality (Cohen et al., 1986). Individuals value good visibility for the well-being it provides them directly, both in the places where they live and work, and in the places where they enjoy recreational opportunities. Millions of Americans appreciate the scenic vistas in national parks and wilderness areas annually. Visibility is also highly valued because of the importance people place on protecting nationally significant natural areas, both now and in the future (i.e., preservation value). Many individuals want to protect such areas for the benefit of future generations, even if they personally do not visit these areas frequently (Chestnut et al., 1994). Tracking changes in visibility provides one measure of the success of efforts to protect such areas from environmental degradation. Society also values visibility because of the significant role it plays in air transportation. Serious episodes of visibility impairment can lead to increased risks in the air transportation industry, particularly in urban areas with high traffic levels (U.S. EPA, 1982b).

Many contingent valuation studies have been performed in an attempt to quantify benefits (or individuals' willingness to pay) associated with improvements in current visibility conditions. The results of several studies are presented in CD table 8-5 (CD, 8-83), table 8-6 (CD, 8-85), and in table VIII-1 (Chestnut et al., 1994). Past studies by Schultze (1983) and Chestnut and Rowe (1990b) have estimated the preservation values associated with improving the visibility in national parks in the Southwest to be quite significant, on the range of approximately $2-6 billion annually (CD, 8-84). Another recent study estimates visibility benefits primarily in the eastern U.S. due to reduced sulfur dioxide emissions under the acid rain program also to be quite significant, in the range of $1.7 - 2.5 billion annually by the year 2010 (Chestnut et al., 1994).

3. Mechanisms of and Contributors to Visibility Impairment

Visibility impairment has been considered the "best understood and most easily measured effect of air pollution" (Council on Environmental Quality, 1978). It is caused by the scattering and absorption of light by particles and gases in the atmosphere. It is the most

TABLE VIII-1. COMPARISON OF RESIDENTIAL VISIBILITY VALUATION STUDY RESULTS

Study	City	Mean WTP ($1990)	Starting VR (miles)	Ending VR (miles)	b coefficient	WTP for 20% changes VR (3)
Eastern CVM Studies						
McClelland et al.	Atlanta and Chicago	Unadj. $39 Partial $25 Full $18	17.6	20	305 196 140	$56 $36 $26
Tolley et al.	Chicago	-$318 $305 $379	9 9 9	4 18 30	367	$67
Tolley et al	Atlanta	-$265 $255 $381	12 12 12	7 22 32	414	$75
Tolley et al	Boston	-$196 $187 $231	18 18 18	13 28 38	372	$68
Tolley et al	Mobile	-$212 $227 $266	10 10 10	5 20 30	275	$68
Tolley et al	Washington, DC	-$314 $323 $410	15 15 15	10 25 35	560	$102
Tolley et al	Cincinnati	-$78 $77 $86	9 9 9	4 19 29	106	$17
Tolley et at	Miami	-$134 $120 $141	13 13 13	8 19 29	226	$41
Rae	Cincinnati	$175	11.4	16.4	531	$97
California CVM Studies						
Brooksire et al.	Los Angeles	$115 $294 $161	2 2 12	12 28 28	105	$19
Loehman et al	San Francisco	-$186 $109	18.6 16.3	16.3 18.6	1172	$214
California Property Value Study						$216-$579
Trijonis et al	Los Angeles					$437-$487
Trijonis et al	San Francisco					

Note: VR - Visual Range

Source: Chestnut et al., 1994.

noticeable effect of fine particles present in the atmosphere. Air pollution degrades the visual appearance of distant objects to an observer, and reduces the range at which they can be distinguished from the background. Ambient particles affect color of distant objects depending upon particle size and composition, the scattering angle between the observer and illumination, the properties of the atmosphere, and the optical properties of the target being viewed.

Fine particles can be emitted directly to the atmosphere through primary emissions or formed secondarily from gaseous precursors. The fine particles principally responsible for visibility impairment are sulfates, nitrates, organic matter, elemental carbon (soot), and soil dust. The efficiency of particles to cause visibility impairment depends on particle size, shape, and composition. Fine particles are effective per unit mass concentration in impairing visibility because their mean diameter is usually comparable to the wavelength of light, a condition that results in maximum light scattering. In the size range from 0.1 to 1.0 μm in diameter, fine particles are more effective per unit mass concentration at impairing visibility than either larger or smaller particles (NAPAP, 1991). Coarse particles (i.e., those in the 2.5 to 10 μm size range) also impair visibility, although less efficiently than fine particles. All particles scatter light to some degree, whereas only elemental carbon plays a significant role in light absorption. In all regions of the country, annual average light extinction is dominated by light scattering as opposed to light absorption (NRC, 1993). Appendix G provides a detailed discussion of several atmospheric optical indices that are used in characterizing visibility impairment and light extinction, including the light extinction coefficient, visual range, and deciview.

Most sulfates, nitrates, and a portion of organics begin as gaseous emissions and undergo chemical transformation in the atmosphere (NAPAP, 1991; CD, 3-2). These particle constituents can readily absorb water from the atmosphere (i.e., are hygroscopic) and grow in size in a nonlinear fashion as relative humidity levels increase. In general, soluble organics are considered to be less hygroscopic than sulfates and nitrates (Sisler, 1993). The relationship between humidity and particle size is a significant factor in visibility impairment in the East, where in many locations average relative humidity exceeds 70% on an annual

average basis and can surpass 80% on many days, particularly in the summer (see more detailed discussion of humidity in section 5).

Light absorption is caused mainly by elemental carbon, a product of incomplete combustion from activities such as the burning of wood or diesel fuel. Light absorption by nitrogen dioxide typically accounts for a few percent of total light extinction in urban areas and is typically negligible in remote areas (CD, 8-13). It contributes to the yellow or brown appearance of urban hazes since it absorbs blue light more strongly than other visible wavelengths. Nitrogen dioxide also may be a factor in isolated plumes from industrial sources in remote locations.

Atmospheric transport of fine particles is a critical factor affecting regional visibility conditions. Fine particles and their precursors can remain in the atmosphere for several days and can be carried hundreds or even thousands of kilometers from their sources to remote locations, such as national parks and wilderness areas (NRC, 1993).

4. Background Levels of Light Extinction

The light extinction coefficient represents the summation of light scattering and light absorption due to particles and gases in the atmosphere. (See Appendix G for a discussion of visibility metrics and the relationship between the light extinction coefficient, visual range, and deciview.) Both anthropogenic and non-anthropogenic sources contribute to light extinction. The light extinction coefficient is represented by the following equation:

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

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

σ_{ag} = light absorption by gases

σ_{sp} = light scattering by particles

σ_{ap} = light absorption by particles (CD, 8-12).

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

a. Rayleigh Scattering

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, CO_2). It accounts for a relatively constant level of light extinction nationally, between 10-12 Mm^{-1} (NAPAP, 1991; U.S. EPA, 1979). The concept of Rayleigh scattering can be used to establish a theoretical maximum horizontal visual range in the earth's atmosphere. At sea level, this maximum visual range is approximately 330 kilometers. Since certain meteorological circumstances can result in visibility conditions that are close to "Rayleigh," it is analogous to a baseline or boundary condition against which other extinction components can be compared.

b. Light Extinction Due to Background Particulate Matter

Light extinction caused by PM from non-anthropogenic 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 background concentrations of PM on an annual average basis, however, when evaluating the relative contributions of anthropogenic and non-anthropogenic sources to total light extinction.

Chapter 4 of the staff paper addresses annual average and 24-hour estimates of background concentrations of PM. Table IV-3 describes the range for annual average regional background $PM_{2.5}$ mass in the East as 2-5 $\mu g/m^3$, and in the West 1-4 $\mu g/m^3$. For PM_{10}, the estimated annual average background concentrations range from 5-11 $\mu g/m^3$ in the East, and 4-8 $\mu g/m^3$ in the West. For background 24-hour $PM_{2.5}$ values, present day observed peak to mean ratios of 2 to 4 can be assumed to apply to the background annual values in table IV-3. This approach suggests that the highest background 24-hour $PM_{2.5}$ levels over the course of a year could be on the order of 15-20 $\mu g/m^3$.

Table VIII-2 from the NAPAP report includes estimates of annual average background concentrations of PM by aerosol constituent, as well as their related contributions to light extinction, expressed in inverse megameters (Mm^{-1}) (NAPAP, 1991). On an hourly or daily basis background concentrations will vary considerably depending on seasonal, meteorological, and geographic factors. The table illustrates that estimated extinction contributions from Rayleigh scattering plus background levels of fine and coarse particles, in

TABLE VIII-2. AVERAGE NATURAL BACKGROUND LEVELS OF AEROSOLS AND LIGHT EXTINCTION

	Average Concentration East $\mu g/m^3$	Average Concentration West $\mu g/m^3$	Error Factor	Extinction Efficiencies[a] m_2/g	Extinction Contributions East Mm^{-1}	Extinction Contributions West Mm^{-1}
Fine Particles ($\leq$2.5μm)						
Sulfates (as NH_4HSO_4)	0.2	0.1	2	2.5	0.5	0.2
Organics	1.5	0.5	2	3.75	5.6	1.9
Elemental Carbon	0.02	0.02	2-3	10.5	0.2	0.2
Ammonium Nitrate	0.1	0.1	2	2.5	0.2	0.2
Soil dust	0.5	0.5	1.5-2	1.25	0.6	0.6
Water	1.0	0.25	2	5	5.0	1.2
Coarse Particles (2.5-10μm)	3.0	3.0	1.5-2	0.6	1.8	1.8
			Rayleigh Scatter		12	11
				Total	26$\pm$7	17$\pm$2.5

[a]The extinction efficiencies are based on the literature review by Trijonis et al. (1986 & 1988). All the extinction efficiencies represent particle scattering, except for elemental carbon where the 10.5 m^2/g value is assumed to consist of 9 m^2/g absorption and 1.5 m^2/g scattering. Note that the 0.6 m^2/g value for coarse particles is a "pseudo-coarse scattering efficiency" representing t total scattering by all ambient coarse particles (2.5 μm) divided by the coarse particle mass between 2.5 and 10 μm.

the absence of anthropogenic emissions of visibility-impairing particles, are 26 plus or minus 7 Mm^{-1} in the East, and 17 plus or minus 2.5 Mm^{-1} in the West. These equate to a naturally-occurring visual range in the East of 150 plus or minus 45 kilometers, and 230 plus or minus 40 kilometers in the West. Excluding light extinction due to Rayleigh scatter, annual average background levels of fine and coarse particles are estimated to account for 14 Mm^{-1} in the East and about 6 Mm^{-1} in the West. Major contributors that reduce visibility from the Rayleigh maximum to the ranges noted above are naturally-occurring organics, suspended dust (including coarse particles), and water. In these ranges of fine particle concentrations, small changes have a large effect on total extinction. Thus, one can see from table VIII-2 that higher levels of background fine particles and associated humidity in the East result in a fairly significant difference between naturally-occurring visual range in the rural East and West.

5. Overview of Current Visibility Conditions

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

Visibility conditions also vary significantly by season of the year. With the exception of remote sites in the northwestern U.S., visibility is typically worse in the summer months. This is particularly true in the Appalachian region, where average extinction in the summer exceeds the annual average by 40% (Sisler et al., 1996).

Figures VIII-1 and VIII-2 present 3-year (March 1992 - February 1995) averages of monitored visibility levels for 44 sites in the IMPROVE (Interagency Monitoring of PROtected Visual Environments) network. (See Appendix G for a description of the aerosol, optical, and scene measurements taken in the IMPROVE network.) The regional variation in current conditions is quite apparent from these figures. Figure VIII-1 expresses conditions in terms of the extinction coefficient. The highest annual average levels are found in the rural

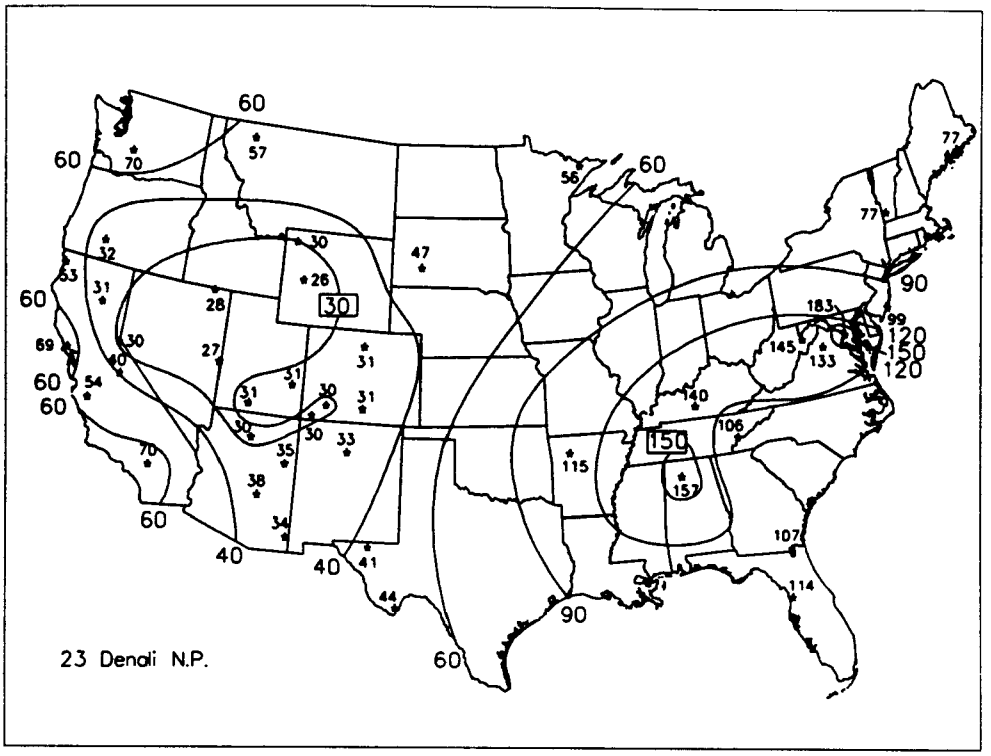


FIGURE VIII-1. AVERAGE LIGHT EXTINCTION COEFFICIENT (IN MM^{-1}) FOR EACH OF THE REPORTED SITES IN THE IMPROVE NETWORK, 1992-1995. (Sisler et al., 1996)

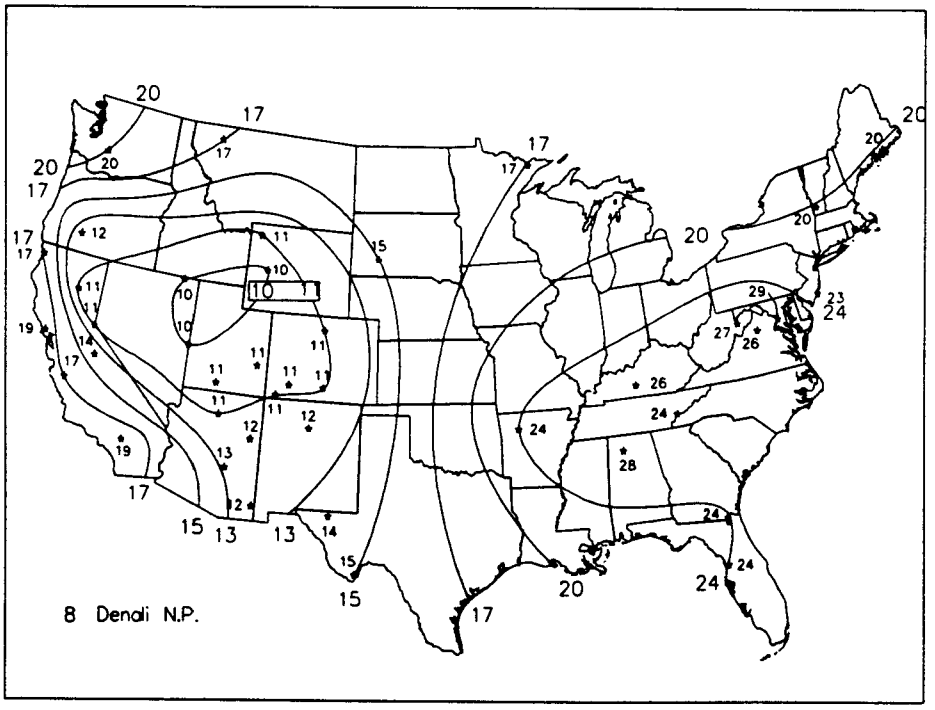


FIGURE VIII-2. ANNUAL AVERAGE VISIBILITY IMPAIRMENT IN DECIVIEWS CALCULATED FROM TOTAL LIGHT EXTINCTION (RAYLEIGH INCLUDED), IMPROVE NETWORK, 1992-1995. (Sisler et al., 1996)

East, where the coefficient ranges from about 100-160 Mm^{-1} (about 23-39 kilometers visual range) for several rural sites south of the Great Lakes and east of the Mississippi River. This means that in certain eastern sites, 3-year average light extinction due to anthropogenic sources is 4 to 6 times natural light extinction levels.

The 3-year average extinction coefficient for many western sites ranges from about 30-70 Mm^{-1} (about 55-150 kilometers visual range), with the lowest extinction found in the intermountain west and Colorado plateau regions. Most of this difference between East and West is due to greater sulfate concentrations and the effect of higher humidity levels on this sulfate in the East (NAPAP, 1991). Studies of historical visibility trends have shown a fairly strong correlation between long-term light extinction levels and sulfur dioxide emissions. This correlation is illustrated for the northeast and southeast U.S. in figure IV-8 and is further discussed in section IV.B. of the staff paper.

Figure VIII-2, which expresses 3-year average visibility conditions in terms of deciviews, shows the same regional variability. Pristine or Rayleigh conditions are represented by a deciview of zero, whereas the highest 3-year average level of impairment in a remote site is 28 deciview in Alabama's Sipsey Wilderness. Under many circumstances, a change of one deciview represents a change perceptible to the average person. By using the deciview scale, the effect of aerosol extinction on human perception is portrayed as a linear scale of visibility degradation. Most of the sites in the intermountain west and Colorado Plateau have impairment of 12 deciviews or less. The northwest and eastern half of the U.S. have values greater than 15 deciviews, with much of the east having values exceeding 23 deciviews.

Figures VIII-3 and VIII-4 present multi-year averages for $PM_{2.5}$ and PM_{10} at IMPROVE sites. Analyses of aerosol constituents from these data are used in determining the light extinction coefficient and deciview. Again, regional variability is apparent, with 3-year average $PM_{2.5}$ levels for most rural western sites in the 2-5 $\mu g/m^3$ range, and levels in the rural East in the 9-15 $\mu g/m^3$ range. Figure VIII-5 compares $PM_{2.5}$ mass to PM_{10} mass for each IMPROVE site. It illustrates that fine PM comprises a larger fraction of PM_{10} in remote eastern (60-70%) versus western (40-50%) locations.

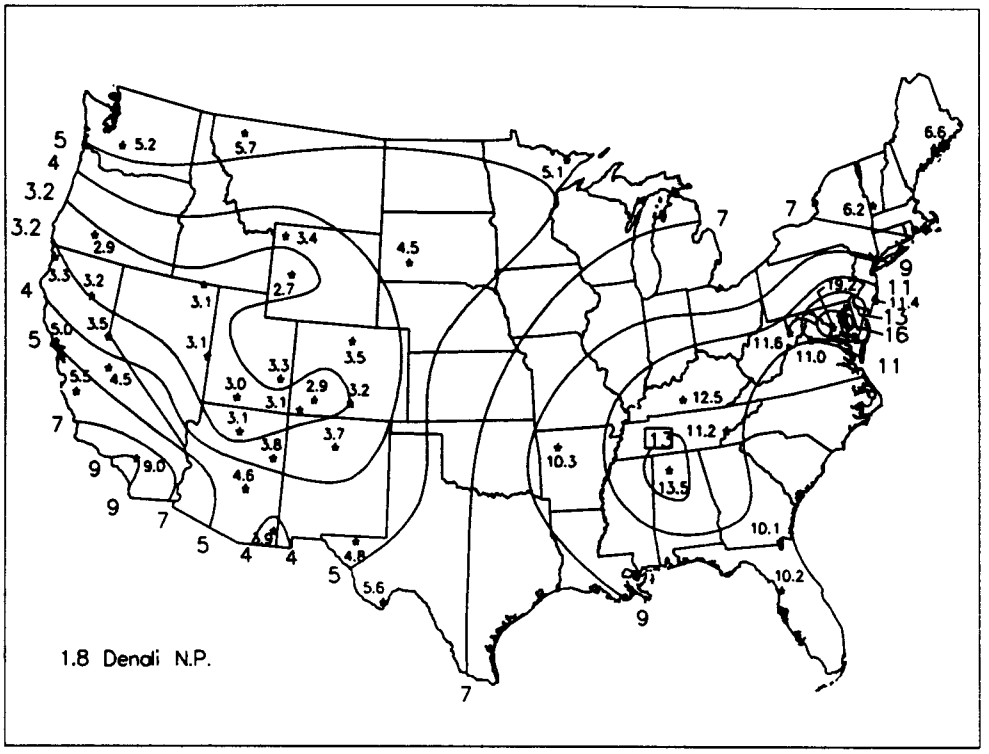


FIGURE VIII-3. AVERAGE $PM_{2.5}$ MASS CONCENTRATION (IN ug/m^3) FOR EACH SITE IN THE IMPROVE NETWORK, 1992-1995. (Sisler et al., 1996)

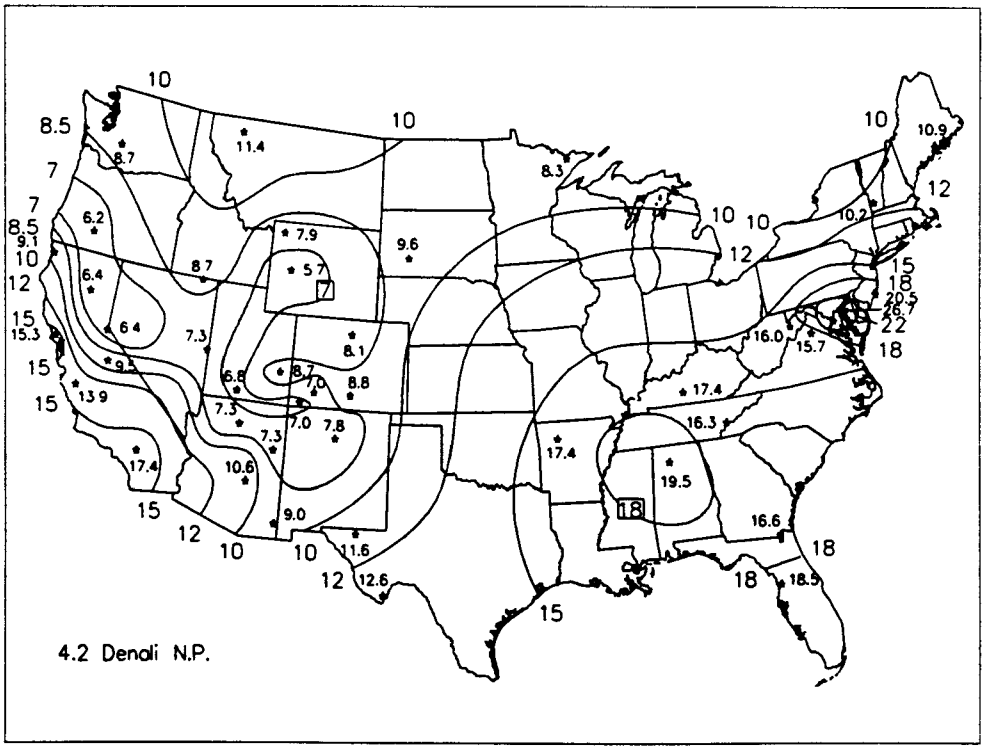


FIGURE VIII-4. AVERAGE PM_{10} MASS CONCENTRATION (IN ug/m^3) FOR EACH SITE IN THE IMPROVE NETWORK, 1992-1995. (Sisler et al., 1996)

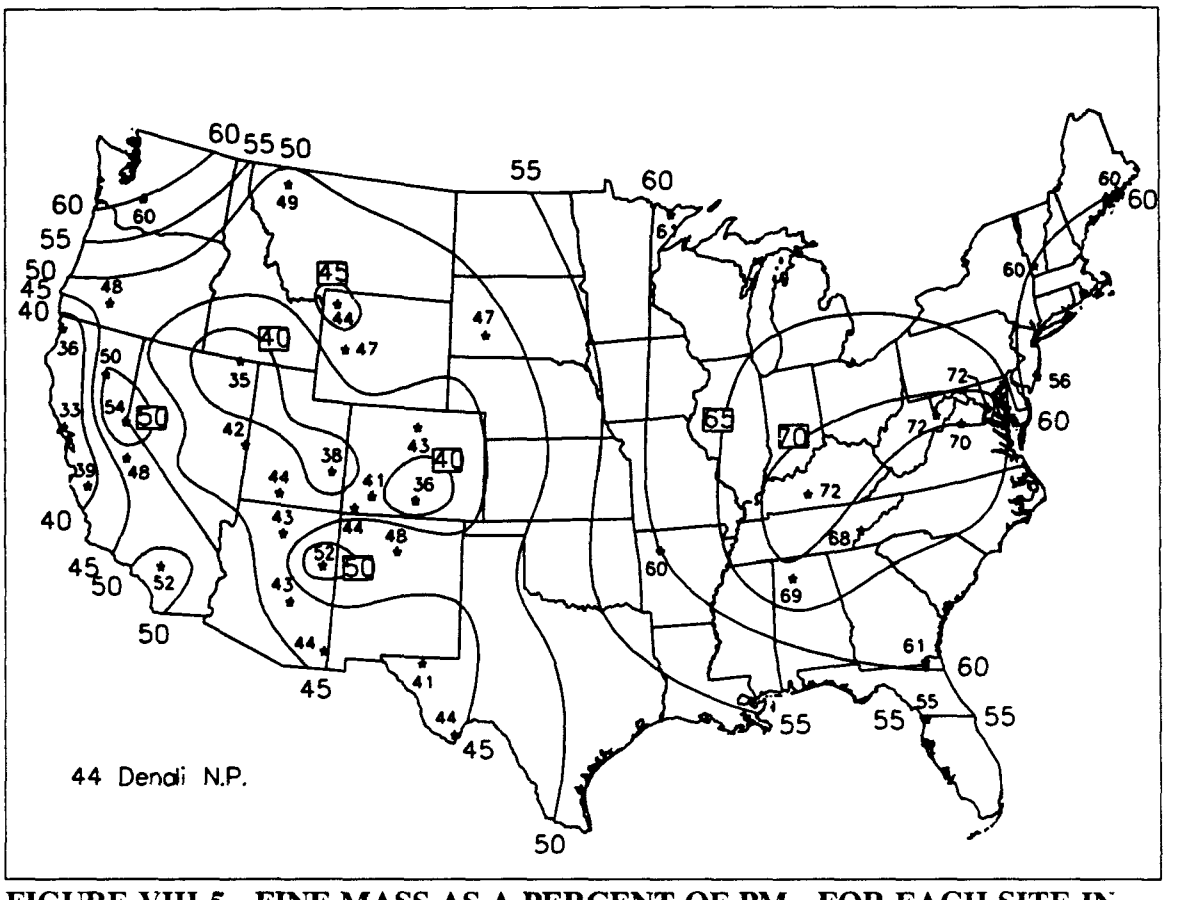


FIGURE VIII-5. FINE MASS AS A PERCENT OF PM_{10} FOR EACH SITE IN THE IMPROVE NETWORK, 1992-1995. (Sisler et al., 1996)

Figures VIII-6a, 6b, 7a, and 7b show the seasonal variability of visibility impairment, expressed in terms of the deciview. One can see that in the rural East, seasonal averages are generally highest in the summer, with values exceeding 30 deciview at Shenandoah National Park and the Sipsey Wilderness in Alabama, and they are generally lowest in the winter. In the Southwest, impairment is slightly higher in the summer and winter, ranging from 10-13 deciview. In the Northwest and northern Rockies, impairment is highest in the autumn and winter. The following subsections further explain significant reasons for the regional variability in visibility impairment.

a. Role of Humidity in Light Extinction

As mentioned previously, humidity plays a significant role in the impairment of visibility by fine particles, particularly in the East, where annual average relative humidity levels are 70-80% as compared to 50-60% in the West (Sisler et al., 1993). Table VIII-2 accounts for relative humidity effects by assigning a separate extinction efficiency for water associated with aerosols. Table VIII-3 illustrates the extinction efficiencies used in a 1996 analysis of data from the IMPROVE network. Total light extinction for sulfate and nitrate is calculated by multiplying the extinction efficiencies by a relative humidity correction factor.

TABLE VIII-3. DRY PARTICLE LIGHT EXTINCTION EFFICIENCY VALUES USED IN 1996 ANALYSIS OF IMPROVE DATA

Aerosol Constituent	Extinction Efficiency (in m^2/g)
Sulfates	3.0 *f(RH)*
Nitrates	3.0 *f(RH)*
Organics	4.0
Soil dust	1.0
Coarse particles	0.6
f(RH) is the relative humidity correction factor. It is the ratio of wet scattering divided by dry scattering.	

Source: Sisler et al., 1996

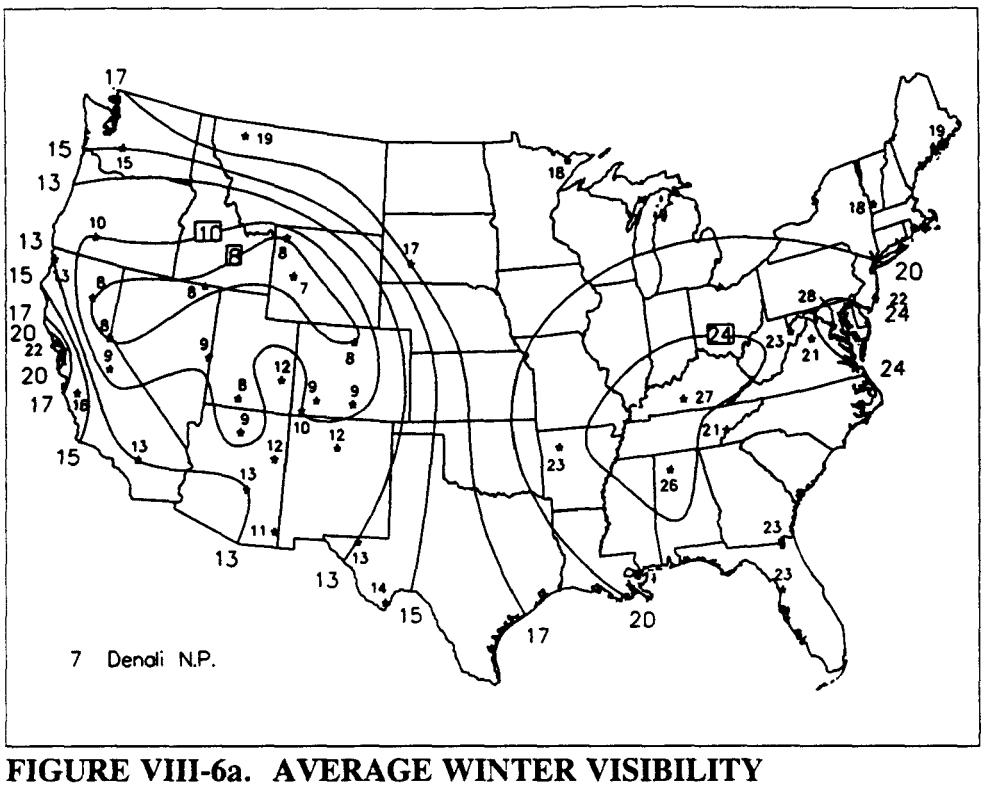


FIGURE VIII-6a. AVERAGE WINTER VISIBILITY IMPAIRMENT IN DECIVIEWS CALCULATED FROM TOTAL LIGHT EXTINCTION (RAYLEIGH INCLUDED), IMPROVE NETWORK, 1992-1995. (Sisler et al., 1996)

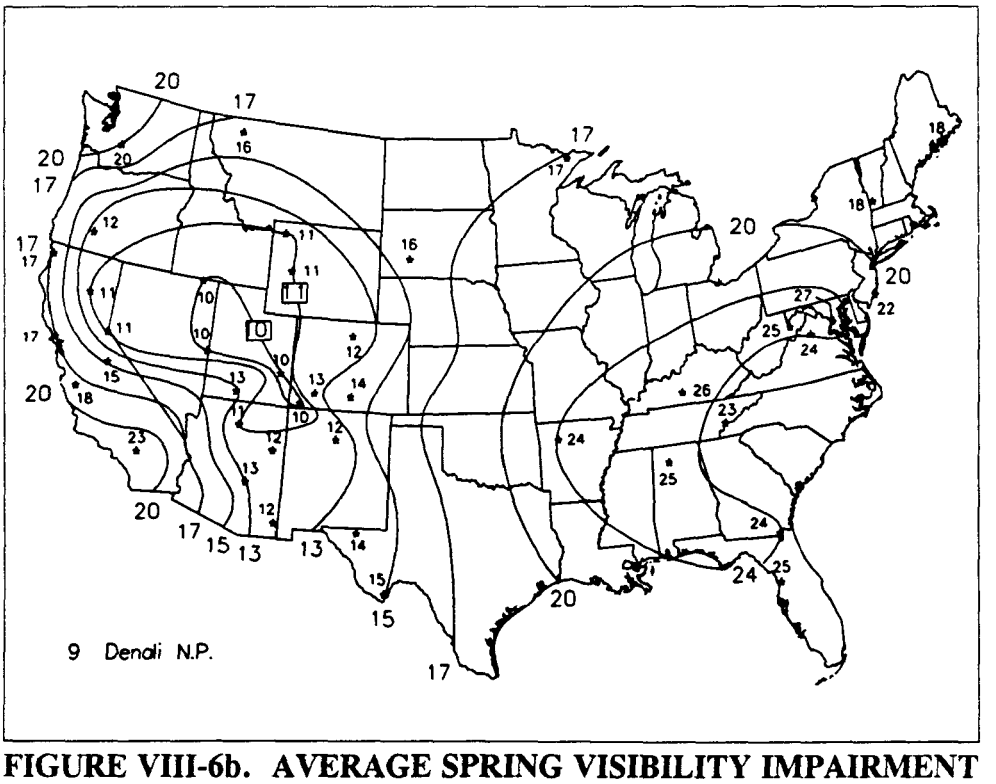


FIGURE VIII-6b. AVERAGE SPRING VISIBILITY IMPAIRMENT IN DECIVIEWS CALCULATED FROM TOTAL LIGHT EXTINCTION (RAYLEIGH INCLUDED), IMPROVE NETWORK, 1992-1995. (Sisler et al., 1996)

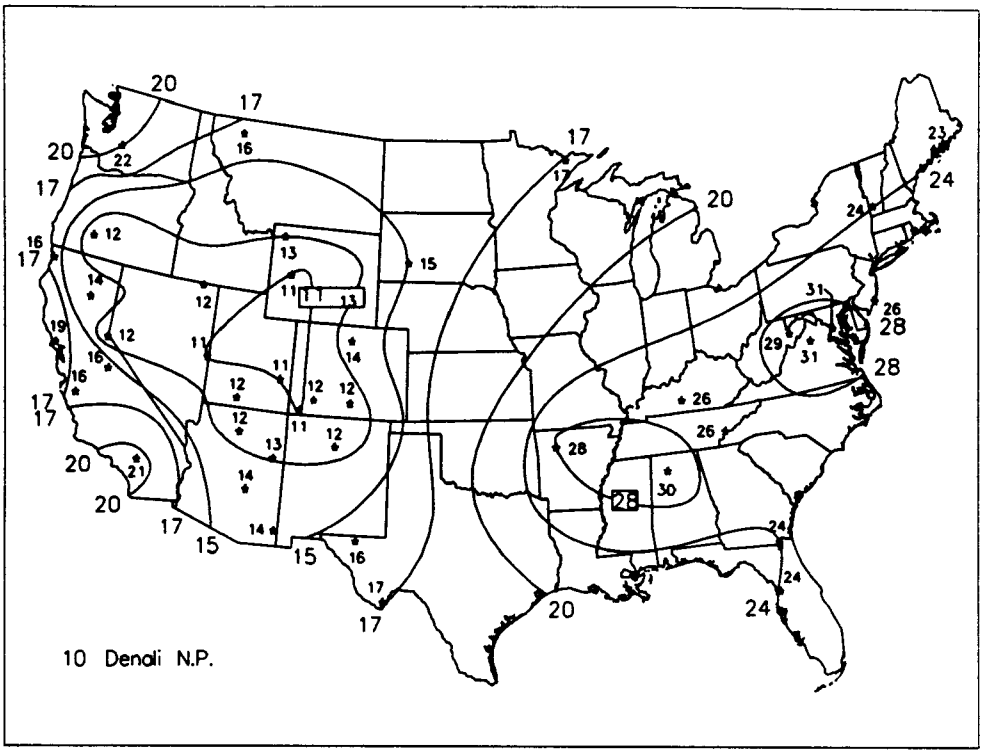


FIGURE VIII-7a. AVERAGE SUMMER VISIBILITY IMPAIRMENT IN DECIVIEWS CALCULATED FROM TOTAL LIGHT EXTINCTION (RAYLEIGH INCLUDED), IMPROVE NETWORK, 1992-1995. (Sisler et al., 1996)

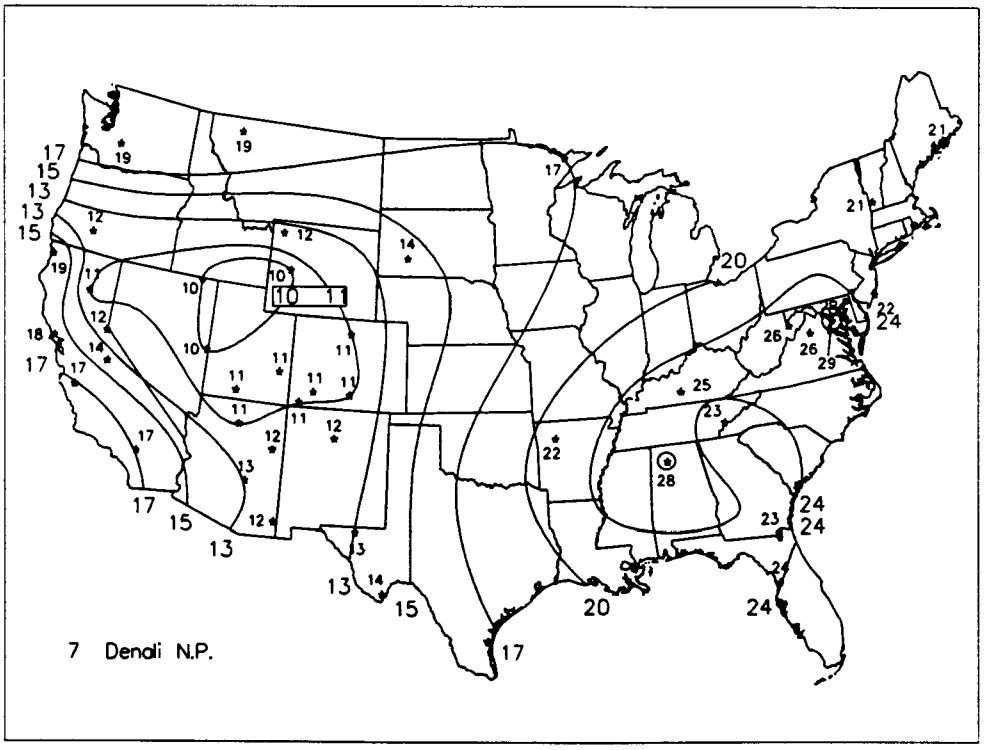


FIGURE VIII-7b. AVERAGE AUTUMN VISIBILITY IMPAIRMENT IN DECIVIEWS CALCULATED FROM TOTAL LIGHT EXTINCTION (RAYLEIGH INCLUDED), IMPROVE NETWORK, 1992-1995. (Sisler et al., 1996)

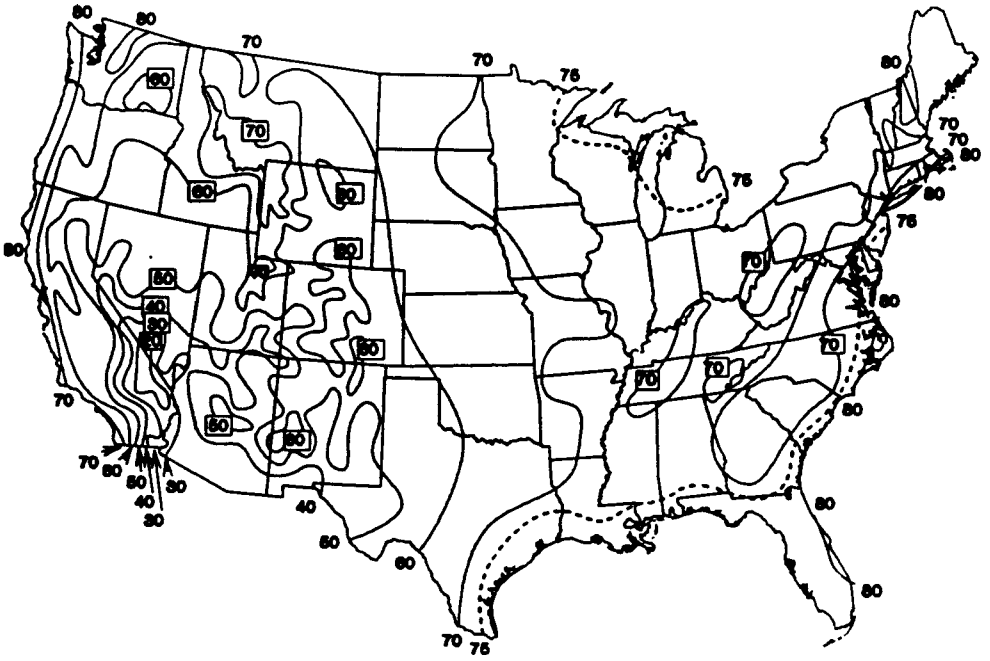
The correction factor represents 1) the hygroscopic nature of the aerosol constituent, and 2) the average annual humidity for the relevant location (Sisler et al., 1993). Light absorption by fine particles can be measured directly by the Laser Integrating Plate Method, or it can be estimated by multiplying elemental carbon mass by an extinction efficiency of 10 m^2/g (Sisler et al., 1996).

Because annual average relative humidity is higher in the East, the same ambient concentration of sulfate, for example, will on average lead to greater light extinction in an eastern location rather than a western one. The top map in figure VIII-8 illustrates the regional variability of annual mean relative humidity nationwide. The bottom map depicts the variability of the relative humidity correction factor used for sulfates in an analysis of IMPROVE data (Sisler et al., 1993). For example, when corrected for humidity, the overall extinction efficiency for sulfates in the East may exceed 11-12 m^2/g, whereas the extinction efficiency for sulfate in the West may be one-third to one-half of that.

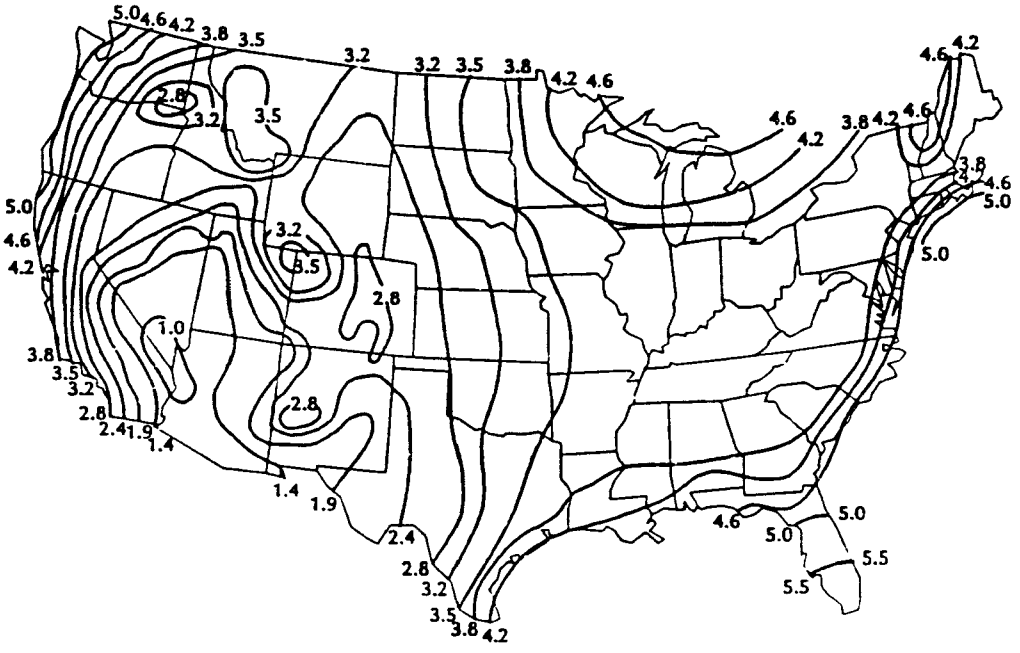
b. Significance of Anthropogenic Sources of Fine Particles

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

It is important to note that even in those areas with relatively low concentrations of anthropogenic fine particles, such as the Colorado plateau, small increases in anthropogenic fine particle concentrations can lead to significant decreases in visual range. This is one reason why Class I areas have been given special consideration under the Clean Air Act. This relationship is illustrated by figure VIII-9, which relates changes in fine particle



(a) Annual mean relative humidity.



(b) Sulfate relative humidity correction factor FT.

FIGURE VIII-8. SPATIAL VARIATION IN AVERAGE RELATIVE HUMIDITY (NOAA, 1978) AND THE SULFATE RELATIVE HUMIDITY CORRECTION FACTOR F_T. (Sisler et al., 1993)

TABLE VIII-4. COMPARISON OF TOTAL LIGHT EXTINCTION TO ESTIMATED BACKGROUND LIGHT EXTINCTION FOR SEVERAL EASTERN AND WESTERN LOCATIONS.

REGION	TOTAL LIGHT EXTINCTION 1988-1994 (in Mm^{-1})		VISUAL RANGE (in km)	
	Annual	Summer	Annual	Summer
Eastern U.S., estimated background light extinction	**26 +/- 7**	**NA**	**150+/- 45**	**NA**
Appalachian	126	182	31	21
Boundary Waters	62	63	63	62
Northeast	77	95	51	41
Washington, D.C.	177	207	22	19
Western U.S., estimated background light extinction	**17 +/- 2.5**	**NA**	**230 +/- 40**	**NA**
Colorado Plateau	32	33	122	119
Cascades	74	73	53	54
Southern California	74	87	53	45
Northern Rockies	57	48	69	82

Sources: Sisler et al., 1996; NAPAP 1991.

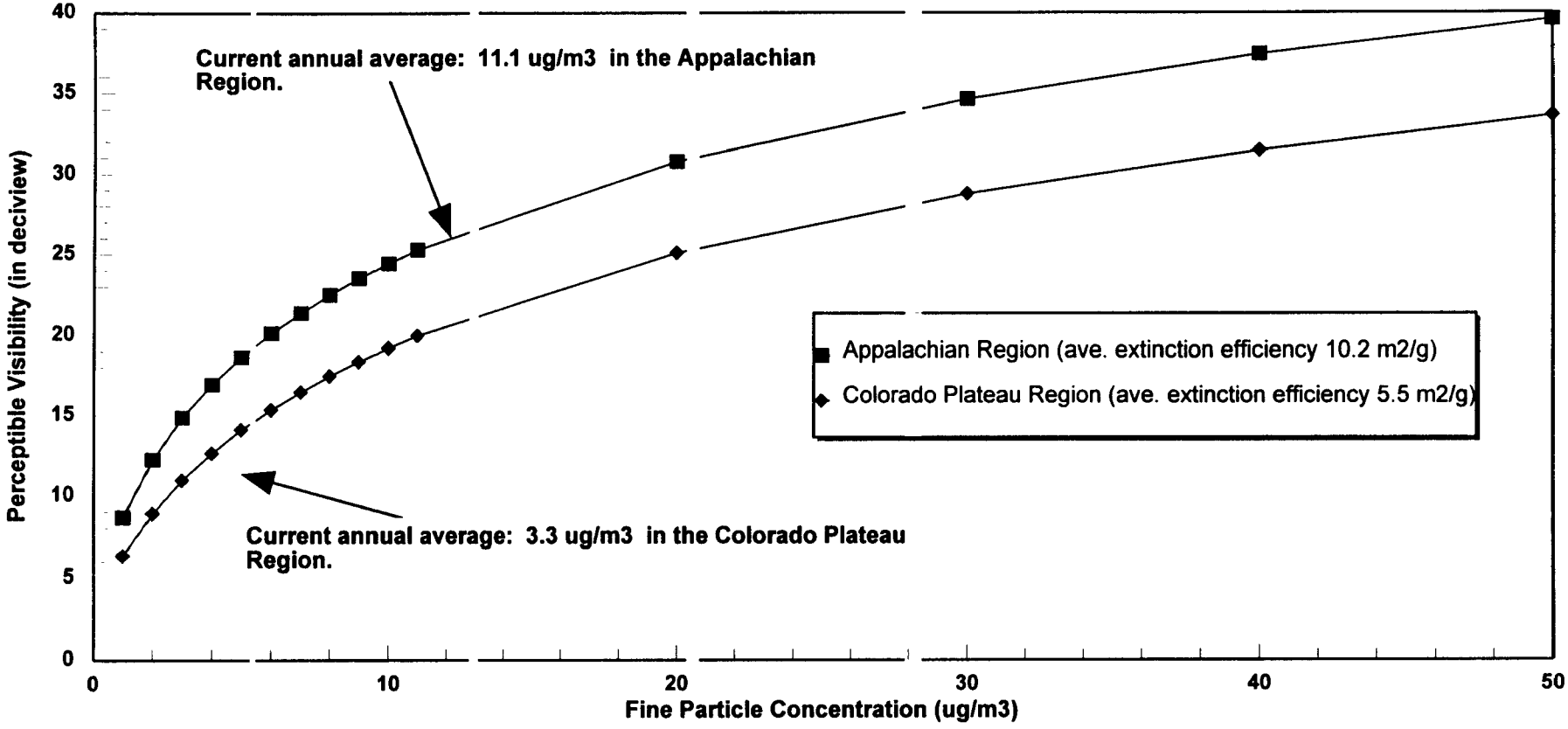


FIGURE VIII-9. PERCEPTIBLE CHANGE IN VISIBILITY AS A FUNCTION OF FINE MASS CONCENTRATION

Note: Average extinction efficiencies are calculated from IMPROVE monitoring program data, March 1988 - February 1994. Changes in total fine particle concentration reflect current mix of constituents.

Appalachian region: Great Smokies, Shenandoah, Dolly Sods.
Colorado Plateau region: Grand Canyon, Bryce Canyon, Canyonlands, Mesa Verde, Bandelier, Petrified Forest.

Under many circumstances, a change of one deciview represents a change perceptible by the average person.

concentrations to perceptible changes in visibility (represented by the deciview metric). The graph shows that in cleaner areas, such as the West, perceptible visibility changes are more sensitive to existing fine particle concentrations than is the case in more polluted areas. In other words, to achieve a given amount of perceived 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 lower concentration areas. This figure 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 (expressed in m^2/g in figure VIII-9) 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 in the Colorado Plateau.

c. Regional Differences in Specific Pollutant Concentrations

As total light extinction levels vary significantly across the country, so does the mix of visibility-impairing pollutants from region to region. Table VIII-5, taken from the 1993 National Research Council study on visibility, shows the estimated contribution of various anthropogenic pollutants to visibility impairment for three main regions of the U.S. The table takes into account relative emissions levels of each pollutant type within each region. This and other analyses (Sisler et al., 1993) show that sulfates are a significant cause of visibility impairment in all parts of the country, but particularly in the East, where they are responsible for about two-thirds of overall light extinction. In the Southwest and Northwest, organics play a larger role, as does elemental carbon. Suspended dust is also a major constituent in the Southwest. The main categories of sources responsible for visibility-impairing fine particle and precursor emissions are listed in table VIII-6 (NRC, 1993).

d. Regional Variation in Urban Visibility

Visibility impairment has been studied in several major cities in the past decade (e.g. Middleton, 1993) because of concerns about fine particles and their potentially significant impacts (e.g., health-related and aesthetic) on the residents of large metropolitan areas. Urban areas generally have higher loadings of fine particulate matter than monitored Class I

TABLE VIII-5. VISIBILITY MODEL RESULTS: ANTHROPOGENIC LIGHT EXTINCTION BUDGETS[a]

	East[b]	Southwest[c]	Northwest[d]
Sulfates	65	39	33
Organics	14	18	28
Elemental carbon	11	14	15
Suspended dust	2	15	7
Nitrates	5	9	13
Nitrogen dioxide	3	5	4

[a]Percentage contribution by specific pollutant to anthropogenic light extinction in three regions of the United States.

[b]Based on Table 9, Table 18, Figure 45, Appendix A, and Appendix E of NAPAP Visibility SOS/T Report (Trijonis et al., 1990). It is assumed that sulfates (3% natural) account for 60% of non-Rayleigh extinction, organics (33% natural) account for 18%, elemental carbon (3% natural) accounts for 10%, suspended dust (50% natural) accounts for 4%, nitrates (10% natural) account for 5%, and nitrogen dioxide (10% natural) accounts for 3%.

[c]Based on Table 9, Table 18, Figure 45, Appendix A, and Appendix E of the NAPAP Visibility SOS/T Report (Trijonis et al., 1990). It is assumed that sulfates (10% natural) account for 33% of non-Rayleigh extinction, organics (33% natural) account for 20%, elemental carbon (10% natural) accounts for 12%, suspended dust (50% natural) accounts for 23%, nitrates (105 natural) account for 8%, and nitrogen dioxide (10% natural) accounts for 4%.

[d]Extinction efficiencies (relative to organics are chosen as 1.5 for sulfates, 2.5 for elemental carbon, 0.3 for fine crustal materials, and 1.5 for nitrates (Trijonis et al., 1988, 1990). Coarse dust extinction is assumed to be three times fine dust extinction (Trijonis et al., 1988, 1990). Natural aerosol particle fractions are assumed to be one-tenth for sulfates, one-third for organics one-tenth for elemental carbon, one-half for crustal materials, and one-tenth for nitrates. These assumptions are applied using the fine mass concentrations in Trijonis et al., (1990). The percentage contribution for nitrogen dioxide is assumed to be 4%.

Source: NRC, 1993.

TABLE VIII-6. PERCENTAGE CONTRIBUTION BY SOURCE CATEGORY TO FINE PARTICLE (AND PRECURSOR) EMISSIONS IN THE EAST, SOUTHWEST, AND NORTHWEST

EAST	SOx	Organic Particles	VOC's	Elemental Carbon	Suspended Dust	NH3	NOx
Electric utilities	78.0	--	--	--	--	--	39
Diesel-fueled mobile sources	1.5	--	--	47	--	--	16
Gasoline vehicles	1.0	34	31	29	--	--	26
Petroleum and chemical industries	4.5	--	11	--	--	--	--
Industrial coal combustion	7.0	--	--	--	--	--	--
Residential wood burning	--	20	13	15	--	--	--
Fugitive dust (on-road/off-road traffic)	--	--	--	--	100	--	--
Feedlots and livestock waste mgmt.	--	--	--	--	--	66	--
Miscellaneous	8.0	46	45	9	--	34	19
SOUTHWEST	**SOx**	**Organic Particles**	**VOC's**	**Elemental Carbon**	**Suspended Dust**	**NH3**	**NOx**
Electric utilities	33	--	--	--	--	--	19
Diesel-fueled mobile sources	12	5	--	52	--	--	23
Gasoline vehicles	5	38	42	31	--	--	32
Petroleum and chemical industries	22	--	12	--	--	--	--
Copper smelters	19	--	--	--	--	--	--
Fugitive dust (on-road/off-road traffic)	--	--	--	--	100	--	--
Residential wood burning	--	8	5	6	--	--	--
Feedlots and livestock waste mgmt.	--	--	--	--	--	75	--
Miscellaneous	9	49	41	11	--	25	26
NORTHWEST	**SOx**	**Organic Particles**	**VOC's**	**Elemental Carbon**	**Suspended Dust**	**NH3**	**NOx**
Electric utilities	30	--	--	--	--	--	8
Diesel-fueled mobile sources	12	--	--	37	--	--	29
Gasoline vehicles	4	15	31	16	--	--	36
Petroleum and chemical industries	19	--	10	--	--	--	--
Residential wood burning	--	22	25	22	--	--	--
Forest management burning	--	45	13	20	--	--	--
Fugitive dust (on-road/off-road traffic)	--	--	--	--	100	--	--
Feedlots and livestock waste mgmt.	--	--	--	--	--	81	--
Primary metallurgical process	8	--	15	--	--	--	--
Organic solvent evaporation	--	--	15	--	--	--	--
Miscellaneous	27	18	6	5	--	19	27

Source: NRC, 1993.

areas, suggesting that visibility impairment in urban areas is typically greater than in rural areas. Monitored annual mean and second highest maximum 24-hour fine particle levels for selected urban areas are listed in Table IV-4. These levels are generally higher than those found in the IMPROVE database for rural Class I areas.

The degree to which different aerosol constituents contribute to overall light extinction in urban areas can vary significantly. Table VIII-7 illustrates the difference between percentage contributions of aerosol constituents to annual average total light extinction in the Washington, DC urban area and the southern California areas. The dominance of sulfate in Washington, DC exhibits a regional effect stemming from sulfur dioxide emissions outside

TABLE VIII-7. PERCENTAGE CONTRIBUTIONS OF AEROSOL CONSTITUENTS TO ANNUAL AVERAGE TOTAL LIGHT EXTINCTION IN THE WASHINGTON, DC AND SOUTHERN CALIFORNIA AREAS.

Location	Sulfate	Nitrate	Organics	Elemental Carbon	Soil and Coarse
Wash, DC	49	16	16	12	7
Southern Calif.	14	44	18	9	14

Source: Sisler et al., 1993

the metropolitan area. In contrast, nitrate plays the greatest role in the overall light extinction levels in the mountainous areas just outside Los Angeles, with most of the nitrate formation in this area coming from nitrogen dioxide emissions within the urban area.

6. Policy Considerations Pertaining to the Effects of PM on Visibility

Impairment of visibility in multi-state regions, urban areas, and Class I areas is clearly an effect of particulate matter on public welfare. The staff has considered a number of factors in assessing appropriate regulatory responses.

An initial question is whether the range of recommended primary standards for fine PM would provide adequate protection against visibility impairment across the country. The

range being considered for an annual PM-fine standard is 12.5 $\mu g/m^3$ to less than 20 $\mu g/m^3$ and the range under consideration for a 24-hour standard is 18 $\mu g/m^3$ to less than 65 $\mu g/m^3$. Table IV-4 presents monitored fine particle annual averages and second highest maximum levels for several major U.S. cities. Analysis of these data suggests that adoption of an annual fine particle standard in the lower half of the recommended range, in combination with adoption of a 24-hour standard in the lower half of the recommended range, would be expected to lead to reductions in annual average fine particle concentrations in many urban areas nationally. Additionally, reductions could be achieved in broader areas in the East if regional attainment strategies are carried out. To examine expected regional visibility improvements resulting from these reductions requires an understanding of the various factors affecting the relationship between fine particle loadings and visibility, such as background levels, humidity, and pollutant mix, as described in section 5 above.

Expected reductions in fine particle concentrations resulting from adoption of the primary fine particle standards in the lower half of the recommended range is likely to result in maintained or improved visibility in many urban areas and in a broader area in the East. As with reductions in fine particle concentrations noted above, improvement of visibility would be greater if regional fine particle attainment strategies are carried out. In its 1993 Report to Congress on the effects of Clean Air Act programs on visibility in mandatory federal Class I areas, EPA examined the impact of expected regional sulfur dioxide reductions under the acid rain program (U. S. EPA, 1993). This report estimated that regional annual average sulfate levels would be reduced over a wide area in the eastern U.S. by the year 2010, resulting in potential improvements in visibility for the region. The analysis projected no expected improvement in the rural West. Moreover, despite projected improvements in visibility, there is no evidence that adoption of the primary fine particle standards in the lower half of the recommended range will eliminate visibility impairment.

The staff has also considered whether the adoption of a national secondary standard would provide adequate and appropriate protection of public welfare across the country. Due to the regional variability in visibility conditions created by background fine particle levels and humidity, the staff has concluded that a national secondary standard would not be the most appropriate means to achieve this objective. The data presented in table VIII-4

indicates that current annual average light extinction levels on the Colorado Plateau (reflecting effects of anthropogenic and background sources of PM) are about equal to background levels (i.e., those levels representing an absence of anthropogenic contributions) in the East. Thus, a national secondary standard set to maintain or improve visibility conditions on the Colorado Plateau would have to be set at or below natural background levels in the East, effectively requiring elimination of all anthropogenic (and some nonanthropogenic) emissions. Conversely, a national secondary standard that would be both attainable and improve visibility in the East would permit further degradation in the West.

An approach which would be more responsive to visibility protection goals, while recognizing these significant regional variations, would be to establish a regional haze program under section 169A of the Clean Air Act. This program, while designed to address the existing adverse effects of fine particles on visibility in Class I areas, would further contribute to visibility improvement in non-Class I areas as well. Section 169A established a national goal of "the prevention of any future, and the remedying of any existing, manmade impairment of visibility in mandatory Class I areas." The EPA is required to establish programs to ensure reasonable progress toward the national goal. These programs are to be implemented by the States and can be regionally specific. Concern with regional visibility impacts to highly valued national parks and wilderness areas in the U.S. led to the inclusion of specific language in section 169B of the 1990 Clean Air Act Amendments, requiring EPA to form the Grand Canyon Visibility Transport Commission. In June 1996, the Commission provided the Administrator with recommendations for regional approaches to protecting visibility. The work of the Commission will be useful to development of a regional haze program under section 169A of the Act.

Much progress has been made in technical areas important to the successful implementation of a regional haze program, including areas such as visibility monitoring, regional scale modeling, and scientific knowledge of the regional effects of particles on visibility. The National Academy of Sciences 1993 report on visibility protection confirmed this point:

> Current scientific knowledge is adequate and control technologies are available for taking regulatory action to improve and protect visibility. However,

> continued national progress toward this goal will require a greater commitment toward atmospheric research, monitoring, and emissions control research and development.

In addition, as noted above, it is expected that the development of a regional haze program would have associated benefits outside of mandatory Class I areas. The National Academy of Sciences concluded the following:

> Efforts to improve visibility in Class I areas also would benefit visibility outside these areas. Because most visibility impairment is regional in scale, the same haze that degrades visibility within or looking out from a national park also degrades visibility outside it. Class I areas cannot be regarded as potential islands of clean air in a polluted sea.

Based on the above considerations, the staff recommends that the Administrator consider establishing a regional haze program under section 169A of the Act, in conjunction with the recommended fine particle primary standards, as the most effective means of addressing the welfare effects associated with visibility impairment. Together, the two programs and associated control strategies should adequately protect against the effects of fine particle pollutants on visibility and make reasonable progress toward the national visibility goal for Class I areas.

C. Effects of PM on Materials Damage and Soiling

The deposition of airborne particles can become a nuisance, reducing the aesthetic appeal of buildings and culturally important articles through soiling, and contribute directly (or in conjunction with other pollutants) to structural damage by means of corrosion or erosion. These potential effects are discussed more fully below. The relative importance of particle size, composition, and other environmental factors (i.e., moisture, temperature, sunlight, and wind) in contributing to the effects is also considered.

1. Materials Damage

Particles affect materials principally by promoting and accelerating the corrosion of metals, by degrading paints, and by deteriorating building materials such as concrete and limestone. Particles contribute to these effects because of their electrolytic, hygroscopic, and acidic properties, and their ability to sorb corrosive gases (principally sulfur dioxide). The

staff review suggests that only chemically active fine mode or hygroscopic coarse mode (mainly sea or road salt) particles contribute to such effects (U.S. EPA, 1986b). While particles have been qualitatively associated with damage to materials, there are insufficient data at present to relate such effects to specific particle pollution levels. The following discussion briefly outlines the available information on PM-related effects associated with each category of material presented in the criteria document.

a. Effects on Metals

The rate of metal corrosion depends on a number of factors, including the deposition rate and nature of the pollutant; the influence of the metal protective corrosion film; the amount of moisture present; variability in the electrochemical reactions; the presence and concentration of other surface electrolytes; and the orientation of the metal surface (CD, Chapter 9). This section briefly discusses the factors affecting metal corrosion set forth in the criteria document.

Nriagu (1978) and Sydberger (1977) conducted studies that highlighted the ability metals have to form a protective film that slows corrosion rates. Metals initially exposed to low concentrations of SO_x corroded at a slower rate than did samples continuously exposed to higher concentrations. This protective corrosion layer may, however, be affected by either dry or wet deposition (CD, Chapter 9).

The rate of metal corrosion decreases in the absence of moisture (CD, Chapter 9). Moisture influences corrosion rates by providing a medium of conduction paths for electrochemical reactions and a medium for water soluble air pollutants. Schwartz (1972) established that the corrosion rate of a metal could increase by 20 percent for each one percent increase in relative humidity above the minimum atmospheric moisture content that allows corrosion to occur (i.e., critical relative humidity). Later studies by Haynie and Upham (1974) and Sydberger and Ericsson (1977) supported Schwartz's theory.

While particles alone have some effect on the early stages of metal corrosion, there is insufficient evidence to relate such effects to specific particle levels. One study (Goodwin et al. (1969)) reported damage to steel, protected with nylon screen, exposed to quartz particles larger than 5 μm; but the exposure time and concentration were not reported. Barton (1958) also found that dust contributed to the early stages of metal corrosion. A number of the

studies evaluated concluded that particulate matter increased the corrosion rate of sulfur dioxides (Sanyal and Singhania, (1956); Yocom and Grappone, (1976); Johnson et al., (1977); Russell, (1976); Walton et al., (1982)). Laboratory studies show mixed results as to whether catalytic species or conductance of the thin-film surface electrolyte is the cause of the increases in corrosion rates (Walton et al., 1982; Skerry et al., 1988 a,b; Askey et al., 1993).

Results of actual field studies have not established a quantitative relationship between particles and corrosion. Thus, the independent effect of particles is not evident since SO_2 is the controlling factor for determining corrosion rate (U.S. EPA, 1986b). Edney et al. (1989) exposed galvanized steel panels to actual field conditions in Research Triangle Park, NC and Steubenville, OH between April 25 and December 28, 1987. The panels were exposed under the following conditions: (1) dry deposition only; (2) dry plus ambient wet deposition; and (3) dry deposition plus deionized water. The average concentrations for SO_2 and particulate matter was 22 ppb and 70 $\mu g/m^3$ and < 1 ppb and 32 $\mu g/m^3$ for Steubenville and Research Triangle Park, respectively. The runoff from the steel panel was analyzed and it was concluded that the dissolution of the steel corrosion products for both sites was likely the result of deposited gas phase SO_2 on the metal surface and not particulate matter.
Another study conducted by Butlin et al. (1992) also demonstrated that the corrosion of mild steel and galvanized steel was SO_2-dependent. Butlin et al. monitored the corrosion of steel samples by SO_2 and ozone under artificially fumigated environments, and NO_2 under natural conditions. Annual average SO_2 concentrations ranged from 2.1 $\mu g/m^3$ in a rural area to 60 $\mu g/m^3$ in one of the SO_2-fumigated locations. Annual average NO_2 concentrations ranged from 1.5 to 61.8 $\mu g/m^3$. The study concluded that corrosion of the steel samples was primarily dependent on the long-term SO_2 concentration and was only minimally affected by nitrogen oxides.

b. Effects on Paint

Paints undergo natural weathering processes from exposure to environmental factors such as sunlight, moisture, fungi, and varying temperatures. In addition to the natural environmental factors, studies show particulate matter exposure may give painted surfaces a dirty appearance (CD, Chapter 9). Several studies also suggest that particles serve as

carriers of other more corrosive pollutants, allowing the pollutants to reach the underlying surface or serve as concentration sites for other pollutants (Cowling and Roberts, 1954).

A number of studies have shown some correlation between particulate matter and damage to automobile finishes. Fochtman and Langer (1957) reported damage to automobile finishes due to iron particles emitted from nearby industrial facilities. General Motors conducted field tests in Jacksonville, Florida to determine the effect of various meteorological events, the chemical composition of rain and dew, and the ambient air composition during the event, on automotive paint finishes. Painted (basecoat/clearcoat technology) steel panels were exposed for varying time periods, under protected and unprotected condition. The researcher concluded that calcium sulfate formed on the painted surface by the reaction of calcium from dust and sulfuric acid contained in rain or dew. The damage to the paint finish increased with increasing days of exposure (Wolff et al., 1990).

Paint films permeable to water are also susceptible to penetration by acid forming aerosols (U.S. EPA, 1995). Baedecker et al. (1991) reviewed studies dealing with solubility and permeability of SO_2 in paints and polymer films. These studies showed permeation and absorption rates varied depending on the formulation of the paint.

Studies reported in the criteria document (Spence et al., (1975); Campbell et al., (1974); Haynie and Spence, (1984); Yocom and Grappone, (1976); and Yocom and Upham, (1977)) support the conclusion that gaseous pollutants contribute to the erosion rates of exterior paints.

c. Effects on Stone

Damage to calcareous stones (i.e., limestone, marble and carbonated cemented stone) has been attributed to deposition of acidic particles. Moisture and salts are considered the most important factors in building material damage (CD, Chapter 9). However, many other factors (such as normal weathering and microorganism damage) also seem to play a part in the deterioration of inorganic building materials. The relative importance of biological, chemical, and physical mechanisms has not been studied to date. Thus, the relative contribution of ambient pollutants to the damage observed in various building stone is not well quantified.

Baedecker et al. (1991) reported that 10 percent of chemical weathering of marble and limestone was caused by wet deposition of hydrogen ions from all acid species. Dry deposition of SO_2 between rain events caused 5 to 20 percent of the chemical erosion of stone, and dry deposition of nitric acid was responsible for 2 to 6 percent of the erosion (Baedecker et al., 1991). Under high wind conditions, particulates result in slow erosion of the surfaces, similar to sandblasting (Yocom and Upham, 1977).

d. Effects on Electronics

Exposure to ionic dust particles can contribute significantly to the corrosion rate of electronic devices, ultimately leading to failure. Particles derived from both natural and anthropogenic sources and ranging in size from tens of angstroms to one μm can cause corrosion of electronics because many are sufficiently hygroscopic and corrosive, at normal relative humidities, to react directly with non-noble metal and passive oxides, or to form conductive moisture films on insulating surfaces to cause electrical leakage. The effects of particles on electronic components were first reported by telephone companies who reported that particles high in nitrates caused corrosion, cracking, and ultimate failure of wire spring relays (Hermance, 1966; McKinney and Hermance, 1969). More recently, Sinclare (1992) and Frankenthal (1993) have reported that anthropogenically-derived particles penetrating into indoor environments can contribute to the corrosion of electronics.

2. Staff Considerations Pertaining to the Effects of PM on Materials Damage

While particles, particularly in conjunction with sulfur dioxide, have been qualitatively associated with damage to materials, there is insufficient data available to relate such damage to specific particle levels in the ambient air. Absent better quantitative data, the staff does not believe the Administrator should consider a separate secondary standard based on materials damage.

3. Soiling

Soiling is the accumulation of particles on the surface of an exposed material resulting in the degradation of its appearance. When such accumulation produces sufficient changes in reflection from opaque surfaces and reduces light transmission through transparent materials, the surface will become perceptibly dirty to the human observer. Soiling can be remedied by cleaning or washing, and depending on the soiled material, repainting.

Determination of what accumulated level of particulate matter leads to increased cleaning or repainting is difficult. For example, Carey (1959) found that the appearance of soiling only occurred when the surface of paper was covered with dust specks spaced 10 to 20 diameters apart. When the contrast was strong, e.g., black on white, it was possible to distinguish a clean surface from a surrounding dirty surface when only 0.2 percent of the areas was covered with specks, while 0.4 percent of the surface had to be covered with specks with a weaker color contrast.

Hancock et al. (1976) found that with maximum contrast, a 0.2 percent surface coverage (effective area coverage; EAC) by dust can be perceived against a clean background. A dust deposition level of 0.7 percent EAC was needed before the object was considered unfit for use. The minimum perceivable difference between varying gradations of shading was a change of about 0.45 percent EAC. Using the information on visually perceived dust accumulation, Hancock et al. (1976) concluded that dustfall rates of less than 0.17 EAC/day would be tolerable to the general public. Similar studies have not been reported for other soiling effects.

Despite the observation that airborne particles soil a wide range of man-made materials, there is only limited information available with respect to size and composition of the culpable particles. In general, the soiling of fabrics and vertical surfaces has been ascribed to fine particles, particularly dark, carbonaceous materials. Soiling of horizontal surfaces may result from deposition of a wide range of particles, including coarse mode dusts.

An important consideration in assessing soiling potential is deposition velocity, which is defined as flux divided by concentration. Deposition velocity is a function of particle diameter, surface orientation and roughness, wind speed, atmospheric stability, and particle density. As a result, soiling is expected to vary with the size distribution of particles within an ambient concentration, whether the surface is positioned horizontally or vertically, and whether the surface is rough or smooth (CD, Chapter 9).

Theoretically, coverage of horizontal surfaces will be related to particle surface areas and deposition velocity. Particle surface areas per unit mass decreases linearly with diameter (assuming spherical particles), while, under quiescent conditions, deposition velocity

increases with the square of the diameter. Under such conditions, large particles would result in more soiling than an equivalent mass of smaller particles. Although second order effects may enhance fine particle deposition relative to larger particles, deposition velocity data still suggest substantially higher deposition on horizontal surfaces for particles larger than 10 μm than for smaller particles (U.S. EPA, 1982b).

The increasing soiling potential associated with increased particle size is mitigated by lighter particle color, effects of rainfall, smaller transport distance from sources and markedly lower penetration of larger particles to indoor surfaces (relative to smaller particles). Because these conflicting factors have not been fully evaluated, it is not possible to make clear particle size divisions with respect to soiling of horizontal surfaces.

The time interval that it takes to transform horizontal and vertical surfaces from clean to perceptibly dirty is generally determined by particle composition and rate of deposition. The process is influenced by the location (sheltered or unsheltered) and spatial alignment of the material, the texture and color of the surface relative to the particles, and meteorological variables such as moisture, temperature, and wind speed.

Haynie and Lemmons (1990) conducted a soiling study in a relatively rural environment in Research Triangle Park, North Carolina. The study was designed to determine how various environmental factors contribute to the rate of soiling of white painted surfaces, which are highly sensitive to soiling by dark particles and represent a large fraction of all man-made surfaces exposed in the environment. Hourly rainfall and wind speed, and weekly data for dichotomous sampler measurements and TSP concentration were monitored. Gloss and flat white paints were applied to hardboard house siding surfaces and exposed vertically and horizontally for 16 weeks, either sheltered or unsheltered from rainfall. Measurements, including reflectance, were taken at 2, 4, 8, and 16 weeks. Based on the results of this study, the authors concluded that: (1) coarse mode particles initially contribute more to soiling of both horizontal and vertical surfaces than fine mode particles; (2) coarse mode particles, however, are more easily removed by rain than are fine mode particles; (3) for sheltered surfaces, reflectance changes are proportional to surface coverage by particles, and particle accumulation is consistent with deposition theory; (4) rain interacts with particles to contribute to soiling by dissolving or desegregating particles and leaving stains; and (5)

very long-term remedial actions are probably taken because of the accumulation of fine rather than coarse particles (Haynie and Lemmons, 1990).

Creighton et al. (1990) reported that horizontal surfaces soiled faster than vertical surfaces and that large particles were primarily responsible for the soiling of horizontal surfaces not exposed to rainfall. Soiling was related to the accumulated mass of particles from both the fine and coarse fraction. Fine mode black smoke and motor vehicle exhaust have been associated with the soiling of building material and facades (Tarrat and Joumard, 1990; Lanting, 1986).

Ligocki et al. (1993) studied the potential soiling of art work in five Southern California museums. The authors concluded that a significant fraction of fine elemental carbon and soil dust particles had penetrated to the indoor atmosphere of the museums studied and may constitute a soiling hazard to displayed art work. The seasonally averaged indoor/outdoor ratios for particulate matter mass concentrations ranged from 0.16 to 0.96 for fine particles and from 0.06 to 0.53 for coarse particles, with lower values observed for building with sophisticated ventilation systems that include filters for particulate removal.

4. Societal Costs

a. Soiling/Property Value

The effect of particles on aesthetic quality depends in part on human perception of pollution. The reduction of aesthetic quality may arise from the soiling of buildings or other objects of historical or social interest from the mere dirty appearance of a neighborhood. A number of studies have indicated that such perceptions of neighborhood degradation are revealed indirectly through effects on the value of residential property. That is, when residential properties similar in other respects are compared, the properties in the more highly polluted areas typically have lower value.

Freeman (1979), reporting on 14 property value studies that used particulate matter or dustfall as one of their pollutant measures, noted that the results generally supported the premise that property values are affected by the full range of particle pollution. He cautioned, however, that direct comparison of the monetary results is not possible since the studies cover a number of cities and use different data bases, empirical techniques, and model specifications.

The extent to which the city-specific results represent soiling as opposed to perceptions of the effects of particles on health and visibility is not clear. Therefore, the results of these studies cannot provide reliable quantitative estimates of the effects of soiling on property values (U.S. EPA, 1982b).

b. Soiling/Materials

Airborne particles soil a wide range of materials in all sectors of the economy. Assuming that these sectors are not as well off in a dirtier state as a cleaner one, soiling will result in an economic cost to society. While the household sector has been examined by a number of investigators, their results have been questioned because of methodology problems and their failure to appropriately address particle size, composition, and deposition rates. As a result, no single study has produced a completely satisfactory estimate of soiling costs for the household sector. It is unfortunate that little or no effort has been expended to account for soiling costs in the commercial, manufacturing, or public sectors. Results from MathTech, Inc. (1983) suggest that soiling costs for the manufacturing sector alone could be significant.

In the review of effects of household soiling, the staff paper has relied principally on Booz, Allen and Hamilton, Inc., (1970); Watson and Jaksch, (1978, 1982) [which was cited in the CD and discussed in more detail in the 1982 criteria document]; and MathTech, Inc., (1983) to derive estimates of household soiling costs. For the year 1970, the estimate for amenity loss due to exterior household soiling was estimated to range form 1 to 3.5 billion dollars (1978 dollars). The 14 $\mu g/m^3$ reduction in U.S. annual TSP levels between 1970 and 1978 was estimated to have resulted in an annual benefit for the year 1978 of 0.2 to 0.7 billion dollars or 14 to 50 million dollars for each $\mu g/m^3$ of reduction (U.S. EPA, 1982a). MathTech, Inc. (1983) estimated household soiling costs in the range of $88.3 million to $1.2 billion (1980 dollars) for attaining the primary PM_{10} standard nationwide. Gilbert (1985) used a household production function framework to design and estimate the short-run costs of soiling. The results were comparable to those reported by MathTech (1983). Finally, McClelland et al. (1991) concluded that households were willing to pay $2.70 per $\mu g/m^3$ change in particle level to avoid soiling effects.

Haynie (1989), using fine and coarse mode particle levels calculated from 1987 EPA AIRS data for PM_{10} and TSP, estimated that $1.74 billion of annual national residential repainting costs could be attributed to soiling (using national average painting costs and frequencies). Haynie and Lemmons (1990) estimated that the national soiling costs associated with repainting the exterior walls of houses probably were within the range of $400 to $800 million a year in 1990. This lower estimate, as compared to Haynie (1989), reflects that households in dirtier areas may not respond with average behavior but mitigate their behavior by (1) accepting greater reductions in reflectance before repainting, (2) washing surfaces rather than painting as often, or (3) selecting materials or paint colors that do not tend to show dirt. Haynie and Lemmons (1990) extrapolated their findings for houses to all exterior paint surfaces and produced a range from $570 to $1,140 million per year.

5. Staff Considerations Pertaining to the Effects of PM on Soiling

It is clear that, at high enough concentrations, particles become a nuisance and result in increased cost and decreased enjoyment of the environment. The available data are limited, however, and do not permit any definitive findings with respect to societal costs or provide clear quantitative relationships between ambient particle loading and soiling. Absent sufficient data, the staff concludes that there is not a sufficient basis to set a separate secondary standard based on soiling effects alone. The recommended suite of primary ambient air quality standards and the regional haze program should reduce the soiling and nuisance effects associated with particle pollution. The effects associated with dustfall are likely to be very localized and thus, more appropriately addressed at the local level.

D. Summary of Staff Conclusions and Recommendations on Secondary NAAQS

This summary of staff conclusions and recommendations for the PM secondary NAAQS draws from the discussions contained in the previous sections of this Staff Paper. The key findings are:

1) Anthropogenic fine particles impair visibility. The level of this impairment varies greatly from East to West, in terms of total loadings, pollutant mix, and the resulting total light extinction. Background levels of fine particles, humidity, and resulting total light extinction vary regionally as well, with the East having generally higher levels than the West.

2) The levels recommended in this staff paper for protection of public health from the adverse effects of fine particles will not completely address the visibility impairment of fine particles on visibility or fully achieve the national visibility goal across the country.

3) Because of regional variations in visibility conditions created by background levels of fine particles, annual average humidity, pollutant mix, and resulting total light extinction, the staff concludes that a national secondary standard to protect visibility would not be an appropriate approach for addressing visibility impairment due to fine particles. Therefore, to address the impairment of visibility from fine particles and to make reasonable progress towards the national visibility goal, the staff recommends that the Administrator consider establishing regional haze regulations under section 169A of the Act.

4) The available data assessed in the CD does not provide an adequate basis to establish a unique national secondary standard to protect against soiling and materials damage effects. The staff recommends setting a secondary standard equivalent to the primary standards for the purposes of addressing soiling and materials damage.

APPENDIX A
CONSIDERATIONS IN SELECTING PARTICLE SIZE CUT POINT FOR FINE PARTICLES

An important decision relating to the choice of indicator is the choice of measurement which in a sense serves as an operational definition of fine particles. The CD concludes that the minimum of mass between the fine and coarse modes lies between 1 and 3 μm, and that the scientific data support a cut point to delineate fine particles in this range (CD, Chapter 3-5). Because of the overlap of fine and coarse particles in this intermodal region, specific cut points are only an approximation of fine particles. Thus, the decision within this range is largely a policy judgement. Although most fine particle (accumulation mode) mass is below 1.0μm, some hygroscopic particles in conditions of high relative humidity may gain water and grow above this size. However, energy considerations normally limit coarse mode particle sizes to greater than 1.0 μm in diameter (CD, 3.1.2).

The main policy choice centers on two options: $PM_{2.5}$ and PM_{1}. Staff recommend the three primary factors to consider in selecting a cut point are consistency with health data, potential for intrusion of mass from the other mode, and availability of monitoring technology.

From a public health perspective, use of a $PM_{2.5}$ cutpoint will result in the capture of all of the potential agents of concern in the fine fraction. For example, the cutpoint of $PM_{2.5}$ captures most sulfates, acids, fine particle metals, organics, and ultrafine particles and accounts for most of surface area, and particle number. Although the CD outlines some conditions (e.g., relative humidity near 100 percent) under which it is possible that hygroscopic particles may grow above 2.5μm, use of the $PM_{2.5}$ cutpoint is still better at capturing the constituents of concern than PM_{1}.

$PM_{2.5}$ has been measured directly in many health studies as described in the CD and Chapter V, Section F above. Significant associations have been reported between $PM_{2.5}$ concentrations and mortality, hospital admissions, cough, upper respiratory infection, lower respiratory infection, asthma status, and pulmonary function changes.

$PM_{2.5}$ measurement technologies are widely available and have been in routine use in the field since the early 1980s. For example, the EPA AIRS database contains $PM_{2.5}$ data from the Inhalable Particle Network (1982-1984), the IMPROVE network (1987 - present), and the NESCAUM network (1988- present). In addition, the California Air Resource Board (CARB) dichotomous sampler network has been collecting $PM_{2.5}$ data routinely since 1980, and many other special studies measuring $PM_{2.5}$ have been conducted across the country. Furthermore, dichotomous samplers allow the coincident measurement of PM_{10} and $PM_{2.5}$, increasing the certainty of comparability between the two measurements.

Measurement of fine particle mass using a 1 μm (PM_1), on the other hand, has not been used in health studies primarily due to lack of available monitoring data. Comparisons between PM_1 and other measurements that were used in the health studies (e.g., PM_{10}) are also not widely available due to lack of available PM_1 monitoring data. Furthermore, PM_1 may not capture as much of the hygroscopic substances such as sulfates which health studies report as having statistically significant associations between sulfate measurements and endpoints including increased mortality and hospital admissions.

PM_1 sampling technologies have been developed and some limited validated data are available from locations such as Phoenix, Arizona. However, the PM_1 samplers have not been widely field-tested to date.

Proponents of the PM_1 option are concerned that the intrusion of particles generated by grinding or crushing (i.e., coarse mode particles) into the daily $PM_{2.5}$ measurement could create spurious NAAQS exceedances. Given the lack of PM_1 data currently available, it is difficult to determine how much intrusion might occur or what areas might be affected during the implementation of a $PM_{2.5}$ NAAQS. The available data show that typically only 5-15 percent (on the order of 1 to 5 $\mu g/m^3$) of the $PM_{2.5}$ mass is attributable to soil-type sources even in dusty areas such as San Joaquin Valley, California, and Phoenix, Arizona. However, this percentage may increase during events such as high winds.

The staff judges that in typical urban areas, the potential for this type of intrusion may be smaller, but without sufficient data these determinations remain very uncertain. A sharper inlet for the Federal Reference Method may help to minimize the intrusion of coarse mode particles into the $PM_{2.5}$ measurement. Although intrusion of coarse mode particles into

daily $PM_{2.5}$ measurements is not anticipated to be significant in most situations, if in light of more data a problem is identified, this issue might be better addressed on a case-by-case basis in the monitoring and implementation programs.

Finally, the staff concludes that $PM_{2.5}$ measurements are more appropriate than some of the measurements historically used in the epidemiological studies (e.g., BS, CoH) although these measurements have been useful in advancing the state of scientific knowledge of particle effects. British Smoke (BS) readings vary more with darkness of particles (i.e., carbon content) than with mass, making associations with mass highly site- and time-specific. The BS method emphasizes control of primary elemental carbon emissions; however, elemental carbon is a minor contributor to fine and total mass in current U.S. atmospheres. Furthermore, lack of consistent relationships between BS reflectance and PM mass measurements diminishes one of the major advantages: BS is not related to the available quantitative health data from U.S. cities with as much certainty as the $PM_{2.5}$ mass measurements although BS is used in many other countries. Using a similar principle to BS, the principle of coefficient of haze (COH) is that visible light is transmitted through (or reflected from as in the case of BS) a section of filter paper before and after ambient air is drawn through it. Thus, COH associations with mass are also highly site- and time-specific.

Thus, because of the consistency with health data, small potential for intrusion, and availability of monitoring technology and existing air quality database, the staff judges that the $PM_{2.5}$ measurement is more appropriate for regulatory purposes than PM_{1}, or historical measurements such as BS or COH.

APPENDIX B

MEASUREMENT METHODS FROM EPIDEMIOLOGY STUDIES

The CD and Chapter V of this Staff Paper summarize health studies which have reported associations between various indicators of PM and health effects. The main mass concentration indicators are TSP, PM_{10}, and $PM_{2.5}$. In addition to $PM_{2.5}$ mass measurements, fine particles have been measured in the U.S. and abroad using a variety of techniques including British or black smoke (BS), coefficient of haze (COH), carbonaceous material (KM), and estimates from visibility measurements (CD, Section 4.2.8).

Studies have also reported associations between health effects and exposure to fractions found predominantly in the fine fraction such as sulfate ($SO_4^=$) and strong acidity (H^+). The CD describes measurement techniques in detail; this section highlights relevant information about other indicators of fine particles (i.e., BS, COH, and KM).

In the past, it was noted that visibly black plumes were emitted by industrial sources; thus, light absorption was adopted as a measure of PM pollution (Chow, 1995). Measurements of the optical properties of particles may be related to gravimetric mass measurements on a site- and time-specific basis with on-site calibrations.

BS preferentially measures elemental carbon particles found in the fine fraction (CD, Section 4.2.8; Baily and Clayton 1980). In addition, the BS inlet design, taken together with its other operating parameters, restricts the size of particles that are sampled. For example, it has been shown in wind tunnel tests that the best estimate of the cut point for BS is 4.5 μm (CD, page 4-52; Waller, 1980; McFarland, 1979). Most particles larger than the cut point of 4.5 μm are either rejected at the inlet or lost in the inlet line (U.S. EPA, 1982a). Furthermore, the BS reading varies more with darkness of particles (i.e., carbon content) than with mass, thus making associations with mass highly case-specific. Because elemental carbon is found predominantly in the fine mass (less than 1.0 μm range), variations in BS are more closely related to fine mass and unlikely to be sensitive to coarse mode particles (NAS, 1980; U.S. EPA, 1982b).

Using a similar principle to BS, COH measures visible light transmitted through (compared to reflected from in the case of BS) a section of filter paper before and after ambient air is drawn through it. The amount of light transmitted is measured by a photocell

(Chow, 1995; Fairley, 1990). In addition, this sampler uses a funnel inlet and a small diameter transport tube nearly identical to the BS sampler. Although the two samplers operate at different flow rates, the particles reaching the filter tape could be expected to have a size range similar to that of the BS instrument (U.S. EPA, 1982a, see Figure 3A-12).

Prior to the 1980s, PM was measured in California by optical reflectance of particles collected on a sample tape (KM). Similar in principle to BS, KM has been shown to be closely related to elemental carbon content in Los Angeles (Kinney and Özkaynak, 1990). Similar to BS, KM is also a fine particle measurement.

Visibility measurements can also be used as a reasonable surrogate to estimate fine particle concentrations because the extinction coefficient is directly related to fine particle mass (CD, page 6-216).

APPENDIX C

PM_{10} NATIONAL CONCENTRATION MAPS AND DEFINITIONS OF REGIONS

Current U.S. PM_{10} levels are illustrated in Figures C-1 and C-2. Figure C-1 shows the fourth highest 24-hour PM_{10} concentration recorded in a county and Figure C-2 depicts highest annual mean PM_{10} concentration using 1992 to 1994 AIRS data in each county for which data completeness criteria were met. Counties not represented with a monitor are left blank.

The following methods were used to calculate the values depicted in the maps. The current single exceedance form of the PM_{10} daily standard allows for an average of one exceedance per year over a three-year period. Thus, the fourth highest concentration is of interest because this value is used to determine attainment with the current daily standard. Seven hundred and twelve counties met the data completeness criterion of at least 75 percent complete data for the period 1992 to 1994. For these counties, all daily concentrations were ordered largest to smallest and the fourth highest PM_{10} concentration was determined for each site. If a county had only one site, then the fourth highest concentration for that site was reported. If a county had more than one site, the site with the maximum fourth highest concentration was used to represent the county.

Figure C-2 shows the maximum annual mean concentration in each county over the three-year period using an average weighted by calendar quarter. Three hundred and eighty counties met the 75 percent data completeness criterion by quarter for 1992 to 1994. Means were calculated for all four calendar quarters for each year in the 3-year period and annual values were calculated based on the quarterly means. The three yearly means were then averaged to obtain one value for each site. If a county had only one site, then the annual mean for that site was reported. If a county had more than one site, the site with the maximum annual mean was used to represent the county.

Figure C-3 shows the regions of the country used in some air quality analyses. Note that state boundaries were used except that California and Texas were split.

Figure C-4 illustrates that a total of 87 different sites reported $PM_{2.5}$ data to AIRS from 1983 to 1993. Over the 11 year period, less than 50 sites reported data to AIRS in any given year. Additional special studies have also monitored $PM_{2.5}$, but these data are not reported in AIRS.

Figure C-1.

PM-10 Air Quality Concentrations, 1992-94

Maximum 4th Highest Daily Concentration

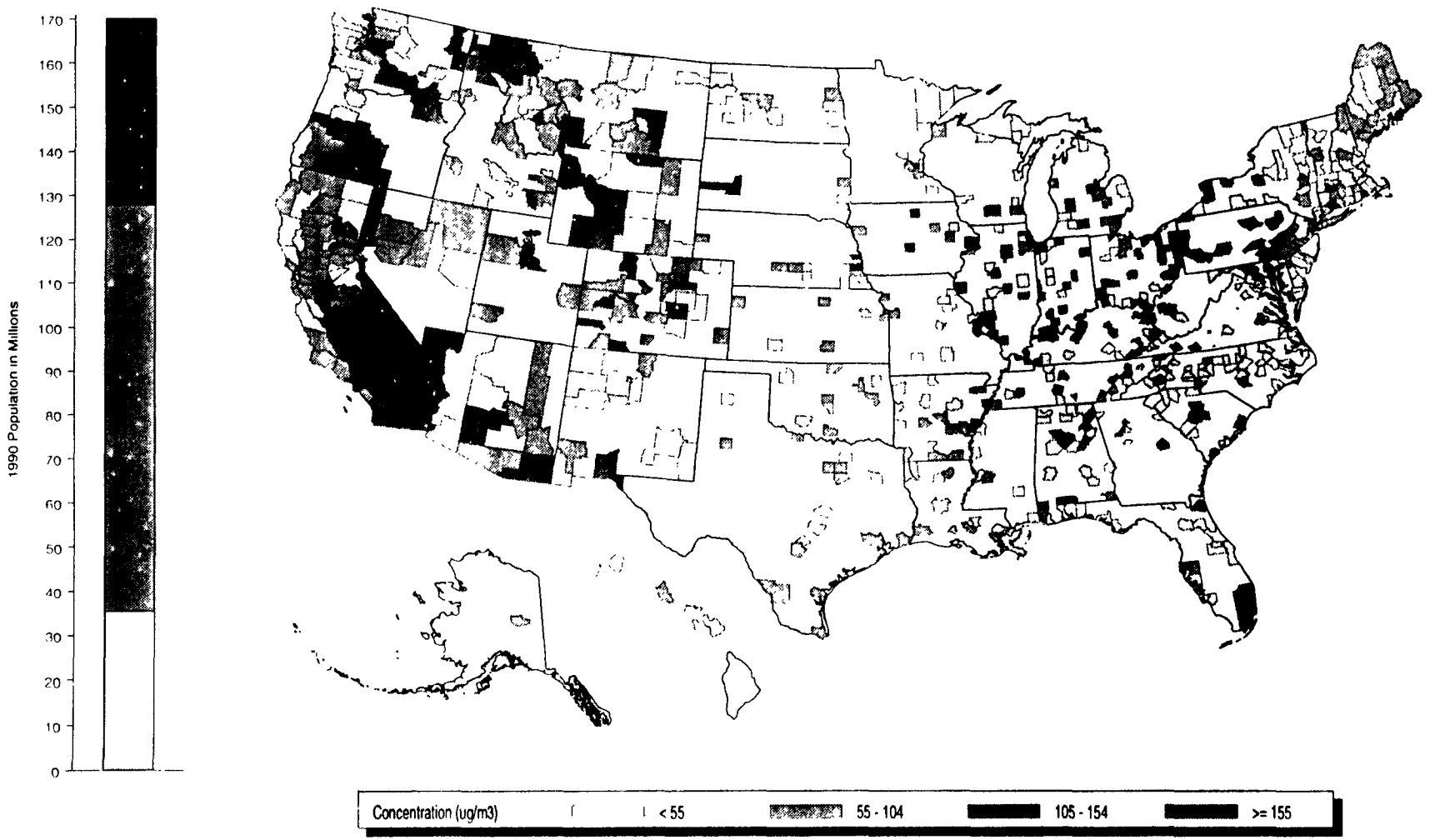


Figure C-2. **PM-10 Air Quality Concentrations, 1992-94**
Maximum Annual Mean

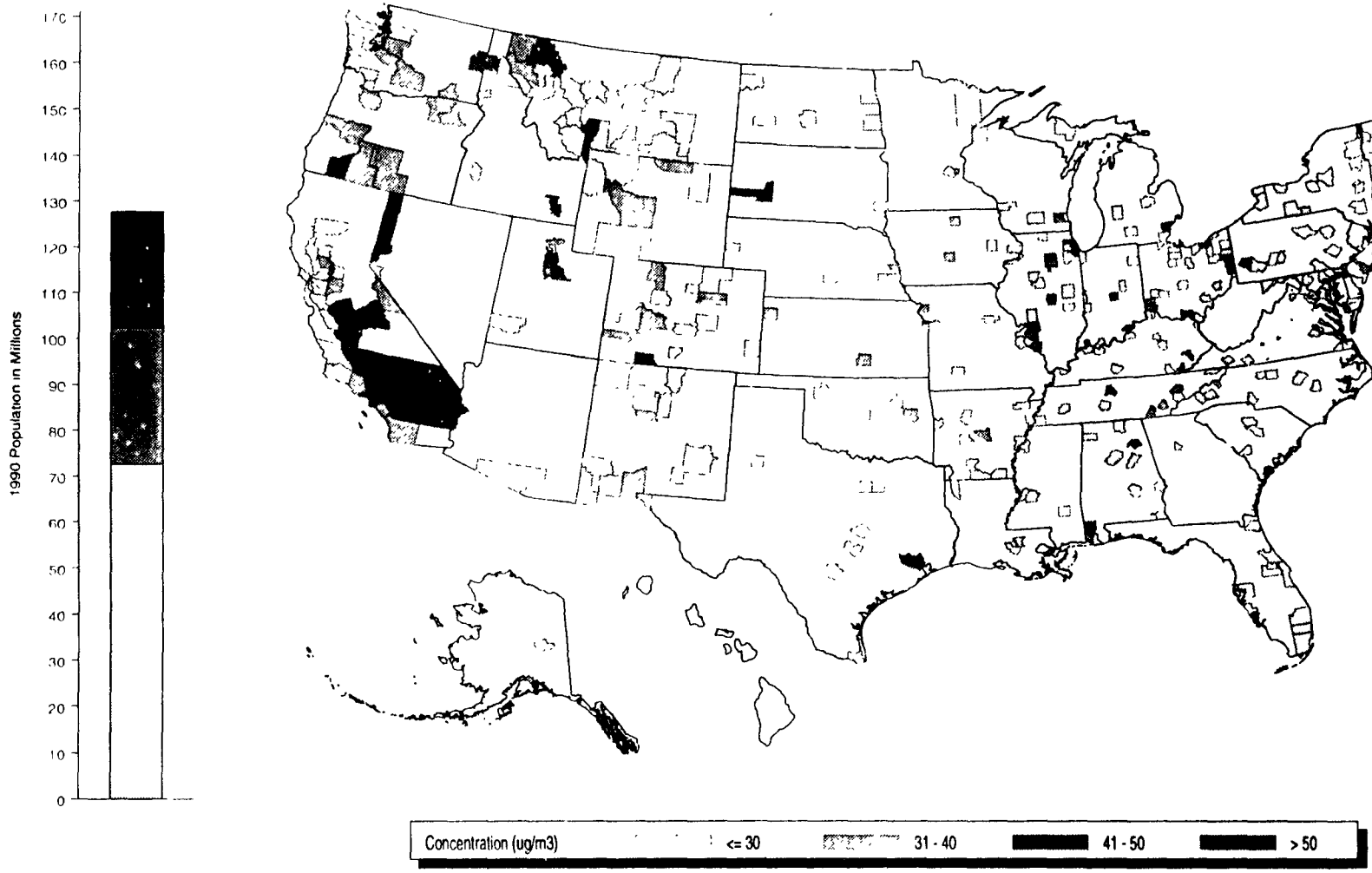
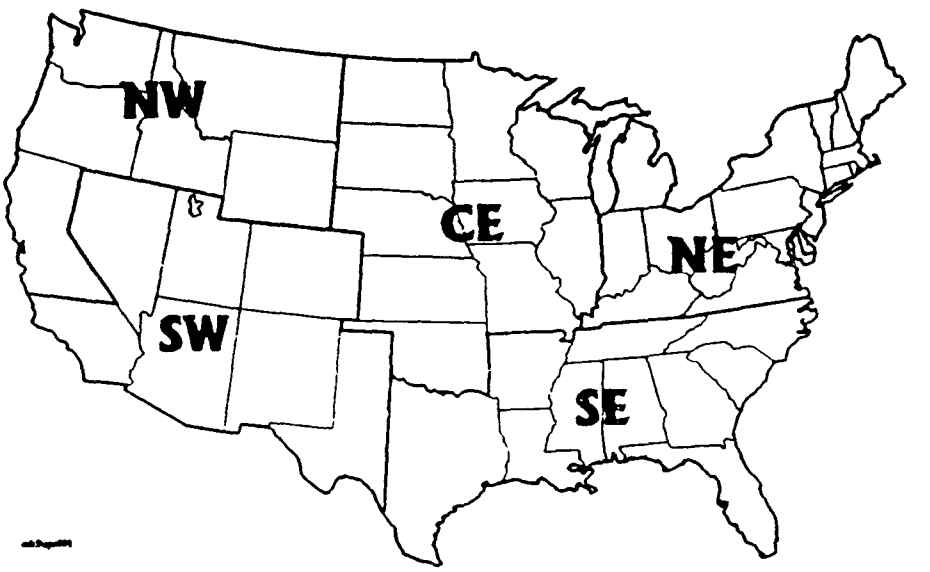


Figure C-3. Regions Used in Air Quality Analyses in this Staff Paper



Region	States
SW	NV UT CO NM AZ TX(West) CA(South)
NW	OR WA ID WY MT CA(North)
CE	OK MO KS NE IA SD ND MN WI IL
NE	IN KY OH MI VA WV PA NY MD NJ CT RI MA VT NH ME DE DC
SE	FL GA AL MS LA TX(East) AR TN NC SC

Figure C-4. PM2.5 AIRS Data Summary, 1983-1993

Figure C-4a. Geographic Distribution of Sites

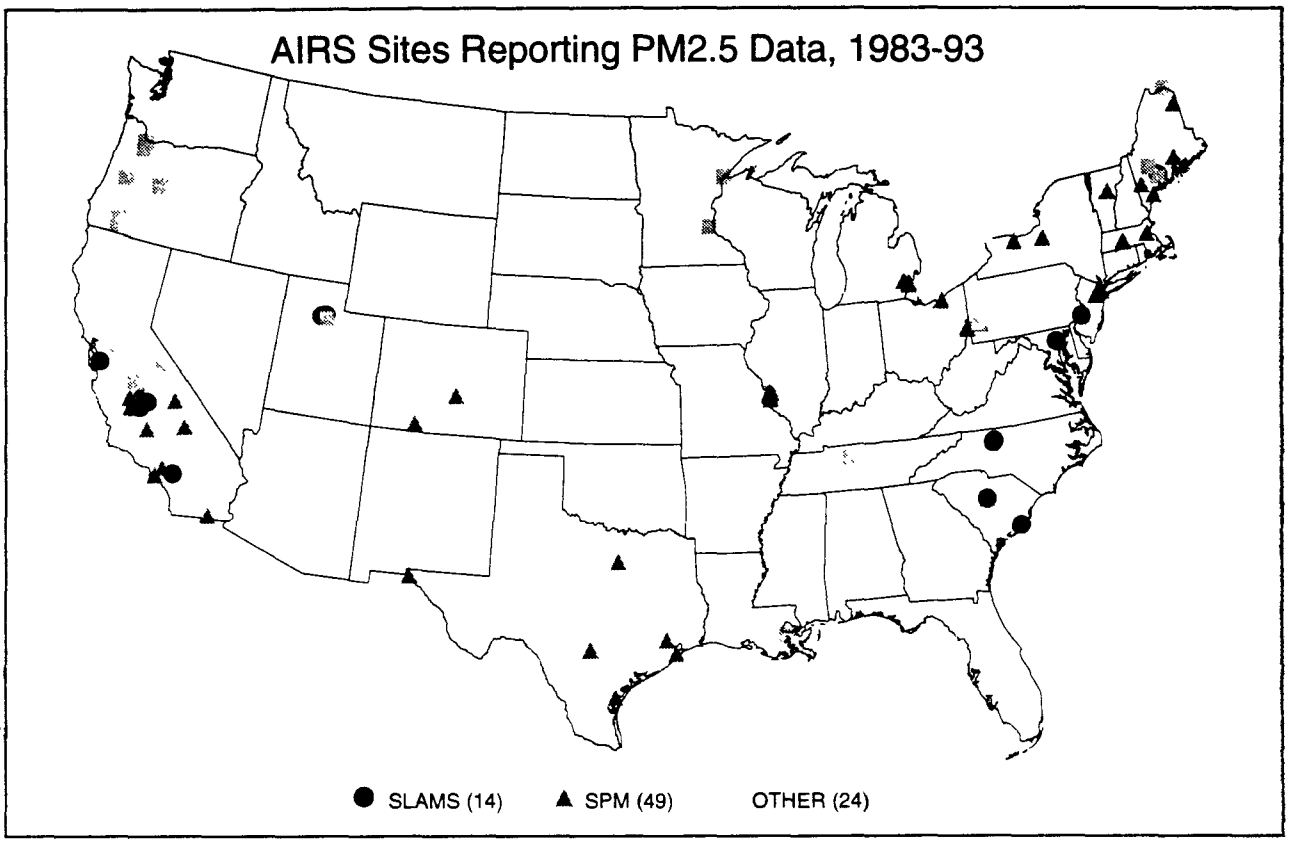
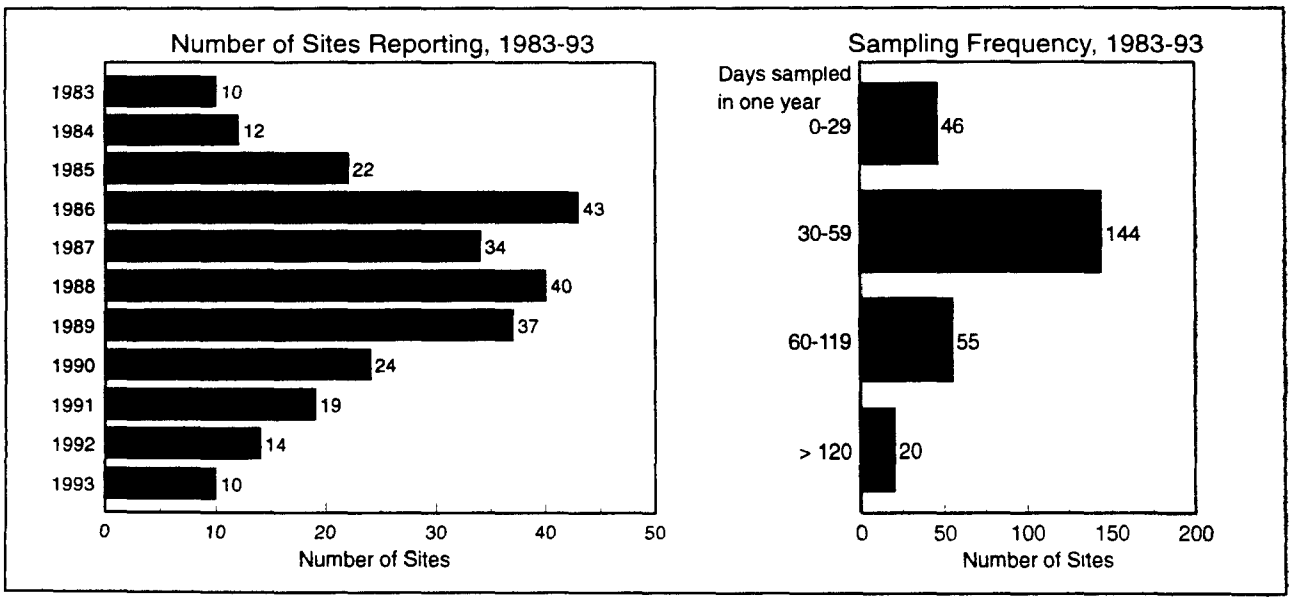


Figure C-4b. Number of Sites and Frequency of Sampling



APPENDIX D

I. HYPOTHETICAL MECHANISMS OF ACTION FOR PM

1. Dosimetric Considerations

Dosimetric considerations formed the principle basis of the approach used for selecting PM_{10} as the indicator of the current standard (pp.23-39, U.S. EPA, 1982b). Exposure can be described, in the context of regulating PM, as the concentration of particles available in the ambient air that a human or animal breathes over a relevant period of time. Dose is the amount of this material that is inhaled and available for deposition at various target sites (e.g., regions of respiratory tract) (CD, p. 10-1). It is the dose that the target site or organ receives upon which manifestation of toxicity depends. The amount of particles deposited or retained in each region of the respiratory tract is governed by exposure concentration, particle diameter and distribution, physico-chemical properties of the inhaled particle (e.g. hygroscopy and solubility), and duration of relevant exposure. In the previous review, such dosimetric considerations, health effects of concern, and aerosol physico-chemical characteristics prompted the Staff with CASAC concurrence to determine that the major risk of commonly occurring outdoor PM was presented by particles of 10 micron or less aerodynamic diameter. Particles of this size are able to penetrate the presumptive targets of PM (tracheobronchial and alveolar regions of the human respiratory tract) (CD, Chapter 10).

The human respiratory tract can be divided into three main regions: (1) extra-thoracic, (2) tracheobronchial, and (3) alveolar regions as shown in Table 10-5 of the CD. They differ markedly in structure, function, size, and sensitivity or reactivity to deposited particles (U.S. EPA, 1982b). Disposition and retention of initially deposited particles depends on clearance and translocation mechanisms that vary with each region of the respiratory tract. Coughing, mucociliary transport, endocytosis by macrophages or epithelial cells, and dissolution and absorption into the blood or lymph are important mechanisms of clearance in the tracheobronchial region. Endocytosis by macrophage or epithelial cells and dissolution of absorption into the blood or lymph are the dominant mechanisms of clearance in the alveolar region.

In essence, ambient particles of 10 μm diameter or less deposit with varying efficiencies in tracheobronchial and alveolar regions of the respiratory tract. Simulations of deposition show that alveolar deposition is fairly uniform for particle between 0.5 and 4.0 μm diameter. Table V-1 of Chapter V is derived from Tables 10-21 and 10-23 of the CD and shows the deposition patterns in the human lung for typical particle distributions found the cities of Philadelphia and Phoenix. This table represents the general population of adult males with normal breathing. The table shows not only do all size fractions below 10 μm diameter have the potential for some deposition in both tracheobronchial and alveolar regions but deposition patterns of the types of particles found in urban areas can be similar in these lung regions under specific conditions.

In regard to sensitive sub-populations, increased deposition and altered clearance may play a role in susceptibility to PM. A detailed discussion of these individuals is presented in section 5-D. Model simulations have suggested that deposition efficiency of particles will be increased in people with COPD and asthma (Anderson, 1990; Miller et al., 1995; Svartengren et al., 1994). Kim et al (1988) demonstrated much greater particle deposition in COPD patients using aerosol re-breathing tests. A compromised lung with greater deposition has a greater probability of interaction of PM with potential targets of PM toxicity and thus increased effects. However, the contribution of such differential deposition of particles to mortality and morbidity has not been elucidated or quantified.

Similarly, differences in dosimetry between animals and humans may be a contributing factor for the apparent differences in animals and human study results. Rodents have a greater deposition of particles in the upper respiratory tract than humans. In addition, models show that humans retain a greater fraction of particles deposited in the alveolar region than do rats or mice. Thus, the differences in deposition patterns of particles between species and between susceptible and nonsusceptible subpopulations could be a contributing factor for the necessity of using relatively high concentrations of larger diameter particles to elicit effects seen in experimental animal studies (CD, Chapter 10).

2. Possible Mechanisms of Action for Health Effects Associated with Ambient Levels of PM Exposure

This discussion focuses on more specific possible mechanisms by which airborne particles may be exerting their effects. Upon deposition, substantial uncertainty still exists as to how particles, alone or in combination with other atmospheric pollutants, produce physiological and ultimately pathological effects. Because both the population affected and PM are heterogenous, the mechanism(s) of action may also be diverse. As shown in the CD (Chapter 13), exposure to particulate matter has been identified as causing a variety of health effects including respiratory symptoms, mechanical changes in lung function, alteration of mucociliary clearance, pulmonary inflammatory responses and morphological alteration in the lung. In addition, from epidemiological studies PM has been reported to be associated with increases in respiratory illness, hospital admissions, and daily mortality.

Consequently, the increasing body of community epidemiological studies finding associations between PM and mortality and morbidity in recent years have prompted a number of authors to advance potential mechanisms of PM toxicity. One major area of interest is pulmonary inflammation. Potential mechanisms for induction of an inflammatory response have been described for: (1) aerosol acidity (Lippmann, 1989a), (2) presence of ultrafine particles (Seaton et al., 1995), and (3) transition metal ions (Tepper et al., 1994). A second area of renewed interest includes examination of the ways particles may affect individuals with preexisting conditions. Frampton et al. (1995) list potential causes of PM induced mortality as being: (1) premature death (i.e., hastening of death for individuals near death within hours or days); (2) increased susceptibility to infectious disease; and (3) exacerbation of chronic underlying cardiac or pulmonary disease. Also of significant interest are new approaches for controlled exposures to particles which are closest to those found under ambient conditions than have been possible in past toxicologic studies (Sioutas et al., 1995). The opportunity to study such particles may be particularly valuable in studying the effects from and potential mechanisms of action for PM exposure. The issue of discrepancies between experimental doses and ambient PM in terms of composition and magnitude of administer dose may be resolved. However, early results of such studies while promising are preliminary and may be valuable for future reviews. A brief summary of

potential mechanisms of toxicity is discussed below. Further discussion is provided in Chapters 11 and 13 of the CD.

The most serious effects associated with community studies of PM appear to be found in individuals who have preexisting conditions. Even in the London episodes, the total amount of inhaled PM by mass eliciting a response in humans was small. Therefore, it is likely that the effect of PM exposure is amplified in conjunction with preexisting conditions that increase risk for PM effects. Given that immunological responses can be quite rapid, consistent with the period between increased PM exposure and an acute effect such as mortality, it is plausible that inflammatory processes can amplify and spread the response from small amounts of PM.

Preexisting inflammation (e.g., from an ongoing infection) of the lung can amplify the inflammatory response to residual fly ash in emphysemic rats (Costa et al., 1995). Indeed, several of the risk factors for PM toxicity involve inflammatory response (e.g., asthma, COPD, and infection). A similar profile of susceptibility may be shown by the only animal deaths recorded during the London Fog of 1952 linked to the fog. These were prize show cattle which suffered from both shipping fever and emphysema. Thus, the cattle which shared susceptibility to the London fog with humans may also share some of the same pre-existing conditions (e.g., COPD and inflammation). A commonly offered explanation of the susceptibility of the show cattle was that they were kept in cleaner stalls and thus had much lower waste ammonia present that might serve to neutralize the high levels of acid aerosol portions of the fog and thus decrease their toxicity. The original report by the Ministry of Health (MOH, 1954), however, also reported cattle death in previous fogs with ordinary stall maintenance and therefore high ambient levels of ammonia that could neutralize acid particles.

Seaton et al., (1995) has proposed the hypothesis that the mechanism of PM involves production of an inflammatory response by ultrafine particles (< 0.02 μm diameter) in the urban particulate cloud. As a result, mediators may be released capable of causing exacerbation of lung disease in susceptible individuals and increased coagulability of the blood. Thus a rationale is provided for the observed increase in cardiovascular deaths associated with urban pollution episodes. Several hematological factors, including plasma

viscosity, fibrinogen, factor VII, and plasminogen activator inhibitor are not only known to be predictive of cardiovascular disease (Lowe, 1993) but to also rise as a consequence of inflammatory reactions. Low grade inflammation has been hypothesized to be particularly important in altering the coagulability of blood as a result of activation of mononuclear cells in the lung (MacNee and Selby, 1993). Activated white cells may initiate and promote coagulation (Helin, 1986) via the final clotting pathway (Ottaway et al., 1984). Alveolar inflammation may also cause the release of interleuken - 6 from macrophages and thus stimulate hepatocyte to secrete fibrinogen (Akira and Kishimoto, 1992). Crapo et al., (1992) has suggested that activation of lung macrophages in the absence of recruited neutrophils leads to acute damage of capillary endothelial cells as well as alveolar lining cells, resulting in intracellular edema, hemorrhage and fibrin deposition.

In support of Seaton's proposed mechanisms is the observation that ultrafine particles cause greater inflammation (assayed by broncho-alveolar lavage) than larger particles of the same substance (Chen et al., 1992; Oberdörster et al., 1992). Fine particles have been shown to be taken up by lung epithelial cells (Stringer et al., 1995) and lung macrophages (Godleski et al., 1995). They have also been shown to produce inflammation *in vitro* (Dye et al., 1995) and *in vivo* (Kodavanti et al., 1995). In addition, metals have been shown to increase the toxicity of particles. Intertracheal instillation of residual oil fly ash into rats also produces an inflammatory response (Jaskot et al., 1995) with Dreher et al., (1995) linking such inflammation to soluble vanadium, iron, and nickel compounds on the particles. Ferric sulfate has been shown to alter pulmonary macrophage function (Skornik and Brain, 1983). In support of an inflammatory component to PM toxicity are several recent reports involving diesel particles which have ascribed observed inflammatory/tumor promoting effects to carbon cores rather than adsorbed organic (CD, Chapter 11, Section 11.5.5). Thus, under this proposed mechanism of PM effect, toxicity may involve a response to PM which involves inflammation.

Aggravation of underlying conditions (chronic cardiopulmonary disease in particular) has been observed in epidemiologic studies as increased hospital admissions for such conditions and decreases in pulmonary function. Aggravation of severity of these conditions has also been hypothesized to explain increases in daily mortality and longitudinal increases

in mortality. Under such a scenario individuals experience more frequent and severe symptoms of their preexisting disease or a more rapid loss of function.

Airflow obstruction could result from laryngeal constriction or broncho-constriction secondary to stimulation of receptors by PM in the extrathoracic or intrathoracic airways. In addition, stimulation of mucous secretion could contribute to mucous plugging in small airways. In pre-existing airway diseases, which feature localized airway narrowing or obstruction, the increased accumulation of PM may lead to hypoxia in the respiratory regions of the lung served by the obstructed airways. In tandem under such condition, there also may be an increased particle deposition and adverse effects on the non-obstructed areas of the lung (CD, p. 11-184). Finally, effects on the surfactant layer in the alveoli by PM may cause increased leakiness in the pulmonary capillaries leading to interstitial edema. Experimentally, acid aerosols have been shown to cause acute effects on pulmonary function among some sensitive individuals. They may induce hyper-reactive airways after 75 $\mu g/m^3$ H_2SO_4 for 3 hours (El Fawal and Schlesenger, 1994). Therefore, the elderly with debilitating disease such as asthma may be stressed by the fine acid aerosols.

In regard to particle size, Thurston et al., (1994b) have reported that hospital admissions for asthma were more strongly associated with fine rather than coarse fraction particles. Aggravation of asthma symptoms has also been reported for fine particles (Ostro et al., 1991; Perry et al., 1983). In studies of cellular and immunological injury with PM inhalation, Kleinman et al. (1995) reports that in eliciting responses 0.2 μm diameter SO_4^{-2} is greater than 0.6 μm diameter NO_3^-, which in turn is greater than 4μm diameter resuspended road dust. Measures of alveolar cord length and cross sectional area were most reduced with the fine sulfate particles which could result in a decrease in compliance or "stiffening" of the lung and smaller inflation volume.

Related to the potential for aggravation of underlying disease by PM is the issues of whether increases in mortality reported to be associated with PM are a result of hastening of imminent death. While this is a plausible and reasonable suggestion, other evidence suggests that it may not explain the full effects of PM on mortality. For example, in interviews with the family members of victims of the London pollution episode of 1952, while some of those victims were reported to having chronic pre-existing conditions and

some having infections, several were reported to have no indication of a life threatening disease process (Ministry of Health, 1954). As reported by the CD (Chapter 13), it appears likely that life shortening from PM exposure is highly variable and could range from days to years. The CD concludes that duration life shortening, lag times, and latent periods of PM-mediated mortality are almost certainly distributed over long time periods. However, confident quantitative determination of specific estimates of years lost to ambient PM exposure is not possible at this time.

There are several potential targets for PM throughout the respiratory tract which may involve stimulation of airway neurological receptors to elicit observed health effects (e.g., bronchoconstriction and mucous secretion). The tracheal bronchial tree has been described as the dominating site for vagal reflexes affecting the airways and most definitely associated with common conditions such as asthma and chronic bronchitis (Widdicombe, 1988). However, respiratory receptors which can effect cardiac as well as other pulmonary effects are distributed through the respiratory tract. For example, "irritant" receptors reside in the epithelium from trachea to respiratory bronchiole, that produce bronchoconstriction and reflex contraction of constrictor muscles of the larynx as well as secretion of tracheal mucous (Widdecombe, 1988). "C" receptors are distributed throughout the tracheobronchial tree and in the alveolar wall, and probably also in the laryngeal mucosa (Sant' Ambrogio, 1982; Coleridge and Coleridge, 1986). They have some of the same actions as "irritant" receptors and are activated by the same group of stimuli (Widdicombe 1988). Most of the lung inflammatory and immunologic conditions such as asthma and chronic bronchitis would probably activate C and irritant receptors, which would interact to cause augmented airway responses (Widdecombe 1988). "J" receptors, which reside in the alveolar wall, can elicit a powerful constriction of the larynx as well as bronchoconstriction. The main activation of these receptors occurs in pathological changes in pulmonary circulation and the alveolar wall rather physiological conditions (Widdcombe, 1974, 1988). Lung pathologic conditions (e.g., edema, pulmonary congestion, pneumothorax, microembolisms and anaphylaxis) as well as various irritant gases (e.g., cigarette smoke, sulfur dioxide, and ammonia) and a wide range

of mediators (e.g., prostaglandins and histamine) have been shown to stimulate lung "irritant" receptors. Irritant gases have been shown to stimulate both lung "irritant" and "J" receptors (Widdecombe 1974, 1988).

Cessation of cardiac activity is often the terminal event in life. Pulmonary responses to PM exposure may include hypoxemia, broncho-constriction, apnea, impaired diffusion, and production of inflammatory mediators that can contribute to cardiovascular perturbation (CD, p. 13-71). For example, hypoxia can precipitate cardiac arrhythmias and other cardiac electrophysiologic responses that may lead to ventricular fibrillation and ultimately cardiac arrest. In addition stimulation of many respiratory receptors have direct cardiovascular effects such as bradycardia and hypertension (C-fibers, nasal receptor or pulmonary J-receptor, and laryngeal receptors) and arrythmia, apnea and cardiac arrest (laryngeal receptors) (CD, p. 13-72).

Particles that may deposit in the lung over time may induce an inflammatory response that could lead to pulmonary fibrosis and impaired pulmonary function. With repeated cycles of acute lung injury by PM and subsequent repair, fibrosis may develop. Persistence of toxic particles may also promote a fibrotic response (CD, p. 13-72). Large lung burdens of particles of even relatively low inherent toxicity have been shown to cause lung cancer in rats (Mauderly et al., 1994). While there is difficulty in elucidating how long-term particle accumulation can induce acute mortality, it may be a factor for the elderly who have been chronically exposed to PM in the work place, those who have resided in heavily industrialized cities before effective control of PM, or smokers. As reported in the previous section, sensitive subpopulations with obstructive pulmonary diseases may have focalized particle accumulation in their lungs due to ventilation abnormalities. However, the mechanism by which prior exposure to particulate could predispose an individual to acute PM effects is unknown.

Impaired respiratory defense has also been proposed as a contributing factor to PM toxicity. Patients with pneumonia have increased risk of mortality and morbidity from PM exposure. Cough, bronchitis, and lower respiratory illness have been reported to be associated with increased ambient particle concentrations (CD, Chapter 12, see below).

Both mucociliary transport and macrophage function are critical to host defense against inhaled pathogens. Potentiation of inflammation and infection from biologically active particles (e.g., spores, fungi, and bacteria) may result from effects on clearance and macrophage function by the acid aerosol component of PM (CD, p. 13-75). Increased risk of infection has been associated with changes in mucociliary clearance (e.g., excessive mucus secretion into the airways can cause airway blockage and reduced clearance). Alveolar macrophages are the primary defense cells of lungs and impairment of their function would also be expected to increase risk of infection. Clearance and macrophage function have been shown experimentally to be affected by constituents of PM, notably fine acid aerosols.

H_2SO_4 and trace metals have been shown to have direct effects on alveolar macrophages in animal experiments (CD, p. 13-75). Kleinman et al. (1995) also reported in their study of cellular and immunological injury by PM that antigen binding to receptors in and respiratory burst activity by macrophages was depressed by exposure to fine (0.2μm diameter) SO_4^{-2} particles. H_2SO_4 has also been shown to affect mucociliary transport and, in combination with ozone, resistance to bacterial infection. However, these effects have been shown at concentrations which are much higher than those reported in the recent epidemiological studies for which PM effects have been reported. Effects mediated through clearance, in particular, would be expected to be manifested over an extended period of exposure rather than a few days. While impaired host defense may not be plausible as a mechanism for mortality associated with short-term fluctuations of PM level, it may contribute to the long-term exposure mortality. In addition, the lag-time reported between PM concentration elevations and general indicators of morbidity (e.g., missed school and work loss days) is consistent with an increased susceptibility to infection which may precipitate respiratory symptoms (see discussion in section V.C).

II. EXTRAPOLATION OF RESULTS FROM LABORATORY STUDIES TO THOSE OF EPIDEMIOLOGIC STUDIES: STRENGTH AND LIMITATIONS OF CONTROLLED HUMAN AND ANIMAL STUDIES

As discussed above, the adverse effects of particulate matter exposure have been shown to be consistent between historical and more recent studies. The effects can be severe and tend to be concentrated in sensitive sub-populations who have pre-existing conditions or characteristics that tend to make them vulnerable to respiratory insult (the very young and old, asthmatics, COPD patients, patients with pneumonia etc). The additional risk of reported mortality and morbidity from particulate matter exposure is relatively small in terms of the whole population. Therefore, large numbers of people must be exposed before effects can be discerned in studies. The question arises as to how to elucidate the mechanism of action of particulate matter in humans. What are the considerations that must be taken into account when an analysis of the body of human clinical data and experimental animal work is done in order to infer a plausible mechanism for particulate matter effects?

1. Numbers of Individuals Affected

An issue of primary concern is that of statistical power. The nature of the effect described in epidemiological work is consistent, and serious, but occurring in a relatively small fraction of the total population (1 in a million increased risk for daily mortality). Therefore, theoretically a relatively large number of animals would be needed to mimic the frequency of response at similar doses. The use of a similar number of animals to mimic the frequency of response to ambient air concentrations of particles which have been associated with effect in humans is impractical. Therefore, in many experimental paradigms, relatively large concentrations are often given investigate the response from a limited number of animals. However, the questionable relevancy and sensitivity of such paradigms limits their use in the determination of the mechanism of action of relatively low changes in concentrations of inhaled particulate matter.

2. Heterogeneity of Human Population

The human population for which the effects are most demonstrable are a sub-population from a genetically heterogenous group. Furthermore, consistency of response is highly variable among the population at risk (e.g., a relatively small group of asthmatics have aggravation of symptoms and not all patients with pneumonia or COPD die as a result of an increase in inhaled particle concentration). The CD suggests that for clinical studies involving asthmatics, differences among subjects may explain in part the differing results between laboratories who study effects of acid aerosols. As an example of differential susceptibility to a respiratory insult, a minority of individuals (3-5%) who are exposed to etiologic agents responsible for hypersensitivity pneumonitis (allergic alveolitis) will develop disease. Determinants of susceptibility for that disease have been described as both the genetic constitution of the individual and the presence of preexisting lung disease. Similar factors probably play a role in susceptibility to inhaled particulate matter effects.

By contrast experimental animals are bred as much as possible to be homogenous genetically so as to give great consistency in response. They are also usually studied in their prime in regard to age and general health. Presence of disease is generally considered to be a confounding factor to be stringently controlled in most animal paradigms. As stated above, those segments of the general population most affected from PM_{10} exposure are the sick, the very young, and the old. Therefore the sensitivity of studies using relatively small numbers of healthy, genetically homogenous, laboratory animals who are in their prime is diminished in exploring mechanism of particulate matter effects.

3. Heterogeneity of PM_{10} Composition

Another key element helps to frame the discussion of the relevance of human clinical studies and experimental animal work to establish a mechanism of action of particulate matter in humans. That is the issue of heterogeneity of both the composition of and exposure to particulate matter. Particulate matter is a broad class of physically and chemically diverse substances (as described in Chapter IV). The PM_{10} fraction is composed of two distinct sub-fraction of particle: fine and coarse particles. PM_{10} samplers collect all of the fine particles

and a portion of the coarse ones. There is a fundamental uncertainty regarding which components or properties of particulate matter is essential to the observed effects in humans.

Coarse particles are typically composed of re-suspended dusts from fields and streets and may contain metal oxides of silica, aluminum, magnesium, titanium, and iron. Coal and oil fly ash, calcium carbonate, sodium chloride, sea salt, small pollen, mold spores, and plant parts may also be present. Fine particles are generally composed of sulfate, nitrate, hydrogen ion, elemental carbon, organic compounds, biogenic organic compounds such as terpenes, and metals such as iron, lead, cadmium, vanadium, nickel, copper, and zinc. Some materials which are more typically found in the coarse fraction, may be also found the fine fraction. Similarly, some materials typically found in the fine fraction may also be in the coarse fraction due to particle growth in conditions of high relative humidity (e.g., sulfates). Additionally, the properties of PM_{10} vary greatly from place to place because of differences in source mixes and atmospheric conditions.

Thus unlike a typical experimental paradigm, where the agent to be studied is isolated and the effects of exposure described in well controlled studies, the heterogeneity of the PM_{10} entity forces a different experimental approach. Typically constituents of the fraction are tested individually to see if effects similar to those observed in humans are reproduced. Consequently, animal studies are further weakened in regard to ability to establish a mechanism of action of particulate matter and to either refute or validate epidemiological observation of effect in humans.

4. Dosimetric Heterogeneity

Finally, dosimetric comparisons between laboratory animals and humans, show that there are significant differences in the respiratory architecture and ventilation of the two which adds additional complication to comparisons of experimental and observed data. Ventilation differences coupled with differences in upper airway respiratory tract structure and size, branching pattern, and structure of the lower respiratory tract occur between species as well as between healthy versus diseased states. These differences may result in significantly different patterns of airflow affecting particle deposition patterns in the respiratory tract (CD, Chapter 13). Additionally, inter-species variability in regard to cell morphology, numbers, types, distribution, and functional capabilities between animal and

human respiratory tracks, leads to differences in clearance of deposited particles which may in turn affect the potential for toxicity. (CD, Chapter 13). Consequently the difficulty of using experimental animal data to investigate particulate matter effects is further defined.

5. Lack of Distinct Disease Pathology

The background levels of cardiopulmonary disease as the cause of death for the general population is very high. Given that COPD and heart diseases are frequent causes of death, it is difficult to discern those who die from the additional effects of particulate matter from those already dying from such diseases and to do autopsy to identify a specific pathology associated with particulate matter caused mortality. Even in historical studies involving higher levels resulting in more pronounced effect it is hard to get an adequate characterization of pathology related to particulate matter effects. Thus without such a characterization of the pathology of particulate matter induced mortality, development and validation of appropriate models to study such effects are more difficult.

6. Lack of Appropriate Equivalents to Epidemiological Endpoints

Animal toxicological equivalents of such epidemiological endpoints as hospital admissions and emergency room visits as an indication of morbidity cannot be obtained. Although mortality can be recreated in a laboratory setting, the relevance of mechanism is currently an issue. In addition, there is question as to what the most appropriate measure of particulate matter is in regard to its toxicity. Specifically is it the inhalable mass which is the most relevant metric of the toxic quantity of particulate matter or is it the number of particles which reaches specific targets? Particles may have low inherent toxicity at one size, yet greater potency at another (CD, Chapter 11). A recent study by Chen et al. (1995) confirmed that the number of particles in the exposure atmosphere not just total mass concentration is an important factor in biological responses following acidic sulfate inhalation (CD, Chapter 11). Specifically, ultrafine particles with a diameter of 20 μm have an approximately 6 order of magnitude increased number than a 2.5 μm diameter particle of the same mass concentration (CD, Section 11). Comparisons of particle number and size are shown in Table 11-1 of the CD.

In addition to considerations of dose (inhalability and appropriate metric), the nature of the response to particles and correlations of the appropriate response to susceptible

population are yet to be resolved. Thus, identification of the dosimeter which induces mortality and morbidity has not been elucidated with consequent difficulty interpretation and design of controlled animal and human studies.

Appendix E

CONCENTRATION-RESPONSE RELATIONSHIPS FOR MODEL SENSITIVITY ANALYSIS IN RISK ASSESSMENT

The interpretation of specific concentration-response relationships is understood to be one of the most problematic issues at this time for the assessment of health risks associated with exposure to ambient PM. The approach to addressing this issue taken in the risk assessment discussed in Chapter VI and in the technical support documents (Abt Associates, 1996a,b) is to consider alternative concentration-response models through a sensitivity analysis. The sensitivity analysis is intended to develop ranges of estimated risks, without attempting to develop any single best estimate of health risks One of the elements needed to frame such a sensitivity analysis is the development of alternative PM concentration ranges over which reported concentration-response functions would be applied. Alternative approaches to identifying appropriate PM concentration cut-points which define the lower end of such ranges are discussed below. The application of these approaches to a number of epidemiological studies using PM_{10} and $PM_{2.5}$ indices of exposure for mortality, hospital admissions, and respiratory effects in children is also presented.

A. Alternative Approaches to Defining Concentration Cutpoints

The characterization and interpretation of observed PM concentration-response relationships are of particular importance in adequately assessing risks from ambient PM Varying degrees of uncertainty exist concerning the PM concentration-response relationship. Such uncertainties may limit the ability to discriminate between a range of plausible alternative concentration-response relationships, and this in turn weakens the ability to estimate potential risks associated with exposure to PM, especially at low ambient concentrations[1]. Key issues for consideration include: 1) what tests and procedures have been done to examine the possibility of linear versus nonlinear dose-response relationships; 2) to what degree do statistical uncertainty and inadequate power preclude exclusion of different alternative concentration-response

[1] The terminology of "low" or "lower" concentrations is used to simply refer to observed PM concentrations generally within the lower half to twenty-five percentile of the reported observations, rather than any concentrations "lower" than those observed

functions; and 3) how factors such as measurement error or copollutants may potentially obscure an underlying concentration-response relationship substantially different and possibly less linear than the reported apparently linear relationship.

Epidemiological investigations of PM generally have taken several approaches to addressing the shape of the concentration-response relationship. A number of investigators have addressed possible non-linearity in this relationship by the use of categorical variables (CD, p. 12-18). Using categorical variables (e.g., quintiles, quartiles) disaggregates the PM concentration spectrum into discrete ranges, and allows risk estimates to be generated independently for each interval. This may increase the likelihood for detecting those ranges of PM concentrations that may be associated with little risk from those associated with substantially higher risk However, by partitioning the PM data into smaller groups, this procedure may increase the impact of measurement error and reduce the statistical power of the analyses. (CD, p.12-18). More recent studies (1993-on) have used various nonparametric approaches--locally estimated smoothing, cubic splines, etc.-- applicable in Generalized Additive Models to allow better assessment of nonlinearities in the PM concentration-response relationships, as well as control for confounders such as weather, season, and time trends (CD, p. 12-19). In addition, potential nonlinearity in these nonparametric concentration-response models are often assessed through statistical tests as well.

In the base case risk analyses described in Chapter VI, reported linear concentration-response functions have been applied across the range of reported PM concentrations, when available, with estimated risk never being quantified below estimate of PM background concentrations. However, given the uncertainty concerning PM concentration-response relationships, especially at lower concentrations, alternatives to the base case assumptions are examined through a sensitivity analysis. Of particular interest is the possibility of substantial nonlinearity -- i.e., a less steep or zero slope in PM concentration-response relationships at lower concentrations. To address such possibilities, concentration-response information from key studies can be assessed to determine for which concentrations it may be most reasonable to posit a reduced or zero slope in the concentration-response relationship.

Several approaches to determine possible cutpoint PM concentrations of particular interest for use in modeling alternative concentration-response relationships are discussed below. Staff recognizes that no consensus exists on the best approach to identify, test, or interpret the effect of such cutpoints on concentration-response information. Detailed evaluation of concentration-response relationships is made more difficult by a lack of information on data densities and confidence intervals (CD, 12-310-311). Given these circumstances, alternative approaches are used to generate a range of potential cutpoints, with no attempt to identify the best or most appropriate cutpoint for risk assessment purposes.

The overall approach taken here is to evaluate the extent to which detailed concentration-response information from key studies suggests statistical limitations or nonlinearities in PM concentration-response relationships over the range of PM concentrations observed in the studies. This evaluation focuses on lower concentrations ranges, given that several concerns raised about PM concentration-response relationships center on whether reported linear functions may be disguising flat or essentially flat relationships (i.e., show no increase in risk) in the lower portions of the concentration-response relationship Three approaches, identified as "lower limit of detection," "minimum mean concentration," and "visual interpretation" are defined below. These approaches have been used to identify reasonable cutpoint concentrations for the concentration-response model sensitivity analysis.

- Lower Limit of Detection: A number of studies present concentration-response information which suggests a generally monotonic increase in response as PM increases (CD, p. 12-23, 12-309). Even if such studies for which the concentration-response information does not suggest a substantially nonlinear relationships across the range of data, the ability to detect any potential effects thresholds or other nonlinearities is limited by the data (CD, p. 12-309-311). For example, plots of RR as a function of the quantile PM concentrations are inherently not able to detect any nonlinearities that may be present within the lowest quantile (CD, p.12-309-310). Thus, for studies that only present concentration-response information in quantile plots and do not show apparent nonlinearities, the maximum concentration (the 20th or 25th percentile value for quintile

and quartile plots, respectively) of the lowest quantile can be considered to be the lower limit of detection of possible nonlinearities.

Reported concentration-response relationships using nonparametric smoothed curves allow a much better assessment of nonlinearities in the concentration-response model (CD p.12-19). Statistical tests can be performed to indicate whether any fluctuations seen in these smoothed curves reflect a substantially nonlinear overall relationship that is statistically discriminable from a linear relationship . Limited numbers of air quality observations can reduce the power of this test, however, and even the visual presentations of smoothed curves are not able to discriminate nonlinearities in regions where there are not enough data points to obtain a stable curve shape (CD, p. 12-310). For studies in which an overall linear relationship cannot be statistically rejected and substantial nonlinearities are not evident, the lower limit for detection of nonlinearity may be considered to be around the 10th percentile. Use of the 10th percentile reflects the greater sensitivity of these smoothing methods compared to quantile analyses to examine whether an observed linear relationship appears to hold toward the lower end of the range of observed concentrations.

- Minimum Mean Concentration: The second approach considered is to use a central tendency concentration as the cutpoint of interest, which is generally available for all studies. The mean (or median) concentration may serve as a reasonable cutpoint of increased PM health risk since at this point there is generally the greatest confidence (i e., the smallest confidence intervals) in the association and the reported RR estimates. The mean concentration considered by staff as most informative to test implications of potential alternative concentration-response functions is the minimum mean concentration associated with a study or studies reporting statistically significant increases in risk across a number of study locations, provided that the monitoring data is sufficient and representative of the area to which the RR estimate is applied. Alternatively, averages of mean concentrations across a group of locations or studies may be more appropriate if location-specific data are inadequate.

- Visual Interpretation: Concentration-response relationships reported by some studies sometimes visually suggest that nonlinearities may exist within the range of the data, even when PM concentrations are significantly associated with health effects in a linear model. Caution is warranted in any visual interpretation of available PM concentration-response information, given the limited information provided and the amount of measurement error that often is involved (CD, p.12-309-311). Use of quantiles can exacerbate this problem as it might increase the likelihood of identifying an apparent nonlinearity in the effect estimate entirely due to increased uncertainty in each quantiles' smaller sample size.

In conjunction with the use of these methods to identify cutpoints for estimating adjusted concentration-response functions, consideration is given to adjustments to the slope of the reported concentration-response relationship. If an underlying nonlinearity is present, the reported slope of a linear concentration-response relationship would change both below the cutpoint concentration (where the reported slope would be too high) and above the cutpoint concentration (where the reported slope would be too low). Adjustments to the slopes of such segments in concentration-response relationships used in this sensitivity analysis are described in the technical support documents (Abt Associates, 1996a,b).

B Concentration Cutpoints from Key Studies

The three methods described above were applied where appropriate to the studies used in the risk assessment (Table VI-2 in section VI.B of this Staff Paper), including both PM_{10} and $PM_{2.5}$ studies where applicable, for mortality, hospital admissions, and respiratory symptoms effects. As outlined below, judgments are necessary to apply such methods, and staff recognizes that other judgments could reasonably be made. However, staff believes that the approach taken here is reasonable and results in selected cutpoints that are useful for the purpose of defining sensitivity analyses that help to address uncertainties in the quantitative assessment of risks based on the available epidemiological evidence. Following the identification of a number of potential cutpoints from these alternative approaches, summarized in Tables E-1 and E-3, the last section condenses this information into a few selected cutpoints, for use in the sensitivity analyses presented in section VI.C of this Staff Paper.

1. Concentration-Response Relationships Associated with Short-Term PM Exposures

The potential concentration cutpoints identified in the following discussion of short-term exposure studies are summarized in Table E-1 for both PM_{10} and $PM_{2.5}$ studies.

a. PM_{10} Mortality Studies

The five studies, conducted in ten locations, included in Table IV-2 which reported PM_{10} mortality relationships were examined.

Lower Limit of Detection: This method was applied to the two studies (Birmingham, Schwartz 1993a; Utah Valley, Pope et al., 1992 and Pope and Kalkstein, 1995) which reported concentration-response relationships between mortality and PM_{10} concentrations. Although some nonlinearity may be evident in the nonparametric smoothed curve reported by Schwartz (1993a; 1994g) in the central portion of the range, from approximately 40 - 60 $\mu g/m^3$ (Fig E-1), these are concentrations at which mortality risk is elevated (Samet et al., 1995). Tests failed to indicate the overall PM-mortality relationship could be statistically discriminated from a possible linear relationship (p value of 0.7 for rejecting linearity). The 10th percentile concentration in Birmingham was reported to be 21 $\mu g/m^3$ (Schwartz, 1993a). The nonparametric smoothed curve reported in Pope and Kalkstein's (1995) reanalysis of Utah Valley mortality (Fig. E-2) was also reported as not significantly different from linear ($p>0.5$). In this study, the 10th percentile concentration was not directly reported but is likely to be approximately 20 $\mu g/m^3$, the approximate midpoint of the lowest quintile reported for Utah Valley by Samet et al. (1995). These concentrations are consistent with the lower limit of detection for nonlinearities of 20 $\mu g/m^3$ PM_{10} identified in the CD discussion of PM mortality exposure-response functions (CD, 12-310).

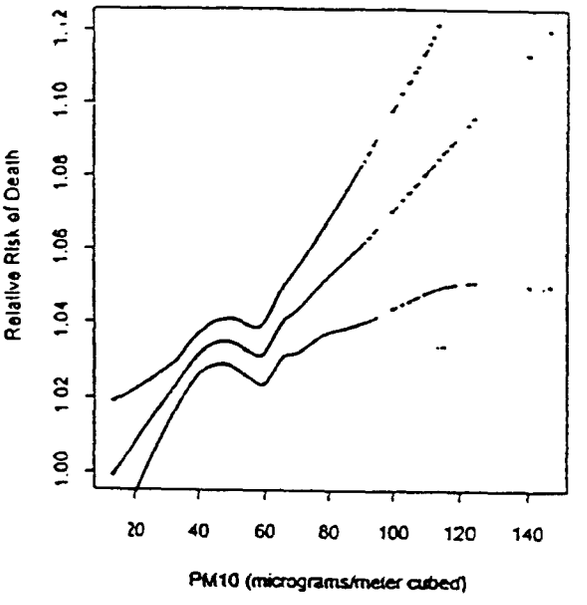
Minimum Mean Concentration: The lowest mean PM_{10} concentration reported in these mortality studies was 30 $\mu g/m^3$, from Schwartz et al. (1996a). This combined mean, averaged across the cities in the study, rather than the lowest mean concentration from any one city in this study, was judged to be appropriate to use for this purpose, since the single monitors used to characterize air quality for each city were sited in locations that may underestimate the average

Table E-1. Potential Concentration Cutpoints of Interest for Assessing the Sensitivity of Risk Estimates Derived from Short-Term Exposure Studies

	TOTAL MORTALITY		HOSPITAL ADMISSIONS		RESPIRATORY SYMPTOMS	
Alternative Approaches	Conc. ($\mu g/m^3$)	Reference	Conc. ($\mu g/m^3$)	Reference	Conc. ($\mu g/m^3$)	Reference
PM_{10} STUDIES						
Lower Limit of Detection	20	Pope & Kalkstein, 1996	19	Schwartz, 1994e	13	Schwartz et al., 1994
	21	Schwartz, 1994g	30	Schwartz & Morris, 1996		
Minimum Mean Concentration	30	Schwartz et al., 1996a	36	Schwartz, 1994f	30*	Schwartz et al., 1994
Visual Interpretation	37	Pope et al., 1992	37	Schwartz, 1994d	-	
	42	Samet et al., 1995				
	43***	Cifuentes and Lave, 1996				
	34-57***	Samet et al., 1995				
$PM_{2.5}$ STUDIES						
Lower Limit of Detection	9	Schwartz, et al., 1996a	13**	Burnett et al., 1995	12	Schwartz et al., 1994
Minimum Mean Concentration	18	Schwartz, et al., 1996a	19	Thurston et al., 1994	18*	Schwartz et al., 1994
			15**	Burnett, et al., 1995		
Visual Interpretation	29***	Cifuentes and Lave, 1996	-		-	
	22-36***	Samet et al., 1995				

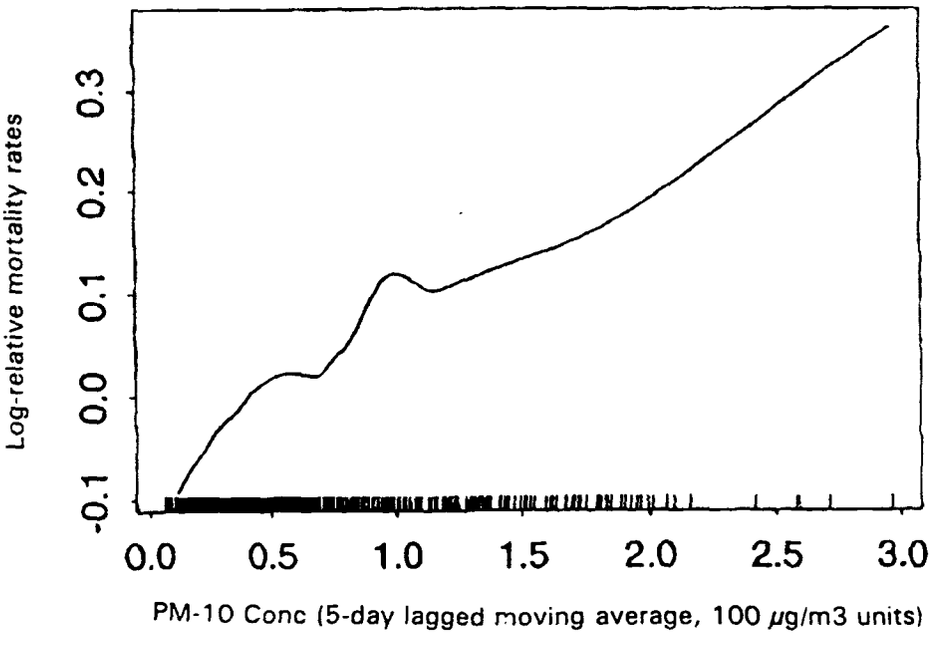
Footnotes: * Median estimate.
** Converted from sulfate data.
*** Converted from TSP data. Range for Samet et al., 1995 reflects elderly and all mortality results, respectively.

FIGURE E-1. RELATIONSHIP BETWEEN RELATIVE RISK OF DEATH AND PM-10 IN BIRMINGHAM (SCHWARTZ, 1994g)



The smoothed plot of the relative risk of death versus PM10 in Birmingham, Alabama, after controlling for smoothed functions of time, temperature, and dew-point temperature (and day-of-week dummy variables) in a generalized additive model. Pointwise one-standard-error confidence intervals are also shown.

FIGURE E-2. RELATIONSHIP BETWEEN RELATIVE RISK OF DEATH AND PM-10 IN UTAH VALLEY (POPE AND KALKSTEIN, 1996)



concentrations experienced across the cities as a whole The mean concentrations in the three cities in which statistically significant results were reported ranged from 24 - 32 $\mu g/m^3$.

Visual Interpretation: A quintile analysis of a Utah Valley study provided by Pope et al., (1992) suggests that any increased risk associated with the second quintile may be less than the increases associated with the three higher concentration quintiles (Fig. E-3). Alternatively, Samet et al. (1995), using quintiles in a slightly different approach, reported that mortality appeared to increase in the two highest quintiles only (Table E-2). This information would suggest a possible cutpoint of interest in the range of 37 (midpoint of quintile showing reducing increased risk in Fig. E-3) to 42 $\mu g/m^3$ (maximum concentration of quintile showing no increase in risk in Table E-2) The staff judges that the weight given these observations should take into consideration the more recent Utah Valley results discussed above, given the greater sensitivity of the nonparametric methods that have been subsequently been applied to the Utah Valley data.

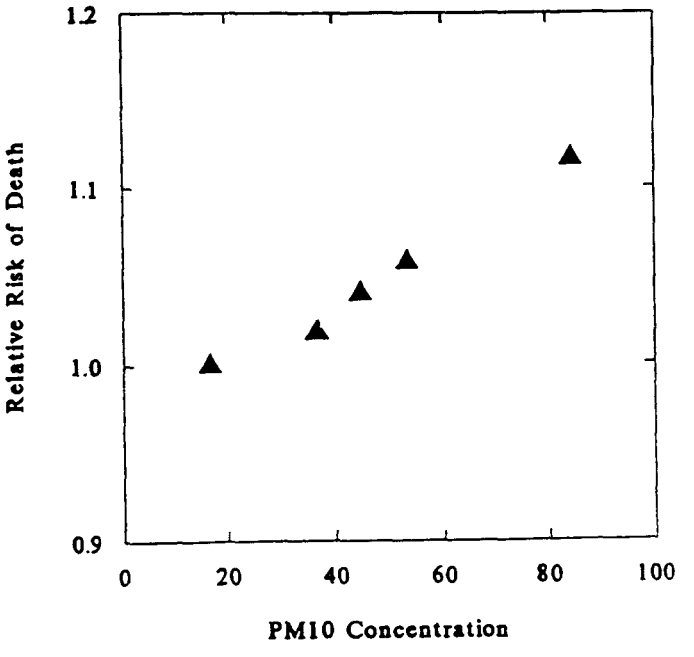
Various analyses have been done on data from Philadelphia examining PM-mortality relationships using TSP as the measure of PM. Table E-1 also contains converted PM_{10} "cutpoint equivalents" from the TSP findings of these studies that examined TSP concentration-response relationships when associated copollutants were included in the model. There are substantial uncertainties both in interpreting this TSP data in relation to smaller particle indicators (PM_{10}, $PM_{2.5}$) (CD, p. 243), especially when evaluation between copollutants is attempted, and inherent in converting TSP findings into estimates of $PM_{2.5}$. The method and issues involved in deriving these PM_{10} "cutpoint equivalents" are discussed in Section C.

b. PM_{10} Hospital Admissions Studies

Studies conducted in seven locations included in Table IV-2 reporting respiratory and cause-specific hospital admissions relationships with PM_{10} were examined.

Lower Limit of Detection: Nonparametric smooth curves of the concentration-response relationships between PM_{10} and pneumonia (Fig. E-4) and COPD hospital admissions in the elderly in Birmingham have been reported by Schwartz (1994e). No apparent nonlinearities are observed, and the relationships are not statistically distinguishable from linearity ($p \geq 0.25$). The 10th percentile concentration is approximately 19 $\mu g/m^3$. A quartile plot of an analysis of cardiac

FIGURE E-3. RELATIONSHIP BETWEEN RELATIVE RISK OF DEATH AND PM-10 IN THE UTAH VALLEY (POPE ET AL., 1992)



Relative risk of death, by quintile of PM_{10} concentration.

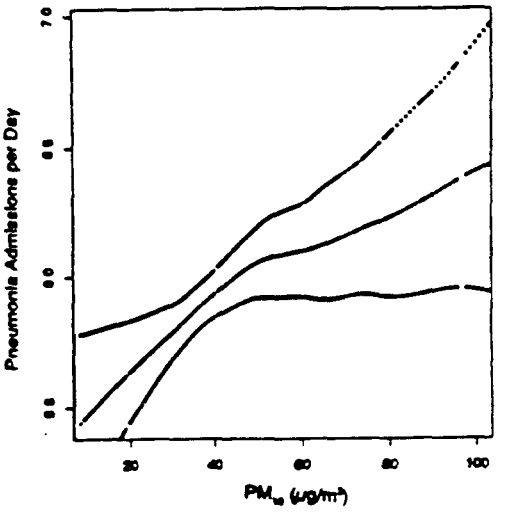
TABLE E-2. RELATIONSHIP BETWEEN RELATIVE RISK OF DEATH AND PM-10 IN UTAH VALLEY (SAMET ET AL., 1995)

Table 28. Relative Risks and Confidence Intervals by Quintile of Five-Day-Lagged Average PM_{10} for Utah Valley Total Mortality, April 1985–December 1989, Controlling for Weather

PM_{10} Quintile	Range ($\mu g/m^3$)	Relative Risk	95% CI[a]
1	11.2–27	1.00	
2	27–34.2	0.98	(0.89. 1.09)
3	34.2–42.2	0.99	(0.89. 1.09)
4	42.2–56.2	1.04	(0.94. 1.16)
5	56.2–296	1.08	(0.97. 1.21)

[a] Corrected for constant over-dispersion.

FIGURE E-4. RELATIONSHIP BETWEEN RELATIVE RISK OF PNEUMONIA ADMISSIONS AMONG THE ELDERLY AND PM-10 IN BIRMINGHAM (SCHWARTZ, 1994e)



Nonparametric smooth of counts of pneumonia admissions (persons per day) versus the concentration of airborne particulate matter with an aerodiameter of ≤10 μm (PM_{10}) after controlling by regression for long-term temporal patterns and weather. The pointwise 95 percent confidence limits of the smooth curve are also shown.

FIGURE E-5. RELATIONSHIP BETWEEN ISCHEMIC HEART DISEASE ADMISSIONS AMONG THE ELDERLY AND PM-10 (SCHWARTZ AND MORRIS, 1996)

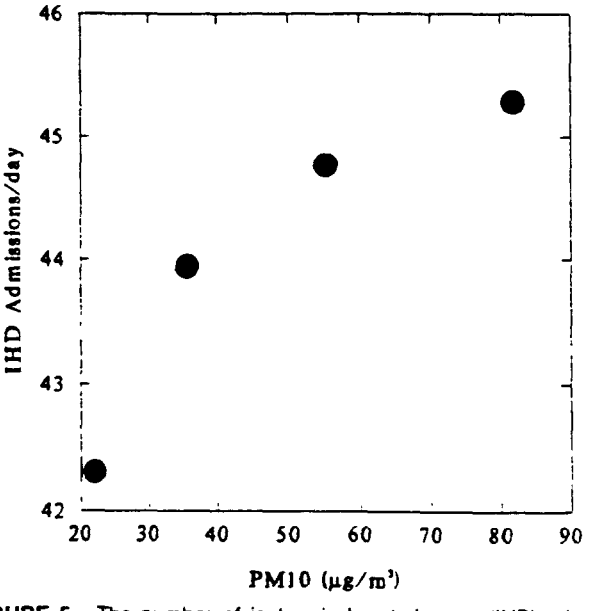


FIGURE 5. The number of ischemic heart disease (IHD) admissions of the elderly in Detroit, Michigan, during 1986–1989 by quartile of particulate matter with an aerodiameter of ≤10 μm (PM_{10}). The plot is after adjusting for all other covariates.

hospital admissions for the elderly in Detroit (Schwartz and Morris, 1996) displays increased risk at and above the second quartile (Fig. E-5), with a 25th percentile concentration of 30 $\mu g/m^3$.

Minimum Mean Concentration: The year-long study with the lowest mean PM_{10} concentration, 36 $\mu g/m^3$, reporting significant associations was the Schwartz (1994f) study of COPD and pneumonia hospital admissions among the elderly in Minneapolis. This compares closely to the mean concentration was reported by Thurston et al. (1994) in their study of summertime hospital admissions in Toronto, with a PM_{10} mean concentration of 33 $\mu g/m^3$ averaged across three summers.

Visual Interpretation: The quartile plot of Schwartz (1994d) for elderly pneumonia hospital admissions in Detroit (Fig. E-6) indicates that pneumonia risk may not increase as sharply for the second quartile of PM concentrations as for subsequent quartiles. The midpoint concentration of this second quartile is 37 $\mu g/m^3$.

c. PM_{10} Respiratory Symptoms Studies

The two studies listed in Table VI-2 reporting PM_{10} associations with respiratory symptoms were examined.

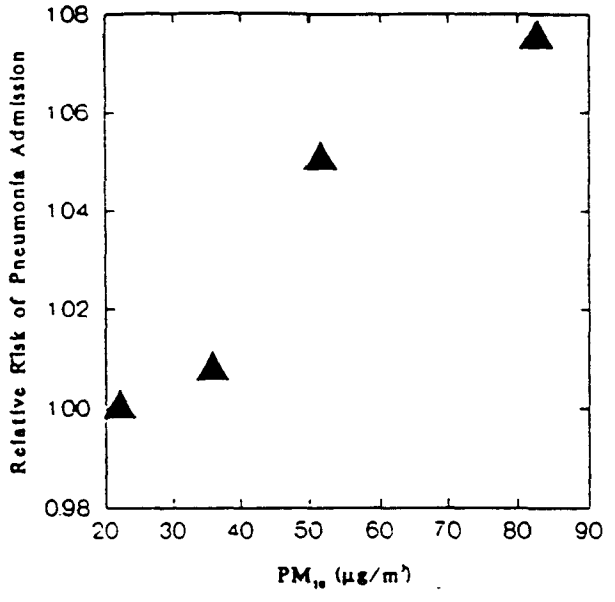
Lower Limit of Detection: The Six City study (Schwartz et al., 1994) provides nonparametric smoothed plots for PM_{10} associations with cough (Fig. E-7) and lower respiratory symptoms (Fig. E-8) Statistical tests of deviations from linearity for these associations are not significant. However, the ability to detect nonlinearities is not likely to extend below the 10th percentile concentration of 13 $\mu g/m^3$ PM_{10}

Minimum Mean Concentration: The Six City study (Schwartz et al., 1994) reports the lower mean PM_{10} concentration of 30 $\mu g/m^3$.

d. $PM_{2.5}$ Mortality Studies

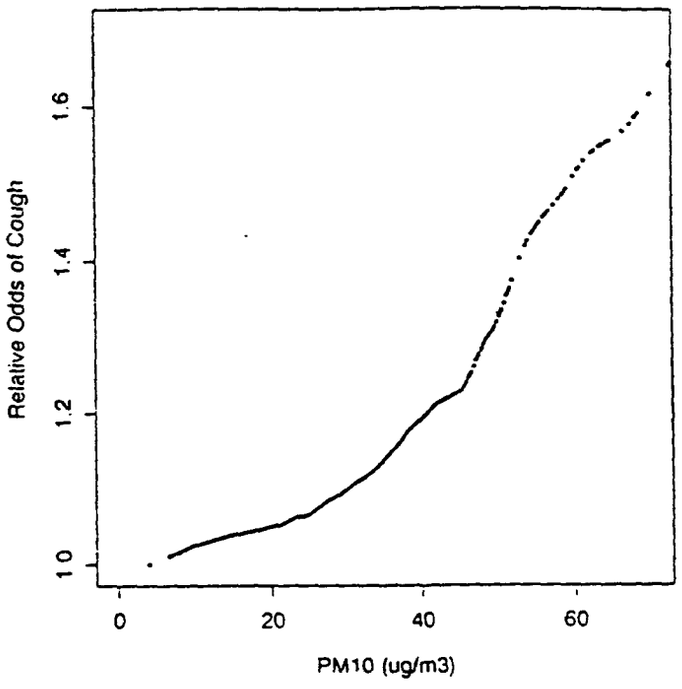
There is less available information concerning $PM_{2.5}$ concentration-response relationships for mortality in comparison to PM_{10}. However, the Harvard Six Cities study (Schwartz et al., 1996a) reports significant associations between $PM_{2.5}$ and mortality in a combined analysis of six cities, as well as associations in individual cities, that indicate that $PM_{2.5}$ mortality associations were relatively consistent in magnitude and statistically significant for three locations (Boston, St. Louis, and Knoxville) with mean concentrations ranging from approximately 16 to 21 $\mu g/m^3$

FIGURE E-6. RELATIONSHIP BETWEEN RELATIVE RISK OF PNEUMONIA ADMISSION AMONG THE ELDERLY AND PM-10 IN DETROIT (SCHWARTZ, 1994d)



The relative risk of pneumonia admissions in the elderly in Detroit, Michigan, by quartile of PM_{10} is shown. The plot is after adjusting for all other covariates. A stepped response with increasing dose is evident, with no evidence for a threshold.

FIGURE E-7. RELATIONSHIP BETWEEN THE ODDS OF COUGH INCIDENCE VERSUS PM-10 CONCENTRATION FROM THE SIX CITY STUDY (SCHWARTZ ET AL., 1994)



Relative odds of incidence of coughing smoothed against 3-d mean PM_{10} ($\mu g/m^3$), controlling for temperature, city, day of the week, and ozone concentration.

FIGURE E-8. RELATIONSHIP OF THE ODDS OF LOWER RESPIRATORY SYMPTOMS INCIDENCE VERSUS PM-10 CONCENTRATION FROM THE SIX CITY STUDY (SCHWARTZ ET AL., 1994)

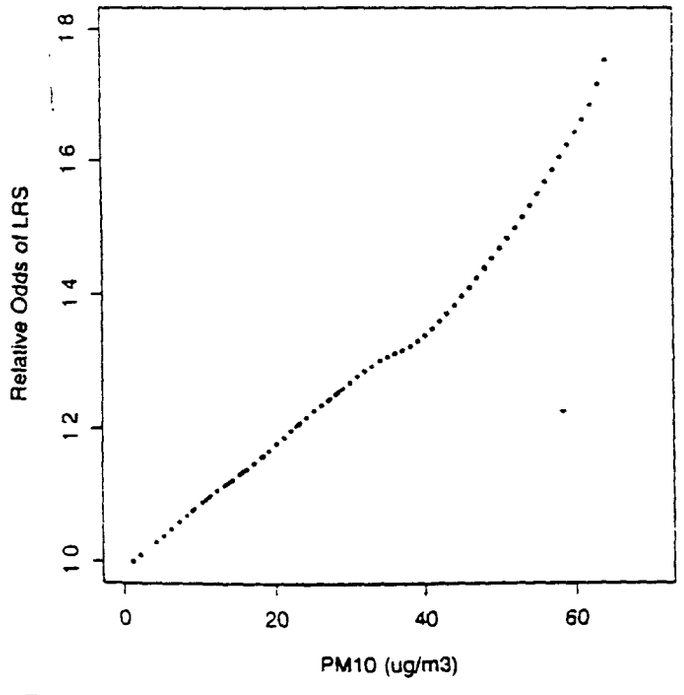


Figure 3. Relative odds of incidence of lower respiratory symptoms (LRS) smoothed against 24-h mean PM_{10} ($\mu g/m^3$) on the previous day controlling for temperature, day of the week, and city.

$PM_{2.5}$ No concentration-response curves were provided, precluding any visual interpretation of results presented in terms of $PM_{2.5}$.

Lower Limit of Detection: For this Six City study, a potential cutpoint could be chosen at the 25th percentile concentration, 9 $\mu g/m^3$, consistent with similar interpretations of studies reporting results in terms of quartile plots.

Minimum Mean Concentration: The $PM_{2.5}$ mean of the combined results from this Six Cities study is 18 $\mu g/m^3$.

Visual Interpretation: Consistent with the approach used above for PM_{10} mortality and discussed more fully in Section C, Table E-1 also gives potential $PM_{2.5}$ "cutpoint equivalents" based on conversions of recent reanalyses of TSP/copollutant concentration-response relationships.

e. $PM_{2.5}$ Hospital Admissions Studies

Minimum Mean Concentration: The only study to examine respiratory hospital admissions directly in terms of $PM_{2.5}$ (Thurston et al., 1994) reported mean concentrations for three summers ranging from approximately 16 to 22 $\mu g/m^3$, with an overall average of approximately 19 $\mu g/m^3$. This is roughly consistent with the more uncertain estimate obtained from the Burnett et al. (1995) study of sulfates and respiratory and cardiac admissions. The mean sulfate concentration of 4.4 $\mu g/m^3$ in that study roughly corresponds to an estimated $PM_{2.5}$ concentration of 15 $\mu g/m^3$.

Lower Limits of Detection: The only study to which this approach can be applied is the Burnett et al. (1995) sulfate study which reports that the respiratory and cardiac hospital admissions from the third quartile were statistically significantly higher than those from the first two quartiles combined. The maximum concentration associated with the bottom two quartiles was approximately 3.0 $\mu g/m^3$ sulfate, the 50th percentile value for the nine Ontario monitoring sites used in the study. To express this finding in terms of a potentially relevant $PM_{2.5}$ cutpoint of interest, a site-specific conversion between SO4 and $PM_{2.5}$ was made using conversion factors for the three largest cities in the study (Toronto, Ottawa, and Windsor), resulting in a $PM_{2.5}$ concentration of roughly 13 $\mu g/m^3$.

f. $PM_{2.5}$ Respiratory Symptoms Studies

Lower Limit of Detection: The Six City respiratory symptoms study (Schwartz et al., 1994) found significant relationships between $PM_{2.5}$ and cough and lower respiratory symptoms in children, although it did not provide either separate quantile or nonparametric smoothed plots for $PM_{2.5}$. Consistent with the approach taken for $PM_{2.5}$ mortality, a potential cutpoint could be chosen at the 25th percentile concentration of 12 $\mu g/m^3$ for this study.

Minimum Mean Concentration: The $PM_{2.5}$ mean concentration for this study (Schwartz et al., 1994) was 18 $\mu g/m^3$.

2. Concentration-Response Relationships Associated with Long-Term PM Exposures

The potential concentration cutpoints identified in the following discussion of short-term exposure studies are summarized in Table E-3 for both PM_{10} and $PM_{2.5}$ mortality studies.

Lower Limit of Detection: The Dockery et al. (1993) Six City study provides plots of long-term mean fine particle concentrations versus adjusted mortality risk for PM_{10} and $PM_{2.5}$. For PM_{10}, increased risks from particles may extend as low as 24 $\mu g/m^3$, the mean concentration for Watertown, which shows an increase in relative risk compared to Portage (Fig. E-9). For $PM_{2.5}$, increased risks may extend as low as 12.5 $\mu g/m^3$, the mean $PM_{2.5}$ concentration for Topeka, which shows a slight increase in relative risk compared to Portage (Fig. E-10).

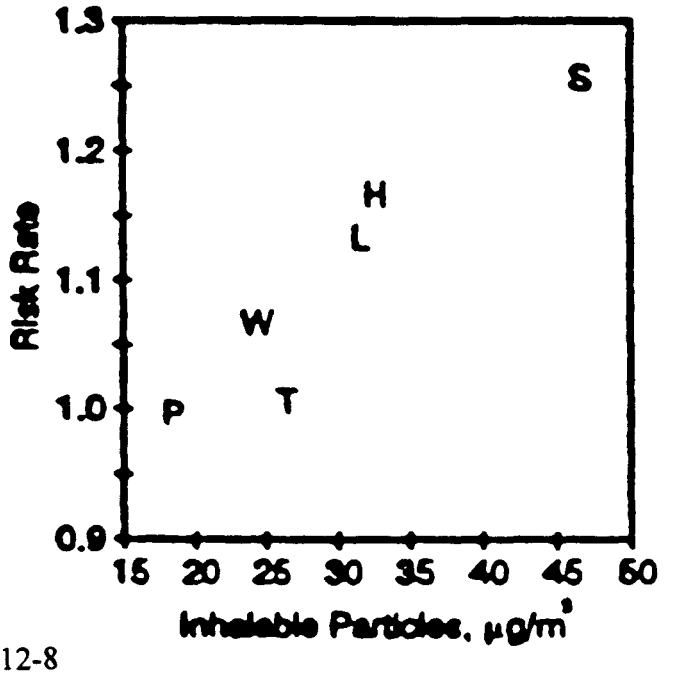
Minimum Mean Concentration: The mean PM_{10} concentration for the Six City study (Dockery et al., 1993) as a whole was 30 $\mu g/m^3$ The mean $PM_{2.5}$ concentration for the Six Cities study (Dockery et al., 1993) and the mean of the median $PM_{2.5}$ concentrations for each city in the ACS study (Pope et al., 1995) were both reported as 18 $\mu g/m^3$.

Visual Interpretation: For PM_{10}, a case might be made from visually inspecting the results of the Six City study (Dockery et al., 1993) that risk consistently increases only beginning with St. Louis, with a long-term PM_{10} mean of approximately 32 $\mu g/m^3$. For $PM_{2.5}$, a similar case might be made that risk consistently increase beginning with Watertown, with a long-term $PM_{2.5}$ mean of approximately 15 $\mu g/m^3$. Such comparisons, however, are limited by the small number of cities in the study. The ACS study (Pope et al., 1995) provides concentration-response information for $PM_{2.5}$ which appears to more consistently increase at concentrations above the median $PM_{2.5}$ concentration of approximately 15 $\mu g/m^3$ (Fig E-11).

Table E-3. Potential Concentration Cutpoints of Interest for Assessing the Sensitivity of Risk Estimates Derived from Long-Term Exposure Studies

Alternative Approaches	TOTAL MORTALITY Conc. ($\mu g/m^3$)	Reference
PM_{10} STUDIES		
Lower Limit of Detection	24	Dockery et al., 1993
Minimum Mean Concentration	30	Dockery et al., 1993
Visual Interpretation	32	Dockery et al., 1993
$PM_{2.5}$ STUDIES		
Lower Limit of Detection	12.5	Dockery et al., 1993
Minimum Mean Concentration	18	Dockery et al., 1993
	18	Pope et al., 1995
Visual Interpretation	15	Dockery et al., 1993
	15	Pope et al., 1995

FIGURE E-9. **RELATIONSHIP BETWEEN MORTALITY RISK RATIOS AND INHALABLE PARTICLES ($PM_{15/10}$) IN THE SIX CITY STUDY (DOCKERY ET AL., 1993)**



Source: CD, Figure 12-8

FIGURE E-10. **RELATIONSHIP BETWEEN MORTALITY RISK RATE RATIOS AND PM-2.5 IN THE SIX CITY STUDY (DOCKERY ET AL., 1993)**

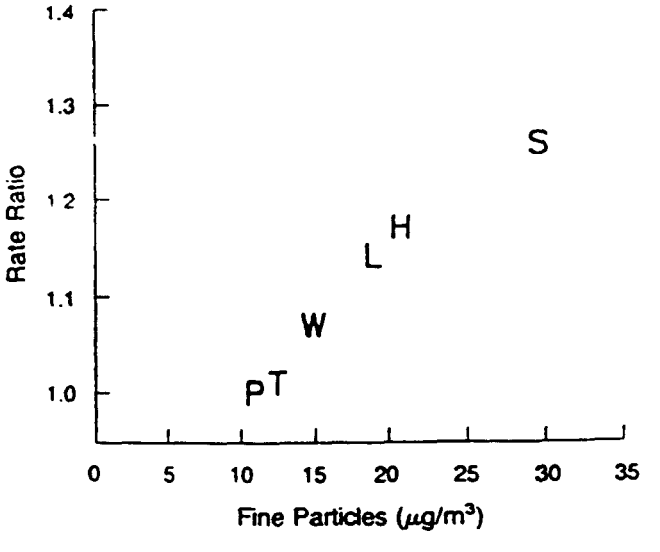
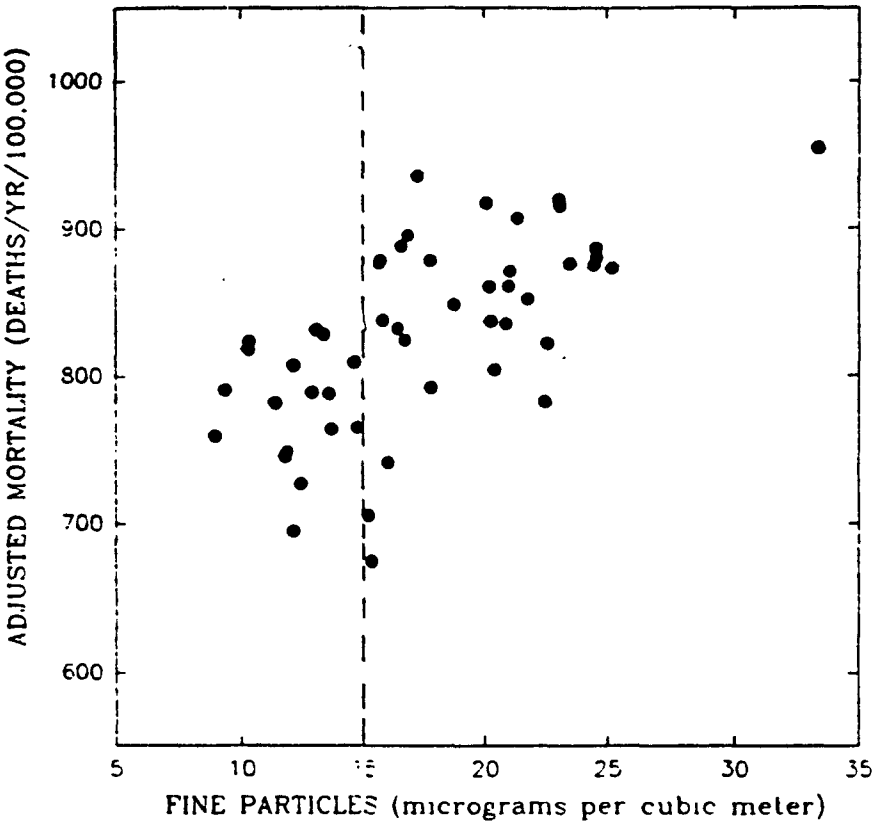


FIGURE E-11. RELATIONSHIP BETWEEN ADJUSTED MORTALITY AND PM-2.5 IN THE AMERICAN CANCER SOCIETY STUDY (POPE ET AL., 1995)



Age-, sex-, and race-adjusted population-based mortality rates for 1980 plotted against mean fine particulate air pollution levels for 1979 to 1983. Data from metropolitan areas that correspond approximately to areas used in prospective cohort analysis.

C. Potential Effects of Copollutants or PM Measurement Error on Concentration- Response Relationships

The approach carried out in the sections above for assessing whether underlying nonlinearities exist in PM concentration-response relationships (e.g., resulting from the presence of biological thresholds) uses existing reported concentration-response relationships. The large majority of these relationships were derived considering ambient PM concentrations alone (e.g., without simultaneous inclusion of copollutants) As discussed in Section V.E., several commentors have raised the issue that if the observed concentration-response relationship reflect PM-health effects relationships in which PM is serving as a proxy for other non-considered factors (e g., the effects of coassociated pollutants, or of total personal exposure to particles) that may causally give rise to health effects, then analyses of observed concentration-response data that do not fully take into account the potential role of these other factors may fail to reveal a genuine underlying nonlinear relationship between ambient PM and health effects. The failure to consider these factors, if they have a genuine causal role, may potentially serve to "disguise" nonlinear concentration-response relationships, and might result in an apparently linear PM concentration-response relationship in cases in which a genuine nonlinear relationship existed.

The two factors advanced as issues of particular concern to consider in this regard have been the influence of coassociated pollutants (Samet et al, 1995; Samet et al., 1996b; Moolgavkar et al , 1995b; Moolgavkar and Luebeck, 1996, Cifuentes and Lave, 1996, Lipfert and Wyzga, 1995b), and the potential influence of different types of measurement error. Measurement error in this context includes concerns over the potential implications that measurements of ambient PM may not accurately reflect total personal exposures to particles, either exposures to all particles or at a mininum a subset of particles including particles of nonambient origin (e.g., from indoor combustion sources). In both the case of potential effects of copollutants and of measurement error, concerns have been raised that available concentration-response relationships may create erroneous estimates of PM-health effects relationships for risk analyses purposes by failing to consider the possibility that these unacknowledged factors may alter the shape of the estimated PM concentration-response relationship.

1. Potential Effects of Copollutants on Determining Effects Thresholds

Several authors have evaluated concentration-response relationships for particles while simultaneously including other combustion source copollutants as variables in the health effects concentration-response regression. Samet et al. (1995) reanalyzed information from Philadelphia for 1973-1980 simultaneously considering SO_2 in the model. One form of presentation they give to their results leads to the question of whether potential TSP effects thresholds exist when copollutants are considered simultaneously. Figure 11 of their report appears to indicate a linear response between mortality and TSP only for TSP > 100 $\mu g/m^3$ (all ages) or TSP > 60 $\mu g/m^3$ (age 65+) (CD, p. 12-311). However, the CD also acknowledges that other approaches undertaken by Samet et al. (1995), such as nonparametric smoothed surfaces simultaneously displaying TSP and SO_2 relationships (CD, pp. 335-344), differs significantly from the simple threshold model shown in their Figure 11 (CD, p. 12-311).

Cifuentes and Lave (1996) analyzed a later period in Philadelphia simultaneously considering two copollutants in the model, SO_2 and O_3. They presented a number of results from several different approaches investigating potential thresholds. The CD finds that Cifuentes and Lave (1996) provides no precise estimate of a change point in the TSP mortality relationship, with the lower portion of a potential cutpoint relationship not showing significance below 60 $\mu g/m^3$ and showing general significance at 90 $\mu g/m^3$ and above (CD, p. 301, Figure 12-32). The study's authors particularly call out the concentration of 78 $\mu g/m^3$ as a concentration below which "the effects of TSP decreased significantly," a concentration representing roughly the midpoint of the range identified by the CD. Although as pointed out by the CD, the methods applied by Cifuentes and Lave do not necessarily imply a slope of zero below the tested cutpoints (CD, pp. 301-302), this central value of 78 $\mu g/m^3$ TSP will be used to summarize the results of their findings in the cutpoint sensitivity analyses for the risk analysis, which does presume a slope of zero below the cutpoint (Appendix F).

To enable the general findings of Samet et al (1995) and Cifuentes and Lave (1996) to be considered in the risk analysis, conversion of their TSP cutpoint findings to fine particles ($PM_{2.5}$) were carried out. Such an approach involves substantial uncertainties both in determining both an appropriate conversion factor to express TSP results as $PM_{2.5}$ as well as the possibility that

substantially different results may have been obtained in the copollutant models if $PM_{2.5}$ data had been available for inclusion in the model rather than the less robust surrogate measure of TSP, especially when discriminations between the particle measure and an associated copollutant are attempted simultaneously in the health model. As indicated by the CD, there is less basis for assuming that analogous results would be obtained for other PM indices, such as PM_{10} or $PM_{2.5}$ (CD, p. 343).

With these concerns in mind, conversion factors were derived from information in Table 6-13 of the CD to allow rough estimates of the potential impacts of application of cutpoints based on the TSP-copollutant analyses of Samet et al. (1995a) and Cifuentes and Lave (1996) to be considered. The Samet et al. (1995) findings were represented by converting the all mortality and elderly 2-D nonparametric smoothed plot findings (reported in Figure 11 of their report) to $PM_{2.5}$ by using the $PM_{2.5}$/TSP ratio (for TSP > 80 $\mu g/m^3$) of 0.36 for the Inhalable Particle Network (IPN), 1979-1983, which provided a rough central estimate $PM_{2.5}$/TSP ratio of 0.36 (CD, Table 6-13) The Cifuentes and Lave (1996) findings were converted to an estimated $PM_{2.5}$ concentration by using the $PM_{2.5}$/TSP ratio available from a site reported to AIRS, 1987-1990 (CD, Table 6-13). Applying these conversions, the Samet et al. (1995) findings could be interpreted as suggesting potential cutpoints in the range of 22 - 36 $\mu g/m^3$ for elderly and all age mortality, respectively, and the Cifuentes and Lave (1996) findings could be interpreted as suggesting the potential for a cutpoint of roughly 29 $\mu g/m^3$ for all age mortality.

Comparable conversions based on Table 6-13 also can be done for PM_{10} , although some additional concern exists for deriving a PM_{10} /TSP conversion factor for Samet et al. (1995) in that the IPN dataset that overlapped the period of study provided information only in terms of PM_{15}. Use of a single monitor operating two years after the study (1982-1983), which was not used in determining the $PM_{2.5}$ conversion factor for Samet et al. (1995) presented previously because the earlier, more extensive network was available, would provide a PM_{10} /TSP conversion factor of approximately 0.57. Use of this factor and a PM_{10} /TSP conversion factor of 0.53 for the AIRS 1987-1991 site provides possible PM_{10} cutpoint concentrations of approximately 34 - 57 $\mu g/m^3$ for the Samet et al. (1995) findings and approximately 43 $\mu g/m^3$ for the Cifuentes and Lave (1996) findings.

For the purposes of sensitivity analyses for the risk analyses, the various cutpoints findings from Samet et al. (1995) and Lave and Cifuentes were represented with a cutpoint of 30 $\mu g/m^3$ $PM_{2.5}$. Given the following considerations: (1) that the Lave and Cifuentes, Samet et al. (1995) findings for the elderly, and the central tendency of the findings for the elderly and all mortality for the two studies combined suggest PM_{10} cutpoints at or below the range of 40 - 45 $\mu g/m^3$, (2) the increased uncertainty in estimating PM_{10} cutpoint equivalents for the Samet et al. (1995) study, and (3) the emphasis of the alternative standards portion of the risk analysis on $PM_{2.5}$, it was judged that there was not a sufficient need to add a separate PM_{10} cutpoint to the sensitivity analyses above 40 $\mu g/m^3$, a concentration that also summarizes the upper end of the analyses of reported concentration-response relationships in Table E-1 (see Summary Section D).

2. Potential Effects of Measurement Error on Determining Effects Thresholds

Another issue to consider in estimating PM concentration-response relationships is the potential effects of measurement error. As discussed in Chapter V, the term measurement error in the broadest sense refers to errors or mis-estimation of several forms that can arise from the use of outdoor monitors to indicate exposure. Measurement error includes both errors resulting from errors in the direct measurement of ambient concentrations, and inaccuracies in the ability of central measurements to proxy for individual exposures, either to ambient pollutant concentrations or potentially the more broad array of particulate pollution from both indoor or outdoor sources to which an individual is personally exposed.

The potential of ambient exposure measurement error (i.e., either error in the direct measurement of ambient concentrations or in the ability of a central monitor to proxy for an individual's exposure to ambient pollutants) to give rise to an apparent more linear-seeming relationship that can disguise an underlying nonlinear relationship has been discussed to some extent in the air pollution and statistics literature (e.g.,Yoshimura, 1990). However, some evidence exists suggesting that the extent of such error may not serve to have large practical significance for current ambient particle concentration-response relationships. As discussed in Section V.E., Schwartz et al. (1996a) reported that statistical relationships between ambient $PM_{2.5}$ concentrations and mortality were observed even when the analysis was restricted to only

days with $PM_{2.5}$ concentrations of 25 $\mu g/m^3$ or below. A number of other studies (Pope, 1991; Schwartz et al.,1993a; Schwartz, 1994d; Schwartz, 1994e; Schwartz, 1994f) have excluded higher PM concentrations (e.g., PM_{10} concentrations above 150 $\mu g/m^3$). The similar or slightly larger relative risks observed in these studies when days with high concentrations are excluded from the analysis suggests that it is unlikely that measurement error is serving to disguise a nonlinear relationship that extends far into the range of observed concentrations. These studies also suggest that any "personal exposure measurement error" (errors in the ability of a central monitor to proxy for an individual's total exposure to indoor and outdoor particles, or some relevant subset of total exposure such as, exposures to all outdoor and indoor combustion sources), if present, may be affecting reported ambient $PM_{2.5}$ concentration-response relationships to only a limited extent. If ambient particle exposures are associated with mortality risk at 25 $\mu g/m^3$ $PM_{2.5}$ or below, it seems unlikely that a nonlinear concentration-response relationship with little or no risk for ambient particles may be being "disguised" by the unacknowledged role of other particle exposures, since relationships between ambient $PM_{2.5}$ and health effects, in general, would not be expected to be influenced by exposures to nonambient indoor sources, which are largely independent of ambient exposures (CD, p.1-10).

To allow for assessment of the potential effects on the risk analysis if measurement errors were found to be substantially affecting the shape of reported concentration-response relationships, cutpoint concentrations and slope adjustments of the type described in Chapter VI can be used to remodel ambient concentration-relationships to reflect hypothetical measurement error. For this purpose, although they were originally derived using the results from other lines of investigation, the cutpoint levels effects selected in Section D of this Appendix, which provide cutpoints across a substantial portion of the lower range of ambient concentrations, can be used to also model the possibility that measurement errors might be obscuring a nonlinear ambient concentration response function with little or no risk in this lower range of concentrations. For example, the possibility that exposure error might be obscuring ambient concentration-response nonlinearities at cutpoints of 10, 18 and 30 $\mu g/m^3$ $PM_{2.5}$ can be examined. Although the very issue raised by concerns about measurement error

is that these reported functions may "disguise" nonlinearity through the operation of errors in measurement of exposure, the results of the analyses in Sections A - C.1 above generated generate a set of potential cutpoints that include substantial PM concentrations, and thus for practical purposes can be used to examine of the potential impacts of substantial measurement error as well.

D. Summary

Staff believes that it is most appropriate to combine the potential concentration cutpoints summarized in Tables E-1 and E-3 into a few cutpoints for the purpose of doing sensitivity analyses. Combining information across studies, effects, and alternative approaches avoids giving undue weight to any particular study or approach. From these efforts, the following specific cutpoints judged of use for illustrating the sensitivity of risk analyses results have been identified:

- Short-term PM_{10} studies: 20, 30, 40 $\mu g/m^3$
- Short-term $PM_{2.5}$ studies: 10, 18, 30 $\mu g/m^3$
- Long-term PM_{10} studies: 24, 30, 32 $\mu g/m^3$
- Long-term $PM_{2.5}$ studies: 12.5, 15, 18 $\mu g/m^3$

These cutpoints were derived for the purposes of obtaining a reasonable range of possible cutpoints for the purposes of investigating the potential sensitivity of the risk analyses results to alternative concentration-response relationships reflecting alternative interpretations of reported relationships, potential changes in the concentration-response relationships from the consideration of copollutants, and/or potential effects of different types of measurement error. The material in Appendix E is not intended to be a critical or rigorous assessment of relative weight of evidence for any particular cutpoints from the available literature.

Appendix F

SENSITIVITY ANALYSES OF KEY UNCERTAINTIES IN THE RISK ASSESSMENT

As indicated in Chapter VI, a number of assumptions are involved in conducting a quantitative risk analysis of the effects of ambient PM, and any such effort involves a number of significant uncertainties. Sensitivity analyses are one approach that can provide insight into the potential effects of uncertainties and selection of alternative input assumptions on the risk analyses results. The results of a number of sensitivity analyses for the risk analyses are presented below. A more detailed discussion of the sensitivity analyses conducted for the PM health risk assessment can be found in the technical support document (Abt Associates, 1996b).

A. Sensitivity Analyses of Key Air Quality Uncertainties

1. Sensitivity Analysis of Alternative Background Concentrations

An important uncertainty concerning the air quality information used in the risk analysis involves estimates of background concentrations (see Table IV-3 for range of estimated background PM_{10} and $PM_{2.5}$ concentrations based on Chapter 4 of the CD). For the base case PM risk estimates, effects were quantified across the range of observations in the original study or to background concentrations, whichever was higher. For the base case risk analysis results reported in Chapter VI, the midpoint of the range of estimated annual background concentrations has been used. Tables F-1A and F-1B show the sensitivity of the risk estimates to using either the low end of the annual background concentration range identified in the CD (5 $\mu g/m^3$ PM_{10} and 2 $\mu g/m^3$ $PM_{2.5}$ in the eastern U.S.) or the high end of the annual background concentration range identified in the CD (11 $\mu g/m^3$ PM_{10} and 5 $\mu g/m^3$ $PM_{2.5}$ in the eastern U.S.) as the estimate for background concentrations rather than the midpoint of the range.

One important point from Table F-1A and F-1B is that the estimates of mortality and bronchitis risks associated with long-term exposure to PM do not change as a result of alternative background concentrations. Because these long-term studies relate health effects to

Table F-1a. Sensitivity Analysis: The Effect of Alternative Background Levels on Predicted Health Effects Associated With "As-Is" PM-10 Philadelphia County, September 1992 - August 1993

Health Effects*			Percent of Total Incidence Associated with PM-10 Above Background**		
			BASE CASE Background = 8 µg/m3	Background = 5 µg/m3	Background = 11 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure		**1.1%** **(0.8 - 1.4)**	1.3% (1.0 - 1.7)	0.9% (0.6 - 1.1)
Hospital Admissions Respiratory	(B) Total Respiratory (>64 years old)		**2.4%** **(1.5 - 3.3)**	2.87% (1.8 - 4.0)	1.9% (1.2 - 2.7)
		(C) COPD (>64 years old)	**3.7%** **(2.5 - 4.7)**	4.4% (3.1 - 5.7)	3.0% (2 1 - 3.8)
		(D) Pneumonia (>64 years old)	**1.9%** **(1.3 - 2.6)**	2.3% (1.6 - 3.1)	1.6% (1.1 - 2.1)
Hospital Admissions Cardiac	(E) Ischemic Heart Disease (>64 years old)		**0.8%** **(0.3 - 1.3)**	1.0% (0.4 - 1.5)	0.6% (0.2 - 1.0)
	(F) Congestive Heart Failure (>64 years old)		**1.4%** **(0.7 - 2.1)**	1.7% (0.8 - 2.5)	1.1% (0.5 - 1.7)
Lower Respiratory Symptoms in Children	(G) Lower Respiratory Symptoms (# of cases) (8 - 12 year olds)		**17.5%** **(15.3 - 19.6)**	20 8% (18.2 - 23.3)	14.2% (12.4 - 15.9)
	(H) Lower Respiratory Symptoms (# of days) (9-11 year old asthmatics)		**6.8%** **(2.4 - 10.9)**	8.2% (2.9 - 13.0)	5.5% (2.0 - 8.8)

* Health effects associated with short-term exposure to PM

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level Background PM-10 is assumed to be 8 ug/m3 .

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter VI for details.

Sources of Concentration-Response (C-R) Functions:

(A) PM-10 C-R function based on pooled results from studies in 10 locations.

(B) PM-10 C-R based on pooled results from 4 functions

(C) PM-10 C-R based on pooled results from 4 functions

(D) PM-10 C-R based on pooled results from 4 functions

(E) Schwartz & Morris, 1995

(F) Schwartz & Morris, 1995

(G) Schwartz, et al., 1994

(H) Pope et al., 1991

Table F-1b. Sensitivity Analysis: The Effect of Alternative Background Levels on Predicted Health Effects Associated With "As-Is" PM-2.5 Philadelphia County, September 1992 - August 1993

Health Effects*		Percent of Total Incidence Associated with PM-2.5 Above Background**		
		BASE CASE Background = 3.5 µg/m3	Background = 2.0 µg/m3	Background = 5.0 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure	**1.8% (1.1 - 2.5)**	2.0% (1 2 - 2.8)	1.6% (1.0 - 2.2)
Hospital Admissions Respiratory	(B) Total Respiratory (all ages)	**2.0% (0.5 - 3.5)**	2 3% (0.6 - 3.9)	1.8% (0.5 - 3.1)
Hospital Admissions Cardiac (>64 years old)	(C) Ischemic Heart Disease***	**0.7% (0.3 - 1.2)**	0.8% (0.3 - 1.3)	0.7% (0.3 - 1.1)
	(D) Congestive Heart Failure***	**1.3% (0.6 - 2.0)**	1.5% (0.7 - 2.2)	1.2% (0.6 - 1.7)
Lower Respiratory Symptoms in Children	(E) Lower Respiratory Symptoms (# of cases) (8-12 years old)	**20.1% (10.3 - 28.3)**	22.2% (11.5 - 31.3)	17.8% (9.2 - 25.2)

* Health effects associated with short-term exposure to PM

** Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level. Background PM-2.5 is assumed to be 3.5 ug/m3.

*** PM-2 5 results based on using PM-2 5 mass as PM-10 mass in the PM-10 functions.

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter VI for details.

Sources of Concentration-Response (C-R) Functions:

(A) PM-2.5 C-R function based on pooled results from 6 locations.

(B) Thurston, et al., 1994

(C) Schwartz & Morris, 1995

(D) Schwartz & Morris, 1995

(E) Schwartz, et al., 1994

annual mean concentrations, and the lowest observed annual mean concentration (the limit used for quantification of risk) is well in excess of current estimates of background (e.g., the range of concentrations observed for the cities in the ACS study (Pope et al., 1995) was 9.0 - 33.4 $\mu g/m^3$ $PM_{2.5}$), the estimates of health risks associated with these endpoints do not change in relation to estimates of background concentrations in the ranges used here (e.g., 2 -5 $\mu g/m^3$ $PM_{2.5}$).

2. Sensitivity of Health Risks Estimates to Alternative Rollback Methods for Simulating Attainment of Alternative Standards

In addition to uncertainties concerning "as is" air quality, there is inherent uncertainty concerning any effort to estimate air quality distributions that would occur upon attaining standards at some future date. In the risk analysis, such uncertainties are introduced both in efforts to model health risks upon attainment of the current standard (Chapter VI, Table VI-8) and upon attainment of alternative $PM_{2.5}$ standards (Chapter VI, Tables VI-12a -13b). The base case analysis assumes that proportional reductions would be observed in air quality concentrations as an area attained either a controlling annual mean or 24-hr standard. A sensitivity analysis was conducted to examine the sensitivity of risk reduction estimates associated with alternative $PM_{2.5}$ standards to an alternative assumption concerning the pattern of air quality rollbacks and the resulting air quality distribution that might be observed in reaching attainment of $PM_{2.5}$ standards (Table F-2). Because $PM_{2.5}$ standards do not currently exist, information on past air quality rollbacks in response to $PM_{2.5}$ standards is not available. However, monitoring information for $PM_{2.5}$ can be examined, although it is uncertain how much of the variation observed between years in the air quality distribution at a location reflects actual control strategies versus more general year-to-year variability. In a preliminary examination of changes in the distribution of $PM_{2.5}$ concentrations from sites with multiple years of data (from AIRS and CARB data sets), Abt Associates found that proportional rollback reasonably approximated the central tendency of variations in $PM_{2.5}$ air quality distributions, however, considerable variation could be observed in this relationship across time and location (see Abt Associates, 1996b for more information).

Table F-2. Sensitivity Analysis: Effect of Alternative Rollback Methods on Predicted Health Effects of PM-2.5

Philadelphia County, September 1992 - August 1993

Health Effects	Total PM-related Incidence	Alternative Standard	Entire AQ distribution reduced equally			Upper 10% of AQ distribution reduced more		
			Rollback Required	Resulting Air Quality (Annual Mean/ 2nd Daily Max)	Change in Total Incidence*	Base Rollback Required**	Resulting Air Quality (Annual Mean/ 2nd Daily Max)	Change in Total Incidence*
(A) Mortality associated with short-term exposure	370 (220 - 510)	15 ug/m3 annual	10.5%	15.0 / 64.4	40 (20 - 60)	14.5%	15.0 / 62.6	30 (20 - 50)
	Percent reduction in PM-related incidence:				10.6%			9.2%
	370 (220 - 510)	50 ug/m3 daily	29.4%	12.3 / 50.0	110 (70 - 170)	18.4%	13.3 / 50.0	70 (40 - 110)
	Percent reduction in PM-related incidence:				29.7%			18.6%
(B) Mortality associated with long-term exposure	900 (560 - 1230)	15 ug/m3 annual	10.5%	15.0 / 64.4	170 (130 - 280)	9.0%	15.0 / 62.6	170 (110 - 240)
	Percent reduction in PM-related incidence:				19.4%			19.4%
	900 (560 - 1230)	50 ug/m3 daily	29.4%	12.3 / 50.0	490 (350 - 770)	18.4%	13.3 / 50.0	350 (220 - 480)
	Percent reduction in PM-related incidence:				54.1%			39.3%

* Health effects incidence was quantified across the range of PM concentrations observed in each study, but now below background PM-2.5 levels, assumed to be 3.5 ug/m3.

(A) C-R function based on studies in 6 locations.
(B) Pope et al., 1995

** The base rollback is the rollback on the lower 90% of the air quality distribution. The upper 10% is reduced by more.

The numbers in parentheses are NOT standard confidence intervals. They are 90% credible intervals based on Monte Carlo analysis that takes into account both statistical uncertainty and possible geographic variability. See text for details.

An attempt to bound the potential effects of alternative PM air quality reduction patterns has been examined in a sensitivity analysis of PM-associated risks by choosing alternative assumptions for modeling $PM_{2.5}$ rollbacks. Table F-2 shows the sensitivity of risks reduction estimates associated with alternative $PM_{2.5}$ standards to the rollback assumption in which the upper 10% of the $PM_{2.5}$ 24-hr air quality concentrations are reduced by a larger amount (a ratio of 1.6) than in the remaining 90% of the distribution of PM air quality concentrations. This alternative rollback case is intended to model a control strategy that preferentially targets peak $PM_{2.5}$ levels. The proportion of preferential reduction in peak concentrations (a 1.6 ratio in reduction for the upper 10% of concentrations) is based on empirical observation of the 99th percentile of observed year-to-year variation in $PM_{2.5}$ air quality among site-years for all available $PM_{2.5}$ monitoring sites with multiyear data from the AIRS or CARB $PM_{2.5}$ datasets.

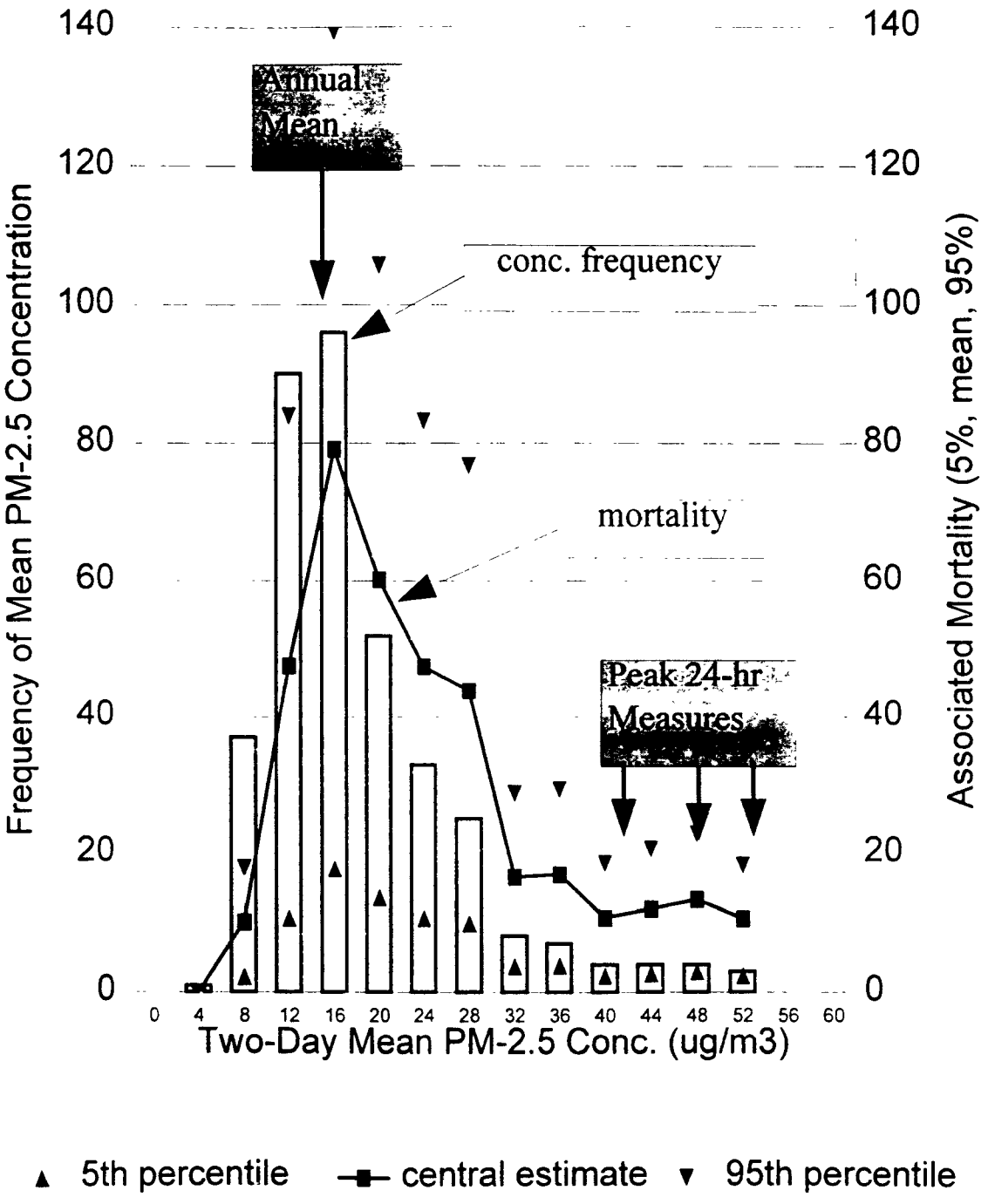
Table F-2 shows for both a proportional rollback and the preferential peak reduction rollback the amount of reduction in $PM_{2.5}$ concentrations necessary to reach alternative standards (for simplicity, the annual and daily standards are considered alone) and the air quality distribution (summarized as the annual mean and 2nd daily max concentration) that is projected to occur upon attainment. In this example, the annual standard provides less of a change in total incidence of health effects, but this is simply a consequence of the annual standard chosen (15 $\mu g/m^3$) being less controlling than the daily standard chosen (50 $\mu g/m^3$) for Philadelphia County (Chapter VI, Table 11b).

More important to consider are the PM-associated risk reductions and resulting air quality observed when the operation of the same standard (annual or daily) is modeled under the two rollback cases rather than any comparison of total incidence reduction between the two standards. The important observation is that estimated changes in incidence of health effects provided by attainment of annual standards are less sensitive to deviation from the base case assumption on rollback than estimated reductions in health effects incidence risk resulting from attainment of a daily standard. For instance, the results in Table F-2 indicate that for a controlling annual standard, past patterns of air quality change would suggest the reduction in health effects from short-term exposures, as represented by mortality from short-term

exposures, could potentially vary more than 35% with a controlling 24-hr standard (mean change in total incidence of 70 versus 110), compared to approximately 25% with a controlling annual standard. For mortality from long-term exposures, this contrast is greater. For example, under a controlling short-term standard estimated risk reduction could potentially vary 30%, while under an annual standard there would be no change in estimated risk reduction. This is a result of the fact that mortality from long-term exposures are related to central estimate air quality measures such as annual mean concentration in the reported concentration-response relationships, thus the distribution of 24-hr concentrations associated with this annual mean concentration does not influence the estimated health risk reduction as long as the same annual mean (in this case, 15 $\mu g/m^3$) is achieved under both rollback conditions.

Figure F-1 illustrates some of the characteristics of the integration of current air quality distributions and reported concentration-response relationships as used to predict the total risk from ambient particle exposures across a year. Figure F-1 shows the relative contribution of different portions of the ambient $PM_{2.5}$ concentration distribution for Philadelphia County to the "as is" mortality health risk from short-term exposures. The Figure shows in bar graph form the proportion of total observed PM-2.5 concentrations across the year (in groups of 4 $\mu g/m^3$ per bar), with the number of days out of the whole year (361 observations) that concentrations fell within each concentration range shown on the left-hand Y axis. On top of this frequency distribution has been overlaid the proportion of "as is" mortality risk under base case assumptions associated with each 4 $\mu g/m^3$ concentration range (Since "as is" mortality risk from short-term exposures was calculated using a two-day mean averaging time, the averaging time used at the largest number of mortality study locations, the proportion of "as is" mortality risk is calculated for each two-day mean interval of 4 $\mu g/m^3$). This Figure shows that for base case assumptions, concentrations in the range of 16-20 $\mu g/m^3$ contribute the largest amount to the estimated mortality risk on an annualized basis for Philadelphia County. Even though concentrations in the range of 44 $\mu g/m^3$ $PM_{2.5}$ and above clearly contribute more mortality per day for these concentrations, the much larger number of days within the 16-20 $\mu g/m^3$ range results in this interval being associated with the largest total risk. Standards with

Figure F-1. Distribution of $PM_{2.5}$ Concentrations and of Estimated Mortality Risks from Short-Term Exposures in Philadelphia County



a 24-hr averaging time are traditionally based on peak air quality statistics, concentrations for which the risk on an individual day is highest, but, as a result of the ambient air quality distribution and the $PM_{2.5}$ concentration-response functions that have been observed, appear to contribute a relatively small amount of the total health risk compared to the distribution as a whole. The annual mean statistic contains information about the aggregate total of all the air quality concentrations, a quantity similar to the quantity of all air quality concentrations minus estimated background that contributes to estimates of annualized mortality risk in the base case risk analysis.

The difference between the air quality distribution as a whole and that estimated to contribute to aggregate annualized health risk will be more pronounced if assumptions about a substantial cutpoint concentration are made. However, even in these cases, the aggregate annualized risk will be a function of the concentrations across a wide portion of the upper end of the $PM_{2.5}$ air quality distribution. Since reducing high concentration days can provide a greater microgram reduction in $PM_{2.5}$ annual average mass for a lesser percentage reduction in air quality, an annual standard will still favor reducing high concentration values. In contrast to the 24-hr standard, however, an annual standard is less likely to allow areas whose air quality concentrations are substantially above those necessary for attainment to reduce concentrations in a fashion that might not result in meaningful risk reduction (e.g., by reducing just a few high peak values). In so doing, an annual controlling standard might be expected to lead to less variation in the risk reduced in different geographic areas having similar initial air quality that reduce PM concentrations to attain a set of $PM_{2.5}$ alternative standards.

Table F-2 conveys this point in a related fashion. Table F-2 shows that under the preferential peak reduction rollback considered, the lower 90% of air quality concentrations are reduced only 18% versus the 30% reduction observed if the entire distribution is reduced evenly. Because the lower 90 percent of the air quality values contribute so substantially to the aggregate annualized risk (Figure F-1), a lesser reduction across this wide range of concentration values leads to less total $PM_{2.5}$ reduction [as reflected by the higher annual mean upon attainment of a daily standard of 50 $\mu g/m^3$ in which lower concentrations have been less

substantially reduced (13.6 $\mu g/m^3$) than when concentrations have been reduced evenly (12.6 $\mu g/m^3$)], and thus less total annual health risk being reduced.

Absent information that allows the possibility to be excluded that PM concentrations through a wide portion of the air quality distribution may contribute to risk, an annual controlling standard is likely to be less sensitive to alternative rollback assumptions. This is in large part because the standard employs an air quality measure (the annual mean) that inherently captures more information reflective of the concentrations across the bulk of the air quality distribution. In general, annual standards would be expected to decrease uncertainty in risk reductions observed for areas that might undergo different air quality rollbacks to reach attainment of $PM_{2.5}$ alternative standards relative to comparably stringent controlling 24-hr standards.

For the special case of modeling the "attainment of current PM_{10} standards" case for Los Angeles County, since the current daily PM_{10} standard is controlling in Los Angeles, it is relevant to consider the potential effects of variations from a proportional rollback for PM_{10} on the risk estimates for alternative $PM_{2.5}$ standards. Variations in the PM_{10} rollback that would result in attainment of the current standards from the proportional rollback assumed could either increase or decrease the amount of risk associated with PM remaining to be affected by alternative $PM_{2.5}$ standards. In addition, the risk estimate for the "attainment of the current standards" case in Los Angeles has an important additional source of uncertainty relating to patterns of reductions. If control strategies to meet the current PM_{10} standards preferentially reduce the coarse fraction of PM_{10} in relation to the fine fraction of PM_{10}, risks associated with $PM_{2.5}$ as an indicator of PM under the "attain current standards" case could be higher and, thus, proportions of estimated risk reduced under the alternative $PM_{2.5}$ standards also would be greater. Alternatively, if control strategies to meet the current standards preferentially reduce the fine fraction, then risks associated with $PM_{2.5}$ as an indicator of PM would be less under the "attain current standards" and the proportion of estimated risks reduced under the alternative $PM_{2.5}$ standards would be less.

B. Sensitivity Analyses of Key Concentration-Response Uncertainties

The area of the risk analysis with the largest number of uncertainties amenable to sensitivity analyses involves the application of PM concentration-response relationships in the risk analysis. The sensitivity of risk estimates for "as is" air quality in Philadelphia has been analyzed to determine the potential impact of alternative analytic approaches to addressing uncertainty in the concentration-response relationships. The following sensitivity analyses about concentration-response relationships are summarized in this Section:

- The effect of alternative assumptions concerning the shape of the concentration-response relationships, especially concerning the effect of cutpoint concentrations below which variations in PM concentration are not associated with increases in risk, is analyzed. Alternative assumptions about the slope of the concentration-response relationship above any presumed cutpoints also is addressed.

- The effect of pooling studies to combine information from a number of studies to apply to the two risk analysis locations is examined. The sensitivity of short-term mortality risk estimates is analyzed, especially with respect to the effects of combining studies that are heterogenous in averaging time.

- The effect of using coefficients for PM obtained simultaneously with other copollutants in the regression model is addressed.

- The effect of alternative assumptions concerning the potential role of air quality previous to that monitored in studies of the effects on mortality associated with long-term exposure is examined.

All of these sensitivity analyses are conducted using "as-is" air quality in Philadelphia County. Further sensitivity analyses are provided in the technical support document (Abt Associates, 1996b).

1. Sensitivity Analyses of Alternative Cutpoint Concentrations

Tables F-3A-E present the results from sensitivity analyses of different alternative cutpoint concentrations for short-term and long-term exposures to PM. The concentrations chosen as cutpoints for these sensitivity analyses were selected from the analysis of potential cutpoints of interest described in Appendix E and summarized in Chapter VI. For the base case analysis, no cutpoint has been assumed. In the sensitivity analyses, various cutpoint concentrations have been examined, and no health risks associated with PM are estimated for

Table F-3a. Sensitivity Analysis: The Effect of Alternative Cutpoint Models on Predicted Health Effects Associated With "As-Is" PM-10 Slope Adjustment Method 1* Philadelphia County, September 1992 - August 1993

Health Effects**		Percent of Total Incidence Associated with PM-10 Above Cutpoint			
		BASE CASE Background = 8 µg/m3	Cutpoint = 20 µg/m3	Cutpoint = 30 µg/m3	Cutpoint = 40 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure	**1.1%** **(0.8 - 1.4)**	0.4% (0.3 - 0 6)	0.2% (0.1 - 0.2)	0.1% (0.0 - 0.1)
Hospital Admissions Respiratory	(B) Total Respiratory (>64 years old)	**2.4%** **(1.5 - 3.3)**	1.3% (0 8 - 1 7)	0.7% (0.4 - 0 9)	0.4% (0.2 - 0.5)
Hospital Admissions Cardiac	(C) Ischemic Heart Disease (>64 years old)	**0.8%** **(0.3 - 1.3)**	0 3% (0.1 - 0.4)	0.1% (0.1 - 0.2	0.1% (0.0 - 0.1)
	(D) Congestive Heart Failure (>64 years old)	**1.4%** **(0.7 - 2.1)**	0 5% (0.2 - 0.2)	0.2% (0.1 - 0.1)	0.1% (0.1 - 0.2)
Lower Respiratory Symptoms in Children	(E) Lower Respiratory Symptoms (# of cases) (8-12 year olds)	**17.5%** **(15.3 - 19.6)**	9.3% (5.4 - 12.7)	6 3% (3.9 - 8.1)	4.7% (3.4 - 5 5)

* Two methods examine the potential impact of a concentration-response function having a steeper slope (i.e., larger coefficient) above specified cutpoints. In both methods the slope below the cutpoint is set = 0, while the slope above the cutpoint is set to be greater than the slope in the original study. In Adjustment Method 1, the cutpoint C-R relationship is modeled to intersect with the original relationship, exceeding the RRs predicted for the original study at higher concentrations. The relationship was modeled to match the reduction in the range of PM concentrations upon application of the cutpoint with an identical percentage increase in the risk observed at the highest concentration. Method 2 estimates a smaller increase in the slope See text for further information.

**Health effects associated with short-term exposure to PM

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter VI for details.

Sources of Concentration-Response (C-R) functions:
(A) C-R function based on pooled results from 10 locations
(B) C-R function based on pooled results from 4 locations.
(C) Schwartz & Morris, 1995
(D) Schwartz & Morris, 1995
(E) Schwartz, et al., 1994

Table F-3b. Sensitivity Analysis: The Effect of Alternative Cutpoint Models on Predicted Health Effects Associated With "As-Is" PM-10 Slope Adjustment Method 2* Philadelphia County, September 1992 - August 1993

Health Effects**		Percent of Total Incidence Associated with PM-10 Above Cutpoint			
		BASE CASE Background = 8 µg/m3	Cutpoint = 20 µg/m3	Cutpoint = 30 µg/m3	Cutpoint = 40 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure	**1.1%** **(0.8 - 1.4)**	0.4% (0 3 - 0.5)	0.1% (0.1 - 0 2)	0.1% (0.0 - 0.1)
Hospital Admissions Respiratory	(B) Total Respiratory (>64 years old)	**2.4%** **(1.5 - 3.3)**	1.0% (0 6 - 1.3)	0.4% (0.3 - 0.6)	0.2% (0.1 - 0.3)
Hospital Admissions Cardiac	(C) Ischemic Heart Disease (>64 years old)	**0.8%** **(0.3 - 1.3)**	0 3% (0.1 - 0.4)	0 1% (0.0 - 0.2)	0.0% (0.0 - 0.1)
	(D) Congestive Heart Failure (>64 years old)	**1.4%** **(0.7 - 2.1)**	0.5% (0 2 - 0.7)	0.2% (0.1 - 0.3)	0.1% (0.0 - 0.1)
Lower Respiratory Symptoms in Children	(E) Lower Respiratory Symptoms (# of cases) (8-12 year olds)	**17.5%** **(15.3 - 19.6)**	7 9% (4.5 - 11.0)	4.1% (2.4 - 5.6)	2 5% (1.5 - 3.2)

* Two methods examine the potential impact of a concentration-response function having a steeper slope (i e., larger coefficient) above specified cutpoints. In both methods the slope below the cutpoint is set = 0, while the slope above the cutpoint is set to be greater than the slope in the original study In Adjustment Method 2, the slope is increased so that the new C-R function estimates the same health risk at the highest observed PM value as the original function Method 1 estimates a larger increase in the slope

**Health effects associated with short-term exposure to PM.

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter VI for details.

Sources of Concentration-Response (C-R) functions:
(A) C-R function based on pooled results from 10 locations.
(B) C-R function based on pooled results from 4 locations.
(C) Schwartz & Morris, 1995
(D) Schwartz & Morris, 1995
(E) Schwartz, et al., 1994

Table F-3c. Sensitivity Analysis: The Effect of Alternative Cutpoint Models on Predicted Health Effects Associated With "As Is" PM-2.5 Slope Adjustment Method 1* Philadelphia County, September 1992 - August 1993

Health Effects**		Percent of Total Incidence Associated with PM-2.5 Above Cutpoint			
		BASE CASE: Background = 3.5 µg/m3	Cutpoint = 10 µg/m3	Cutpoint = 18 µg/m3	Cutpoint = 30 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure	**1.8% (1.1 - 2.5)**	1.1% (0.6 - 1.5)	0.5% (0.3 - 0.6)	0.1% (0.1 - 0 2)
Hospital Admissions Respiratory	(B) Total Respiratory (all ages)	**2.0% (0.5 - 3.5)**	1.4% (0.4 - 2 4)	0.8% (0.2 - 1.4)	0.4% (0.1 - 0.7)
Hospital Admissions Cardiac	(C) Ischemic Heart Disease (>64 years old)	**0.7% (0.3 - 1.2)**	0.4% (0.1 - 0 6)	0.2% (0.1 - 0.3)	0.1% (0.0 - 0.1)
	(D) Congestive Heart Failure (>64 years old)	**1.3% (0.6 - 2.0)**	0.6% (0.3 - 1 0)	0.4% (0.2 - 0 5)	0.1% (0.1 - 0.2)
Lower Respiratory Symptoms in Children	(E) Lower Respiratory Symptoms (8 - 12 years old)	**20.1% (10.3 - 28.3)**	13.1% (7.1 - 18.5)	9.7% (5.6 - 13.0)	6.5% (5.2 - 7.1)

* Two methods examine the potential impact of a concentration-response function having a steeper slope (i e., larger coefficient) above specified cutpoints. In both methods the slope below the cutpoint is set = 0, while the slope above the cutpoint is set to be greater than the slope in the original study. In Adjustment Method 1, the cutpoint C-R relationship is modeled to intersect with the original relationship, exceeding the RRs predicted for the original study at higher concentrations. The relationship was modeled to match the redu the range of PM concentrations upon application of the cutpoint with an identical percentage increase in the risk observed at the highest concentration. Method 2 estimates a smaller increase in the slope. See text for further information.

** Health effects associated with short-term exposure to PM.

Sources of Concentration-Response (C-R) functions:
(A) C-R function based on pooled results from six locations.
(B) Thurston, et al., 1994
(C) Schwartz & Morris, 1995
(D) Schwartz & Morris, 1995
(E) Schwartz et al., 1994

The numbers in parentheses for pooled functions are NOT standard confidence intervals.
All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability.
See text in Chapter VI for details.

Table F-3d. Sensitivity Analysis: The Effect of Alternative Cutpoint Models on Predicted Health Effects Associated With "As Is" PM-2.5 Slope Adjustment Method 2* Philadelphia County, September 1992 - August 1993

Health Effects**		Percent of Total Incidence Associated with PM-2.5 Above Cutpoint			
		BASE CASE: Background = 3.5 µg/m3	Cutpoint = 10 µg/m3	Cutpoint = 18 µg/m3	Cutpoint = 30 µg/m3
Mortality (all ages)	(A) Associated with short-term exposure	**1.8%** **(1.1 - 2.5)**	1.0% (0 6 - 1 4)	0.4% (0 2 - 0.6)	0.1% (0.1 - 0 2)
Hospital Admissions Respiratory	(B) Total Respiratory (all ages)	**2.0%** **(0.5 - 3.5)**	1 2% (0 3 - 2.1)	0.6% (0.2 - 1.1)	0 2% (0.1 - 0 4)
Hospital Admissions Cardiac	(C) Ischemic Heart Disease (>64 years old)	**0.7%** **(0.3 - 1.2)**	0.4% (0.2 - 0.6)	0.2% (0.1 - 0.3)	0.1% (0.0 - 0 1)
	(D) Congestive Heart Failure (>64 years old)	**1.3%** **(0.6 - 2.0)**	0.7% (0 3 - 1.0)	0.3% (0.1 - 0.5)	0.1% (0.0 - 0.1)
Lower Respiratory Symptoms	(E) Lower Respiratory Symptoms (8 - 12 years old)	**20.1%** **(10.3 - 28.3)**	12.1% (6 5 - 17 2)	6.9% (3.8 - 9 6)	3.6% (2.3 - 4 5)

* Two methods examine the potential impact of a concentration-response function having a steeper slope (i.e., larger coefficient) above specified cutpoints. In both methods the slope below the cutpoint is set = 0, while the slope above the cutpoint is set to be greater than the slope in the original study In Adjustment Method 2, the slope is increased so that the new C-R function estimates the same health risk at the highest observed PM value as the original function Method 1 estimates a larger increase in the slope.

**Health effects associated with short-term exposure to PM

The numbers in parentheses for pooled functions are NOT standard confidence intervals.
All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability.
See text in Chapter VI for details.

Sources of Concentration-Response (C-R) functions:
(A) C-R function based on pooled results from six locations
(B) Thurston, et al., 1994
(C) Schwartz & Morris, 1995
(D) Schwartz & Morris, 1995
(E) Schwartz et al., 1994

Table F-3e. Sensitivity Analysis: The Effect of Differing Cutpoints on Estimated Mortality Associated with Long-term Exposure to PM-2.5 Philadelphia County, September 1992 - August 1993

	BASE CASE **Lowest Observed = 9 ug/m3**	Cutpoint = 12.5 ug/m3	Cutpoint = 15 ug/m3	Cutpoint = 18 ug/m3
(A) Mortality associated with long-term exposure	**4.6%** **(2.8 - 6.2)**	2.4% (1.5 - 3.3)	0.8% (0.5 - 1.1)	0.0% (0.0 - 0.0)

(A) Pope et al., 1995

Health effects incidence was calculated down to the lowest level observed in the study (9 ug/m3).
No adjustments to the slope were performed.

The numbers in parentheses for pooled functions are NOT standard confidence intervals.
All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability.
See text in Chapter VI for details.

any days whose 24-hr concentrations are below the specified cutpoint concentration. In addition, the slope of the relationship above the cutpoint has been remodeled using one of two approaches. For both approaches, the relationship is assumed to begin at zero increased risk at the cutpoint concentration, and to extend upward with an increased slope compared to the original reported relationship (see Fig. VI-6). In Approach 1 it is assumed that the new slope would increase to an extent where the increased health risk predicted at the highest concentration is increased proportional to the proportion of the range of original concentrations that fall below the cutpoint. While this adjustment produces a slope resembling those generally posited to result in a model incorporating a cutpoint (e.g., Fig VI-6), there is no clear guidance on how to most appropriately model changes in slope for purposes such as the PM risk analysis (where, for instance, primary datasets are not readily available).

In light of this uncertainty, a second approach, involving a more minimal adjustment to slope (labeled "Approach 2" on Figure VI-6) also has been carried out as a potential lower bound for an adjusted slope. In Approach 2, the concentration-response relationship has been remodeled to begin at zero at the cutpoint and intersect with the same health risk estimated at the highest concentrations observed in the original relationship. As cutpoints are chosen that exclude successively larger number of observations, it is expected that the milder degree of increased slope represented by Approach 2 would be less likely to be observed.

Figure F-2 suggests that relatively mild increases in slope may be observed for some TSP concentration-response relationships compared to a linear model meta analysis from the CD. However, other TSP concentration-response relationships which examined cutpoints well within the range of data observed a pattern of increased slope more like that modelled in Approach 1 (Philadelphia 1983-88, which included SO_2 and O_3 in the analysis, compared with a meta analysis of PM coefficients from models including copollutants).

As might be expected, Tables F-3A - D indicate that the two slope adjustment approaches agree mostly closely at the lowest cutpoint concentration. In addition, these tables suggest that the method of adjusting the slope of the remaining relationship is less important to the estimates of health risk than the choice of cutpoint concentration itself. The higher the cutpoint, the greater the proportion of observations for each city that is associated with no increase in risk. Depending on judgments concerning the weight to be given the estimates at

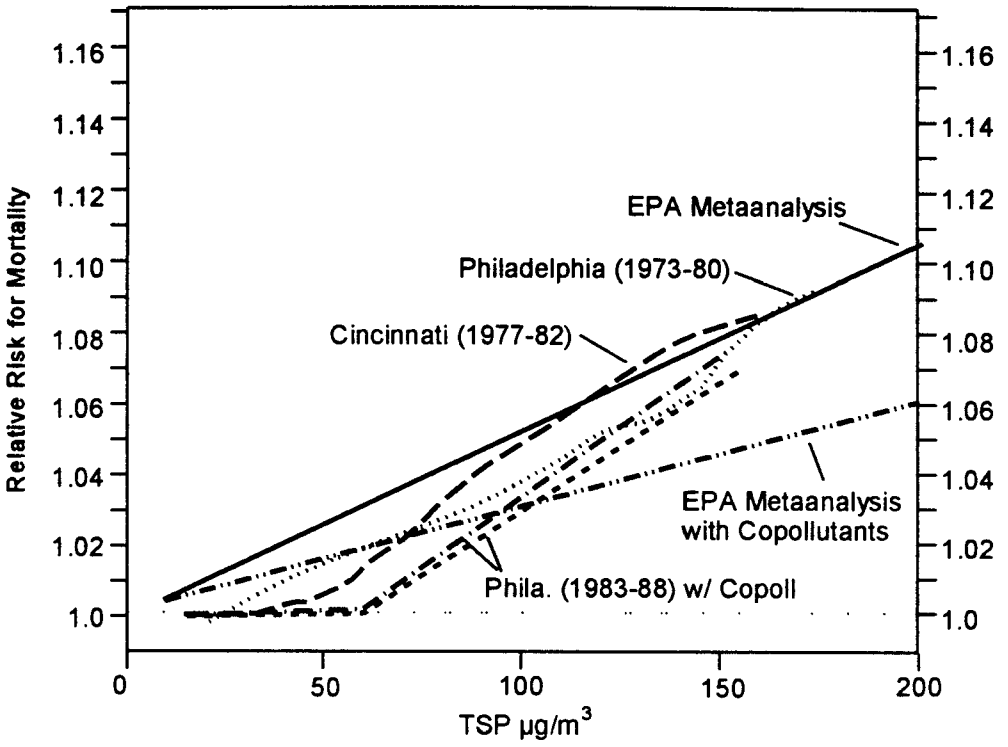


Figure F-2. Comparison of Smoothed Nonlinear and Linear Mathematical Models for Relative Risk of Total Mortality Associated with Short-Term TSP Exposure (CD, Figure 13-6). Curves show smoothed nonparametric models for Philadelphia (based on Schwartz 1994b) and for Cincinnati (based on Schwartz, 1994a), and piecewise linear models for Philadelphia (based on Cifuentes and Lave, 1996). Solid curve shows linear model from EPA metaanalysis using studies with no copollutants, dash-dot curve shows linear model from EPA metaanalysis using studies with SO_2 as a copollutant (described in CD Chapter 12).

higher cutpoint concentrations, assumptions concerning cutpoint concentrations can make a substantial difference in the estimates of risks associated with PM.

For the concentration-response relationship of mortality from long-term exposures (Table F-3E), the upper cutpoint eliminates estimated risk for Philadelphia County because Philadelphia County's annual mean concentrations are below 18 $\mu g/m^3$. For health risks both from short-term and long-term exposures, the sensitivity of estimates of risks would be expected to vary with location, especially for locations with substantially different overall PM air quality (e.g., Los Angeles County).

2. Effect on Pooled Concentration-Response Analyses Using Studies with Different Averaging Times

In their review of the PM mortality literature, the CD pointed out that heterogeneity in averaging time is an important factor to consider in assessing results (CD, p.12-72). In the PM risk analysis estimates from a number of studies have been pooled for several endpoints. For the mortality pooled analysis, studies that used averaging times ranging from 1 to 5 day mean PM concentrations have been included. Table F-4 disaggregates the pooled analysis to examine the effect of restricting the estimates of mortality risk to those studies using only the same averaging time (with the exception of the three-day and five-day mean studies, which were combined). Results vary considerably over averaging times. In the base case analysis, two-day mean air quality concentrations were used to estimate mortality, since the largest number of functions used that averaging time. Table F-4 indicates that using two-day mean concentrations to represent Philadelphia County PM_{10} concentrations results in an increase in the risk estimates predicted by the single study that reported results related to a one-day mean concentration (Kinney et al., 1995), and a slight increase in the risk predicted for the set of two studies using three- to five-day mean concentrations (Schwartz, 1993 and Pope et al., 1992). However, the Table also indicates that applying an alternative averaging time, such as one-day or five-day mean concentrations, results in no apparent difference in estimated risk from the base case two-day mean assumption.

Table F-4. Sensitivity Analysis: Effect of Combining Different Averaging Times In Pooled Short-Term Exposure Mortality Functions on Predicted Health Effects Associated With "As-Is" PM-10 Philadelphia County, September 1992 - August 1993

	Percent of Total Incidence Associated with PM-10 Above Background*			
	BASE CASE Studies Using All Averaging Times (10 studies)**	Studies using 1-day average PM (1 study)	Studies using 2-day average PM (7 studies)	Studies using 3-5 day average PM (2 studies)
Matching study and data averaging times	**2-day average PM**	1-day average PM	2-day average PM	5-day average PM
	1.1% (0.8 - 1.4)	0.4% (0.0 - 0.8)	1.0% (0.5 - 1.5)	1.8% (1.3 - 2.4)
Using 2-day average PM data	2-day average PM	2-day average PM	2-day average PM	2-day average PM
	same	0.4% (0.0 - 0.8)	same	1.9% (1.3 - 2.4)
Using 1-day average PM data	1-day average PM			
	1.1% (0.8 - 1.4)			
Using 5-day average PM data	5-day average PM			
	1.1% (0.8 - 1.4)			

*Health effects incidence was quantified across the range of PM concentrations observed in each study, when possible, but not below background level Background PM-10 is assumed to be 8 ug/m3

** The base case is a random-effects pooled function used with 2-day average PM data All other pooled functions are also random effects, except the pooled function derived from studies using 3-5 day average PM data, for which a fixed effects model was used, since it is not possible to calculate a random effects model for those two functions

The numbers in parentheses for pooled functions are NOT standard confidence inter All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chapter VI for details.

The studies that contribute to the pooled function are:

1-day: Kinney et al., 1995 (Los Angeles)
2-day: Ito and Thurston, 1996 (Chicago)
Schwartz et al. 1996 (Boston, MA; Knoxville, TN; St. Louis, MO; Steubenville, OH; Portage, WI; Topeka, KS)
3-day: Schwartz 1993 (Birmingham, AL)
5-day: Pope et al., 1992 (Utah Valley)

3. Effect of Using Concentration-Response Relationships Simultaneously Considering Copollutants

PM is part of a mix of combustion source pollutants originating from a variety of stationary and mobile sources and, thus generally occurs along with other pollutants generated by combustion sources (e.g., sulfur oxides, nitrogen oxides, volatile organic compounds) or produced through the transformation of these pollutants (e.g., O_3). Such copollutants could either serve as potential confounders of the observed PM-health associations or as effect modifiers that influence the magnitude of PM associated effects. The studies used in the risk analysis provide PM coefficients from areas with widely varying levels of copollutants. One approach to controlling for the potential effects of copollutants is to include copollutants simultaneously in the model with PM when estimating the PM coefficient for a health endpoint. However, this method may be limited by collinearity in the pollutants of interest (Samet et al., 1996b). (For a fuller treatment of copollutants, potential confounding, and the significance of observed variations across study locations, see Chapter V and CD, Chapters 12 and 13).

The base case analysis used concentration-response relationships estimated without inclusion of copollutants, and it is not possible to directly estimate the sensitivity of the base case results taking into account the effect of simultaneous inclusion of copollutants, since not all the studies used for the base case examined copollutants in this manner. As an alternative, the sensitivity of individual study estimates in relationship to inclusion of copollutants is examined in Tables F-5A and F-5B. Table F-5A provides a comparison of the coefficients for studies that reported PM coefficients both with and without inclusion of copollutants, and Table F-5B provides the risk estimates obtained from applying those coefficients to Philadelphia County in the risk analysis. The results in these two tables provide a more general sense of how much of an effect inclusion of copollutants typically has on the magnitude of the health risk estimates and, thus, potentially on the base case results. The results for many, but not necessarily all, of the studies are consistent with the assessment in the CD that PM effect sizes and their statistical uncertainty in most studies showed little sensitivity to the adjustment for copollutants (CD, p.13-55).

Table F-5a. Sensitivity Analysis: Effect of Copollutants
Relative Risks for Change of 50 ug/m3 PM-10 or 25 ug/m3 PM-2.5

Health Effects Associated with Short-Term Exposure		Study, Pollutant, & Location	Relative Risk No Copollutant	Relative Risk with Daily Average SO2	Relative Risk with Daily 1-hour Maximu CO	Relative Risk with Daily Average O3	Relative Risk with Daily 1-hour Maximum O3
Mortality		Ito & Thurston 1995, PM-10 Chicago	**1.02** **(1.02 - 1.04)**			1.02 (1.01 - 1.03)	
		Kinney et al., 1995, PM-10 Los Angeles	**1.02** **(1.00 - 1.05)**		1.02 (0.99 - 1.04)		1.02 (1.00 - 1.05)
		Pope 1994, PM-10 Utah Valley, summer only	1.11 (0.95 - 1.31)			1.14 (0.96 - 1.37)	1.19 (1.00 - 1.43)
Hospital Admissions	All respiratory (all ages)	Thurston et al., 1994, PM-2.5 Ontario, Canada	**0.086*** **(0.024 - 0.15)**				0.045* (-0.028 - 0.12)
	All respiratory (ages >64)	Schwartz 1995, PM-10 New Haven	**1.06** **(1.01 - 1.12)**	1.07 (1.02 - 1.13)		1.09 (1.01 - 1.18)	
		Schwartz 1995, PM-10 Tacoma	**1.10** **(1.04 - 1.16)**	1.11 (1.03 - 1.19)		1.12 (0.99 - 1.26)	
	Pneumonia (ages >64)	Schwartz 1994, PM-10 Minneapolis/St. Paul				**1.08** **(1.02 - 1.14)**	
		Schwartz 1994, PM-10 Detroit				**1.06** **(1.03 - 1.09)**	
	COPD (ages >64)	Schwartz 1994, PM-10 Detroit				**1.10** **(1.06 - 1.16)**	
	Ischemic Heart Disease	Schwartz & Morris 1995, PM-10 Detroit	**1.028** **(1.011 - 1.047)**	1.024** (1.005 - 1.043)	1.025 (1.007 - 1.044)		
	Congestive Heart Failure	Schwartz & Morris 1995, PM-10 Detroit	**1.050** **(1.024 - 1.077)**		1.038 (1.011 - 1.064)		

Results presented in bold come from functions used in the base case analysis.
The number of significant digits given for each relative risk is the same as the number reported in the original study.

* Thurston et al. 1994 provides a function relating changes in PM to changes in the number of cases.
The relative risk calculated from this coefficient may vary widely from location to location, depending on baseline incidences.
Therefore, the coefficient, adjusted to a rate per 100,000 people, is reported, instead of a relative risk.

** Based on 1-hour maximum SO2.

Table F-5b. Sensitivity Analysis: Effect of Copollutants on Predicted Health Effects Associated With "As-Is" PM* Philadelphia County, September 1992 - August 1993

			Percent of total incidence associated with PM above background				
Health Effects		Study & Location	with no copollutant	with daily average SO2	with daily 1-hour maximum CO	with daily average O3	with daily 1-hour maximum O3
Mortality		Ito & Thurston 1996, PM-10 Chicago	**0.8% (0.3 - 1.3)**			0.6% (0.2 - 0.9)	
		Kinney et al., 1995, PM-10 Los Angeles	**0.4% (0.0 - 0.8)**		0.3% (-0.0 - 0.7)		0.4% (0.0 - 0.8)
		Pope 1994, PM-10 Utah Valley, summer only	3.0% (-1.5 - 7.2)			3.7% (-1.3 - 8.3)	4.8% (-0.2 - 9.4)
Hospital Admissions	All respiratory (ages >64)	Thurston et al., 1994, PM-2.5 Ontario, Canada	**NA NA**				NA NA
	All respiratory (ages >64)	Schwartz 1995, PM-10 New Haven	**2.4% (0.3 - 4.5)**	1.9% (0.6 - 3.4)		2.4% (0.4 - 4.6)	
		Schwartz 1995, PM-10 Tacoma	**3.2% (-0.2 - 6.4)**	2.9% (1.0 - 4.7)		3.2% (-0.2 - 6.4)	
	Pneumonia (ages >64)	Schwartz 1994, PM-10 Minneapolis/St. Paul				**2.2% (0.6 - 3.8)**	
		Schwartz 1994, PM-10 Detroit				**1.6% (0.7 - 2.5)**	
	COPD (ages >64)	Schwartz 1994, PM-10 Detroit				**2.8% (1.5 - 4.2)**	
	Ischemic Heart Disease	Schwartz & Morris 1995, PM-10 Detroit	**0.8% (0.3 - 1.3)**	0.7%** (0.1 - 1.2)	0.7% (0.2 - 1.2)		
	Congestive Heart Failure	Schwartz & Morris 1995, PM-10 Detroit	**1.4% (0.7 - 2.1)**		1.1% (0.3 - 1.8)		

Results presented in bold come from functions used in the base case analysis.

* Health effects associated with short-term exposure to PM. Incidence was quantified across the range of PM concentrations observed in each study, but not below background PM levels, assumed to be 8 ug/m3 for PM-10 and 3.5 ug/m3 for PM-2.5.

** Based on 1-hour maximum SO2.

The numbers in parentheses for pooled functions are NOT standard confidence intervals. All numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability. See text in Chaptet VI for details.

Two substantial uncertainties remain concerning copollutants and the method of controlling for their effects through simultaneous inclusion in the health risk model. First, to what degree is it possible that the associated copollutant does not have a bona fide independent effect on mortality separate from PM? If the copollutant does not have an independent effect on mortality, then changes in the PM coefficient resulting from inclusion of the second pollutant may just be the results of collinearity between the pollutants and may not accurately reflect the underlying PM coefficient. Second, if the changes seen with inclusion of copollutants actually do reflect a bona fide improvement in the estimate of the PM effect, then is it possible simultaneous inclusion of additional copollutants would further reduce the coefficient? As pointed out by Samet et al. (1996b) and in Chapter V, examination of effects within a single location may often be limited by collinearity between pollutants and comparison across geographic areas may be required for a fuller assessment of the potential effects of copollutants on reported PM concentration-response relationships.

4. Sensitivity Analysis Concerning Reduction in the Slope of Concentration-Resposer Relationships for Risks from Long-Term Exposures

Two major concerns have been raised concerning whether the slope of the concentration-response relationships from recent studies of mortality from long-term exposures (Dockery et al., 1993, Pope et al., 1995) may be misestimated. One major uncertainty concerning the studies of health risks associated with long-term exposures to PM for adults is the potential relevance of air quality concentrations previous to the period of monitoring in the study. If long-term air quality concentrations previous to the period being monitored: 1) are relevant for a substantial portion of the population for the endpoint being studied, and 2) are substantially different than concentrations monitored during the study, then the actual long-term concentration-response relationship may be substantially different than that observed in the reported study (CD, p.13-34). The second major uncertainty relates to whether inadequate control of potential confounders may substantial alter the reported concentration-response relationships (CD, pp. 12-140-43, 12-165, 12-176-178).

The question of the degree to which previous (from years to decades) air quality exposures might have affected mortality risk is complex.[1] In addition, quantitative information on the levels of previous air quality concentrations is difficult to ascertain, especially for $PM_{2.5}$. The CD reports that for the monitoring data reported in the Six City mortality study, downward trends in $PM_{2.5}$ mass are evident for four of the six cities (CD, p. 13-14).

Given these uncertainties in developing a quantitative basis for a sensitivity analyses concerning historical air quality, Table F-6 simply shows the potential impact of mortality risk estimates associated with long-term exposures if one assumes that previous air quality concentrations reduce the observed slope of the PM concentration-response relationship by 33% (modeling the case if relevant previous $PM_{2.5}$ concentrations averaged approximately 50% higher than that monitored in the study period) and by 50% (modeling the case if relevant previous $PM_{2.5}$ concentrations were twice as high). As expected, positing that the most important $PM_{2.5}$ concentrations in regards to effects on mortality risk occurred before the study monitoring period leads directly to similarly proportional reductions (approximately 33% and

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[1] Judging the extent to which previous air quality may be a significant concern for the estimates of risk from long-term exposures requires consideration of both of past air quality variability *and* of the relevant exposure period that might be expected to affect mortality risk for a substantial portion of the cohort population The CD notes that a detailed investigation of temporal relationships has not been attempted in the cohort studies, but also notes that if responses reflect primarily the last few years of integrated exposure then the concurrent average monitoring data would be reasonably predictive (CD, p. 12-171, 12-181). Some findings from air pollution epidemiology suggest recent exposures may be of primary importance. The reduction in mortality incidence observed with a reduction in PM concentrations for 14 months in Utah Valley suggests that a significant amount of the mortality of substantial prematurity associated with particles in that location did not appear dependent on exposures over the span of years, since changes in mortality rates could be observed with a relatively brief temporal change (a 14 month period of reduced concentrations) in long-term average PM pollution

Observations of the temporal relationship of exposure to mortality risk for a large portion of cardiovascular mortality (deaths from myocardial infarction) and for lung cancer from cohort studies on active cigarette smoke exposure suggest that elevated risks for myocardial infarction generally return to close to baseline nonsmoking relative risks within three to ten years (Rosenberg et al., 1985; 1990) and that much of the lung cancer risk is reduced close to the risk for never smokers (compared to the marked elevation in relative risk for lung cancer among current smokers) within 10-15 years after cessation of smoking (USEPA, 1992, Table 4-6 and 4-7). The significance of these findings to air pollution effects cannot be assumed, since quite distinct mechanisms for cigarette smoking and particular matter exposure and mortality from cardiovascular and lung cancer causes may be likely. However, the smoking cohort studies show that in one area in which the temporal relationship of exposure to mortality risk from cardiovascular and lung cancer causes has been examined, evidence suggests recent exposures may be substantially more important than less recent exposures

Table F-6. Sensitivity Analysis: The Effect of Concentration-Response Function Slope on Estimated Mortality Associated with Long-term Exposure to PM-2.5*
Philadelphia County, September 1992 - August 1993

Health Effect**	**BASE CASE Assuming AQ as reported**	Assuming relevant AQ 50% higher*	Assuming relevant AQ twice as high***
(A) Mortality associated with	**4.6%**	3.4%	2.3%
long-term exposure	**(2.8 - 6.2)**	(2.1 - 4.7)	(1.4 - 3.2)

*This Table illustrates the sensitivity of mortality risk associated with long-term exposure (A) Pope et al., 1995 if concentration-response function slope were adjusted to reflect possible effects of previous air quality or potential confounders not addressed in the original PM health effects model.

**Health effects incidence was calculated down to the lowest level observed in the study.

*** Adjusted function from Pope et al., 1995. Had historical air quality (AQ) been 50% higher, the relative risk calculated by the study would have been two thirds of that reported. Had historical air quality been twice as high, the relative risk calculated would have been half that reported.

The numbers in parentheses for pooled functions are NOT standard confidence intervals.
All the numbers in parentheses are interpreted as 90% credible intervals based on uncertainty analysis that takes into account both statistical uncertainty and possible geographic variability.
See text in Chapter VI for details.

50%) in the estimates of long-term mortality risk. To the extent that the estimates of mortality risks from long-term exposure reflect the net sum of acute events that take place over that year (which will occur when increases in daily death rates associated with acute events are not subsequently canceled by decreases ("harvesting") (CD p.12-139), this component of mortality risk from long-term exposures risk is not sensitive to assumptions about previous air quality.

Similar slope reductions can also serve to model concerns about uncontrolled confounding. The CD provides as an example how inclusion of additional ecological variables can attentuate the PM2.5-mortality relationship observed in a initially simply age- and race-adjusted dataset. The direction and extent of change in slope that might be observed by control of such confounders in a prospective cohort design, which features individual data for some risk factors is not certain (CD, pp. 12-176-77), however for the purposes of sensitivity analyses reductions in slope of 33-50% for the long-term studies will be assumed appropriate appropriate to reflect the viewpoint that exhibits substantial concerns about residual uncontrolled confounding in these studies. These would result in the same proportional reductions of approximately 33-50% in the estimates of long-term mortality risk (relative to base case assumptions) as when this slope reduction was considered as a sensitivity analysis for the potential effects of previous air quality.

Appendix G

MEASURES OF VISIBILITY IMPAIRMENT AND LIGHT EXTINCTION

Several atmospheric optical indices and approaches can be used for characterizing visibility impairment and light extinction. The CD discusses several indicators that could be used in regulating air quality for visibility protection, including: 1) light extinction (and related parameters of visual range and deciview) calculated from measurements of fine particle constituents and their associated scattering and absorption; 2) light extinction measured directly by transmissometer; 3) light scattering by particles, measured by nephelometer; 4) fine particle mass concentration; 5) contrast transmittance (CD, 8-125).

In conjunction with the National Park Service, other Federal land managers, and State organizations, EPA has supported since 1986 a monitoring protocol utilizing a combination of the first four measurements. This long-term visibility monitoring network is known as IMPROVE (Interagency Monitoring of PROtected Visual Environments. The following discussion briefly describes the IMPROVE protocol and provides rationale supporting use of the light extinction coefficient, derived from both direct optical measurements and measurements of aerosol constituents, for purposes of implementing air quality management programs to improve visibility.

IMPROVE provides direct measurement of fine particles and precursors that contribute to visibility impairment at more than 40 mandatory Federal Class I areas across the country. The IMPROVE network employs aerosol, optical, and scene measurements. Aerosol measurements are taken for PM_{10} and $PM_{2.5}$ mass, and for key constituents of $PM_{2.5}$, such as sulfate, nitrate, organic and elemental carbon, soil dust, and several other elements. Measurements for specific aerosol constituents are used to calculate "reconstructed" aerosol light extinction by multiplying the mass for each constituent by its empirically-derived scattering and/or absorption efficiency. Knowledge of the main constituents of a site's light extinction "budget" is critical for source apportionment and control strategy development. Optical measurements are used to directly measure light extinction or its components. Such measurements are taken principally with either a transmissometer, which measures total light extinction, or a nephelometer, which measures particle scattering (the largest human-caused component of total extinction). Scene characteristics are recorded 3 times daily with 35

millimeter photography and are used to determine the quality of visibility conditions (such as effects on color and contrast) associated with specific levels of light extinction as measured under both direct and aerosol-related methods. Because light extinction levels are derived in two ways under the IMPROVE protocol, this overall approach provides a cross-check in establishing current visibility conditions and trends and in determining how proposed changes in atmospheric constituents would affect future visibility conditions.

The light extinction coefficient has been widely used in the U.S. for many years to describe visibility conditions and the change in visibility experienced due to changes in concentrations of air pollutants. As noted earlier, the extinction coefficient can be defined as the fraction of light lost or redirected per unit distance through interactions with gases and suspended particles in the atmosphere. Direct relationships exist between measured ambient pollutant concentrations and their contributions to the extinction coefficient. The contribution of each aerosol constituent to total light extinction is derived by multiplying the aerosol concentration by the extinction efficiency for that aerosol constituent. Extinction efficiencies vary by type of aerosol constituent and have been obtained through empirical studies. For certain aerosol constituents, extinction efficiencies increase significantly with increases in relative humidity.

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

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

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

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

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

Visual range is a measure of visibility that is inversely related to the extinction coefficient. Visual range can be defined as the maximum distance at which one can identify a black object against the horizon sky. The colors and fine detail of many objects will be lost at a distance much less than the visual range, however. Visual range has been widely used in air transportation and military operations in addition to its use in characterizing air quality. Because it is expressed in familiar units and has a straightforward definition, visual range is likely to continue as a popular measure of atmospheric visibility (Pitchford and Malm, 1994). Conversion from the extinction coefficient to visual range can be made with

the following equation (NAPAP, 1991):

$$\text{Visual Range} = 3.91/\sigma_{ext}$$

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

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

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

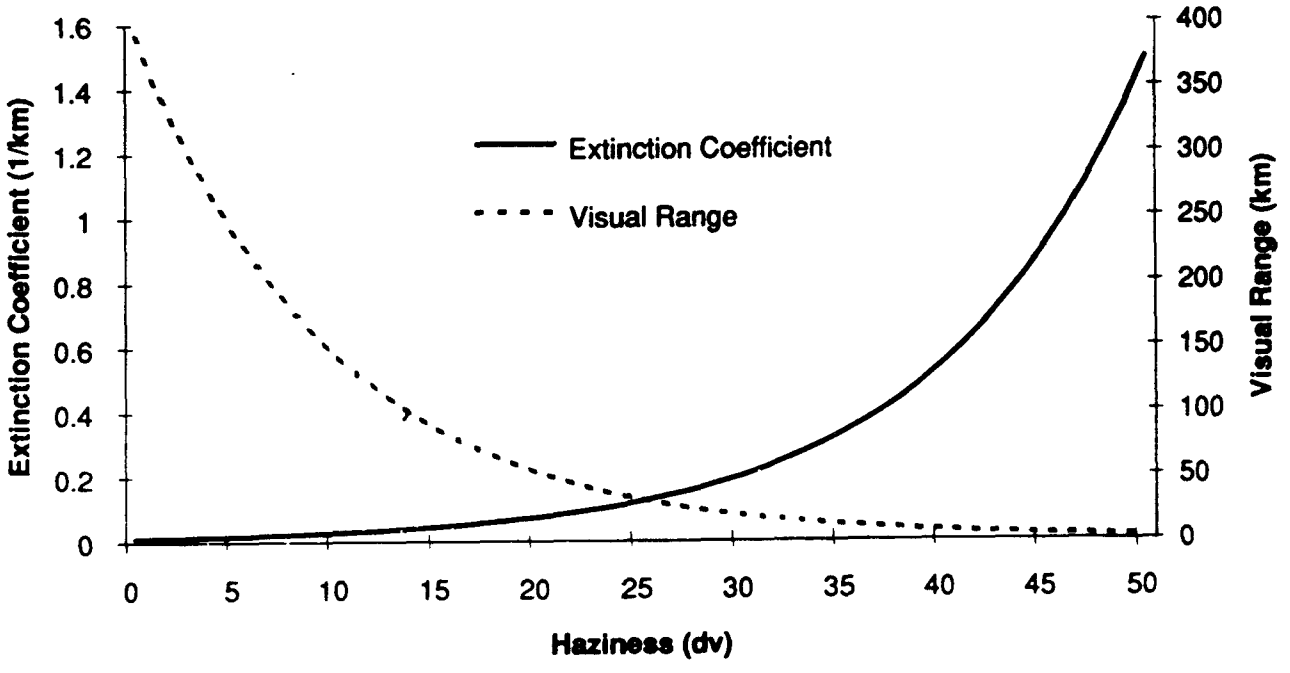

FIGURE G-1. VISUAL RANGE AND EXTINCTION COEFFICIENT AS A FUNCTION OF HAZINESS EXPRESSED IN DECIVIEW

Source: Pitchford and Malm, 1994

APPENDIX H
CLEAN AIR SCIENTIFIC ADVISORY COMMITTEE
CLOSURE LETTERS

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

March 15, 1996

EPA-SAB-CASAC-LTR-96-005

Honorable Carol M. Browner
Administrator
U.S. Environmental Protection Agency
401 M. Street SW
Washington, DC 20460

Re Closure by the Clean Air Scientific Advisory Committee (CASAC) on the draft Air Quality Criteria for Particulate Matter

Dear Ms. Browner:

The Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board (SAB) has held a series of public meetings during its peer review of the Agency's draft documents which will form part of the basis for your decision regarding the National Ambient Air Quality Standards (NAAQS) for Particulate Matter (PM). The Committee has held public meetings on December 12-13, 1994 (planning and introductory issues); August 3-4, 1995 (review of the initial draft Criteria Document); December 14-15, 1995 (review of the revised draft Criteria Document and the first draft of the Staff Paper); and February 29, 1996 (review of the revised draft Criteria Document - specified chapters only). A review of the revised draft Staff Paper is planned for May 16-17, 1996. The primary Agency draft documents that we have reviewed are the: a) *Air Quality Criteria for Particulate Matter* (the Criteria Document prepared by the National Center for Environmental Assessment - Research Triangle Park, NC - ORD), and b) *Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information* (the Staff Paper prepared by the Office of Air Quality Planning and Standards - Research Triangle Park, NC - OAR).

As part of our review process, we have kept you informed of our findings through two letter reports: a) *Clean Air Scientific Advisory Committee (CASAC) Comments on the April 1995 draft Air Quality Criteria for Particulate Matter* (EPA-SAB-CASAC-LTR-95-005; August 30, 1995); and b) *Clean Air Scientific Advisory Committee (CASAC) Comments on the November, 1995 Drafts of the Air Quality Criteria for Particulate Matter and the Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information (OAQPS Staff Paper)*, (EPA-SAB-CASAC-LTR-96-003, January 5, 1996)

The Clean Air Scientific Advisory Committee, supplemented by a number of expert Consultants (hereinafter referred to as the Panel) reviewed a revised draft of the PM Criteria Document and a first draft of the Staff Paper for Particulate Matter at a meeting on December 14-15, 1995 in Chapel Hill, NC. At that meeting and in subsequent written comments by individual members which were provided to EPA Staff, the Panel made numerous recommendations for improving the draft document. The Panel was impressed with the breadth and scope of the latest revision of the draft Criteria Document and agreed that, except for Chapters 1 (Executive Summary), 5 (Sources and Emissions), 6 (Air Quality), and 13 (Integrative Synthesis), only minor revisions would be necessary to make the remainder of the draft Criteria Document satisfactory for providing an adequate scientific basis for regulatory decisions on PM based on available information. However, the Panel felt that Chapters 1, 5, 6, and 13 required major revisions which the Panel would need to review again.

On February 29, 1996, the Panel again met in Chapel Hill, NC to review revised drafts of Chapters 1, 5, 6, and 13 of the Criteria Document. While Chapter 13 can be improved, as suggested below, I want to take this opportunity, on behalf of the entire Panel, to commend Dr. Lester Grant and his staff in the National Center for Environmental Assessment (NCEA) for producing its best ever example of a true integrative summary of the state of knowledge about the health effects of airborne PM and the associations between the effects and the various available indices of PM exposure. NCEA has outlined some of the options for your subsequent choice of available PM indicators for a NAAQS by examining the degrees of association between various health indices and PM indicators including total suspended particulate (TSP), thoracic particulate (PM_{10}), fine particulate ($PM_{2.5}$), sulfate particulate ($SO_4^=$), acid particulate (H^+) and carbonaceous particulate (BS and CoH), with available knowledge from dosimetry, results of controlled human exposure studies in humans and laboratory animals, and mechanistic understandings. This thorough review and evaluation also provides an important starting point for focussing the future PM research program on studies that can better identify the compositional and particle size characteristics of the most biologically active agents within the PM_{10}. We were especially impressed that this integrative summary could be produced in the short time period since our review of the initial rough draft in December 1995.

This letter is a summary of our findings and conclusions from the February 29th meeting. Our comments reflect our satisfaction with the improvements made in the scientific quality and completeness of these chapters. The changes made in these chapters are consistent with our earlier recommendations. However, the Panel provided additional comments to your staff at the meeting and subsequently in writing. Although we feel that it is essential to have these additional comments considered for incorporation in the Criteria Document, we did not feel that it was essential to review another revised version and, thus, we came to closure on the entire Criteria Document anticipating incorporation of our suggested changes. It was our consensus that although our understanding of the health effects of PM is far from complete, a revised Criteria Document which incorporates the Panel's latest comments will provide an

adequate review of the available scientific data and relevant studies of PM. With the incorporation of our suggested changes, the revised Criteria Document will be very comprehensive and will provide an adequate scientific basis for regulatory decisions on particulate matter based on available information. However, a number of members have expressed concern that since we are closing on the Criteria Document before we will be able to see the revised version, we have no assurance that our comments will be incorporated. I will return to this concern later.

I would like to summarize for you the Panel's major comments on Chapters 1, 6, and 13. There were no major comments on Chapter 5. In Chapter 6, Panel members raised issues concerning the definition and level of background PM concentrations. The Panel has provided the Agency with guidance in the written comments to resolve these concerns. This is an important issue because some studies suggest effects at levels which approach background concentrations.

Of the 17 members of the Panel present, five were satisfied with Chapter 13 as is, four had no substantive comments because their expertise was outside of Chapter 13, and eight had some substantive comments on one or more aspects of the chapter which I summarize below. The members who were satisfied with the chapter praised the Agency for making a compelling case for $PM_{2.5}$ being the best available surrogate index for the causal agent. They thought EPA presented a large body of consistent and coherent studies and that they were appropriately presented as an integrative synthesis. The issues raised by the other Panel members regarding Chapter 13 fell into three categories. First, several Panel members felt that additional discussions of the inherent errors associated with air sampling, estimating human exposure from central monitoring data, and relating these data to excess mortality and morbidity were necessary so that the uncertainties of the relative risk estimates would be better appreciated.

Second, about half of the Panel members expressed concern that the case made in the Criteria Document for $PM_{2.5}$ being the best available surrogate for the principal causative agent in PM_{10} may be overstated, and that EPA has not adequately justified its rejection of other alternative explanations discussed next. In addition, it needs to be acknowledged that large particles (e.g., $d \approx 4.0\ \mu m$) may be responsible for acute respiratory effects, especially in susceptible groups such as asthmatics.

Third, several Panelists pointed out that a number of recently published (or in-press) studies (including the Health Effects Institute study), which were conducted to critically evaluate some of the epidemiological studies using alternative models or including additional gaseous pollutant data, present a different perspective of the PM/mortality issue than the one presented in this chapter. Collectively, these reanalyses have confirmed the reproducibility of the earlier studies, but they also present a more complicated relationship in which causality does not appear to be unambiguously attributed to any single pollutant let alone a specific portion of the PM. EPA on the other hand emphasized a PM causal conclusion based on the pattern of

associations across multiple sites having different pollutant mixtures. These results need to be discussed adequately in Chapter 13.

Our only comments on Chapter 1, the Executive Summary, were that it reflect the revisions that have been recommended for Chapters 6 and 13.

As mentioned above, Panel members have expressed concern that the Agency may not be responsive to some of our comments or may misinterpret them since we will not have another opportunity to review the final document. This concern is another unfortunate consequence of the court- mandated "accelerated" time schedule, but nevertheless, it is a real concern. We anticipate being advised of text changes made in response to our concerns prior to or at the May 16-17, 1996 meeting, and we can advise you afterward about whether our concerns have been adequately addressed by the Agency.

On behalf of the Panel, I would like to thank EPA staff for their considerable efforts in preparing the Criteria Document on the accelerated schedule. We look forward to seeing the revised final version once it is completed. The Panel also looks forward to reviewing the revised Staff Paper during the public meeting presently scheduled for May 16-17, 1996.

Sincerely,

George T. Wolff

Dr. George T. Wolff, Chair
Clean Air Scientific
Advisory Committee

March 20, 1996

Honorable Carol M. Browner
Administrator
U. S. Environmental Protection Agency
401 M Street SW
Washington DC 20460

re: Supplement to the Closure Letter from the Clean Air Scientific Advisory Committee

Dear Ms. Browner:

The co-signers of this letter are members of the Particulate Matter Criteria Document Review Panel and consultants to the Clean Air Scientific Advisory Committee (CASAC) of the Science Advisory Board, U.S. EPA. This letter is not being sent as a minority report to the CASAC closure letter, but as a supplement to address some of the concerns raised in the CASAC letter. We were selected for the CASAC review of the Particulate Matter Criteria Document because of our combined expertise in the interpretation of epidemiological studies, our understanding of the literature on the human health effects of particulate air pollution, and our familiarity with the use of air monitoring data in analyzing human health effects. As individuals, we have been extensively involved in conducting studies of population exposure to air pollution and evaluating the human health effects of this exposure.

As noted in the closure letter to you on the draft Air Quality Criteria for Particulate Matter from the Chair of CASAC, the Panel members praised the EPA criteria document for its excellent integrative synthesis of the literature. Overall, most panel members concluded that the document made a persuasive case that population exposure to particulate matter (PM) is causally associated with excess mortality and morbidity in the U. S. even at concentrations at and below the existing primary air quality standard. While the cosigners of this letter are in agreement with this judgment, we are aware that some of our Panel colleagues have reservations about this important conclusion. Our purpose in this supplementary letter is to make explicit our reasons for reaching our conclusion, in order to assist the staff of the National Center for Environmental Assessment in addressing the reservations of our colleagues. We also

intend our comments to aid the staff of the Office or Air Quality Planning and Standards in preparing its staff paper in support of a revised particulate air quality standard.

The closure letter from the Chair of CASAC notes that the concerns of Panel members who are not in full agreement with the above conclusion fall into three categories:

1. Uncertainties in the human health risks of particulate air pollution, arising from errors in air monitoring, from estimating human exposure from central monitoring data, and from relating these data to excess mortality and morbidity.

2. Concern that the case for $PM_{2.5}$ being the best available surrogate for the principal causative agent in particulate air pollution may be overstated, and that EPA has not adequately justified its rejection of other alternative explanations.

3. Recently published studies that appear to contradict, or at least to present a different perspective on, the conclusions reached by EPA in its integrative synthesis of the literature.

Regarding these concerns overall, the writers of this letter wish to make it clear that we are not arguing that $PM_{2.5}$ is the causal agent of the observed excess mortality and morbidity associated with particulate air pollution. In our judgment, the studies reviewed in the criteria document, specifically those considered in Chapter 12 (Epidemiological Studies), are persuasive in demonstrating a causal relationship between particulate air pollution, as measured by different methods in the various studies, and excess mortality and morbidity. However, the evidence does not allow us to conclude that a specific physical or chemical component of the particulate mass is clearly the responsible causal agent. Our conclusion is analogous to making the assertion that cigarette smoke is a cause of lung cancer and nonmalignant respiratory disease,even though the specific causal agent in cigarette smoke has not been identified among the many chemicals known to be present in cigarette smoke.

The reasons for concluding that particulate air pollution is causally related to excess mortality and morbidity have been well stated in the integrative synthesis (Chapter 13) of the criteria document. For heuristic purposes, we will summarize these reasons here, and cite locations in Chapter 13 where supporting sentences and paragraphs are presented:

- A large number (20) of epidemiological time-series studies have consistently found a statistically significant association between daily variation in particulates and total mortality in cities of the U.S., Canada, Latin America, the U.K. and continental Europe. These findings argue against the associations being attributable to statistical sampling variation, i.e. the role of chance (Section 13.4.1.1).

- The results of these time-series studies cannot be attributed to the vagaries of statistical modeling (Section 13.4.3.2), nor to confounding by season or weather (Section 13.4.3.3).

- The results of the time-series studies cannot be attributed to other criteria air pollutants. The mortality effect of particulates is found whether or not other pollutants are present at elevated concentrations, though it is difficult to separate the effects of particulates from other pollutants when the latter covary with particulates. The most persuasive evidence that the causal agent is some component of the airborne particulate mass is in studies of cities or seasons where other pollutants are present at very low concentrations. Across the range of the 20 studies mentioned above, particulate air pollution is the only pollutant that is consistently associated with excess daily mortality, and the estimate of its effect is relatively stable when adjusted for the presence of co-pollutants. There are exceptions to this stability, particularly in those cities where particulate and gaseous air pollutants are highly intercorrelated. But no monitored air pollutant, other than particulate matter, can account for the consistently observed excess mortality in these studies (Section 13.4.3.4). Excess morbidity from cardiopulmonary diseases has also been observed in a considerable number of studies (Section 13.4.1.2), and the morbidity relationship with ambient particulate concentrations is stronger overall and more consistent than for any other air pollutant.

- There is considerable coherence between the observed mortality and morbidity effects of particulate air pollution. Not only is excess mortality from cardiovascular and respiratory diseases observed, but on days of higher particulates excess hospitalizations for cardiovascular and respiratory diseases are reported. These mortality and morbidity excesses are strongest in populations that would be expected to be more susceptible to the effects of air pollution, particularly the elderly. The relation of particulates with mortality is strongest also for cardiopulmonary diseases rather than for other disease categories. On days of high particulates, there is an increased proportion of deaths from chronic obstructive pulmonary disease, pneumonia, heart disease and deaths among the elderly than on days of low particulates. These findings

are supportive of a causal role for particulate air pollution, since they are health endpoints one would most anticipate from exposure by the inhalation route (Section 13.4.3.5 and Section 13.5.1).

Given the striking consistency of the above studies, their robustness to variations in statistical modeling, the coherence among different but closely related health endpoints, and the empirical elimination of any alternative explanation for the findings, we conclude that a causal interpretation for particulate air pollution exposure is reasonable and defensible. This conclusion is further supported by longitudinal cohort studies of populations in which a geographical gradient in particulate air pollution was associated with a corresponding gradient in total mortality, in cardiopulmonary mortality and in lung cancer. These studies carefully controlled for other individual risk factors for these health endpoints (Section 13.4.1.1).

With specific reference to the first category of concern expressed by our Panel colleagues, although population exposure to air pollution cannot be perfectly estimated based on central monitoring , these inherent errors in exposure estimation are more likely to cause an underestimation of the adverse health effects associated with pollution exposure, particularly in longitudinal cohort studies where individual risk factors and exposures are directly related to health effects. Thus the consistent positive findings cannot be attributed to exposure measurement error. Furthermore, there is growing evidence that fine particles are more uniformly distributed over large geographic areas than are coarse particles (Section 13.2.4), that measurements at one site give a reasonable estimate of the fine particulate concentrations across a city (Section 13.2.6), and that fine particles penetrate and have longer lifetimes indoors than coarse particles (Section 13.2.6). This evidence supports using ambient measures of fine particulates at a central site as an acceptable estimate of the average exposure of people in the community (Section 13.2.6). For these reasons, we judge that uncertainties arising from air monitoring and human exposure estimation do not negate the consistent excess mortality and morbidity associations discussed above.

With regard to the second concern of our Panel colleagues, we believe that the case has been made that fine particulates, as measured by $PM_{2.5}$, are the best surrogate currently available for the component of particulate air pollution that is associated with excess mortality and morbidity. We emphasize once again that we are not claiming that $PM_{2.5}$ is the causal agent, but rather that $PM_{2.5}$ is a better measure, than any alternative metric, of the complex in the particulate mass that is causing excess mortality and morbidity. Distinguishing between PM_{10} and $PM_{2.5}$ is difficult, given the high correlation between these two pollutants in both time and

space. In many studies, either metric will provide nearly the same estimate of the exposure-response relationship. However, a number of recent re-analyses of mortality and morbidity have been performed to address the issue of whether fine or coarse particulates (the latter indexed by subtracting $PM_{2.5}$ from PM_{10}) more consistently predicts a relationship with adverse health effects. These studies, as reviewed Section 13.4.1.1 and Tables 13-3, 13-4 and 13-5 of the Criteria Document, conclude that excess mortality, hospital admissions for respiratory diseases and decreased lung function are more strongly and consistently associated with fine rather than with coarse mode particulates. These findings are also supported by earlier studies in the U.K. in which British Smoke measurements, which primarily reflect the contribution of the fine particle mode, were consistently associated with excess mortality. Finally, several characteristics of fine mode particles, as opposed to the coarse mode, are more consistent with the observed excess mortality and morbidity observed in epidemiological studies. As noted earlier, these characteristics are: (1) fine particulates are more uniform in distribution than the coarse mode across urban areas, (2) fine particulates penetrate into indoor environments more completely than coarse particles, and (3) fine particulates have a more prolonged residence time in indoor air than coarse particles. These points are discussed in Section 13.7, Summary and Conclusions. Given that a causal association of excess mortality and morbidity with particulate air pollution has been established, we concur with staff's judgments that fine particulates are the best available surrogate for the population exposures associated with these health effects.

With regard to the third concern of our Panel colleagues, some studies have recently been published that are interpreted as contradicting the conclusion that particulate air pollution is causally associated with excess mortality and morbidity. We agree that, in its revision of the criteria document, EPA needs to address these apparent discrepancies more explicitly, and we offer the following comments to assist staff in that task.

First, the Health Effects Institute (HEI) reanalysis does not contradict any of the above conclusions. The HEI analysis conclusively demonstrated that the positive findings from the original studies selected for reanalysis were replicable, were not an artifact of statistical modeling, and were not confounded by idiosyncrasies in the method to control for season or weather. The HEI investigators then proceeded to apply their statistical modeling procedure to data from Philadelphia. They reported moderately high intercorrelations between particulates, as measured by total suspended particulate (TSP) measurements, and several of the pollutant gases, and, as expected, found that under these conditions, they could not attribute the observed exposure-response mortality relationships to TSP alone. They further observed that the TSP and SO_2 effects were not independent of one another, and that the TSP effect was stronger in some

seasons of the year and at some concentrations of SO_2, while the SO_2 effect was stronger in other seasons and at some concentrations of TSP. The HEI investigators appropriately concluded that, because of the high intercorrelations between pollutants in Philadelphia, mortality effects could not be attributed solely to particulates. More importantly, in their further report on this phase of their study, they concluded that "insights into the effects of individual criteria pollutants can be best gained by assessing effects across locations having different pollutant mixes and not from regression modeling of data from single locations" ("Air Pollution and Mortality in Philadelphia, 1974-1988", interim report dated February 9, 1996). The EPA Criteria Document undertakes this assessment of effects across locations having different pollutant mixes, and this assessment was discussed above (in the third bulletted paragraph)

One published reanalysis (Moolgavkar S: Epidemiology 1995; 6: 476-484) of the Philadelphia mortality data set has been interpreted as contradicting the findings of the original study (Schwartz J & Dockery DW: Am Rev Resp Dis 1992: 145: 600-604), which concluded that particulates were positively associated with variations in daily mortality. However, the HEI re-analysis, reported above, confirmed the findings of the original study, but, more importantly, noted that it was not possible in Philadelphia to attribute the mortality effect exclusively to particulates or individual gaseous pollutants, due to their high intercorrelations, as previously discussed. Separation of the effects of these pollutants requires analyses in a variety of locations with different pollutant mixes.

Presentations and papers by Lipfert and Wyzga (Inhalation Toxicology 1995; 7: 671-689) discuss uncertainties in identifying responsible pollutants in epidemiological studies. The latter article raises the important issue of measurement error, but in applying its analysis to the Philadelphia data set, it encounters the same problem of intercorrelated pollutants and the inability to partition health effects exclusively or primarily to one of the pollutants. Similarly, the analysis of the Philadelphia data set by Li and Roth (Inhalation Toxicology 1995; 7: 45-58) purports to show that a panoply of seemingly conflicting findings is produced with different modeling strategies, but this paper is superseded by the HEI report, which shows conclusively that the confounding effect of weather was appropriately controlled in the original analysis, and that the original results are not an artifact of the modeling strategy.

Finally, among papers considered as not supporting the main conclusion of the EPA criteria document, that of Styer et al. (Environ Health Perspec 1995; 103: 490-497) fitted separate regressions to each month of the year and found significant particulate effects only in a few of the months. But such partitioning of data in small time segments is considered to be

inappropriate because it results in a significant loss of statistical power and thus a loss of sensitivity to the moderate relative risk associated with ambient air pollution and a loss of ability to separate the effects of one pollutant as opposed to another.

There are several reasons why the mortality and morbidity effects of particulate air pollution will not be the same in all cities and at all seasons of the year. Therefore, there will not be total agreement among all published studies in the magnitude of the adverse effect per unit of particulate exposure. The reasons for these variations in estimates of the exposure-response relationship are several (as discussed in Section 13.4.1.1): (1) the toxicity of particulates likely depends on size distribution and chemical composition, and these characteristics vary among geographic areas. (2) local populations differ in demographic and socioeconomic characteristics, and these differences will be likely to modify the health effects of particulate exposures. (3) the health status of communities differs among geographic areas, and thus the susceptibility of populations to the same level of particulate air pollution will vary. (4) average levels of copollutants will vary across geographic areas, and these may cause small or moderate variations in the particulate effect. In spite of these considerations, there is a remarkable consistency in the body of epidemiological studies, showing a positive exposure-response association between particulates and mortality and morbidity. In our judgment, EPA has appropriately synthesized this evidence and drawn a responsible public health conclusion, namely, that particulate concentrations at current levels are causally associated with excess mortality and morbidity. Furthermore, we agree that fine particulates, as currently indexed by $PM_{2.5}$, are the most appropriate indicator for the component of the particulate air mass to which these adverse effects are attributed. We also agree that some adverse health effects may be related to the coarse particulate mode, and that therefore it is desirable to consider fine and coarse mode particulates as separate candidates for air quality standards. This is the final conclusion of Chapter 13 of the Criteria Document, and we hope that our discussion will assist the EPA staff in presenting firmer support for their conclusion.

Sincerely,

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New York University

Jan Stolwijk, Professor
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Carl Shy, Professor and Chair
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c: Members of the Particulate Matter Criteria Document Review Panel

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

June 13, 1996

EPA-SAB-CASAC-LTR-96-008

Honorable Carol M. Browner
Administrator
U.S. Environmental Protection Agency
401 M. Street SW
Washington, DC 20460

Subject: Closure by the Clean Air Scientific Advisory Committee (CASAC) on the Staff Paper for Particulate Matter

Dear Ms. Browner:

The Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board (SAB) has held a series of public meetings during its peer review of the Agency's draft documents which will form part of the basis for your decision regarding the National Ambient Air Quality Standards (NAAQS) for Particulate Matter (PM). The Committee has held public meetings on December 12-13, 1994 (planning and introductory issues); August 3-4, 1995 (review of the initial draft Criteria Document); December 14-15, 1995 (review of the revised draft Criteria Document and the first draft of the Staff Paper); February 29, 1996 (review of the revised draft Criteria Document - specified chapters only, and the Office of Air Quality Planning and Standards (OAQPS) Risk Assessment Plan); and May 16-17, 1996 (review of the revised draft Staff Paper). The primary Agency draft documents that we have reviewed are the: a) *Air Quality Criteria for Particulate Matter* (the "Criteria Document" prepared by the National Center for Environmental Assessment - Research Triangle Park, NC - ORD), b) *Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information* (the "Staff Paper" prepared by the Office of Air Quality Planning and Standards - Research Triangle Park, NC - OAR), and c) *A Particulate Matter Risk Analysis for Philadelphia and Los Angeles* (draft), 1996, Prepared by Abt Associates for US EPA.

As part of our review process, we have kept you informed of our findings through three letter reports: a) *Clean Air Scientific Advisory Committee (CASAC) Comments on the April 1995 draft Air Quality Criteria for Particulate Matter* (EPA-SAB-CASAC-LTR-95-005; August 30, 1995); b) *Clean Air Scientific Advisory Committee (CASAC) Comments on the November, 1995 Drafts of the Air Quality Criteria for Particulate Matter and the Review of the National Ambient Air Quality Standards for Particulate*

Matter: Policy Assessment of Scientific and Technical Information (OAQPS Staff Paper), (EPA-SAB-CASAC-LTR-96-003, January 5, 1996), and c) *Closure by the Clean Air Scientific Advisory Committee (CASAC) on the draft Air Quality Criteria for Particulate Matter* (EPA-SAB-CASAC-LTR-96-005, March 15, 1996).

The Clean Air Scientific Advisory Committee, supplemented by a number of expert Consultants (hereinafter referred to as the "Panel"), reviewed a first draft of the Staff Paper for Particulate Matter at the December 14 and 15, 1995 meeting in Chapel Hill, NC. At that meeting and in subsequent written comments by individual members which were provided to EPA Staff, the Panel made numerous recommendations for improving the draft document. The Panel met again on May 16, 1996 in Chapel Hill, NC and on May 17, 1996 in Research Triangle Park, NC to review a revised draft of the Staff Paper and the recommendations contained within the Staff Paper for the level and form of the proposed PM NAAQS. This letter is a summary of our findings and conclusions from that meeting.

It was the consensus of the Panel that although our understanding of the health effects of PM is far from complete, the Staff Paper, when revised, will provide an adequate summary of our present understanding of the scientific basis for making regulatory decisions concerning PM standards. Seventeen of the twenty-one Panel members voted for closure. There were two no votes, one abstention, and one absence. However, most of the members who voted for closure did so under the assumption that the Agency would make significant changes to the next version of the Staff Paper which is due by July 15, 1996 (a court ordered mandate). The desired changes have been articulated to your staff at the meeting and subsequently in writing.

The Panel endorses the EPA Staff's recommendation not to establish a separate secondary PM NAAQS for regulating regional haze and agrees that there is an inadequate basis for establishing a secondary NAAQS to reduce soiling and material damage effects.

The attached table (Table I) summarizes the Panel members' recommendations concerning the form and levels of the primary standards. Although some Panel members prefer to have a direct measurement of coarse mode PM ($PM_{10-2.5}$) rather than using PM_{10} as a surrogate for it, there is a consensus that retaining an annual PM_{10} NAAQS at the current level is reasonable at this time. A majority of the members recommend keeping the present 24-hour PM_{10} NAAQS, at least as an option for the Administrator to consider, although those commenting on the form of the standard strongly recommended that the form be changed to one that is more robust than the current standard. There was also a consensus that a new $PM_{2.5}$ NAAQS be established, with nineteen Panel members endorsing the concept of a 24-hour and/or an annual $PM_{2.5}$ NAAQS. The remaining two Panel members did not think any $PM_{2.5}$ NAAQS was justified. However, as indicated in Table I, there was no consensus on the level, averaging time, or form of a $PM_{2.5}$ NAAQS. At first examination of Table I, the diversity of opinion is obvious and appears to defy further characterization. However,

the opinions expressed by those endorsing new $PM_{2.5}$ NAAQS can be classified into three broad categories. Four Panel members supported specific ranges or levels within or toward the lower end of the staff's recommended ranges. Seven Panel members supported specific ranges or levels near, at, or above the upper end of staff's recommended ranges. Eight other Panel members declined to select a specific range or level, but most had comments which appear as footnotes in Table I.

A number of Panel members based their support for a $PM_{2.5}$ NAAQS on the following reasoning: there is strong consistency and coherence of information indicating that high concentrations of urban air pollution adversely affect human health, there are already NAAQS that deal with all the major components of that pollution except $PM_{2.5}$, and there are strong reasons to believe that $PM_{2.5}$ is at least as important as $PM_{10-2.5}$ in producing adverse health effects.

Part of this diversity of opinion can be attributed to the accelerated review schedule. While your staff is to be highly commended for producing such quality documents in such a short period of time, the deadlines did not allow adequate time to analyze, integrate, interpret, and debate the available data on this very complex issue. Nor does a court-ordered schedule recognize that achieving the goal of a scientifically defensible NAAQS for PM may require iterative steps to be taken in which new data are acquired to fill obvious and critical voids in our knowledge. The previous PM NAAQS review took eight years to complete.

The diversity of opinion also reflects the many unanswered questions and uncertainties associated with establishing causality of the association between $PM_{2.5}$ and mortality. The Panel members who recommended the most stringent $PM_{2.5}$ NAAQS, similar to the lower part of the ranges recommended by the Staff, did so because they concluded that the consistency and coherence of the epidemiology studies made a compelling case for causality of this association. However, the remaining Panel members were influenced, to varying degrees by the many unanswered questions and uncertainties regarding the issue of causality. The concerns include: exposure misclassification, measurement error, the influence of confounders, the shape of the dose-response function, the use of a national $PM_{2.5}/PM_{10}$ ratio to estimate local $PM_{2.5}$ concentrations, the fraction of the daily mortality that is advanced by a few days because of pollution, the lack of an understanding of toxicological mechanisms, and the existence of possible alternative explanations.

In recommending that the staff carry out a risk assessment, it was the expectation of CASAC that the risk assessments would narrow the diversity of opinion by evaluating how all of the uncertainties propagate throughout the entire model. However, not all of the uncertainties could be included and the combined effect of all of them could not be examined. The Panel recommended that additional analyses be conducted to present combined uncertainties. However, currently the risk assessments are of limited value in narrowing the diversity of opinion within the Panel.

The Panel is unanimous, however, in its desire to avoid being in a similar situation when the next PM NAAQS review cycle is under way by a future CASAC Panel. The Agency must immediately implement a targeted research program to address these unanswered questions and uncertainties. It is also essential that we obtain long-term $PM_{2.5}$ measurements. CASAC is ready to assist the Agency in the development of a comprehensive research plan that will address the questions which need answers before the next PM review cycle is completed. We understand that your staff is preparing a PM research plan for our review later this summer. We look forward to providing our comments on this important matter.

CASAC recognizes that your statutory responsibility to set standards requires public health policy judgments in addition to determinations of a strictly scientific nature. While the Panel is willing to advise you further on the PM standard, we see no need, in view of the already extensive comments provided, to review any proposed PM standards prior to their publication in the Federal Register. In this instance, the public comment period will provide sufficient opportunity for the Panel to provide any additional comment or review that may be necessary.

Thank you for the opportunity to present the Panel's views on this important public health issue. We look forward to your response to the advice contained in this letter.

Sincerely,

George T. Wolff

Dr. George T. Wolff, Chair
Clean Air Scientific Advisory Committee

TABLE I
Summary of CASAC Panel Members Recommendations
(all units $\mu g/m^3$)

		$PM_{2.5}$ 24-hr	$PM_{2.5}$ Annual	PM_{10} 24-hr	PM_{10} Annual
Current NAAQS		N/A	N/A	150	50
EPA Staff Recommendation		18 - 65	12.5 - 20	150[13]	40 - 50
Name	**Discipline**				
Ayres	M.D.	yes[2]	yes[2]	150	50
Hopke	Atmos. Sci.	20 - 50[3]	20 - 30	no	40 -50[4]
Jacobson	Plant Biologist	yes[2]	yes[2]	150	50
Koutrakis	Atmos. Sci.	yes[2,5,6]	yes[2,5,6]	no	yes[4]
Larntz	Statistician	no	25-30[7]	no	yes[2]
Legge	Plant Biologist	≥ 75	no	150	40 - 50
Lippmann	Health Expert	20 - 50[3]	15 - 20	no	40 - 50
Mauderly	Toxicologist	50	20	150	50
McClellan	Toxicologist	no[8]	no[8]	150	50
Menzel	Toxicologist	no	no	150	50
Middleton	Atmos. Sci.	yes[2,3,12]	yes[2,5]	150[3,13]	50
Pierson	Atmos. Sci.	yes[2,9]	yes[2,9]	yes[4]	yes[4]
Price	Atmos. Sci./ State Official	yes[3,10]	yes[10]	no[3,4]	yes[4]
Shy	Epidemiologist	20 - 30	15 - 20	no	50
Samet[1]	Epidemiologist	yes[2,11]	no	150	yes[2]
Seigneur	Atmos. Sci.	yes[3,5]	no	150[13]	50
Speizer[1]	Epidemiologist	20 - 50	no	no	40 - 50
Stolwijk	Epidemiologist	75[7]	25-30[7]	150	50
Utell	M.D.	≥65	no	150	50
White	Atmos. Sci.	no	20	150	50
Wolff	Atmos. Sci.	≥75[3,7]	no	150[3]	50

[1] not present at meeting; recommendations based on written comments
[2] declined to select a value or range
[3] recommends a more robust 24-hr. form
[4] perfers a $PM_{10-2.5}$ standard rather than a PM_{10} standard
[5] concerned upper range is too low based on national $PM_{2.5}/PM_{10}$ ratio
[6] leans towards high end of Staff recommended range
[7] desires equivalent stringency as present PM_{10} standards

[8] if EPA decides a $PM_{2.5}$ NAAQS is required, the 24-hr. and annual standards should be 75 and 25 $\mu g/m^3$, respectively with a robust form

[9] yes, but decision not based on epidemiological studies

[10] low end of EPA's proposed range is inappropriate; desires levels selected to include areas for which there is broad public and technical agreement that they have $PM_{2.5}$ pollution problems

[11] only if EPA has confidence that reducing $PM_{2.5}$ will indeed reduce the components of particles responsible for their adverse effects

[12] concerned lower end of range is oo close to background

[13] the annual standard may be sufficient; 24-hr level recommended if 24-hour standard retained

NOTICE

This report has been written as part of the activities of the Science Advisory Board, a public advisory group providing extramural scientific information and advice to the Administrator and other officials of the Environmental Protection Agency. The Board is structured to provide balanced, expert assessment of scientific matters related to problems facing the Agency. This report has not been reviewed for approval by the Agency and, hence, the contents of this report do not necessarily represent the views and policies of the Environmental Protection Agency, nor of other agencies in the Executive Branch of the Federal government, nor does mention of trade names or commercial products constitute a recommendation for use

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TECHNICAL REPORT DATA

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1 REPORT NO	2	3 RECIPIENT'S ACCESSION NO
EPA-452/R-96-013		

4 TITLE AND SUBTITLE	5 REPORT DATE
Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information -- OAQPS Staff Paper	July 1996
	6 PERFORMING ORGANIZATION CODE
7. AUTHOR(S) Bachmann, J.D.; Caldwell, J.C.; Damberg, R.J.; Edwards, C.; Koman, T.; Martin, K.; Polkowsky, B.; Richmond, H.M.; Smith, E.; Woodruff, T.	8 PERFORMING ORGANIZATION REPORT NO
9. PERFORMING ORGANIZATION NAME AND ADDRESS Office of Air and Radiation Office of Air Quality Planning and Standards U.S. Environmental Protection Agency Research Triangle Park, North Carolina 27711	10 PROGRAM ELEMENT NO
	11. CONTRACT/GRANT NO.
12. SPONSORING AGENCY NAME AND ADDRESS	13 TYPE OF REPORT AND PERIOD COVERED Final
	14 SPONSORING AGENCY CODE

15. SUPPLEMENTARY NOTES

16. ABSTRACT

This staff paper evaluates and interprets the updated scientific and technical information that EPA staff believes is most relevant to the review of primary and secondary national ambient air quality standards for particulate matter (PM). This assessment is intended to bridge the gap between the scientific review in the 1996 criteria document and the judgements required of the Administrator in setting ambient air quality standards for PM. The major staff recommendations presented in the staff paper for consideration by the Administrator include: (1) the current PM standards should be revised in light of evidence showing effects in areas that attain current NAAQS; (2) PM_{10} remains an appropriate indicator, but the fine ($PM_{2.5}$)and coarse fractions of PM_{10} should be regulated separately; (3) two $PM_{2.5}$ standards should be established: a 24-hour standard with a more robust form and a level selected from a range of 20-65 $\mu g/m^3$, and an annual expected mean standard selected from a range of 12.5-20 $\mu g/m^3$; (4) consideration should be given to the use of spatial averaging across multiple monitors for $PM_{2.5}$ standards; (5) an annual PM_{10} standard should be retained for control of coarse fraction particles, alone or in combination with a 24-hour PM_{10} standard; (6) the level of the annual standard should be selected from a range of 40-50 $\mu g/m^3$; if a 24-hour standard is retained, the level should remain at 150 $\mu g/m^3$, but with a more robust form; and, (7) secondary standards for PM should be set equal to the primary standards to address soiling and nuisance; consideration should be given to addressing remaining visibility impairment issues through regional haze regulations.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS		b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
Particulate Matter	Mortality	Air Quality Standards	
PM	Morbidity		
Air Pollution	Exposure Assessment		
Health Effects	Risk Assessment		
Welfare Effects			

18 DISTRIBUTION STATEMENT	19 SECURITY CLASS *(Report)*	21 NO OF PAGES
	20 SECURITY CLASS *(Page)*	22 PRICE

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