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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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, April 1995 External Review Draft; 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 primary standards 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 ...."

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 μm (so-called total suspended particulate or TSP). The primary standards (measured by the indicator TSP) were 260 $\mu g/m^3$, 24-hour average, not to be exceeded more than once per year, and 75 $\mu g/m^3$, annual geometric mean. The secondary standard (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 10 microns.[3] EPA also

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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).

[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

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 on February 15, 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 the review and any revision of the PM NAAQS by January 31, 1997, with a proposed decision on the NAAQS required by June 30, 1996 (*American Lung Association v. Browner*, CIV-93-643-TUC-ACM (D. Ariz., October 6, 1994)).

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, and identified key issues to be addressed in this Staff Paper and 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

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suspended particulate matter of aerodynamic size less than or equal to 10 μm.

EPA's National Center for Environmental Assessment (NCEA) hosted three public peer-review workshops on drafts of key chapters of a revised CD. The NCEA subsequently released a complete draft of the revised CD for CASAC and public review, and CASAC reviewed the draft at a public meeting held on August 3-4, 1995. The NCEA is currently revising the draft CD in response to CASAC and public comments.

III. APPROACH

This Staff Paper is based on the scientific evidence reviewed in the revised CD and takes into account CASAC and public comments on that document to the extent possible. The staff has also considered comparative air quality analyses and exposure-response assessments in evaluating the appropriateness of revising the current primary NAAQS and in assessing potential alternative NAAQS. Technical and economic analyses examining visibility impairment, soiling and materials damage, and possible ecosystem effects have also been considered in evaluating the appropriateness of 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

To facilitate meeting the court-ordered schedule, the staff identified several possible policy alternatives to provide a basis for commencing initial analytical assessments of air quality, human exposure, and health risks. In so doing, the staff recognized that additional alternatives might need to be analyzed as the review process continued (e.g., as a result of CASAC and public review of drafts of the CD and this Staff Paper).

1. Primary Standards

Many health scientists have expressed the opinion that observed epidemiological associations between PM_{10} and health effects may actually be primarily the result of exposures to the fine particle fraction of PM_{10} (particles that are approximately an order of magnitude smaller than 10 μm in aerodynamic diameter and generated largely from combustion processes). 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 provides a summary of differences among the various fractions of PM_{10}, relevant physical and chemical properties, air quality status and trends for both PM_{10} and fine particles, and characterizations of average "background" concentrations.

Chapter V discusses biological mechanisms of toxicity, sensitive subpopulations, the nature of health effects associated with PM, evaluations of the evidence, and concentration-response information. Chapter V also presents staff judgments concerning which effects are

important for the Administrator to consider in selecting appropriate primary standards and uncertainties surrounding the specific agents of concern.

Drawing on these factors and on information contained in the previous chapters, Chapter VI presents staff conclusions and recommendations for the Administrator to consider in reaching a decision on revision of the primary NAAQS. The chapter addresses alternative averaging times, forms, pollutant indicators, and levels, with a summary section that presents the staff's overall recommendations for a suite of primary standards.

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

IV. AIR QUALITY CONSIDERATIONS

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 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.

Examining air quality information is important to understanding possible human exposures and potential impacts on health and welfare. This chapter briefly discusses the chemical and physical properties of PM in the atmosphere and presents historical and current PM_{10} and $PM_{2.5}$ levels.

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[1]In this staff paper, particle size or diameter refers to aerodynamic diameter. 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.

A. Multi-modal Size Distribution: Fine and Coarse Fractions of PM

The health and environmental effects of PM and the fate of the components are strongly related to the size of the particles. The aerodynamic size of particles plays a major role in determining the extent to which they are able to penetrate into the respiratory tract and how they behave once inside (e.g., how far the particles are able to penetrate, the extent to which the body's clearance mechanisms are effective in removing the particles, and where they are deposited as discussed in Chapter V). Furthermore, size is one of the most important parameters in determining atmospheric lifetime and visibility impact of particles. Atmospheric lifetime is also important to health effects because of its relationship to exposure. Differences in surface area, number of particles, chemical composition, water solubility, formation process, and emission sources also vary with particle size. Thus, size is an important parameter in characterizing PM, and particle diameter has been used to define the present standard.

The multi-modal distribution of particles based on diameter has long been recognized (Whitby, Husar, and Liu, 1972; Whitby et al., 1975; Willeke and Whitby, 1975; National Research Council, 1977; U.S. EPA, 1982a; U.S. EPA, 1982b; U.S. EPA, 1986b; CD Chapter 3). Particles can be divided into fine and coarse modes based on particle size and formation mechanism.

Although particles display a consistent multi-modal distribution over several physical metrics such as surface area and mass, the specific distributions may vary over place, conditions, and time because of different sources, atmospheric conditions, and topography. Figure IV-1 illustrates an idealized mass distribution of the two distinct modes related to particle size: fine and coarse particles. Fine particles can be further classified as nuclei, ultrafine, and accumulation modes. The fine mode also accounts for most of the surface area and number of particles, as shown in Figure IV-2.

The CD concludes that an appropriate cut point 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 Chapter 3; Miller et al., 1979). However, the CD states that the data do not provide a clear or obvious choice of cut point and that some overlap occurs

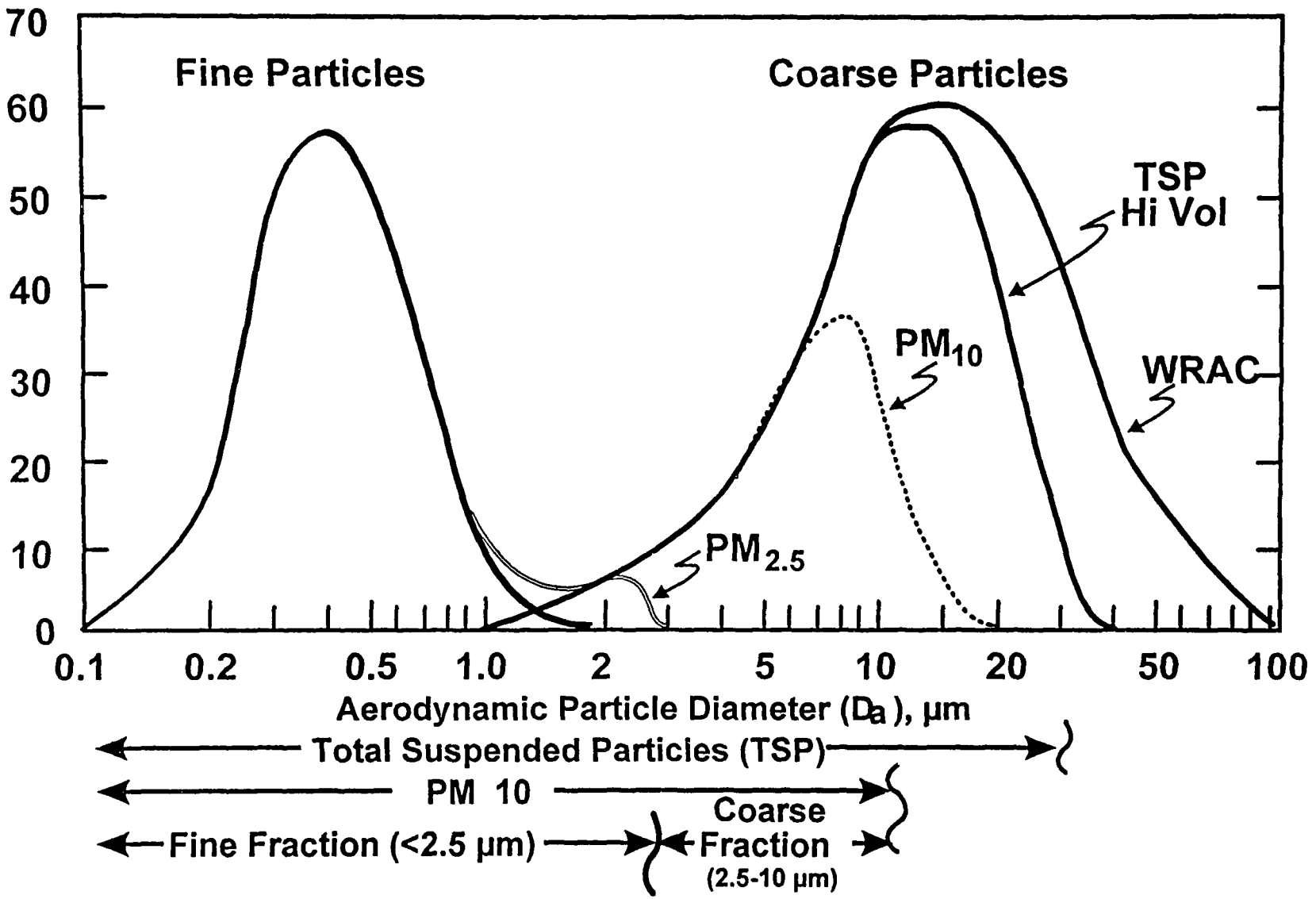


Figure IV-1. Idealized Fine and Coarse Mode Particle Mass

The dotted lines represent the mass captured by different monitors. For example, PM10 mass would be proportional to the portion of the coarse mode under the curve (the coarse fraction) and all of the fine fraction.

Figure IV-2. Multi-modal Distribution of Particulate Matter

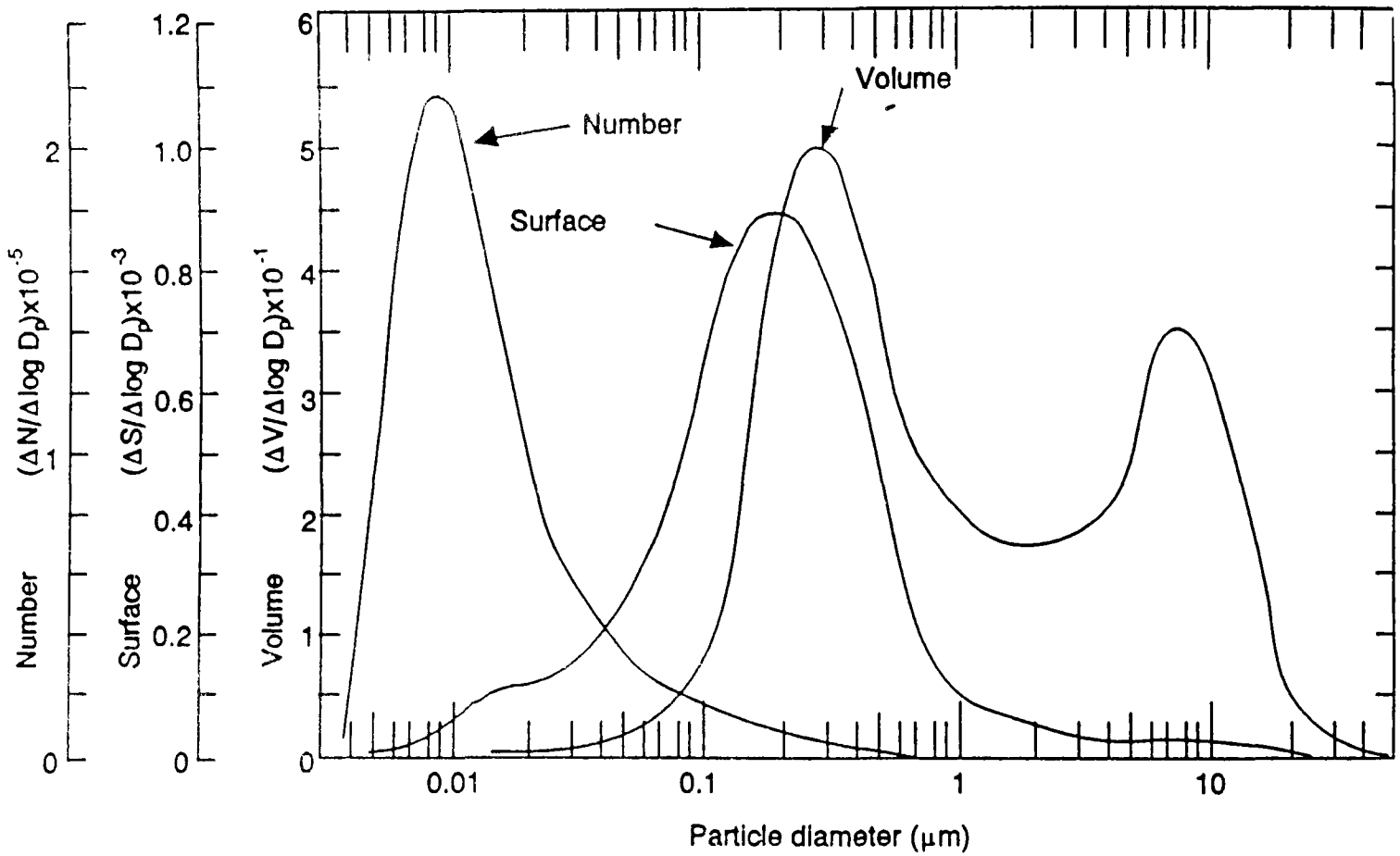


Figure IV-2. These normalized plots of number, surface and volume (mass) distributions from Whitby (1975) show a bimodal mass distribution in a smog aerosol. Historically, such particle size plots were described as consisting of a coarse mode (2.5 to 15 μm), a fine mode (0.1 to 2.5 μm), and a nuclei mode ($<$ 0.05μm). The nuclei mode would currently fall within the ultrafine particle range (0.005 to 0.1 μm).

between the modes. Appendix A outlines the policy considerations in choice of 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, $PM_{2.5}$ is used to refer to gravimetric measurements with a 2.5 μm cut point while fine particles will be used more generally to refer to the fine mode. Because of the possible overlap of fine and coarse modes in the intermodal region (i.e., 1 - 3 μm), $PM_{2.5}$ is only an approximation for fine particles. Moreover, monitor design, measurement temperature, and inlet efficiency can also affect which particles are included in the definitions of the various size fractions. Sampling method may also affect the amount of semivolatile organics and nitrates and particle-bound water included in the measurement. In addition to $PM_{2.5}$ mass measurements, fine particles have been measured in the U.S. and abroad using a variety of techniques resulting in a variety of indicators including British Smoke (BS), Coefficient of Haze (COH), carbonaceous material (KM), and estimates from visibility measurements. 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 carbonaceous particles found in the fine fraction. 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 is 4.5 μm (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. In the U.S., elemental carbon is found predominantly in the fine mass ($<$ 1.0 μm range) (NAS, 1980; U.S. EPA, 1982b).

Using a similar principle to BS, the principle of COH measurement 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 and the amount of light transmitted is

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

Fraction	Description
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
$PM_{2.5}$	Particles with an upper 50 percent cut point of aerodynamic diameters[1] less than 2.5 μm, a measurable approximation for fine particles[2]
Coarse particles, generally	Coarse mode particles which are mostly mechanically generated through crushing or grinding
Coarse fraction of PM_{10}	Particles with an upper 50 percent cut point of aerodynamic diameters between 2.5 μm and 10 μm
PM_{10}	Particles with an upper 50 percent cut point of aerodynamic diameters less than 10 μm, including fine fractions and part of the general coarse mode
Total Suspended Particles (TSP)	Particles with an upper 50 percent cut point of aerodynamic diameters less than approximately 50 μm; highly wind speed dependent

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

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 Ozkaynak, 1990). Similar to BS, KM is also a fine particle measurement.

B. Fine and Coarse Fractions' Distinct Properties

In addition to being distinguishable based on size considerations, fine and coarse particles have many other distinct properties, as summarized in Table IV-2. Fine and coarse particles can be differentiated by their chemical composition, solubility, acidity, sources and formation processes, atmospheric lifetime, infiltration indoors, and transport distances. Furthermore, fine and coarse particle mass concentrations generally are poorly correlated (Wilson et al., 1995; SAI, 1995).

1. Chemical Composition, Solubility, and Acidity

Figure IV-3 shows the synthesis of the available published data on the chemical composition in U.S. cities broken down by region between $PM_{2.5}$ and coarse fraction particles which together comprise PM_{10} (CD, Chapter 6). Sulfates and organic carbon dominate fine particles (together accounting for between 50 to over 90 percent of fine mass); whereas, minerals dominate the coarse fraction of PM_{10} (ranging from over 50 percent to 70 percent of coarse fraction mass) (CD, Chapter 6). Differences across the country in sources and atmospheric conditions contributes to this variability.

In general, fine and coarse particles have different solubility and acidity. With the exception of carbon and some organic compounds, fine particles are largely soluble in water and hygroscopic (i.e., they readily take up and retain water), although some are deliquescent (i.e., they remain crystalline until a certain relative humidity is exceeded at which point they become hygroscopic). The fine particle mode also contains most of the acid particles (CD,

TABLE IV-2. COMPARISON OF FINE VERSUS COARSE MODE PARTICLES

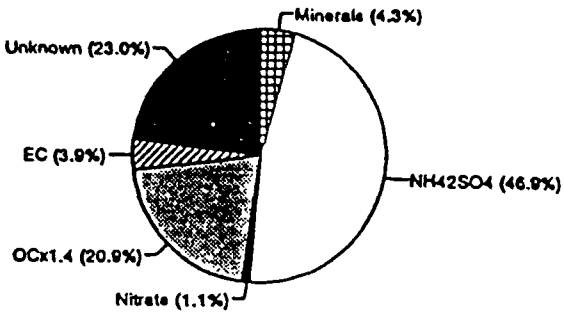
	Fine Mode	Coarse Mode
Formed from:	Gases	Large solids/droplets
Formed by:	Chemical reaction; Nucleation, Condensation, Coagulation. Evaporation of fog and cloud droplets in which gases have dissolved and reacted.	Mechanical disruption (crushing, grinding etc.), Evaporation of sprays, Suspension of dusts.
Composed of:	Sulfate, $SO_4^=$; Nitrate, NO_3^-; Ammonium, NH_4^+; Hydrogen ion, H^+; Elemental carbon, C; Organic compounds; PNA; Metals, Pb, Cd, V, Ni, Cu, Zn; Particle-bound water; Biogenic organics.	Resuspended dusts, Soil dust, street dust. Coal and oil fly ash. Metal oxides of Si, Al, Mg, Ti, Fe. $CaCO_3$, NaCl, sea salt, Pollen, mold spores, Plant parts.
Solubility:	Significant fraction soluble, hygroscopic and deliquescent; some portions insoluble.	Largely insoluble and non-hygroscopic.
Sources:	Combustion of coal, oil, gasoline, diesel, wood. Atmospheric transformation products of NO_x, SO_2, and organics including biogenic organics, e.g., terpenes. High temperature processes, smelters, steel mills, etc.	Resuspension of soil tracked onto roads and streets. Suspension from disturbed soil, e.g., farming, mining. Resuspension of industrial dusts. Construction, 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: Wilson et al., 1995; CD Chapter 3

Figure IV-3. Chemical Composition of $PM_{2.5}$, Coarse Fraction, and PM_{10} by Region

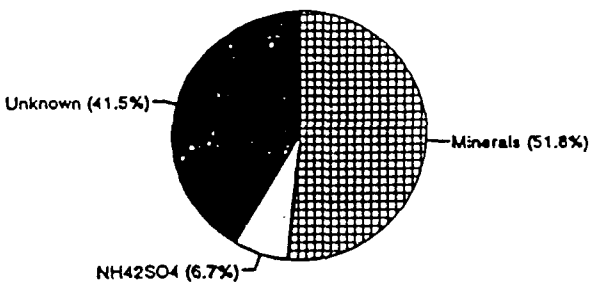
EASTERN U.S. URBAN AREAS

$PM_{2.5}$



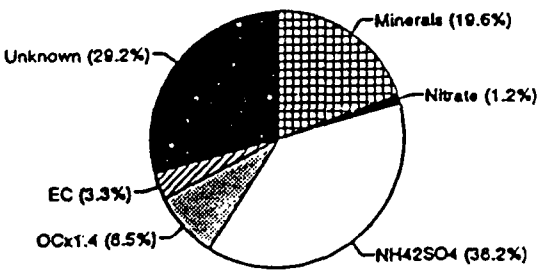
Nitrate based on 3 studies.

Coarse Fraction



Insufficient Nitrate, OC, and EC data available.

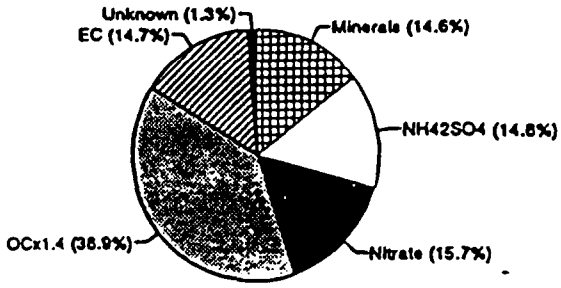
PM_{10}



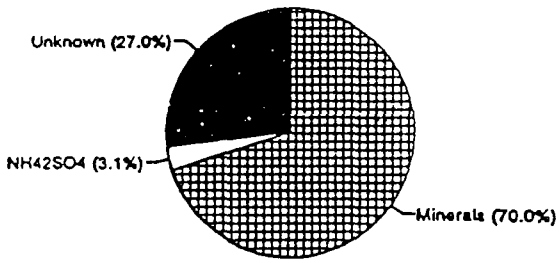
Nitrate based on 2 studies.

WESTERN U.S. URBAN AREAS

$PM_{2.5}$

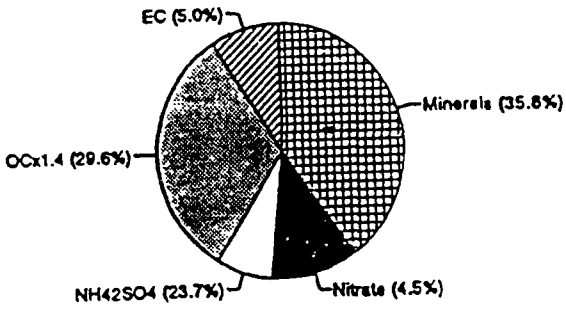


Coarse Fraction



Insufficient Nitrate, OC, and EC data available.

PM_{10}



Reconstructed sum = 106%

Chapter 3). By contrast, coarse particles are mostly insoluble, non-hygroscopic, and generally basic (CD, Chapter 3).

Fine particles also display regional differences in chemical composition. For example, fine particles in the Eastern U.S. are comprised of relatively more sulfate (47 percent) than the west (15 percent). Many western sites have a lower sulfate and a larger nitrate contribution. Nitrates contribute 1 percent to the Eastern U.S. mass and 15 percent to Western U.S. mass for fine particles. Western U.S. urban areas have twice the proportion of organic carbon compounds as found in Eastern U.S. urban areas.

The proportions presented in this figure do not show relative concentrations; however, the relative concentrations become apparent when the fine and coarse fractions are aggregated into the PM_{10} figures. (Concentrations for selected cities and time periods studied for chemical composition are also presented in Appendix B tables; national levels are presented below.) Concentrations of coarse fraction particles are generally higher in the Western and Southwestern U.S. than in the Eastern U.S. PM_{10} is a composite of fine and coarse particles with sulfates dominating the eastern U.S. and minerals being more important in the Western U.S. One of the primary reasons for the heterogeneity of PM_{10} composition is the fundamental underlying differences between fine and coarse particles. As presented in the CD, Appendix B portrays the available published data on chemical constituents of PM_{10}, fine particles, and coarse particles, focusing on cities in which health studies were conducted. Other than noting that mineral and soil compounds tend to dominate the coarse fraction, no discernable patterns in chemical composition of the $PM_{2.5}$ could be determined based on this limited database that used several different analytical techniques.

2. Sources and Formation Processes

Fine and coarse particles generally have distinct sources and formation mechanisms although there may be some relatively small overlap. Fine particles are usually formed from gases in two ways: (1) nucleation (i.e., formation of particles from low vapor pressure substances, produced either from combustion or from chemical reaction of gases) and (2) condensation of gases onto existing particles. Particles formed from nucleation also coagulate to form relatively larger particles, although these particles normally do not grow

above 1.0 μm in aerodynamic diameter by these processes (CD, Chapter 3). Particles formed as a result of chemical reaction of gases are termed secondary particles because the direct emission from a source is a gas (e.g., SO_2 or NO) that is subsequently converted to a low vapor pressure substance in the atmosphere. Examples of fine particles include species such as sulfates, organics, and ammonium. Transformation from gases to particles requires substantial interaction in the atmosphere. Such transformation can take place locally, during prolonged stagnations, or during transport over long distances. Moisture, sunlight, temperature, and the presence or absence of fogs and clouds affect transformation. In general, particles formed from these types of secondary processes will be more uniform in space and time than those that result from primary emissions. Although primary particles are also found in the fine fractions (the most common being particles less than 1.0 μm in aerodynamic diameter from combustion sources), secondary particles are predominately found in the fine range, which is one of the reasons fine particles are more uniform in space and time than coarse particles.

By contrast, most of the coarse fraction particles are emitted directly as particles resulting from mechanical disruption such as crushing, grinding, evaporation of sprays, suspensions of dust from construction and agricultural operations. Simply put, coarse particles are formed by breaking up bigger particles into smaller ones. Some combustion-generated particles such as fly ash and soot are also found in the coarse fraction.

3. Atmospheric Lifetime, Transport, and Infiltration Indoors

Larger particles deposit more rapidly than small particles, affecting transport, infiltration of particles formed outdoors into homes and buildings where people spend most of their time, and thus are exposed to PM. Measurements of indoor PM levels are especially important because people on average spend 21 hours each day indoors and thus a large amount of their exposure to PM may occur inside (CD, Chapter 7; Robinson and Nelson, 1995).

Every particle attains an equilibrium between gravity and atmospheric resistance at its terminal settling velocity.[2] Compared to other sizes, fine particles with aerodynamic diameters between 0.1 and 1.0 μm have the minimum terminal settling velocity of particles. Figure IV-4 shows that fine particles will remain suspended for much longer times (on the order of days to weeks for fine particles as opposed to minutes to hours for coarse particles) and will travel much farther (i.e., hundreds to thousands of kilometers) than coarse fraction particles (i.e., kilometers to tens of kilometers) (CD Chapter 3; Watson, Rogers, and Chow, 1995). Therefore, fine particles have generally longer atmospheric lifetimes than coarse particles.

Fine particles originating outdoors infiltrate into homes and buildings to a greater degree than coarse particles (CD, Chapter 7; Lioy et al., 1990). Figure IV-5 presents the relative contribution of indoor particles by source category. Approximately two thirds of indoors PM_{10} concentrations originated outdoors. Even more of the fine particles proportionately are of outdoor origin, about three quarters. Anuszewski et al. (1992) show that light scattering particles measured by nephelometry have very high correlation between indoor and outdoor concentrations ($R^2 = 0.9$). Spengler et al. (1981) found that for the Harvard Six City study, long-term mean infiltration of outdoor-origin $PM_{3.5}$ was 70 percent for homes without air conditioning and 30 percent in homes with air conditioning. (CD Chapter 7). Koutrakis et al. (1992) using New York State data of homes without smoking or fireplaces found that 60 percent of the $PM_{2.5}$ mass was from outdoor sources (CD Chapter 7). Thus, ambient particles penetrate indoors and are available to be breathed into the lungs.

Specific constituents of fine particles penetrate well indoors as well. Suh et al. (1994) also provide evidence that ambient fine particles penetrate indoors in State College, PA. The correlations between personal[3] and outdoor sulfate measurements were high

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[2]The settling velocity increases as the square of the particle diameter or when the particle density increases. For small particles, vertical air movements caused by turbulence can counteract the gravitational settling velocity and such particles can remain suspended for days (Willeke and Whitby 1975).

[3]Personal exposure monitoring is usually accomplished by the subject carrying a portable device as the subject goes about routine daily activities.

Figure IV-4. Atmospheric Lifetime of Particles Based on Size

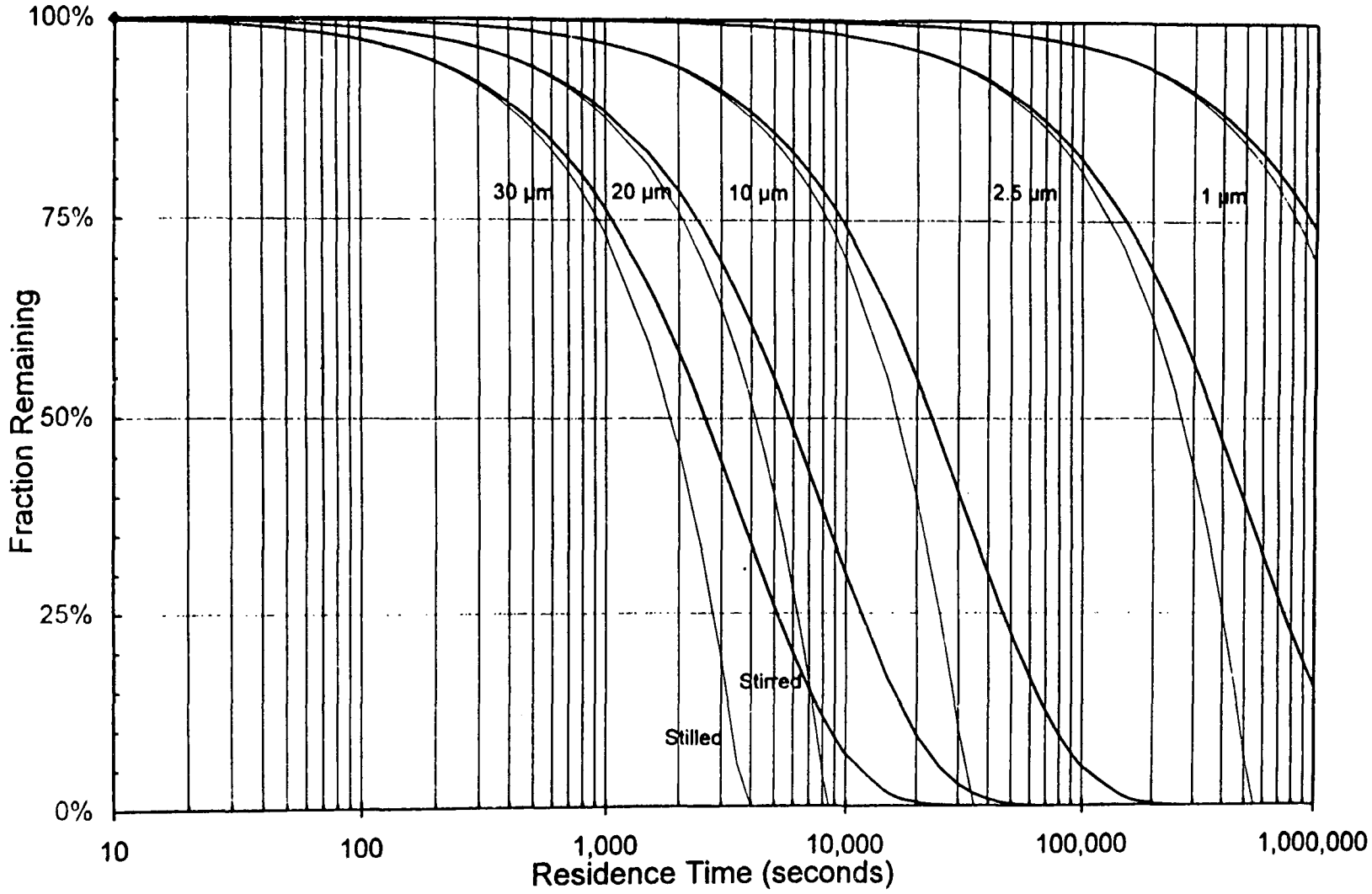
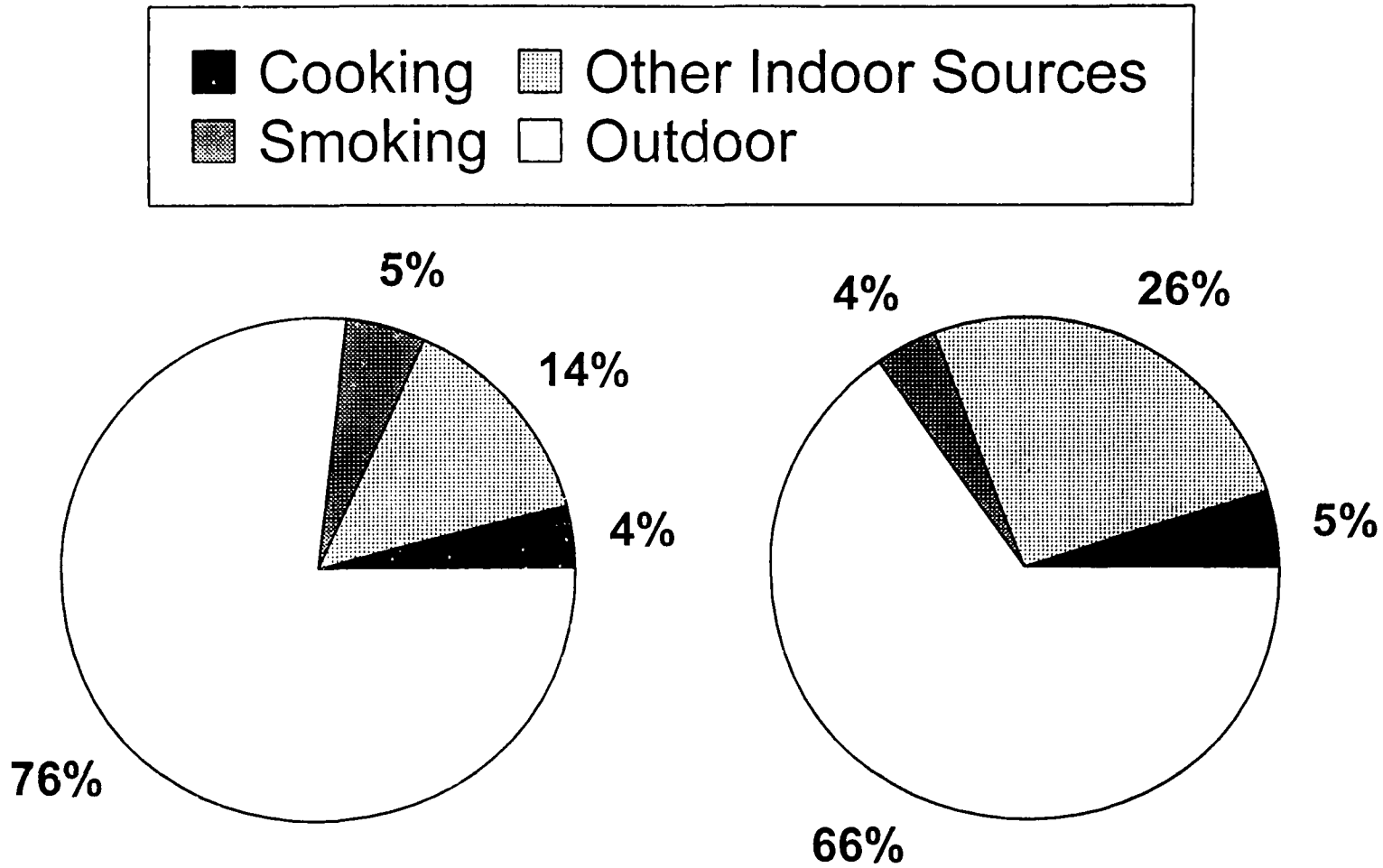


Figure IV-4. Aging times for homogeneously distributed particles of different aerodynamic ciameters in a 100 meter deep mixed layer. Gravitational settling is assumed for bothe still and stirred chamber models (Watson, Rogers, and Chow 1995).

Figure IV-5. Sources of PM2.5 and PM10 in Homes



PM2.5
N= 352 samples

PM10
N= 350 samples

Source: PTEAM Study, Riverside, CA

(R^2 = 0.92) despite the negligible indoor sources of sulfur. The correlation between personal and ambient acidity (H^+) was lower (R^2 = 0.38 and R^2 = 0.63 when corrected for personal ammonia), but still meaningful given that acidity is virtually all of outdoor origin.

4. Correlations between $PM_{2.5}$ and Coarse Fraction

Daily monitored concentrations of $PM_{2.5}$ correlate poorly with daily concentrations of the coarse fraction as shown in the scatter plot in Figure IV-6a using over 11,000 data points from locations across the U.S. The estimated R-squared statistic is close to zero (R^2 = 0.08), indicating that almost no linear relationship exists between daily averages of $PM_{2.5}$ and the coarse fraction. For the annual average concentration shown in Figure IV-6b, where one would expect to see a higher correlation because the averaging smooths some of the day-to-day variability, there is still almost no correlation between annual $PM_{2.5}$ and coarse fraction as shown by the low R-squared statistic (R^2 = 0.11). Note that in some specific instances, fine and coarse fractions may be correlated (e.g., driving a vehicle down a dusty road would produce fine particle emissions from the exhaust and coarse emissions from the road dust). However, overall the fine and coarse fractions are poorly correlated.

In summary, the fine and coarse fractions of PM_{10} are distinct entities with differing chemical composition, sources and formation processes, atmospheric lifetimes, infiltration indoors, and transport distances. $PM_{2.5}$ and coarse fraction mass concentrations are generally poorly correlated.

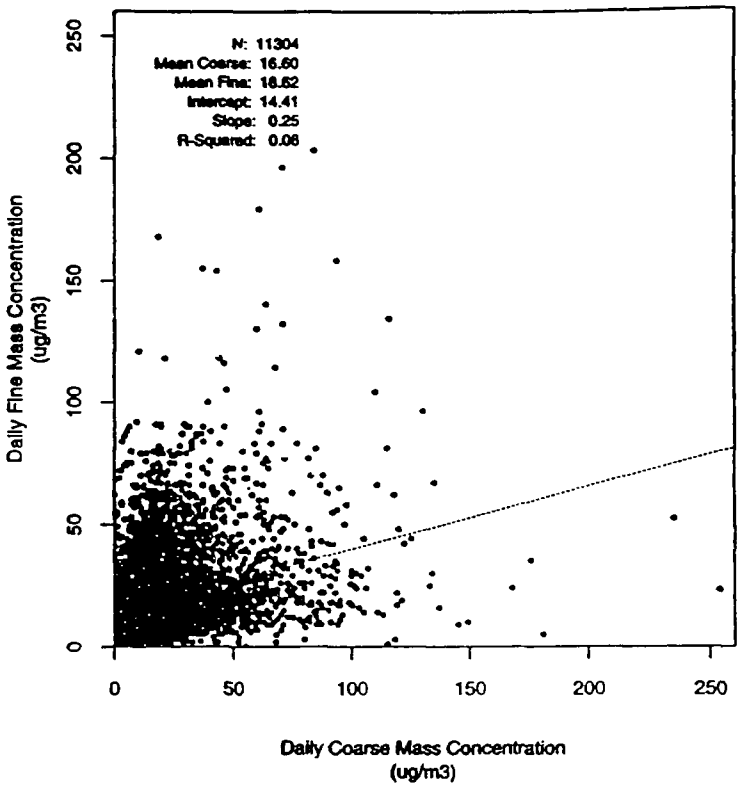
C. Trends of U.S. PM Levels

States and local air pollution control agencies have been collecting PM_{10} data using EPA-approved reference samplers and reporting these data to EPA's publicly available Aerometric Information Retrieval System (AIRS) since mid-1987. Trends may readily be examined for the 6-year period from 1988 to 1993 as illustrated in Figures IV-7 and IV-8. 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, respectively. The ninetieth

Figure IV-6. Scatter Plot of $PM_{2.5}$ and Coarse Fraction Concentrations

6a. Daily $PM_{2.5}$ v. Coarse Fraction Concentrations



6b. Annual Average $PM_{2.5}$ v. Coarse Fraction Concentrations

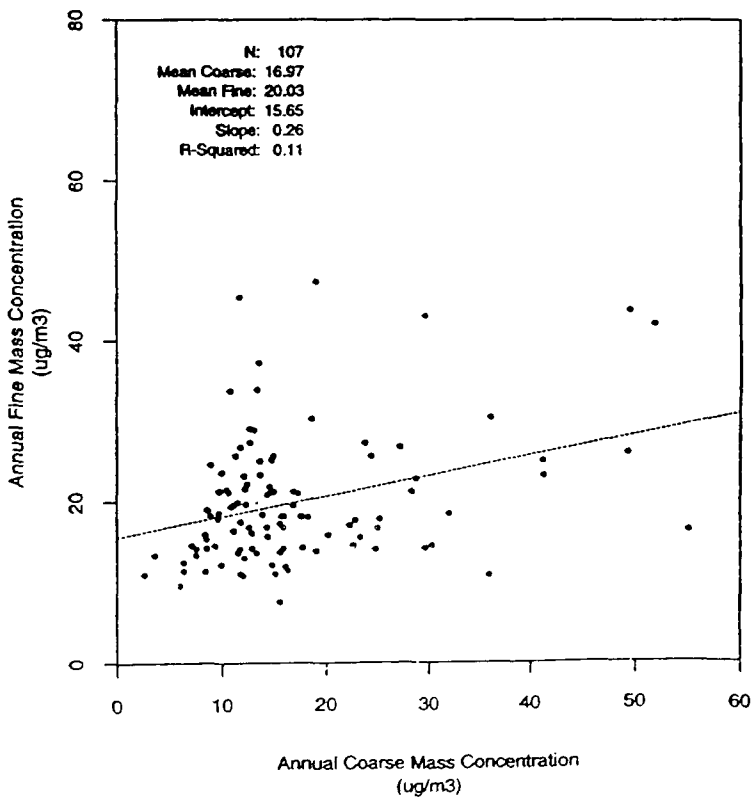


Figure IV-7. **PM-10 TREND, 1988-1993**
(90th PERCENTILE)

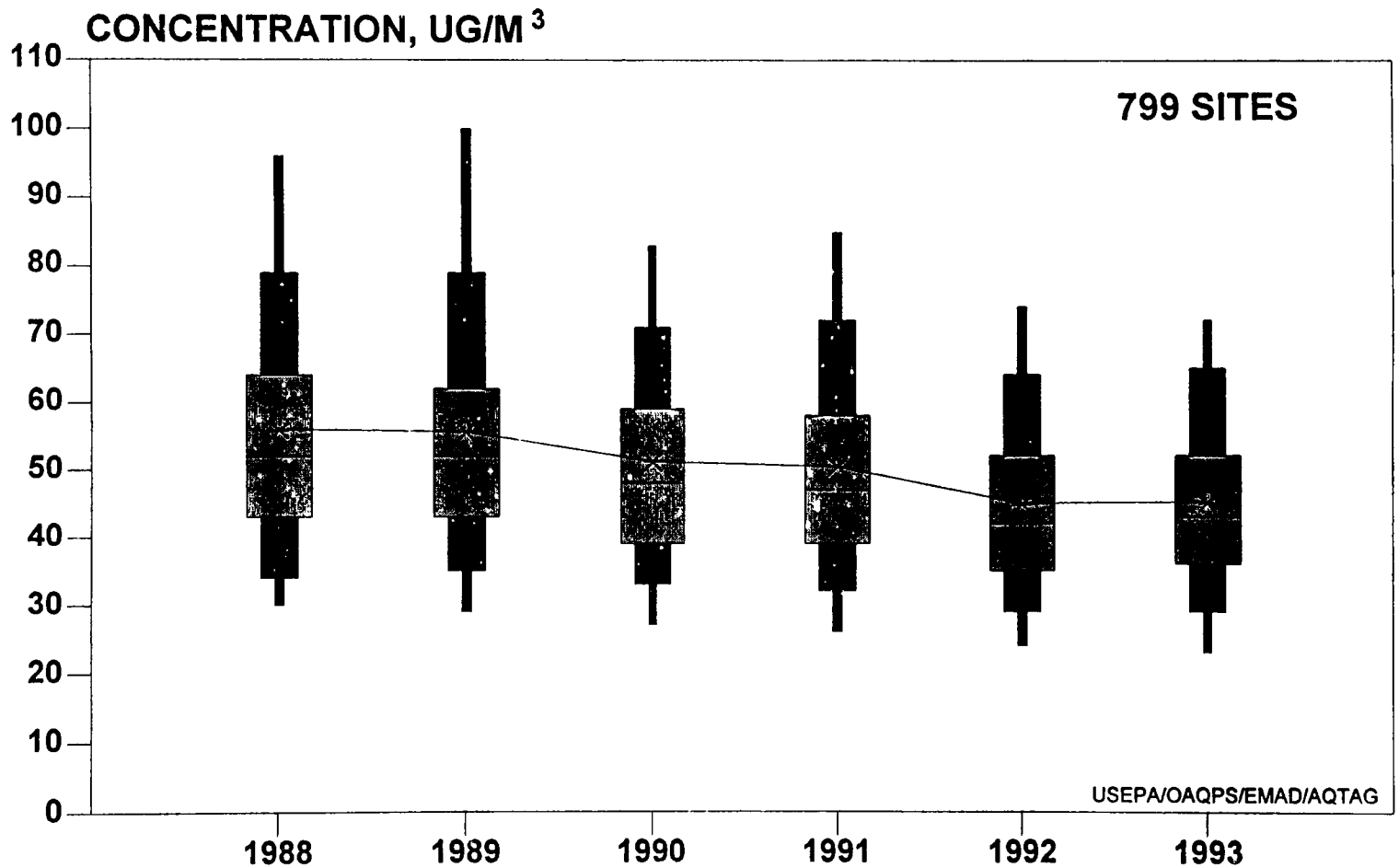
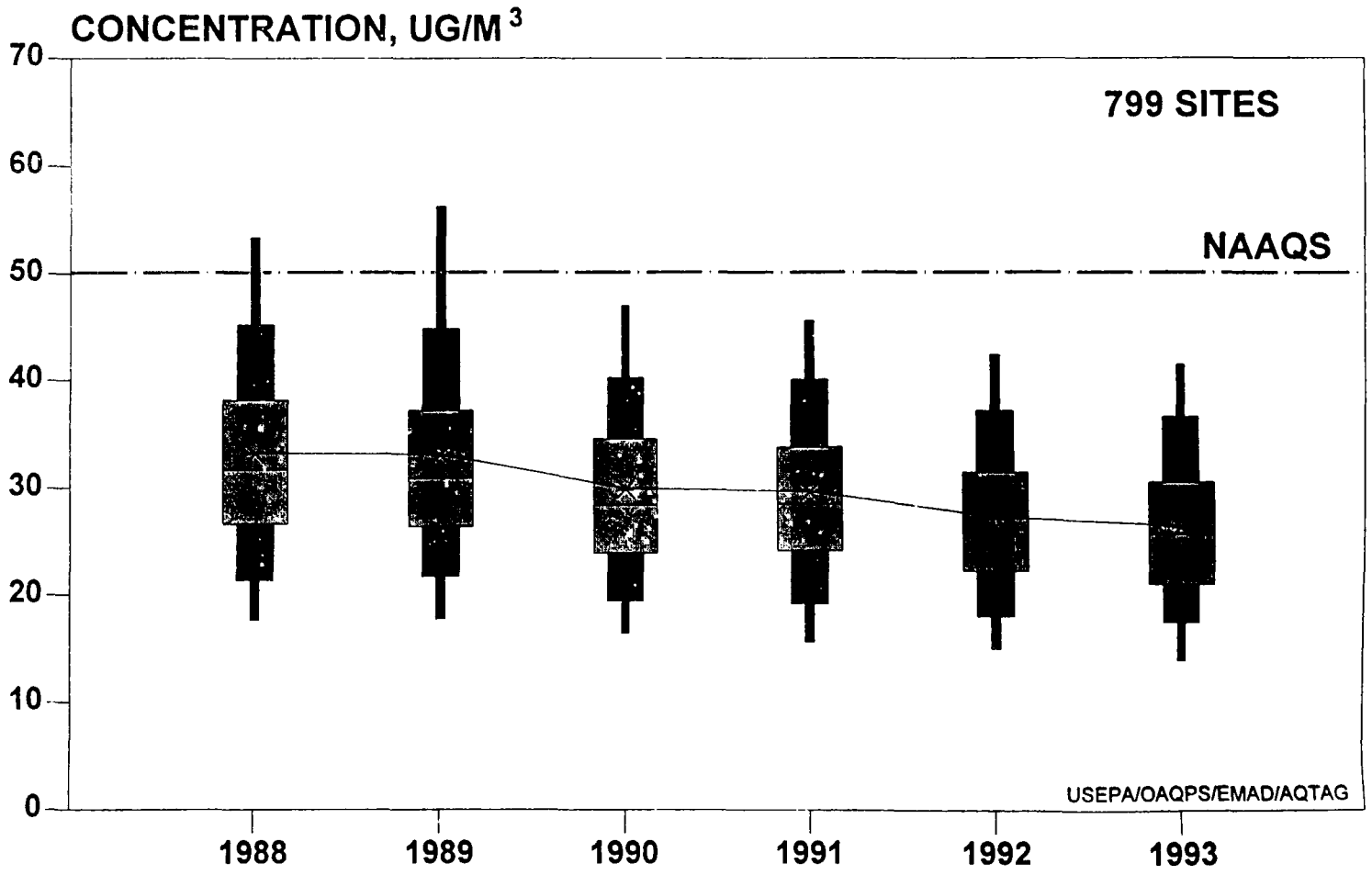


Figure IV-8. **PM-10 TREND, 1988-1993 (ANNUAL ARITHMETIC MEAN)**



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. 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, 1993).

In 1990 EPA designated 70 areas nonattainment for PM_{10}, and 13 additional areas were added in 1994 for a total of 83 PM_{10} nonattainment areas. Based on air quality data for 1992 to 1994, 37 of these are eligible for redesignation to attainment. Figure IV-9 shows the areas in nonattainment as of September 1994, also indicating the prevalent contributing sources and size of population residing in nonattainment areas.

The data for $PM_{2.5}$ concentrations are more limited than the PM_{10} data. Figure IV-10 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. Appendix C summarizes the databases which were assembled by EPA to support the air quality analyses in this Staff Paper. Fine particle trends are not available because the number of sites measuring $PM_{2.5}$ is small compared to the PM_{10} database and the sampling period is restricted to a few years. Furthermore, $PM_{2.5}$ is measured using non-standard sampling frequencies and non-standard sampling equipment. Thus, data are not sufficient to produce fine particle trends.

D. Current U.S. PM Levels

Current U.S. PM_{10} levels are illustrated in Figures IV-11 and IV-12. Figure IV-11 shows the annual mean PM_{10} concentration, and Figure IV-12 depicts the second highest 24-hour PM_{10} concentration in each county for which data were available and data completeness criteria were met. Each figure shows a snapshot of measured air quality data from 1993 for

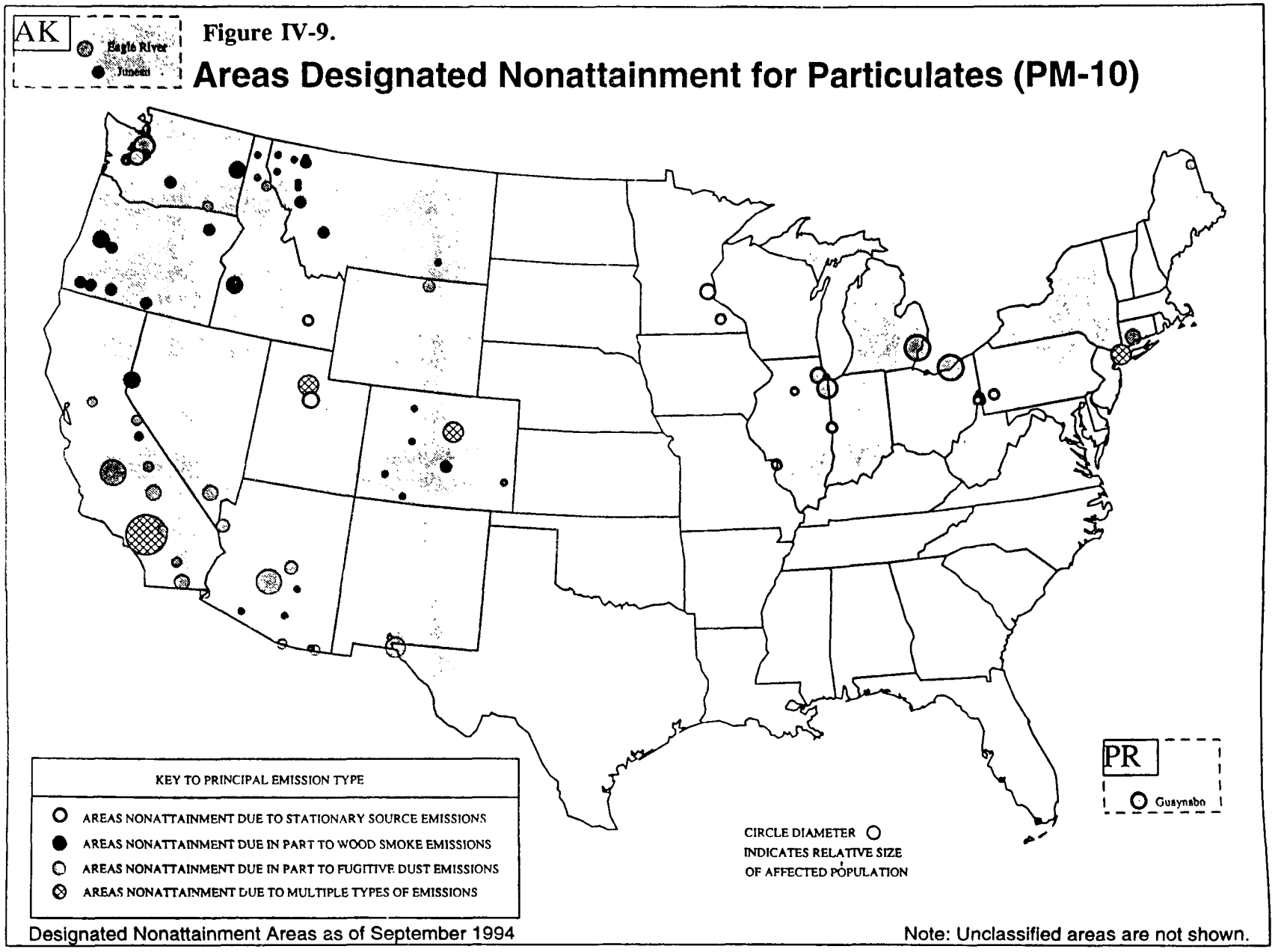


Figure IV-9.
Areas Designated Nonattainment for Particulates (PM-10)

Figure IV-10. $PM_{2.5}$ AIRS Data Summary, 1983-1993

Figure IV-10a. Geographic Distribution of Sites

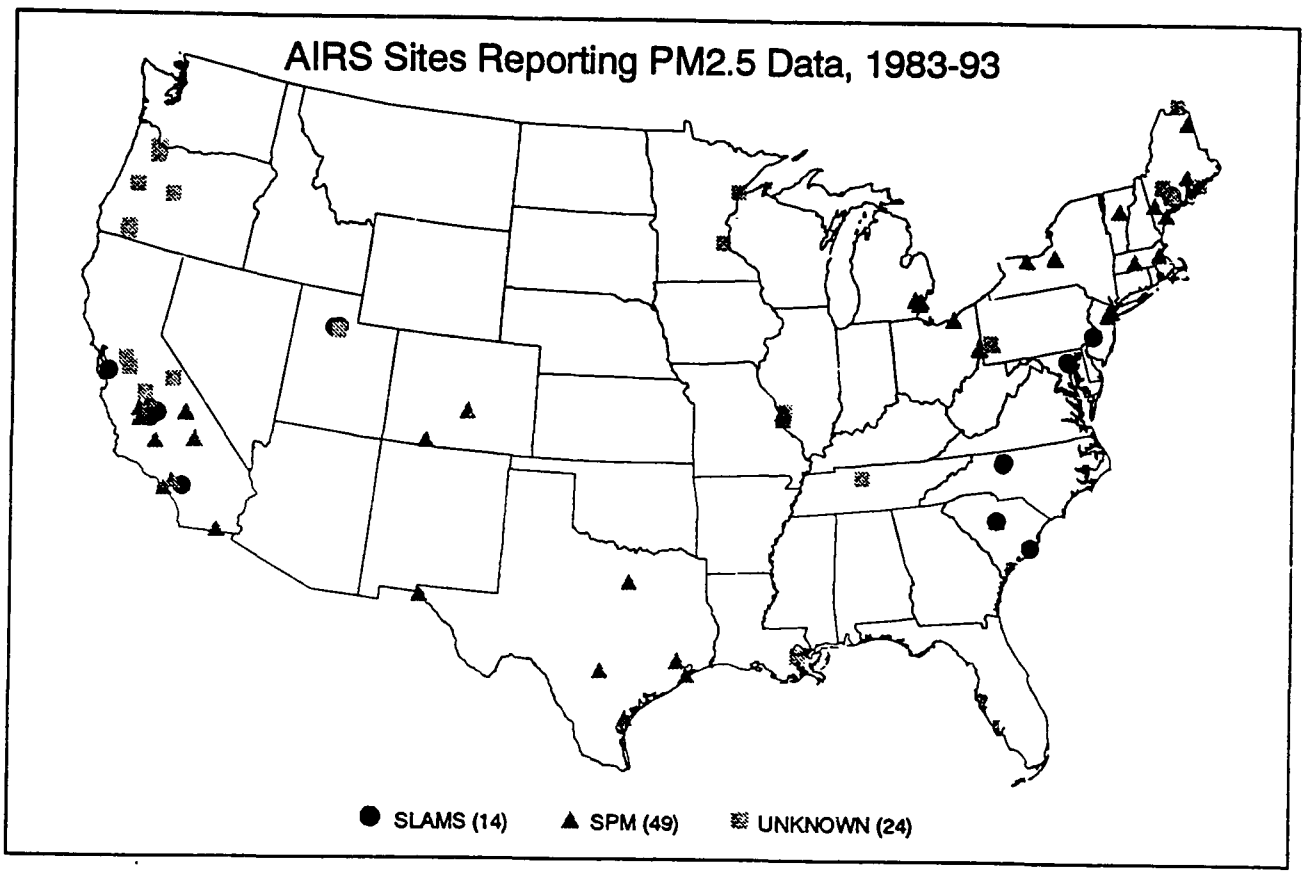


Figure IV-10b. Number of Sites and Frequency of Sampling

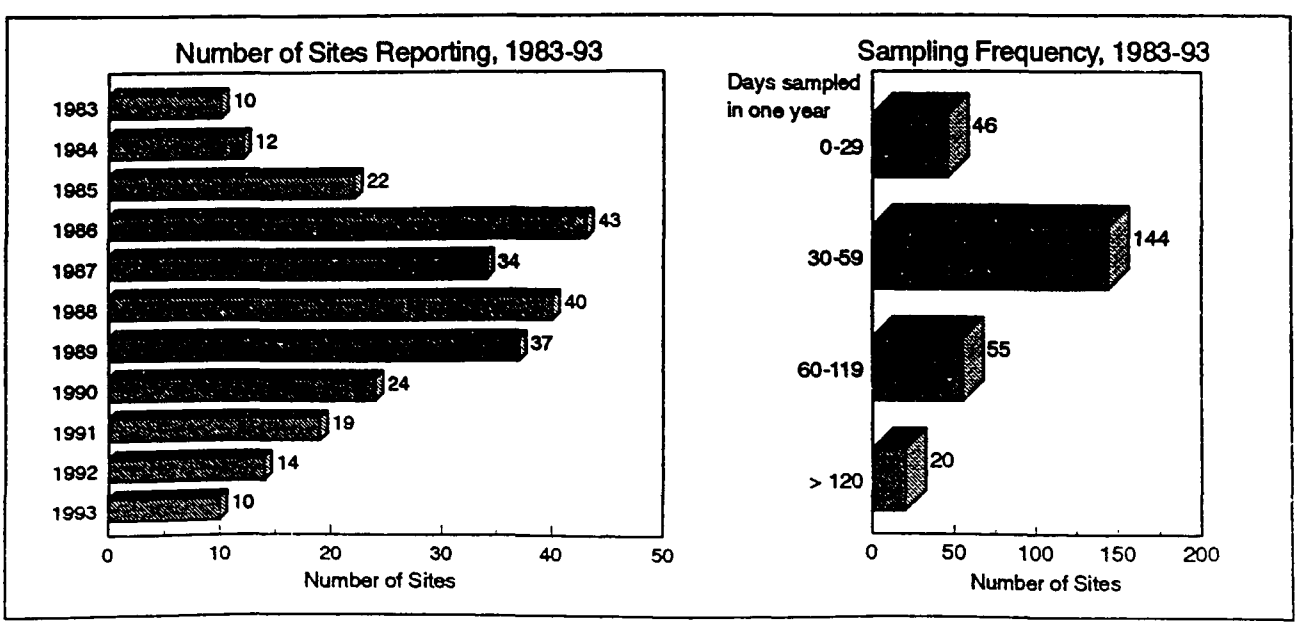


Figure IV-11. **PM-10 Air Quality Concentrations, 1993**
Highest Annual Mean

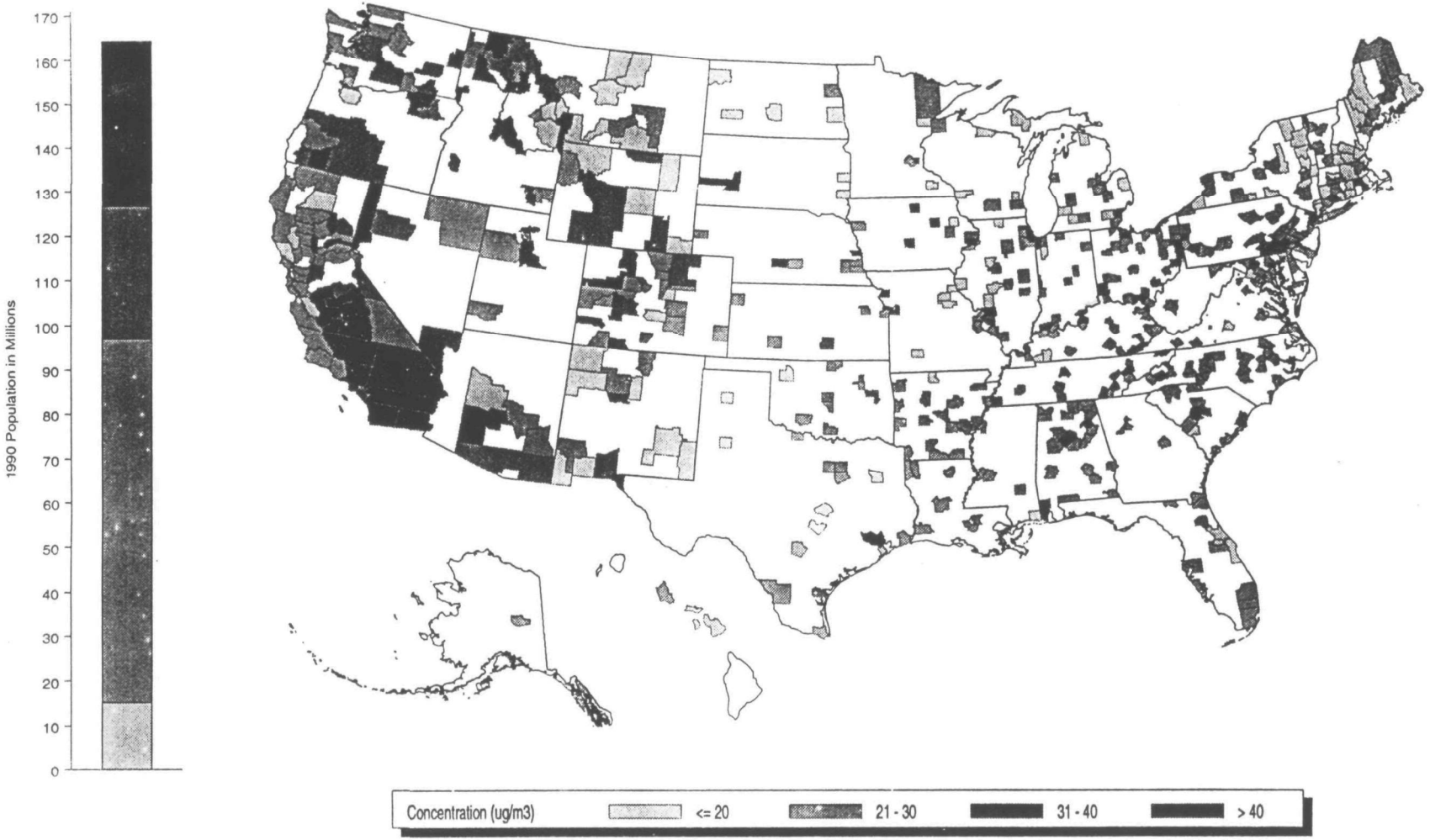
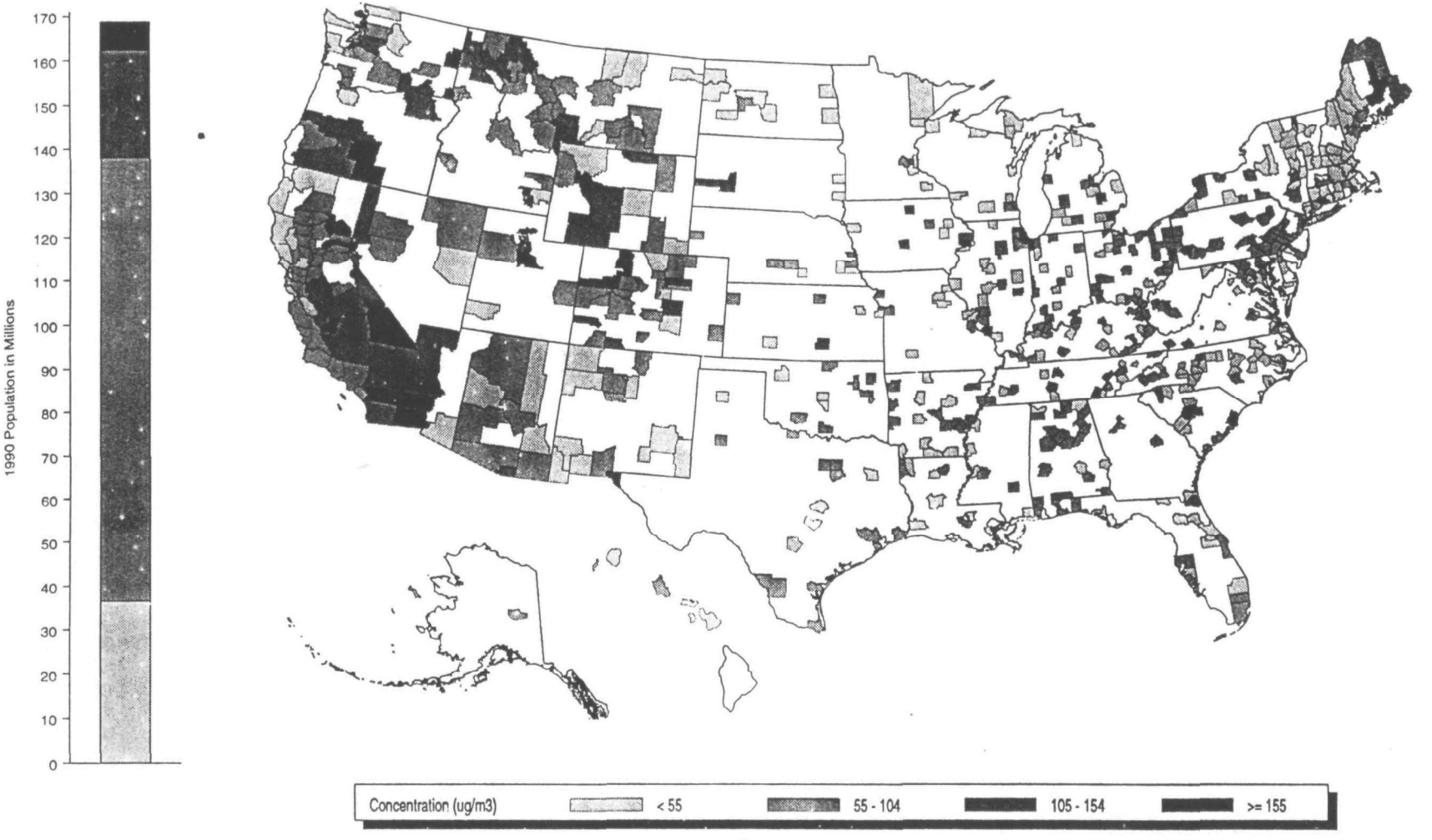


Figure IV-12. **PM-10 Air Quality Concentrations, 1993**
Highest Second Max 24-Hour Average



the 647 counties in the U.S. which have at least one monitor.[4] Counties not represented with a monitor are left blank.

Although $PM_{2.5}$ levels are more difficult to illustrate than PM_{10}, the large PM_{10} dataset can be scaled to augment the monitored $PM_{2.5}$ data. In the locations where both PM_{10} and $PM_{2.5}$ were measured, it is possible to discern a relationship between the $PM_{2.5}$ and PM_{10} values and other factors and then to apply that relationship to compose a morecomplete picture of $PM_{2.5}$ levels across the U.S. This approach was used to create Figures IV-13 and IV-14, which depict annual mean and 24-hour values, respectively, of $PM_{2.5}$ concentrations predicted from PM_{10} values and other factors on a county basis. More detail of this approach and the associated uncertainties are addressed in Appendix C.

E. Air Quality Comparisons of $PM_{2.5}$ and PM_{10}

In contrast to fine and coarse fractions which are distinct entities, fine particles are a subset of PM_{10}. $PM_{2.5}$ and PM_{10} may be related using mass ratios, correlations, and day-to-day variation. Because of the limited $PM_{2.5}$ monitoring compared to PM_{10} monitoring, the conclusions are dependent on the available data. Geographic differences from the Eastern U.S. and the Western U.S. are discernable in the relationships between $PM_{2.5}$ and PM_{10}.

1. Mass Concentration Ratios of $PM_{2.5}$ to PM_{10}

Ratios of daily $PM_{2.5}$ to PM_{10} mass concentrations show what percentage of the PM_{10} is attributable to fine particles. The national ratio of daily $PM_{2.5}$ to PM_{10} is 0.58, indicating that based on the available data, almost 60 percent of the PM_{10} mass is fine particles. The percentage varies as much as by a factor of 2 depending on the region and season (SAI, 1995). The ratios also vary significantly by time period and site location because of changes in sources over time.

Table IV-3 presents the medians of 24-hour $PM_{2.5}/PM_{10}$ mass concentration ratios by region and season. The table shows significant variability by region.

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[4]For counties with more than one monitor, the monitor with the highest concentration is used for plotting purposes. In Figure IV-11, only annual means with at least seventy-five percent data completeness are used. Note that tests for attainment and nonattainment of the PM_{10} standard are generally based on 3 years of data. Since these figures represent only 1 year of data, no conclusion can be made concerning a county's attainment or nonattainment status using these figures alone.

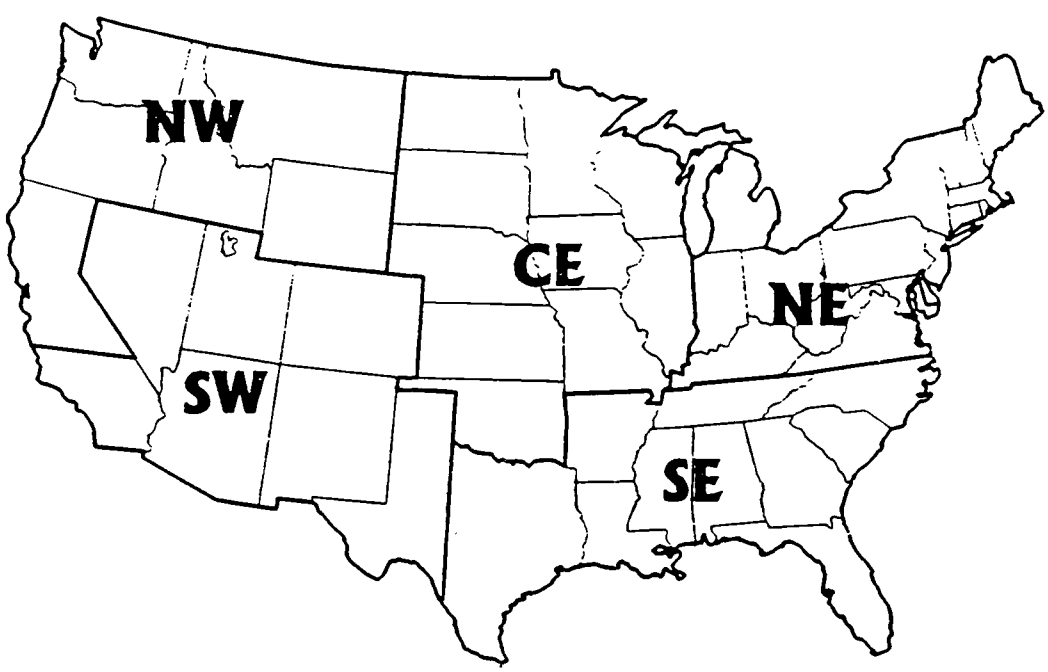
Figure IV-13. **Predicted Fine Particle Concentrations, 1993**
Highest Annual Mean

Figure IV-14. **Predicted Fine Particle Concentrations, 1993**
Highest Second Max 24-Hour Average

Table IV-3. Median of 24-hour $PM_{2.5}/PM_{10}$ Mass Concentration Ratios

Region	Season				
	Spring	Summer	Fall	Winter	All
CE	0.59	0.62	0.61	0.69	0.63
NE	0.61	0.62	0.63	0.65	0.63
SE	0.54	0.53	0.55	0.62	0.57
NW	0.34	0.26	0.40	0.58	0.36
SW	0.31	0.33	0.38	0.43	0.35
All regions	0.55	0.57	0.59	0.63	0.58

Source: SAI, 1995; using coincident monitored data from 1988–1993.



For example, the Eastern U.S. shows less variability in the ratio across season than the West (especially the Northwest). The seasonal ratios range from 0.61 to 0.65 in the Northeast while the ratios range from 0.26 to 0.58 in the Northwest. Moreover, the Eastern ratios for all seasons combined (0.57-0.63) are almost twice that found in the West (0.35-0.36), indicating that fine particles are a more dominant part of PM_{10} in the East due to the types of sources.

One of the major reasons $PM_{2.5}$ and PM_{10} do not compare well in the Western U.S. is the higher mass contribution of the coarse fraction in the arid Southwestern and Western U.S. However, most of the health studies have been conducted in the Eastern U.S., where $PM_{2.5}$ and PM_{10} correlate well and $PM_{2.5}$ contributes well over half of the daily particle mass or in Western urban locations, which are dominated by combustion sources, as discussed further in Chapter V. The regional differences in the 24-hour mass concentration ratios also reflect a difference in the correlation between daily $PM_{2.5}$ and PM_{10} values, driven primarily by the regional differences in fine and coarse fractions.

2. Correlations Between $PM_{2.5}$ and PM_{10}

The $PM_{2.5}/PM_{10}$ mass concentration ratios presented above are averages over observed concentration levels in a given region. Figure IV-15a displays the relative 24-hour concentrations of $PM_{2.5}$ and PM_{10} over increasing concentrations. Figure IV-15b presents a similar comparison using annual mean concentrations. As presented in Table IV-4, nationally $PM_{2.5}$ correlates much better with PM_{10} than with the coarse fraction, as evinced by the higher R-squared statistic for both daily and annual comparisons.

Table IV-5 presents annual and summertime correlations of daily measurements of $PM_{2.1}$ with PM_{10} and daily measurements of sulfates with PM_{10} in 24 North American Cities. These measurements are all part of a single study so that the uncertainties about combining sampling protocols are minimized. Daily $PM_{2.1}$ is well correlated with PM_{10} in most Eastern U.S. cities (generally R^2 statistic ranges from approximately 0.6 to 0.8 with one value near 0.3). Daily $PM_{2.1}$ is somewhat correlated with PM_{10} in the Canadian and California sites (R^2 range 0.4 to 0.6). The Northern sites' correlations increase in the summer while the California correlations decrease in the summer. California sites also show

Figure IV-15. Scatter Plot of $PM_{2.5}$ and PM_{10} Concentrations

Figure IV-15a. Daily $PM_{2.5}$ v. PM_{10} Concentrations

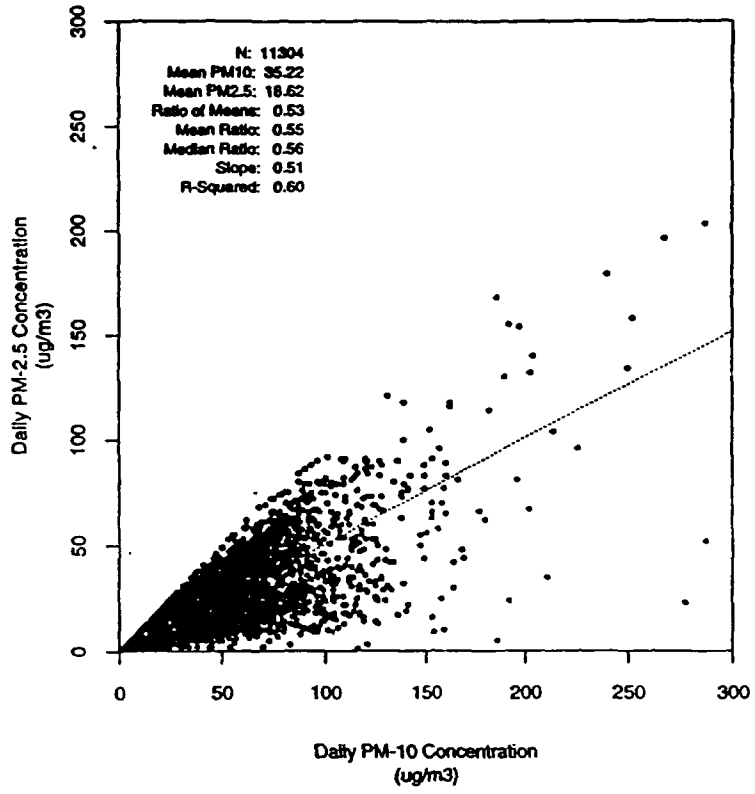


Figure IV-15b. Annual $PM_{2.5}$ v. PM_{10} Concentrations

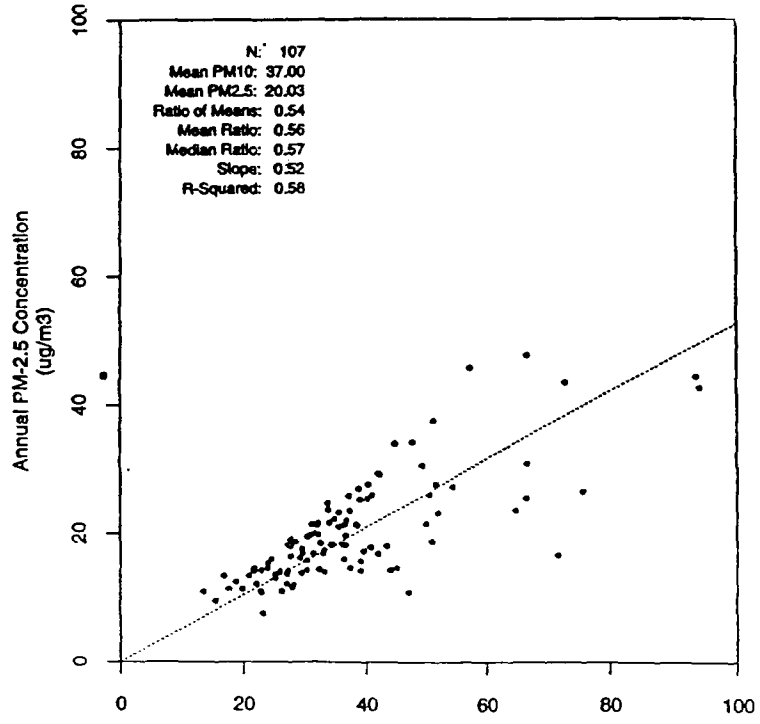


Table IV-4. Correlations of Fine, Coarse Fraction and PM_{10}

	$PM_{2.5}/PM_{10}$ R-squared Statistic	$PM_{2.5}$/Coarse Fraction R-squared Statistic	Sample Size, N
Daily Values	0.60	0.08	11,304
Annual Values	0.58	0.11	107

Source: SAI, 1995; Using monitored data from 1985 to 1993

Table IV-5. Correlations of $PM_{2.1}$ with PM_{10} and Sulfates with PM_{10} in 24 North American Cities

SITE	Year Round Daily Correlations ($PM_{2.1}$,PM_{10})	Year Round Daily Correlations (SO_4,PM_{10})	Summer (May - Sept.) Daily Correlations ($PM_{2.1}$,PM_{10})	Summer (May - Sept.) Daily Correlations (SO_4,PM_{10})	Annual $PM_{2.1}$ Mass Conc. ($\mu g/m^3$)
Eastern U.S. Sites					
Oak Ridge, TN	0.72	0.40	0.79	0.36	17.1
Hendersonville,TN	0.34	0.34	0.48	0.42	16.4
Morehead, KY	0.64	0.61	0.61	0.59	20.0
Blacksburg, VA	0.67	0.61	0.72	0.71	N/A
Charlottesville, VA	0.76	0.62	0.83	0.67	16.0
Zanesville, OH	0.59	0.76	0.55	0.77	16.9
Athens, OH	0.81	0.62	0.92	0.66	N/A
Parsons, WV	0.62	0.29	0.85	0.64	17.0
Uniontown, PA	0.76	0.38	0.81	0.34	21.0
Penn Hill, PA	0.62	0.69	0.62	0.67	19.3
State College, PA	0.81	0.77	0.90	0.88	N/A
Northern Sites					
Dunnville, ON	0.50	0.53	0.55	0.52	16.2
Leamington, ON	0.61	0.55	0.77	0.74	N/A
Newtown, CT	0.61	0.52	0.79	0.69	13.8
Egbert, ON	0.49	N/A	0.59	N/A	N/A
Pembroke, ON	0.55	0.49	0.76	0.62	10.4
West Coast Sites					
Simi Valley, CA	0.56	0.46	0.50	0.24	18.2
Livermore, CA	0.52	0.10	0.41	0.04	15.3
Monterey, CA	0.42	0.06	0.11	0.00	9.4
Rural Sites					
Springdale, AR	0.31	0.17	0.23	0.15	14.4
Aberdeen, SD	0.17	0.00	0.50	0.00	N/A
Yorkton, SK	0.11	0.02	0.11	0.07	N/A
Penticton, BC	0.46	0.08	0.49	0.06	10.0

Source: Spengler (1995)

a much lower correlation of sulfates with PM_{10}, especially in the summer. Sulfate correlations with PM_{10} are notably lower on the West Coast and in some of the rural sites than in the northern or eastern sites. Rural sites also generally show poor correlations between $PM_{2.1}$ and PM_{10}.

The $PM_{2.5}$ component contributes more to the day-to-day variation in PM_{10} values. Figure IV-16 presents a time series of the $PM_{2.5}$, coarse fraction, and PM_{10} values in Philadelphia, PA, in 1990. It can be seen that on some days, $PM_{2.5}$ and the coarse fraction increase and decrease together, and on other days they move in opposite directions. However, in most periods, $PM_{2.5}$ dominates the day-to-day variability in the PM_{10} mass, and the coarse fraction acts almost as a constant. Wilson et al. (1995) present similar conclusions for Philadelphia for 1992 and 1993, reporting a high correlation between daily $PM_{2.5}$ and PM_{10} (R^2 = 0.90); a moderate correlation between the coarse fraction and PM_{10} (R^2 = 0.35); and a low correlation between fine and coarse faction (R^2 = 0.11). To understand how fine particles, PM_{10}, and TSP compare an analysis of AIRS data in Philadelphia in 1982 was conducted. The daily values of $PM_{2.5}$ to PM_{10} are better correlated (R^2 = 0.90) than $PM_{2.5}$ and TSP (R^2 = 0.58). A moderate correlation was again observed between the coarse fraction and PM_{10} (R^2 = 0.44).

Figure IV-17 presents a similar time series plot of the $PM_{2.5}$, coarse fraction, and PM_{10} values in St. Louis, MO, in 1993. This graph also suggests that much of the day-to-day variability of PM_{10} mass is driven by the day-to-day variability of $PM_{2.5}$. Wilson et al. (1995) also examined St. Louis during the period 1988 to 1993 using PM_{15} (which are similar to PM_{10} measurements but would be expected to contain more coarse fraction mass) and reported results similar to Philadelphia. A high correlation between daily $PM_{2.5}$ and PM15 was reported (R^2 = 0.85); a moderate correlation between the coarse fraction and PM_{10} (R^2 = 0.55); and a low correlation between fine and coarse faction (R^2 = 0.19).

Based on a wider examination of $PM_{2.5}$ data available from the AIRS database, which tends to contain urban and suburban locations, Wilson et al. (1995) reported that in most cities where $PM_{2.5}$ concentration exceeds the coarse fraction concentration, the variation in daily PM_{10} concentrations at any individual site characteristic of the community

Figure IV-16. Philadelphia, PA, 1990

PM10, PM2.5, and Coarse Fraction

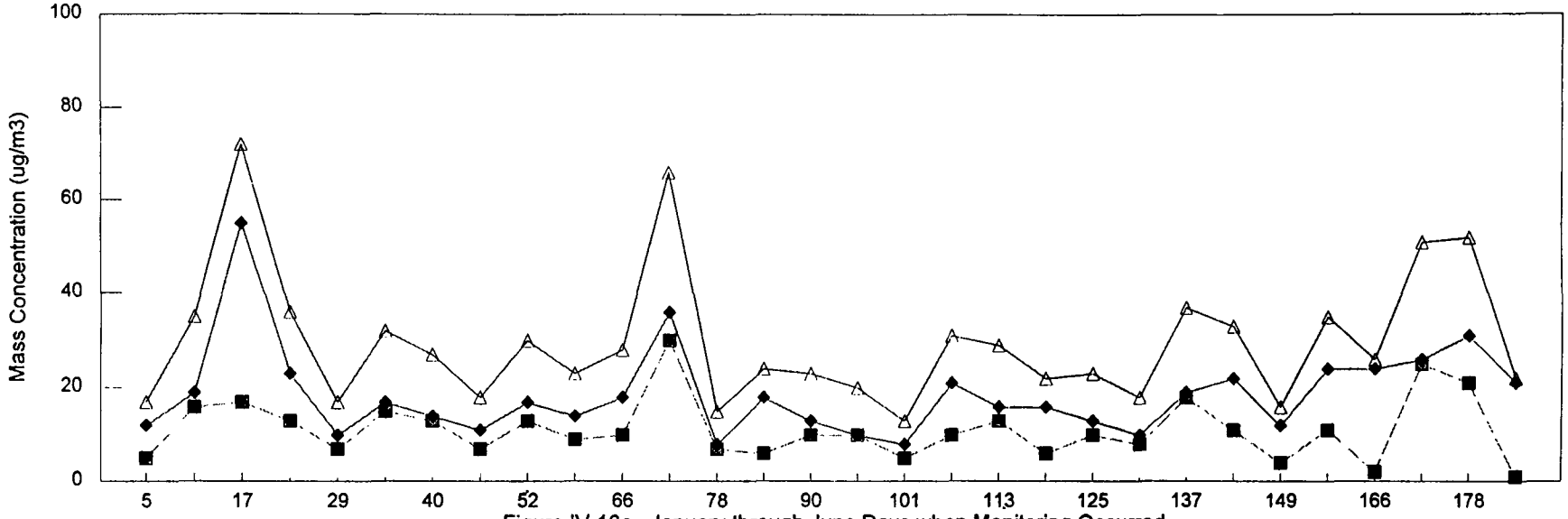


Figure IV-16a. January through June Days when Monitoring Occurred

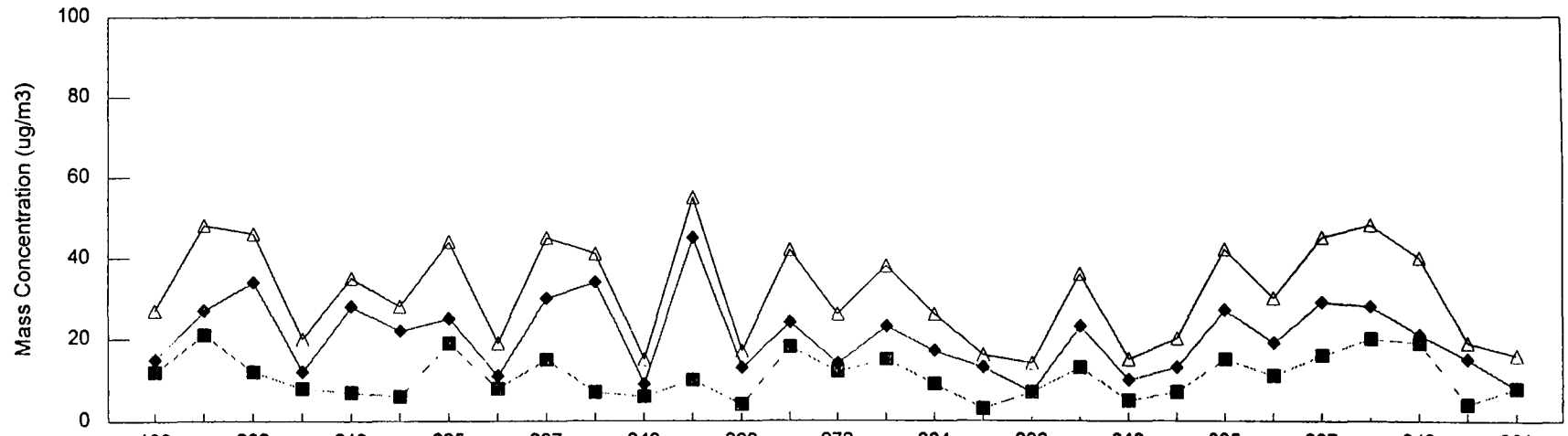


Figure IV-16b. July through December Days when Monitoring Occurred

Note: Sampling occurred on a one-in-six day schedule

Coarse Mass — PM-2.5 — PM-10

Figure IV-17. St. Louis, MO, 1993

PM10, PM2.5, and Coarse Fraction

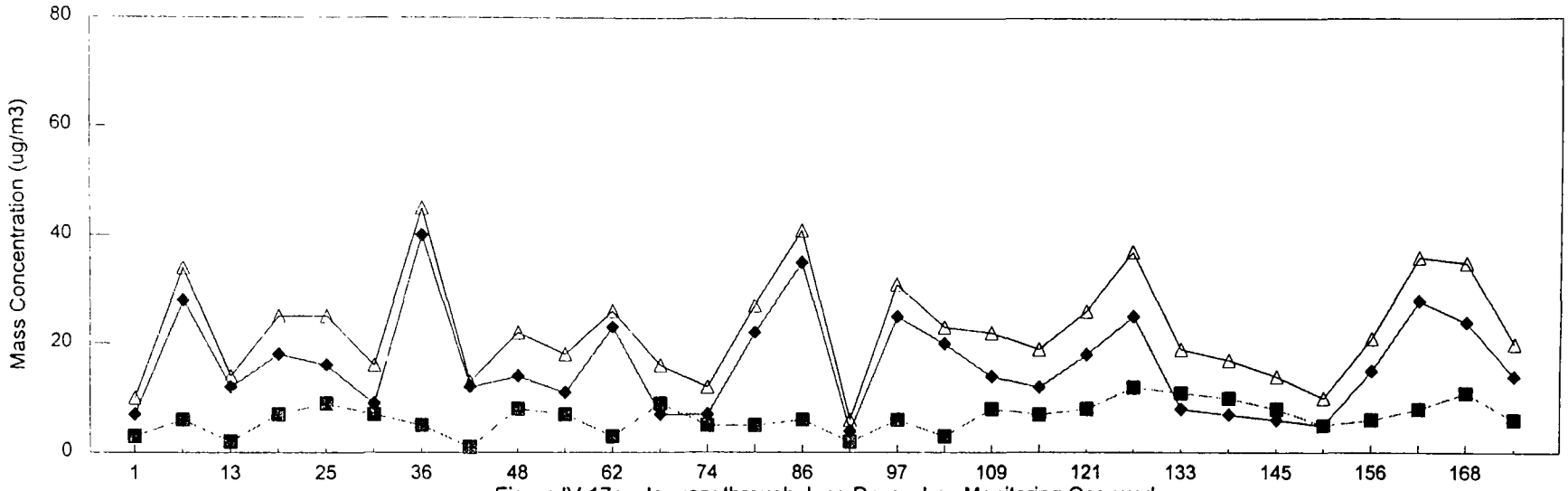


Figure IV-17a. January through June Days when Monitoring Occurred

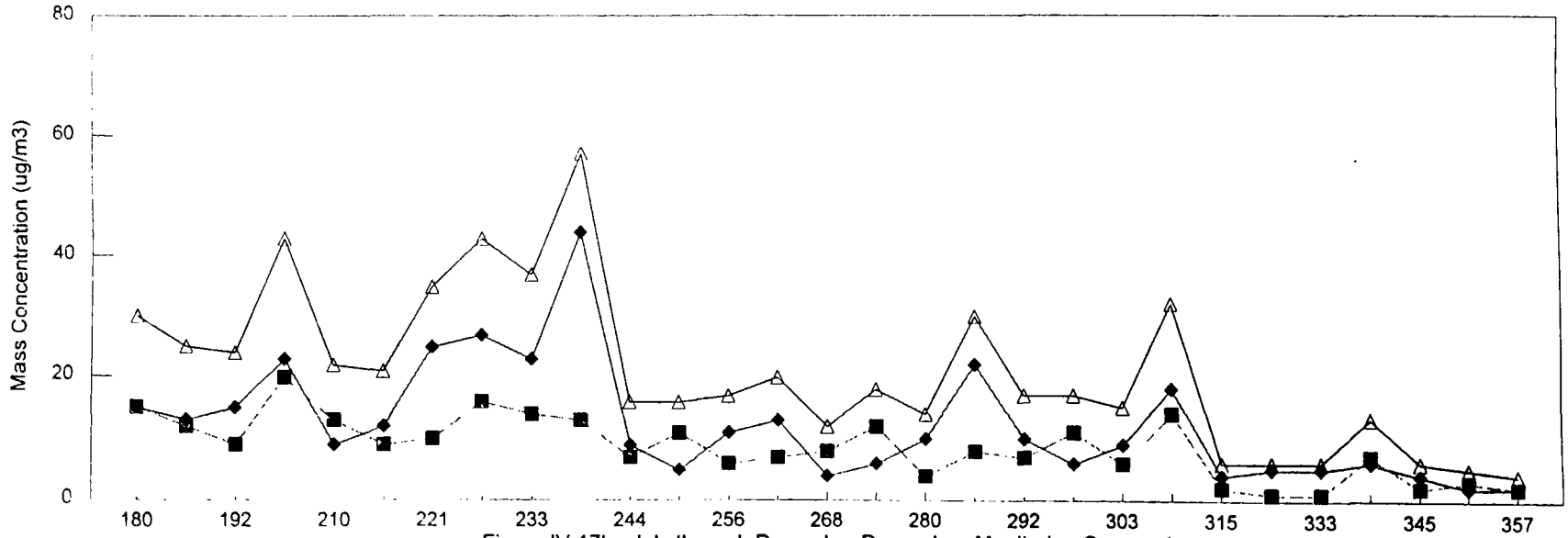


Figure IV-17b. July through December Days when Monitoring Occurred

Note: Sampling occurred on a one-in-six day schedule

■ Coarse Mass ◆ PM-2.5 △ PM-10

will be dominated by the variability in $PM_{2.5}$. Wilson and colleagues also conclude 75 percent of the data sets in AIRS support the proposition that although epidemiological correlations have been made with TSP and PM_{10}, it is possible that the variability in the fine particles, not the variability in PM_{10} or the coarse fraction, produces a correlation with the health endpoints.

In addition to the day-to-day variation in $PM_{2.5}$, the day-to-day relationship between PM concentrations monitored outdoors at a monitoring station to measurements of personal exposure is important to interpreting the time series community health studies. Personal exposure to outdoor pollutants can vary considerably from the concentrations measured at a monitoring station. Typically, PM personal exposure measurements are higher than the outdoor PM concentrations due to indoor sources of particles such as cooking, cleaning and smoking. However, unless there is a day to day correlation between personal exposure and other factors such as systematic measurement error or personal behaviors, individual personal exposure will be higher on days when outdoor PM levels are high and lower on days when outdoor PM levels are lower.

The CD reports a major new finding which differentiates between the low correlations of PM personal exposure measurements with PM outdoor monitoring found in cross-sectional exposure study designs versus time series designs (CD, Chapter 7). Janssen et al. (1995) point out that the low correlations observed in most of the other studies reported in the literature were cross-sectional (i.e., calculated on a group level), and were therefore mostly determined by the variation between subjects. A more relevant model is the serial correlation reported by Janssen et al. (1995) and noted by Lioy et al. (1990) for personal and outdoor correlations.

These exposure studies report good serial correlations and poor cross-sectional correlations that are consistent with the other cross-sectional results reported in the literature. For instance, Janssen et al. (1995) report good serial correlations between personal exposure measurements and stationary monitors for PM_{10} (median R^2 = 0.40, and as high as 0.96 for children and median R^2 = 0.25, and as high as 0.85 for adults). Excluding days with exposure to environmental tobacco smoke raised the median R^2 for PM_{10} from 0.40 to 0.53 for children and from 0.25 to 0.50 for adults. Even better results were obtained for the serial correlations of $PM_{2.5}$ (median R^2 = 0.74 for children). Thus, these results suggest that a substantial amount of the day-to-day variation in personal exposure to fine particles can be attributed to day-to-day variations in outdoor $PM_{2.5}$ concentrations. The link to the health database will be explored further in Chapters V and VI.

3. Ratio of Highest Daily Value to Annual Mean

Short-term variability in PM_{10} varies by region (CD Chapter 6). The highest daily value (or peak) versus the mean value is seen in the northern and western continental US, especially in the winter. The lowest variation prevails over the warm season in the Southwest and Southeast. The CD concludes that the southern areas are more uniformly covered by summertime PM_{10}; whereas, the northern states experience more episodic high concentrations of PM_{10} (CD Chapter 6).

Staff further examined the ratios between the highest daily value in a year and the annual mean and their dependence on year, region, and precipitation (relative to the long term average precipitation for the given location). The results, summarized in Table IV-6, indicate that for $PM_{2.5}$ and PM_{10}, the average ratio of the highest daily value to annual

TABLE IV-6. Summary of Ratios of Highest Annual Day to Annual Mean for $PM_{2.5}$ and PM_{10}.

Summary Statistic	$PM_{2.5}$		PM_{10}	
	Mean	Std. Dev.	Mean	Std. Dev.
Highest-Annual-Day-to-Annual-Mean Ratio	3.08	0.91	3.01	1.53
Second-Highest-Annual-Day-to-Annual-Mean Ratio	2.52	0.62	2.41	0.78
Second-Highest-3-Year-Value-to-Mean Ratio	3.27	0.80	3.11	1.50
Third-Highest-3-Year-Value-to-Mean Ratio	2.98	0.72	2.80	1.25
Fourth-Highest-3-Year-Value-to-Mean Ratio	2.80	0.67	2.60	0.81

The 3-year ratios were based on 1991–1993 data only, but the annual peak-to-mean ratios used all available data.

mean is about 3, with standard deviations of about 0.9 and 1.5, respectively. No clear patterns were discernable in the variation by year on the highest daily value-to-annual mean ratios. The most noticeable effect of location was that Northwest sites tended to have higher ratios compared to other regions, and based on analysis of annual precipitation, dry years tended to have higher ratios than normal or wet years. Since the sites in the Northwest also tended to be the sites with dry years, it is not clear whether location or precipitation is the more important factor in determining the size of the ratio of highest daily value to annual mean. In other words, the location and precipitation effects are highly correlated so it is hard to separate out these two effects.

Table IV-7 presents more detailed distribution of the values for PM_{10}. Likewise, Table IV-8 presents the same information for $PM_{2.5}$. There is more certainty associated with the PM_{10} analyses than the $PM_{2.5}$ analyses because of the larger data base for PM_{10} (approximately 8,400 data points from across the country).

Table IV-7. Distribution of Highest 24-Hour-Value-to-Annual Mean Ratio for PM_{10}

TABLE 7a. Distributions of highest-annual-peak-to-mean ratios, compared by region: PM 10.

Cumulative Fraction	Northwest	Central	Northeast	Southeast	Southwest	ALL
Minimum	1.29	1.40	1.32	1.20	1.35	1.20
1	1.62	1.73	1.63	1.58	1.60	1.62
5	1.97	1.91	1.91	1.81	1.81	1.88
10	2.17	2.03	2.04	1.90	1.96	2.01
20	2.43	2.21	2.19	2.02	2.17	2.19
30	2.66	2.37	2.33	2.15	2.35	2.35
40	2.86	2.49	2.46	2.28	2.57	2.51
50	3.09	2.63	2.58	2.41	2.78	2.67
60	3.38	2.80	2.72	2.57	3.02	2.86
70	3.76	3.04	2.90	2.78	3.37	3.10
80	4.30	3.36	3.12	3.07	3.73	3.49
90	5.51	3.94	3.58	3.54	4.67	4.17
95	7.36	4.57	4.12	4.02	5.58	5.08
99	13.98	7.06	5.83	4.95	8.16	8.27
Maximum	43.87	16.54	16.09	7.14	15.12	43.87
Mean	3.72	2.89	2.75	2.59	3.13	3.01
Std. Dev	2.62	1.05	0.86	0.71	1.40	1.53
Number	1704.00	1083.00	3045.00	1160.00	1413.00	8405.00

TABLE 7b. Distributions of second highest-annual-peak-to-mean ratios, compared by region: PM 10.

Cumulative Fraction	Northwest	Central	Northeast	Southeast	Southwest	ALL
Minimum	0.94	1.08	0.82	1.05	1.15	0.82
1	1.30	1.37	1.37	1.35	1.36	1.35
5	1.66	1.70	1.69	1.60	1.65	1.66
10	1.84	1.80	1.81	1.70	1.76	1.78
20	2.07	1.94	1.95	1.81	1.90	1.92
30	2.23	2.04	2.05	1.88	2.02	2.03
40	2.39	2.12	2.14	1.95	2.12	2.14
50	2.55	2.21	2.23	2.03	2.25	2.24
60	2.74	2.32	2.33	2.13	2.41	2.37
70	2.98	2.46	2.46	2.23	2.62	2.54
80	3.31	2.67	2.62	2.39	2.91	2.76
90	3.89	2.97	2.91	2.62	3.35	3.19
95	4.70	3.35	3.20	2.89	3.94	3.67
99	7.36	4.19	4.09	3.52	5.16	5.06
Maximum	26.88	5.65	9.32	6.21	7.33	26.88
Mean	2.80	2.33	2.31	2.12	2.45	2.41
Std. Dev	1.22	0.54	0.51	0.43	0.76	0.78
Number	1704.00	1083.00	3045.00	1160.00	1413.00	8405.00

Table IV-8. Distribution of Highest 24-Hour-Value-to-Annual Mean Ratio for $PM_{2.5}$

TABLE 8a. Distributions of highest-annual-peak-to-mean ratios, compared by region: PM 2.5.

Cumulative Fraction	Northwest	Central	Northeast	Southeast	Southwest	ALL
Minimum	1.55	1.57	1.53	1.67	1.55	1.53
1	1.96	1.57	1.66	1.67	1.62	1.62
5	2.18	2.16	1.90	1.76	1.85	1.91
10	2.37	2.49	2.08	2.01	1.97	2.07
20	2.71	2.61	2.30	2.17	2.15	2.30
30	2.95	2.72	2.47	2.29	2.31	2.54
40	3.20	2.89	2.63	2.50	2.54	2.73
50	3.43	2.99	2.79	2.77	2.72	2.91
60	3.72	3.09	2.94	2.91	2.95	3.11
70	3.84	3.34	3.11	3.15	3.26	3.39
80	4.28	3.78	3.34	3.39	3.70	3.78
90	4.96	4.18	3.85	4.64	4.13	4.32
95	5.20	4.50	4.33	5.19	4.51	4.86
99	6.44	4.64	5.00	5.66	5.89	5.81
Maximum	7.05	4.64	5.76	5.66	7.34	7.34
Mean	3.53	3.13	2.88	2.95	2.91	3.08
Std. Dev	0.96	0.68	0.72	0.99	0.92	0.91
Number	117.00	38.00	110.00	53.00	153.00	471.00

TABLE 8b. Distributions of second highest-annual-peak-to-mean ratios, compared by region: PM 2.5.

Cumulative Fraction	Northwest	Central	Northeast	Southeast	Southwest	ALL
Minimum	1.53	1.43	1.17	1.16	1.36	1.16
1	1.61	1.43	1.39	1.16	1.38	1.39
5	1.88	1.46	1.65	1.41	1.72	1.71
10	2.09	1.92	1.80	1.71	1.79	1.85
20	2.35	2.11	2.04	1.94	1.93	2.01
30	2.51	2.28	2.13	2.08	1.99	2.15
40	2.60	2.35	2.24	2.18	2.10	2.28
50	2.86	2.41	2.35	2.27	2.23	2.40
60	3.05	2.57	2.56	2.30	2.36	2.59
70	3.24	2.72	2.67	2.55	2.56	2.73
80	3.43	2.96	2.77	2.68	2.75	3.00
90	3.79	3.18	3.12	3.05	3.17	3.34
95	3.99	3.48	3.25	3.34	3.43	3.64
99	4.46	3.64	4.39	5.15	3.83	4.44
Maximum	4.54	3.64	4.84	5.15	4.08	5.15
Mean	2.90	2.50	2.43	2.36	2.35	2.52
Std. Dev	0.65	0.50	0.54	0.65	0.54	0.62
Number	117.00	38.00	110.00	53.00	153.00	471.00

F. Background PM Levels

Some PM is suspended in the atmosphere through mechanisms independent of the activities of people. The range of concentrations can vary greatly over time and location. The literature contains limited information on monitored or estimated values of background average concentrations of PM for various averaging times (annual, seasonal, daily) or various locations in the United States. In the literature there is also a large disparity in definitions of "background" concentrations, and estimates of natural components of the PM are scarce. There is no standardized terminology regarding the concept of PM background.[5]

Based on the CD's review of the limited literature, defining background levels of PM and determining what part of PM is attributable to natural phenomena is a multi-dimensional and complex concept. Background levels of PM vary by geographic location, altitude and season. For the purposes of this document, background PM is defined as the range 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. An estimate of background PM_{10} annual average concentrations across the U.S. ranges between 4.5 and 6.3 $\mu g/m^3$ including associated water (NAPAP, 1991). Similarly, an estimate of background fine PM annual average concentrations across the U.S. ranges between 1.5 and 3.3 $\mu g/m^3$ including associated water (NAPAP, 1991). There is a definite geographic trend to these levels with the lower value applicable to the Western and the higher value 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. Most of this background estimate is believed to be due to natural sources.

The natural component of the background arises from physical processes of the atmosphere that entrain fine particles of crustal material (i.e., soil) as well as emissions of organic particles resulting from natural combustion sources such as wildfire. In addition

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[5]The literature refers to many different terms for background including; "clean background", "average background," "continental background," "clean continental background," and "background." None of these studies defined a specific approach to determine whether anthropogenic sources were contributing to the background.

certain vegetation can emit fine organic aerosols as well as their precursors. The exact magnitude of this natural part for a given geographic location can not be precisely determined because the magnitude of the long-range transport of anthropogenic precursor emissions is not well known. Only broad estimates for longer averaging times can be developed at this time.

For the purposes of determining how background concentrations affect visibility, a welfare consideration linked to fine particle concentrations, it is necessary to know the contribution of each particle species to the estimated background concentrations because the light extinction coeeficient varies by species. Estimates of background concentrations by species have been developed using rural monitoring and consideration of anthropogenic emissions, including precursors, to the species. Fine particle background also varies by location and season, and these variations are important to assessing the relationship between fine particle concentrations and visibility effects. These estimates and their importance to developing a recommendation on the appropriateness of a national secondary standard to address visibility effects can be found in Chapter VII.

V. CRITICAL ELEMENTS IN THE REVIEW OF THE PRIMARY STANDARDS

The health information most relevant to the review of the primary standards for PM is presented in this chapter. It builds upon the integrative summary developed in the last review (U.S. EPA, 1982b; 1986b), focusing on perspectives drawn from the significant new body of information that has accumulated in the intervening years. The chapter begins with a discussion of mechanisms of action, including the penetration, deposition, and clearance of the major fractions of outdoor PM discussed in the previous chapter, as well as the possible physiological and pathological responses to these particulate substances drawn from animal, controlled human, and epidemiological studies. Past and recent evidence useful in identifying potential sensitive populations is then discussed. Key findings from recent evidence regarding the potential effects of PM are then outlined. Since a large proportion of the evidence is from community epidemiological studies, the coherence and strength of the epidemiological evidence is assessed. Finally, those studies most useful for developing quantitative assessment of the potential health effects of PM are presented.

A. Mechanisms

In this section, possible mechanisms of constituents of PM which may produce observed health effects, and the relevant information concerning such mechanisms will be described. First, dosimetric considerations are discussed which helped to form the basis of the current standard. Then a discussion of the most recent information concerning dosimetry, as it pertains to the elucidation of the potential mechanisms of PM effect, is presented. Finally, a discussion is provided concerning the possible mechanisms by which PM may contribute to observed effects, recognizing that most of the controlled studies used exposures to particulate substances much higher than found in contemporary atmospheres.

1. Dosimetric Considerations

a. Current Standard

This discussion describes dosimetric information and concepts upon which selection of the indicator in the current standard is based. Such 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. 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, Chapter 10). The amount of particles deposited or retained in each region of the respiratory tract is governed by exposure concentration, the particle diameter and distribution, and physico-chemical properties of the inhaled particle (e.g., hygroscopy and solubility), as well as species specific features of anatomy (airway geometry, ventilation rate and physiology) (CD, Chapter 13). It is the dose that the target site or organ receives, upon which manifestation of toxicity depends. In the previous review, based on dosimetric considerations and on aerosol physico-chemical characteristics, the staff, with CASAC concurrence, determined that the major risk of commonly occurring outdoor PM was presented by those particles that penetrate to the tracheobronchial and alveolar regions of the human respiratory tract (U.S. EPA, 1982b). Consequently, the determination of what is able to be deposited in these areas, how long it stays there, and its toxicity are of great importance in developing risk assessments for particle exposures and subsequent strategies for the prevention of PM health effects.

As discussed in Chapter IV of this document, the mass and volume of typically observed ambient particles tend to be distributed in two distinct size modes described as fine mode and coarse mode particles (see Figure IV-1). While the fine mode contains most of the surface area and numbers of particles, and about 1/3 to 1/2 of PM volume and mass, the coarse mode, by comparison, contains much smaller numbers of particles and about 1/2 to 2/3 of particulate volume and mass. (See Figure IV-2).

The human respiratory tract can be divided into three main regions: (1) extra-thoracic, (2) tracheobronchial, and (3) alveolar regions as shown in Table V-1. They differ markedly in structure, function, size, and sensitivity or reactivity to deposited particles (U.S. EPA, 1982b). In addition, there are differences in deposition and clearance of PM in the regions of the lung. In humans, the principal mechanisms for deposition of particles of differing diameter are inertial impaction (2 - 100 μm diameter), sedimentation (0.5 - 2.0 μm diameter), and diffusion ($<$ 0.5 μm diameter). A qualitative assessment of the deposition of

TABLE V-1. MAJOR REGIONS OF THE RESPIRATORY TRACT (Mort Lippmann, 1982 Staff Paper)

Region	Description	Principal Size Range (s) Deposited	Major Mechanisms of Deposition	Major Mechanisms of Clearance	Normal Clearance Times for Insoluble Particles
Extrathoracic (ET)	The head and pharynx, down to and including the larynx. Subdivisions: Nose breathing Anterior nares Ciliated nasal passages Nasopharynx Mouth breathing Mouth, oropharynx	~1 - >100μm[a] hygroscopic aerosols > ~0.3μm	Impaction	Mucociliary action to G.I. tract Sneezing, blowing, wiping to exterior Dissolution to blood-stream or mucous	minutes longer(nares)
Thoracic (TB+AL)					
Tracheobronchial (TB)	Ciliated conducting airways from the tracheal to the terminal ciliated bronchioles. (~ 16 generations).	~0.2 - 15μm[b]	Impaction Sedimentation Interception (Fibers)	Mucociliary action, coughing to G.I. Tract Dissolution to blood-stream or mucous	hours
Alveolar (AL) (Pulmonary)	Gas exchange region including unciliated airways and alveoli. Subdivisions: Respiratory bronchioles, alveolar ducts, alveolar sacs, atria, and alveoli.	<20 μm[c]	Diffusion(<1 μm) Sedimentation Impaction (>2 μm)	Phagocytosis to TB region, lymphatic system Dissolution to blood-stream, other fluids	weeks-years

[a]"Inspirability" Extrapolation by ISO, 1981.
[b]Extrapolation of NYU data by Miller et al., 1979.
[c]Figures 11-8, 11-9 criteria document.

typical ambient particles is summarized in the 1982 Staff Paper (U.S. EPA, 1982b). Based on deposition for normal nasal breathing, over half of the total mass distribution of inhaled PM would be deposited in the extra-thoracic region, most of this being coarse particles. Up to half of the hygroscopic fine mass (e.g., sulfates that grow to 2-4 μm diameter) is predicted to also be deposited and dissolved in this same region. Smaller fractions (5-25 percent) of hygroscopic and non-hygroscopic fine particles (mostly $\leq$ 1 μm diameter) would be deposited in the tracheobronchial and alveolar region respectively. A similar fraction of coarse particles (2.5 - 8 μm diameter) would be deposited in the same regions (U.S. EPA, 1982b).

In essence, regional deposition of ambient particles in the respiratory tract does not occur at divisions that clearly correspond to the distribution of size of particles that occur in the atmosphere. Nevertheless, little coarse particle mass of diameter larger than 15 μm is deposited in the tracheobronchial region and little mass greater than 10 μm in diameter is deposited in the alveolar region. Particles smaller than 10 μm in diameter can be deposited with varying efficiencies in both regions. A more complete discussion of possible responses to particle deposition, potential mechanisms of those responses as well as regional deposition is discussed in the 1982 Staff Paper (see Table 5-2 in Appendix F).

The above generalizations regarding typical particle deposition in normally breathing adults are subject to great variability. Deposition into specific regions of the respiratory tract and lung can be influenced by changes in respiratory flow rate, respiratory frequency, and tidal volume. Consequently, the activity level of an individual may result in a change in the mode of breathing (mouth versus nasal breathing) and can significantly alter regional as well as total respiratory tract deposition of inhalable particles. Mouth breathing results in decreased removal of particles by the upper respiratory tract allowing deeper penetration into the lung. In addition, among normal adults subjects, baseline rates of deposition of particles vary. Disease states have the potential for increased deposition of particles as constriction and inflammation of airways or the increased buildup of mucous may in turn increase local particle deposition. Asthmatics, patients with bronchitis, and cigarette smokers have been shown to have increased deposition of particle in the tracheobronchial region. Furthermore,

models of tracheobronchial deposition also suggests enhanced particle deposition of fine and coarse fractions for children as compared to adults. (U.S. EPA, 1986b, CD, Chapter 10).

Deposition in the tracheobronchial region of coarse mode particles tend to be elevated at the bifurcations where epithelial nerve endings are concentrated. Such nerve endings, which connect to mechanical stimulation receptors, may cause reflex coughing and broncho-constriction upon stimulation by particles. Consequently, individuals with increased deposition rates (e.g., asthmatics) may experience even further increases in deposition of particle due to broncho-constriction, altered clearance and buildup of fine and coarse fraction particle at bifurcations.

Clearance is the removal of deposited particles from lung regions. The clearance mechanisms of each region of the respiratory system are distinctly different as are the expected residence times of any given inhaled particle. Alveolar macrophages engulf particles deposited in lung parenchyma, and then either migrate to the terminal bronchioles where they are transported via the mucocilliary escalator or migrate into the interstitium of the lungs to the lymph nodes. Particle size has been reported to affect ingestion by macrophages. Ultrafine particles of size 20 nm diameter are less effectively phagocytized by macrophage than are larger particles of 200 nm diameter (Oberdörster, 1992). However, once ingested, the clearance of particle-laden alveolar macrophages via the mucocilliary system may not be affected by particle size if solubility and cytotoxicity of the particle are low (CD, Chapter 10).

Poorly soluble fine and coarse particles deposited in the alveolar region would be expected to have clearance times on the order of weeks to months or longer. By comparison, clearance of particles in the tracheobronchial region may take hours to days. However, once deposited it is not certain whether transport rate and therefore clearance of poorly soluble particles is independent of the nature of the particle (size, shape, and composition) (CD, Chapter 13). While it is plausible that differential clearance may have a role in specific susceptibility to PM effects and may be integral to mechanisms of PM toxicity, there is insufficient information to support specific mechanisms (CD, Chapter 10).

In conclusion, size divisions, according to the regional deposition of particles in the human respiratory tract and the distribution of particles found in the atmosphere, form the basis of the approach used for selecting the size fraction of the current standard, namely PM_{10}. The risk of adverse health effects associated with extra-thoracic deposition of commonly found particles larger than a nominal size of 10 μm in diameter was judged to be sufficiently low that they were excluded from the primary particulate standard. Selection of a size fraction for the standard based on a nominal 10 μm indicator was supported by (1) the overlap that occurs in deposition in tracheobronchial and alveolar regions of the lung of both particle modes, (2) the overlap of size ranges in maximum efficiency for alveolar deposition (2-4 μm in diameter), (3) chemical heterogeneity of the fine and coarse particle modes, and (4) the potential for coarse insoluble particles to cause broncho-constriction, altered clearance, and alveolar tissue damage.

b. Recent Dosimetry Considerations of Interest

Knowledge of the effects of disease states on deposition and clearance may assist in characterizing susceptible populations to PM and help elucidate possible mechanisms for susceptibility. Variability of clearance may act as a contributing factor to susceptible populations (age, sickness, smokers, etc.) of PM effects. Greater deposition of particles in subjects with various lung conditions is verified in studies by Kim et al. (1988). Model simulations of compromised lungs have been shown to have greater number of particles deposited per alveolus (Miller et al., 1995). More recent studies also suggest that large differences in clearance rates among different individuals to equivalent chronic exposures of poorly soluble particles may result in large variations in respiratory tract burdens. Consequently, deposition and clearance patterns of particles in the lung may influence the type of response elicited. However, the contribution that differential deposition and clearance of the components of PM might make to observed mortality has not been elucidated or quantified.

Differences in what dose animals or humans receive from a particular concentration of PM is important in attempts at extrapolation of observed effects between species. The greater complexity of the nasal passages coupled with the obligate nasal breathing of rodents

has been suggested to result in greater deposition in the upper respiratory tract of rodents than in humans breathing orally or even nasally (Dahl, 1991), especially of coarse particles (Miller et al., 1995). In regard to smaller diameter particles, model simulations suggest that humans retain more alveolar particles than rats or mice (CD, Chapter 13). Anderson et al. (1990) has shown increased deposition of ultrafine particles (0.02 - 0.24 μm diameter) in patients with chronic obstructive pulmonary disease (COPD). 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). Consequently, dosimetry information adds support for the uncertainty in extrapolation between human studies and experimental animal studies.

The ratio of number of particles to alveolar macrophages can be compared between particles of differing size when dose as measured by mass is kept constant. Under such conditions the larger number of smaller particles per unit mass have a higher probability of interacting with alveolar macrophages. Accordingly, a compromised lung with greater deposition has a greater probability of macrophage or alveolar surface area interaction. Thus, there is support for a potential increased toxicity of smaller particles by increased deposition in a subpopulation at risk and an increased probability of interaction with potential targets of toxicity via increased numbers of particles and surface area.

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. Both fine and coarse fraction particles have the potential for deposition in the tracheobronchial and alveolar regions of the respiratory system and thus have access to potential respiratory targets. The

previous staff assessment of the literature found evidence to support the following potential mechanisms of toxicity for particles deposited in the thoracic region (U.S. EPA, 1982b):

- Chemical and mechanical irritation/stimulation resulting in bronchoconstriction by a variety of fine and coarse particles;
- Enhanced sensitivity to subsequent bronchoconstrictive agents by sulfuric acid;
- Altered clearance rates, increased mucous production by deposited material, including cigarette smoke, sulfuric acid, and dusts;
- Increased deposition and slowed clearance at bronchial bifurcations;
- Direct damage to tissues by acids;
- Decreased oxygen transport and probable increased resistance of blood flow through pulmonary capillaries;
- Death of macrophages resulting in release of proteolytic enzymes that damage alveolar tissues, by silica, other coarse dusts;
- Damage to macrophages, other host defense mechanisms by surface coating of toxic materials;
- Combined effect of exposure and slowed clearance of particles; and,
- Enhancement of damage to lung function by childhood respiratory infections.

As noted in the original presentation, this summary of potential mechanisms is for qualitative purposes only; many of the mechanistic studies supporting these suggestions involve exposures significantly higher than those encountered under ambient conditions.

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). Using this method, preliminary results suggest that it may be important in the development of an appropriate experimental paradigm for elucidating mechanisms of PM mortality observed in humans. Salient aspects of each of these areas is discussed briefly below.

a. Inflammation

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 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). Lipfert (1994) in describing animal deaths as a result of the London Fog of 1952 reports that the only documented animal deaths were among fat prize cattle which had a tendency to suffer from "shipping fever" and that sheep and pigs were unaffected both by shipping fever and the fog. 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. The original report by the Ministry of Health (MOH, 1954), however, not only confirmed the presence of the "fever" in the cows affected by the London fog but also reported cattle death in previous fogs with ordinary stall maintenance and therefore high ambient levels of ammonia that could neutralize acid particles.

Pathology from a limited sample of the affected cattle included capillary engorgement with more limited alveolar wall thickening, swelling of alveolar epithelium and presence of an exudate containing fine particles and neutrophils in some bronchioli. The MOH report

also noted that twelve of the more serious cases from the London fog episode were of fat young cattle in prime condition who after slaughter were shown to have emphysema, commencing pneumonia, and slight enteritis. The report concludes that there may be some anatomical or physiological peculiarity which renders fat cattle liable to develop pulmonary 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).

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 are 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). As discussed below in section C, metals

have been shown to increase the toxicity of particles. Installation 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). Thus, under this proposed mechanism of PM effect, toxicity may involve a response to PM which involves inflammation.

b. Aggravation of Underlying Condition

Aggravation of severity of underlying chronic lung disease has 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 (CD, Chapter 13). Impaired respiratory function may be a way in which PM exerts effects. As stated previously, acid aerosols have 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 (Fuwal and Schlesenger, 1994). Therefore, the elderly with debilitating disease such as asthma may be stressed by the fine acid aerosols. In an epidemiological study, 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.

A hastening of imminent death has also been proposed as one of the mechanisms of PM induced mortality. 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). Moreover, the CD points out that in prospective cohort and time series studies, life shortening due to PM exposure is more than just a hastening of imminent death (CD, Chapter 13). As shown in the time series studies, increased mortality can be detected typically within a few days of increases in ambient PM concentration (Samet et al., 1995). Therefore, the short time period between mortality and elevations in PM is consistent with exacerbation of a preexisting condition rather than initiation of life-threatening symptoms by PM alone.

c. Inflammation and Bronchoconstriction

Recently a methodology has been developed for concentration of ambient particles for the purposes of exposing experimental animals to specific size fractions of the ambient PM which may be associated with observed effects in humans (Sioutas et al., 1995). Such a method is particularly valuable in studying the effects from and potential mechanism of action for PM exposure as the issue of discrepancies between experimental doses and ambient PM in terms of composition and magnitude of administered dose can be resolved. Preliminary results have been reported to show that short-term exposure (6 hours) to concentration of ambient particles (fine particles of 0.1-2.5 μm diameter), which are 30 times that of normal air (300 - 4 $\mu g/m^3$) , produce no effects on mortality or morbidity parameters in healthy animals (Syrian Hamsters). Rats with monocrotyline-induced pulmonary vascular/inflammation (Costa et al., 1994; White and Roth, 1989) or chronic bronchitis, as well as their appropriate controls, also received similar exposure to concentrated ambient particles for three days. Death was reported to occur during the exposure without visible change in behavior and also overnight most significantly in animals with chronic bronchitis exposed to the concentrated ambient particles. All animals were reported to exhibit inflammation, however animals with chronic bronchitis exposed to the concentrated ambient particles also exhibited significant broncho-constriction. Animals with bronchitis who died

displayed the most pathological evidence of broncho-constriction followed by survivors with bronchitis, and finally animals dying in the monocrotyline treatment group. In these studies, highest mortality was reported to be correlated with inflammation plus broncho-constriction. Thus, it has been hypothesized that airway responses together with preexisting inflammation play a role in the observed mortality in humans after increases in PM exposure. This hypothesis is consistent with the findings in the affected cattle in the London episode and the profile of susceptibility described in the epidemiological literature describing acute mortality. These findings also support the hypothesis that ambient particle share a different physico-chemical composition than artificial particles used in human clinical and animal experimental work. Therefore, it may be that multiple components of PM or other pollutants in the ambient atmosphere must be present together in order for the full potential of PM toxicity to be expressed.

d. Particle Accumulation

Another hypothesis for the mechanism of PM effects involves particle accumulation of large lung burdens of poorly soluble particles. 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 particle overload can induce acute mortality, it may be a factor for former and current smokers among the elderly. It may also be a factor for the elderly who have been chronically exposed to PM in the work place or those who have resided in heavily industrialized cities before effective control of PM (CD, Chapter 13). Populations with prior exposure to large particle concentrations such as smokers, workers exposed to high particle levels, or those living in highly industrialized cities with a history of numerous increases in ambient particle concentration have increased risk for mortality from PM exposure (CD, Chapter 12). Therefore, while available evidence does not support the mere accumulation of large burdens of PM in the lung as a mechanism for reported PM effects, it is plausible that increased particle burdens from past exposure could further augment the insult from recent increases in ambient particle concentration. However, the mechanism by which prior exposure to particulate could predispose an individual to acute PM effects is unknown.

e. Impaired Respiratory Defense

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 mucocilliary transport and macrophage function are critical to host defense against inhaled pathogens. Increased risk of infection has been associated with changes in mucocilliary 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, Chapter 11, see below). 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 mucocilliary 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 below in section V.C).

B. Sensitive Subpopulations

There are groups within the total population that consistently show susceptibility to adverse health effects from PM exposure. These groups are the same as those which succumbed to air pollution during "catastrophic" historical episodes and which are most susceptible to effects during routine fluctuations in PM. They are described below in Table V-2.

TABLE V-2: SENSITIVE POPULATION SUBGROUPS

Individuals with Chronic Obstructive Pulmonary Diseases (COPD): Asthma Bronchitis Bronchiectasis Emphysema
Individuals with Cardiovascular Disease
Individuals with Infections: Influenza Pneumonia
Elderly
Children
Smokers
Mouth or Oral-nasal Breathers

A discussion of sensitive subpopulations and their occurrence in the general population is described in Table 5-3 of U.S. EPA (1982b). This discussion focuses on characteristics of sensitive subpopulations identified above to be most at risk for adverse health effects from PM exposure and how those characteristics support the plausibility of

observed effects from epidemiologic studies. Such sub-populations may experience effects at lower levels of PM than the general population and the subsequent magnitude of effect may be greater. Enhanced susceptibility may be due to differences in dosimetry, tissue sensitivity, or both.

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.

COPD is the most common pulmonary cause of death (fourth leading cause of death overall) and is a major cause of disability. COPD incidence increases with age of the population (CD, Chapter 11). Patients with COPD have a larger relative risk of mortality from PM exposure than the general population (CD, Chapter 12, see below). COPD is characterized by airway obstruction in which there is increased resistance to airflow during forced expiration. Airway obstruction is seen with such conditions as chronic bronchitis, bronchiectasis (irreversible focal bronchial dilatation), emphysema, asthma, and bronchiolitis. Two other forms of COPD, emphysema and chronic bronchitis, may result in chronic inflammation of distal airways as well as destruction of the lung parenchyma (CD, Chapter 13). In addition, COPD causes a reduction of ventilatory reserves which may be expected to predispose such patients to affecters of pulmonary function. COPD patients may also have hyper-responsiveness to physical and chemical stimuli, altered distribution of PM resulting in greater concentrations of PM in well ventilated areas, and impaired host defense mechanisms as identified through increased rates of respiratory infection. Rates of regional clearance appear to be reduced in humans with COPD (CD, Chapter 11).

Asthma is a particular form of COPD. There are approximately 13 million people in the U.S. with asthma and that number is increasing (National Center for Health Statistics, 1994). In addition, mortality from asthma has been rising in recent years (Gergen and Weis, 1992) and air pollution has been implicated as a causative factor (CD, Chapter 11). Asthma is a lung disease characterized by (1) airways obstruction that is reversible, but not so

completely 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, Chapter 13). [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. Asthmatics have been shown to have increased deposition of PM after bronchoconstriction. A similar pattern is observed in subjects with other forms of COPD. The sputum of asthmatics has a low pH indicative of a potential loss of buffering of acidic particles (CD, Chapter 13).

Physical findings in COPD are highly variable especially in early stages (chest x-ray findings, cough and sputum production, and wheezing all vary in character and intensity). Small airway disease can also be extensive yet not appreciably affect spirometric pulmonary function tests (FEV_1). Therefore, the use of physical findings to accurately gage the effects of changes of ambient air concentrations of PM is problematic. Accordingly, there may be difficulty in detecting effects from particles through the use of pulmonary function tests, such as the FEV_1.

There appears to be increased risk from death and morbidity (increased hospital admissions) due to cardiovascular causes associated with exposure to increased PM concentration. Bates (1992) has postulated three ways in which pollutants could affect cardiovascular mortality statistics: (1) acute airways disease misdiagnosed as pulmonary edema, (2) increased lung permeability, leading to pulmonary edema in people with underlying heart disease and increased left atrial pressure, and (3) acute bronchiolitis or pneumonia induced by air pollutants precipitating congestive heart failure in those with pre-existing heart disease (CD, Chapter 11). Patients with COPD, a subpopulation already identified as being at increased risk from PM effects, have a tendency to have arrhythmias (irregularity of the heart beat). In addition, enlarged airspaces have been hypothesized to increase blood flow resistance through the pulmonary capillary network thereby increasing

cardiac stress (U.S. EPA, 1982b). As with COPD, the preexisting condition of ischemic heart disease occurs at high frequency in the general population and contributes significantly to total mortality. The pathophysiology of many lung diseases is related to cardiac function. Specifically, increased hospital admissions for ischemic heart disease and congestive heart failure associated with PM may result from exacerbation of cardiovascular disease through respiratory effects (Schwartz and Morris, in press). The inability to adequately perfuse tissues is central to the pathophysiology of both myocardial infarction and congestive heart failure. The diminished ability of lungs to oxygenate blood (e.g., through airway obstruction and bronchoconstriction associated with COPD or with aging), as well as pulmonary edema and increased left atrial pressure for those with pre-existing cardiovascular disease may increase demands on the myocardium and thus precipitate ischemic cardiac events or congestive heart failure in susceptible individuals. Results from animal studies suggest that pulmonary hypertension or inflammation may increase susceptibility to effects of particles on the lungs (Costa et al., 1994). In addition, terminal events in patients with end stage COPD are often cardiac in nature and may be recorded as a cardiovascular death rather than as a respiratory cause.

2. Individuals with Infections

Controlled exposures of individuals with influenza to high concentrations of ammonium nitrate induced increased sensitivity of the respiratory epithelium as compared to uninfected subjects (Utell et al., 1980). Respiratory infection is also a risk factor for exacerbation of asthma and increased susceptibility of cardiopulmonary patients to stress (Fishman, 1976). Increased infant mortality has been reported from pneumonia concurrent with increased PM in Rio De Janeiro, Brazil (Penna and Duchiade, 1991). Frequently the immediate cause of death in persons compromised by heart and lung diseases is pneumonia or other respiratory infection (Samet et al., 1995). Consequently, because of the interrelatedness of these diseases the specific causes of death may not always be readily discernible (Samet et al., 1995).

3. The Elderly

There is currently little information on how aging in the absence of pathology might make the elderly more susceptible to the effects of ambient particles (Cooper et al., 1991). There are, however, decreases in pulmonary functions associated with aging that may decrease the capacity of the elderly to withstand respiratory insult. Older people already have decreased oxygen exchange capacity. The PaO_2 (partial pressure for arteriolar oxygen) for a healthy 20-year old breathing room air is about 90 mmHG whereas the normal PaO_2 at age 70 is about 75 mmHg. This physiologic decrease with age is partly the result of a decrease in lung elastic recoil (e.g., senile emphysema) leading to closure of small airways in the tidal volume range. Increasing age is accompanied by decreases in lung volume, FEV_1, flow velocity/volume curves, resting cardiac output, and cardiac output reserve (Kenny, 1989). In addition, little is known about possible interactions between numerous medications that the elderly typically take and exposure to ambient pollutants which may potentiate adverse effects. In regard to animal studies, older animals as well as those with chronic illness have been reported to have a more limited ability to adapt to stressors which may include air pollution (CD, Chapter 11).

4. Children

Children have the potential to be inherently more susceptible to the effects of PM as they show a greater incidence of asthma and decreased immunological protection (CD, Chapter 13). For those who are under 20 years of age, asthma rates have increased approximately 45 percent from 1980 to 1987 (50 per 1000 persons) (NIH, 1991). In addition, children may spend more time outdoors at higher ventilation rates via increased activity and have subsequent increased inhalation of outdoor pollutants. The changes in dosimetry between children and adults are discussed below in section V.C. Infants in particular have been hypothesized to be a susceptible 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). Recent studies in North America have not found increased mortality or morbidity in infants, although they were reportedly at increased risk of mortality in the London air pollution episode of 1952 and,

more recently, to be at greater risk of mortality from pneumonia with elevated particulate pollution levels in Rio De Janeiro (Penna and Duchiade, 1991).

5. Smokers

As discussed in the CD, smoking has been identified as a key etiologic factor in primary risk factors discussed above (e.g., COPD, increased risk for cardiovascular disease, and respiratory infection) for PM effects. Environmental tobacco smoke (ETS) has been shown to increase the risk to children of lower respiratory tract infections (bronchitis and pneumonia) and increased frequency and severity of asthma exacerbations. These are also risk factors of adverse heath effects from increased PM exposure.

6. Mouth or Oronasal Breathers

Although not necessarily a readily identifiable group distinct from those above, dosimetric considerations indicate that individuals who, due to disease, increased activity, or other reason habitually breathe through the mouth are at increased risk due to increased penetration of both fine and coarse particles. Approximately 15 percent of the population may fall into this category (Niinimaa et al., 1980; U.S. EPA, 1994c).

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 updates and expands upon findings presented in the previous Staff Paper (U.S. EPA, 1982b, 1986b). It identifies and describes the principal health effects associated with PM, emphasizing the substantial amount of recent information most pertinent to the review of the current PM standards. Evidence for such associations drawn from epidemiological studies, controlled human exposures, and animal toxicology is discussed and evaluated in the Criteria Document (CD) and below. Based on the scientific information discussed and evaluated in the CD and in this staff paper, the key health categories associated with PM are listed in Table V-3.

TABLE V-3. PARTICULATE MATTER EFFECTS OF CONCERN
Increased Mortality
Aggravation of Existing Respiratory and Cardiovascular Disease Hospital Admissions and Emergency Department Visits School Absences Work Loss Days Restricted Activity Days
Respiratory Mechanics and Symptoms
Altered Clearance and Other Host Defense Mechanisms
Morphological Damage
Cancer

The majority of effects listed above have been consistently associated with PM exposure from a large body of community epidemiological evidence. The strengths and weakness of epidemiological studies in general are discussed in some detail in the CD, and outlined briefly below. Particularly important issues concerning uncertainties in the recent community studies of the health effects PM are presented in section V.D of this chapter. Epidemiological studies identify site-, time-, and monitor-specific associations of incidence of diseases or effects and risk factors; they do not demonstrate causality or provide clear evidence of the mechanisms of such diseases or effects. Specifically, the community epidemiological studies focus on showing whether associations exist, rather than on how they might be explained at a pathogenic or mechanistic level. Experimental work in laboratory animals and humans also helps to generate data from which to develop hypotheses concerning mechanisms of PM effect which can in turn aid in the design of epidemiological studies. If the exact pathogenic mechanisms are not known, however, as is the case of cardiovascular disease and cigarette smoking, well-conducted, consistent, and coherent epidemiology studies

may still provide strong evidence of effects. Nevertheless, because groups rather than individuals are studied, uncertainties regarding cause and effect are increased. Consequently, a variety of approaches and analyses are applied to assess whether chance could have determined the findings. An advantage of such studies is that the population of concern (humans) is examined under realistic exposure conditions. Additionally, large number of subjects with a range of susceptibilities may be observed.

Qualitative support for some of these epidemiologic observations has been reported for specific components of the ambient particle milieu 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 continuum of changes including: respiratory symptoms such as wheeze and coughing, changes in pulmonary function, altered mucocillary clearance, inflammation, changes in lung morphology and tumor formation (CD, Chapter 11). 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.

Typically, experimental animal toxicology studies are designed to develop information for understanding the mechanistic steps following particle deposition and health effects from specific constituent(s) of the PM milieu. In the case of the study of the effects of PM, there are several difficulties in using human clinical studies and experimental work in animals to elucidate mechanisms of effects from PM exposure. These limitations hinder the interpretation of this body of work with regard to determining either the risk of adverse effects from particles in humans or in determining the mechanism of action of particles at ambient levels. However, these studies do illustrate the potential for particles to cause adverse effects and aid in development of proposed mechanisms for observed PM effects. For example, this information shows that the site of respiratory tract deposition (and hence particle size) clearly influences health outcome and that toxicity can vary greatly by chemical species (e.g., cadmium toxicity differs from that of sulfuric acid).

A more complete discussion of the interpretation of such work can be found in Appendix D and includes difficulties in reproducing the effects of PM in an appropriate

animal model given the (a) numbers of individuals affected, (b) lack of distinct disease pathology, (c) lack of appropriate equivalents to epidemiological endpoints, and (d) the heterogeneity of the human population, physico-chemical composition of PM, and dosimetry (within the human population and between humans and animals). In addition, studies using particles generated in the laboratory are probably not an accurate reproduction of the complex physico-chemical characteristics of particles found in the ambient air along with varying amounts of pollutant gases (Sioutas et al., 1995). The lack of an effect of any one of the chemical constituents of PM in experimental systems should always be interpreted with caution given that responses to the differing components of mixtures may be synergistic, additive, or antagonistic, and may vary with particle size and surface characteristics.

Key evidence illustrating each of the major effects categories listed in Table V-3 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

Reports of the effects of ambient PM on health date back to the dramatic pollution episodes of Belgium's industrial Meuse Valley; Donora, Pennsylvania; and London, England. In these cases, winter weather inversions led to very high particle concentrations (Firket, 1931; Ciocco and Thompson, 1961) which were associated with large increases in mortality and morbidity, especially among individuals with preexisting cardio-pulmonary conditions. Analyses of a series of episodes in London indicated excess mortality occurring with abrupt increases in particles, including sulfuric acid, accompanied simultaneously by high levels of SO_2 (U.S. EPA, 1969, p. 154). As noted above, livestock were also severely affected. In studies of these episodes, indicators of PM such as British Smoke (BS) were used which preferentially measures carbonaceous particles found in the fine fraction, as discussed in Chapter IV.

During the review of the PM standards that culminated in 1987, this collection of predominantly episode studies was augmented by several more extensive time-series analyses examining the PM pollution/mortality relationship across 14 London winters. These studies

differed from the original studies by examining the mortality/PM relationship using more sophisticated statistical techniques to examine mortality during routine variations in PM and sulfur dioxide levels. These analyses showed a continuum of response across PM levels and suggested 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 with air pollution than SO_2 (Mazumdar and Sussman, 1981).

ii. Recent Findings

Beginning in 1987, two important developments took place. Investigators started to use more sophisticated statistical techniques, originally based on econometric techniques, which allowed the evaluation of the association between short-term variations in PM and mortality. Secondly, more information on PM levels became available in cities throughout the U.S., through implementation of extensive monitoring networks, which allowed studies of short-term PM levels and health effects. Since then numerous epidemiological studies have reported an association between short-term exposures to PM and mortality. In these studies, investigators have observed an association between daily or several day averages in concentration of PM (as TSP, PM_{10}, or $PM_{2.5}$) and mortality in communities across the U.S. and in several locations outside the U.S.. Of 29 studies published between 1988 and 1995, 25 have found a positive association between increases in ambient PM concentration and mortality (Appendix E). These studies are consistent with the earlier analyses of the 14 London winters, but extend to lower concentrations and a large number of areas with differing climate, particle composition, and varying amounts of SO_2 and other gaseous pollutants.

A summary of the studies using a variety of PM indicators which the CD concluded were most appropriate for quantitative assessments are presented in Table V-4. These studies have reported a consistent association between changes in PM levels and mortality, finding a 1.5 percent to 19.0 percent increase in daily mortality associated with a 50 $\mu g/m^3$ increase of PM concentrations (Table V-4). These studies have been conducted in a number of different geographic locations in North America. Two in the west (Utah Valley and Los

TABLE V-4. EPIDEMIOLOGICAL STUDIES OF SHORT-TERM PM EXPOSURE MORTALITY STUDIES: COMPARISON OF RELATIVE RISK (RR) ESTIMATES FOR TOTAL MORTALITY FROM 50 $\mu g/m^3$ CHANGE IN PM_{10}

Study Location	Reference	PM_{10} ($\mu g/m^3$) Mean	PM_{10} ($\mu g/m^3$) Maximum	Other Pollutants In Model	Lag Times, d	RR per 50 $\mu g/m^3$	95 Percent Confidence Interval
Athens, Greece	Touloumi et al. (1994)	78	306	None	1 d	1.034	(1.025, 1.044)
				SO_2, CO	1 d	1.015	(1.00, 1.03)
Birmingham, AL	Schwartz (1993)	48	163	None	$\leq$ 3 d	1.05	(1.01, 1.10)
Chicago, IL	Ito et al. (1995)	38	128	O_3, CO	$\leq$ 3 d	1.025	(1.005, 1.05)
Chicago, IL	Styer et al. (1995)	37	365	None	3 d	1.04	(1.00, 1.08)
Kingston, TN	Dockery et al. (1992)	30	67	None	$\leq$ 3 d	1.085	(0.94, 1.25)
				O_3	$\leq$ 3 d	1.09	(0.94, 1.26)
Los Angeles, CA	Kinney et al. (1995)	58	177	None	1 d	1.025	(1.00, 1.055)
				O_3, CO	1 d	1.017	(0.99, 1.036)
Santiago, Chile	Ostro et al. (1995a)	115	367	None	1 d	1.04	(1.035, 1.06)
				None	$\leq$ 4 d	1.07	(1.04, 1.10)
				None, Poisson	1 d	1.022[a]	(1.003, 1.042)
				SO_2, Poisson	1 d	1.026[a]	(1.005, 1.047)
				NO_2, Poisson	1 d	1.043[a]	(1.020, 1.066)
				O_3, Poisson	1 d	1.026[a]	(1.005, 1.047)
St. Louis, MO	Dockery et al. (1992)	28	97	None	$\leq$ 3 d	1.08	(1.005, 1.15)
				O_3	$\leq$ 3 d	1.06	(0.98, 1.15)
Toronto, ON Canada	Ozkaynak et al. (1994)	40	96	None	0 d	1.025	(1.015, 1.034)
Utah Valley, UT	Pope et al. (1992)	47	297	None	$\leq$ 4 d	1.08	(1.05, 1.11)
				None, winter	$\leq$ 4 d	1.085	(1.03, 1.35)
				None, summer	$\leq$ 4 d	1.11	(0.92, 1.35)
				Max O_3, summer	$\leq$ 4 d	1.19	(0.96, 1.47)
				Avg O_3, summer	$\leq$ 4 d	1.14	(0.92, 1.41)

Relative risk calculated from parameters given by author assuming a 50 $\mu g/m^3$ increase in PM_{10} or its equivalent.
[a]Calculated on basis of 50 $\mu g/m^3$ increase, from 50 to 100 $\mu g/m^3$.

Angeles), two in the south (Kingston and Birmingham), one in the north (Toronto), and two in the midwest (Chicago and St. Louis). Each of these locations differ in pollution patterns and weather patterns. For example, Chicago and St. Louis have higher SO_2 levels than Utah Valley or Los Angeles. In addition, Birmingham has a more humid climate than Los Angeles. Yet each study finds an association between increased mortality and PM that is relatively consistent with other studies. It is of note that the coefficient of increased risk implied by the 1952 episode in London (1.06) is consistent with those reported for the current studies (Schwartz et. al., 1994).

Investigators have found a trend towards association between directly measured levels of $PM_{2.5}$ and short-term mortality in seven cities studied to date (Table V-5). The association was statistically significant in four considered individually and in a group of six when considered together (Table V-6). Associations between fine particles and mortality have been found in four other areas using other surrogate indicators for fine particulates. Each of these studies found a statistically significant positive association between surrogates for fine particle mass and mortality.

The Schwartz et al. (in press) study evaluated the relative contribution of different size fractions to risk of excess mortality from PM exposure within one study design. The combined $PM_{2.5}$ results of the Schwartz et al. (in press) study presented in Table V-5 are compared to other particle indicators in Table V-6. In the analysis of all of the cities combined, both PM_{10} and $PM_{2.5}$ were significantly associated with daily mortality, while the coarse fraction mass coefficient was not statistically significant.

iii. Specific Causes of Mortality Associated with PM

Several studies have examined associations between PM level and mortality by cause of death. In these studies, the investigators have reported stronger associations with respiratory and cardiovascular causes of death, and deaths in the elderly (Styer et al., 1995; Ostro, 1995a; Schwartz, 1994a; Pope et al., 1992). Results from these studies suggest that the strongest association of increased risk of mortality from PM are for those individuals with preexisting respiratory conditions. Table V-7 summarizes the relative risks for total mortality and respiratory and cardiovascular causes of death, and mortality among the elderly

TABLE V-5. RECENT EPIDEMIOLOGICAL SHORT-TERM FINE PARTICLE EXPOSURE MORTALITY STUDIES

Study	Fine Particle Indicator	Relative Risk: Per 25 $\mu g/m^3$ $PM_{2.5}$ Increase	Relative Risk: BS, KM, COH or $SO_4^=$ Converted to RR per 50 $\mu g/m^3$ PM_{10}	95% Confidence Interval
Dockery et al., 1992 St. Louis, MO	$PM_{2.5}$	1.04		(1.00 - 1.07)
Dockery et al., 1992 Kingston, TN	$PM_{2.5}$	1.04		(0.97 - 1.12)
Schwartz et al., in press Boston, MA	$PM_{2.5}$	1.06		(1.04 - 1.07)
Schwartz et al., in press Knoxville, TN	$PM_{2.5}$	1.04		(1.01 - 1.07)
Schwartz et al., in press St. Louis, MO	$PM_{2.5}$	1.03		(1.01 - 1.04)
Schwartz et al., in press Steubenville, OH	$PM_{2.5}$	1.03		(1.00 - 1.06)
Schwartz et al., in press Portage, WI	$PM_{2.5}$	1.03		(0.99 - 1.08)
Schwartz et al., in press Topeka, KS	$PM_{2.5}$	1.03		(0.95 - 1.11)
Touloumi et al., 1994 Athens, Greece	BS		1.03	(1.025 - 1.044)
Kinney and Ozkaynak, 1991 Los Angeles, CA	KM		1.02	(1.00 - 1.055)
Fairley, 1990 Santa Clara County, CA	CoH		1.02*	(1.01 - 1.03)
Ozkaynak et al., 1994 Toronto, Canada	$SO_4^=$		1.025	(1.015 - 1.034)

*RR per 1200 CoH

TABLE V-6. SHORT-TERM PM EXPOSURE AND MORTALITY STUDY IN COMBINED SIX-CITY ANALYSIS RELATIVE RISK FOR A 25TH TO 75TH PERCENTILE INCREASE IN ALTERNATIVE MEASURES OF PARTICULATE AIR POLLUTION FROM SCHWARTZ et al., in press.

Particle Measure	25th - 75th Percentile Range	Estimated Increase in Mortality	95% Confidence Interval	T Statistic
PM_{10}	22.1 $\mu g/m^3$	1.9%	(1.2%, 2.5%)	5.73
$PM_{2.5}$	13.7$\mu g/m^3$	2.0%	(1.5%, 2.7%)	7.13
CM*	10.6 $\mu g/m^3$	0.5%	(-0.1%, 1.1%)	1.68
SO_4	5.8 $\mu g/m^3$	1.2%	(0.7%, 1.8%)	4.87
H^+	18.9 nm/m^3	0.2%	(-0.6%, 0.9%)	0.45

*CM = coarse fraction (PM_{10} minus $PM_{2.5}$)

TABLE V-7. COMPARISON OF TOTAL MORTALITY AND CAUSE-SPECIFIC MORTALITY FOR SHORT-TERM EXPOSURE STUDIES

Study	Total Mortality, Relative Risk per 50 $\mu g/m^3$ PM10	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)
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		
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)
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.

for the studies evaluating PM. Respiratory related deaths generally describe presence of acute respiratory illness (e.g., symptoms involving the upper respiratory tract and pneumonia), as well as COPD and pneumoconioses. Studies presented in Table V-7 also suggest that the elderly are at higher risk of mortality from PM exposure (Styer et al., 1995; Ostro et al., 1995a).

iv. Experimental Animal Studies

Studies of short-term exposure to specific components of PM have been conducted in an attempt to reproduce mortality from short-term exposure. The vast majority of such studies have found mortality only at concentrations well above ambient levels, even in sensitive species (e.g., guinea pig) and appear to be of little relevance to the effects observed in humans. Lethality is used as an endpoint via a severe irritation response (e.g., laryngeal or bronchial spasm) in healthy animals. Preliminary findings involving concentrated ambient particles and mortality in animals at lower concentrations are discussed above in section V.B.

b. Mortality From Long-Term Exposures to PM

i. Recent Findings

In the last review, staff evaluated a large number of cross-sectional studies that found associations between mortality and long-term exposures to various indicators of PM. These as well as more recent cross sectional studies are summarized in Table V-8. Staff concluded that such studies provided only suggestive evidence of long-term mortality. Less weight was given to these studies because of a number of unaddressed potential confounders and methodological problems inherent with such ecologic approaches. In the recent literature, however, three prospective cohort studies have reported results that may lend additional support to the earlier results. The results of these studies (Abbey et al., 1991; Dockery et al., 1993; Pope et al., 1995) are presented in Table V-9 and are 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. (Harvard Six City Study). Extensive information was obtained regarding individual level potential confounders such as smoking, social and economic status, and occupation. After adjustment for these co-variates, the authors found several measures of PM, (PM_{10}, $PM_{2.5}$ and sulfates) were significantly associated with increases of mortality.

TABLE V-8. COMMONLY-BASED CROSS-SECTIONAL MORTALITY STUDIES (SINCE 1980)

Source	Health Outcome	Time Period/ No. Units	PM Indicators	PM Mean ($\mu g/m^3$)	PM Range/ (Std. Dev.)	Sites Per City	Mean City Pop.	Model[1] Type	PM Lag Structure	Other Pollutants	Other Factors	Relative Risk[2] at TSP = 100, SO_4 = 15	RR. Confidence Interval	Elasticity
Özkaynak and Thurston (1989) Table VI	Total mortality	1980 98 SMSA	TSP	78	(26)	1	NA	OLS sep.	none	none	Pct. ≥ Age 65, median age,	1.012 TSP	(0.96, 1.06)	0.01
			SO_4	11.1	(3.4)						Pct. nonwhite, pop. density, Pct. poor, pct. w/ 4 yrs college.	1.17 SO_4	(1.09, 1.24)	0.086
Özkaynak and Thurston (1987) Table VII	Total mortality	1980, 38 SMSA	PM_{15}	38	(7.3)	1	NA	OLS sep.	none	none	Same as above.	1.059 PM_{15}	(0.95, 1.16)	0.045
			$PM_{2.5}$	20	(3.8)							1.085 $PM_{2.5}$	(0.96, 1.21)	0.068
Lipfert et al. (1988) Table 24	Total mortality	1980 172-185 cities	Fe,	1.2	(0.61)	1	57,500	OLS sep.	none	none	Pct. ≥ Age 65, birth rate;	1.044 Fe	(1.02-1.07)	0.041
			SO_4	9.5	(3.5)						Pct. Afr.-Amer. pop. density, pct. poor; Pct. pop. change, pct. w/ 4 yrs. college; Pct. Hispanic, adj., cig., sales; Pct. prior res., hard water	1.13 SO_4	(1.06-1.20)	0.071
Lipfert et al. (1988) Table 24	Total mortality	1980 68 cities	PM_{15}	38	(121)	1	57,500	OLS sep.	none	none	Same as above.	1.036 PM_{15}	NS[3]	0.027
			$PM_{2.5}$	18	(6)							1.082 $PM_{2.5}$	NS[3]	0.059
Lipfert et al. (1988) Page 60	Total mortality	1980 122 cities	TSP	88	(29)	1	about 60,000	OLS joint	10 years	none	Pct. ≥ Age 65, birth rate, pct. nonwhite, pop. density, pct. poor, adj. cig. sales, pct. w/ 4 yrs. college	about 1.0	NS[3]	NS
			SO_4	9.0	(1.8)							1.072 SO_4	(1.0, 1.14)	0.037
Lipfert (1993a) Regr. 6.1, 6.2	Mortality from natural causes	1980 149 SMSA	TSP	68	(17)	10.6 (TSP)	928,000	OLS sep.	none	none	Pct. ≥ Age 65, Pct. Afr.-Amer., Pct.	1.038 TSP	(0.97, 1.10)	0.026
			SO_4	9.3	(3.1)						Hispanic, Pct. other nonwhite, pct. poor, pct. pop. change, adj. cig. sales, pct. w/ 4 yrs. college, hard water, heating degr. days pop. density	1.059 SO_4	(0.99, 1.12)	0.037

TABLE V-8 (cont'd). COMMONLY-BASED CROSS-SECTIONAL MORTALITY STUDIES (SINCE 1980)

Source	Health Outcome	Time Period/ No. Units	PM Indicators	PM Mean ($\mu g/m^3$)	PM Range/ (Std. Dev.)	Sites Per City	Mean City Pop.	Model Type	PM Lag Structure	Other Pollutants	Other Factors	Relative Risk[1] at TSP = 100, SO_4 = 15	RR. Confidence Interval	Elasticity
Lipfert (1993) Regr. 13.1, 13.3	Mortality from natural causes	1980 62 SMSA	PM_{15}	38	(29)	1	928,000	OLS sep.	none	none	Same as above	1.036 PM_{15}	(0.98, 1.10)	0.027
			$PM_{2.5}$	18	(4.5)							1.060 $PM_{2.5}$	(0.99, 1.13)	0.043
Lipfert (1993) Regr. 9.1, 9.3	Mortality from natural causes	1980 62 SMSA	TSP	68	(17)	10.6 (TSP)	928,000	Log-linear	none	none	Same as above without other nonwhite, heating degr. days, pop. density	1.066 TSP	(1.006, 1.13)	0.044
			SO_4	9.3	(3.1)							1.021 SO_4	NS	0.012
Lipfert (1993) Regr. 13.5	Major CVD	1980 62 SMSA	SO_4 (IP)	4.3	(2.5)	1	928,000	OLS	none	none	Same as above with other nonwhite, heating degree days, pop. density	1.04 SO_4	NS	0.011
Lipfert (1993) Regr. 12.1	Major CVD	1980 62 SMSA	SO_4 (IP)	4.3	(2.5)	1	928,000	OLS	none	none	Pct. ≥ Age 65, median age, pct. nonwhite, pop. density, pct., poor pct. w/ 4 yrs. college	1.19 SO_4	(1.03, 1.35)	0.054
Lipfert (1993) Regr. 10.3, 10.4	COPD	1980 149 SMSA	non-TSP	56.4	(18)	10.6	928,000	Log-linear	none	none	Pct. ≥ Age 65, pct. Afr.-Amer., Pct. Hispanic, pop. density, pct. poor, adj. cig. sales	1.50 TSP	(1.22, 1.83)	0.23
			TSP	68.5	(17)							1.43 TSP	(1.20, 1.71)	0.25

[1]All regression models used PM indicators one at a time (separate models) except as noted.
[2]At TSP = 100 $\mu g/m^3$, SO_4 = 15 $\mu g/m^3$, corrected for migration.
[3]NS = not statistically significant, confidence limits not available.

TABLE V-9. PROSPECTIVE COHORT MORTALITY STUDIES

Source	Health Outcome	Population	Time Period/ No. Units	PM Indicators	PM Mean ($\mu g/m^3$)	PM Range/ (Std. Dev.)	Sites Per City	Total Deaths	Model Type	PM Lag Structure	Other Pollutants	Other Factors	Relative Risk[1] at SO_4 = 15, PM_{15} = 50, $PM_{2.5}$ = 25	RR. Confidence Interval	Elasticity
Abbey et al. (1991)	Total mortality from disease	Calif. 7th Day Adventist	1977-82 Defined by air monitoring sites	24 h TSP > 200	102	25-175 (annual avg)	NA	845	Cox proportional hazards	10 yrs	none	age, sex, race, smoking, education, airway disease	0.99 TSP[1]	(0.87-1.13)[1]	NS[2]
Dockery et al. (1993) p. 1758	Total mortality	White adult volunteers in 6 U.S. cities[3]	1974-91	PM_{15} $PM_{2.5}$ SO_4	29.9 18 7.6	18-47 11-30 5-13	1	1429	Cox proportional hazards	none	none	age, sex, smoking, education, body mass, occup. exposure hypertension[4], diabetes[4]	1.42 PM_{15} 1.31 $PM_{2.5}$ 1.46 SO_4	(1.16-2.01) (1.11-1.68) (1.16-2.16)	0.25 0.22 0.23
Pope et al. (1995) Table 2	Total mortality	American Cancer Society, adult volunteers in U.S.	1982-89 $PM_{2.5}$ 50 cities SO_4 151 cities	$PM_{2.5}$ SO_4	18.2 11[5]	9-34 4-24	1 1	20,765 38,963	Cox proportional hazard	none	none	age, sex, race, smoking, education, body mass, occup. exposure, alcohol consumption, passive smoking, climate[4]	1.17 $PM_{2.5}$ 1.10 SO_4	(1.09-1.26) (1.06-1.16)	0.117 0.077

[1]For 1,000 h/yr > 200 $\mu g/m^3$.
[2]NS = non significant, confidence limits not shown.
[3]Portage, WI; Topeka, KS; Watertown, MA; Harrisman-Kingston, TN; Steubenville, OH.
[4]Used in other regression analyses not shown in this table.
[5]Value may be affected by filter artifacts.

The adjusted increases in risk (26 percent) between the cities with highest and lowest levels of air pollution were nearly equal for PM_{10}, $PM_{2.5}$ and sulfates.

A similar 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). In this study, the association between multi-year concentrations of sulfates and $PM_{2.5}$ and mortality was evaluated. As in the Six City Study, individual level information 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 increase in mortality and those of sulfate associated with a 15 percent increase in mortality between cities with the least and most polluted air.

A third prospective cohort study was also conducted in California by Abbey et al., 1991 (California Seventh Day Adventist Study), which did not find a significant association between total mortality and TSP. However, this study has less statistical power than the other two studies because the California Seventh Day Adventist study reports a smaller number of deaths (60 percent of that reported in the Harvard Six City study and 4 percent in the ACS study). In addition, TSP was used as the measure of exposure, which does not spatially correlate as well as PM_{10} or fine particles.

ii. Specific Causes of Mortality

Both the Harvard Six City and the ACS studies evaluated specific causes of mortality associated with PM as shown in Table V-10. As with the short-term studies, the increase in risk of mortality associated with particle matter was mostly attributed to increases in cardiopulmonary mortality. The Harvard Six City study reported a 37 percent increase in cardiopulmonary mortality associated with $PM_{2.5}$, after adjusting for covariates, between the most polluted and least polluted city. Similarly, the ACS study reported a 31 percent increase in cardiopulmonary mortality associated with $PM_{2.5}$, after adjusting for covariates, between the most polluted and least polluted city.

However, unlike the short-term exposure studies, an association was also found between lung cancer mortality and PM levels, though the results were not always statistically

TABLE V-10. RELATIVE RISK BETWEEN THE MOST POLLUTED AND LEAST POLLUTED CITIES FOR 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 Relative Risk Per 18.6 $\mu g/m^3$ Increase in PM2.5	Non-Smokers Relative Risk Per 18.6 $\mu g/m^3$ Increase in PM2.5	Former Smokers Relative Risk Per 18.6 $\mu g/m^3$ Increase in PM2.5	Current Smokers Relative Risk Per 18.6 $\mu g/m^3$ Increase in PM2.5
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)
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 PM2.5 (Steubenville, OH, average 29.6 $\mu g/m^3$) and the lowest level of PM2.5 (Portage, WI, 11.0 $\mu g/m^3$).

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

Endpoint	Total Population Relative Risk Per 24.5 $\mu g/m^3$ in PM2.5	Non-Smokers Relative Risk Per 24.5 $\mu g/m^3$ in PM2.5	Current and Former Smokers Relative Risk Per 24.5 $\mu g/m^3$ in PM2.5
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 PM2.5 of the 47 cities examined.

significant. The Harvard Six City Study reported a positive but nonsignificant increase in mortality for lung cancer between the most polluted and least polluted cities as measure by $PM_{2.5}$. The ACS study did not find an increase in lung cancer associated with $PM_{2.5}$, but did find a statistically significant 36 percent (95 percent confidence interval of 11 percent to 66 percent) increase in lung cancer mortality between the cities with the highest and lowest levels of sulfates. Both the ACS study and the Harvard Six City study found no other associations between PM levels and causes of mortality other than cardiopulmonary.

These two studies also evaluated the association between PM level and total and cause-specific mortality by smoking status (Table V-10). 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 Harvard Six City Study compared risk of mortality associated with PM for the total population, former smokers, current smokers and nonsmokers. There was a positive association between mortality and fine particles for all categories in the Harvard Six City study, though the association was only statistically significant for the former smokers and current smokers (Table V-10a). In the ACS study, statistically significant positive associations were reported for total and cardiopulmonary mortality for nonsmokers and the population of people who were current or former smokers (Table V-10b). Estimated pollution-related mortality risk was as high for never-smokers as it was for current and former smokers.

c. Extent of Mortality Displacement

Given the inevitability of death, an important consideration is the length of time for which death has been advanced ("mortality displacement" or "prematurity of death") in these studies. Findings of significant prematurity in PM-associated deaths would further heighten concern about exposure to PM. From the description of the sensitive subpopulations for PM exposure, 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 exposure to PM. Such a pattern is often seen for some other environmental insults, such as high temperature (Kalkstein, 1991). Increased mortality has been reported in short-term exposure studies primarily, but not exclusively, among the elderly (i.e., 65 years of age

or older) or individuals with preexisting respiratory disease. However, Schwartz (1994b); Samet et al., (1995); and Bates (1992) also note that the sensitive subpopulations for PM effects could be continually changing as people contract disease and recover. This observation supports the hypothesis that death might be substantially premature if a person becomes seriously ill and without the extra stress of PM would otherwise have recovered. It is plausible that both of these types of life-shortening (i.e., a few days to much longer term mortality displacement) may be observed in the studies because of the heterogeneity of the population studied.

It is very difficult to determine the amount of time death is being advanced. Schwartz (1994c) has reported an increase in sudden deaths for individuals who were not hospitalized on days with high PM levels in Philadelphia. In this case, where it may be assumed that patients with current life-threatening symptoms of disease would be more likely to be in a hospital, it is difficult to be confident that only short-term displacement of mortality is occurring.

Direct evidence from short-term exposure studies concerning the degree of mortality displacement observed is limited. Spix et al., (1993) reported a statistical test for whether PM mortality effects might be affecting those for whom death was imminent. They report some evidence consistent with this hypothesis, but it was not statistically significant. The authors speculate, on the other hand, that PM may also lead to the extra stress that causes the death of a seriously ill person who may have otherwise recovered.

If the effect of PM is only to advance imminent death among particularly sensitive individuals, the excess daily deaths during periods of high PM concentrations would be canceled by subsequent mortality reductions. Thus, in this case, long-term differences in mortality would not be expected to be observed. Pope et al. (1992) reported observable long-term differences in mortality in the Utah Valley for a 44-month period in which particle levels averaged 15 $\mu g/m^3$ higher than an intervening 13-month period of lower PM pollution. The difference observed (3 percent higher mortality in the high PM period) was consistent with that predicted from models of mortality associations with short-term PM exposures, although the observed differences may have related both to short-term and long-term

exposure effects. Because this difference in mortality was observable across a 13-month period, this observation suggests that the associations between PM and mortality in this location are not restricted solely to mortality displacement on the order of a few days or weeks, for which no systematic difference in observed mortality would be expected.

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. Lippmann and Thurston (in press) have suggested that the mean life-shortening from PM exposure from these studies is on the order of two years. Furthermore, they point out that the average life shortening implies that many individuals have lives shortened by many years.

2. Aggravation of Existing Respiratory and Cardiovascular Disease

It is reasonable to anticipate that if associations between PM and mortality are observed, that the same kinds of community based observational studies should find increased morbidity with elevated levels of PM. This is indeed the case. Given the mortality results as well as the earlier mechanistic and sensitive populations discussions, it is also not surprising that the majority of such studies tend to find effects linked to populations with respiratory or cardiovascular disease. Numerous studies have observed associations between PM and responses ranging from severe effects such as increased hospitalization for respiratory and cardiovascular conditions, to moderate exacerbation of respiratory conditions and changes in pulmonary function. The key evidence for such effects is summarized below.

a. Hospital Admissions and Emergency Department Visits

A number of epidemiological studies report a positive association between short-term exposures to PM and hospital admissions for respiratory-related and cardiac diseases. Hospital admissions and emergency room visits for these diseases are an indication of their incidence in the population. Table V-11 is a summary of the results for admissions for all respiratory disease. Tables V-12 to V-14 show studies which investigated associations of PM levels with specific respiratory or cardiovascular diseases such as COPD (emphysema, chronic bronchitis, bronchiectasis, asthma etc,), pneumonia, and heart disease. As with the mortality studies, associations between PM and hospital admissions have been observed in

TABLE V-11. EPIDEMIOLOGICAL STUDIES OF HOSPITAL ADMISSIONS FOR RESPIRATORY DISEASE

Study	PM Indicator	PM Mean & Range	Other Pollutants Measured	Weather & Other Factors	Pollutants in model	Result* (Confidence Interval)
Burnett et al. (1994) All ages in Ontario, Canada, 1983-1988	sulfate	sulfate means ranged from 3.1 to 8.2 $\mu g/m^3$	Ozone	Temperature	ozone	1.03** (1.02, 1.04)
Thurston et al. (1994) All ages in Toronto, Ontario, Canada, July and August, 1986-1988	$PM_{2.5}$, sulfate, PM_{10}, and TSP	mean sulfate ranged 38 to 124 (nmole/m^3), PM_{10} 30 to 39 $\mu g/m^3$, TSP 62 to 87 $\mu g/m^3$	Ozone, H+, SO_2, NO_2	Temperature	none	PM_{10} 1.09 (0.96, 1.22) PM_{10} 1.01 (0.87, 1.15)
Thurston et al. (1992) All ages in Buffalo, Albany, New York City, July and August, 1988-1989	sulfate, H+	mean sulfate ranged 6.9 to 9.6 $\mu g/m^3$, maximum 42 PM_{10}	Ozone, H+	Temperature	ozone	(not given for PM measures)
Schwartz (in press) Elderly in New Haven, CT, 1988-1990	PM_{10}	mean = 41, 10% tile = 19, 90% tile = 67	Ozone, SO_2	Temperature and dew point adjusted for in the moving average	none SO_2 (2 day lag) ozone (2 day lag)	1.06 (1.00, 1.13) 1.07 (1.01, 1.14) 1.09 (1.00 - 1.20)
Schwartz (in press) Elderly in Tacoma, WA, 1988-1990	PM_{10}	mean = 37, 10% tile = 14, 90% tile = 67	Ozone, SO_2	Temperature and dew point adjusted for in the moving average	none SO_2 O_3 (2 day lag)	1.10 (1.03, 1.17) 1.11 (1.02, 1.20) 1.12 (0.97 - 1.29)
Schwartz (in press) Spokane, WA	PM_{10}	mean = 46, 10% tile = 16, 90% tile = 83	Ozone		none	1.085 (1.036 - 1.136)
Pope (1989) All ages in Utah Valley, UT	PM_{10}	mean = 45.8, ranged from 11 to 365	none	Temperature		(statistically significant results (RR) not given)

* Relative risk calculated from parameters given by author assuming a 50 $\mu g/m^3$ increase in PM_{10} or 100 $\mu g/m^3$ increase in TSP.
** Relative risk per 14 $\mu g/m^3$ sulfate.

TABLE V-12. EPIDEMIOLOGICAL STUDIES OF HOSPITAL ADMISSIONS FOR COPD

Study	PM Indicator	PM Mean & Range	Other pollutants measured	Weather & Other Factors	Pollutants in model	Result* (Confidence Interval)
Sunyer et al. (1993) ER admissions 14 years and older in Barcelona, 1985-1989	black smoke	winter 33% tile = 49, 67% tile = 77, summer 33% tile = 36, 67% tile = 55	SO_2	min temp, day of week and year	none	winter: 1.15 (1.09, 1.21) summer: 1.05 (0.98, 1.12)
					SO_2	winter: 1.05 (1.01, 1.09) summer: 1.01 (0.97, 1.05)
Schwartz (1994f) Elderly in Minneapolis, MN, 1986-1989	PM_{10}	mean = 36, 10% tile = 18, 90% tile = 58	Ozone	8 categories of temp. & dew pt., month, year, lin. & quad. time trend	none	1.25 (1.10, 1.44)
Schwartz (1994e) Elderly in Birmingham, AL, 1986-1989	PM_{10}	mean = 45, 10% tile = 19, 90% tile = 77	Ozone	7 categories of temp. & dew pt., month, year, lin. & quad. time trend		1.13 (1.04 - 1.22)
Schwartz (1994d) Elderly in Detroit, MI	PM_{10}	mean = 48, 10% tile = 22, 90% tile = 82	Ozone	Temp., month, lin. & quad. time trend	Ozone	1.11 (1.04 - 1.17)

* Relative risk calculated from parameters given by author assuming a 50 $\mu g/m^3$ increase in PM_{10} or 100 $\mu g/m^3$ increase in TSP.

TABLE V-13. EPIDEMIOLOGICAL STUDIES OF HOSPITAL ADMISSIONS FOR PNEUMONIA

Study	PM Indicator	PM Mean & Range	Other pollutants measured	Weather & Other Factors	Pollutants in model	Result* (Confidence Interval)
Schwartz (1994f) Elderly in Minneapolis, MN 1986-1989	PM_{10}	mean = 36, 10% tile = 18, 90% tile = 58	Ozone	8 categories of temp. & dew pt., month, year, lin. & quad. time trend	none	1.08 (1.01,1.15)
Schwartz (1994e) Elderly in Birmingham, AL 1986-1989	PM_{10}	mean = 45, 10% tile = 19, 90% tile = 77	Ozone	7 categories of temp. & dew pt., month, year, lin. & quad. time trend	none	1.09 (1.03, 1.15)
Schwartz (1994d) Elderly in Detroit, MI 1986-1989	PM_{10}	mean = 48, 10% tile = 22, 90% tile = 82	Ozone	Temp, month, lin. & quad. time trend	ozone	1.06 (1.02, 1.10)

* Relative risk calculated from parameters given by author assuming a 50 $\mu g/m^3$ increase in PM_{10} or 100 $\mu g/m^3$ increase in TSP.

TABLE V-14. EPIDEMIOLOGICAL STUDIES OF HOSPITAL ADMISSIONS FOR HEART DISEASE

Study	PM Indicator	PM Mean & Range	Other pollutants measured	Weather & Other Factors	Pollutants in model	Result* (Confidence Interval)
Schwartz and Morris (in press) Elderly in Detroit, MI 1986-1989 Ischemic Heart Disease	PM_{10}	mean = 48, 10% tile = 22, 90% tile = 82	SO_2, CO, ozone	Temp, month, lin. & quad. time trend	none	1.06 (1.02, 1.10)
					ozone, CO, SO_2	1.06 (1.02, 1.10)
Burnett et al. (1995) All ages in Ontario, Canada, 1983-1988 Cardiac disease admission	sulfate	means ranged from 3.0 to 7.7 in the summer and 2.0 and 4.7 in the winter	Ozone	Temperature included in separate analyses by summer and winter	none	1.04 (1.03, 1.06)
					ozone	1.04 (1.03, 1.05)

* Relative risk calculated from parameters given by author assuming a 50 $\mu g/m^3$ increase in PM_{10} or 100 $\mu g/m^3$ increase in TSP.

numerous communities throughout North America (Birmingham, Detroit, Spokane, Tacoma, New Haven, Utah Valley, New York State, Ontario, Canada). Of the 12 studies, 11 of them reported statistically significant, positive associations between PM level and increased risk of admission to the hospital including evaluating cause-specific admissions for respiratory diseases. The studies find a 3-24 percent increase in hospital admissions for respiratory disease associated with a 50 $\mu g/m^3$ increase in PM_{10}. Specifically, studies reported 6-9 percent increase in admissions for pneumonia and an 11-25 percent increase for COPD for the elderly associated with a 50 $\mu g/m^3$ increase in PM. A recent study of hospital admissions for cardiovascular illness (Schwartz and Morris, in press) reported that PM was positively and significantly associated with daily admissions for ischemic heart disease, with SO_2, CO, and ozone making no independent contribution to the effect. In the same study PM and CO showed independent association for congestive heart failure admissions.

When viewed together, these studies demonstrate an association between hospital admissions for respiratory and cardiac causes and PM. Evaluating the cause-specific associations suggests a greater effect on admissions for COPD. These results are consistent with those of the mortality studies, which also found a stronger association between respiratory related mortality and PM than total 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. It is not clear whether the effects result from aggravation of chronic disease (e.g., COPD), acute infection, or non-specific symptomatic effects. Nevertheless, the results of these studies show consistent associations between such measures of morbidity and increasing levels of PM. Ransom and Pope (1992) have reported a statistically significant positive association between PM levels and school absences. Respiratory conditions are the most frequent cause of school absences (CD, Chapter 12). In addition, three other studies report positive significant associations between PM and work loss days and restricted activity days (Ostro, 1983; Ostro and Rothschild, 1989; Ostro, 1987). A study by Ostro and Rothschild (1989) reported positive

significant associations between PM and respiratory-related restricted activity days. All of these studies report a two to four week lag time between elevations on 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., mortality), but PM may elicit effects which are exhibited at a later time. This result is also consistent with the hypothesis of increased susceptibility to infection resulting from exposure to PM.

3. Respiratory Mechanics and Symptoms

Further exploration of the PM/health effect association shows PM is also associated with effects on measures of lung function and 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. Acute Pulmonary Function Changes and Respiratory Symptoms from Short-term Exposures

i. Community Air Pollution Studies

In community epidemiological studies, associations between PM and acute pulmonary function changes are also observed, consistent with respiratory-related effects observed in the previously described mortality and morbidity studies. Table V-15 presents the results of the studies. The functions studied include forced vital capacity (FVC), forced expiratory capacity for one second (FEV_1) and for three quarters of a second ($FEV_{0.75}$), and peak expiratory flow rate (PEFR).

Table V-16 provides a summary of studies reporting acute respiratory disease symptoms associated with short-term PM exposures. These studies found associations between short-term exposures of PM and upper respiratory symptoms (e.g., hoarseness, sore throat), lower respiratory symptoms (chest pain, phlegm, and wheeze), fever, cough, and acute respiratory illness. Eleven of these studies were conducted in children. Four of the studies evaluated respiratory symptoms in all children (Schwartz et al., 1994; Hoek and Brunekreef, 1993; Hoek and Brunkekreef,1995; Schwartz et al., 1991) and all but one found

TABLE V-15. EPIDEMIOLOGICAL STUDIES OF ACUTE PULMONARY FUNCTION CHANGES

Study	PM Indicator	PM Mean & Range ($\mu g/m^3$)	Other Pollutants Measured	Function Examined	Decrease* (Confidence Interval)
ADULTS					
Pope and Kanner (1993) Study of adults in the Utah Valley from 1987 to 1989	PM_{10}	PM_{10} daily mean was 55 and ranged from 1 to 181	Limited monitoring of SO_2, NO_2, and ozone	FEV_1 FVC	29 ml (7,51) 15 ml (-15,45)
CHILDREN					
Koenig et al. (1993) Study of asthmatic and non-asthmatic elementary school children in Seattle, WA in 1989 and 1990	$PM_{2.5}$	$PM_{2.5}$ ranged from 5 to 45	none	Asthmatics FEV_1 FVC Non-asthmatics FEV_1 FVC	 42 ml (12, 73) 45 ml (20, 70) 4 ml (7, 51) 15 ml (-15, 45)
Dockery et al. (1982) School age children in Steubenville, OH, measured at three times between 1978 and 1980	TSP	up to 455	SO_2	FVC $FEV_{0.75}$	8.1 ml 1.8 ml Note: decreases were statistically significant
Neas et al. (1995) Study of 83 children in Uniontown, PA, in the summer of 1990	PM_{10}, $PM_{2.5}$	Mean PM_{10} = 36, max. = 83 Mean $PM_{2.5}$ = 25, max. = 88 $\mu g/m^3$	O_3, SO_2, sulfate, H^+	PEFR per 25 $\mu g/m^3$ $PM_{2.5}$	23.1 (-0.3, 36.9)

*Decreases in lung function calculated from parameters given by author assuming a 50 $\mu g/m^3$ increase in PM_{10} or 100 $\mu g/m^3$ increase in TSP.
FEV_1 Forced expiratory capacity for 1 second.
FVC Forced vital capacity.
PEFR Peak expiratory flow rate.

TABLE V-15. EPIDEMIOLOGICAL STUDIES OF ACUTE PULMONARY FUNCTION CHANGES

Study	PM Indicator	PM Mean & Range ($\mu g/m^3$)	Other Pollutants Measured	Function Examined	Decrease* (Confidence Interval)
Quackenboss et al. (1991) Asthmatic children aged 6 to 15 years in Tucson, AZ, measured in May and November, 1988	$PM_{2.5}$	Not given	NO_2	PEFR	375 ml/s Note: these are diurnal rather than daily changes
Pope et al. (1991) Study of asthmatic children in the Utah Valley	PM_{10}	PM_{10} ranged from 11 to 195	SO_2, NO_2, ozone	PEFR	55 ml/s (24, 86)
Pope and Dockery (1992) Study of non-asthmatic symptomatic and asymptomatic children in the Utah Valley	PM_{10}	PM_{10} ranged from 11 to 195	SO_2, NO_2, ozone	Symptomatic PEFR	30 ml/s (10, 50)
				Asymptomatic PEFR	21 ml/s (4, 38)
Hoek and Brunekreef (1993) Study of children aged 7 to 12 in Wageningen, Netherlands	Black smoke, PM_{10}	range of PM_{10} was 30 to 144	SO_2, NO_2	PEFR	41 ml/s (-8, 90)
Roemer et al. (1993) Study of children with chronic respiratory symptoms in The Netherlands	Black smoke, PM_{10}	range of PM_{10} was 30 to 144	SO_2, NO_2	PEFR	34 ml/s (9, 59)

*Decreases in lung function calculated from parameters given by author assuming a 50 $\mu g/m^3$ increase in PM_{10} or 100 $\mu g/m^3$ increase in TSP.
FEV_1 Forced expiratory capacity for 1 second.
FVC Forced vital capacity.
PEFR Peak expiratory flow rate.

TABLE V-15. EPIDEMIOLOGICAL STUDIES OF ACUTE PULMONARY FUNCTION CHANGES

Study	PM Indicator	PM Mean & Range ($\mu g/m^3$)	Other Pollutants Measured	Function Examined	Decrease* (Confidence Interval)
Dassen et al. (1986) School age children in The Netherlands, measured in November, 1984 and January, 1985	RSP (PM_{10}), TSP	TSP and RSP both exceeded 200	SO_2	FVC FEV_1 PEFR	slopes not given but FVC, FEV_1, and PEFR were significantly reduced during episodes
Studnicka et al. (1995) Study of 133 children at a summer camp in southern Austria in 1991	PM_{10}	Means ranged from 6.6 to 10.7 $\mu g/m^3$	H^+, SO_2, ammonia	FVC FEV_1 PEFR	17.5 ml/s (64,0, 99,0) 66.5 ml/s (-10,0, 143.0) 99 ml/s
Hoek and Brunekreef (1994) Study of children in 4 towns in The Netherlands	No. of sites not given 24-hr PM_{10} measured	PM_{10} mean was 45, range was 14-126 $\mu g/m^3$	SO_2, NO_2, sulfate, nitrate, HONO	FVC FEV_1 PEFR	-0.5 ml/s (-3.5, 2.5) 5.0 ml/s (-1.0, 11.0) 41.0 ml/s (12.5, 69.5)
Dusseldorf et al. (1994) Study of 32 adults in a steel plant in Wijkaan Zee, The Netherlands	PM_{10} measured at 3 sites	PM_{10} mean was 54, range was 4 - 137 $\mu g/m^3$	Iron, Mn, sodium, silicon	PEFR evening PEFR morning	45 ml/s (9, 81) 77 ml/s (34, 119)

*Decreases in lung function calculated from parameters given by author assuming a 50 $\mu g/m^3$ increase in PM_{10} or 100 $\mu g/m^3$ increase in TSP.
FEV_1 Forced expiratory capacity for 1 second.
FVC Forced vital capacity.
PEFR Peak expiratory flow rate.

TABLE V-16. EPIDEMIOLOGICAL STUDIES OF ACUTE RESPIRATORY DISEASE

Study	PM Indicator	PM_{10} ($\mu g/m^3$) Mean	Minimum	Maximum	Other Pollutants Measured	Weather & Other Factors	Other Pollutants in Model	RR per 50 $\mu g/m^3$ increase* (Confidence Interval)
CHILDREN								
Schwartz et al. (1994) 300 elementary school children in Six-Cities in U.S., 1984-1988	PM_{10}	30	13**	53***	Ozone, NO_2, SO_2	Temperature	SO_2, O_3	Cough+ 1.51 (1.12, 2.05)
	$PM_{2.5}$	18	7**	37***				Upper respiratory+ 1.39 (0.97 - 2.01) Lower respiratory+ 2.03 (1.36 - 3.04)
Pope et al. (1991), asthmatic school children (4th, 5th grade) in the Utah Valley, winter 1989-1990	PM_{10}	46	11	195	NO_2, SO_2, and ozone. Values were well below the standard	Variables for temperature and time trend	none	Upper respiratory+ 1.20 (1.03, 1.39) Lower respiratory+ 1.28 (1.06, 1.56)

* Relative risk calculated from parameters given by author assuming a 50 $\mu g/m^3$ increase in PM_{10} or 100 $\mu g/m^3$ increase in TSP.
** 10th percentile.
*** 90th percentile.
**** Odds ratio for increase is shortness of breath for a 56 $\mu g/m^3$ increase in PM_{10}.
\+ Statistically significant with 95 percent confidence interval.

TABLE V-16. EPIDEMIOLOGICAL STUDIES OF ACUTE RESPIRATORY DISEASE

Study	PM Indicator	PM_{10} ($\mu g/m^3$) Mean	Minimum	Maximum	Other Pollutants Measured	Weather & Other Factors	Other Pollutants in Model	RR per 50 $\mu g/m^3$ increase* (Confidence Interval)
Pope and Dockery (1992), symptomatic non-asthmatic children in the Utah Valley, winter 1990-1991	PM_{10}	76	7	251	None	Variable for low temperature	none	Upper respiratory+ 1.20 (1.03, 1.39) Lower respiratory+ 1.27 (1.08, 1.49) Cough+ 1.29 (1.12, 1.48)
						5-Day Moving Average		Upper respiratory 1.30 (1.06, 1.58) Lower respiratory 1.40 (1.14, 1.70) Cough 1.42 (1.17, 1.73)
Pope and Dockery (1992), asymptomatic children in the Utah Valley, winter 1990-1991	PM_{10}	76	7	251	None	Variable for low temperature	none	Upper respiratory 0.99 (0.78, 1.26) Lower respiratory 1.13 (0.91, 1.39) Cough+ 1.18 (1.00, 1.40)
						5-Day Moving Average		Upper respiratory 1.04 (0.75, 1.43) Lower respiratory 1.21 (0.92 - 1.59) Cough 1.35 (1.07 - 1.72)

* Relative risk calculated from parameters given by author assuming a 50 $\mu g/m^3$ increase in PM_{10} or 100 $\mu g/m^3$ increase in TSP.
** 10th percentile.
*** 90th percentile.
**** Odds ratio for increase is shortness of breath for a 56 $\mu g/m^3$ increase in PM_{10}.
\+ Statistically significant with 95 percent confidence interval.

TABLE V-16. EPIDEMIOLOGICAL STUDIES OF ACUTE RESPIRATORY DISEASE

Study	PM Indicator	PM_{10} ($\mu g/m^3$) Mean	Minimum	Maximum	Other Pollutants Measured	Weather & Other Factors	Other Pollutants in Model	RR per 50 $\mu g/m^3$ increase* (Confidence Interval)
Ostro (1995b) Study of 83 African-American Asthmatic Children in Los Angeles, CA	PM_{10}	56	20	101	Ozone, NO_2, SO_2	Temperature, humidity, pollens, molds	ozone	Shortness of breath 1.58 (1.05 - 2.30)**** No effect on cough or wheeze
Hoek and Brunekreef (1993), respiratory disease in school children aged 7 to 12 in Wageningen, Netherlands, winter 1990-1991	PM_{10}	N/A	N/A	110	Max SO_2 = 105 $\mu g/m^3$, max NO_2 = 127 $\mu g/m^3$	Variable for ambient temperature and day of study	none	Upper respiratory+ 1.14 (1.00, 1.29) Lower respiratory 1.06 (0.86, 1.32) Cough 0.98 (0.86, 1.11)
Schwartz et al. (1991) Study of acute respiratory illness in children in 5 German communities, 1983-1985	TSP	17-56	5-34**	41-118***	median SO_2 levels ranged from 9 to 48 $\mu g/m^3$, median NO_2 levels ranged from 14 to 5 $\mu g/m^3$	Most significant terms of day of week, time trend, and weather (terms not listed)	none (TSP was not significant when NO_2 added to model)	1.26 (1.12, 1.42)+
Braun-Fahrlander et al. (1992) Study of preschool children in four areas of Switzerland	TSP	(not given)			SO_2, NO_2, and ozone levels not given	city, risk strata, season, temperature (not given)	none	Upper respiratory+ 1.55 (1.10, 2.24)

* Relative risk calculated from parameters given by author assuming a 50 $\mu g/m^3$ increase in PM_{10} or 100 $\mu g/m^3$ increase in TSP.
** 10th percentile.
*** 90th percentile.
**** Odds ratio for increase is shortness of breath for a 56 $\mu g/m^3$ increase in PM_{10}.
\+ Statistically significant with 95 percent confidence interval.

TABLE V-16. EPIDEMIOLOGICAL STUDIES OF ACUTE RESPIRATORY DISEASE

Study	PM Indicator	PM_{10} ($\mu g/m^3$) Mean	Minimum	Maximum	Other Pollutants Measured	Weather & Other Factors	Other Pollutants in Model	RR per 50 $\mu g/m^3$ increase* (Confidence Interval)
Roemer et al. (1993) Study of children with chronic respiratory symptoms in Wageningen, The Netherlands	PM_{10}	6 days above 110 $\mu g/m^3$			SO_2 and NO_2 means not given	(not given)	none	Cough (not given)
Hoek & Brunekreef (1995) Study of respiratory symptoms in 300 children in Deane & Enkhulzen, The Netherlands	PM_{10}, sulfates	48 36	13 11	124 136	ozone, nitrate	trend, day of week, humidity	none	Upper respiratory symptoms Logistic regression coefficient -0.0014 (-0.0032 - 0.0004) Similar results for lower respiratory symptoms
ADULTS								
Ostro et al. (1991) Study of adult asthmatics in Denver, Colorado November 1987 to February 1988	$PM_{2.5}$	22	0.5	73	nitric acid, sulfates, nitrates, SO_2, and hydrogen ion	day of survey, day of week, gas stove, minimum temperature	none	Cough 1.09 (0.57, 2.10)

* Relative risk calculated from parameters given by author assuming a 50 $\mu g/m^3$ increase in PM_{10} or 100 $\mu g/m^3$ increase in TSP.
** 10th percentile.
*** 90th percentile.
**** Odds ratio for increase is shortness of breath for a 56 $\mu g/m^3$ increase in PM_{10}.
\+ Statistically significant with 95 percent confidence interval.

TABLE V-16. EPIDEMIOLOGICAL STUDIES OF ACUTE RESPIRATORY DISEASE

Study	PM Indicator	PM_{10} ($\mu g/m^3$) Mean	Minimum	Maximum	Other Pollutants Measured	Weather & Other Factors	Other Pollutants in Model	RR per 50 $\mu g/m^3$ increase* (Confidence Interval)
Pope et al. (1991), asthmatics age 8-72 in the Utah Valley, winter 1989-1990	PM_{10}	46	11	195	NO_2, SO_2, and ozone. Values were well below the standard	Variables for low temperature and time trend	none	Upper respiratory 0.99 (0.81, 1.22) Lower respiratory 1.01 (0.81, 1.27)
Ostro et al. (1993) Study of non-smoking adults in Southern California	sulfate fraction and COH	8	2	37	ozone, mean = 7 pphm, range = 1 to 28	temperature, rain humidity	none	Sulfates: Upper respiratory 0.91 (0.73, 1.15) Lower respiratory 1.48 (1.14, 1.91)
Dusseldorf et al. (1994) Study of adults near a steel mill in The Netherlands	PM_{10}, iron, sodium, silicon, and manganese	54	4	137	Geometric mean iron = 501 ng/m^3, manganese = 17 ng/m^3, silicon = 208 ng/m^3	(not given)	none	Cough+ 1.14 (0.98, 1.33)

* Relative risk calculated from parameters given by author assuming a 50 $\mu g/m^3$ increase in PM_{10} or 100 $\mu g/m^3$ increase in TSP.
** 10th percentile.
*** 90th percentile.
**** Odds ratio for increase is shortness of breath for a 56 $\mu g/m^3$ increase in PM_{10}.
\+ Statistically significant with 95 percent confidence interval.

positive significant associations with PM with one or more symptoms. Two of the studies evaluated respiratory symptoms in asthmatic children (Pope et al., 1991, Ostro, 1995b) and found significant positive associations with PM, although in the Ostro (1995b) study, the effect could not be separated from ozone. A study of symptomatic and asymptomatic children found significant positive associations with all symptoms in the symptomatic children, and positive, but not significant associations in the asymptomatic children (Pope and Dockery, 1992). The four studies in adults were inconsistent. Therefore, these studies suggest that vulnerable individuals, such as symptomatic children or children with asthma, may have aggravation of symptoms associated with PM.

ii. Controlled Exposures to Laboratory Aerosols

In general, bronchoconstriction and associated symptoms may be induced by chemical or mechanical irritation by inert dusts, re-suspended urban dust, coarse organic dusts, fine acid aerosols, and fine particles in combination with pollutant gases (U.S. EPA, 1982b). Specifically, exposure to high levels of re-suspended dust consisting of dust particles ranging in size from 0.5-10 μm diameter with a composition consisting of crustal material, sulfates, and volatile material has also been shown to induce bronchoconstriction (U.S. EPA, 1982a).

In controlled human clinical and animal studies, acidic aerosols have been a primary focus of research of PM effects. Table 11.2 of the CD provides a summary of the controlled human exposures to acid aerosols and other particles and shows acid aerosol effects from short-term exposures (less than 24 hours). Most of the human clinical studies examine the effect on lung function from exposure to H_2SO_4 aerosols. The endpoints most commonly measured are symptoms and pulmonary function tests which are well standardized in regard to their use in these studies (Utell et al., 1993). The advantages of such studies include the opportunity to study the species of interest (humans), and the ability to control exposure in regard to pollutant concentration, aerosol characteristics, temperature, and relative humidity (CD, Chapter 11).

In these studies healthy young subjects seem to tolerate relatively large concentrations of H_2SO_4 particles without deleterious effect; whereas, asthmatics experience decrements in lung function at relatively low concentrations. Asthma severity in studies of acid aerosols

and other particles are presented in Table 11-3 of the CD. 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, Chapter 11). 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 only slightly above peak ambient levels (e.g., less than 100 $\mu g/m^3$) (Koenig et al., 1989; CD, Chapter 11). Lung function effects in asthmatic subjects are correlated with hydrogen ion content of the sulfate aerosol (CD, Chapter 11) and affected by neutralization by oral ammonia (Utell et al., 1983b; 1989) and buffering capacity of the aerosol (Fine et al., 1987b). In addition, results from recent studies have suggested aerosols of submicron particle size may alter lung function to a greater degree than large particle aerosols in asthmatic subjects (CD, Chapter 11; Avol et al., 1988a,b,; Utell et al., 1983b) albeit at larger concentrations than found to affect adolescent asthmatics. Human studies have also suggested potentiation of effects (airway responsiveness) of H_2SO_4 aerosol exposure with ozone exposure (Linn et al., 1994; Frampton et al., 1995; CD, Chapter 11).

However, studies cited in an Acid Aerosols Issue Paper (U.S. EPA, 1989) have not demonstrated synergistic or interactive effects between sulfates and SO_2 exposure (CD, Chapter 11). Indeed, given the low solubility of SO_2 in acid aerosol, significant interactions would not be anticipated in the deeper regions of the lungs, to which SO_2 alone has difficulty penetrating (U.S. EPA, 1994c). Reflex bronchoconstriction by high peaks of SO_2 could, however, increase the deposition of particles in the tracheobronchial region by narrowing the conductive airways.

As described in the CD, human studies of PM are limited as they tend to use pulmonary function as the endpoint of response and do not examine airway inflammation or other more sensitive indicators related to pulmonary function changes. There are also limits as to the kind of experiments which can be done using human subjects as exposures must be without significant harm.

Many laboratory animal studies have also been conducted using acid aerosol exposures with the most recent studies on effects on pulmonary mechanical function

presented in Table 11-4 of the CD. Some other earlier work is also presented. The CD suggests that acidic sulfates exert their action throughout the respiratory tract with response and location of effect dependent on particle size, mass, and number concentration (CD, Chapter 13). In regard to the particle size, the CD suggests, that at high concentrations which are above lethal threshold, large particles are more effective in eliciting response, while at sublethal levels, smaller particles are more effective (CD, Chapter 11). Issues of dosimetry and use of an inappropriate dose metric hinder interpretation of such work (see Appendix D for further discussion).

In contrast to mortality as an endpoint, the dyspneic response in a sensitive sub-population of guinea pigs to relatively lower concentrations of H_2SO_4 is similar to the asthma response in humans (e.g., rapid onset with similar obstructive lung function changes) (CD, Chapter 11). In addition, in controlled animal experiments, both acute and chronic exposure of laboratory animals to H_2SO_4 at exposure well below lethal ones have been shown to produce functional changes in the respiratory tract (CD, Chapter 11) and, therefore, whatever the underlying mechanism, the results of pulmonary function studies indicate that H_2SO_4 is a broncho-active agent.

b. Long-Term Exposure to PM

i. Chronic Pulmonary Function Changes

A number of epidemiological studies investigated the association between long-term exposure to PM and pulmonary function change (Table V-17). The results are equivocal with three studies reporting no association and one finding a decrease in lung function associated with increasing PM levels. In regard to experimental animal studies, long-term exposure to H_2SO_4 is also associated with alteration in pulmonary function (changes in distribution of ventilation and respiratory rates in monkeys) albeit at high concentration (CD, Chapter 11). In addition hyper-responsive airways have been induced with repeated exposures to 250 $\mu g/m^3$ H_2SO_4 in rabbits (CD, Chapter 11).

ii. Chronic Respiratory Disease

Table V-18 summarizes studies investigating the association between chronic respiratory disease and long-term exposure to PM. Four of the studies reported an

TABLE V-17. EPIDEMIOLOGICAL STUDIES OF CHRONIC PULMONARY FUNCTION CHANGES

Study	PM Type & No. Sites	PM Mean & Range	Other Pollutants Measured	Weather & Other Factors	Decrease* (Confidence Interval)
CHILDREN					
Neas et al. (1994) Study of lung function in children in 6 cities in the U.S. Data collected from 1983-1988.	$PM_{2.5}$, sulfate fraction	Not given	SO_2, NO_2, and ozone	City, gender parental education, history of asthma, age, height, weight	FVC and FEV_1 not changed. Values could not be converted to mls.
Dockery et al. (1989) Study of lung function in children in 6 cities in the U.S. Survey done 1980-1981.	PM_{15}, sulfate fraction	PM_{15} means ranged from 20 to 59 $\mu g/m^3$	SO_2, NO_2	City, gender, parental education, history of asthma, age, height, weight	No significant relationship found with PM_{10}
Ware et al. (1984) Study of lung function in children in 6 cities in the U.S. Survey done 1974-1977	TSP	TSP means ranged from 39 to 114 $\mu g/m^3$	SO_2, NO_2	City, gender, parental education, history of asthma, age, height, weight	Non-significant changes of 0.06% (-0.27, 0.39) for first round and -.09% (-0.42, 0.24) for second round
ADULTS					
Ackermann-Liebrich et al. (in press) Study of 9,651 adults in 8 areas of Switzerland done in 1991	TSP, PM_{10}	PM_{10} mean was 21.2, ranged from 10.1 to 33.4	SO_2, NO_2, TSP, O_3	Height, weight, age, gender, atopic status	A significant 3.4% decrease in FVC and a 1.6% decrease in FEV1 was found in healthy non-smokers. Similar results were found for non-smokers and former smokers.

* Decreases in lung function calculated from parameters given by author assuming a 50 $\mu g/m^3$ increase in PM_{10} or 100 $\mu g/m^3$ increase in TSP.

TABLE V-18. EPIDEMIOLOGICAL STUDIES OF CHRONIC RESPIRATORY DISEASE

Study	PM Type & No. Sites	PM Mean & Range	Overall Symptom Rate	Model Type & Lag Structure	Other pollutants measured	Other Covariates	Other pollutants in model	Result* (Confidence Interval)
CHILDREN								
Ware et al. (1984) Study of respiratory symptoms in children in 6 cities in the U.S. Survey done 1974-1977	Daily monitoring of TSP, SO_2, NO_2, and ozone at each city	City TSP means ranged from 39 to 114 $\mu g/m^3$	Cough, .08, Bronchitis .08, Lower resp. .19	Logistic regression	SO_2, NO_2, and ozone	age, gender, parental education, maternal smoking	none	Cough 2.75 (1.92, 3.94) Bronchitis 2.80 (1.17, 7.03) Lower resp. 2.14 (1.06, 4.31)
Dockery et al. (1989) Study of respiratory symptoms in children in 6 cities in the U.S. Survey done 1980-1981	Daily monitoring of PM_{15}, sulfate fraction at each city	City PM_{15} means ranged from 20 to 59 $\mu g/m^3$	Cough, .02 to .09, Bronchitis .04 to .10, Lower resp. .07 to .16	Logistic regression	SO_2, NO_2, and ozone	age, gender, maternal smoking	none	Cough 5.39 (1.00, 28.6) Bronchitis 3.26 (1.13, 10.28) Lower resp. 2.93 (0.75, 11.60)
Neas et al. (1994) Study of children aged 7 to 11 from six cites in U.S. Survey done 1983-1986.	$PM_{2.5}$	Not given	Not given	Logistic regression	NO_2	household smoking, gas stove, age, gender	none	Cough 1.08 (0.76, 1.53) Bronchitis 1.32 (0.98, 1.79) Lower resp. 1.23 (0.98, 1.55)
ADULTS								
Abbey et al. (1995a,b,c) Study of AOD**, bronchitis, and asthma in adult Seventh Day Adventist	Daily monitoring of TSP, PM_{10}, visibility at 9 sites in northern and southern California	Not given	AOD = 11.8% Bronchitis = 7.2%	Multi-logistic regression	SO_4, O_3, SO_2, NO_2	Age, gender, education, previous symptoms	none	1.23 AOD** (0.91, 1.65) 1.39 Bronchitis (0.99, 1.92)

* Estimates calculated from data tables assuming a 50 $\mu g/m^3$ increase in PM_{10} or 100 $\mu g/m^3$ increase in TSP.
**Airway obstructive disease (AOD) is defined to include symptoms of chronic bronchitis, emphysema, and asthma.

association between PM levels and lower respiratory illnesses, chronic cough, and bronchitis. Three of the studies found associations in children, while one found an association between PM level and a measure of chronic respiratory disease in adults. The results from these studies are consistent and supportive of those reported for short-term studies. The implications of respiratory disease in children is discussed above in section V.B.

iii. Lasting Physiological Effects

Symptoms from long-term exposure to PM in humans include cough, chronic bronchitis (characterized by persistent cough and phlegm production), and lower respiratory illness. As stated by the CD, the presence of PM, which increases the risk for respiratory symptoms and related respiratory morbidity, is important because of associated public health concerns with regard to both the immediate and longer term symptoms produced and 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) and is also increased by PM exposure. Survivors of the Donora, Pennsylvania pollution episode with prior chronic disease and those who became acutely ill during the episode, had higher subsequent rates of mortality and illness (Ciocco and Thompson, 1961).

The 1982 Staff Paper concluded that community epidemiological studies (Ware et al., 1981; Dockery et al., 1981; 1989) and occupational studies of bronchitis in workers exposed to high dust levels (Morgan, 1978) suggested that high concentrations of long-term particle exposure is associated with an increase in prevalence of bronchitis. A description of respiratory diseases and related impairments associated with occupational exposures to particles was presented in Table B-1 of the 1982 Staff Paper (Appendix F). Lippmann (1981), drawing analogies between the effects of cigarette smoke and acids on clearance, has hypothesized that repeated acid exposure may have a role in the etiology of bronchitis, at least at concentrations that existed in the historical London episodes.

4. Morphological Damage

Traditional epidemiology has not been used to evaluate the extent to which PM directly alters lung tissues and components. Morphological alterations associated with

exposure to acid aerosols are outlined in Table 11-5 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, Chapter 11). Schwartz et al., (1977) reports that using very high concentrations ($\geq$ 70 mg/m^3) of H_2SO_4 at comparable particle size, rats and monkeys were quite resistant while guinea pigs and mice were the more sensitive species as measured by morphological endpoints. Animal studies of fine (0.3 μm) diameter to ultrafine (0.04 μm) diameter H_2SO_4 aerosols (300 μg/m^3), have shown 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).

Silica has long been considered to be a major occupational health hazard where exposure to crystalline silica is associated with pulmonary inflammation and fibrosis (CD, Chapter 11). The differing forms of silica (amorphous versus crystalline) are thought to have differential potential for toxicity but data on amorphous forms is limited (CD, Chapter 11). 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.

Some risk of long-term exposure to coarse dusts is suggested by autopsy studies of farm workers and residents in the Southwest, desert dwellers, and zoo animals and humans exposed to various crustal dusts near or slightly above current ambient levels. These studies find that those exposures may result in a silicate pneumonoconiosis. Responses ranged from the buildup of particles in macrophage with no clinical significance to possible pathological fibrotic lesions.

5. Altered Clearance and Other 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, Chapter 11). The effects of acidic sulfates on respiratory tract clearance are summarized in Table 11-7 of the CD. Relatively high H_2SO_4 exposure has also been associated with changes in clearance and macrophage function in experimental animals and in clearance in humans (CD, Chapter 11). Alteration of mucociliary clearance due to acid aerosols has also been reported and discussed in an Acid Aerosol Issue Paper (U.S. EPA, 1989). The direction and magnitude of the effect are 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, Chapter 11). In addition, the acidity of the aerosol has been reported to affect mucociliary clearance (CD, Chapter 11). Acid aerosols have been shown to elicit a slowing in clearance that lasts several months following multiple exposures (Lippmann, 1981). In regard to antigenic stimulation, guinea pigs have been reported to show increased sensitivity to inhaled antigen (ovalbumin) with concurrent H_2SO_4 exposure (CD).

Alveolar macrophages not only play a 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, Chapter 11). Studies by Chen et al. (1992a) suggest that effects in alveolar macrophage function may be dependent on particle size (increased phagocytosis with fine and decreased phagocytosis with ultrafine aerosols). Other studies have tried to use animal models to demonstrate an effect of H_2SO_4 exposure on susceptibility to bacterial infection. In such studies, large concentrations were required to elicit effects in previously healthy animals. In addition, acute exposures of up to 5 mg/m^3 of H_2SO_4 aerosols alone have not been demonstrated to enhance susceptibility to bacterially-mediated respiratory disease in mice (See Table 11-8 in the CD). However, Gardiner et al. (1977) reported increased susceptibility to infection by first ozone (0.1 ppm) and then H_2SO_4 (0.9 mg/m^3) in mice.

Fiber optic bronchoscopy with broncho-alveolar lavage (BAL) is a useful technique for sampling the lower airways of humans in clinical studies and can provide a relatively sensitive measure of inflammation (CD, Chapter 11). Only one study to date has utilized

bronchoscopy to evaluate responses to acid aerosols. Healthy nonsmokers were exposed to acid aerosol and were exercised after oral ammonia was neutralized. No evidence of airway inflammation or changes in markers for host defense were detected in BAL samples collected 18 hours after exposure (Frampton et al., 1992). Culp et al. (1995) determined that the acid aerosol exposure did not alter mucus composition in subjects in the Frampton study.

Metals have been reported to affect clearance and other host defence systems. Nickel inhalation has been shown to impair macrophage function and increase incidence of pneumonia in laboratory animals (CD, Chapter 11). Loading of particles with certain transition metals, such as iron, may have the potential to enhance particle toxicity, acute inflammation, and nonspecific bronchial responsiveness. Silica particles have been reported to be rendered more toxic when complexed with iron. Rats fed with iron depleted diets exhibited less inflammation and fibrotic injury after such exposures (Ghio et al., 1994; 1992; Ghio and Hatch, 1993). However there is some difficulty in extrapolating the *in vitro* experimental paradigms used in these studies to ambient exposure situations.

6. Cancer

Studies of long-term exposure have reported associations between mortality from lung cancer and PM levels. As reported above, using indicators of fine particle mass, ($PM_{2.5}$ and sulfates), increased risk of mortality from lung cancer has been reported. It is not clear whether such associations relate to causation of the disease, or whether individuals with cancer are more susceptible to other effects of particles.

Studies of the potential of particles to cause cancer as well as noncancer respiratory effects have been studied in laboratory animals. All major types of airborne PM may contain absorbed organic compounds which enhance the toxicity of the particles. Specifically these compounds may be mutagenic or carcinogenic to animals and may contribute in some degree to the incidence of human cancer associated with exposure to urban air pollution (CD, Chapter 11). Polycyclic aromatic hydrocarbons (PAHs) are perhaps the best studied class of potential carcinogens in PM. As noted in the 1982 Staff Paper, organic extracts with potential carcinogenic activity are preferentially found in the fine fraction. For example,

diesel exhaust particles and gasoline engine particles are examples of particles whose organic extracts have proven to be mutagenic and tumorigenic.

In addition to absorbed organic compounds, particles themselves have been shown to induce a carcinogenic response. Inhalation of talc (NTP, 1992) and carbon black aerosols (Stoker and Mauderly, 1994) have been associated with induction of lung tumors. Furthermore, it has been suggested that the insoluble carbon core of diesel particles is as at least as important as the organic compounds and possibly more so for lung tumor induction at high particle concentrations (> 2000 mg/m^3) (CD, Chapter 11). However it is not clear that this effect is seen at lower levels. Extrapolation to human risk from extraction studies are difficult because of different species and age, route of exposure (e.g., no inhalation assays in animals), physico-chemical properties of the material, and exposure concentration.

A large body of information has focused on toxicology of other constituents of PM such as metals, trace elements, and silica. Metals contained in PM are all toxic under specific conditions of exposure. Many are carcinogens as well as causing decreases in respiratory function. In addition, silica has been reported to cause lung tumors following chronic exposures in rats (CD, Chapter 11).

D. Strength and Coherence of Epidemiological Evidence

The majority of the evidence of the effects of PM on health comes from epidemiological studies. In assessing these studies for review of the primary standard, the strength and quality of the epidemiological studies must be evaluated. The CD outlines the major criteria to be used in evaluating the scientific quality of the epidemiological studies and to assist 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. In developing staff conclusions and recommendations for primary standards, it is important to evaluate the most critical aspects of the epidemiological studies. Accordingly, the discussion below summarizes the consistency and coherence of the recent studies as a group, and outlines observations on

potential confounding by weather, other pollutants, exposure considerations, as well as sensitivity to model specification.

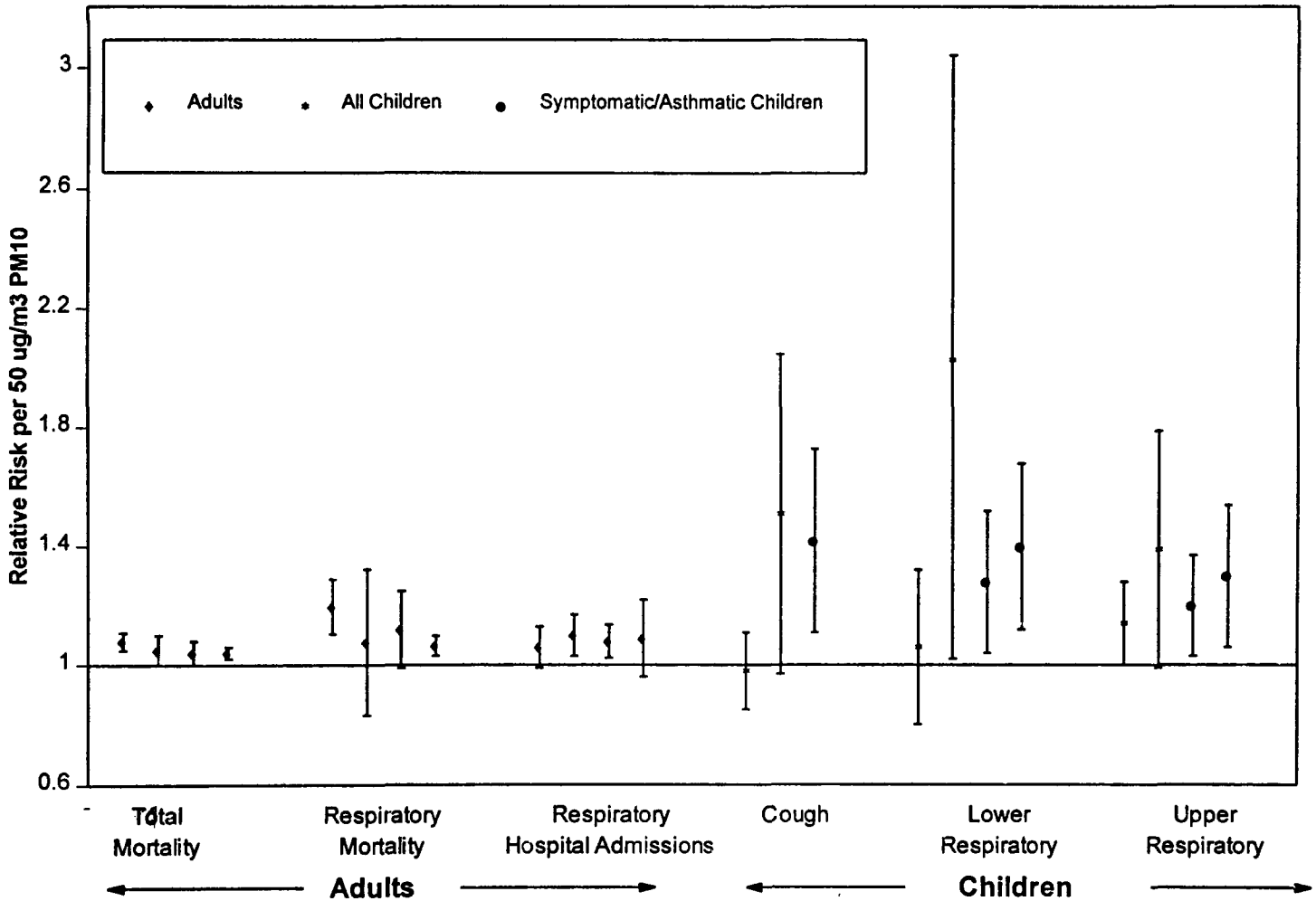
1. Consistency and Coherence

As noted above, numerous recent studies have evaluated the relationship between PM and health effects. Figure V-1 displays the results of selected studies for a variety of health effects. These studies have been conducted in a number of geographic locations, using a variety of air quality measurements and statistical techniques, and with varying temporal relationships. The strength of the association is enhanced by qualitatively and quantitatively consistent associations between PM levels and health effects found by different investigators in different geographical locations. In the early stages of this review, issues were raised about the reproducibility of the original studies finding effects at low levels in several U.S. cities. To resolve these issues, the Health Effects Institute (HEI) funded a reanalysis of data sets from Philadelphia, Utah Valley, Santa Clara, St. Louis, Kingston and Birmingham (Samet et al., 1995). The HEI reanalyses produced independent results that closely agreed and confirmed the original investigators' results in all six locations[1]. Evaluated in isolation, individual studies may have specific weaknesses, which have been discussed previously in the CD. When viewed together, however, the studies show consistent effects of PM on health.

In addition, this collection of studies shows substantial coherence in the types of observed effects. The mortality studies indicate respiratory-related and cardiovascular deaths are a major contributor to the PM/mortality relationship. These findings are supported by the morbidity studies finding an association between respiratory and cardiovascular hospital admissions. Related, but less severe health effects include symptoms and functional changes indicating aggravation of respiratory diseases such as bronchitis and asthma, as well as acute respiratory illness as evidenced by cough and other lower respiratory tract symptoms. The impact of these events are also observed in the association with restricted activity days, work

[1]Additional observations and insights from the extended HEI analyses are included in subsequent sections. HEI typically receives half of its funds from U.S. EPA and half from 28 manufacturers and marketers of motor vehicles and engines in the U.S.

FIGURE V.1. RELATIONSHIP BETWEEN RELATIVE RISK PER 50 $\mu g/m^3$ PM_{10} AND SPECIFIC CAUSES OF MORTALITY AND MORBIDITY IN ADULTS AND CHILDREN



Total Mortality and Respiratory Mortality

1. Pope et al. (1992)
2. Schwartz (1993)
3. Styer et al. (1995)
4. Ostro et al. (1995a)

Respiratory Hospital Admissions

1. Schwartz (in press) New Haven, CT
2. Schwartz (in press) Tacoma, WA
3. Scwartz (in press) Spokane, WA
4. Thurston et al. (1994) Ontario, Canada

Cough, Lower Respiratory, and Upper Respiratory

1. Hoek and Brunekreef (1993)
2. Schwartz et al. (1994)
3. Pope et al. (1991), asthmatic children
4. Pope and Dockery (1992), symptomatic children

loss days and school absences. Such a coherence of effects adds to the strength and consistency of the association (Bates, 1992).

2. Model Sensitivity

Chapter 12 of the CD discusses concerns that have been raised regarding the extent to which the observational results are sensitive to the statistical modeling approach used to analyze the data. Investigators have applied a variety of statistical techniques to evaluate the relationship between observed health effects and variations in short-term exposure to PM (Table C.1, Appendix E) with almost all approaches indicating consistent positive associations. The investigators from the recent analyses sponsored by the HEI developed a new statistical method for analyzing data. Using this technique, corrected t-statistics were reported to be greater than those reported by the original investigators. This result would indicate that the original investigators underestimated the statistical significance of the PM/mortality relationship. They confirmed the PM_{10}/mortality relationship was relatively insensitive to statistical technique.

3. Exposure Misclassification

A significant difficulty in interpretation of the epidemiological studies, particularly for quantitative purposes, is the determination of uncertainties and possible biases introduced by using outdoor monitors to estimate population exposures. It is important to examine the possible effect that exposure misclassification may have on the reported associatins 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 below focuses on the relationship between the monitored pollutant levels and the actual exposure and on how the error in the measurements might bias the reported associations.

Chapter IV discusses the overall relationship between outdoor monitors and personal exposure and the properties of fine particles. Apart from errors associated with the outdoor measurements themselves, questions have been raised about the number of monitors needed to represent the population. Because fine particles are spatially homogeneous, the uncertainties associated with studies using a limited number of monitors to represent daily

fluctuations in community concentrations are reduced. In contrast, the coarse fraction which is less spatially homogeneous across a city, is not as well represented by a limited number of monitoring stations.

Moreover, because some of the sensitive populations in the short-term mortality and hospital admissions studies (i.e., the elderly and those with pre-existing disease) may spend even more time indoors than the general population, the extent to which outdoor fluctuations are found to affect indoor and personal exposures is of particular importance. Examination of indoor and personal exposures calculated on a group level provide equivocal results. Correlations are often poor because they mainly reflect variation between subjects (Janssen et al., 1995, CD, Chapter 7). However, as discussed in Chapter IV and the CD, studies of serial correlations are more relevant in determining how well personal exposure can be represented by a monitoring station for short-term exposure studies. At least two exposure studies that used serial correlations (Janssen et al., 1995; Lioy et al., 1990) noted good serial correlations between outdoor PM_{10} and personal exposures (e.g., R^2 of 0.4 to 0.53 for children), and even better serial correlations for outdoor and personal exposure for $PM_{2.5}$ (e.g.,R^2 of 0.74 for children) (Janssen et al., 1995). The strength of this correspondence between outdoor concentrations and personal exposure levels on a day-to-day basis reduces, but does not completely eliminate concerns about the use of outdoor monitors in the short-term studies.

Given the potential error in pollution and other covariate measurements, consideration should be given to the effect of these errors on the association between exposure and outcome. This measurement error can often bias the association toward the null. However, the association can also be influenced by the relationship between particulate matter and the other covariates, which can bias the association in either direction. For example, Schwartz and Morris (in press) address this issue in their study of cardiovascular hospital admissions and PM, CO, and weather in Detroit, MI. In this case, the correlation between CO and the weather variables and PM was small. In addition, the correlation between PM and weather variables was also small. Such low correlations may imply that it is likely significant portions of bias comes from the errors in measuring the pollutants, which would decrease the

association between particulate matter levels with hospital admissions. The authors point out, however, that this does not mean that the estimated magnitude of the associations was unbiased.

It is possible that the reported associations would be biased upward if there is a significant positive correlation between the covariates. Such bias would result in an increased chance of finding a false positive finding. Thus, when reviewing the potential for exposure misclassification, it is important to consider potential correlation between the covariates, since it can influence the effect estimate. Schwartz (1994b) reviewed the reported correlations between PM, temperature, and SO_2. The correlations between PM and temperature were less than 0.5 in 9 of the 10 study areas. Similarly, the correlations with SO_2 in 5 of the 8 study areas were less than 0.4. Consistent with the implications of the previous example (Schwartz and Morris, in press), these low correlations suggest that much of the error results from the measurement of PM.

While the precise effect that measurement error will have on the particulate matter/health effect associations is unknown, it is possible to estimate the likely influences measurement error will have on the association, and thereby exclude unlikely scenarios. Given the good correlation between ambient monitors and personal exposure, these observations suggest that the association is more likely to be biased downward. Nevertheless, a comprehensive, formal treatment of exposure misclassification in particulate matter effects studies is an important research need.

4. Confounding in Short-term Studies

Potential confounders of the PM/health effects relationship are those independent risk factors related to both PM concentrations and the health effect of interest. Inadequate control for confounding can result in incorrect interpretations assuming the effect is a result of the observed risk factor, when a third variable (the confounder) is really responsible. In short-term exposure studies, major covariates associated with daily changes in health effects, such as weather, season and other pollutants (e.g., SO_2) correlated with PM need to be considered. In contrast, as discussed in the next section, the long-term prospective cohort studies and cross-sectional studies need to consider risk factors that may vary among

communities (e.g., smoking levels). A number of methods are used by epidemiologists to address or reduce confounding, with varying degrees of success. A summary of the major issues relevant to recent PM studies is outlined below.

a. Weather

Weather is an important confounder in short-term PM studies because fluctuations in weather are associated with both changes in PM levels and health effects observed in the studies. The relationship between weather and mortality often follows a U-shaped curve with increases in mortality in cold and hot weather conditions. PM levels can be high under either temperature regime; some areas have peak levels in the summer (e.g., Los Angeles), and some in the winter (e.g., Utah Valley). The independent effect of PM separate from weather can be ascertained both by evaluating multiple studies conducted in areas with varying weather and by assessing the methods individual studies use to adjust for weather. Taken together, positive consistent associations between PM and mortality and morbidity effects have been observed in dry and humid climates (e.g., Los Angeles versus Birmingham), cold and warm climates (e.g., Toronto versus Birmingham) and areas where PM is highest in the winter (e.g., Utah Valley, Minneapolis), and the summer (e.g., Birmingham), or are elevated in both seasons (e.g., Los Angeles) (Schwartz et al., 1994b).

On an individual study basis, investigators have used a variety of approaches to separate the effects of PM and weather. For example, some studies have excluded a portion of the extreme weather days, as defined by temperature and/or humidity, from the analysis (Schwartz, 1994a; Schwartz and Morris (in press). Many investigators use statistical methods to adjust for weather when modelling 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). Finally, an examination of the sensitivity of the PM_{10}/health effect relationship

to varying degrees of control for weather has been examined (Schwartz (in press), Schwartz et al., in press, Kinney et al., 1995). Several studies reported that the effects of weather had on mortality were distinct but largely separate from the effects of PM in the areas studied; elimination of all weather variables from the PM-mortality models did not substantially affect the size of the observed associations between PM and mortality (Schwartz et al., in press; Schwartz and Dockery, 1992a, 1992b). All the studies use some method to adjust for weather, and report consistent, positive associations between PM and health effects.

The HEI analysis included a careful evaluation of the sensitivity of the mortality/TSP association to specification of weather in their reanalysis of Philadelphia (Samet et al., 1995). The investigators used a nonparametric surface to model the relationship between mortality and temperature and dewpoint, and compared it to the linear terms used by Schwartz and Dockery (1992a). They found association between mortality and TSP was relatively insensitive to the specification of weather in the model.

A number of investigators and commenters believe that the most recent analyses of short-term PM and mortality have controlled adequately for weather (Moolgavkar et al., 1995; Schwartz, 1994; Dockery and Pope, 1994). Recommendations have been made, however, to examine further the use of synoptic classifications (Kalkstein, 1991) associated with health stress as an approach to assessing weather as a confounder of PM effects. Analyses of this kind have been recently completed for Utah Valley and are being submitted for publication. The unpublished results are reported to be consistent with the above assessment (Pope and Kalkstein, in press).

b. Confounding by Other Pollutants

One of the considerations in evaluating these studies is whether the health effects are associated with air pollution in general, of which PM is a portion, or whether the health effects are independently exerted by PM. Suggestive, but not conclusive, support for PM effects comes from a comparison across multiple areas and studies. If PM is truly acting independently, then a consistent association should be observed in a variety of locations of

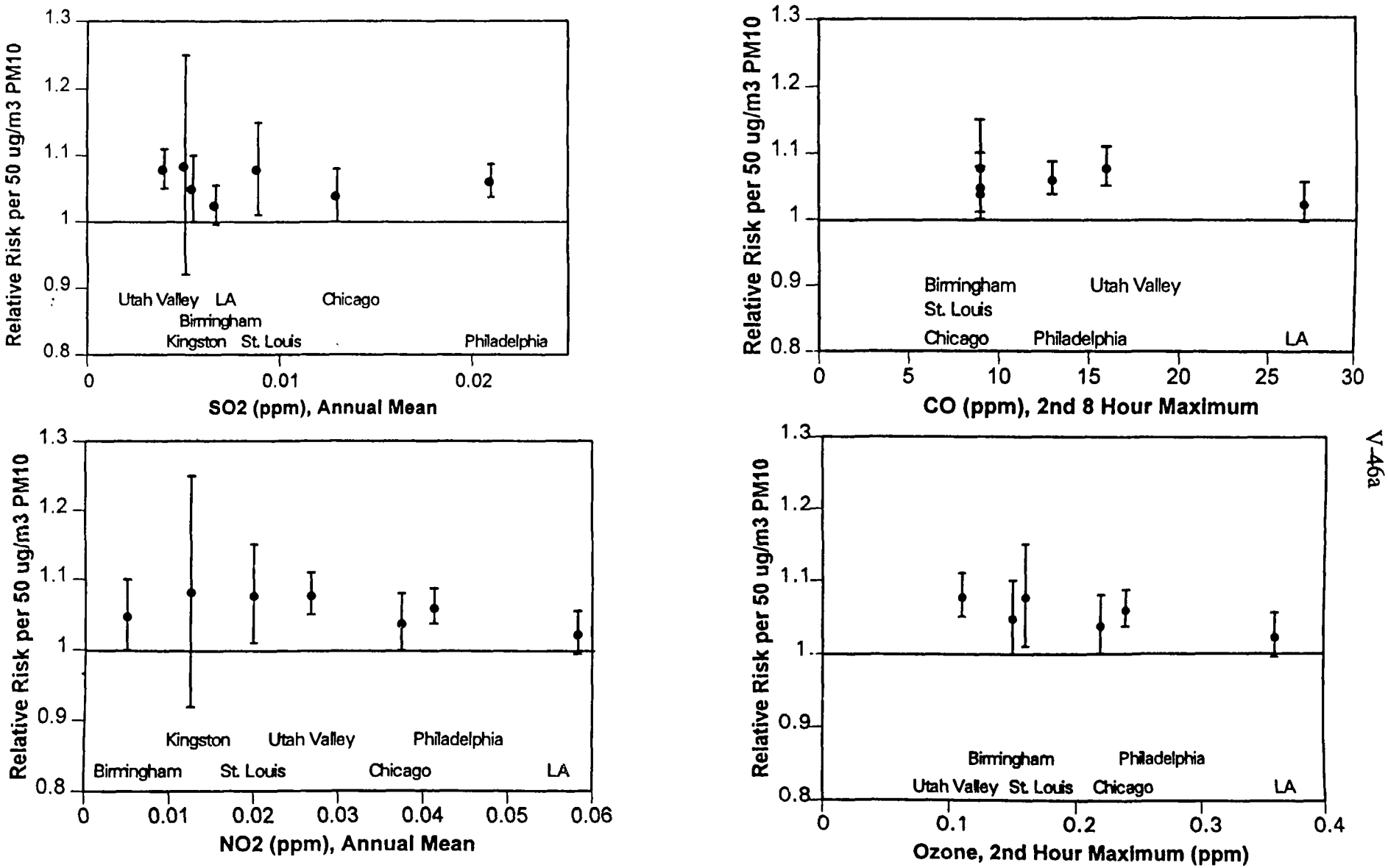
differing relative proportions of particles and potential gaseous pollution confounders. If instead the observed PM effect results from confounding from another pollutant, the associations with PM would be expected to be consistently high in areas with high concentrations of the confounder, and consistently low in areas with lower concentrations of the confounder. Figure V-2 shows the reported relative risk of particle effects and associated levels of SO_2, NO_2, ozone, and CO for a number of study cities where such data were readily available. The relative risks are those reported in each of the studies, unadjusted for the other pollutants. The figure indicates that the association with PM remains consistent through a wide range of concentrations of these potentially confounding pollutants. Although further studies are needed in this area, the available body of data show some associations for these other criteria pollutants, but the results are not as consistent as for PM. While it is possible that different pollutants may serve to confound particles in different areas[2], it seems unlikely that the confounding would lead to such similar associations and relative risk numbers for particles. Specific observations relating to the potential for confounding from SO_2, ozone, CO and NO_2 are outlined below.

i. Sulfur Dioxide (SO_2)

SO_2, having been present at high concentrations with PM during some historical episodes, especially in the well-documented wintertime smogs in London, has long been seen as a prime candidate for potential confounding. Reanalyses of the 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 exclusion (studies in areas with low SO_2 levels) and more direct means. Other pertinent information comes from SO_2 and PM air

[2]In this interpretation of the results, 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-2. RELATIONSHIP BETWEEN RELATIVE RISK OF MORTALITY ASSOCIATED WITH PM_{10} AND LEVELS OF SO_2, CO, NO_2, AND OZONE.



Data on SO_2, CO, NO_2 and O_3 are from the EPA Trends Report, except for annual averages of SO_2 and NO_2 in Kingston and St. Louis, which are from Dockery et al (1992). The detection limit is used for cities reporting nondetects (Utah Valley (SO_2), and Birmingham (NO_2)).

quality relationships and studies of the penetration of SO_2, alone and in combination with particles, to the respiratory tract.

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 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., 1995; Schwartz 1992a; Saldiva et al., 1994). A similar analysis 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 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; Moolgavakar, 1995b; Li and Roth, 1990; Samet et al., 1995). 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 evaluated the association between TSP and mortality in Philadelphia between 1973-1980 (1992a). 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. They evaluated the associations separately by season because of potential confounding by season. Modeled individually with mortality, 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 in Philadelphia.

HEI evaluated both of these data sets (Samet et al., 1995). Although overall results were similar to those of the original authors, the techniques used in this study 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 study to attribute such effects solely to particles. The combined pollutant mortality relationships are of some interest (See Figure V-13 below in section V.E). 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.

The original investigators' response to HEI suggest 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 (Wilson et al., 1995) 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.

In evaluating the findings in Philadelphia, an important consideration is the evidence on the mechanisms of toxicity of particles as compared to SO_2 alone. Although quantitative support is lacking, the discussion of controlled human and animal studies of particles indicate that smaller particles can more effectively penetrate to the portions of the lung where irritation or other interactions with lung tissues might produce significant effects. (See section V.A above). Beyond reflex bronchoconstriction observed only at very high peak levels, however, gas-phase SO_2 is generally efficiently removed in the extrathoracic region in humans (U.S. EPA, 1994c). It is hard to posit significant cardio-pulmonary effects for low

concentrations of a substance that does not effectively reach the bronchial or alveolar regions. However, one mechanism by which SO_2 can be transported deeper into the lung is absorption or dissolution onto the surfaces of atmospheric particles (U.S. EPA, 1982b). In this case, the complex HEI results might be 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 exclusion, i.e. 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 (Figure V-2a). As discussed above, consistent significant associations between particle level and mortality are found in cities of differing SO_2 levels (Figure V.2a). The figure also shows the relative risk of mortality associated with PM_{10} remains relatively unchanged in areas of low SO_2 versus high SO_2, further supporting the robustness of the association between PM_{10} and mortality. 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, 19992a,b) although the two areas have comparable SO_2 levels.

There are studies in a few cities where SO_2 and PM are highly correlated and it is more difficult to ascribe the observed mortality effects to a single pollutant (e.g., Philadelphia). In such cases, consideration of the observed relationships and relevant information on air quality, dosimetry, and mechanisms suggest that there is not an independent effect of SO_2 that does not involve some particle fractions. 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).

ii. Ozone

Increased ozone concentrations in the summer or ozone season is a potential confounder in areas with concurrent high PM levels, (e.g., large regions of eastern North America and Los Angeles). In areas where ozone peaks often occur in the same time of year as high PM concentration, covariate adjustment has often been used to try to distinguish effects from other pollutants. A number of studies using such methods have found PM to be a stronger predictor of mortality than ozone (Dockery et al., 1992; Saldiva et al., 1995; Kinney et al., 1995; Ostro et al., 1995). Adjusting for the presence of ozone did not affect the associations with PM and mortality. For example, in Los Angeles, which has the highest concentrations of ozone studied (Figure V-2), the investigators found no significant association between ozone and mortality in models that included PM (Kinney, 1995). In Santiago, a negative correlation exists between ozone and PM, and a positive association was not observed between ozone and mortality across a full year even without PM in the model, despite summertime values of ozone twice the U.S. standard (Ostro et al., 1995). Finally, in Utah Valley, ozone and PM were also negatively correlated, resulting in a strengthened PM effect being observed upon inclusion of ozone as a covariate (Pope et al., 1995, Table V-3).

As discussed above, Figure V-2d shows the relative risk for mortality from exposure to PM by the 2nd hour maximum ozone value in each of the areas (exception of St. Louis, where the value is the maximum daily average). The relative risk remains relatively constant across the studied areas. Additionally, in some locations the potential for ozone to confound the effects caused by PM is minimized by the low concentrations observed during seasons which show a robust PM effect. Examples include Utah Valley and Santa Clara, where ozone levels are minimal in the winter when the PM levels are high (Pope et al., 1992; 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 ozone levels.

There is a higher potential for ozone confounding for the risk of respiratory-related morbidity, because multiple studies have demonstrated apparent separable associations between respiratory effects and PM and ozone concentrations. The respiratory-related hospital admission studies often find ozone and PM are each singularly associated with respiratory-related admissions (Schwartz, 1994d; Schwartz (in press); Burnett et al., 1994). Table V-11 shows the relative risk associated with a 50 ug/m^3 increase in PM_{10} and hospital admissions before and after inclusion of ozone in the model in New Haven, CT and Tacoma, WA (Schwartz, in press). 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 ozone. Potential for ozone confounding for cardiac-related hospital admissions appears to be much less. Two studies have reported that PM is associated with cardiac hospital admissions but ozone is not (Burnette et al., 1995; Schwartz and Morris, in press).

iii. 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 positively associated with mortality. However, there was a high correlation between CO and PM. 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 results from these studies are inconsistent. Because of the nature of urban sources of CO as well as indoor sources, exposure misclassification may introduce significant problems. 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 Schwartz and Morris's study (in press) 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.

iv. 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, 1995, Ozkaynak, 1994). Mixed results were reported concerning the association between NO_2 and mortality. Kinney and Ozkaynak (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., 1995. 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., 1992).

While the association between NO_2 and health effects in these studies is inconclusive, the association between PM and health effects remains positive and consistent, both across study areas with varying levels of NO_2 (Figure V-2c) and after controlling for NO_2 in the model (Ostro et al., 1995; Saldiva et al., 1995; Schwartz et al., 1994).

c. Summary of Short-Term Confounders

Evaluated individually, each study has potential confounding problems. Concerns continue relating to treatment of weather, other pollutants, and particularly, measurement errors and exposure misclassification. However, there are over 50 studies finding positive, significant associations between short-term PM levels and mortality and morbidity endpoints. Many of these studies have been conducted in areas across the U.S. and Canada, where meteorological and pollution patterns vary distinctly. Yet, the studies find a consistent, positive association between PM and mortality and morbidity effects. The breadth of these studies and insights drawn from air quality and controlled effects studies suggest that it is unlikely that the PM effect can be entirely attributed to one or more of the known potential confounders. Moreover, although the available evidence can not be conclusive, it also seems unlikely that the effect is due to some unknown non-particulate confounder.

5. Confounding in Long-term Exposure Studies

In the long-term studies, differences in health effects are evaluated across communities with differing levels of pollution. Unlike the short-term exposure studies, which must consider confounders that fluctuate temporally with PM levels, these studies need to consider confounders that may vary among communities and that are important risk factors for the health effect of interest. The studies evaluate a range of health effects from mortality to morbidity endpoints, but mostly focus on respiratory and cardiovascular related endpoints. The recent cohort studies represent a substantial advance in the study of the effects of long-term exposure to air pollutants, because individual information on potential confounders such as age, sex, smoking habits, education, occupation, and other risk factors can be taken into account. Several specific risk factors are discussed below:

1) Smoking represents the largest potential confounder since it is highly associated with the same effects observed with PM. Smoking has either been controlled for by adjusting for the presence of smoking and pack-years smoked in the analysis, or by exclusion of smokers from the analysis. Increased risk of mortality is seen after adjusting for smoking. In addition, associations between mortality and morbidity effects and PM continue to be observed when the analysis is restricted to never smokers (Pope et al., 1995; Dockery et al., 1993) or involve only non-smokers (Abbey et al., 1991-5).

2) Similar to smoking, individuals with occupational exposure to PM (e.g., dust, gases, fumes, asbestos, chemical acid solvents, coal or stove dust) appear to be at increased risk from particle effects ((Dockery et al., 1993; Abbey et al., 1991, 1995), although the relationship continues to hold after adjustment for occupational exposures (Dockery et al., 1993; Pope et al., 1995). Pope et al. (1995) also found that once the analysis adjusted for cigarette smoking, adjustments for occupational exposure made little difference in the observed PM-mortality associations.

3) Weather is a potential confounder in short-term exposure studies, because short-term fluctuations in weather are associated with health effects. Since long-term exposure studies evaluate changes in health effects over longer periods of time, short-term variations in weather are less a concern in these studies. However, if there is a difference in total mortality over the year due to different cumulative impacts of weather and season in differing locations, this could lead to some potential confounding. Pope et al. (1995) found that daily high, low, or mean temperature was not correlated with $PM_{2.5}$ concentrations across the fifty cities he studied, and that inclusion of indicator variables for generally "hot" and "cold" cities did not substantially affect the particulate matter/mortality associations.

4) The possibility of confounding by other potential confounders needs to be assessed in relationship to the controls that were used. For instance, individual controls on body-mass index and education would serve as substantial controls on the most relevant diet and lifestyle factors.

In regard to residual confounding, those studies using a larger number of cities or locations would be expected to provide better estimates of effects, since the chances that a potential confounder would be substantially associated along the same gradient as the pollutant in question should be expected to decrease as the number of additional areas and distinctive locations increases.

5) With regard to copollutants, mortality effects associated with long-term exposures to air pollutants were more strongly associated with the levels of inhalable, fine, or sulfate particles than with the levels of sulfur dioxide or nitrogen dioxide (Dockery et al., 1993). In addition, no apparent relationship with long-term ozone concentrations was observed, although the small differences in ozone levels among the cities limited the power to detect ozone-health effects associations (Dockery et al., 1993). The Seventh Day Adventist study found that both the development and aggravation of chronic respiratory disease symptoms was associated with long-term exposures to several indicators of particles (TSP and estimated PM_{10} and $PM_{2.5}$) (Abbey et al., 1995b). In contrast, long-term exposures to sulfur dioxide or nitrogen dioxide did not have effects on chronic respiratory disease symptoms, while ozone was associated with aggravation of asthma symptoms, but not the development of aggravation of chronic bronchitis (Euler et al., 1988; Abbey et al., 1993, 1995). The lack of independent effects for copollutants at the levels observed in these long-term studies indicate that these copollutants are not likely to be confounding the effects observed between particles and mortality and morbidity.

E. Concentration-Response Relationships

This section presents a staff assessment of quantitative information on concentration-response relationships between health effects and ambient PM. As discussed below for short- and long-term studies, the staff has focused on selected individual studies to gain insight, to the degree possible, from the available concentration-response information as to where a clear and consistent increase in risk may be discerned. This assessment is intended to provide information useful in deriving appropriate ranges of concentrations for consideration in selecting ambient air quality standard levels. In so doing, the staff does not intend to

imply that such an assessment can or should be used to attempt to identify a threshold of effects below which there is no increased risk. Such population thresholds may not exist within the range of ambient PM concentrations, and, as previously discussed, epidemiological studies are limited in their ability to discern such thresholds even if they do exist.

Further, the staff recognizes that the inherent uncertainties in each individual study, such as those associated with specification of relevant exposures and potential confounding by other environmental and/or personal variables, substantially limit the conclusions that can appropriately be drawn from any one study. Thus, in this assessment, the staff's interest is ultimately on the coherence of results from all the studies rather than solely on each study considered independently. This quantitative assessment of concentration-response relationships is considered in context with the entire body of health effects and air quality evidence in identifying a quantitative basis for drawing conclusions and recommendations about appropriate ranges of levels for consideration in setting PM standards, as presented in Chapter VI.

1. Criteria for Assessment

The epidemiological studies of short- and long-term exposures to PM are most straightforwardly interpreted as indicating whether a general, global association exists between health effects and the varying concentrations of particulate concentrations observed in the studies. However, to gain additional insight useful in assessing policy alternatives for standard setting, staff has looked to several types of information from the studies in assessing concentration-response relationships between exposures to PM and health effects. As discussed below, such information includes the range of PM concentrations over which the general relationship between particles and health effects are observed, the central tendency of that range, and the pattern and consistency of increases in risk observed within the range of PM concentrations.

1) Information on the range of concentrations observed in the study helps bound the particle concentrations with which there is a possible association with the observed health effects. However, it is often not possible to make clear

inferences as to whether the observed risk of health effects is uniformly associated with increasing particle concentrations, or is more strongly associated with concentrations of PM above a certain level.

2) Information on the central tendency (median or mean) of the range can provide an indication where the observed association between particles and health effects has the least statistical uncertainty, although this value may not reflect a point at which risk is clearly elevated, especially if the relationship between health effects and particle concentrations is substantially non-linear.

3) The most useful information for the assessment of concentration-response is provided by presentations of the observed relationships between risk and particle concentrations for various segments of the overall range. Such presentations typically take the form of dividing the data into smaller percentile ranges (such as quartiles or quintiles) and reporting the associated risk for each division of the range, or nonparametric smoothed curves that apply weighted averages of the effects around a particular concentration. Both of these approaches allow the existence of substantial nonlinearities in the observed relationship to be assessed, and have been used by some investigators to identify concentrations associated with elevated health risk (e.g., Samet et al., 1995; Schwartz, 1993a).

However, for reasons discussed in the CD, the staff recognizes that these approaches have inherent limitations and need to be interpreted with caution. For example, observed differences in risk across discrete or continuous segments of the concentration range may be more reflective of relatively smaller numbers of observations in one or more of the segments than of real differences in observed risk.

The specific studies considered in this assessment were selected on the basis of the evaluation of the quality of the studies presented in the CD, findings of significant associations between PM exposures and health effects, and the availability in the study of information that provides insight into the pattern of concentration-response within the range of the study.

2. Concentration-Response Relationships from Short-Term Studies

The selected studies listed in Table V-19, which report associations between short-term (24-hour average) exposure to PM and health effects, provide quantitative concentration-response information showing the pattern of increased risk within the range over which increased relative risk is reported. Included in this list of selected studies are studies that report associations between PM, as measured by PM_{10}, $PM_{2.5}$, sulfates, and TSP, and mortality, hospital admissions, and respiratory symptoms. These selected studies are part of the much larger number of studies that have been evaluated in the CD and summarized in section V.C. They are discussed below with regard to the nature of the concentration-response relationships observed in the studies and the insights that can be gained with regard to the PM concentrations where clear and consistent increases in risk can be discerned.

a. $PM_{10}/PM_{2.5}$ Studies

The most recent investigation of the effects of daily PM_{10} and $PM_{2.5}$ concentrations on mortality examined the associations between several indicators of particles and daily mortality in six cities (Schwartz et al., in press). As discussed above in section V.C. and summarized in Table V-6, $PM_{2.5}$ showed the strongest and most consistent association with mortality, coarse fraction particles did not show a statistically significant[3] association, and the statistically significant association observed between PM_{10} and mortality has been interpreted by the authors as resulting primarily from fine particles (Schwartz et al., in press). Positive associations between $PM_{2.5}$ and mortality were observed for each of the six cities, and this association was statistically significant for the three cities experiencing the highest daily mortality in the study. Although concentration-response curves were not presented for each city, the authors reported an additional analysis that excluded all $PM_{2.5}$ concentrations of 30 $\mu g/m^3$ and above in all the cities (approximately the top 10% of values). The association remained statistically significant when concentrations of 30 $\mu g/m^3$ $PM_{2.5}$ and above were excluded.

[3]Statistical significance is reported at the 95% level throughout this section unless otherwise noted.

TABLE V-19. CONCENTRATION-RESPONSE INFORMATION FROM SELECTED SHORT-TERM EXPOSURE STUDIES

Study	Mean or Median Measured Concentration ($\mu g/m^3$ of measured pollutant)	Concentration Range ($\mu g/m^3$ of measured pollutant)	Minimum Clear Increased Risk Level ($\mu g/m^3$ of measured pollutant)
$PM_{10}/PM_{2.5}$ Studies			
Mortality -- 6 Cities			
Schwartz et al., in press: PM-2.5	18	<4 - >44	<30
PM-10	25 (median)	8-69	
Mortality -- St. Louis			
Dockery et al., 1992: PM-10	28	1-97	33
PM-2.5	18	1 - 75	-
Samet et al., 1995: PM-10	"	"	44
PM-2.5	"	"	-
Mortality -- Utah Valley			
Pope et al., 1992: PM-10	47	up to 297	46
Samet et al., 1995 PM-10	"	"	50
Mortality -- Birmingham			
Schwartz, 1993a,1994g: PM-10	48	up to 163	60
Samet et al., 1995 PM-10	"	"	53
Hospital Admissions -- Birmingham			
Schwartz et al., 1994e: PM-10	45	19 - 77	20 - 45
Hospital Admissions -- Detroit			
Schwartz, 1994d: PM-10	48	22 - 82	52
Hospital Admissions -- Detroit			
Schwartz and Morris, in press PM-10	48	22 - 82	37
Respiratory Symptoms -- 6 Cities			
Schwartz et al., 1994: PM-10	30	<13 - 117	≤30
PM-2.5	18 (median)	<7 - 86	-
Respiratory Symptoms -- Utah Valley			
Pope and Dockery, 1992: PM-10	76 (mean)	up to 251	55
Sulfate Studies			
Hospital Admissions -- Ontario			
Burnette et al., 1994, 1995	3 (median)	<1.5 - >23	4 - 8
TSP Studies			
Mortality -- Philadelphia			
Samet et al., 1995	77	22 - 338	100

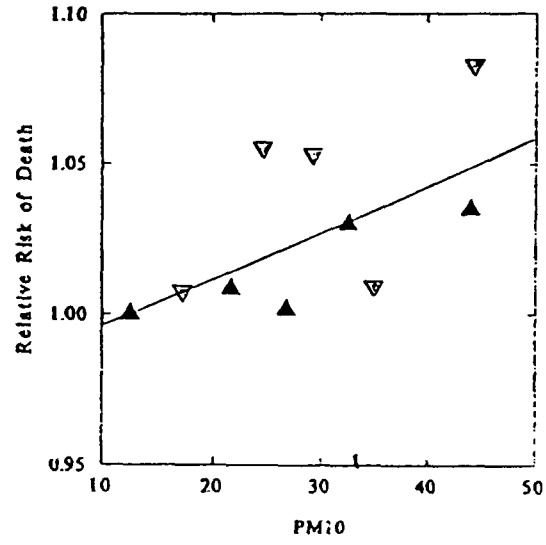
These results from Schwartz et al. (in press) are generally consistent with the earlier findings of Dockery et al. (1992). Dockery et al. (1992) reported an earlier analysis of one of six cities, St. Louis, that included only one-fifth of the observations for this location that were later included in the Schwartz et al. (in press) study. Positive associations were reported between $PM_{2.5}$ and mortality that reached marginal statistical significance, but the strongest associations over the study period were reported between PM_{10} and mortality. Concentration-response information for PM_{10} was presented in the form of a quintile plot showing the association between daily particle concentrations and mortality (Figure V-3[4]). Examination of this information suggests that risk may not increase in a clear and consistent pattern until the fourth quintile, with a mean concentration of approximately 33 $\mu g/m^3$ PM_{10}. A reanalysis of these data (Samet et al., 1995), which associated quintiles of PM_{10} with mortality in a slightly different manner, reported that risk was not elevated until perhaps the highest quintile (Table 22 in Samet et al., 1995), with a mean concentration of approximately 44 $\mu g/m^3$ PM_{10} (as reported in the original study). A positive but marginally significant association was also reported with $PM_{2.5}$, although no $PM_{2.5}$ concentration-response relationship was presented.

Pope et al. (1992) reported an association between PM_{10} and mortality in the Utah Valley, as shown in Figure V-4. Although generally linear, risk does not appear to clearly and consistently increase until the third quintile, with a mean concentration of about 46 $\mu g/m^3$ PM_{10}. Reanalysis of the Utah Valley data by Samet et al., (1995) found no pattern of increased risk until the fourth quintile, with a mean concentration of approximately 50 $\mu g/m^3$ PM_{10} (Table 28 in Samet et al., 1995). Thus, while the two analyses differ somewhat concerning risk at the lowest concentrations, where uncertainty would be expected to be greatest, the analyses appear to agree well concerning the PM_{10} concentrations, 46-50 $\mu g/m^3$, associated with a clear and consistent increase in risk.

An association between PM_{10} and mortality was also reported for Birmingham (Schwartz et al., 1993a). The concentration-response relationship was reported as a

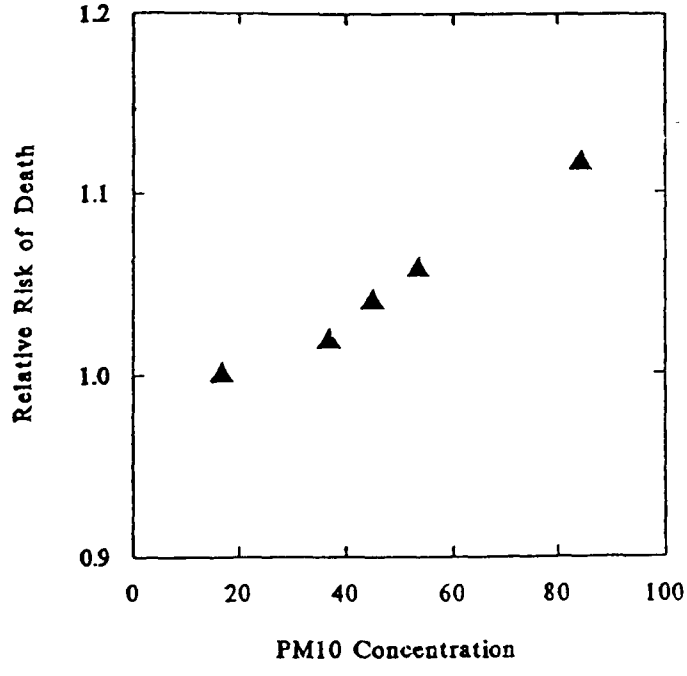
[4]Figures V-3 through V-18 in this section include annotations as presented in the original references.

FIGURE V-3. RELATIONSHIP BETWEEN RELATIVE RISK OF MORTALITY AND PM-10 IN ST. LOUIS AND EASTERN TENNESSEE (DOCKERY ET AL., 1992)



Relative risk of mortality of quintile of PM_{10} concentrations on the previous day, separately for St. Louis (▲) and eastern Tennessee (▽). The lowest quintile in each area is taken as the reference category. Straight line is the weighted mean regression.

FIGURE V-4. RELATIONSHIP BETWEEN RELATIVE RISK OF DEATH AND PM-10 IN THE UTAH VALLEY (POPE ET AL., 1992)



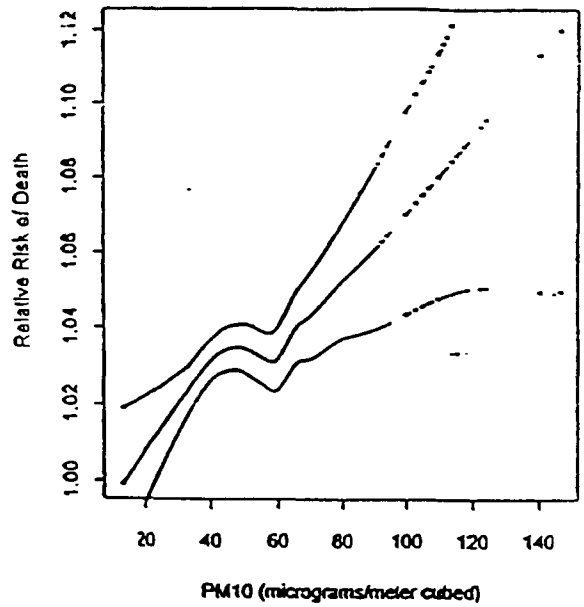
Relative risk of death, by quintile of PM_{10} concentration.

nonparametrically smoothed plot, with a presentation in a subsequent publication showing the 95% confidence limits (Schwartz, 1994g), as shown in Figure V-5. This plot shows an increase in relative risk over the range of approximately 20-40 $\mu g/m^3$ PM_{10}, with a flattening in the concentration-response relationship between approximately 40 - 60 $\mu g/m^3$. Risk appears to increase in a clear and consistent pattern at concentrations of 60 $\mu g/m^3$ and above. A reanalysis of this study which presented information in terms of quartiles found risk to be elevated for the third and fourth quartiles (Table 31 in Samet et al., 1995), with the third quartile having a mean concentration of approximately 53 $\mu g/m^3$ PM_{10}.

Three recent studies have reported concentration-response information for associations between PM_{10} and respiratory hospital admissions for pneumonia and COPD, primarily in the elderly. Schwartz (1994d) reported an association between PM_{10} and respiratory hospital admissions for pneumonia and for COPD in Detroit. A quartile plot of pneumonia admissions in the elderly, Figure V-6, appears to suggest that clear and consistent risk from PM_{10} is associated with the third quartile, with a mean of approximately 52 $\mu g/m^3$. For COPD admissions, which are less frequent than pneumonia admissions, the concentration-response relationship is less consistent (Figure V-7). Increased risk is apparent at the second quartile, with a mean of approximately 34 $\mu g/m^3$ PM_{10}, decreases but is still elevated for the third quartile (52 $\mu g/m^3$ PM_{10}), and clearly increases above the third quartile. Thus, for respiratory hospital admission in Detroit, risk appears to clearly and consistently increase at 52 $\mu g/m^3$ PM_{10} and above, with a likelihood of elevated risk occurring at concentrations below that point, especially for COPD admissions.

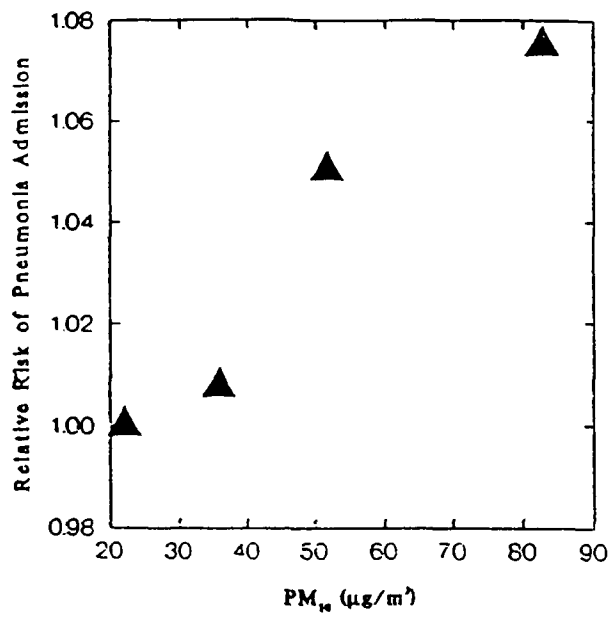
Schwartz (1994e) reported associations between PM_{10} and respiratory hospital admissions in Birmingham. Nonparametric smoothed curves of pneumonia admissions in the elderly (Figure V-8) and COPD admissions (Figure 6 in Schwartz, 1994e) appear to show increasing risk throughout the range of the concentration-response relationship. When nonlinear terms were compared with a linear term for PM_{10}, no statistical significant improvement in model fit was seen (Schwartz, 1995e). Taking into account the lack of nonlinearities, staff considers that a clear and consistent increase in risk can most appropriately be judged as occurring within the lower range of concentrations from 45

FIGURE V-5. RELATIONSHIP BETWEEN RELATIVE RISK OF DEATH AND PM-10 IN BIRMINGHAM (SCHWARTZ, 1993a)



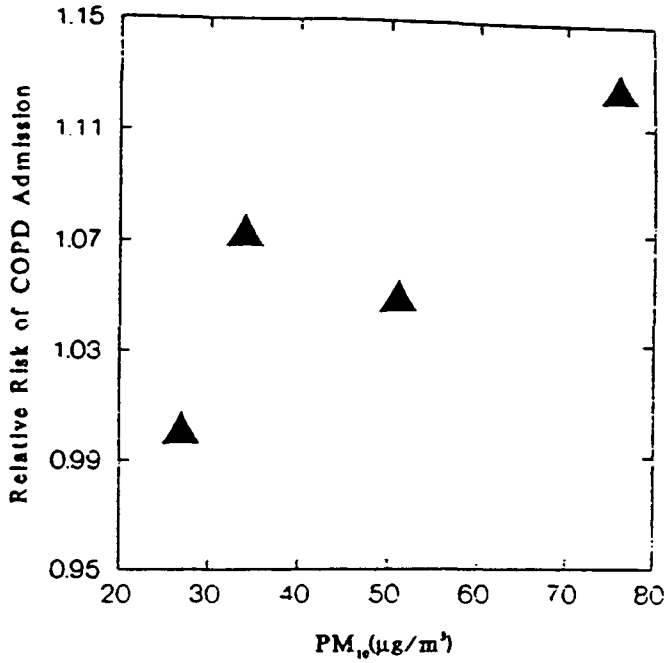
The smoothed plot of the relative risk of death versus PM10 in Birmingham, Alabama, after controlling for smoothed functions of time, temperature, and dew-point temperature (and day-of-week dummy variables) in a generalized additive model. Pointwise one-standard-error confidence intervals are also shown.

FIGURE V-6. RELATIONSHIP BETWEEN RELATIVE RISK OF PNEUMONIA ADMISSION AMONG THE ELDERLY AND PM-10 IN DETROIT (SCHWARTZ, 1994d)



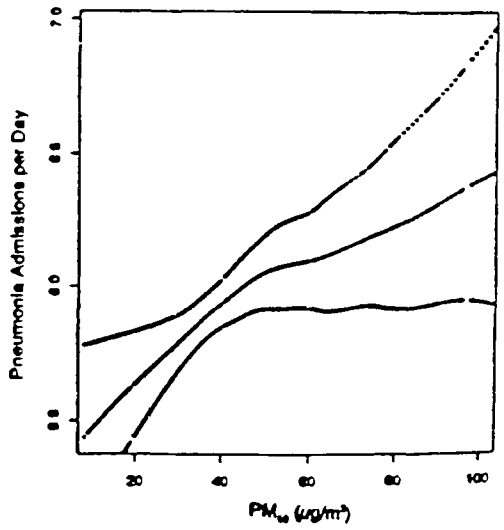
The relative risk of pneumonia admissions in the elderly in Detroit, Michigan, by quartile of PM_{10} is shown. The plot is after adjusting for all other covariates. A stepped response with increasing dose is evident, with no evidence for a threshold.

FIGURE V-7. RELATIONSHIP BETWEEN RELATIVE RISK OF COPD ADMISSIONS AMONG THE ELDERLY AND PM-10 IN DETROIT (SCHWARTZ, 1994d)



The relative risk of COPD 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 V-8. 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.

$\mu g/m^3$, the median concentration, down to 20 $\mu g/m^3$, where the lower 95% confidence interval for this nonparametric smoothed curve includes no increase in risk. Additional uncertainty in the relationship may result from the lack of inclusion of ozone as a covariate, although ozone was not found to have a statistically significant association with hospital admissions in Birmingham (Schwartz, 1994e). The nonparametric smoothed curve for COPD admissions is similar but steeper to that for pneumonia admissions, although COPD admissions are a more infrequent outcome.

Cardiac hospital admissions have also been associated with PM_{10} for the elderly in Detroit (Schwartz and Morris, in press). Figure V-9 shows a quartile plot of the relationship between PM_{10} and hospital admissions for ischemic heart disease, controlling for carbon monoxide and other covariates. Increased risk is apparent at the second quintile, with a mean of approximately 37 $\mu g/m^3$ PM_{10}.

Schwartz et al. (1994) reported associations for PM_{10} and $PM_{2.5}$ with respiratory symptoms in children, including cough and lower respiratory symptoms, across six cities. The nonparametric smoothed plot presented in the study for the PM_{10} relationship with cough is shown in Figure V-10. Cough incidence increases throughout the range, and the slope of increase appears to be diminished at the lowest PM_{10} concentrations. However, the overall relationships for cough and for lower respiratory symptoms were tested for deviations from linearity, which was not found to be significant (Schwartz et al., 1994), implying substantial nonlinearities were not present in the relationship between PM_{10} and cough and lower respiratory symptoms. Interpretation of this plot is made more difficult by the absence of confidence intervals. Staff judges that clear and consistent risk can most appropriately be characterized as occurring at or below the median PM_{10} concentration of 30 $\mu g/m^3$, where statistical uncertainty would be expected to be the least. With regard to lower respiratory symptoms, elevated risk is observed across almost the entire range of PM_{10} concentrations (Figure 3 in Schwartz et al., 1994), but caution in interpretation is appropriate given the relatively small number of observations for this health endpoint.

Pope and Dockery (1992) found PM_{10} to be associated with upper respiratory symptoms, lower respiratory symptoms, and cough in Utah Valley during a winter of

FIGURE V-9. RELATIONSHIP BETWEEN ISCHEMIC HEART DISEASE ADMISSIONS AMONG THE ELDERLY AND PM-10 (SCHWARTZ AND MORRIS, IN PRESS)

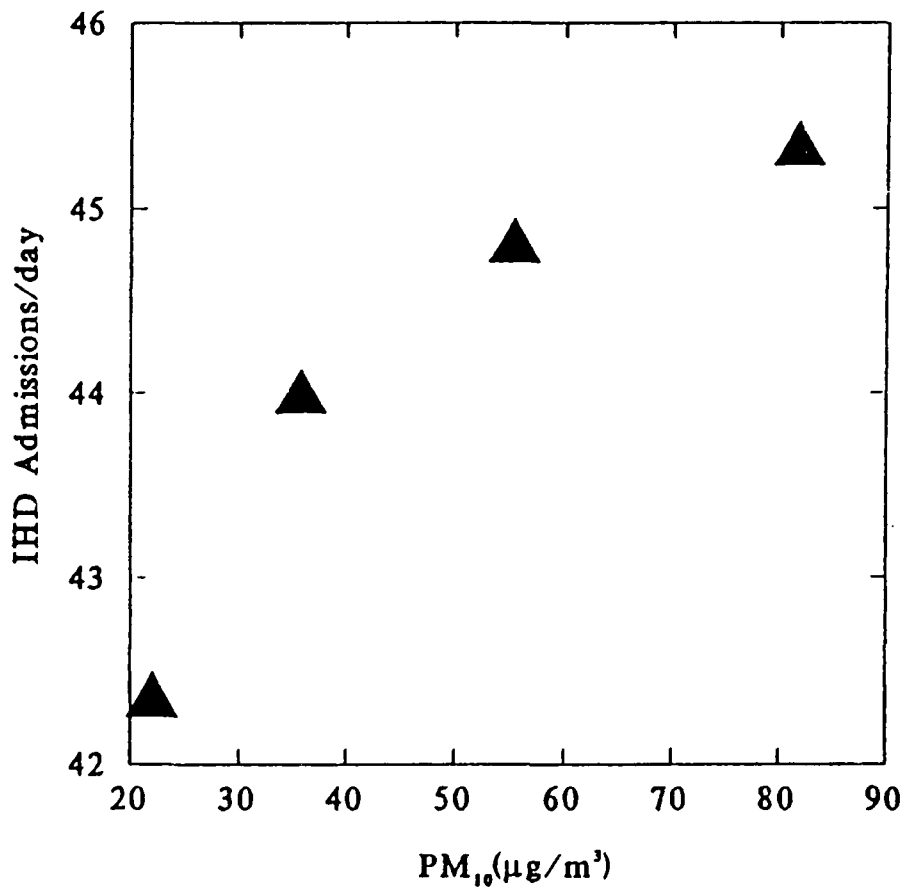
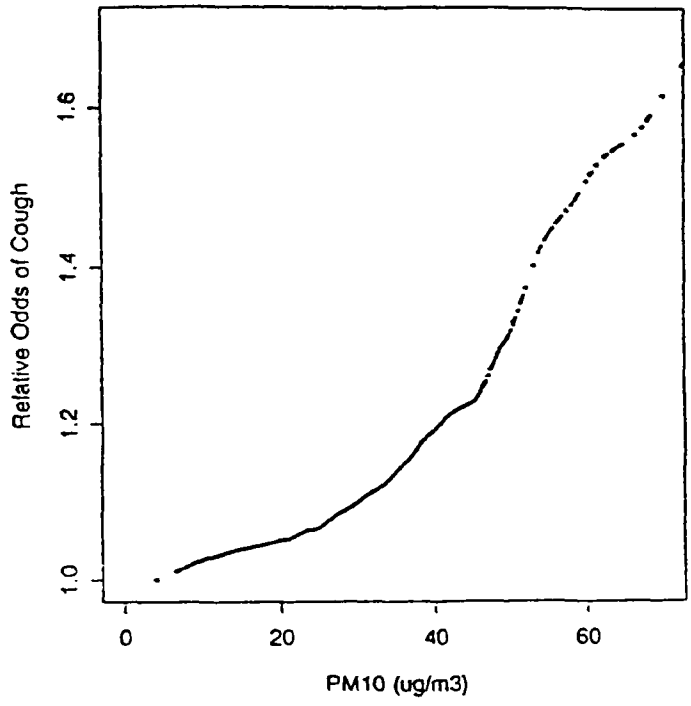


FIGURE V-10. RELATIONSHIP BETWEEN THE ODDS OF COUGH INCIDENCE VERSUS PM-10 CONCENTRATION FROM THE SIX CITY STUDY (SCHWARTZ ET AL., 1994)



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 V-11. RELATIONSHIP BETWEEN PERCENT OF CHILDREN REPORTING SYMPTOMS AND PM-10 IN UTAH VALLEY (POPE AND DOCKERY, 1992)

MEAN % REPORTING RESPIRATORY SYMPTOMS FOR QUARTILES OF PM_{10} LEVELS*

			Mean % Reporting					
			Symptomatic			Asymptomatic		
Quartile	No. of Days	PM_{10} Mean†	Upper	Lower	Cough	Upper	Lower	Cough
1	25	25	21	11	12	17	10	9
2	25	55	27	13	16	18	11	11
3	25	89	27	16	18	15	10	11
4	25	141	33	20	24	18	14	16

* The quartiles were based on 5-day moving averaged PM_{10} levels, which ranged from 13 to 209 $\mu g/m^3$, with the 1st, 2nd, and 3rd quartiles equal to 39, 67, and 107 $\mu g/m^3$, respectively.

† This equals the mean PM_{10} level for the 25 days in each quartile.

relatively high particle concentrations. The associations were stronger for children identified as having a history of mild respiratory symptoms. Figure V-11 shows the percent incidence of health effects by quartile of PM_{10}. Risk for all three respiratory symptom categories increases consistently for children with symptomatic histories at the second quartile of PM_{10}, which averaged 55 $\mu g/m^3$, with risk not increasing in asymptomatic children until the fourth quartile.

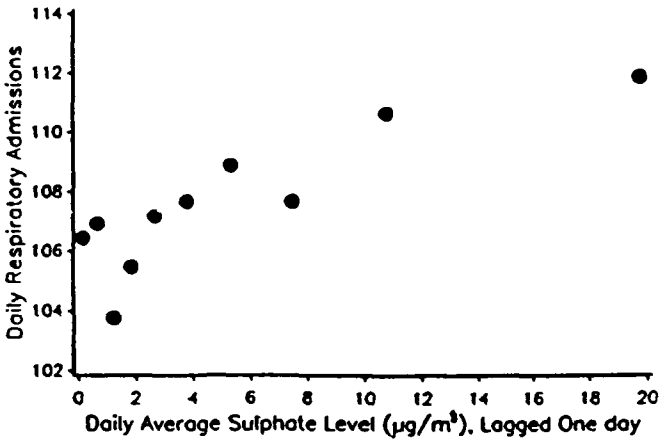
b. Sulfate Studies

Sulfates and acid aerosols have been reported in a number of studies to be associated with respiratory hospital admissions, as summarized in Table V-9. Two studies particularly useful for assessing quantitative concentration-response relationships have reported an association between sulfates and hospital admissions for all ages while simultaneously controlling for ozone (Burnette et al., 1994, 1995). In these studies, sulfates were reported to be associated with respiratory and cardiac hospital admissions in 168 hospitals across Ontario. Figure V-12 shows a decile plot of daily respiratory admissions and sulfate level from these studies. By inspection, this plot suggests that the observed risk for respiratory hospital admissions becomes more clearly and consistently elevated within a concentration range of 4-8 $\mu g/m^3$ as sulfate. From a separate analysis, the daily average hospital admission rates for respiratory and cardiac illness, associated with the third quartile of sulfate concentrations (averaging 4.1 $\mu g/m^3$) were reported to be significantly different from the corresponding rate for the lower 50% of concentrations (averaging 1.5 $\mu g/m^3$) (Burnette et al., 1995).

c. TSP Studies

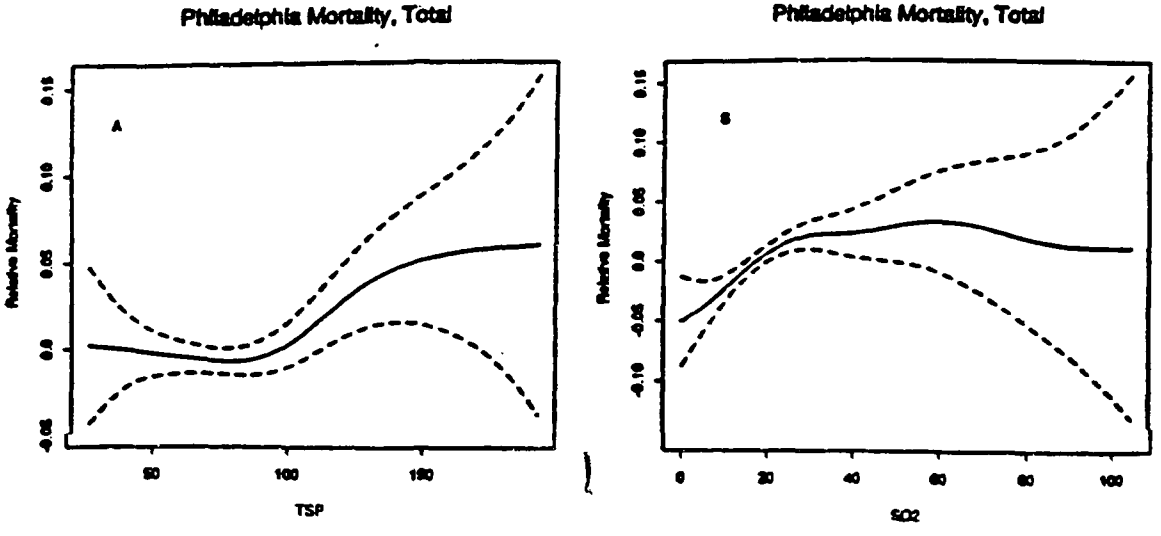
A number of studies have been conducted examining the relationship between TSP and various health endpoints, especially mortality. In general, because of the inclusion of non-inhalable particles within the TSP indicator, the CD concludes that TSP is a less reliable indicator for evaluating health effects of PM. However, because of the substantial amount of daily air quality data for TSP and other pollutants available in Philadelphia, several investigators have extensively reanalyzed this database with respect to associations between air pollution and premature mortality in Philadelphia (Samet et al., 1995, Moolgavkar et al.,

FIGURE V-12. RELATIONSHIP BETWEEN RESPIRATORY HOSPITAL ADMISSIONS AND SULFATE CONCENTRATIONS IN ONTARIO (BURNETTE ET AL., 1994)



Average number of adjusted respiratory admissions among all 168 hospitals bydecile of the daily average sulfate level ($\mu g/m^3$) lagged 1 day

FIGURE V-13. RELATIONSHIP BETWEEN RELATIVE MORTALITY AND TSP OR SO_2 IN PHILADELPHIA (SAMET ET AL., 1995)



Plots of estimated nonparametric effects of TSP and SO_2 on log-relative rates of total and age-specific mortality for Philadelphia, 1973–1980. Curves were estimated using generalized additive models with 4 degrees of freedom per predictor.

1995, Schwartz and Dockery, 1992; Dockery, Schwartz and Pope, 1995; Li and Roth, 1990).

Samet et al. (1995) reanalyzed the Philadelphia dataset used by Schwartz and Dockery (1992), covering the years of 1973 -1980, to examine in detail the relationship between TSP and other covariates such as weather, season, time trend variables, and SO_2 in explaining variations in daily mortality. The authors largely confirm the findings of Schwartz and Dockery (1992) concerning the association between TSP and daily mortality in Philadelphia, with important differences concerning the effects of season and the relative effects of TSP and SO_2. In terms of concentration-response relationships, Samet et al. (1995) find some evidence that a nonlinear, nonadditive surface of TSP and SO_2 better predicts mortality than the linear additive models used by the original investigators. When TSP and SO_2 were considered simultaneously in an additive model but allowed to vary nonlinearly, TSP was associated with log-relative mortality most clearly at concentrations of 100 $\mu g/m^3$ and above, while SO_2 exhibited a roughly linear increase in the log-relative mortality rate until about 40 parts per billion, after which there was little change (Figure V-13). The authors themselves do not interpret the possible significance of an association with SO_2 that is restricted to the lowest concentrations.

Drawing from the discussion of confounding in section V.D., the staff believes it would not be plausible to interpret these results as indicating an association between SO_2 acting alone and mortality. Dockery et al. (1995) suggest that the most parsimonious interpretation is that both pollutants are likely serving as surrogates for the real pollutant of concern. They suggest fine particles are likely to be more closely associated with the observed health effects. Given that TSP by definition includes particles too large to be inhaled, and, thus, is clearly an imperfect surrogate for particles of interest for respiratory effects, it would not be surprising that other indicators such as SO_2, often associated at low concentrations with fine particles, might be better associated than TSP with observed health effects. Further, they suggest that if two variables are serving as proxies for a third, local nonlinearities, apparent interactions, or both are likely to occur.

Samet et al. (1995) further examined the effects of simultaneously regressing TSP and SO_2, and found that both TSP and SO_2 had independent effects, or, in models with more extensive controls for long-term time trends, that TSP remained significant but SO_2 did not [the original findings of Schwartz and Dockery (1992)]. Furthermore, although graphs of the concentration-response relationship for the later period, 1981-1988, are not provided, Samet et al. (1995) report that the association between TSP and mortality is stronger over this period than the association between SO_2 and mortality. Based on the analyses and interpretations discussed above, to the extent that TSP can serve as a surrogate for inhalable or fine particles that may be associated with the observed mortality effects, analyses of TSP in Philadelphia do not appear to suggest a clear and consistent increase in mortality risk until concentrations of approximately 100 $\mu g/m^3$ TSP.

3. Concentration-Response Relationships from Long-Term Studies

Several studies reporting associations between long-term (annual average) exposure to PM and health effects, listed in Table V-20, also provide quantitative concentration-response information showing the pattern of increased risk within the range over which increased relative risk is reported. Included in this list of selected studies are studies that report associations between PM, as measured by $PM_{2.5}$, PM_{15}, and TSP, and mortality and aggravation of chronic respiratory symptoms. These selected studies are part of the much larger body of studies that have been evaluated in the CD and summarized in section V.C. They are discussed below with regard to the nature of the concentration-response relationships observed in the studies and the insights that can be gained with regard to the PM concentrations where clear and consistent increases in risk can be discerned.

As discussed in section V.D., studies of long-term exposures are subject to different types of confounders and employ different means of controlling for confounding than short-term exposure studies. In addition, the evaluation of concentration-response relationships specifically from studies of health effects due to long-term exposures is subject to additional uncertainty related to specification of relevant exposures.

With regard to controlling for confounding variables in long-term studies, the recent prospective cohort studies represent a substantial advance in study design in comparison with

TABLE V-20. CONCENTRATION-RESPONSE INFORMATION FROM SELECTED LONG-TERM EXPOSURE STUDIES

Study	Concentration Range ($\mu g/m^3$ of measured pollutant)	Minimum Clear Increased Risk Level ($\mu g/m^3$ of measured pollutant)
$PM_{2.5}$ Studies		
Mortality -- 6 Cities Dockery et al., 1993	11 - 30	15 - <30
Mortality -- ACS 50 Cities Pope et al., 1995	9 - 33	>15
$PM_{2.5}$ / PM_{15} Study		
Bronchitis in Children -- 6 Cities Dockery et al., 1989: PM-15 PM-2.5	20 - 59 12 - 37	38 22[1]
TSP Study		
Chronic Bronchitis -- 53 Cities Schwartz et al., 1993	48 - 130	75

[1]PM-2.5 concentration provided in the study for city (St. Louis), with minimum long-term concentration showing clear and consistent risk (Figure V-16).

previous cross-sectional studies in that information about each subject's age, body-mass index, lifestyle habits, etc., can be taken into account. This improvement is important since these studies are not restricted to one area, but rather compare relative risks in terms of measures of air quality from different cities. As a result, a number of risk factors that can vary across locations are introduced by including subjects from different geographic areas, with potentially different diets, exposures to other environmental variables, age distributions, and other population characteristics. Studies that involve a large number of locations or cities (e.g., Pope et al., 1995, Schwartz et al., 1993) or that assign individual exposure estimates to each member of the cohort (Abbey et al., 1995a) may have a greater likelihood of minimizing residual confounding from unaddressed variables.

With regard to uncertainties in specifying relevant exposures, questions arise as to the significance and levels of air quality concentrations prior to the study period, and as to the appropriateness of the averaging time used by the investigator. Given the decreasing patterns of PM concentrations observed over this century, associating risk observed in a recent study period solely with contemporaneous air quality could serve to magnify, on a $\mu g/m^3$ mass basis, the apparent risk of current PM levels. This uncertainty is addressed in the CD in terms of the potential implications of using longer historical periods of exposure in analyzing long-term relative risks.

a. $PM_{2.5}$ Studies

The most significant recent studies concerning long-term effects of exposure to PM are two prospective cohort studies that have directly examined the relationship between $PM_{2.5}$ and mortality in cohorts of individuals across a number of U.S. cities. In a study of mortality over a 14-16 year period involving 8,111 adults living in six U.S. cities, Dockery et al. (1993) reported an association between the average $PM_{2.5}$ concentrations in these cities and mortality. Concentration-response information from this Harvard Six City study was provided in the form of a plot of increased risk of death by city as a function of the annual average $PM_{2.5}$ concentration (Figure V-14). This plot shows the relationship between $PM_{2.5}$ concentrations and differences in death rates across the cities among the cohorts after controlling for confounders. The observed association is highly significant ($p < 0.005$), with

FIGURE V-14. RELATIONSHIP BETWEEN MORTALITY RISK RATE RATIOS AND PM-2.5 IN THE SIX CITY STUDY (DOCKERY ET AL., 1993)

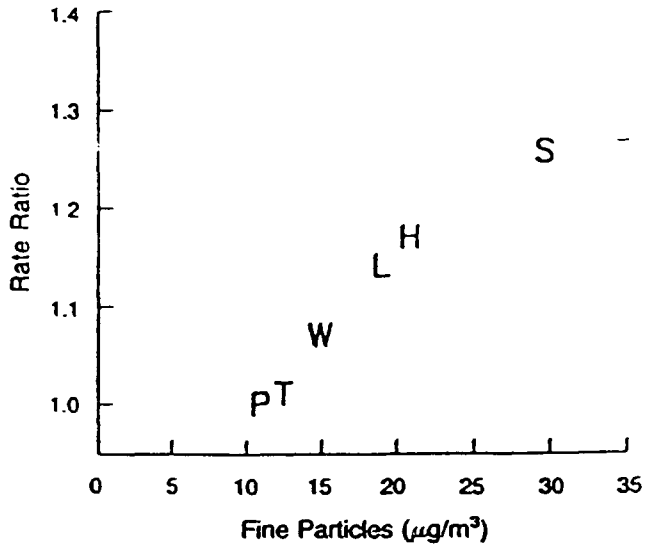
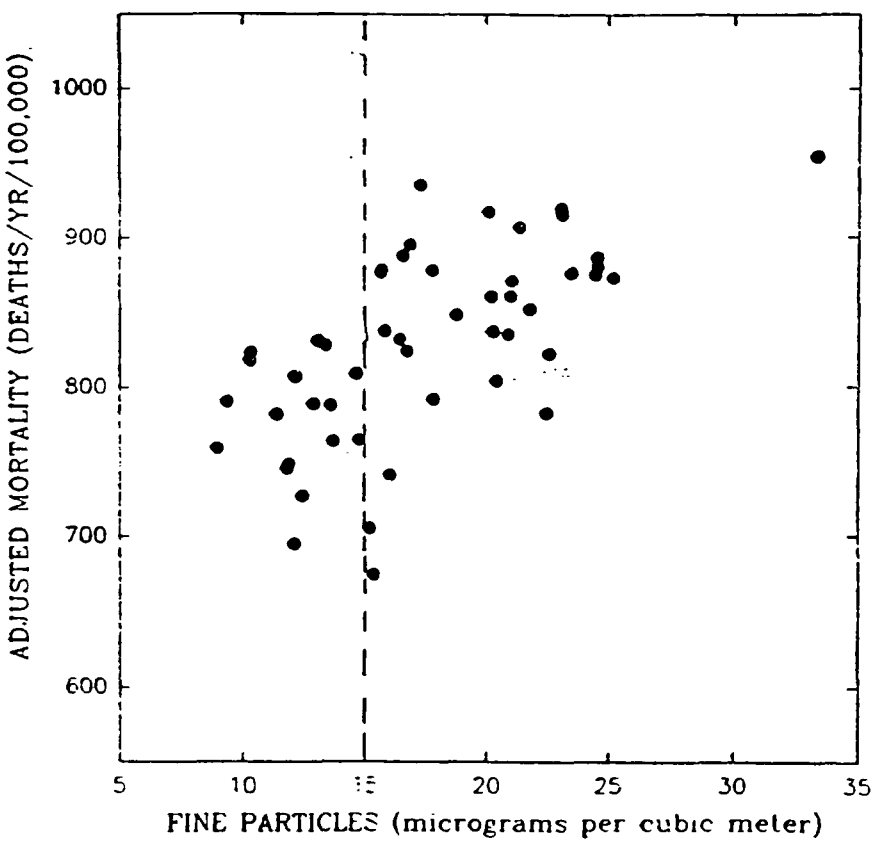


FIGURE V-15. RELATIONSHIP BETWEEN ADJUSTED MORTALITY AND PM-2.5 IN THE AMERICAN CANCER SOCIETY STUDY (POPE ET AL., 1995)



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.

apparent linearity across the cities, and the comparison of differences in death rates between the highest and lowest cities (1.26) is in exact agreement with the relative risk from the reported regression equation. The clearest increased risk is observed for Steubenville, at 30 $\mu g/m^3$ long-term $PM_{2.5}$ concentration, the only city for which statistical significance on an individual city basis was obtained. However, the highly significant relationship identified across all six cities suggests that increased risk of mortality in this study is likely to be associated across a wider portion of the range than only for the city which independently was statistically significant. From this plot, it appears that a clear and consistent pattern of increased risk is evident for the four cities with the highest annual average $PM_{2.5}$ concentrations. Of the four cities showing clear increase in risk, the lowest concentration among these four cities is associated with Watertown, MA, which has an average concentration of approximately 15 $\mu g/m^3$ $PM_{2.5}$.

A particular strength of this study is the long period of air quality measurements it incorporates, and the fact that these measurements came from monitors specifically sited to represent the exposure of the cohort as a whole. For fine particles, 6 to 9 years of $PM_{2.5}$ concentrations were available, beginning in 1980 (four to six years into the study), and ending in 1985-1988 (three to seven years before the end of the study). While it would be ideal to have air quality monitoring extend throughout the period of study as well as considerably before, only approximately one-fifth of the deaths had occurred in the cohort before fine particle monitoring had begun. For other particle indicators evaluated in this study, such as TSP, measurements went further back in time, but measurements of $PM_{2.5}$ extended furthest into the later years of the study, in which mortality was the greatest (Figures 1 and 2 in Dockery et al., 1993).

Questions remain as to whether previous fine particle concentrations for these cities varied substantially from concentrations monitored in this study. Nearly all the cities showed a slight decrease in fine particle concentrations over the course of the study (Figure 1 of Dockery et al., 1993). Assessment of historical levels of fine particles, as indicated by airport visibility measurements discussed in the CD, suggests that, unlike indicators of larger particles which may have been at dramatically higher concentrations in several cities during

the 1960s, fine particles are likely to have been roughly equal to or only somewhat higher than the concentrations measured during the study. This comparison is necessarily uncertain due to the use of airport visibility data as a surrogate for $PM_{2.5}$ and the potential for significant variability in long-term $PM_{2.5}$ trends among cities. Nevertheless, even if longer-term historical concentrations are relevant for a substantial portion of the cohort, it is not expected that inclusion of historic $PM_{2.5}$ concentrations would substantially alter the nature of the associations between $PM_{2.5}$ and mortality risks seen in the study, although the impact on quantitative study results is uncertain.

A large-scale study by Pope et al. (1995) involving from three hundred thousand to half a million adults living in from fifty to one hundred fifty cities nationwide also reported associations between $PM_{2.5}$ and mortality. The ACS study sought to determine if the relationship between mortality and fine particles, as measured by $PM_{2.5}$ and sulfates, observed in the Harvard Six City study, could be confirmed in a larger study including ten to thirty times the number of locations and thirty to seventy times the number of cohort members. The ACS study also collected detailed information on a greater number of potential confounding risk factors than in the Harvard Six City study.

In the ACS study the association between mortality and $PM_{2.5}$ concentrations was evaluated in fifty cities. The $PM_{2.5}$ concentration-response information from the ACS study (Figure V-15) is based on a simple ecologic analysis, rather than a full cohort analysis, including adjustments only for differences among cities in age, sex, and race. In contrast, the concentration-response assessment in the Harvard Six City study used the strength of the prospective cohort approach, comparing $PM_{2.5}$ concentrations against the adjusted mortality rate for each city's cohort, with the adjustment based on the outcome of the control of important risk factors on an individual basis for each individual in the cohort. Thus, the $PM_{2.5}$ concentration-response information for mortality risk in the ACS study does not incorporate the degree of control for confounding that the Harvard Six City study does, even though the full ACS study cohort analysis incorporates control for more potential risk factors on an individual basis than in the Harvard Six City study.

In further comparison to the Harvard Six City study, the estimates of long-term $PM_{2.5}$ concentrations in the ACS study are likely to be somewhat more uncertain because the duration of monitoring was not as long, and the monitors used had not been sited with the population exposure of the cohort in mind. Data from the inhalable particle network for the period 1979-1983 was used to estimate long-term exposure of the cohort in relation to mortality in the cohort occurring from 1982-1989.

While recognizing these uncertainties, the ecological analysis from the ACS study does appear to suggest that risk increases clearly and consistently at median $PM_{2.5}$ concentrations above 15 $\mu g/m^3$ $PM_{2.5}$. Given the uncertainties inherent in its analyses of concentration-response information, the ACS study, drawing from a much larger number of cities, can be characterized as supporting the findings of Dockery et al. (1993) concerning clear and increased risk above 15 $\mu g/m^3$ $PM_{2.5}$.

b. Supporting Studies With Other Indicators

Two additional long-term studies using other indicators of PM, including PM_{15} and TSP, provide concentration-response information for associations with respiratory illness in children and adults. Although these studies appear more uncertain or potentially less useful for assessing concentration-response relationships with fine particles, they are included here to provide information on whether the ranges of increased risk observed from the cohort mortality studies are consistent with relevant findings from other studies of associations between long-term exposure and chronic morbidity.

The effects of long-term exposures to particles on respiratory symptoms in children in the six cities has been studied in relation to a number of PM indicators (Dockery et al., 1989). PM_{15} was observed to be statistically significantly associated with increased acute bronchitis symptoms among the entire cohort, with $PM_{2.5}$ showing the second strongest but marginally significant association (95% CI for Odds Ratio = 0.8 to 5.9). When a subset of children with a history of asthma or persistent wheeze was analyzed separately, PM_{15} and $PM_{2.5}$ showed consistent, marginally significant associations (CI= 0.9 - 15.5 for PM_{15}; 0.9 - 13.2 for $PM_{2.5}$). This study used the same monitoring network as did Dockery et al. (1993), which was specifically sited to measure the cohort's exposure to fine particles. Because this

study is restricted to children 7-11 years of age, concerns about the relevance of previous air quality are diminished.

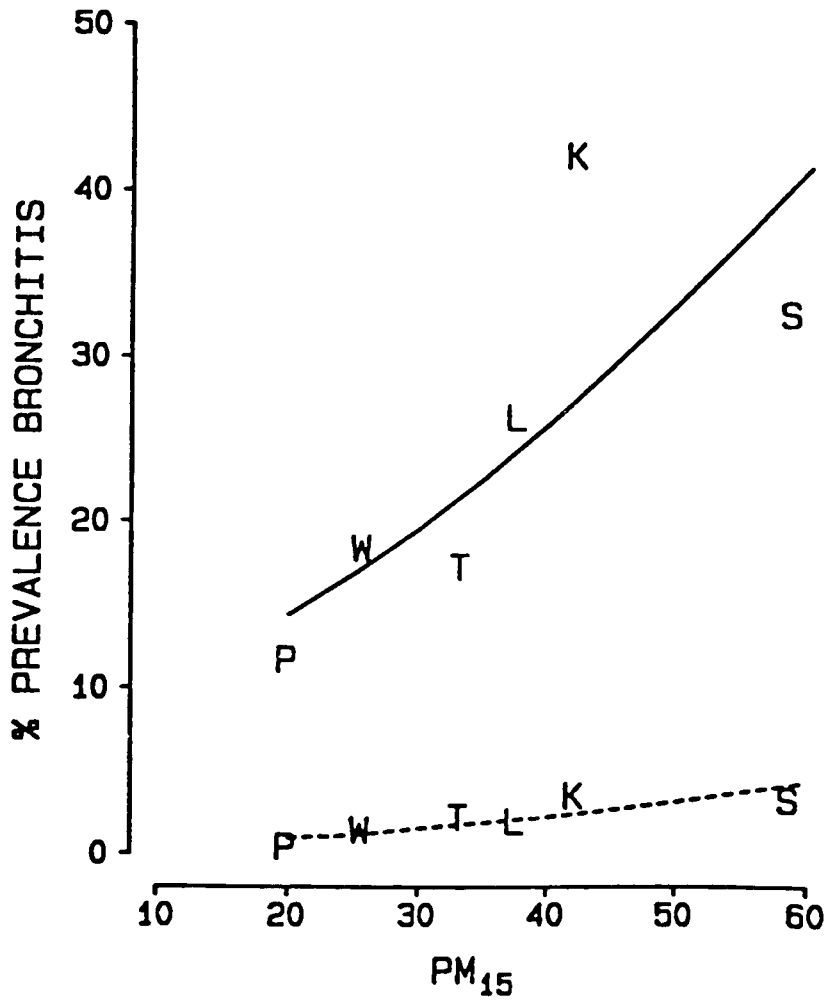
Given that in five of the six cities $PM_{2.5}$ comprises between 58 to 68% of the PM_{15} mass, $PM_{2.5}$ most likely contributes at least partially to the PM_{15} associations seen with bronchitis symptoms. Speizer (1989) suggests that the effects on bronchitis symptoms observed in this study might be most strongly associated with acid aerosols (H+), a component of the fine particle fraction (Figure 3 in Speizer, 1989; Figure 12-16 in the CD). This may be especially true for the cities with the highest particle concentrations. However, the one city that shows relatively large coarse particle contributions, Topeka, has an elevated prevalence of bronchitis as well.

Figure V-16 shows a plot of the prevalence of bronchitis versus PM_{15} concentration, with the solid line representing those with a history of asthma or persistent wheeze. The cities with the three highest concentrations, where the magnitude of increased risk is greatest, had annual mean PM_{15} concentrations ranging from 37 - 58 $\mu g/m^3$ and $PM_{2.5}$ concentrations ranging from 22 - 37 $\mu g/m^3$, although the pattern of increased risk is not consistent across these three cities.

A cohort study involving information from the First National Health and Nutrition Survey (NHANES) of 6000 adults in 53 cities adults reported associations between chronic bronchitis and a physician-diagnosed respiratory illness and TSP (Schwartz et al., 1993). Figure V-17 shows the relative risk of chronic bronchitis versus TSP concentration, and Figure V-18 shows the relative risk of doctor diagnosed respiratory illness versus TSP. In both, risk appears to be increasing clearly and consistently at the second quartile, with a mean concentration of approximately 75 $\mu g/m^3$ TSP.

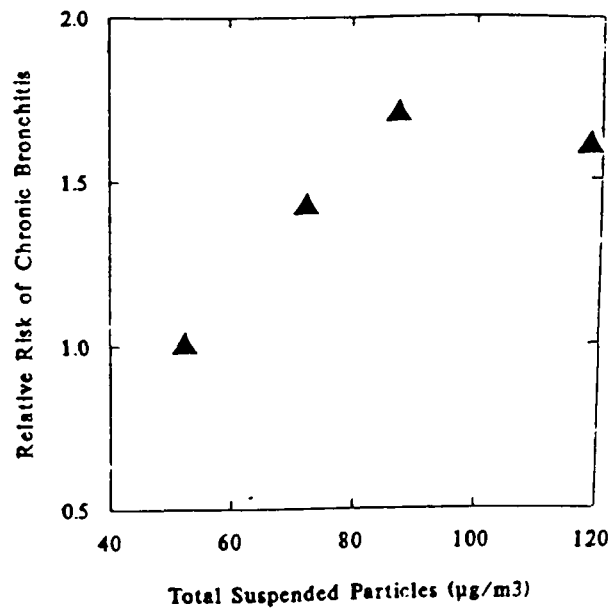
This study is based only on the TSP concentrations from the year prior to the NHANES assessment of effects. To the extent that chronic bronchitis prevalence may involve longer exposures, and that past TSP concentrations were potentially much higher in some of the study area cities, these concentration-response relationships may contain significant uncertainty in the specification of relevant exposures. To the extent that TSP may be serving in part as a surrogate for fine particles, however, the uncertainties in the

FIGURE V-16. RELATIONSHIP BETWEEN BRONCHITIS PREVALENCE AND PM-15 IN THE SIX CITIES STUDY (DOCKERY ET AL., 1989).



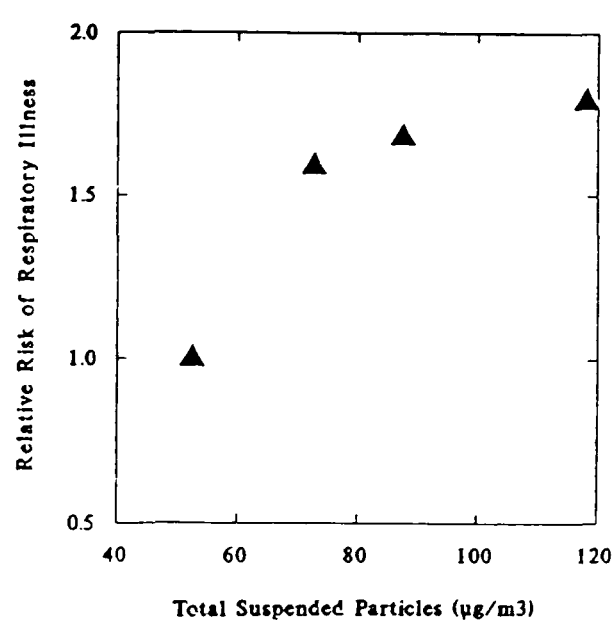
City-specific prevalence of reported bronchitis versus annual mean PM_{15} concentrations ($\mu g/m^3$) stratified by reported asthma or persistent wheeze. Upper curve (*solid line*) is the logistic fit for children with reported asthma or wheeze, and lower curve (*dashed line*) is the logistic fit for those without (see figure 1 for city labels).

FIGURE V-17. RELATIONSHIP BETWEEN RELATIVE RISK OF CHRONIC BRONCHITIS AND TSP FOR NHANES I SURVEY SUBJECTS (SCHWARTZ, 1993b)



The relative risk of chronic bronchitis by quartiles of TSP exposure, after controling for age, race, sex, and cigarette smoking.

FIGURE V-18. RELATIONSHIP BETWEEN RELATIVE RISK OF RESPIRATORY ILLNESS AND TSP FOR NHANES I SURVEY SUBJECTS (SCHWARTZ, 1993b)



The relative risk of a diagnosis of respiratory illness by the examining physician by quartile of TSP exposure, after controlling for age, race, sex, and cigarette smoking.

concentration-response relationships would be expected to be less, given the smaller variation seen in historical fine particle trends.

VI. STAFF CONCLUSIONS AND RECOMMENDATIONS ON PRIMARY NAAQS

This chapter presents staff conclusions and recommendations for the Administrator to consider in deciding whether to retain or revise the PM NAAQS. Drawing from the information and analyses discussed in Chapters IV and V, this chapter addresses the major components needed to specify an ambient standard: averaging time, form, pollutant indicator, and level. Staff conclusions and recommendations on each of these interrelated components are based on considering how the components of an individual standard and how 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 judgement. A final decision must draw upon scientific information about health effects and risks, as well as judgements about how to deal with the range of uncertainties that are inherent in the 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 health effects are likely through levels at which scientists generally agree that effects may occur but the likelihood and magnitude of the response becomes 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.

A. Averaging Time and Form of the Standards

The current primary PM NAAQS include both a 24-hour standard with a statistical form and an annual standard with an 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 that time. Since the last review, numerous researchers have extended the epidemiological database linking health effects with both short-term (from less than 1 day to up to 5 days) and long-term (from generally a year to several years) exposures to PM. This body of

evidence, summarized in Chapter V, provides increased support for both short-term and long-term standards, as discussed below.

1. Short-term Standards

a. Averaging Time

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. In some geographic areas the observed health effects are 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. The 24-hour averaging time effectively protects 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 a longer averaging time, such as 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, it is not clear whether the majority of effects that have been associated with daily exposure to PM, including mortality and various measures of morbidity, would occur after

only short duration exposures. Moreover, 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. Further, the staff believes that additional research is needed to examine short duration exposures.

Based on the above discussion, the staff recommends that consideration be given to retaining the current 24-hour averaging time as the most appropriate to address health effects associated with short-term (from less than 1-day to up to 5-day) exposures to PM.

b. Form

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 was selected to be a one-expected-exceedance form, 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 and, thus, recommends that consideration be given to retaining the current statistical form for a 24-hour PM standard.

2. Long-term standards

a. Averaging Time

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) and respiratory symptoms and illness (e.g., chronic bronchitis and cough in children). The CD recognizes the importance of the presence of PM in the lungs because of associated public health concerns about both immediate and longer term symptoms produced and the long-term potential for increased risk 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) and is also increased by PM exposure. Therefore, serious health consequences are associated with long-term PM exposures.

In addition, human lung deposition data show that both coarse and fine fractions of PM_{10} are able to penetrate and deposit in the gas exchange portion of the lung, an area in which insoluble particles are retained for long periods (i.e., months to years). Given that particles can be retained in the lung for long periods of time, several of the plausible mechanisms of toxicity support the need for a long-term averaging time to protect against health effects. Specifically, it is possible that accumulation of large lung burdens of particles over time could be a possible mechanism or that long-term exposures could lead to impaired respiratory defenses. Moreover, chemically active substances on particle surfaces, such as acids or carcinogens, could cause damage before they are cleared, which, if not repaired, could lead to cumulative damage. Although the specific mechanisms of toxicity of PM remain speculative at this time, the staff believes that information on lung deposition and clearance provides additional support for concern with long-term PM exposures.

The staff has also considered whether, for some effects (e.g., chronic mortality), relevant exposure periods might better reflect the cumulative effects of PM exposures over a number of years. In such cases, an annual average would provide effective protection against long-term exposures to PM that exceed a year. Further, the staff has considered that air quality studies show significant seasonal variability in exposures to PM. However, the staff believes that the available community studies do not support a clear association for seasonal exposures that would be distinct from 24-hour or annual exposures.

As discussed above, the staff believes that an annual averaging time is consistent with the effects observed in community epidemiological studies, as well as insights into potential effects of long-term exposure from studies of toxicology and dosimetry. Further, the staff does not believe that multi-year or seasonal averaging times would be more effective or supportable than an annual averaging time. Thus, the staff recommends that consideration

be given to retaining the current annual averaging time as the most appropriate to address health effects associated with long-term exposures to PM.

b. Form

As part of the last review, the annual standard was changed from a geometric mean to an arithmetic mean of the daily averages. 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.A. EPA, 1982b). The staff continues to believe that this rationale is sound and, thus, recommends that consideration be given to retaining the current arithmetic average form for an annual PM standard.

B. Particulate Matter Indicator

1. General Considerations

Faced with clear evidence of adverse health effects in heavily polluted areas in the decades after World War II, public health authorities in the U.S. and Great Britain pushed for reductions in general particulate matter as indexed by available monitors, despite the fact that the identity of causative agents and mechanisms was not well understood. That these efforts were successful is evidenced in the elimination of classical pollution episodes, greatly improved air quality, and reduced health risks (U.S. EPA, 1982b). As the review of U.S. air quality trends in Chapter IV indicates, these improvements have continued under the current PM_{10} standards, in areas with differing combinations of particulate and gaseous air pollution. Furthermore, concentrations of some of the more innately toxic components (e.g., trace metals, benzo(a)pyrene (BaP)) also declined in previously polluted areas (U.S. EPA, 1982b). Previous decisions to control particles as a general class, although heterogeneous in physical and chemical composition, appear to have been good public health policy even though "particulate matter" is toxicologically undefined. Based of the evaluation of the updated scientific information in this review, the staff continues to believe that separate general PM standards (as opposed to combined PM and SO_2 standards) remains an appropriate policy choice for protecting public health.

The most recent summary of scientific information in the CD and outlined in Chapters IV and V also 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 large body of new community studies and improvements in human exposure and air quality presented in the CD and outlined in Chapters IV and V above, however, have significantly expanded the information regarding associations between contemporary community air pollution containing particles and morbidity and mortality in sensitive populations. Even with the presence of other pollutants in the airsheds 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 information on PM_{10}, fine particles, coarse fraction particles, and acid aerosols. Because statistical associations between these indicators and health indices have been observed at concentrations below the current standards, the adequacy of both the indicator and the levels for the PM_{10} standards have been questioned (Schwartz and Dockery, 1992a; Dockery et al., 1993; Lippmann and Thurston, 1995). If the constituents other than the most harmful fractions of PM_{10} are captured by samplers and targeted for reduction by health protection strategies, less effective health protection will occur. Therefore, proper identification of the components of PM_{10} most likely responsible for the observed health effects is critical to maximize 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 provide for more effective protection of public health.

2. Alternative Refinements for PM_{10} Indicator

The staff bases its conclusions and recommendations for indicator(s) on the integration of information in three key areas:

- Assessment of the totality of the evidence from epidemiology, toxicology and human clinical studies;
- Consideration of the information on dosimetry and potential mechanisms of toxicity; and
- Air quality and exposure analyses related to interpretation of the health studies.

Key aspects of each of these related areas have been drawn from the studies contained in the CD and summarized in Chapter V. Based on these consideration as well as the earlier assessment of indicators (U.S. EPA, 1982b; 1986b), the staff has identified the following major policy options for further refinements to the PM_{10} indicator:

- Specifying an additional size division based on the observed bimodal ambient air size distribution, that is, between fine and coarse fraction particles less than 10 μm; and,
- Adding a chemical class indicator based on sulfates and aerosol acidity.

Each of these approaches is discussed and evaluated briefly below. The Administrator must select the appropriate indicator(s) and consider how the indicator will relate to providing health protection in conjunction with the other components of a standard (i.e., averaging time, form, and level).

a. Indicators for the Fine and Coarse Fractions of PM_{10}

Recognition that ambient particle mass and volume are typically distributed bimodally has long led to the suggestion that the health (and other) effects of the two modes should be treated separately (NAS, 1977; Miller et al., 1979; Wilson and Suh, 1995; Pope et al., 1995; Schwartz et al., 1995). From an atmospheric chemistry perspective, the two modes can be thought of as separate pollutant classes with distinct properties and origins. Although both size fractions are implicated in health effects, the important differences in their physical and chemical properties make it less clear whether the relative risk presented by the two modes should be considered to be equivalent. In the previous review, staff gave serious consideration to separating the two classes, ultimately concluding that the limited number of and nature of the community epidemiology studies available at that time could not be used to support more than a single size specific indicator (U.S. EPA, 1982b; 1986b).

In this review, the staff concludes that the significantly expanded community epidemiology exposure and air quality information provide a sufficient basis for considering separation of fine and coarse fraction particles. In addition, the experience in implementing the PM_{10} standards provides insights as the relative effectiveness and efficiency of standards based on that indicator in limiting the risks presented by fine and coarse fraction particles. As outlined above, staff continues to believe that both fractions of PM_{10} present health risks that must continue to be addressed by ambient standards. Key considerations regarding indicators for each of these fractions are discussed below.

i). Fine Particles

The staff assessment of the basis for a separate fine particle standard is summarized as follows.

1) The fundamental physical and chemical characteristics of the fine fraction are summarized in Chapter IV. Fine particles tend to originate from nucleation (i.e.,

formation of particles from low vapor pressure substances, produced either from combustion or from chemical reaction of gases) and condensation of gases onto existing particles. As a result of their physical properties, fine particles share a number of properties important to assessing health risk. These include physical properties such as high surface area and number, a more uniform distribution in urban and regional scales, long atmospheric lifetimes, and increased ability to infiltrate indoors. Although fine particles are chemically heterogenous, as a group they are generally acidic and a major fraction is soluble and hygroscopic, while the coarse fraction is generally basic and insoluble.

These differences in sources and properties also have a profound influence on the nature of control strategies. Conceptually, the PM_{10} indicator was intended to provide adequate protection from both fine and coarse fraction particles. Areas which exceed the current PM NAAQS have significant amounts of coarse fraction mass. Specifically, the PM_{10} indicator registers a substantial amount of coarse particle mass (e.g., up to 70 percent in the western half of the country where most exceedances of the current standards occur and around 35 percent in the eastern U.S.). In practice, it is often easier for sources and regulators to control preferentially the locally generated coarse fraction. Therefore, it is not clear that a PM_{10} indicator, unless set at an unnecessarily stringent level, provides adequate, efficient protection from the effects of fine particles.

2) Although only qualitative information exists on potential mechanisms of toxicity, both past and current assessments in this area suggest that the distinct physical and chemical properties of fine particles may contribute to enhanced risk for several possible mechanisms. The enhanced effects with decreasing size in some toxicologic studies of laboratory generated aerosols and the substantial surface area for potential adsorption of irritant gases has long been noted as a reason for increased concern for fine particles (Natush and Wallace, 1974; U.S. EPA, 1982b; NAS, 1977a). Although the relevance of the particle composition can be questioned, recent reports of the acute toxicity of freshly generated ultrafine particles at low concentrations show that

severe pulmonary inflammation and death are observed in rats after brief exposures (Oberdörster et al., 1995). In this case, the large number of particles per unit mass may have been critical. Preliminary work using an ambient particle concentrating device developed by Sioutas et al. (1995) finds lethal effects in compromised animals from ambient aerosol between 0.2 and 2.5 μm at much lower concentrations than reported for pure compounds in other laboratory studies.

That common physical properties (surface area and number) shared by fine particles in disparate areas might be instrumental in particle toxicity is also indirectly supported by one of the most interesting features of the recent body of epidemiologic literature. As noted in Chapter V, a large number of studies conducted in cities in the U.S., Europe, and South America have found strikingly similar relative risks for particles when normalized to a comparable indicator and concentration. Substantial differences are observed (or reasonably expected) in the chemical composition of fine and coarse fraction particles and pollutant gases among such areas. Although far from conclusive, this similarity is suggestive that some consistent characteristic of all particles, such as surface area, might be involved if these associations are causal. In this respect, fine particles would present a greater risk than coarse fraction particles.

Several chemical classes of concern such as most of the acids and sulfates are found predominantly in the fine fraction. In general, acids are found to be more acutely irritating than are materials in the coarse fraction. Kleinman et al. (1995) reported the relative toxicity of high concentrations of fine particle components (sulfate and nitrate) in animal tests to be greater than resuspended road dust. Some transition metals and organics associated with the fine fraction have been shown to cause effects in toxicological experiments. Loading particles with certain transition metals such as iron, vanadium, or nickel may have the potential to enhance particle toxicity, acute inflammation, and non-specific bronchial responsiveness. Adsorbed organic compounds may also enhance toxicity of particles. Diesel exhaust particles and gas engine particles are examples of particles whose organic extracts have been proven to be mutagenic and tumorigenic in animals.

3) As noted above, particle deposition in the tracheobronchial and alveolar regions of the human lung is of greatest concern. For quiescent breathing in normal adults, most of the mass of insoluble fine particles tend to penetrate to the alveolar region, where they may accumulate over time. Hygroscopic fine sulfates and acids, however, grow in the respiratory tract, resulting in significant deposition in the tracheobronchial region. Furthermore, soluble particles that deposit in the alveolar region are available to cause damage before they are cleared, damage which if not repaired can contribute to cumulative effects. Both tracheobronchial and alveolar deposition of fine particles may be enhanced for sensitive populations such as individuals with chronic or acute respiratory disease, and for small children. Fine particles penetrate and deposit more efficiently in the alveolar region.

4) Direct epidemiological evidence distinguishing the relative effects of fine versus coarse fraction particles is limited. Nevertheless, a coherent body of epidemiology studies has shown associations of health effects with fine particles measured by $PM_{2.5}$, BS, KM, CoH, and with classes of compounds found mostly in the fine fraction, such as sulfates and associated acids. Associations with fine particles have been reported across the full spectrum of health effects described in Chapter V, including mortality associated with short-term and long-term exposures, hospital admissions, respiratory symptoms, decreases in lung function, school absences, and work absences. These data alone provide a substantial basis for quantitative assessments needed to establish a standard level. In addition, staff believes that the large epidemiological database relating to associations between health effects of short-term elevations of PM_{10} is best interpreted as primarily representing risks from fine particles, as discussed below. Historical pollution catastrophes such as those in London, England, in the 1950's were most likely caused by increases in levels of fine particles.

Several recent studies have shown stronger relationships for $PM_{2.5}$ than PM_{10} with both excess mortality and hospital admissions (Dockery et al., 1993; Pope et al., 1995; Thurston et al., 1994; Schwartz 1995). Studies examining other morbidity endpoints reported statistically significant associations with PM_{10}, although the

associations with $PM_{2.5}$ were suggestive of a trend but not statistically significant (Dockery et al., 1989). The most direct comparison of the acute effects of fine and coarse fraction particles is provided by the analysis of daily mortality in six cities by Schwartz et al., (1995). In this analysis, both $PM_{2.5}$ and PM_{10} were positively and significantly associated with fine particles, while the association with coarse particles was small and insignificant. While this does not show that coarse particles do not cause acute effects, it does indicate the mortality effects are best related to the fine fraction.

Because of their physical properties, fine particles are more likely than coarse particles to contribute to exposures measured in the epidemiology studies. Examinations of air quality aspects of the time series PM_{10} mortality studies[1] also support this suggestion:

- As described in Chapter IV, air quality properties of $PM_{2.5}$ in the areas studied imply that daily measures of PM_{10} are driven by underlying changes in $PM_{2.5}$ levels. First, $PM_{2.5}$ is highly correlated with PM_{10} in study locations. Furthermore, the day-to-day variation of $PM_{2.5}$ concentration is predominantly greater than the day-to-day variation in coarse fraction concentration in these areas; thus, day-to-day variations in PM_{10} levels are driven by variations in fine particle levels, and the coarse fraction is much less variable. Finally, in the Eastern U.S., where most studies were conducted, $PM_{2.5}$ represents a large fraction of the PM_{10} mass (63 percent of mass on average in the Eastern U.S.). In specific Western U.S. cities studies, fine particles were used as measurements (e.g., Los Angeles, CA; Santa Clara County, CA; Denver, CO) or fine particles tend to dominate the PM_{10} measurements (e.g., woodsmoke communities in winter and stagnation conditions of low wind speed in conjunction with combustion sources).

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[1]As described in the Criteria Document (Chapter 12), time series analyses attempt to use statistical techniques to relate short-term variations in air quality measurements to short-term variations in health endpoints over time, typically using a 24-hour period as a unit of measure (e.g., daily PM concentrations and daily deaths or hospital admissions) while controlling for confounding factors.

- Epidemiological studies associate outdoor levels of PM to health endpoints. In general, stationary monitor fine particles levels are better related to personal exposure measures than are coarse fraction levels. The spatially homogeneous nature of fine particles means community-oriented monitors on which most studies are based may better represent fine than coarse particles. Coarse particles are higher near sources and then levels drop off with distance from the source. Thus, the community-based monitor may not represent exposure to coarse particles as well as exposure to fine particles.[2]

  Moreover, outdoor-origin fine particles are better able to infiltrate indoors in air conditioned or heated areas than are coarse particles; once inside, fine particles will remain suspended for longer periods of time. Thus, outdoor fine particles contribute more to exposure because people spend a significant amount of time indoors. It follows that time series associations between variations in particle concentrations at outdoor monitors and both personal exposures and related health effects in populations that spend most of their time indoors are more likely due to fine particles. The recent time series studies providing a direct comparison between outdoor PM_{10} and $PM_{2.5}$ and personal exposure show that both indicators are significantly related to variations in personal exposure, but that fine particles show substantially stronger associations (Janssen et al., 1995). By inference, the coarse fraction association must be smaller even than for PM_{10}.

  Consequently, daily fluctuations in PM_{10} concentrations measured in the epidemiology studies are likely to be better associated with fluctuations in fine particle concentrations. However, insufficient monitoring data prevent this conclusion from being tested directly in most areas. In most of the areas where the relative health significance of fine and coarse fraction particles were

[2]This does not imply that the coarse fraction does not contribute to the observed health effects, only that the time series studies would not be the most effective tool to elucidate coarse fraction associations.

· examined directly, fine particles were more statistically significant and stable predictors of the health effects than coarse fraction particles.

ii. Coarse Fraction of PM_{10}

As noted above, the staff continues to believe in the need to provide against the potential health effects of coarse fraction particles. Coarse fraction particles deposit in both the tracheobronchial and alveolar region. 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.

Although none of the available community epidemiology studies has been able to isolate high concentrations of coarse fraction particles, occupational studies, laboratory exposures and autopsy studies suggest a number of concerns remain about potential risks from the coarse fraction. Occupational studies suggest that some effects, specifically industrial bronchitis, may be associated with prolonged occupational exposure to coarse particle insoluble mineral dusts (e.g., greater than 5 μm aerodynamic diameter) (U.S. EPA, 1982b; Morgan, 1978). Laboratory studies of potential mechanisms find that high levels of coarse insoluble dusts can result in responses such as bronchoconstriction, altered clearance, and alveolar tissue damage (U.S. EPA, 1982b). Autopsy studies of animals and humans exposed to various ambient crustal dusts at or slightly above ambient levels typical in the Western U.S. suggest those exposures result in silicate pneumonoconiosis (U.S. EPA, 1982b). Responses ranged from build up of particles in macrophages with no clinical significance to possible pathological fibrotic lesions.

Although much of the recent epidemiology suggests greater effects from fine particles, it is premature to ascribe all of the effects observed in the PM_{10} studies to fine particles. This is particularly true for effects in children (Schwartz et al., 1994; Dockery et al., 1989), who spend more time in outdoor activity and hence receive less protection from both building and nasal removal mechanisms. In such studies, however, it is not possible to provide separate estimates for coarse fraction effects levels. In addition, the existing ambient data base for coarse fraction particles is quite small. On the other hand, the monitoring network

for PM_{10} is large, and some of the epidemiological studies can be used to assess PM_{10} levels. If fine particle standards were established, then PM_{10} would serve as a *de facto* indicator for coarse fraction particles. As noted above, in many areas, 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. Therefore, if a fine particle indicator were chosen, the staff would still recommend retention of PM_{10} as the indicator to protect against the risks of coarse fraction particles.

b. Chemical Class Indicators -- Sulfates and Acid Aerosols

Another option for a PM indicator is a chemical class such as sulfates and acids aerosols, metals, and organics. Each of these classes is predominantly found in the fine fraction. Following the conclusion of the last PM standards review, the CASAC recommended that special consideration be given to only one of these classes -- acid aerosols. Accordingly, EPA prepared a comprehensive review of acid aerosols, including sulfate aerosols (U.S. EPA, 1989). The review concluded that the scientific and technical basis was not sufficient to support a separate NAAQS for acid aerosols. The more recent information in the CD prompted reexamination of this issue.

As presented in the CD and Chapter V, there are substantial health data concerning sulfates and acid aerosols. This section examines their potential use as PM indicators. Both sulfates and acids reside almost exclusively in the fine particle fraction and are usually closely associated with each other.

Sulfates have a long, well-documented history of associations with health effects. Some of the earliest reports of health effects from PM in the United States used sulfate as the air quality measurement. Sulfates and fine particle mass are highly correlated in many areas of the country, especially in the Eastern U.S. As discussed in the CD and Chapter V, epidemiology associations have been reported between sulfates and mortality and serious morbidity endpoints such as hospital admissions.

Toxicological studies and human clinical studies of pH neutral or nearly neutral sulfate salts, however, have not been able to reproduce the health effects observed in the

epidemiological studies, even at relatively high concentrations (Lippmann and Thurston, 1995). Sulfate aerosols containing strong acids such as sulfuric acid (H_2SO_4) do produce functional and structural changes in healthy subjects consistent with the effects observed in the epidemiological studies (CD, Chapter 11-13). Consequently, although sulfate itself is not innately of health concern, it is closely linked to acid species which have been shown in toxicological, human clinical, and epidemiological studies to be of concern. Based on these factors, some investigators (Lippmann and Thurston, 1995) suggest that sulfates would be an appropriate indicator for PM.

A sulfate indicator would not capture all of the potential agents of concern as effectively as a fine particle indicator. PM associations with daily mortality have been observed in locations where sulfate concentrations would be expected to be low (Fairley, 1990; Schwartz, in press (Spokane); Schwartz et al., 1993). A sulfate indicator would not address these risks as effectively as a general fine particle standard although a sulfate indicator might capture some of the contributing components. While sulfates might be a useful proxy for acidic species in the fine fraction, it would only include these classes. Concern exists for the potential role of transition metals, organic compounds, ultrafine particles, particle surface area, or number. $PM_{2.5}$, on the other hand, would effectively capture all of these agents.

Acid aerosols are currently regulated to varying degrees by current NAAQS for PM, sulfur dioxide, and nitrogen oxides. Recent epidemiology studies have reported associations between acid aerosols and health effects (See CD Table 12-3). However, several PM epidemiological studies in areas where acid concentrations are low or minimal have still seen consistent particle effects (Fairley, 1990; Pope, 1991; Pope, 1992; Dockery et al., 1992). While several studies show acids to be as well or even more strongly correlated with health effects than particle mass measurements, some other studies have found general particle measures to be better correlated (Dockery et al., 1992; Schwartz et al., 1994).

An acid standard would focus health protection measures on acids, leaving out control of organics, transition metals, non-acidic ultrafine particles, and other components of PM of potential concern. While fine or ultrafine acid aerosol clearly may play a role in the

observed health effects associations, the data do not provide support for any single mechanism or pollutant. Therefore, using sulfates or other acid indicators would not address these risks as effectively as a general fine particle standard. Moreover, because sulfates form such a large fraction of fine mass in the Eastern U.S., an appropriate fine particle standard would focus attention on control of sulfates and associated acids.

This review suggests that a large number of different particulate substances may produce a variety of responses in animals and humans. Identification and control of each of the many ambient aerosol components would be difficult to accomplish, time-consuming, and would place excessive monitoring, compliance, and other requirements on effected agencies and industries with no clear potential for improving public health protection over general particulate regulation. Given current knowledge and technical capabilities, separate national standards for chemical classes, including sulfates and acid aerosols, would be difficult to support and implement, again with no obvious advantages for improving health protection over an appropriately set fine particle standard.

3. Staff Conclusions and Recommendations for a Particle Indicator

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

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. The recent health evidence and implementation experience with PM_{10} have, however, prompted the staff to reconsider previous conclusions regarding further refinements to the indicator.

2) Any refinement of the PM_{10} indicator should be based on the integration of the totality of the evidence from epidemiology, toxicology and human clinical studies; consideration of respiratory tract deposition data and potential mechanisms of toxicity; and air quality and exposure analyses.

3) The staff finds that the available information is sufficient to further refine the indicator for the current primary standard indicator, PM_{10}. In order to provide

more effective and efficient health protection, the staff recommends a distinction be made between standards for fine particle and for coarse fraction particles.

4) Movement to chemical class indicators is not advisable during this review. Of the particulate chemical classes, most is known about sulfates and acids. In the past reviews, staff has concluded that insufficient data exist to set a separate national sulfate or acid aerosol standard (U.S. EPA 1989; 1986b; 1982b). The staff continues to concur with these judgments. Additional components of fine particles have also been demonstrated to be of health concern. Because sulfates form such a large fraction of fine mass in many areas, an appropriate fine particle indicator would result in control of sulfate-related species.

Based on these conclusions, the staff makes the following recommendations with respect to reviewing the adequacy of the current indicator for PM:

1) The PM_{10} indicator should be retained. PM_{10} is the most appropriate surrogate for additional protection from potential risks of coarse fraction particles. A separate coarse particle indicator is not appropriate because of the much larger data base associated with PM_{10}.

2) A fine particle indicator should also be added to provide more effective protection against the risk posed by fine particles.

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 particles. Thus, the decision within this range is largely a policy judgement. Based on considerations of consistency with health data, the limited potential for intrusion of coarse particles into the fine fraction, and availability of monitoring technology, the staff recommends using a $PM_{2.5}$ gravimetric measurement, which will be further specified in the Federal Reference Method and equivalency program.

From a public health perspective, $PM_{2.5}$ captures all of the potential agents of concern in the fine fraction. For example, $PM_{2.5}$ captures most sulfates, acids, fine particle metals, organics, and ultrafine particles and accounts for most of surface area, and particle number. $PM_{2.5}$ has been used directly in many health studies as described in the CD and Chapter V. $PM_{2.5}$ has some potential for intrusion of particles generated by grinding or crushing (i.e., coarse mode particles) into the daily $PM_{2.5}$ measurement. 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 subsequent data reveal problems in this regard, this issue might be better addressed on a case-by-case basis in the monitoring and implementation programs. Furthermore, $PM_{2.5}$ measurement technologies are widely available and have been in use since the 1970s.

PM_1, on the other hand, has not been used in health studies primarily due to lack of available monitoring data. PM_1 could reduce possible intrusion of coarse mode particles in some situations, but it might not capture all of the fine mode in other situations. PM_1 sampling technologies have been developed; however, the PM_1 samplers have not been widely field-tested to date. Thus, the staff recommends the use of $PM_{2.5}$ as the fine particle indicator.

C. Level of the Standards

Selecting a suite of PM ambient air quality standards that provide an adequate margin of safety remains a difficult challenge for the decision maker despite recent new studies and analyses since the last review that provide significant relevant information and insights. Although we can now somewhat better characterize PM, it remains a pollutant class with chemical and physical characteristics that vary with geographic location, source mix, meteorology, and time. This aspect of PM inherently makes interpretation of epidemiological evidence difficult and diminishes the utility of controlled human and animal studies in making quantitative judgments about appropriate ranges of standard levels. In addressing this issue, the staff recognizes, as in past reviews, that although the scientific literature supports the notion that various mixes of particles pose risks to health, the basis for

any general PM standards is largely a public health policy judgment. The staff believes that by considering policy alternatives that focus control on those particles with the greatest potential for causing health effects of concern, primarily through $PM_{2.5}$ standards as recommended above, a more effective and efficient policy response would result than that associated with the current suite of standards.

1. General Considerations and Approach

In developing an approach to formulating recommendations on appropriate ranges of standard levels, the 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 can do no more than observe site-, time-, and monitor-specific associations between levels of a given pollutant and health responses. Further, such studies cannot be expected to 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 risks, and the associated uncertainties, along a continuum of exposures using the full range of available health and exposure data from the specific key and supporting studies.

3) Relative to other single pollutants for which NAAQS have been set, establishing appropriate ranges of levels for general PM 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 mixture of particles. However, even without any additional chemical-specific evidence, the staff believes that the large uncertainties inherent in setting general PM standards do not preclude our identifying appropriate ranges of policy alternatives from which specific PM 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 for the recommended indicators and averaging times is based on: 1) staff assessments of the quantitative concentration-response relationships suggested by specific epidemiological studies identified in the CD as appropriate for quantitative assessment purposes; 2) consideration of how these studies may be applied in developing ranges specifically for $PM_{2.5}$ and PM_{10} standards; and 3) qualitative consideration of the uncertainties and other key factors that affect the margins of safety associated with ranges of standard levels. This approach recognizes that final decisions about appropriate PM standard levels must draw not only on scientific information about health effects and risks, but also on policy judgments about when observed effects become adverse from a public health perspective and how to deal with the range of uncertainties that are inherent in the evidence and assessments.

These staff assessments and considerations are discussed below for both 24-hour and annual $PM_{2.5}$ standards, as well as for PM_{10} standards. The following discussions are based on information in the CD and in Chapters IV and V of this Staff Paper.

2. Specific Considerations and Conclusions for $PM_{2.5}$ Standards

a. 24-Hour $PM_{2.5}$ Standard

i. Levels of Interest from Short-term Epidemiological Studies

As discussed in section V.E, selected epidemiological studies provide quantitative concentration-response information useful in developing an appropriate range of standard levels for consideration in setting a new 24-hour $PM_{2.5}$ standard. In the last PM standards review, studies were not available that directly used the PM_{10} indicator that was established based on that review. In this review, by contrast, several studies are available that directly use the currently recommended $PM_{2.5}$ indicator (Schwartz et al., in press; Dockery et al., 1992; Schwartz et al., 1994). A larger number of studies using PM_{10}, as well as reanalyses of some datasets by independent reviewers, are also now available to provide concentration-response information (Pope et al., 1992; Schwartz, 1993a, 1994g; Schwartz et al., 1994e; Schwartz, 1994d; Schwartz and Morris, in press; Pope and Dockery, 1992; Samet et al., 1995). These key $PM_{2.5}$ and PM_{10} studies are supported by other studies that used sulfates and TSP as indicators of exposure (Burnette et al., 1994, 1995; Samet et al., 1995). These selected studies were the basis for staff's assessment to discern concentrations of the measured pollutants at which risk increases in a clear and consistent pattern in these studies, as presented in Table V-19.

To gain insight into the coherence of these short-term studies with regard to estimated 24-hour $PM_{2.5}$ concentrations at which clear and consistent increases in risk may occur, staff converted all such estimates in terms of the measured pollutants into estimates of $PM_{2.5}$, shown in Table VI-1. In so doing, staff is aware that this approach is premised on assuming that $PM_{2.5}$ concentration-response relationships for these studies would be similar to those observed for other measured particle indicators. Further, staff recognizes that translating exposures measured in terms of one particle indicator to $PM_{2.5}$ exposures incorporates additional uncertainties beyond those inherent in specifying exposures directly in terms of a measured particle indicator. To minimize this uncertainty to the extent possible, site-specific conversion factors were used wherever available. Taking into account these caveats, staff

TABLE VI-1. ESTIMATED LEVELS OF MINIMUM CLEAR INCREASED RISK IN TERMS OF MEASURED OR ESTIMATED $PM_{2.5}$ (24-HOUR AVERAGE)

Study/PM Indicator	Minimum Clear Increased Risk Level for $PM_{2.5}$ ($\mu g/m^3$ of measured or estimated $PM_{2.5}$)
MORTALITY -- 6 Cities - PM-2.5 Schwartz et al., in press	<30
MORTALITY -- St. Louis - PM-2.5/PM-10 Dockery et al., 1992 Samet et al., 1995	21[1] 28[1]
MORTALITY -- Utah Valley - PM-10 Pope et al., 1992 Samet et al., 1995	27[2] 29[2]
MORTALITY -- Birmingham - PM-10 Schwartz, 1993a, 1994g Samet et al., 1995	34[3] 30[3]
MORTALITY -- Philadelphia - TSP Samet et al., 1995	34[4]
HOSPITAL ADMISSIONS -- Birmingham - PM-10 Schwartz et al., 1994e	11 - 26[3]
HOSPITAL ADMISSIONS -- Detroit - PM-10 Schwartz, 1994d	33[5]
HOSPITAL ADMISSIONS (Cardiac) -- Detroit - PM-10 Schwartz and Morris, in press	23[5]
HOSPITAL ADMISSIONS -- Ontario - Sulfate Burnette et al., 1994, 1995	13 - 18[6]
RESPIRATORY SYMPTOMS -- 6 Cities - PM-2.5 Schwartz et al., 1994	≤18[7]
RESPIRATORY SYMPTOMS -- Utah Valley - PM-10 Pope and Dockery, 1992	32[2]

[1]Concentration to PM-2.5 from PM-10 quartile done by using a site-specific PM-2.5/PM-10 ratio for the period of study (0.64).

[2]Conversion to PM-2.5 used nationwide PM-2.5/PM-10 ratio for all seasons (0.58) from SAI, 1995, because urbanized Utah Valley judged not well represented by other Southwest sites.

[3]Minimum clear risks indicates lowest concentration on nonparametric smoothed curve where a clear and consistent increase in risk is evident. Conversion to PM-2.5 done using the PM-2.5/PM-10 ratio for Southeast region from SAI, 1995 (0.57).

[4]Conversion to PM-2.5 done by applying median PM-2.5/TSP ratio available for 1982 in Philadelphia (0.34), using data from the inhalable particle network.

[5]Conversion to PM-2.5 done using PM-2.5/PM-10 ratio (0.63) for central U.S. from SAI, 1995.

[6]Conversion to PM-2.5 done using site-specific regression equations to convert from sulfate to PM-10 for the three major cities in the study and converting the PM-10 values to PM-2.5 using a nationwide PM-2.5/PM-10 regression equation for Canada (CEPA/FPAC Working Group, 1995; Brook et al., in press). The results agree closely with those obtained using a sulfate/PM-2.5 ratio for Toronto from Thurston et al., 1994.

[7]Conversion to PM-2.5 from PM-10 done by using site-specific ratio for the period of study (0.6).

believes that this approach provides useful insight beyond that which can be obtained by only looking at each study independently.

As seen in Table VI-1, the selected short-term $PM_{2.5}$ and PM_{10} studies most useful for evaluating quantitative concentration-response relationships appear to show strong coherence in terms of measured and estimated $PM_{2.5}$ concentrations at which the risk of observed mortality and morbidity effects appears to clearly and consistently increase. From the mortality studies, risk appears to clearly and consistently increase at $PM_{2.5}$ concentrations somewhat below 30 $\mu g/m^3$. The morbidity studies show a wider range of estimates for concentrations of clear and consistent risk, ranging from somewhat below 20 $\mu g/m^3$ to somewhat above 30 $\mu g/m^3$. When the sulfate and TSP studies are considered as surrogates for $PM_{2.5}$, these studies yield estimates of $PM_{2.5}$ concentrations where risk increases in a clear and consistent pattern that are within the range of that seen in the $PM_{2.5}$ and PM_{10} studies. Thus, this staff assessment suggests that, when considered together, the short-term epidemiological studies are quantitatively coherent and support the judgment that evidence of increased mortality and morbidity risks become more apparent when daily $PM_{2.5}$ concentrations reach approximately 20 $\mu g/m^3$ to somewhat below 30 $\mu g/m^3$.

The above discussion of 24-hour $PM_{2.5}$ levels at which increased risks of various health effects are likely is based on interpreting the quantitative study results as being primarily attributable to changes in $PM_{2.5}$ levels. The staff believes such an interpretation is reasonable in light of the entire body of evidence and given efforts in the original studies and reanalyses to control for potential confounding and to characterize exposures in terms of $PM_{2.5}$ concentrations in selected studies where such information was available. However, the staff recognizes that various issues have been raised that call into question using these study results directly as a basis for establishing a range of levels for a 24-hour $PM_{2.5}$ standard. Alternative interpretations of the studies, for example, raise the possibility that the observed effects may in part be more appropriately attributed to specific components within the mixture of PM or to other pollutants in the mixture of ambient air in general, despite extensive efforts to control for such confounders. Further, limitations on matching exposures of the population subgroup experiencing effects to the ambient measurements of PM, and the

conversions of measured concentrations of other indicators into $PM_{2.5}$ concentrations that have been used in developing exposure-response relationships for $PM_{2.5}$ call into question the specific levels at which effects are likely to be experienced. Finally, the degree of prematurity of death observed in the mortality studies is an issue in regard to the extent to which such prematurity -- if it is, for example, on the order of a few days -- should be considered a matter of public health concern.

These issues are discussed further below. Taking into account all such questions, however, the staff considers it appropriate at a minimum to interpret the short-term quantitative studies as a body of evidence that, when taken together with information from controlled human and animal studies, supports the following conclusion: adverse public health effects, including premature mortality and increased morbidity, are likely to occur in various sensitive population subgroups at 24-hour $PM_{2.5}$ concentrations below those that occur when the current 24-hour PM_{10} NAAQS is attained. Thus, the staff concludes that a $PM_{2.5}$ concentration that is approximately equivalent to 150 $\mu g/m^3$ PM_{10} represents a clear level of concern where health effects can with confidence be attributed to PM exposures. This approximately equivalent $PM_{2.5}$ concentration varies from about 55 to about 95 $\mu g/m^3$ for western regions and eastern regions, respectively, across the U.S., with the national average conversion yielding a 24-hour $PM_{2.5}$ level of about 85 $\mu g/m^3$ as the approximate equivalent of the current 24-hour PM_{10} NAAQS. Setting a 24-hour $PM_{2.5}$ standard at this level would give maximum weight to the issues and uncertainties mentioned above by, in essence, completely discounting the quantitative study results, and no weight to the coherence of the entire body of evidence that has become available since the last review of the PM standards. Such a standard could not be construed as being at all precautionary in nature, nor to provide any identifiable margin of safety. Instead, the staff believes that the issues and uncertainties mentioned above should be considered in the context of deciding what standard within the range recommended below will, in the Administrator's judgment, provide an adequate margin of safety.

ii. Margin of Safety Considerations for a 24-Hour $PM_{2.5}$ Standard

As discussed above, there are a number of issues that the staff believes are relevant and appropriate to consider in deciding what standard will provide and adequate margin of safety.

An effect of "air pollution" on mortality is now broadly accepted by the scientific community (Moolgavkar, 1995, Samet et al., 1995), but differing views remain as to the degree to which the risk of premature mortality can be quantitatively associated with PM. Further, associations between air pollutants other than PM have been well-established for some of the morbidity effects linked to PM, resulting in uncertainties in the degree to which the risk of observed morbidity effects can be quantitatively associated with PM. As discussed in section V.D, when the health effects of PM are evaluated across the entire body of evidence, not just looking at each study independently, concerns are lessened by the absence of consistent and plausible confounding by other candidate pollutant(s). Further, investigations into plausible mechanisms, elucidating how exposure to ambient levels of PM may result in the observed effects have not yet established definite pathways from exposure to effects at ambient PM concentrations. Therefore, substantial uncertainty will remain concerning the agent(s) responsible for the observed associations.

A related uncertainty concerns the degree to which effects associated with PM can be attributed primarily to the fine particle fraction of PM. Although the existing health literature contains more studies using PM_{10} as a general indicator of particles than it does studies of $PM_{2.5}$, the staff concludes that the existing $PM_{2.5}$ studies strongly implicate fine particles, and as discussed in Chapter V, that much of the larger body of PM_{10} studies can be interpreted as linking effects to the fine particle component of PM_{10}. Further, there are differing views as to the degree to which specific chemical classes within $PM_{2.5}$ may be primarily associated with observed effects. The staff believes this issue concerning specific chemical classes should be considered in judging the desirability of a more general surrogate indicator (i.e., one that is likely to include the various physical properties and chemical constituents causally linked to effects) as a practical guide to the development of effective and efficient control strategies.

Another relevant factor concerns the uncertainties associated with using fixed-point outdoor monitoring networks, and area-wide conversions between a measured indicator and estimates for another indicator of PM exposure, to specify exposures in quantitative analyses of exposure-response relationships. Although recent studies have become more sophisticated in the application of central, fixed-site monitoring to community-wide measures of health effects, significant uncertainties necessarily remain. As discussed in Chapter IV, the importance of this uncertainty is lessened for fine particles, which have good serial correlation between central monitors and personal exposure (Janssen et al., 1995), are generally more uniformly distributed across a community than other PM indicators, and penetrate more efficiently indoors where much of individual exposures to PM occur.

Uncertainties in specifying exposures carry over into analyses of the exposure-response relationships directed toward gaining insight into patterns of increasing risk over relevant PM concentration ranges. The staff has attempted to minimize the impact of such uncertainties concerning concentration-response by evaluating available information from individual studies to attempt to discern where the clearest and most consistent patterns of increased risk are evident, and, further, to consider the coherence of these analyses in the context of the entire body of evidence.

Another factor to be addressed in margin of safety considerations includes the uncertainties and differing views with regard to the degree of life shortening that is reflected in the mortality events associated with short-term PM exposure. The extent of life shortening in these studies is difficult to specify, with some authors reporting that at least a portion of the mortality effects from short-term exposures may involve little displacement of mortality (Spix et al., 1993). Other findings from short-term exposure studies (Pope et al., 1992) and from long-term exposure studies (Dockery et al., 1993; Pope et al., 1995) suggest that substantial displacement of mortality may be occurring in some cases. The staff believes that assessing the public health significance of this effect ultimately involves a general policy judgment that should be made in the context of considering what standard will provide an adequate margin of safety.

b. Annual $PM_{2.5}$ Standard

i. Levels of Interest from Long-term Epidemiological Studies

Staff has used the same approach to identify concentration levels of interest for an annual $PM_{2.5}$ standard as described above for a 24-hour $PM_{2.5}$ standard. Although only four long-term studies were judged to provide quantitative concentration-response information useful in developing an appropriate range of consideration for an annual $PM_{2.5}$ standard, two of these studies directly examined the effects of $PM_{2.5}$ exposure on mortality (Dockery et al., 1995; Pope et al., 1993). These $PM_{2.5}$ mortality studies are supported by studies of morbidity effects (e.g., chronic bronchitis in children and adults) using PM_{15} and TSP (Dockery et al., 1989; Schwartz et al., 1993). These selected studies were the basis for staff's assessment to discern concentrations of the measured pollutants at which risk increases in a clear and consistent pattern in these studies, as presented in Table V-20.

To gain insight into the coherence of these long-term studies with regard to estimated annual $PM_{2.5}$ concentrations at which clear and consistent increases in risk may occur, staff converted all such estimates in terms of the measured pollutants into estimates of $PM_{2.5}$, shown in Table VI-2. In so doing, staff is aware that this approach is premised on assuming that $PM_{2.5}$ concentration-response patterns would be similar to those observed for other measured particle indicators. Further, staff recognizes that translating exposures measured in terms of one particle indicator to $PM_{2.5}$ exposures incorporates additional uncertainties beyond those inherent in specifying exposures directly in terms of a measured particle indicator. Taking into account these caveats, staff believes that this approach provides useful insight beyond that which can be obtained by only looking at each study independently.

The two strongest studies of the effects of long-term exposures to $PM_{2.5}$ are the Harvard Six City (Dockery et al., 1993) and ACS (Pope et al., 1995) mortality studies. As seen in Table VI-2, the Harvard Six City study suggests a range of clear and consistent risk of from 15 $\mu g/m^3$ to below 30 $\mu g/m^3$ $PM_{2.5}$. Available concentration-response information from the ACS study, which examined cities with a similar range of long-term particle concentrations to the Harvard Six City study, provides evidence of clear risk at

TABLE VI-2. ESTIMATED LEVELS OF MINIMUM CLEAR INCREASED RISK IN TERMS OF MEASURED OR ESTIMATED $PM_{2.5}$ (ANNUAL AVERAGE)

Study/PM Indicator	Minimum Clear Increased Risk Level for $PM_{2.5}$ ($\mu g/m^3$ of measured or estimated $PM_{2.5}$)
MORTALITY -- 6 Cities - PM-2.5 Dockery et al., 1993	15 - <30
MORTALITY -- ACS 50 Cities - PM-2.5 Pope et al., 1995	>15
BRONCHITIS IN CHILDREN -- 6 Cities - PM-2.5/PM-10 Dockery et al., 1989	22
CHRONIC BRONCHITIS -- 53 Cities - TSP Schwartz et al., 1993	23[1]

[1]Derived from applying conversion factors for PM-10/TSP (0.5) and PM-2.5/PM-10 (0.6) for late 1970's - early 1980's data and previous as given in the 1986 SPA (pgs. 8 & 11) (U.S. EPA, 1986b.) Ratios may have differed somewhat over the study period (1970-1974).

concentrations above 15 $\mu g/m^3$ $PM_{2.5}$. Thus, the ACS study provides support that effects may be occurring at the lower end of the Harvard Six City study range.

The findings of these mortality studies, which associate $PM_{2.5}$ with mortality of sufficient prematurity to give rise to differences in overall annualized mortality rates, lessens the likelihood that mortality events associated with $PM_{2.5}$ solely involve only very brief shortening of life. Further, the ability to look across regions of substantially different PM composition allows for more confident focus on $PM_{2.5}$ as being primarily responsible for the observed effects (Dockery et al., 1993). Staff notes that questions raised in the CD about the appropriate exposure period to use in examining long-term mortality effects add to the uncertainty in interpreting the magnitude of risk and the concentration at which risk increases in a clear and consistent pattern suggested by these studies. Staff believes that these important uncertainties dealing with mortality effects should be addressed as part of the margin of safety considerations.

Coherence between the mortality and morbidity studies is more difficult to judge because the morbidity effects can be less confidently ascribed primarily to $PM_{2.5}$ and require uncertain conversions from other PM indicators to arrive at estimates of $PM_{2.5}$. However, when expressed in terms of $PM_{2.5}$, the morbidity studies suggest estimates of minimum $PM_{2.5}$ concentrations of clear and consistent risk from 22 to 23 $\mu g/m^3$. These morbidity studies support the judgment that clear and consistent risk from long-term exposures to $PM_{2.5}$ is likely to occur well below 30 $\mu g/m^3$ annual average $PM_{2.5}$.

ii. Margin of Safety Considerations for an Annual $PM_{2.5}$ Standard

Staff has identified several factors that are relevant and appropriate to consider in assessing margin of safety considerations, as discussed below.

Similar to the short-term studies, uncertainties in attributing observed effects specifically to PM, rather than to other co-pollutants, and more specifically to $PM_{2.5}$, are an important factor in margin of safety considerations. Several long-term studies have found PM to be a stronger indicator for many health effects from long-term exposures than other potentially confounding pollutants (Abbey et al., 1995c; Dockery et al., 1993; Dockery et al., 1989), as well as $PM_{2.5}$ being an equal or better indicator than other measures of PM

(Dockery et al., 1993; Abbey et al., 1995c). However, as long as mechanistic investigations have not established definite pathways from exposure to effects, uncertainty will remain concerning the agent(s) responsible for the observed effects. Further, uncertainties about specific causal agents should be addressed in considering the desirability of a more general surrogate indicator; i.e., one that is likely to include the various physical properties and chemical constituents causally linked to effects, as a practical guide to the development of effective and efficient control strategies.

With regard to specifying exposures, long-term studies have the same uncertainties as do short-term studies concerning the use of centrally-located, fixed-site monitors, generally compounded by the comparison across different urban areas. The current long-term mortality and morbidity cohort studies address this problem by controlling other potential confounding risk factors. Some also site monitors with a goal of characterizing the study population exposure (Dockery et al., 1989; 1993) or use fixed monitoring information broken down on a subject-by-subject basis (Abbey et al., 1991; 1993). However, uncertainties with regard to specification of exposures necessarily remain for long-term studies.

An important uncertainty involves whether cumulative effects from a long period of exposure to PM pollution, not fully captured in the monitoring done for a study, might be most relevant for some of the observed health effects. If a large proportion of subjects were responding to cumulative exposures over a period of years, and the air quality concentrations prior to the studies were substantially higher than during the studies, this circumstance could lead to an overestimate of the effects of PM on health. Examination of historical visibility records suggests that historical levels of fine particles do not appear to be substantially higher than that used in a key mortality study (Dockery et al., 1993). Further, some morbidity studies addressed this question by either testing the effects of particle concentrations several years previous to the effect (Abbey et al., 1995b) or by restricting their study population to young children (Dockery et al., 1989). In general, however, the current lack of knowledge concerning the most relevant exposure periods for the effects associated with long-term exposure to PM suggests that exposure estimates for long-term studies incorporate greater uncertainty than those from short-term studies.

Another factor to be considered is the uncertainty relating to the degree to which the long-term studies are detecting additional increased risk of chronic mortality or other endpoints, or are just measuring the cumulative impact of associations with short-term exposures. Given uncertainties concerning the measure of exposures, both current and historical, for the long-term studies, as well as corresponding uncertainties in the short-term exposure studies, a comparative assessment of the magnitude of risk observed by both study approaches is difficult. However, any mortality resulting from short-term exposures that is substantially premature should be reflected in the mortality events measured in long-term studies. Thus, the long-term studies can be interpreted as indicating either that a significant portion of the mortality associated with short-term exposures involves significant prematurity, or, more likely, that the mortality associated with long-term exposures includes both chronic and acute mortality effects.

3. Staff Recommendations for $PM_{2.5}$ Standards

The following staff recommendations are based most directly on the above discussion, but take into account the entire body of evidence presented in the CD and in Chapter V. These recommendations suggest ranges of levels for 24-hour and annual $PM_{2.5}$ standards that the staff believes would protect against the various adverse health effects associated with exposures to PM that have been reported in a large number of epidemiological studies and supported by evidence from controlled human and animal studies.

a. <u>24-Hour $PM_{2.5}$ Standard</u>

With regard to a 24-hour $PM_{2.5}$ standard, the staff offers the following recommendations:

1) The upper end of the range of consideration for a new 24-hour $PM_{2.5}$ standard should be below the $PM_{2.5}$ concentration that is approximately equivalent to the level of protection provided by the current PM_{10} standard. An approximately equivalent 24-hour $PM_{2.5}$ concentration, about 85 $\mu g/m^3$ on average in the U.S., contains no identifiable margin of safety and should not be considered as an appropriate standard alternative. While the uncertainties inherent in the most recent epidemiological studies are important margin of safety

considerations, the staff believes there is no basis for discounting the quantitative results from these studies completely in selecting an upper end of range of consideration. Thus, staff recommends that weight be given to the coherence of the entire body of epidemiological evidence, with support from controlled human and animal studies, in limiting consideration to a level below 85 $\mu g/m^3$ for a new 24-hour $PM_{2.5}$ standard.

2) The lower end of the range of consideration for a new 24-hour $PM_{2.5}$ standard be 25 $\mu g/m^3$. The key and supporting epidemiological studies relied upon to assess the point at which increased risks of adverse health effects can be clearly seen, taken together, suggest a range of levels from somewhat below 20 $\mu g/m^3$ to somewhat over 30 $\mu g/m^3$. In considering a 24-hour $PM_{2.5}$ level of 25 $\mu g/m^3$ as the lower end of the range, the staff places significant weight on the coherence of the study results, even in light of inherent uncertainties and alternative interpretations possible for each study considered independently. The staff believes that a 24-hour $PM_{2.5}$ standard set at this level would be precautionary in nature in protecting against a full range of short-term effects associated with the identified sensitive subgroups of the population, giving 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.

3) In selecting a level for a 24-hour $PM_{2.5}$ standard within this range, the Administrator should consider the degree and nature of protection that will 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 should be considered in selecting the levels for each standard. One possible policy approach would be to view an annual $PM_{2.5}$ standard as serving as the primary driving force for control programs that would effectively lower the entire distribution of $PM_{2.5}$ concentrations, thus serving to protect not only against long-term effects but also short-term effects as well. With this approach, the

24-hour $PM_{2.5}$ standard would 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. 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. The comparative air quality data presented in Chapter IV (Table IV-8) shows approximately 90% of the monitored $PM_{2.5}$ annual-peak-to-mean ratios below 3.34 on average nationwide (with a range from 3.1 in the southeast to 3.8 in the northwest). Thus, considering the joint protection afforded by both 24-hour and annual standards suggests, for example, that a 24-hour $PM_{2.5}$ level of about 65 $\mu g/m^3$ might be an appropriate complement to a 20 $\mu g/m^3$ annual $PM_{2.5}$ standard.

b. Annual $PM_{2.5}$ Standard

With regard to an annual $PM_{2.5}$ standard, staff offers the following recommendations:

1) Staff recommends that the upper end of the range of consideration for a new annual $PM_{2.5}$ standard should be below 30 $\mu g/m^3$. This level, which is approximately equivalent on average across the U.S. to the level of the current annual PM_{10}, contains no identifiable margin of safety and should not be considered as an appropriate standard alternative. While the uncertainties inherent in the most recent epidemiological studies are important margin of safety considerations, staff believes there is no basis for discounting the quantitative results from these studies completely in selecting an upper end of range of consideration. Thus, the staff recommends that weight be given to the coherence of the entire body of long-term epidemiological evidence, with mechanistic support from controlled human and animal studies, in limiting consideration to a level below 30 $\mu g/m^3$ for a new annual $PM_{2.5}$ standard.

2) Staff recommends that the lower end of the range of consideration for a new annual $PM_{2.5}$ standard be 15 $\mu g/m^3$. A standard set at this level would place greater weight on the full range of exposure-response relationships from the

key mortality studies and on the staff's assessment of the levels at which mortality risk clearly increases. In recommending this lower level of consideration, staff recognizes that this level does not represent a threshold below which there is no risk of premature mortality. Rather, the staff judges that below this level the uncertainties in historical air quality and relevant periods of exposure, as well as potential confounding by other environmental and/or personal lifestyle factors, become sufficiently great as to serve as an appropriate bound for a policy choice intended to avoid unacceptable risk.

3) The staff recommends that in selecting a level for an annual $PM_{2.5}$ standard within this range, the Administrator consider the degree and nature of protection that will be afforded by a new 24-hour $PM_{2.5}$ standard. The joint protection provided by a suite of standards that includes both 24-hour and annual $PM_{2.5}$ standards should be considered in selecting the levels for each standard.

4. Staff Conclusions and Recommendations for PM_{10} Standards

The following staff conclusions and recommendations are based on a reexamination of the previous conclusions regarding PM_{10} standards in light of the above recommendations regarding fine particle standards and the body of evidence presented in the CD and Chapters IV and V of this paper. The staff makes the following conclusions and recommendations regarding PM_{10} standards:

1) Assuming a fine particle standard is established, the major function of the PM_{10} standard is to protect against the known and anticipated effects associated with coarse fraction particles in the size range of 2.5 to 10 μm. It is difficult to discern the incremental effects of coarse fraction particles from fine particles in available community epidemiology studies.

2) Based on the assessment of the available studies, the staff finds that the best evidence for drawing quantitative conclusions for PM_{10} comes from a long-term cohort study of acute bronchitis in children in the Harvard Six City study (Dockery et al., 1989). This study found somewhat better associations with

PM_{15} than with $PM_{2.5}$ over the entire cohort. This suggests that coarse fraction particles, in combination with fine particles, may have influenced the observed effects. From a mechanistic perspective, it is possible that prolonged deposition of coarse fraction particles could be elevated in children, who are more prone to be active outdoors than adults. Moreover, the major site of thoracic deposition, the tracheobronchial region, would be expected to be the region involved in the effects. It is of note that Dockery et al. (1989) is an update of the study by Ware et al., (1986), which formed the main basis for the level of the current annual PM_{10} standard. The staff concludes there is no basis in the more recent study to alter the level and form of current annual PM_{10} standard, which is 50 $\mu g/m^3$, annual arithmetic mean.

3) The staff also considered other potential effects of annual coarse particle exposures that were not evaluated in community epidemiology studies. These include altered clearance, industrial bronchitis, and alveolar tissue damage; such effects have been reported at levels much above those that would be permitted under the standard. At levels nearer, but likely above the level of the standard, autopsy studies found silicate pneumoconiosis associated with particles that by analyses were consistent with exposure to crustal materials. While these findings support retention of the current PM_{10} standard, they provide no basis for changing the level.

4) 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. As noted above, the present assessment of the more numerous recent short-term studies has led to the staff conclusion that such effects are far more likely to be associated with fine particles. Accordingly, the staff recommends that these effects should be addressed with a fine particle standard.

5) Available community studies conducted in areas with elevated coarse fraction concentrations suggest that short-term effects occur at concentrations well above the current standard. Johnson et al., (1992) found that an episode of

very high concentrations of volcanic ash had little effect on pulmonary function in a panel of school children. Hefflin et al., (1994) studied hospital admissions in an agricultural community in eastern Washington. Short-term exposures to PM_{10} were associated with increased respiratory effects across the year, which included both fine and coarse exposures. During a two week period in which two windblown dust episodes occurred with particle levels exceeding 1,000 $\mu g/m^3$, only hospital admissions for upper respiratory infections showed an increase of even marginal significance. The authors conclude that dust in the community has only a small effect on general respiratory health.

6) Based on the lack of clear evidence for short-term effects of coarse fraction particles at or near the level of the current PM_{10} standard, the staff examined the relative protection afforded by the annual standard. Based on the typical 24 hour to annual ratios in Chapter IV, the annual standard would be expected to provide substantial protection against the occurrence of high coarse fraction concentrations. Using the average ratio of 3 (Table IV-6), an annual average of 50 $\mu g/m^3$ would have a daily maximum of 150 $\mu g/m^3$ or less. In areas with more variability, the ratios can be larger, but are still likely to limit the peak concentrations of coarse fraction particles from human derived activities to levels below those expected to be associated with adverse health effects. Using the data from Table IV-7, 95% of areas with an annual mean of 50 $\mu g/m^3$ would have 24-hour maxima below 250 $\mu g/m^3$.

7) In summary, the staff recommends retention of the current annual PM_{10} standard of 50 $\mu g/m^3$, annual arithmetic mean. This standard is judged to provide adequate protection against the long-term and short-term effects associated with the presence of coarse fraction particles.

D. Summary of Staff Recommendations

The major staff conclusions and recommendations made in sections VI.A-C above are briefly summarized below:

1) General PM standards remain a reasonable public health policy choice.

2) While the current PM_{10} standards provide protection from both fine and coarse mode particles that are capable of penetrating to the most sensitive regions of the human respiratory tract, the tracheobronchial and alveolar regions, the staff concludes that the fine fraction of PM_{10} is more likely to contain those physical and chemical properties and components associated most strongly with a broad array of adverse public health effects, including premature mortality as well as various measures of increased morbidity in children and other sensitive populations. Further, retaining PM_{10} as the sole indicator of PM would continue to direct control efforts towards coarse fraction particles, which available evidence suggests are of lower risk to health than the fine fraction. Thus, a new fine particle indicator is recommended, to include those particles with an aerodynamic diameter less than a nominal 2.5 μm, in addition to retention of a PM_{10} indicator to continue to limit coarse mode particle pollution.

3) Staff recommends replacing the current 24-hour PM_{10} standard with a new 24-hour $PM_{2.5}$ standard, with a statistical expected exceedance form, and supplementing the current annual PM_{10} standard with a new annual $PM_{2.5}$ standard, with an arithmetic average form. This suite of indicators and averaging times is judged to be sufficient to address all the components of PM that have been associated with adverse health effects.

4) Based on the staff's assessment of the short-term epidemiological data, the range of 24-hour $PM_{2.5}$ levels of interest is 25 to less than 85 $\mu g/m^3$. Given the coherence of the body of short-term epidemiological evidence for increased risk in areas where the current standard is attained and the seriousness of the potential health effects, the upper end of the above range is judged to represent a clear level of concern with no identifiable margin of safety and should not be considered as an appropriate standard alternative. Although some degree of increased risk to public health below 25 $\mu g/m^3$ cannot be excluded,

consideration of the entire body of epidemiological and controlled human and animal studies do not suggest increased health risks of consequence below this level. The uncertainties inherent in the epidemiological evidence, in particular with regard to potential confounding by other environmental factors, limitations on quantifying exposures, and differing judgments as to the public health significance of the degree of prematurity of death observed in the mortality studies, should be considered in evaluating margins of safety associated with alternative 24-hour $PM_{2.5}$ standards in the range of 25 $\mu g/m^3$ to somewhat below 85 $\mu g/m^3$.

5) Based on the staff's assessment of the long-term epidemiological data, the range of annual average $PM_{2.5}$ levels of interest is 15 to less than 30 $\mu g/m^3$. Given the recent large-scale studies directly associating long-term $PM_{2.5}$ exposure with premature mortality, the supporting evidence from other chronic morbidity studies, and the seriousness of the potential health effects, the upper end of the above range is judged to represent a clear level of concern with no identifiable margin of safety and should not be considered as an appropriate standard alternative. Although some degree of increased risk to public health below 15 $\mu g/m^3$ cannot be excluded, when uncertainties in historical exposures and relevant exposure periods are considered, the epidemiological evidence does not suggest increased risk of consequence below this level. The uncertainties inherent in the epidemiological evidence, in particular with regard to potential confounding by other environmental and personal/lifestyle factors and limitations on quantifying exposures, should be considered in evaluating margins of safety associated with alternative annual $PM_{2.5}$ standards in the range of 15 $\mu g/m^3$ to somewhat below 30 $\mu g/m^3$.

6) When selecting final $PM_{2.5}$ standard levels, consideration should be given to the joint protection afforded by the 24-hour and annual standards taken together. For example, an annual $PM_{2.5}$ standard at 20 $\mu g/m^3$ would be expected to result in substantially reduced 24-hour levels, potentially limiting

maximum 24-hour levels to less than about 65 $\mu g/m^3$ in approximately 90% of the areas, thus adding to the protection against short-term effects afforded by a 24-hour standard.

7) Based on staff assessments of air quality, toxicological, and epidemiological evidence available since the last review, retention of the current annual PM_{10} standard is recommended to provide ongoing control of coarse mode particles for protection against known and anticipated adverse health effects associated with both 24-hour and annual exposures.

VII. 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. 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.

The chapter does not address in detail 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. Types of Visibility Impairment

Visibility effects are manifested in two principal ways: (1) as local impairment (e.g., plumes and localized hazes) and (2) as regional haze. Local-scale impairment is defined as impairment that is "reasonably attributable" to a single source or group of sources. This type of impairment is considered to be an effect primarily due to nearby sources, although it can be part of a larger, regional impairment. Visibility impairment in some urban areas can

be dominated by local rather than regional sources, depending on site-specific geographical and meteorological factors.

The second category of impairment, regional haze, is produced from a multitude of sources and impairs visibility in every direction over a large area, such as an urban area, or possibly over several states. Objects on the horizon are masked and the contrast of nearby objects is reduced. In some cases, the haze may be elevated and appear as layers of discoloration. Multiple sources may combine over many days to produce haze, which is often regional in scale. The fate of regional haze is a function of meteorological and chemical processes, sometimes causing fine particle loadings to remain suspended in the atmosphere for long periods of time and to be transported long distances from their sources.

2. Social Valuation of Visibility

Visibility is an air quality-related value essential 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. Thousands of Americans appreciate the scenic vistas in national parks and wilderness areas annually. Visibility is also highly valued, however, because of the importance people place on protecting nationally significant natural areas, both now and in the future. Many individuals want to protect such areas for the benefit of future generations, even if they personally do not visit these areas very frequently (Chestnut et al., 1994). 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 table VII-1. As an example of the potential value attributable to improvements in visibility, a recent study estimates visibility

Table VII-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.[5]	Atlanta and Chicago	Unadj. $39 Partial $25 Full $18	17.6	20	305 196 140	$56 $36 $26
Tolley et al.[6]	Chicago	-$318 $305 $379	9 9 9	4 18 30	367	$67
Tolley et al[6]	Atlanta	-$265 $255 $381	12 12 12	7 22 32	414	$75
Tolley et al[6]	Boston	-$196 $187 $231	18 18 18	13 28 38	372	$68
Tolley et al[6]	Mobile	-$212 $227 $266	10 10 10	5 20 30	275	$68
Tolley et al[6]	Washington, DC	-$314 $323 $410	15 15 15	10 25 35	560	$102
Tolley et al[6]	Cincinnati	-$78 $77 $86	9 9 9	4 19 29	106	$17
Tolley et at[6]	Miami	-$134 $120 $141	13 13 13	8 19 29	226	$41
Rae[7]	Cincinnati	$175	11.4	16.4	531	$97
California CVM Studies						
Brooksire et al.[8]	Los Angeles	$115 $294 $161	2 2 12	12 28 28	105	$19
Loehman et al[9]	San Francisco	-$186 $109	18.6 16.3	16.3 18.6	1172	$214
California Property Value Sutdy						$216-$579
Trijonis et al[10]	Los Angeles					$437-$487
Trijonis et al[10]	San Francisco					

Note: VR - Visual Range

benefits due to reduced sulfur dioxide emissions under the acid rain program to be quite significant, in the range of $1.7 - 2.5 billion annually by the year 2010 (Chestnut et al., 1994).

3. Visibility-Impairing Particles

Visibility impairment has been considered the "best understood and most easily measured effect of air pollution" (Council on Environmental Quality, 1978). It is perhaps the most noticeable effect of fine particles (i.e., those with an aerodynamic diameter less than 2.5 μm) present in the atmosphere. Fine particles are effective in impairing visibility because the mean diameter of fine particles is usually less than or only slightly greater than the wavelength range of visible light, and light scattering is at a maximum when the wavelength is comparable to the particle diameter. Fine particles in the size range from 0.1 to 1.0 μm in diameter are more effective per unit mass concentration at impairing visibility than either larger or smaller particles (NAPAP, 1991).

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. Particles affect color of distant objects depending upon particle size, composition, scattering angle between observer and illumination, and optical characteristics of the background target.

Fine particles, which can be emitted directly to the atmosphere through primary emissions or formed secondarily from gaseous precursors, impair visibility by scattering or absorbing light. Different types of particles have varying efficiencies in causing visibility impairment.

The fine particles (and associated particulate water) principally responsible for visibility impairment are sulfates, nitrates, organic matter, elemental carbon (soot), and soil dust. Coarse particles (i.e., those in the 2.5 to 10 μm size range) also impair visibility, although less efficiently than fine particles. Sulfates and nitrates readily absorb water from the atmosphere and grow in size in a nonlinear fashion as relative humidity levels increase. Soluble organics are considered to be less hygroscopic than sulfates and nitrates (Sisler, 1993). All of these particles scatter light. Light absorption is caused mainly by elemental carbon.

Sulfates, nitrates, and some organic particles begin as gaseous emissions and undergo chemical transformation in the atmosphere. These 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. Metrics for Expressing Visibility Impairment and Light Extinction.

Daytime visibility is determined the competition between transmitted radiance and path radiance. Transmitted radiance comes from the object being viewed and contains all of the information about that object available to the observer. Path radiance is scattered by the atmosphere into the line of sight, and contains no information about the object being viewed. When path radiance dominates transmitted radiance, as in a dense fog, visibility is poor. Path radiance is used in models that calculate visibility as a function of particle concentration, composition, and size.

Light extinction is an optical property of each point in the atmosphere and is closely linked to air quality. In itself, light extinction is not the best indicator of visibility for a specific scene. However, light extinction is a useful measure of haze because it represents the effect that gases and aerosols have on changes in path radiance. Therefore, light extinction is a useful indicator of visibility for regulatory purposes.

The light 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. This coefficient is typically expressed in terms of inverse kilometers or megameters (km^{-1} or Mm^{-1}). Direct relationships exist between the concentrations of particle types and their contributions to the extinction coefficient. By apportioning the extinction coefficient to different particle types, one can estimate changes in visibility due to changes in constituent concentrations (Pitchford and Malm, 1994).

The extinction coefficient is inversely related to visual range. Conversion from light extinction coefficient to visual range can be done with the following equation: Standard visual range (in kilometers) = 3.912 / light extinction coefficient (in M^{-1})(NAPAP, 1991). Visual range can be defined as the maximum distance (i.e., miles or kilometers) at which one

can identify an object against a uniform background. 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).

Another important visibility metric is the deciview, which measures perceived haziness. 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 may be useful for defining perceptible changes in future regulatory programs. Figure VII-1 illustrates the relationships between these three visibility metrics.

5. Overview of Current Visibility Conditions

Visibility conditions vary regionally, as a function of background levels of fine particles, average relative humidity levels, and generally higher anthropogenic particle loadings in the East as opposed to the West. An overview of current visibility conditions, developed from monitored aerosol concentrations and expressed in terms of the light extinction coefficient, is provided in figure VII-2 (Sisler et al., 1993). Median standard visual range in the rural mountain and desert areas of the Southwest average 130-190 km, whereas median visual range in the rural areas south of the Great Lakes and east of the Mississippi River is between 20 and 35 km.

Most of this six-fold difference between East and West is due to greater sulfate concentrations in the East and the effect of higher humidity levels 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. Figure VII-3 illustrates this correlation for the southeastern U.S.

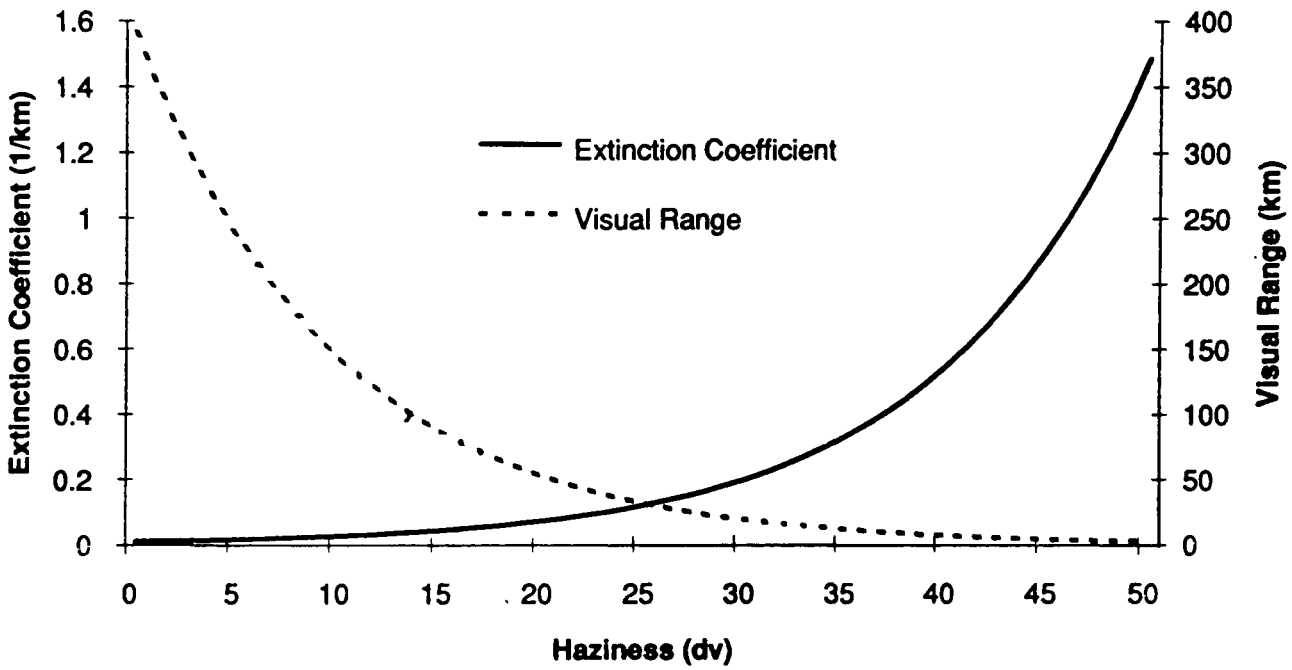
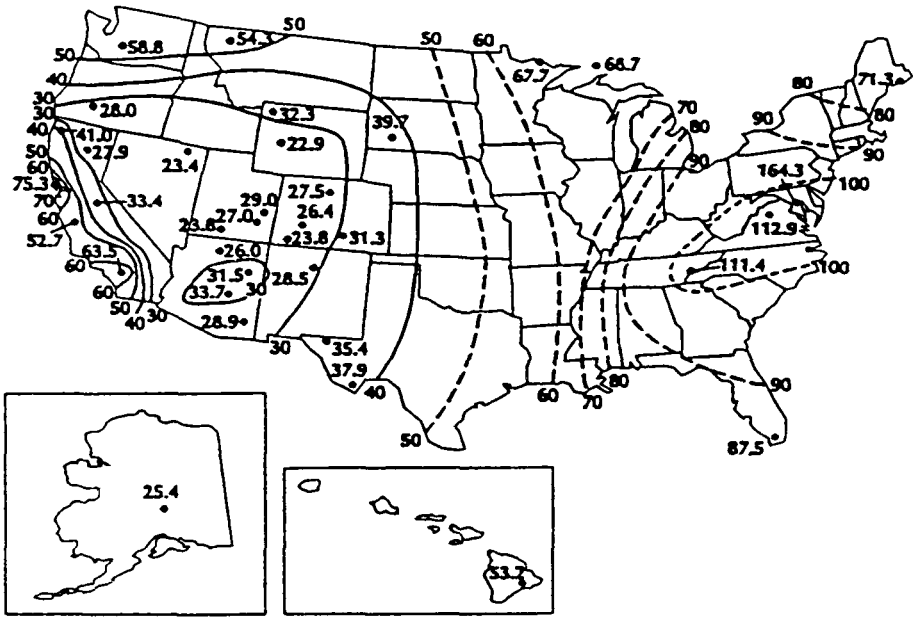
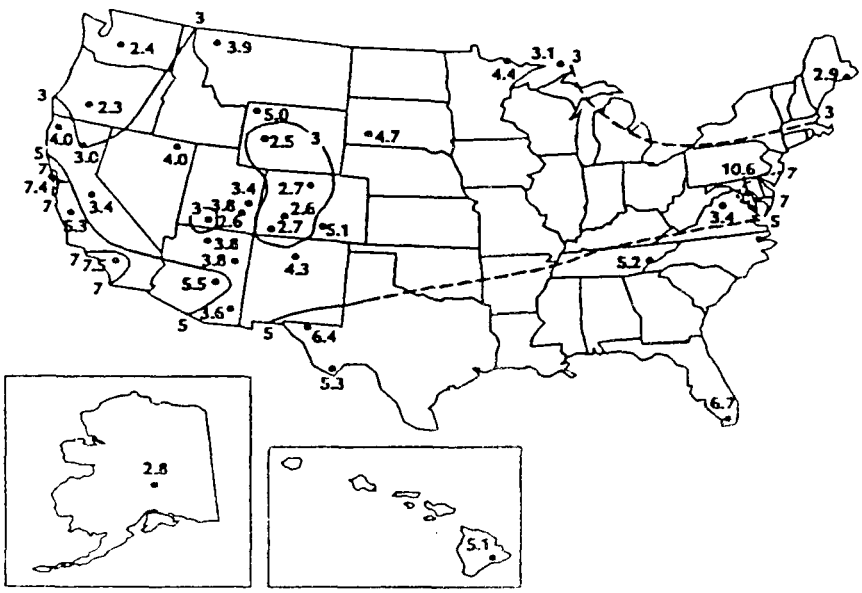


Figure VII-1. Visual range and extinction coefficient as a function of haziness expressed in deciview.



(a). Total light extinction b_{ext} (Mm^{-1})



(b). Extinction due to coarse particles and fine soil (Mm^{-1})

Figure VII-2. Average reconstructed light extinction coefficient (Mm^{-1}) calculated from the aerosol concentrations measured during the first three years of IMPROVE, March 1988 through February 1991. The various panels of this figure show total extinction (including Rayleigh scattering due to air) and the contributions due to the various aerosol components: coarse particles and fine soil, sulfate, organic carbon, nitrate, and light-absorbing carbon.

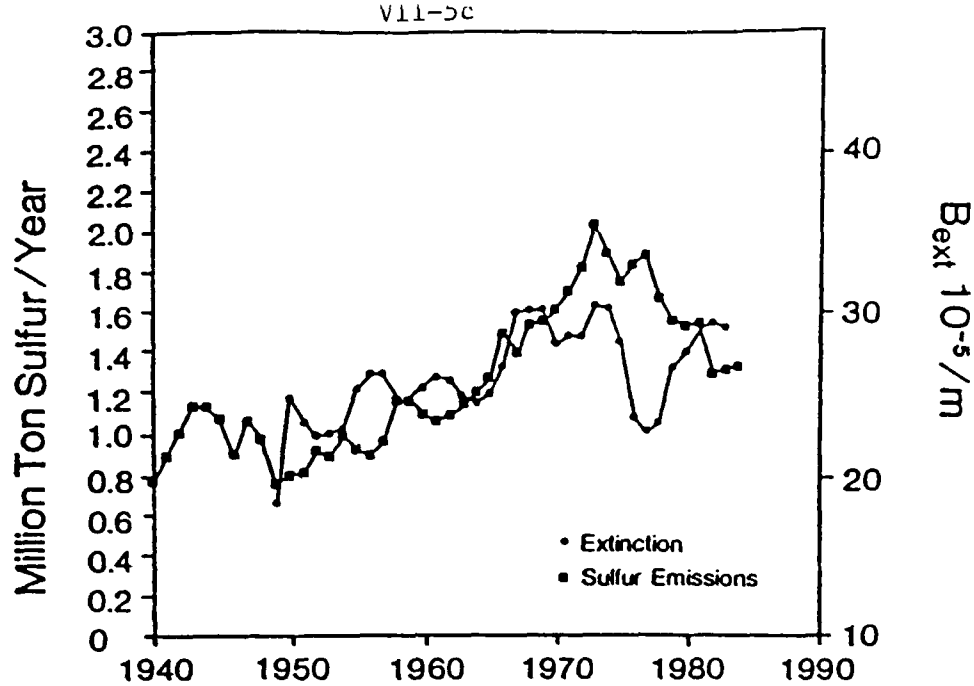


Figure 7a. Comparison of sulfur emission trends (▪) and extinction coefficient (•) for the sougheastern U.S. region during winter months.

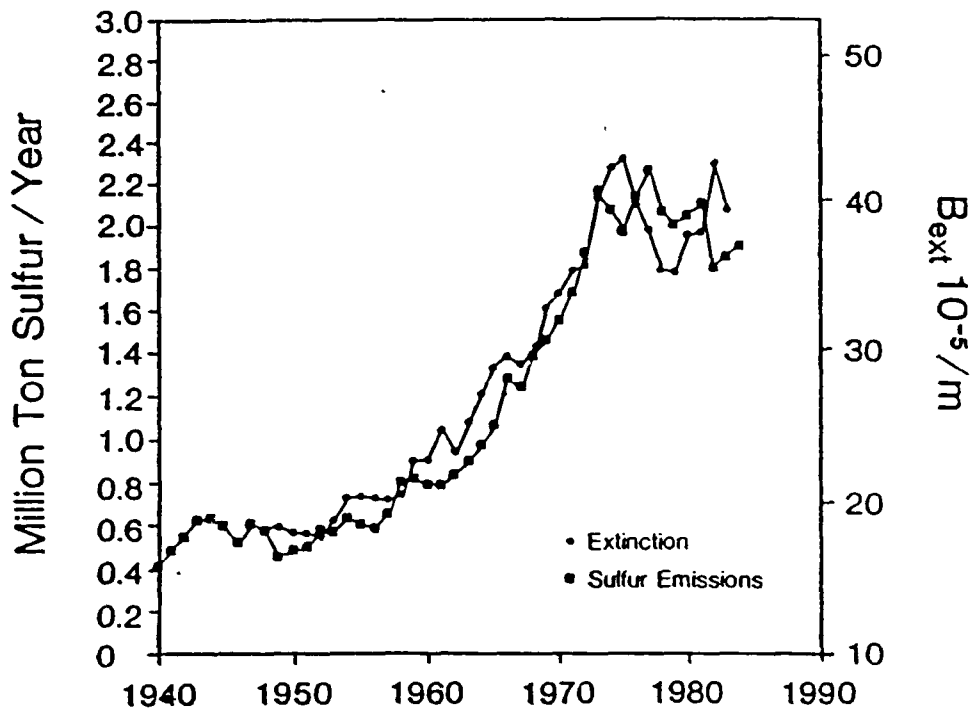


Figure VII-3. Comparison of sulfur emission trends (▪) and extinction coefficient (•) for the southeastern U.S. region during the summer months.

The following sections further explain the reasons for regional variability in visibility impairment.

6. Estimated Background Levels of Fine Particles and Associated Light Extinction

Total light extinction is determined by the combined effects of fine particles from background (i.e., natural) sources, Rayleigh scattering, and fine particles from anthropogenic sources. The 1990 National Acid Precipitation Assessment Program report estimated annual average background levels of fine particles by particle type, and related contributions to light extinction, expressed in inverse megameters (Mm^{-1}) (NAPAP, 1991). Table VII-2, shows that background fine particle concentrations are estimated at 3.32 $\mu g/m^3$ in the East and 1.47 $\mu g/m^3$ in the West.

The contribution of each particle type to total light extinction is derived by multiplying the estimated average concentration by the extinction efficiency for that particle type. Extinction efficiencies used in table VII-2 are for "dry" particles only. Associated water is expressed as a separate particle type.

Other studies have used slightly different extinction efficiencies for dry particles than those found in table VII-2. For example, Sisler et al. (1993) used the dry extinction efficiencies in table VII-3 in an analysis of monitoring data collected from the Interagency Monitoring of Protected Visual Environments (IMPROVE) visibility monitoring network, a cooperative program in place since 1986 involving several Federal agencies and State representatives.

7. 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). Two ways that the effect of humidity on light extinction can be represented are by including water as a separate particle type (as is done in table VII-2), or by multiplying particle-specific extinction efficiencies by correction factors representing 1) the hygroscopic nature of the particle, and 2) the average annual humidity for the relevant location (Sisler et al., 1993).

Table VII-2

Average Natural Background Levels of Aerosols and Light Extinction

	Average Concentration		Error Factor	Extinction Efficiencies[a]	Extinction Contributions	
	East $\mu g/m^3$	West $\mu g/m^3$		m^2/g	East Mm^{-1}	West Mm^{-1}
FINE PARTICLES (≤2.5 μm)						
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	$1^1/_2$-2	1.25	0.6	0.6
Water	1.0	0.25	2	5	5.0	1.2
COARSE PARTICLES (2.5-10 μm)						
	3.0	3.0	$1^1/_2$-2	0.6	1.8	1.8
			RAYLEIGH SCATTER		12	11
				TOTAL	26 ± 7	17 ± 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 the total scattering by all ambient coarse particles (≤ 2.5 μm) divided by the coarse particle mass between 2.5 and 10 μm.

Particle Type	Extinction Efficiency (in m^2/g)
sulfates	3.0
organics	3.0
elemental carbon	10.0
nitrates	3.0
soil dust	1.0
coarse particles	0.6

Source: Sisler et al. 1993

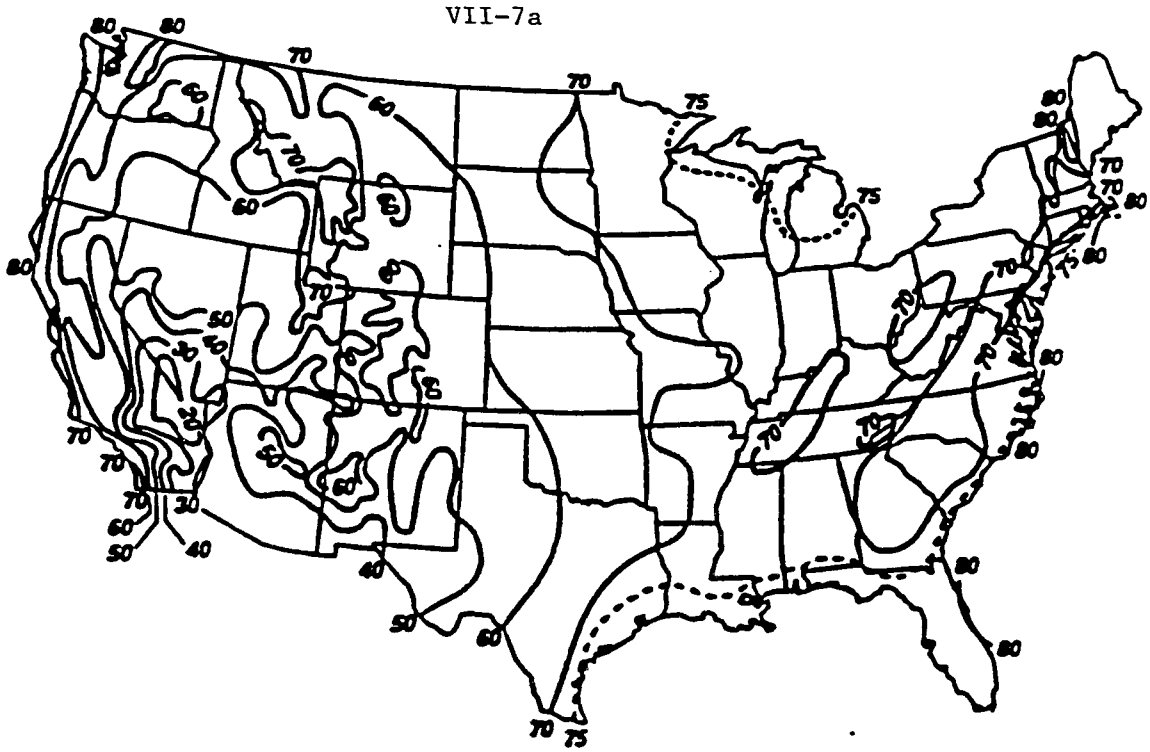
Table VII-3. Dry particle light extinction efficiency values used in 1993 analysis of IMPROVE data.

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 VII-4 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 the IMPROVE analysis (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.

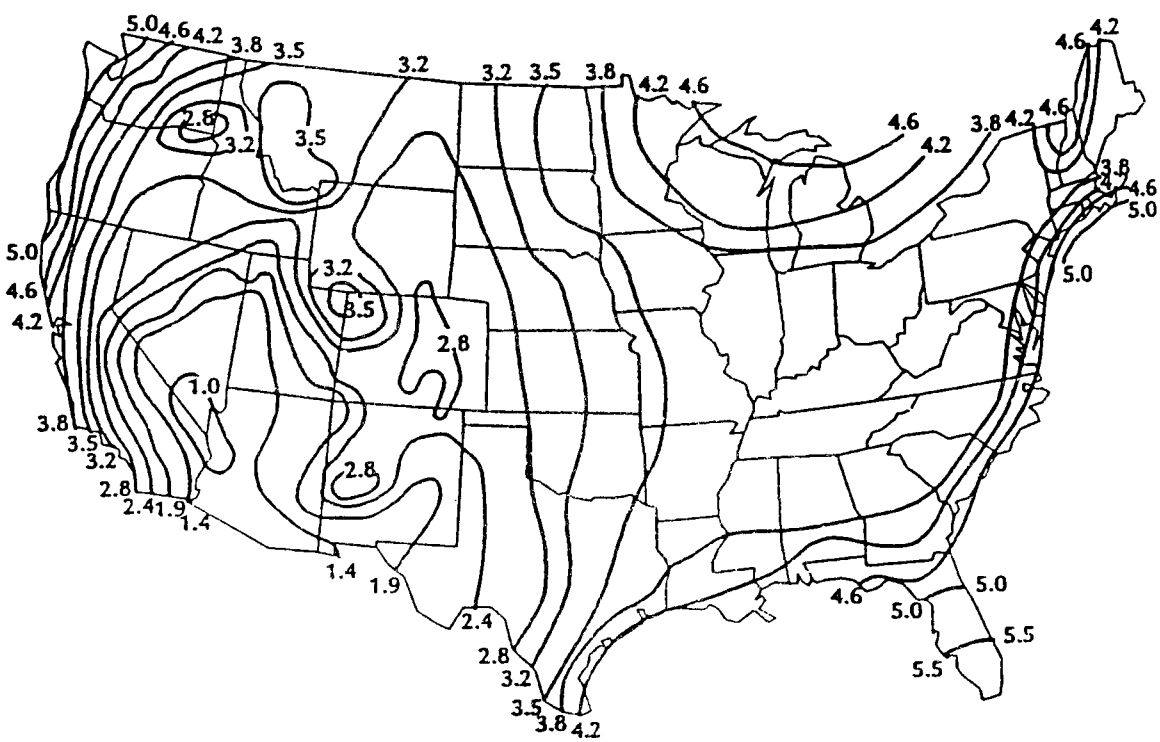
8. Rayleigh Scattering

Table VII-2 shows the contribution to total light extinction from "Rayleigh scatter." Rayleigh scattering represents the degree of light extinction found in a particle-free atmosphere, caused by the gas molecules that make up "blue sky" (e.g., N_2, 0_2, CO_2)(U.S. EPA, 1979). Rayleigh scattering thus can be used to establish a maximum horizontal visual range in the earth's atmosphere, absent visibility-impairing particles. At sea level, this maximum visual range is approximately 326 kilometers (equivalent to light extinction of 12 Mm^{-1}). While Rayleigh scattering can be shown to establish this maximum visual range, it should not be considered to contribute to visibility impairment. Rather, only fine and coarse particles from natural and anthropogenic sources should be considered responsible for impairing visibility (from the maximum visual range "baseline" established by Rayleigh scattering).

Estimated extinction contributions from Rayleigh scattering <u>and</u> background levels of fine and coarse particles, in the absence of anthropogenic emissions of visibility-impairing particles, are estimated to result in a visual range in the East of 150+/- 45 kilometers and 230 +/- 40 km in the West. NAPAP (1991) estimated that the major contributors to nonanthropogenic light extinction levels in the East are Rayleigh scattering (46%), organics (22%), water (19%), and suspended dust, including coarse particles (9%). The major contributors in the West are Rayleigh scattering (64%), suspended dust (14%), organics (11%), and water (7%). Thus, higher levels of background fine particles in the East result in a fairly significant difference between maximum visual range estimates in the East and West.



(a) Annual mean relative humidity.



(b) F_T.

Figure VII-4. Spatial variation in average relative humidity (NOAA, 1978) and the sulfate *RH* correction factor F_T.

9. Significance of Anthropogenic Sources of Fine Particles

The concentrations of background fine particles are generally small when compared with concentrations of fine particles from anthropogenic sources. The same relationship holds true when one compares light extinction due to background fine particles with light extinction due to anthropogenic fine particles. 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).

This fact is well-documented by data from the IMPROVE visibility monitoring network (Sisler et al., 1993). Table VII-4 compares total light extinction (from background and anthropogenic sources) with light extinction due only to natural sources for several locations across the country. The table demonstrates the significant role in most parts of the country of anthropogenic emissions in overall light extinction as compared to background fine particle levels.

It should be noted 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. National concern with protecting visibility in the highly valued national parks and wilderness areas in this region 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. The Commission is required to provide the Administrator with recommendations for protecting regional visibility in the future.

10. Regional Differences in Anthropogenic Pollutant Levels

While total light extinction levels vary significantly across the country, so does the mix of visibility-impairing pollutants from region to region. Table VII-5, taken from the 1993 National Academy of Sciences (NAS) 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

REGION	TOTAL LIGHT EXTINCTION, 1988-1991 (in Mm^{-1})		VISUAL RANGE (in km)	
	Annual	Summer	Annual	Summer
Eastern U.S., estimated natural light extinction	**26 +/- 7**	**NA**	**150+/- 45**	**NA**
Appalachian	112	193	35	20
Boundary Waters	68	73	58	54
Northeast	71	88	55	44
Washington, D.C.	164	192	24	20
Western U.S., estimated natural light extinction	**17 +/- 2.5**	**NA**	**230 +/- 40**	**NA**
Colorado Plateau	27	29	145	135
Cascades	59	68	66	58
Southern California	63	76	62	51
Northern Rockies	54	46	72	85

Source: Sisler et al. 1993

Table VII-4. Comparison of total light extinction to estimated natural light extinction for several eastern and western locations.

Table VII-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%.

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 source categories responsible for visibility-impairing fine particle and precursor emissions are listed in table VII-6 (NAS, 1993).

11. 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., aesthetic and health-related) on the many residents of large metropolitan areas. Urban areas generally have higher loadings of fine particulate matter than monitored class I areas, and they demonstrate significant variability in the degree to which different pollutant types contribute to overall light extinction. Table VII-7 illustrates annual average and second-highest maximum fine PM levels for various cities in the U.S.

Table VII-8 illustrates the difference between percentage contributions of particle types 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 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.

12. Staff Considerations Pertaining to the Effects of PM on Visibility

Impairment of visibility in multi-state regions, urban areas, and class I areas (i.e., certain national parks, wilderness areas, and international parks as described in section 162(a) of the Act) is clearly an adverse effect on public welfare. The staff has considered a number of factors in assessing an appropriate regulatory response.

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 15 $\mu g/m^3$ to less than 30 $\mu g/m^3$ and the range under consideration for a 24-hour standard is 25 $\mu g/m^3$ to less than 85 $\mu g/m^3$.

Table VII-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: National Research Council, Protecting Visibility in National Parks and Wilderness Areas, National Academy Press, 1993.

LOCATION	NUMBER OF MONITORS	ANNUAL MEAN, RANGE OF HIGHEST VALUES FOR DIFFERENT MONITORS, YEARS 1983-1993 (in $\mu g/m^3$)	SECOND HIGHEST MAXIMUM 24-HOUR VALUE, RANGE OF HIGHEST VALUES FOR DIFFERENT MONITORS, YEARS 1983-1993 (in $\mu g/m^3$)
Archuleta County, CO	1	22.7	48
Baltimore, MD	2	24.7 - 26.1	45 - 106
Boston, MA	1	20.2	55
Cleveland, OH	1	28.4	55
Columbia, SC	2	22.3 - 33.0*	43 - 60
Dallas-Fort Worth, TX	1	13.6	37
Detroit, MI	4	17.3 - 25.4*	44 - 73
El Paso, TX	1	14.5	58
Fresno, CA	3	15.4 - 26.0	58 100
Houston, TX	1	16.6	37
Los Angeles-Long Beach, CA	2	27.5 - 32.0	70 - 88
Minneapolis, MN	1	13.6	38
Nashville, TN	1	28.6	64
New York, NY	4	21.9 - 47.0*	51 - 91
Philadelphia, PA	1	26.0	47
Pittsburgh, PA	2	25.7 - 26.6	59 82
Portland, OR	5	10.2 - 17.2	19 - 60
Portland, ME	1	39.9*	50
Riverside, CA	2	11.0 - 43.3	22 - 114
Salt Lake City, UT	3	14.0 - 52.9*	14 - 91
St. Louis, MO	6	14.7 16.9	38 - 49
Steubenville, OH	4	16.6 - 25.7	42 - 81
Syracuse, NY	2	20.0 - 40.8*	47 - 72
Winston-Salem, NC	2	23.4 24.7	51 - 56

Source: U.S. EPA, monitored values from AIRS database

*These data are unreliable due to incomplete and/or unrepresentative monitoring.

Table VII-7 **Annual Average and Second Highest Maximum Fine PM Levels for Selected U.S. Cities.**

Location	Sulfate	Nitrate	Organics	Elemental Carbon	Soil and Coarse
Wash, DC	49.0	16.0	16.2	11.9	6.9
So. Calif.	14.4	44.4	18.2	9.0	13.9

Source: Sisler et al. 1993

Table VII-8. Percentage contributions of particle types to annual average total light extinction in the Washington, D.C. and southern California areas.

Table VII-7 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, and possibly in broader areas in the East if regional attainment strategies are emphasized. To examine any expected visibility improvement resulting from these reductions requires an understanding of the relationship between fine particle loadings and visibility. Figure VII-5 shows that visibility change is sensitive to current fine particle concentrations and their associated extinction efficiency. To achieve a given amount of visibility improvement, a larger reduction in fine particle concentration is required in areas with higher existing concentrations, such as the East. Expected reductions in fine particle concentrations resulting from adoption of the primary fine particle standards in the lower half of the recommended range is expected to result in maintained or improved visibility in many urban areas and in a broader area in the East. Improvement of visibility would be greater if regional fine particle attainment strategies are emphasized. 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 sulfate levels would be reduced over a wide area in the eastern U.S. by the year 2010, resulting in potential improvements in annual average visibility for the region. The analysis projected no expected improvement in the rural West. Any additional regional strategies to attain fine particle standards could lead to further visibility improvements, particularly in the East. However, there is no evidence that adoption of the primary fine particle standards in the lower half of the recommended range will eliminate adverse impacts of fine particles on visibility.

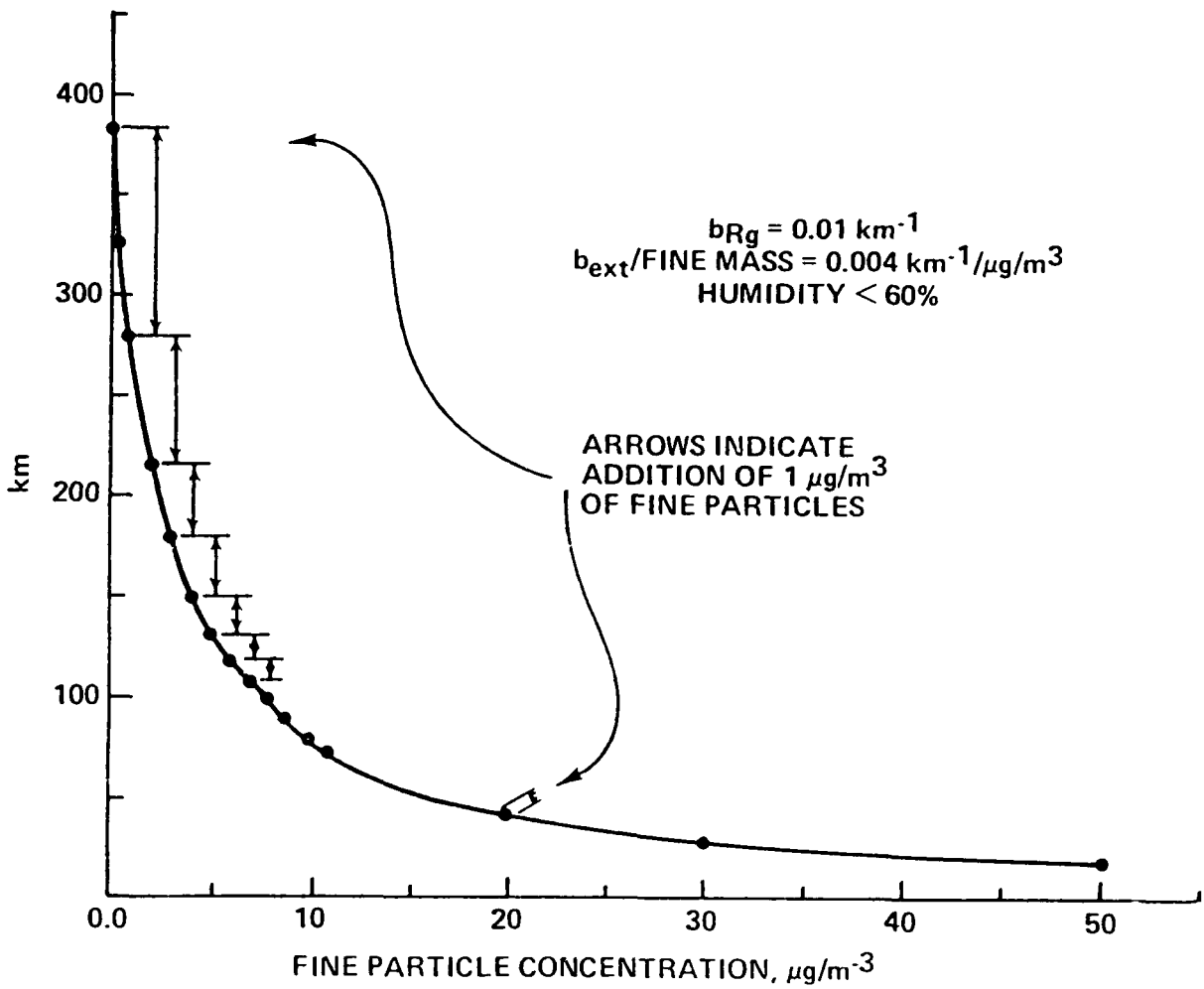


Figure VII-5. Effects of fine particle increments on calculated visual range. Addition of 1 μg/m3 to a clean atmosphere reduces visual range by 30 percent. Addition of the same amount when background visual range is 35 km (20 miles) produces a 3 percent reduction.

The staff has also considered whether the adoption of a national secondary standard would provide adequate protection of public welfare across the country. Due to the regional variability in background fine particle levels, the staff has concluded that a national secondary standard could not achieve this objective. The data presented in table VII-4 indicates that current annual average light extinction levels on the Colorado plateau are about equal to background levels in the East. In other words, a national secondary standard set to maintain or improve visibility conditions in the Colorado plateau would have to be set at or below natural background levels in the East. Conversely, a national secondary standard that would be both attainable and improve visibility in the East would permit further degradation in the West.

A more promising option is to establish a regional haze program under section 169A of the Clean Air Act, which would address the existing adverse effects of fine particles on visibility in both class I and non-class I areas. 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.

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, it is expected that the development of a regional haze program will have associated benefits outside of mandatory class I areas. The National Academy of Sciences concluded that:

> 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 adverse effects of fine particle pollutants on visibility.

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 on 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 link between ambient pollutant concentrations and damage to various building stones is difficult to quantify.

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 nationally. The level of this impairment varies greatly from East to West, in terms of total loadings, pollutant mix, and resulting total light extinction. Background levels of fine particles and humidity vary regionally as well, with the East having higher levels than the West.
2) Because of regional variations in natural 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 the most effective approach for addressing visibility impairment. Therefore, the staff recommends that the Administrator consider establishing regional haze regulations under section 169A of the Act.
3) The available data assessed in the CD does not provide an adequate basis to establish a national secondary standard to protect against soiling and materials damage effects.

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APPENDIX A

Considerations in Selecting Particle Size Cut Point for Fine Particles

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. 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. 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. The main policy choice centers on two options: $PM_{2.5}$ and PM_1. 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.

From a public health perspective, $PM_{2.5}$ captures all of the potential agents of concern in the fine fraction. For example, $PM_{2.5}$ 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 hygroscpic particles may grow above 2.5μm, $PM_{2.5}$ is still better able to capture them than PM_1.

$PM_{2.5}$ has been used directly in many health studies as described in the CD and Chapter V above (see Table V-5). Associations have been reported between exposures to $PM_{2.5}$ and mortality, hospital admissions, cough, upper respiratory infection, lower respiratory infection, and asthma status, and pulmonary function changes (although not all of these associations are statistically significant).

$PM_{2.5}$ 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 (1989-present), and 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 using $PM_{2.5}$ 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.

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. Health studies report 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 the PM 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 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) reading varies more with darkness of particles (i.e., carbon content) than with mass, making associations with mass highly site- and time-specific. Using a similar principle to BS, the principle of 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. 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.

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

Chemical Composition Data for Particulate Matter

PM2.5 COMPOSITION (24-h AVG) EASTERN U.S. Units = ug/m3

Ref	1	1	2(b)	3	4(c)	5(d)	5(d)	6,7	8(a)	8(a)
Site	Smoky Mtns	Shenandoah	Camden	Philadelphia	Deep Creek	Raleigh	Roanoke	Watertown	Hartford	Boston
Dates	9/20-26/78	7/23-5/08/80	7/14-8/13 '82	7/25-8/14/94	8/83	1/85-3/85	10/88-2/89	5/79-6/81	1980	1980
Hours			6am-6pm-6am		4x daily	7am-7pm-7am	7am-7pm-7am	0000-0000		
Dur	12	12	12	24	6	12	12	24	24	24
Number	12	28	50	21	98			354	2	1
Mass	24.00	27.00	28.70	32.18	40.00	30.30	19.90	14.90	26.75	34.80
OC	2.22	0.44	2.05	4.51	1.45	10.00	7.30			
EC	1.10	1.12	1.87	0.76	0.18	0.50	1.50			
Nitrate	0.30		<0.48		0.57					
Sulfate	12.00	13.60	11.20					6.50		
Acidity								20.3		
Al	<0.054	<0.105	0.053	0.114		0.009	0.176		0.035	
As	<0.003	<0.003	0.001		0.001	0.001	0.002			0.002
Ba										
Br	0.018	0.008	0.029	0.009	0.005	0.028	0.005	0.088	0.036	0.020
Ca	0.016	0.035	0.040	0.058	0.048	0.018	0.047	0.041	0.070	0.070
Cd			0.002							
Cl	<0.010	0.010	0.003	0.026		0.007	0.053	0.084		
Cr			0.002			0.000	0.001		0.003	0.004
Cu	0.003	0.005		0.007		0.020	0.007		0.043	0.035
Fe	0.028	0.054	0.091	0.127	0.058	0.044	0.114	0.074	0.125	0.121
K	0.040	0.061*	0.101	0.060	0.044	0.159	0.177		0.171	0.096
Mg				0.023						
Mn			0.006	0.003	0.003	0.003	0.012	0.004	0.007	0.001
Mo			0.001			0.001	0.001			
Na			0.146	0.070	0.034					
Ni			0.011	0.007				0.009	0.010	0.012
P				0.015						0.009
Pb	0.097	0.052	0.249	0.019	0.048	0.096	0.027	0.329	0.510	0.285
Rb										
S	3.744	4.539	4.200	3.251	6.700	1.729	1.177	1.800	2.219	3.869
Sb			0.079		0.001					
Se	0.001	0.001	0.002	<0.002	0.003	0.002	0.002	0.001	0.001	0.001
Si	0.038	0.116	0.103	0.165	0.150	0.076	0.077	0.100	0.177	0.144
Sn			<0.012							
Sr			<0.002							
Ti	<0.006	<0.010	<0.027	<0.042					0.002	
V	<0.004	<0.010	0.013	<0.013	0.001	0.003	0.004	0.022	0.017	0.020
Zn	0.009	0.011	0.082	0.041	0.013	0.015	0.083		0.079	0.046

References are listed in Table 1 Appendix. Associated notes are explained in Table 1.

* Values for this size fraction are calculated from the average measured values reported for the other two size fractions.

PM2.5 COMPOSITION (24-h AVG) WESTERN U.S. Units = ug/m3

Ref	8(a)	9(g)	9(g)	10(i)	11(j)	5(d)	12(f)	8(a)	8(a)	8(a)
Site	Res.Tr.Pk	Los Angeles	Los Angeles	San Joaquin Valley	Phoenix	Boise	Nevada	Tarrant CA	Five Points C	Riverside CA
Dates	1980	Summer'87	Fall'87	6'88-6'89	10/13/89-1/17/90	12/86-3/87	11/86-1/87	1980	1980	1980
Hours						7am-7pm-7a	00-2400			
Dur	24	4,5 and 7	4 and 6	24	6 h, 2x/day	12	24	24	24	24
Number	3	11 days	6 days	~35	~100 days		24	6	3	4
Mass	28.77	41.10	90.20	29.89	29.37	35.70	56.92	57.05	31.80	35.18
OC		8.27	18.46	4.87	10.10	12.70	19.97			
EC		2.37	7.28	3.24	7.47	1.70	15.17			
Nitrate		4.34	22.64	8.17	3.60		2.43			
Sulfate		9.41	4.38	3.00	1.33		1.67			
Acidity										
Al	0.073	0.035	0.250	0.152	0.130	0.102	0.275	0.177	0.239	0.036
As	0.002	0.022	0.015		<0.020	0.002	0.001			
Ba		0.015	0.043	0.012	<0.106		0.013			
Br	0.007	0.013	0.065	0.010	0.011	0.014	0.033	0.102	0.015	0.037
Ca	0.035	0.022	0.335	0.096	0.170	0.026	0.215	0.455	0.150	0.301
Cd				<0.007	<0.018					
Cl		0.093	0.453	0.094	0.365	0.122	0.145		0.004	0.009
Cr		0.022	0.025	0.003	0.003	0.001	0.002	0.002	0.001	
Cu	0.016	0.063	0.273	0.096	0.015	0.011	0.010	0.047	0.024	0.040
Fe	0.120	0.099	0.557	0.180	0.216	0.022	0.310	0.316	0.216	0.127
K	0.148	0.041	0.217	0.188	0.207	0.145	0.280	0.186	0.244	0.120
Mg		0.024	0.075							
Mn	0.003	0.016	0.043	0.006	0.023	0.002	0.015	0.032	0.005	0.007
Mo					<0.006	0.002				
Na		0.202	0.466							
Ni	0.001	0.005	0.007	0.016	0.003		0.006	0.003	0.025	0.007
P	0.042	0.060	0.046	0.007	<0.051		0.041		0.007	
Pb	0.106	0.038	0.185	0.029	0.039	0.045	0.115	0.619	0.087	0.376
Rb				0.001	<0.0025		0.001			
S	2.835	2.832	1.998	1.242	0.437	0.603	0.765	2.578	1.129	1.653
Sb				<0.002	<0.033					
Se	0.002	0.013	0.011	0.001	<0.002	0.001	0.000		0.001	0.001
Si	0.350	0.052	0.520	0.460	0.430	0.069	0.860	0.583	0.656	0.234
Sn				<0.015	<0.028					
Sr		0.019	0.028	0.002			0.004			
Ti		0.005	0.060	0.017	<0.030		0.043	0.010	0.005	
V		0.006	0.007	0.015	<0.016	0.001	0.009		0.006	0.003
Zn	0.018	0.090	0.298	0.078	0.056	0.019	0.033	0.095	0.016	0.029

References are listed in Table 1 Appendix. Associated notes are explained in Table 1.

* Values for this size fraction are calculated from the average measured values reported for the other two size fractions.

PM2.5 COMPOSITION (24-h AVG) CENTRAL U.S. Units = ug/m3

Ref	8(a)	8(a)	8(a)	8(a)	8(a)	5(d)	13	14(m)	14(aa)	15
Site	San Jose CA	Honolulu	Winnemucca	Portland	Seattle	Albuquerque	Denver	Urban Denver	Non-urban Denver	Chicago
Dates	1980	1980	1980	1980	1980	12/84-3/85	1/11-30/82	11/87-1/88	11/87-1/88	7/94
Hours						7am-7pm-7am	6am-6pm-6a	9am-4pm-9am	9am-4pm-9am	0800-0800
Dur	24	24	24	24	24	12	12	7 & 17	7 & 17	24
Number	6	1	5	4	1		~26	~136	~150	16
Mass	36.28	21.10	9.68	37.18	10.70	20.60	20.73	19.67	10.35	13.57
OC						13.20	7.11	7.25		5.39
EC						2.10	2.15	4.41		1.31
Nitrate							2.22	3.96		
Sulfate							2.06	1.55		
Acidity										
Al	0.123	1.127	0.361	0.581	0.002	0.077	0.394	0.037		0.046
As	0.001			0.012	0.006		<0.002			<0.003
Ba							0.031			<0.091
Br	0.188	0.017	0.006	0.093	0.019	0.085	0.103	0.018		0.004
Ca	0.089	1.024	0.243	0.154	0.037	0.059	0.047	0.058		0.045
Cd							0.006	0.005		<0.029
Cl	0.050	0.518		0.021		0.036	0.052	0.141		0.011
Cr	0.003	0.004		0.009	0.002		<0.009	0.003		<0.005
Cu	0.043	0.018	0.026	0.072	0.024		0.010	0.017		0.011
Fe	0.148	0.726	0.231	0.270	0.098	0.045	0.079	0.111		0.089
K	0.248	0.371	0.149	0.218	0.080	0.074	0.079	0.077		0.061
Mg										0.012
Mn	0.006	0.020	0.003	0.052	0.004		0.011	0.012		0.005
Mo						0.000				<0.002
Na										0.022
Ni	0.006	0.002	0.001	0.027	0.006		0.003	0.002		<0.001
P	0.013	0.002		0.017	0.006		0.043			0.008
Pb	0.891	0.071	0.042	0.422	0.215	0.237	0.326	0.075		0.027
Rb							<0.003			
S	0.852	0.313	0.358	1.944	0.831	0.507	0.709	0.642		1.321
Sb								0.004		<0.042
Se				0.001	0.001			0.001		<0.001
Si	0.292	2.363	0.914	0.377	0.092	0.076	0.277	0.272		0.074
Sn								0.006		<0.049
Sr							<0.003	0.001		
Ti		0.063	0.009	0.005			<0.027	0.009		<0.029
V	0.002	0.001		0.014						<0.009
Zn	0.061	0.011	0.011	0.081	0.059	0.007	0.046	0.031		0.052

References are listed in Table 1 Appendix. Associated notes are explained in Table 1.

* Values for this size fraction are calculated from the average measured values reported for the other two size fractions.

PM2.5 COMPOSITION (24-h AVG) CENTRAL U.S. Units = ug/m3

Ref	16	6,7	17	6,7	6,7	6,7	8(a)	8(a)	8(a)	8(a)
Site	Houston	Harriman	Harriman	Kingston	Portage	Topeka	El Paso	Inglenook	Braidwood	Kansas City KS
Dates	9/10-19/80	5/80-5/81	9/85-8/86	5/80-6/81	3/79-5/81	8/79-5/81	1980	1980	1980	1980
Hours		0000-0000		0000-0000	0000-0000	0000-0000				
Dur	12	24	24	24	24	24	24	24	24	24
Number	20	256	330	169	271	286	10	8	1	8
Mass	38.60	20.80	21.00	24.60	11.00	12.50	27.16	32.03	28.20	25.66
OC	5.68									
EC	1.42									
Nitrate	0.59									
Sulfate	14.61	8.10	8.70		6.81	6.05				
Acidity		36.1	36.1		10.5	11.6				
Al	0.123						0.155	0.082	0.089	0.091
As	<0.005						0.025	0.001		0.003
Ba	0.048									
Br	0.055	0.038		0.044	0.011	0.045	0.070	0.040	0.003	0.027
Ca	0.155	0.150		0.120	0.045	0.250	0.332	0.326	0.084	0.519
Cd	<0.003									
Cl	0.032	0.021		BQL	0.027	0.031		0.003		
Cr	<0.005						0.001	0.002		0.004
Cu	0.028						0.036	0.032	0.024	0.032
Fe	0.162	0.120		0.097	0.049	0.090	0.134	0.281	0.071	0.189
K	0.119						0.127	0.408	0.052	0.311
Mg										
Mn	0.014	0.017		0.010	0.003	0.004	0.004	0.037	0.001	0.006
Mo										
Na	<0.38									
Ni	0.004	BQL		BQL	BQL	BQL	0.001	0.001	0.001	0.002
P	0.028							0.008		0.013
Pb	0.465	0.180		0.194	0.061	0.163	0.481	0.309	0.041	0.180
Rb	<0.002									
S	4.834	2.500		2.400	1.400	1.100	0.823	2.655	2.060	1.816
Sb	0.006									
Se	<0.002	0.002		0.002	0.001	0.000	0.002	0.001	0.001	0.001
Si	0.210	0.120		0.200	0.075	0.190	0.436	0.685	0.220	0.434
Sn	<0.005									
Sr	<0.002									
Ti	<0.014						0.003			0.004
V	<0.008	BQL		BQL	BQL	BQL				
Zn	0.084						0.055	0.133	0.011	0.034

References are listed in Table 1 Appendix. Associated notes are explained in Table 1.

* Values for this size fraction are calculated from the average measured values reported for the other two size fractions.

PM2.5 COMPOSITION (24-h AVG) CENTRAL U.S. Units = ug/m3

Ref	8(a)	8(a)	8(a)	8(a)	8(a)	8(a)	8(a)	18(k)	6,7	17	6,7
Site	Minneapolis	Kansas City MO	Akron	Cincinnati	Buffalo	Dallas	St. Louis	St. Louis	St. Louis	St. Louis	Steubenville
Dates	1980	1980	1980	1980	1980	1980	1980	8-9/76	9/79-6/81	9/85-8/86	4/79-4/81
Hours									0000-0000		0000-0000
Dur	24	24	24	24	24	24	24	6-12	24	24	24
Number	6	3	7	2	14	4	5		306	311	499
Mass	15.50	16.77	36.09	29.80	38.75	28.93	23.06	34.00	19.00	17.70	29.60
OC											
EC											
Nitrate											
Sulfate									8.10	8.00	12.80
Acidity									10.3	9.7	25.2
Al			0.046	0.062	0.192	0.111	0.119	0.203			
As	0.004	0.007	0.012	0.013	0.009	0.033	0.003	0.002			
Ba								0.020			
Br	0.047	0.064	0.039	0.024	0.003	0.223	0.025	0.132	0.078		0.042
Ca	0.103	0.213	0.110	0.062	0.218	0.691	0.090	0.132	0.101		0.097
Cd								0.004			
Cl								0.087	0.052		0.092
Cr	0.001	0.002	0.010	0.003	0.002	0.005		0.006			
Cu	0.035	0.021	0.037	0.024	0.026	0.043	0.018	0.029			
Fe	0.087	0.140	0.609	0.174	0.671	0.248	0.076	0.275	0.190		0.590
K	0.092	0.142	0.268	0.136	0.310	0.125	0.126	0.261			
Mg											
Mn	0.005	0.006	0.085	0.011	0.033	0.015	0.002	0.036	0.021		0.029
Mo											
Na											
Ni	0.001	0.001	0.006	0.004	0.008	0.002	0.002	0.004	0.003		0.005
P			0.059	0.043	0.060	0.018	0.020	0.001			
Pb	0.308	0.369	0.412	0.343	0.359	1.066	0.277	0.688	0.327		0.216
Rb								0.000			
S	0.907	0.763	3.419	2.876	3.706	1.514	2.333	4.655	2.100		4.700
Sb								0.006			
Se	0.001		0.008	0.005	0.005		0.002	0.004	0.002		0.005
Si	0.169	0.177	0.522	0.328	0.241	0.442	0.170	0.458	0.160		0.290
Sn								0.009			
Sr								0.002			
Ti			0.009	0.003		0.007		0.112			
V					0.001	0.002		0.002	BQL		0.011
Zn	0.045	0.046	0.150	0.053	0.078	0.054	0.023	0.101			

References are listed in Table 1 Appendix. Associated notes are explained in Table 1.

* Values for this size fraction are calculated from the average measured values reported for the other two size fractions.

PM10 COMPOSITION (24-hr AVG) EASTERN U.S. Units = ug/m3

Ref	1(o,q)*	1(o,q)*	2(b)*	3(ab)*	4(c)	5(d)	5(d)	6,7(p,q)	8(a,q)	8(a,q)
Site	Smoky Mtns	Shenandoah	Camden	Philadelphia	Deep Creek	Raleigh	Roanoke	Watertown	Hartford	Boston
Dates	9/20-26/78	7/23-5/08/80	7/14-8/13 '82	7/25-8/14/94	8/83	1/85-3/85	10/88-2/89	5/79-6/81	1980	1980
Hours			6am-6pm-6am		4x daily	7am-7pm-7a	7am-7pm-7a	0000-0000		
Dur	12	12	12	24	6	12	12	24	24	24
Number	12	28	50	21	98			354	2	1
Mass	29.60	34.40	40.10	40.60				24.20	54.60	140.40
OC	2.22	0.44	2.05	4.51						
EC	1.10	1.12	2.29	0.76						
Nitrate	0.30		0.57							
Sulfate	12.00	14.38	11.20					8.94		
Acidity										
Al	BQL	0.311	0.603	0.439					1.910	3.458
As	BQL		0.001							0.003
Ba										
Br	0.023	0.011	0.044	0.012				0.110	0.082	0.045
Ca	0.338	0.339	0.400	0.479				0.250	0.934	1.139
Cd			0.002							
Cl	BQL	0.189	0.072	0.073				0.389	0.302	0.301
Cr			0.002						0.011	0.008
Cu	0.003	0.011		0.021					0.069	0.058
Fe	0.146	0.212	0.581	0.479				0.350	1.195	1.733
K	0.148	0.190	0.252	0.160					0.481	0.629
Mg				0.126						
Mn		BQL	0.017	0.010				0.009	0.028	0.030
Mo			0.001							
Na			0.146	0.206						
Ni	BQL	BQL	0.015	0.009				0.011	0.015	0.034
P				0.042					0.033	0.025
Pb	0.111	0.061	0.303	0.032				0.405	0.681	0.462
Rb										
S	3.744	4.539	4.430	3.251				2.000	2.647	4.371
Sb			0.260							
Se	0.001	0.001	0.002					0.001	0.001	0.001
Si	0.618	0.929	1.713	1.098				1.100	4.694	6.904
Sn			BQL							
Sr			0.002							
Ti	0.018	0.017	0.065	0.030					0.096	0.154
V	BQL	BQL	0.020					0.022	0.025	0.028
Zn	0.009	0.017	0.112	0.092					0.133	0.100

References are listed in Table 1 Appendix. Associated notes are explained in Table 1.

* Values for this size fraction are calculated from the average measured values reported for the other two size fractions.

PM10 COMPOSITION (24-hr AVG) WESTERN U.S. Units = ug/m3

Ref	8(a,q)	9(g)	9(g)	10(i)	11(j)*	5(d)	12(f)	8(a,q)*	8(a,q)*	8(a,q)*
Site	Res.Tr.Pk	Los Angeles	Los Angeles	San Joaquin Valley	Phoenix	Boise	Nevada	Tarrant CA	Five Points CA	Riverside CA
Dates	1980	Summer'87	Fall'87	6'88-6'89	10/13/89-1/17/90	12/86-3/87	11/86-1/87	1980	1980	1980
Hours						7am-7pm-7a	00-2400			
Dur	24	4,5 and 7	4 and 6	24	6 h, 2x/day	12	24	24	24	24
Number	3	11 days	6 days	~35	~100 days		24	6	3	4
Mass	36.93	67.40	98.70	74.05	62.45			100.90	124.37	106.20
OC		11.61	23.35	10.59	14.56					
EC		3.19	8.49	5.62	8.30					
Nitrate		9.47	27.50	10.55	4.46					
Sulfate		11.28	5.39	3.62	2.34					
Acidity										
Al	0.679	0.758	0.847	3.570	2.67			2.407	7.317	3.549
As	0.002	0.007	0.019		BQL					
Ba		0.070	0.127	0.051	0.01					
Br	0.010	0.016	0.072	0.015	0.01			0.149	0.019	0.065
Ca	0.121	0.585	1.190	1.057	2.10			4.543	1.786	5.082
Cd					BQL					
Cl		1.119	0.880	0.487	0.56				0.026	0.173
Cr	0.002	0.023	0.042	0.010	0.01			0.007	0.007	0.005
Cu	0.026	0.022	0.178	0.087	0.04			0.077	0.037	0.061
Fe	0.302	0.836	2.192	1.633	1.47			1.257	3.275	2.015
K	0.216	0.237	0.460	0.820	0.88			0.441	1.437	1.081
Mg		0.335	0.287		BQL					
Mn	0.006	0.033	0.063	0.037	0.05			0.067	0.055	0.049
Mo					BQL					
Na		1.632	0.518		BQL					
Ni	0.001	0.005	0.005	0.010	0.01			0.006	0.037	0.013
P	0.042	0.187	0.099	0.059	0.05			0.002	0.155	0.144
Pb	0.119	0.084	0.251	0.061	0.06			0.786	0.105	0.489
Rb				0.004	BQL					
S	3.058	3.353	2.262	1.463	0.62			2.888	1.422	2.373
Sb					BQL					
Se	0.002	0.008	0.010	0.001	BQL				0.001	0.001
Si	1.737	2.040	2.162	8.037	7.44			5.791	16.657	7.778
Sn					BQL					
Sr		0.018	0.024	0.014	0.01					
Ti	0.021	0.077	0.165	0.147	0.14			0.093	0.277	0.182
V		0.005	0.009	0.014	BQL				0.013	0.003
Zn	0.025	0.114	0.293	0.094	0.09			0.147	0.032	0.059

References are listed in Table 1 Appendix. Associated notes are explained in Table 1.

* Values for this size fraction are calculated from the average measured values reported for the other two size fractions.

PM10 COMPOSITION (24-hr AVG) CENTRAL U.S. Units = ug/m3

Ref	8(a,q)*	8(a,q)*	8(a,q)*	8(a,q)*	8(a,q)*	8(a,q)*	13(q)*	14(m)	14(aa)	15(s)*
Site	San Jose CA	Honolulu	Winnemucca	Portland	Seattle	Albuquerque	Denver	Urban Denver	Non-urban Denver	Chicago
Dates	1980	1980	1980	1980	1980	12/84-3/85	1/11-30/82	11/87-1/88	11/87-1/88	7/94
Hours						7am-7pm-7am	6am-6pm-6am	9am-4pm-9am	9am-4pm-9am	0800-0800
Dur	24	24	24	24	24	12	12	7 & 17	7 & 17	24
Number	6	1	5	4	1		~26	~136	~150	16
Mass	66.68	46.90	65.42	117.55	36.00		56.46			28.54
OC							7.11			5.39
EC							2.15			1.31
Nitrate							2.22			
Sulfate							2.45			5.46
Acidity										
Al	2.053	2.992	6.925	6.932	2.296		3.294			0.269
As	0.001			0.014	0.008		<0.004			<0.0043
Ba							0.089			<0.130
Br	0.250	0.023	0.010	0.121	0.033		0.127			0.011
Ca	0.771	1.981	2.177	1.459	0.585		0.705			0.761
Cd							0.018			<0.041
Cl	0.480	1.456	0.176	0.197	0.228		1.287			0.047
Cr	0.009	0.009	0.006	0.019	0.005		<0.018			<0.0073
Cu	0.071	0.025	0.043	0.109	0.041		0.018			0.017
Fe	1.214	1.384	1.995	2.059	1.001		1.033			0.432
K	0.508	0.665	1.200	0.805	0.231		0.727			0.161
Mg										0.118
Mn	0.027	0.034	0.044	0.108	0.022		0.031			0.013
Mo										<0.0041
Na										0.022
Ni	0.014	0.005	0.003	0.036	0.007		0.008			<0.0018
P	0.045	0.002		0.028	0.006		0.155			0.035
Pb	1.119	0.093	0.063	0.537	0.292		0.424			0.032
Rb							0.005			
S	1.109	0.571	0.573	2.371	0.952		0.709			1.363
Sb							<0.004			<0.059
Se				0.001	0.001		<0.004			<0.0017
Si	5.506	6.129	12.817	12.505	4.424		7.737			0.813
Sn							<0.004			<0.070
Sr							0.009			
Ti	0.086	0.130	0.173	0.191	0.091		0.09			0.019
V	0.002	0.001		0.018			<0.004			<0.013
Zn	0.105	0.019	0.026	0.119	0.093		0.085			0.090

References are listed in Table 1 Appendix. Associated notes are explained in Table 1.

* Values for this size fraction are calculated from the average measured values reported for the other two size fractions.

PM10 COMPOSITION (24-hr AVG) CENTRAL U.S. Units = ug/m3

Ref	16(q)*	6,7(p,q)	17*	6,7(p,q)	6,7(p,q)	6,7(p,q)	8(a,q)*	8(a,q)*	8(a,q)*	8(a,q)*
Site	Houston	Harriman	Harriman	Kingston	Portage	Topeka	El Paso	Inglenook	Braidwood	Kansas City KS
Dates	9/10-19/80	5/80-5/81	9/85-8/86	5/80-6/81	3/79-5/81	8/79-5/81	1980	1980	1980	1980
Hours		0000-0000		0000-0000	0000-0000	0000-0000				
Dur	12	24	24	24	24	24	24	24	24	24
Number	20	256	330	169	271	286	10	8	1	8
Mass	63.40	32.50	30.00	35.40	18.20	26.40	76.21	72.45	56.90	70.33
OC	8.78									
EC	1.42									
Nitrate	2.22									
Sulfate	15.52	11.14	8.70	13.63	7.29	6.60				
Acidity			36.1							
Al	1.216						2.903	2.508	2.020	2.144
As	<0.015						0.037	0.001	0.002	0.003
Ba	0.139									
Br	0.091	0.052		0.056	0.014	0.055	0.103	0.061	0.006	0.036
Ca	2.935	1.800		0.960	0.380	2.400	3.964	2.924	1.490	4.371
Cd	<0.012									
Cl	0.398	0.050		0.018	0.083	0.031	0.043	0.003		
Cr	0.007						0.004	0.006	0.002	0.010
Cu	0.046						0.083	0.059	0.044	0.048
Fe	0.766	0.690		0.360	0.230	0.580	0.946	1.474	0.727	0.989
K	0.289						0.623	0.717	0.355	0.660
Mg										
Mn	0.035	0.038		0.027	0.009	0.020	0.027	0.078	0.018	0.026
Mo										
Na	<1.49									
Ni	0.008	0.001		ND	0.001	0.001	0.002	0.003	0.002	0.005
P	0.128							0.030	0.014	0.013
Pb	0.589	0.237		0.234	0.074	0.203	0.672	0.388	0.054	0.237
Rb	<0.006									
S	4.83	2.500		2.400	1.500	1.200	1.072	2.969	2.632	2.031
Sb	0.006									
Se	<0.003	0.002		0.002	0.001	0.000	0.003	0.001	0.002	0.001
Si	3.200	2.000		1.900	0.980	2.500	5.813	6.997	5.987	4.976
Sn										
Sr										
Ti	0.036						0.080	0.116	0.083	0.076
V	<0.045	ND		ND	ND	ND				
Zn	0.142						0.112	0.188	0.023	0.060

References are listed in Table 1 Appendix. Associated notes are explained in Table 1.

* Values for this size fraction are calculated from the average measured values reported for the other two size fractions.

PM10 COMPOSITION (24-hr AVG) CENTRAL U.S. Units = ug/m3

Ref	8(a,q)*	8(a,q)*	8(a,q)*	8(a,q)*	8(a,q)*	8(a,q)*	8(a,q)*	18(x)*	6,7(p,q)	17*	6,7(p,q)
Site	Minneapolis	Kansas City MO	Akron	Cincinnati	Buffalo	Dallas	St. Louis	St. Louis	St. Louis	St. Louis	Steubenville
Dates	1980	1980	1980	1980	1980	1980	1980	8-9/76	9/79-6/81	9/85-8/86	4/79-4/81
Hours									0000-0000		0000-0000
Dur	24	24	24	24	24	24	24	6-12	24	24	24
Number	6	3	7	2	14	4	5		306	311	499
Mass	46.35	58.43	70.90	62.95	83.32	61.55	56.82	62.00	31.40	27.60	46.50
OC											
EC											
Nitrate											
Sulfate									11.14	8.00	17.60
Acidity										9.7	
Al	2.191	2.284	2.555	2.972	3.000	1.405	3.956	1.412			
As	0.005	0.010	0.015	0.013	0.009	0.039	0.004	0.003			
Ba								0.054			
Br	0.069	0.093	0.064	0.041	0.015	0.274	0.046	0.179	0.099		0.052
Ca	1.674	3.967	1.541	1.374	2.768	4.127	1.874	2.949	1.600		1.120
Cd								0.005			
Cl	0.293	0.530	0.572	0.103	0.728	0.029	0.053	0.344	0.145		0.303
Cr	0.003	0.006	0.024	0.005	0.017	0.010	0.001	0.015			
Cu	0.057	0.036	0.055	0.038	0.048	0.066	0.032	0.043			
Fe	0.831	1.119	2.249	1.057	2.711	0.968	0.663	1.493	0.770		2.200
K	0.402	0.503	0.592	0.499	0.516	0.335	0.417	0.653			
Mg											
Mn	0.031	0.031	0.129	0.032	0.111	0.035	0.019	0.071	0.040		0.068
Mo											
Na											
Ni	0.002	0.003	0.011	0.007	0.017	0.004	0.004	0.009	0.005		0.008
P			0.059	0.080	0.060	0.018	0.020	0.099			
Pb	0.406	0.478	0.509	0.442	0.467	1.318	0.372	0.877	0.415		0.259
Rb								0.002			
S	1.131	1.043	3.870	3.265	4.471	1.754	2.612	5.188	2.300		5.500
Sb								0.007			
Se	0.001		0.008	0.005	0.005		0.002	0.005	0.002		0.005
Si	4.848	4.986	5.531	6.961	2.916	3.652	4.638	4.928	2.100		2.300
Sn								0.010			
Sr								0.009			
Ti	0.062	0.074	0.116	0.099	0.051	0.058	0.058	0.587			
V					0.001	0.002		0.006	ND		0.013
Zn	0.072	0.086	0.219	0.201	0.121	0.084	0.044	0.175			

References are listed in Table 1 Appendix. Associated notes are explained in Table 1.

* Values for this size fraction are calculated from the average measured values reported for the other two size fractions.

COARSE COMPOSITION (24-hr AVG) EASTERN U.S. Units = ug/m3

Ref	1(o)	1(o)	2(b)	3(ab)	4(c)	5(d)	5(d)	6,7(o,p)*	8(a,o)	8(a,o)
Site	Smoky Mtns	Shenandoah	Camden	Philadelphia	Deep Creek	Raleigh	Roanoke	Watertown	Hartford	Boston
Dates	9/20-26/78	7/23-5/08/80	7/14-8/13 '82	7/25-8/14/94	8/83	1/85-3/85	10/88-2/89	5/79-6/81	1980	1980
Hours			6am-6pm-6am		4x daily	7am-7pm-7a	7am-7pm-7am	0000-0000		
Dur	12	12	12	24	6	12	12	24	24	24
Number	12	28	50	21	98			354	2	1
Mass	5.60	7.40	11.40	8.42				9.30	27.85	105.60
OC			<3.00							
EC			0.42							
Nitrate			0.57							
Sulfate		0.78	<0.90					2.44		
Acidity										
Al	<0.300	0.311	0.550	0.325					1.875	3.458
As	<0.001	<0.002								0.001
Ba										
Br	0.005	0.003	0.015	0.003				0.022	0.046	0.025
Ca	0.322	0.304	0.360	0.421				0.209	0.864	1.069
Cd			<0.006							
Cl	<0.012	0.179	0.069	0.047				0.305	0.302	0.301
Cr			<0.009						0.008	0.004
Cu	<0.005	0.006		0.014					0.026	0.023
Fe	0.118	0.158	0.490	0.352				0.276	1.070	1.612
K	0.108	0.129	0.151	0.100					0.310	0.533
Mg				0.104						
Mn		<0.006	0.011	0.006				0.006	0.021	0.029
Mo										
Na				0.136						
Ni	<0.002	<0.003	0.004	0.002					0.005	0.022
P				0.027					0.033	0.016
Pb	0.014	0.009	0.054	0.013				0.076	0.171	0.177
Rb										
S	<0.560	<0.711	0.230	BQL				0.200	0.428	0.502
Sb			0.181							
Se	<0.0006	<0.001	<0.0015	BQL						
Si	0.580	0.813	1.610	0.933				1.000	4.517	6.760
Sn			<0.009							
Sr			0.002							
Ti	0.018	0.017	0.065	0.030					0.094	0.154
V			0.007	BQL					0.008	0.008
Zn	<0.004	0.006	0.030	0.052					0.054	0.054

References are listed in Table 1 Appendix. Associated notes are explained in Table 1.

* Values for this size fraction are calculated from the average measured values reported for the other two size fractions.

COARSE COMPOSITION (24-hr AVG) WESTERN U.S. Units = ug/m3

Ref	8(a,o)	9(g)*	9(g)*	10(i)*	11(j)	5(d)	12(f)	8(a,o)	8(a,o)	8(a,o)
Site	Res.Tr.Pk	Los Angeles	Los Angeles	San Joaquin Valley	Phoenix	Boise	Nevada	Tarrant CA	Five Points CA	Riverside CA
Dates	1980	Summer'87	Fall'87	6'88-6'89	10/13/89-1/17/90	12/86-3/87	11/86-1/87	1980	1980	1980
Hours						7am-7pm-7a	00-2400			
Dur	24	4,5 and 7	4 and 6	24	6 h, 2x/day	12	24	24	24	24
Number	3	11 days	6 days	~35	~100 days		24	6	3	4
Mass	8.17	26.30	8.50	44.17	33.09			43.85	92.57	71.03
OC		3.34	4.89	5.71	4.46					
EC		0.82	1.21	2.38	0.84					
Nitrate		5.13	4.86	2.38	0.86					
Sulfate		1.87	1.01	0.62	0.37					
Acidity										
Al	0.606	0.723	0.597	3.418	2.539			2.230	7.078	3.513
As		BQL	0.004	0.000	<0.002					
Ba		0.055	0.084	0.040	<0.077					
Br	0.003	0.003	0.006	0.006	0.002			0.047	0.004	0.028
Ca	0.086	0.563	0.854	0.961	1.929			4.088	1.636	4.781
Cd					<0.016					
Cl		1.026	0.426	0.393	0.194				0.022	0.164
Cr	0.002	0.002	0.017	0.007	0.008			0.005	0.006	0.005
Cu	0.010	BQL	BQL	BQL	0.021			0.030	0.013	0.021
Fe	0.182	0.737	1.635	1.453	1.259			0.941	3.059	1.888
K	0.068	0.196	0.243	0.632	0.669			0.255	1.193	0.961
Mg		0.311	0.212	0.000						
Mn	0.003	0.017	0.021	0.031	0.032			0.035	0.050	0.042
Mo					<0.005					
Na		1.431	0.052	0.000						
Ni		BQL	BQL	BQL	0.003			0.003	0.012	0.006
P		0.127	0.053	0.052	0.038			0.002	0.148	0.144
Pb	0.013	0.046	0.066	0.032	0.022			0.167	0.018	0.113
Rb					0.003					
S	0.223	0.520	0.264	0.222	0.178			0.310	0.293	0.720
Sb					<0.030					
Se		BQL	BQL	0.000	<0.002					
Si	1.387	1.988	1.642	7.577	7.013			5.208	16.001	7.544
Sn					<0.026					
Sr		BQL	BQL	0.012	0.014					
Ti	0.021	0.072	0.106	0.130	0.121			0.083	0.272	0.182
V		BQL	0.003	BQL	<0.014				0.007	
Zn	0.007	0.024	BQL	0.016	0.034			0.052	0.016	0.030

References are listed in Table 1 Appendix. Associated notes are explained in Table 1.

* Values for this size fraction are calculated from the average measured values reported for the other two size fractions.

COARSE COMPOSITION (24-hr AVG) CENTRAL U.S. Units = ug/m3

Ref	8(a,o)	8(a,o)	8(a,o)	8(a,o)	8(a,o)	5(d)	13(o)	14(m)	14(ab)	15(s)
Site	San Jose CA	Honolulu	Winnemucca	Portland	Seattle	Albuquerque	Denver	Urban Denver	Non-urban Denver	Chicago
Dates	1980	1980	1980	1980	1980	12/84-3/85	1/11-30/82	11/87-1/88	11/87-1/88	7/94
Hours						7am-7pm-7am	6am-6pm-6a	9am-4pm-9am	9am-4pm-9am	0800-0800
Dur	24	24	24	24	24	12	12	7 & 17	7 & 17	24
Number	6	1	5	4	1		~26	~136	~150	16
Mass	30.40	25.80	55.74	80.38	25.30		35.73			14.97
OC										
EC										
Nitrate										
Sulfate							0.39			
Acidity										
Al	1.930	1.865	6.564	6.351	2.294		2.900			0.223
As				0.002	0.002					<0.0013
Ba							0.058			<0.038
Br	0.062	0.006	0.004	0.028	0.014		0.024			0.007
Ca	0.682	0.957	1.934	1.305	0.548		0.658			0.716
Cd							0.012			<0.012
Cl	0.430	0.938	0.176	0.176	0.228		1.235			0.036
Cr	0.006	0.005	0.006	0.010	0.003		<0.009			<0.0024
Cu	0.028	0.007	0.017	0.037	0.017		0.008			0.006
Fe	1.066	0.658	1.764	1.789	0.903		0.954			0.344
K	0.260	0.294	1.051	0.587	0.151		0.648			0.101
Mg										0.106
Mn	0.021	0.014	0.041	0.056	0.018		0.021			0.008
Mo										<0.0017
Na										<0.017
Ni	0.008	0.003	0.002	0.009	0.001		0.005			<0.0007
P	0.032			0.011			0.113			0.027
Pb	0.228	0.022	0.021	0.115	0.077		0.099			0.005
Rb							0.005			
S	0.257	0.258	0.215	0.427	0.121		<0.48			0.043
Sb										<0.017
Se										<0.0006
Si	5.214	3.766	11.903	12.128	4.332		7.460			0.739
Sn										<0.021
Sr							0.009			
Ti	0.086	0.067	0.164	0.186	0.091		0.090			0.019
V				0.004						<0.004
Zn	0.044	0.008	0.015	0.038	0.034		0.039			0.038

References are listed in Table 1 Appendix. Associated notes are explained in Table 1.

* Values for this size fraction are calculated from the average measured values reported for the other two size fractions.

COARSE COMPOSITION (24-hr AVG) CENTRAL U.S. Units = ug/m3

Ref	16(o)	6,7(o,p)*	17	6,7(o,p)*	6,7(o,p)*	6,7(o,p)*	8(a,o)	8(a,o)	8(a,o)	8(a,o)
Site	Houston	Harriman	Harriman	Kingston	Portage	Topeka	El Paso	Inglenook	Braidwood	Kansas City KS
Dates	9/10-19/80	5/80-5/81	9/85-8/86	5/80-6/81	3/79-5/81	8/79-5/81	1980	1980	1980	1980
Hours		0000-0000		0000-0000	0000-0000	0000-0000				
Dur	12	24	24	24	24	24	24	24	24	24
Number	20	256	330	169	271	286	10	8	1	8
Mass	24.80	11.70	9.00	10.80	7.20	13.90	49.05	40.43	28.70	41.67
OC	3.10									
EC										
Nitrate	1.63									
Sulfate	0.91	3.04			0.48	0.55				
Acidity										
Al	1.093						2.748	2.426	1.931	2.284
As	<0.006						0.012		0.002	0.003
Ba	0.091									
Br	0.036	0.014		0.012	0.003	0.010	0.033	0.021	0.003	0.029
Ca	2.780	1.650		0.840	0.335	2.150	3.632	2.598	1.406	3.754
Cd	<0.006									
Cl	0.366	0.029		0.018	0.056	0.000	0.043			0.530
Cr	0.007						0.003	0.004	0.002	0.004
Cu	0.018						0.047	0.027	0.020	0.015
Fe	0.604	0.570		0.263	0.181	0.490	0.812	1.193	0.656	0.979
K	0.170						0.496	0.309	0.303	0.361
Mg										
Mn	0.021	0.021		0.018	0.006	0.016	0.023	0.041	0.017	0.025
Mo										
Na	<0.74									
Ni	0.004	0.001		BQL	0.001	0.001	0.001	0.002	0.001	0.002
P	<0.1							0.022	0.014	
Pb	0.124	0.057		0.040	0.013	0.040	0.191	0.079	0.013	0.109
Rb	<0.003									
S	<1.29	BQL		BQL	BQL	BQL	0.249	0.314	0.572	0.280
Sb	<0.009									
Se							0.001		0.001	
Si	2.990	1.880		1.700	0.905	2.310	5.377	6.312	5.767	4.809
Sn	<0.009									
Sr	<0.008									
Ti	0.036						0.077	0.116	0.083	0.074
V	<0.03									
Zn	0.058						0.057	0.055	0.012	0.040

References are listed in Table 1 Appendix. Associated notes are explained in Table 1.

* Values for this size fraction are calculated from the average measured values reported for the other two size fractions.

COARSE COMPOSITION (24-hr AVG) CENTRAL U.S. Units = ug/m3

Ref	8(a,o)	8(a,o)	8(a,o)	8(a,o)	8(a,o)	8(a,o)	8(a,o)	18(k,r)	6,7(o,p)*	17	6,7(o,p)*
Site	Minneapolis	Kansas City MO	Akron	Cincinnati	Buffalo	Dallas	St. Louis	St. Louis	St. Louis	St. Louis	Steubenville
Dates	1980	1980	1980	1980	1980	1980	1980	8-9/76	9/79-6/81	9/85-8/86	4/79-4/81
Hours									0000-0000		0000-0000
Dur	24	24	24	24	24	24	24	6-12	24	24	24
Number	6	3	7	2	14	4	5		306	311	499
Mass	30.85	41.67	34.81	33.15	44.57	32.63	33.76	28.00	12.40	9.90	16.90
OC											
EC											
Nitrate											
Sulfate									3.04		4.80
Acidity											
Al	2.191	2.284	2.509	2.910	2.808	1.294	3.837	1.209			
As	0.001	0.003	0.003			0.006	0.001	0.001			
Ba								0.034			
Br	0.022	0.029	0.025	0.017	0.012	0.051	0.021	0.047	0.021		0.010
Ca	1.571	3.754	1.431	1.312	2.550	3.436	1.784	2.817	1.499		1.023
Cd								0.001			
Cl	0.293	0.530	0.572	0.103	0.728	0.029	0.053	0.257	0.093		0.211
Cr	0.002	0.004	0.014	0.002	0.015	0.005	0.001	0.009			
Cu	0.022	0.015	0.018	0.014	0.022	0.023	0.014	0.014			
Fe	0.744	0.979	1.640	0.883	2.040	0.720	0.587	1.218	0.580		1.610
K	0.310	0.361	0.324	0.363	0.206	0.210	0.291	0.392			
Mg											
Mn	0.026	0.025	0.044	0.021	0.078	0.020	0.017	0.035	0.019		0.039
Mo											
Na											
Ni	0.001	0.002	0.005	0.003	0.009	0.002	0.002	0.005	0.002		0.004
P				0.037				0.098			
Pb	0.098	0.109	0.097	0.099	0.108	0.252	0.095	0.189	0.088		0.043
Rb								0.002			
S	0.224	0.280	0.451	0.389	0.765	0.240	0.279	0.533	0.200		0.800
Sb								0.001			
Se								0.001			
Si	4.679	4.809	5.009	6.633	2.675	3.210	4.468	4.470	1.940		2.010
Sn								0.001			
Sr								0.007			
Ti	0.062	0.074	0.107	0.096	0.051	0.051	0.058	0.475			
V								0.004	BQL		0.002
Zn	0.027	0.040	0.069	0.148	0.043	0.030	0.021	0.074			

References are listed in Table 1 Appendix. Associated notes are explained in Table 1.

* Values for this size fraction are calculated from the average measured values reported for the other two size fractions.

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APPENDIX C

Summary of $PM_{2.5}$ Air Quality Databases and Model for Predicting $PM_{2.5}$

Appendix C. $PM_{2.5}$ Databases and Models for Predicting $PM_{2.5}$ from PM_{10} Values

PM2.5 Databases

Most of the air quality analyses used all available PM_{10} and $PM_{2.5}$ data (1982–1993) from sites with both PM measurements in the AIRS, IMPROVE, NEPART and Inhalable Particle Network (IPN) monitoring networks, and some analyses used data from the California Air Resources Board (CARB) Dichot and Southern California Air Basin (SCAB) Intensive Monitoring Network databases. Table C-1 describes the concurrent PM_{10} and $PM_{2.5}$ databases. See SAI February 1995 report for more details.

For the peak-to-mean analyses, the combined data from the AIRS, IMPROVE, IPN, CARB Dichot and SCAB Intensive Monitoring Network databases were used. NEPART data were not used because of frequent cases with $PM_{2.5}/PM_{10}$ ratios above 1 (due to the use of different measuring instruments).

Predicting Daily $PM_{2.5}$ Values

Because there are more PM_{10} data than fine particle data, some efforts were made to predict $PM_{2.5}$ concentrations from PM_{10} values. However, variation monitoring techniques and variation in the coarse fraction can complicate the ability to predict daily values, especially in areas with relatively large coarse fractions. Predictions of annual $PM_{2.5}$ values are more reliable than estimates of daily $PM_{2.5}$ values.

In view of the difficulties in predicting daily $PM_{2.5}$ values from PM_{10} mass alone, alternative approaches to the ratio estimators were used to provide better estimates of $PM_{2.5}$ based on PM_{10} and other available data such as region, season, and windspeed.

Table 1. Concurrent PM_{10} and $PM_{2.5}$ Data Bases

STUDY	WHO	WHERE	# SITES	WHEN	REGION/ LANDUSE	NOTES
IMPROVE	NPS	Class I areas	56	since 10/89	rural	data in-house
NESCAUM (NEPART)	NESCAUM	Class I areas	7	since 9/88	NE, rural	only $PM_{2.5}$ is described in reference
AIRS		Mostly Northeastern Urban sites	66, (8 rural)	since '83	mostly NE, urban	
IPN	EPA	All regions, but most in northeast	36, (6 rural)	1/82 - 1/84	mostly NE, urban	dichots
CARB Dichot Network	CARB	California sites	7	routine since 1/80	W, mostly urban?	
SJVAQS	DRI	San Joaquin Valley, CA	6	6/88 - 6/89	SW, urban & rural	3 urban, 3 rural seasonal data summary in-house
SNAPS-II	DRI	Reno, NV	1	2/87 - 3/87	SW, urban	data (graph) in-house (JAPCA 1990)
Phoenix PM_{10}	DRI	Phoenix, AZ	8	9/89 - 1/90	SW, urban	data summary in-house
Phoenix Urban Haze	DRI	Phoenix, AZ	4	9/89 - 1/90	SW, urban	6-hr samples (morning/afteroon); data summary in-house
Tucson PM_{10}	DRI	Tucson, AZ	4	9/89 - 1/90	SW, urban	data summary in-house
Tucson Urban Haze	DRI	Tucson, AZ	1	9/89 - 1/90	SW, urban	6-hr samples (morning/afternoon)
SCAB Intensive Monitoring	SCAQMD	Downtown Los Angeles, CA	1	1/86 - 12/86	SW, urban	data in-house

Table 1. Concurrent PM_{10} and $PM_{2.5}$ Data Bases (concluded)

STUDY	WHO	WHERE	# SITES	WHEN	REGION/ LANDUSE	NOTES
Neighborhood-Scale Rubidoux	DRI	Riverside, CA	3	1/88 - 1/89	SW, urban	PM_{10} only?
Oregon AQSN	ODEQ	Oregon sites	< 25	since '83	NW, rural + urban	dichots at some locations
Jefferson County SIP	Ohio EPA	Steubenville, OH (and WV sites)	5	1/90 - 12/90	NE, urban/ industrial	dichots
PREVENT	UC Davis	Mt. Ranier Nat'l Park	3	6/90 - 9/90	NW, rural	
Shenandoah Visibility	U.C. Davis	Shenandoah Nat'l Park	2	7/91 - 9/91	NE, rural	
MOHAVE	U.S. EPA	CA, NV, AZ sites	31	9/91 - 8/92	SW, rural + urban?	
AUSPEX	DRI	San Joaquin Valley, CA (and other rural)	10	14 episodes 7/90 - 8/90	W, rural	5-hr and 7-hr samples (morning/afternoon)
CADMP	DRI	California sites	10	since 5/88	W, rural	12-hr samples (6am-6pm; 6pm-6am)
Acid Aerosol	Harvard	Uniontown, PA	1	6/91 - 8/91	NE, industrial	Indoor/outdoor study
Forest Response	USDA, DRI	Barton Flats, CA	1	since 10/90	W, rural	12-hr samples (6am-6pm; 6pm-6am)
Puget Sound Air Toxics	NSI	Tacoma Tideflats	2	12/89 - 1/90	NW, urbal/ industrial	12-hr samples (7am-7pm; 7pm-7am)

By significantly augmenting the database of $PM_{2.5}$ concentrations, these regression predictions could be used to provide more complete information on the impact of proposed alternative $PM_{2.5}$ NAAQS formulations. The applicability of these results would depend upon the strength of the regression model (predictive accuracy).

For the regression modeling the 1988–1993 AIRS PM database was combined with wind speed data (daily maximum and daily mean) for the nearest monitor, and AIRS daily maximum hourly ozone data (for the nearest monitor within 20 miles).

The models included regressions on selected measured factors and variables such as season, region, wind speed, and daily maximum hourly ozone; however, the predictions of daily $PM_{2.5}$ values still has uncertainties as represented by a low R squared correlation statistic ($R^2 = 0.4$) and by high mean squared errors of the predicted ratio (0.025) and $PM_{2.5}$ concentration.

The best model was selected using a stepwise regression procedure to avoid including terms that would increase the complexity of the model without significantly improving the model accuracy. This model expresses the mean ratio as the sum of terms for season, land use, region, log daily maximum hourly ozone concentration, log daily maximum wind speed, log daily mean wind speed, some of their two-factor interactions and quadratic terms, together with a multiple of PM_{10}. (An interaction measures the extent to which the effect of one variable varies with the level of another variable. For example, the inclusion of a season/region interaction term means that the model assumes that the mean ratio for a season is different for different regions.)

The final model had a R-squared statistic of 0.4. The "typical" error, as defined by the root mean square error divided by the mean measured value, is about 30 percent for the ratio and about 40 percent for the predicted $PM_{2.5}$. Table C-2 gives more information about the regression models, including a list of the independent variables. For more details about this table and about the regressions, see SAI, 1995.

TABLE C-2. Summary of GLM ratio predictions for 1988–1993 using 3 regression models.

Model	Effects[1]	Range of Predicted Ratios	R-Square	Mean Square Error	
				Predicted Ratio	Predicted $PM_{2.5}$
1. Full	S,R,L,O,P,W,M,SR, SL,SO,SW,SM,RL,RO, RW,RM,LO,LW,LM, OO,OW,OM,WW,WM, MM	0.125 to 1.005	0.402	0.0254	63.20
2. Best	S,R,L,O,W,M,SR,SL, SO,RL,RO,RW,RM, LO,MM,OO,P	0.164 to 0.997	0.399	0.0254	63.01
3. PM Only	S,R,L,SR,LR,SL	0.241 to 0.785	0.345	0.0259	69.43

[1] S = Season; R = Region; L = Land use; P = PM_{10}; O = Log Ozone; W = Log Maximum Wind Speed; M = Log Mean Wind Speed.

All predictions are based on 4,158 observations, of which 2,221 had $PM_{10} > 30\ \mu g/m^3$.

Table C-3. Regression Coefficients

Description	Coefficient	Std. Error
Intercept	1.3959925348	0.1474182834
l1	0.3073145307	0.0956879182
r1	-0.3776503620	0.1309241123
r2	-0.1340208501	0.0539672399
r3	-0.1666333810	0.0402760651
r4	-0.0279404757	0.0229316883
s1	-0.1074828313	0.0378706642
s2	0.0008228475	0.0228386788
s3	0.0378687052	0.0229151628
M	0.0077850304	0.0246859620
W	-0.0571402112	0.0247969456
O	-0.3013312495	0.0597227364
MM	-0.0166049496	0.0076897974
OO	0.0272285221	0.0074196893
P	-0.0005619142	0.0001437664
r1*s1	-0.0063374696	0.0108643150
r2*s1	-0.0234241617	0.0060784225
r3*s1	-0.0022432622	0.0036520019
r4*s1	-0.0039536140	0.0025380381
r1*s2	-0.0034235951	0.0072239755
r2*s2	0.0106803520	0.0037837339
r3*s2	0.0030315657	0.0024723039
r4*s2	0.0013630935	0.0016068555
r1*s3	-0.0316804924	0.0134740214
r2*s3	0.0050697115	0.0051831044
r3*s3	-0.0041470165	0.0033092445
r1*O	0.0448202236	0.0247292661
r2*O	-0.0014492506	0.0105907134
r3*O	0.0457880844	0.0075674780
r4*O	0.0063878655	0.0043171802
r1*M	-0.0162975221	0.0401053217
r2*M	-0.0566163387	0.0187467621
r3*M	0.0015303057	0.0125849727
r4*M	-0.0048482228	0.0096187141
l1*O	-0.0839335316	0.0230356244
s1*O	0.0328830035	0.0086496791
s2*O	0.0006064914	0.0052878957
s3*O	0.0025819148	0.0054471383
l1*r3	0.0243145834	0.0082318869
r1*W	0.0962484064	0.0428591060
r2*W	0.0682528572	0.0222493632
r3*W	-0.0026358922	0.0157960596
r4*W	-0.0120560988	0.0108329495
l1*s1	0.0304714189	0.0147528462
l1*s2	-0.0087578574	0.0089204190
l1*s3	0.0130235897	0.0082105759

P = PM_{10}, O = log (daily maximum hourly ozone),
W = log (daily maximum wind speed),
M = log (daily mean wind speed) r1-r4, s1-s3,
l1 are coded region, season, and land-use variables

APPENDIX D

Strengths and Limitations of Experimental Human and Animal Studies

Appendix D

STRENGTHS 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, as discussed above in section V.A.1., 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, Section 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, Section 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, Section 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, Section 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

Epidemiological Evidence of Short-term Exposure Mortality Effects

TABLE 12-4. SUMMARIES OF PUBLISHED PM_{10}-ACUTE MORTALITY EFFECTS STUDIES BASED ON VARIOUS PM MEASURES

Health Outcome	Synthesis Study	Location	Original PM Measurement	Mean Equivalent PM_{10}	Percent Change Per 10 $\mu g/m^3$ PM_{10} Equivalent	95 Percent Confidence Interval
Total Mortality	Ostro (1993)	London UK	BS	80	0.3	(0.29, 0.31)
		Steubenville OH	TSP	61	0.6	(0.44, 0.84)
		Philadelphia PA	TSP	42	1.2	(0.96, 1.44)
		Santa Clara CA	COH	37	1.1	(0.73, 1.51)
	Dockery and Pope (1994)	St. Louis MO	PM_{10}	28	1.5	(0.1, 2.9)
		Kingston TN	PM_{10}	30	1.6	(-1.3, 4.6)
		Birmingham AL	PM_{10} (3d)	48	1.0	(0.2, 1.5)
		Utah Valley UT	PM_{10} (5d)	47	1.5	(0.9, 2.1)
		Philadelphia PA	TSP (2d)	40	1.2	(0.7, 1.7)
		Detroit MI	TSP	48	1.0	(0.5, 1.6)
		Steubenville OH	TSP	61	0.7	(0.4, 1.0)
		Santa Clara CA	COH	35	0.8	(0.2, 1.5)
Respiratory Mortality	Dockery and Pope (1994)	Birmingham AL	PM_{10} (3d)	48	1.5	(-5.8, 9.4)
		Utah Valley UT	PM_{10} (5d)	47	3.7	(0.7, 6.7)
		Philadelphia PA	TSP (2d)	40	3.3	(0.1, 6.6)
		Santa Clara CA	COH	35	3.5	(1.5, 5.6)
Cardiovascular Mortality	Dockery and Pope (1994)	Birmingham AL	PM_{10} (3d)	48	1.6	(-1.5, 3.7)
		Utah Valley UT	PM_{10} (5d)	47	1.8	(0.4, 3.3)
		Philadelphia PA	TSP (2d)	40	1.7	(1.0, 2.4)
		Santa Clara CA	COH	35	0.8	(0.1, 1.6)

TABLE 12-3. SUMMARIES OF RECENTLY PUBLISHED EPIDEMIOLOGICAL STUDIES RELATING HUMAN MORTALITY TO AMBIENT LEVELS OF PARTICULATE MATTER

PM Measure (Concentrations)	Study Description	Results and Comments	Reference
KM (mean = 25; SD = 11)	Total, respiratory, and cardiovascular mortality in Los Angeles County during 1970 to 1979 related to O_3, CO, SO_2, NO_2, HC, daily max. temperature, relative humidity, and KM (a particulate matter metric of optical reflectance by particles, related to the ambient carbon concentration). Low pass filter used to eliminate short-wave, so that only long-wave associations are studied.	Frequency domain analyses indicated significant short- and long-wave associations with KM. The filtered (i.e., long-wave) data analysis indicated that air pollution (including KM) was significantly associated with seasonal variations in LA mortality.	Shumway et al. (1988)
TSP (OECD Method) (Lyons, France: 3 year mean = 87 $\mu g/m^3$) (Marseilles, France 3 y mean = 126 $\mu g/m^3$)	Daily total, respiratory, and cardiac mortality for persons ≥65 years of age tested for associations with SO_2 and TSP during 1974 to 1976 in Lyons and Marseilles, France. Temperature also considered in analyses.	No significant mortality associations found with TSP, while SO_2 was reportedly associated with total elderly deaths in both cities. Seasonality addressed by analyzing deviations from 3-year average of 31-day running means of variables. However, lags of temperature not considered and probable seasonal differences in winter/summer temperature-mortality relationship not addressed.	Derriennic et al. (1989)
BS (mean = 90.1 $\mu g/m^3$) (24-h avg. daily max. = 709 $\mu g/m^3$)	Daily total mortality analyzed for associations with BS, SO_2, and H_2SO_4 in London, England, during 1963 to 1972 winters. Mean daily temperature and relative humidity also considered.	PM, SO_2, and H_2SO_4 all indicated as having significant associations with mortality (0, 1 day lag). Temperature also correlated (negatively) with mortality, but with a 2-day lag. Seasonality addressed by studying only winters and by applying a high-pass filter to the series and analyzing residuals.	Thurston et al. (1989)

TABLE 12-3 (cont'd). SUMMARIES OF RECENTLY PUBLISHED EPIDEMIOLOGICAL STUDIES RELATING HUMAN MORTALITY TO AMBIENT LEVELS OF PARTICULATE MATTER

PM Measure (Concentrations)	Study Description	Results and Comments	Reference
COH (monthly mean range = 9 to 12)	Daily total, respiratory, cancer, and circulatory associations with daily COH in Santa Clara County, CA, during 1980 to 1982 and 1984 to 1986 winters. Daily mean temperature and relative humidity at 4 PM also considered.	An association found between COH and increased mortality, even after making adjustments for temperature, relative humidity, year, and seasonality.	Fairley (1990)
BS	Daily total mortality in Athens, Greece, and surrounding boroughs during 1975 to 1987 related to BS, SO_2, NO_2, O_3, and CO_2 using multiple regression.	During winter months 1983 to 1987, the daily number of deaths was positively and statistically significantly associated with all pollutants, but the association was strongest with BS.	Katsouyanni et al. (1990a)
BS (annual mean range = 51.6 to 73.3 $\mu g/m^3$) (maximum daily value = 790 $\mu g/m^3$)	For the period 1975 to 1982 in Athens, Greece, 199 days with high SO_2 (>150 $\mu g/m^3$) were each matched on temperature, year, season, day of week, and holidays with two low SO_2 days. Mortality by-cause comparisons made between groups by analysis of variance by randomized blocks. BS correlated with SO_2 at r = 0.73, but not directly employed in the analysis.	Mortality was generally higher on high SO_2 days, with the difference being most pronounced for respiratory conditions. BS levels for each group not provided, and BS-SO_2 confounding not addressed, limiting interpretability of results.	Katsouyanni et al. (1990b)

TABLE 12-3 (cont'd). SUMMARIES OF RECENTLY PUBLISHED EPIDEMIOLOGICAL STUDIES RELATING HUMAN MORTALITY TO AMBIENT LEVELS OF PARTICULATE MATTER

PM Measure (Concentrations)	Study Description	Results and Comments	Reference
KM (mean = 25; SD = 11)	Shumway et al. (1988) 1970 to 1979 Los Angeles mortality dataset analyzed using a high-pass filter to allow investigation of short-wave (acute) associations with environmental variables (by removing seasonality effects). Environmental variables considered in regression analyses included temperature, relative humidity, extinction coefficient, carbonaceous particulate matter (KM), SO_2, NO_2, CO, and O_3.	Analyses demonstrated significant associations between short-term variations in total mortality and pollution, after controlling for temperature. Day-of-week effects found not to affect the relationships. The results demonstrated significant mortality associations with O_3 lagged 1 day, and with temperature, NO_2, CO, and KM. The latter three pollutants were highly correlated with each other, making it impossible to separately estimate particulate matter associations with mortality.	Kinney and Ozkaynak (1991)
TSP (mean = 87 $\mu g/m^3$) (24-h avg. range: 46 to 137 $\mu g/m^3$, 5th to 95th percentiles)	Total deaths in Detroit, MI, 1973 to 1982 analyzed using Poisson methods. Environmental variables considered included TSP, SO_2, O_3, temperature, and dew point. Seasonality controlled via multiple dummy weather and time variables.	Significant associations reported between TSP and mortality in autoregressive Poisson models (RR of 100 $\mu g/m^3$ TSP = 1.06). However, most TSP data estimated from visibility, which is best correlated with the fine aerosol (and especially sulfate) portion of the TSP. Thus, results suggest a fine particle association.	Schwartz (1991)
TSP (mean = 77 $\mu g/m^3$) (max. = 380 $\mu g/m^3$) (5th to 95th percentiles = 37 to 132 $\mu g/m^3$)	Total and cause specific daily mortality in Philadelphia, PA during 1973 to 1980 related to daily TSP and SO_2 (n $\approx$2,700 days). No other pollutants considered in the analysis. Poisson regression models, using GEE methods, included controls for year, season, temperature, and humidity. Autocorrelation addressed via autoregressive terms in model.	Strongest associations found with pollution on the same and prior days. Total mortality (mean = 48/day) estimated to increase 7% (95% C.I. = 4 to 10%) for a 100 $\mu g/m^3$ increase in TSP. Cause-specific effects of TSP were larger (as %). SO_2 associations were non-significant in simultaneous models with TSP, but correlations of their coefficients not reported	Schwartz and Dockery (1992a)

TABLE 12-3 (cont'd). SUMMARIES OF RECENTLY PUBLISHED EPIDEMIOLOGICAL STUDIES RELATING HUMAN MORTALITY TO AMBIENT LEVELS OF PARTICULATE MATTER

PM Measure (Concentrations)	Study Description	Results and Comments	Reference
TSP (mean = 69 $\mu g/m^3$) (5th to 95th percentiles = 32 to 120 $\mu g/m^3$)	Age and cause-specific daily mortality in Philadelphia, PA during 1973 and 1990 related to daily TSP, SO_2, and O_3. Other environmental variables included were: temperature, barometric pressure, humidity, and precipitation. Various models employed, including poisson and autoregessive. Prefiltering methods also applied to remove long-waves in data.	TSP effect found only in winter season. TSP never significant in by-cause analyses of those <15 or ≥ 65 years of age. TSP effects weakened by the addition of other pollutants (TSP-SO_2 r = 0.57). However, the inclusion of barometric pressure and precipitation in these models may have acted as surrogates for PM, potentially confounding results. Correlations between TSP and these variables not presented.	Li and Roth (1995)
TSP (mean = 111 $\mu g/m^3$) (24-h avg. range: 36 to 209 $\mu g/m^3$, 10th to 90th percentiles)	Daily total mortality in Steubenville, OH, between 1974 to 1984 related to TSP, SO_2, temperature, and dew point. Poisson regression employed, because of very low death counts/day (mean = 3.1). Regressions controlled for season by including dummy variables for winter and spring, and autoregressive methods also used to address any remaining autocorrelation.	In regressions controlling for season and weather, previous day's TSP was a significant predictor of daily mortality. SO_2 was less significant in regressions, becoming nonsignificant when entered simultaneous with TSP. Auto-regressive models gave similar results.	Schwartz and Dockery (1992b)
TSP (mean = 113 $\mu g/m^3$) (10th to 90th percentiles = 38 to 212 $\mu g/m^3$)	Daily mortality in Steubenville, OH during 1974 to 1984 related to TSP, SO_2, temperature, and dew point (to allow comparisons of results with Schwartz and Dockery, 1992b). Poisson method employed. Analyses done overall and by-season.	In single pollutant models, the TSP coefficient was the same as Schwartz and Dockery (1992b), but TSP effects were found to be attenuated by SO_2 inclusion in the model. SO_2 was also attenuated by the addition of TSP. It is concluded that TSP and SO_2 effects cannot be separated in this dataset. Intercorrelations among these variables not presented.	Moolgavkar et al. (1995)

TABLE 12-3 (cont'd). SUMMARIES OF RECENTLY PUBLISHED EPIDEMIOLOGICAL STUDIES RELATING HUMAN MORTALITY TO AMBIENT LEVELS OF PARTICULATE MATTER

PM Measure (Concentrations)	Study Description	Results and Comments	Reference
BS (mean = 90.1 $\mu g/m^3$) (range = 0 to 350 $\mu g/m^3$)	Further analysis of London, England data (1965 to 1972) examined by Thurston et al. (1989). Spectral and advanced time series methods applied, including prewhitening and auto- regressive (AR) moving average (MA) methods. Environmental variables considered included BS, SO_2, H_2SO_4, temperature, and relative humidity.	Estimated pollution mean effect of 2 to 7% of all London winter deaths (mean = 281/day). However, the various pollutants' effects were not separated. Independent model test on the 1962 episode confirmed the appropriateness of such methods. Long-wave addressed by considering winters only and by prewhitening the data.	Ito et al. (1993)
BS (range = 50 to 250 $\mu g/m^3$)	Daily total mortality in Athens, Greece, during July, 1987 (when a major "heat wave occurred) compared to the deaths in July during the previous 6 years. Environmental variables considered included: temperature, discomfort index (DI), BS, SO_2, and BS. Confounding effects of day-of-week, month, and long-term trends addressed via dummy variables in OLS regression models.	Mean daily temperature above 30 °C found to be significantly associated with mortality. The main effects of all air pollutants nonsignificant, but the interaction between high air pollution and temperature were significant for SO_2 and suggestive ($p < 0.20$) for ozone and BS.	Katsouyanni et al. (1993)
Suspended Particles (SP) (range = 10 to 650 $\mu g/m^3$)	Daily total mortality in Erfurt, East Germany, during 1980 to 1989 (median = 6/day) related to SO_2, SP, T, RH, and precipitation. SP measurements made only 1988 to 1989. Autoregresssive Poisson models employed (due to low deaths/day) also included indicator variables for extreme temperatures and adjustments for trend, season, and influenza epidemics.	Both SO_2 and SP were found to be significantly associated with increased mortality. In a simultaneous regression, SP remained significant while SO_2 did not. Correlations of these coefficients not provided, however. Pollution effect size similar to that for meteorology.	Spix et al. (1993)
BS (mean = 83 $\mu g/m^3$) (range = 18 to 358 $\mu g/m^3$)	Daily total mortality in Athens, Greece, during 1984-1988 (mean = 38/day) related to BS, SO_2, CO, T, and RH. Autoregressive OLS models employed also included indicator variables for season, day of week, and year.	BS, SO_2, and CO all found to be individually significantly associated with increased mortality. In simultaneous regressions, the size of all coefficients declined, with SO_2 still significant and BS approaching significance. CO was no longer significant, but was highly correlated with BS (r = 0.74) in the data.	Touloumi et al. (1994)

TABLE 12-3 (cont'd). SUMMARIES OF RECENTLY PUBLISHED EPIDEMIOLOGICAL STUDIES RELATING HUMAN MORTALITY TO AMBIENT LEVELS OF PARTICULATE MATTER

PM Measure (Concentrations)	Study Description	Results and Comments	Reference
TSP (mean = 52 $\mu g/m^3$; SD = 19.6 $\mu g/m^3$)	Daily total and cause-specific mortality in Cincinnati, OH, (mean total = 21/day) during 1977 to 1982 related to TSP, temperature, and dew point. Poisson model employed with dummy variables for each month and for eight (unspecified) categories of temperature and dew point. Linear and quadratic time trend terms also included. Spline and nonparametric models also applied. Autocorrelation not directly addressed.	TSP was significantly associated with increased risk of total mortality. The relative risk was higher for the elderly and for those dying of pneumonia and cardiovascular disease. However, the analysis failed to consider other pollutants, and there remains the potential for within-month, long-wave confoundings.	Schwartz (1994a)
TSP (mean = 375 $\mu g/m^3$) (maximum = 1,003 $\mu g/m^3$)	Daily deaths during 1989 in two residential areas in Beijing, China, (mean total deaths = 21.6/day) related to TSP and SO_2 using Poisson methods. Controlling indicator variables for quintiles of temperature and humidity, as well as for Sunday also included. Long-wave confounding and autocorrelation not directly addressed. However, season-specific results presented.	Significant mortality associations found for ln (SO_2) and ln (TSP). Associations were strongest for chronic respiratory diseases. In simultaneous regressions, SO_2 was significant, but not TSP. However, the two pollutants were highly correlated with each other (r = 0.6), as well as with temperature. In season-specific analyses, both pollutants were significant in summer, but only SO_2 in winter.	Xu et al. (1994)
PM_{10} (mean = 47 $\mu g/m^3$) (24 h max. = 365 $\mu g/m^3$) (5 day max. = 297 $\mu g/m^3$)	Total, respiratory, and cardiovascular mortality in Utah County, UT, during 1985 to 1989 related to 5-day moving average PM_{10}, temperature, and humidity. Time trend and a random year terms also included in autoregressive Poisson models employed. Seasonality not directly addressed in this basic model, but the addition of four seasonal dummy variables changed results little.	A significant positive association between total non-accidental mortality and PM_{10} was observed, the strongest association being with the 5-day moving average of PM_{10}. The association was largest for respiratory disease, the next largest for cardiovascular, and the lowest for all other. Association noted below 150 $\mu g/m^3$ PM_{10}. The possible influence of other pollutants discussed, but not directly addressed.	Pope et al. (1992)

TABLE 12-3 (cont'd). SUMMARIES OF RECENTLY PUBLISHED EPIDEMIOLOGICAL STUDIES RELATING HUMAN MORTALITY TO AMBIENT LEVELS OF PARTICULATE MATTER

PM Measure (Concentrations)	Study Description	Results and Comments	Reference
PM_{10} St. Louis, MO: (mean = 28 $\mu g/m^3$) (24 hr max. = 97 $\mu g/m^3$) Kingston/Harriman, TN (mean = 30 $\mu g/m^3$) (24 h max. = 67 $\mu g/m^3$)	Total mortality in St. Louis, MO, and Kingston/Harriman, TN (and surrounding counties), during September 1985 to August 1986 related to PM_{10}, $PM_{2.5}$, SO_2, NO_2, O_3, H^+, temperature, dew point, and season using auto-regressive Poisson models.	Statistically significant daily mortality associations found with PM_{10} and $PM_{2.5}$ in St. Louis, but not with other pollutants. In Kingston/Harriman, PM_{10} and $PM_{2.5}$ approached significance, while other pollutants did not. Seasonality was reduced by season indicator, variables, but within season long wave cycles not directly addressed.	Dockery et al. (1992)
PM_{10} (mean = 48 $\mu g/m^3$) (24 h max. = 163 $\mu g/m^3$)	Total daily mortality in Birmingham, AL, from August 1985 to December 1988 related to PM_{10}, temperature, and dew point. Poisson models employed addressed seasonal long wave influences by the inclusion of 24 sine and cosine terms having periods ranging from 1 mo to 2 years. Autoregressive linear models also applied.	Significant associations found between total mortality and prior day's PM_{10}. Various models gave similar results, as did eliminating all days with $PM_{10} > 150$ $\mu g/m^3$. However, the possible role of other pollutants not evaluated.	Schwartz (1993)
PM_{10} (mean = 40 $\mu g/m^3$) (24 h max. = 96 $\mu g/m^3$)	Total, cardiovascular, cancer, and respiratory mortality in Toronto, Canada, during 1972 to 1990 related to PM_{10}, TSP, SO_4, CO, O_3, temperature, and relative humidity. Nineteen-day moving average filtered data used in OLS regressions. Sixty-three hundred and three PM_{10} values estimated based on TSP, SO_4, COH, visibility (B_{ext}) and temperature data, using model developed from 200 PM_{10} sampling days during the period.	Significant associations found between all pollutants considered and mortality, after controlling for weather and long wave influences. However, it was not possible to separate the PM_{10} association from other particulate measures considered. Simultaneous PM and ozone regressions gave significant coefficients for each, but intercorrelations among the pollutants not presented.	Ozkaynak et al. (1994)

TABLE 12-3 (cont'd). SUMMARIES OF RECENTLY PUBLISHED EPIDEMIOLOGICAL STUDIES RELATING HUMAN MORTALITY TO AMBIENT LEVELS OF PARTICULATE MATTER

PM Measure (Concentrations)	Study Description	Results and Comments	Reference
PM_{10} (mean = 58 $\mu g/m^3$) (24 h max. = 177 $\mu g/m^3$)	Total mortality in Los Angeles, CA, during 1985 to 1990 related to PM_{10}, O_3, CO, temperature, and relative humidity. Poisson models employed addressed seasonal long-wave influences by including multiple sine and cosine terms ranging from 1 mo to 2 years in periodicity. OLS and long linear models also tested. Winter and summer analyzed separately also.	Association between PM_{10} and mortality found to be only mildly sensitive to modeling method. CO also individually significant. The addition of either CO or O_3 lowered the significance of PM_{10} in model somewhat, but the PM_{10} coefficient was not as affected, indicating minimal effects on the PM_{10} association by other pollutants in this case.	Kinney et al. (1995)
PM_{10} (mean = 38 $\mu g/m^3$) (24 h max. = 128 $\mu g/m^3$)	Total mortality in Los Angeles, CA and Chicago, IL during 1985 through 1990 related to PM_{10}, O_3, and temperature. Analysis focused on importance of monitor choice to modeling results. Poisson models used addressed seasonal long wave influences by including multiple sine/cosine terms ranging from 1 mo to 2 years in periodicity.	Average of multiple sites' PM_{10} found to be significantly associated with mortality in each city after controlling for season, temperature and ozone. Other pollutants and relative humidity not yet considered. Individual sites' PM_{10} varied from non-significant to strongly significant. Also, dividing the data by season diminished the significance of the multi-site average PM_{10} in mortality regressions. Both site selection and sample size concluded to influence results.	Ito et al. (1995)

TABLE 12-3 (cont'd). SUMMARIES OF RECENTLY PUBLISHED EPIDEMIOLOGICAL STUDIES RELATING HUMAN MORTALITY TO AMBIENT LEVELS OF PARTICULATE MATTER

PM Measure (Concentrations)	Study Description	Results and Comments	Reference
PM_{10} (mean = 115 $\mu g/m^3$) (24 h max. 367 $\mu g/m_3$)	Total, respiratory, and cardiovascular daily deaths/day (means = 55, 8, and 18, respectively) in Santiago, Chili during 1989 through 1991 related to PM_{10}, O_3, SO_2, NO_2, temperature and humidity. Seasonal influences addressed by various methods, including seasonal stratification, the inclusion of sine/cosine terms for 2.4, 3, 4, 6, and 12 month periodicities, prefiltering, and the use of a nonparametric fit of temperature. Log of PM_{10} modeled using OLS with first order autoregressive terms.	Significant association found between PM_{10} and daily mortality, even after addressing potential confounders (e.g., weather), other pollutants, lag structure, and outliers. Strongest associations found for respiratory deaths. SO_2 and NO_2 also significantly associated individually, but only PM_{10} remained significant when all were added simultaneously to the regression. Correlations of the coefficients not reported.	Ostro et al. (1995a)
PM_{10} (mean = 82.4 $\mu g/m^3$) (24 h avg. SE = 38.9 $\mu g/m^3$)	Respiratory mortality among children < 5 years old (mean = 3/day) in Sao Paulo, Brazil during May 1990 through April 1991 related to PM_{10}, SO_2, NO_x, O_3, CO, temperature, humidity, and day of week. Season addressed by including seasonal and monthly dummy variables in regressions. Mortality data adjusted for non-normality via a square root transformation.	Significant association found between respiratory deaths and NO_x, but no other pollutants. No such association found for non-respiratory deaths. However, auto-correlation not addressed. Also, inter-correlations of the pollutant coefficients not reported (but NO_x - PM_{10} correlation = 0.68)	Saldiva et al. (1994)
PM_{10} (mean = 82.4 $\mu g/m^3$) (24 h avg. SE = 38.9 $\mu g/m^3$)	Total mortality among the elderly (≥ 65 years old) (mean = 63/day) in Sao Paulo, Brazil during May 1990 through April 1991 related to two day avg. of PM_{10}, SO_2, NO_x, O_3, and CO, and to temperature, humidity, and day of week. Season addressed by including seasonal and monthly dummy variables. Temperature addressed using three discrete dummy variables.	Significant associations found between total elderly deaths and all pollutants considered. In a simultaneous regression, PM_{10} was the only pollutant which remained significant. The PM_{10} coefficient actually increased in this regression, suggesting interpollutant interactions. Correlations of the pollutant coefficients not provided.	Saldiva et al. (1994)

TABLE 12-3 (cont'd). SUMMARIES OF RECENTLY PUBLISHED EPIDEMIOLOGICAL STUDIES RELATING HUMAN MORTALITY TO AMBIENT LEVELS OF PARTICULATE MATTER

PM Measure (Concentrations)	Study Description	Results and Comments	Reference
PM_{10} (Cook County median = 37 $\mu g/m^3$; max = 365 $\mu g/m^3$) (Salt Lake County median = 35 $\mu g/m^3$; max = 487 $\mu g/m^3$)	Total, respiratory, circulatory, and cancer mortality in Cook County, 1985 to 1990. Elderly, total by race and sex also evaluated. Poisson regression with seasonal adjustments, meteorological variables, and pollen tested. In Salt Lake Count, total and elderly mortality. One daily station in Cook County and two daily monitoring stations in Salt Lake County, plus multiple __-day stations.	Average and single site PM_{10} were significant predictions of PM_{10} in Cook County for total, elderly, cancer, and elderly white mortality, marginal for respiratory, circulatory, and elderly black. Significant Fall and Spring mortality in Cook County, not Summer or Winter. No significant effects in Salt Lake County. No copollutants.	Styer et al. (1995)
PM_{10} (variable by month and year)	Reanalysis of Utah County mortality, 1985 to 1992, broken down by year, season cause and place of death. PM_{10} was entered as a dichotomous variable, less or greater than 50 $\mu g/m^3$. No adjustment for copollutants or for weather in Poisson regression, except for daily minimum temperature. Poisson regression, not GEE.	Variations in RR did not appear to be associated with high or low PM_{10} days. High RR for cancer deaths, age < 60, at home. Highest RR in spring. Increased RR for sudden infant death syndrome. Patterns appear noncausal.	Lyon et al. (1995)

APPENDIX F

Miscellaneous Tables of Effects Information

TABLE 11-2. CONTROLLED HUMAN EXPOSURES TO ACID AEROSOLS AND OTHER PARTICLES

Ref.	Subjects	Exposures[1]	MMAD[2] μm	GSD[3] μm	Duration	Exercise	Temp °C	RH[4] %	Symptoms	Lung Function	Other Effects	Comments
Anderson et al. (1992)	15 healthy 15 asthmatic 18 to 45 years	1): air 2): H_2SO_4 ≈ 100 μg/m^3 3): carbon black ≈ 200 μg/m^3 4): acid-coated carbon with ≈ 100 μg/m^3 H_2SO_4	1.0	2	60 min.	V_E ≈ 50 L/min	22	50	Healthy subjects more symptomatic in air.	Largest decrements in FVC with air exposure.	No change in airway responsiveness	Smoking status of subjects not stated.
Aris et al. (1990)	19 asthmatic 20 to 40 years	Mouthpiece study: HMSA[5] 0 to 1000 μM + H_2SO_4 50 μM vs H_2SO_4 50 μM	6.1			100 W on cycle		100	HMSA did not increase symptoms in comparison with H_2SO_4 alone.	No effects on SRaw[6]		
		Chamber study: HMSA 1 mM + H_2SO_4 5 mM vs H_2SO_4 5 mM	≈7		1 h		≈25					
Aris et al. (1991a)	10 healthy nonsmokers 21 to 31 years ozone sensitive	HNO_3 500 μg/m^3 or H_2O, or air followed by ozone 0.2 ppm	≈6		2 h 3 h	50 min of each h 40 L/min	22	100	No effects of fog exposure	No direct effects of fog exposures. Greatest decrements when ozone preceded by air.	No change in airway responsiveness	Fog may have reduced ozone effects on lung function.
							22	50				
Aris et al. (1991b)	18 asthmatics 23 to 37 years	Mouthpiece study: H_2SO_4 vs NaCl, ≈3000 μg/m^3 with varying particle size, osmolarity, relative humidity	0.4 vs ≈6		16 min	With & without exercise.	≈24	<10 vs 100	No effects	Increases in SRaw with low RH conditions; no pollutant-related effects		Postulated that effects seen in other studies due to secretions or effects on larynx
		Chamber study: H_2SO_4 vs NaCl, 960 to 1400 μg/m^3 with varying water content	6		1 h	100 W on cycle	≈27					

TABLE 11-2 (CONT'D). CONTROLLED HUMAN EXPOSURES TO ACID AEROSOLS AND OTHER PARTICLES

Ref	Subjects	Exposures[1]	MMAD[2]	GSD[3]	Duration	Exercise	Temp °C	RH[4]	Symptoms	Lung Function	Other Effects	Comments
Avol et al. (1988a)	21 healthy 21 asthmatic 18 to 45 years	Air H_2SO_4: Healthy: 363, 1128, 1578 $\mu g/m^3$ Asthmatic: 396, 999, 1,460 $\mu g/m_3$	0.85 to 0.91	2.4 to 2.5	1 h	10 min X 3 47 to 49 L/min	21	50	Healthy: Slight increase in cough with highest concentrations. Asthma: dose-related increase in lower resp. sx.	Healthy: No effects on lung function or airway reactivity. Asthma: ↓FEV_1 0.26 L with H_2SO_4 1,460 $\mu g/m^3$		
Avol et al. (1988b)	22 healthy 22 asthmatic 18 to 45 years	H_2O H_2SO_4: Healthy: 647, 1,100, 2,193 $\mu g/m^3$ Asthmatic: 516, 1,085, 2,034 $\mu g/m_3$	9.7 to 10.7		1 h	10 min X 3 41 to 46 L/min	9	100	Dose-related increase in lower resp. sx. in both groups.	Healthy: No effects on lung function. Asthma: ↓peak flow 16% at 2,034 $\mu g/m^3$ H_2SO_4 .	No effects on airway responsiveness	Half the subjects received acidic gargle; no difference in effects.
Avol et al. (1990)	32 asthmatics 8 to 16 years	Air H_2SO_4 46, 127, and 134 $\mu g/m^3$	0.5	1.9	40 min	30 min rest, 10 min exercise 20L/min/m^2	21	48	No pollutant effect	No pollutant effect. One subject increased SRaw 14.2% with acid exposure.		Did not reproduce findings of Koenig et al., 1983.
Balmes et al. (1988)	12 asthmatics responsive to hypoosmolar saline aerosol 25 to 41 years	Mouthpiece, 5,900 to 87,100 μ/m^3: NaCl 30 mOsm H_2SO_4 30 mOsm HNO_3 30 mOsm $H_2SO_4+HNO_3$ 30 mOsm H_2SO_4 300 mOsm	≈5 to 6	1.5		At rest	≈23			Concentration of acid aerosol required to increase SRaw by 100% lower than for NaCl. No difference between acid species.		Exposures did not mimic environmental conditions. No mitigation by oral ammonia.

TABLE 11-2 (CONT'D). CONTROLLED HUMAN EXPOSURES TO ACID AEROSOLS AND OTHER PARTICLES

Ref.	Subjects	Exposures[1]	MMAD[2]	GSD[3]	Duration	Exercise	Temp °C	RH[4]	Symptoms	Lung Function	Other Effects	Comments
Culp et al. (1995)	16 healthy 20 to 39 yrs	NaCl 1000 $\mu g/m^3$ H_2SO_4 1,000 $\mu g/m^3$	0.9	1.9	2 h	10 min X 4 ≈40 L/min	22	40			Mucins from bronchoscopy: no effects on mucin recovery or changes in glycoproteins	
Fine et al. (1987b)	8 asthmatics 22 to 29 yrs	Mouthpiece: Buffered and unbuffered HCl and H_2SO_4 at varying pH	5.3 to 6.2	1.6 to 1.8		At rest			Cough with inhalation of unbuffered pH 2 aerosols	≈50% increase in airway resistance with buffered acid aerosols at pH 2. Little response to unbuffered acids.		Titratable acidity important determinant of response to acid aerosols.
Fine et al. (1987a)	10 asthmatics 22 to 34 yrs	Mouthpiece: Na_2SO_3 0 to 10 mg/ml, pH 9, 6.6, 4; buffered acetic acid pH 4; SO_2 0.25 to 8 ppm	5.6 to 6.1	1.6 to 1.7		At rest				For Na_2SO_3, broncho-constriction greater at lower pH; no response to acetic acid.		Suggests effects related to release of SO_2 or bisulfite, but not sulfite.
Frampton et al. (1992)	12 healthy 20 to 39 yrs	NaCl 1,000 $\mu g/m^3$ H_2SO_4 1,000 $\mu g/m^3$	0.9	1.9	2 h	10 min X 4 ≈40 L/min	22	40	4/12 subjects: throat irritation with acid exposure.	No pollutant effects	BAL findings: No effects on cell recovery, lymphocyte subsets, AM function, fluid proteins.	
Frampton et al. (1995)	30 healthy 30 asthmatics 20 to 42 yrs	NaCl or H_2SO_4 100 $\mu g/m3$ followed by ozone 0.08, 0.12, or 0.18 ppm	0.45 0.64	4.05 2.50	3 h 3 h	10 min X 6. Healthy: 33 to 40 L/min; asthmatics: 31 to 36 L/min	21	40	No pollutant effects	Healthy subjects: no significant effects. Asthmatics: ozone dose-response following H_2SO_4 pre-exposure, but not NaCl		
Green et al. (1989)	24 healthy 18 to 35 yrs	Air; activated carbon 510 $\mu g/m^3$; HCHO 3.01 ppm; carbon 510 $\mu g/m^3$ + HCHO 3.01 ppm	1.4	1.8	2 h	15 of each 30 min., 57 L/min	22	65	Increased cough with carbon + HCHO	No direct effects of carbon. Additive effects of carbon + HCHO on FVC, FEV_3, peak flow; decrements less than 5%.		

TABLE 11-2 (CONT'D). CONTROLLED HUMAN EXPOSURES TO ACID AEROSOLS AND OTHER PARTICLES

Ref.	Subjects	Exposures[1]	MMAD[2]	GSD[3]	Duration	Exercise	Temp °C	RH[4]	Symptoms	Lung Function	Other Effects	Comments
Hanley et al (1992)	22 asthmatics 12 to 19 yrs	Mouthpiece: 1): Air; H_2SO_4 70, 130 $\mu g/m^3$ 2): Air; H_2SO_4 70 $\mu g/m^3$ with and without lemonade	0.72	1.5	40 min. 45 min.	10 min 30 min ≈30 L./min	22	65	No effects	Significant decreases in FEV_1 (≈37 ml/μmol H+) and FVC at 2 to 3 min but not 20 min after exposure.	Significant correlation between baseline airways responsiveness and $\Delta FEV_1/H^+$ (R^2=0.3).	Large variability in oral NH_3 levels.
Koenig et al (1989)	9 asthmatics with exercise-induced broncho-spasm 12 to 18 yrs	Mouthpiece: Air; H_2SO_4 68 $\mu g/m^3$; SO_2 0.1 ppm; $H_2SO_4+SO_2$; HNO_3 0.05 ppm			40 min.	10 min.	25	65	No effects	↓FEV_1 6% after H_2SO_4 compared with 2% after air.		
Koenig et al (1992)	14 asthmatics with exercise-induced broncho-spasm 13 to 18 yrs	Mouthpiece: Air; H_2SO_4 35 or 70 $\mu g/m^3$	0.6	1.5	45 or 90 min.	≈23 L/min	22	65		↓FEV_1 6% after H_2SO_4 35 $\mu g/m^3$ for 45 min, 3% after 70 $\mu g/m^3$ (NS). Smaller changes after 90 min exposures.		Responses unrelated to $C \times T \times \dot{V}_E$
Koenig et al (1993)	8 healthy 9 asthmatic 60 to 76 yrs	Mouthpiece: Air; $(NH_4)_2SO_4$ ≈70 $\mu g/m^3$; H_2SO_4 ≈74 to 82 $\mu g/m^3$ with and without lemonade	0.6	1.5	40 min	10 min 17.5 L/min for asthmatics, 19.7 for healthy	22	65		No significant effects. Correlation between increase in resistance and oral ammonia levels in asthmatics (R^2 = 0.575).		

TABLE 11-2 (CONT'D). CONTROLLED HUMAN EXPOSURES TO ACID AEROSOLS AND OTHER PARTICLES

Ref.	Subjects	Exposures[1]	MMAD[2]	GSD[3]	Duration	Exercise	Temp °C	RH[4]	Symptoms	Lung Function	Other Effects	Comments
Koenig et al. (1994)	28 asthmatics 12 to 19 yrs	Mouthpiece: Air; ozone 0.12 ppm+NO_2 0.3 ppm; ozone 0.12 ppm+NO_2 0.3 ppm+H_2SO_4 68 $\mu g/m^3$: ozone 0.12 ppm+NO_2 0.3 ppm+HNO_3 0.05 ppm	0.6	1.5	90 min X 2 days	V_E 3 X resting	22	65	No pollutant effects	No pollutant effects	No effects on airway responsiveness	6 subjects with moderate or severe asthma did not complete protocol
Kulle et al. (1986)	20 healthy 20 to 35 yrs	Air; activated carbon 517 $\mu g/m^3$; SO_2 0.99 ppm; carbon 517 $\mu g/m^3$ + SO_2 0.99 ppm.	1.5	1.5	4 h	15 min × 2, 35 L/min	22	60	No symptoms related to carbon exposure	No direct or additive effects of carbon exposure		
Laube et al. (1993)	7 healthy 20 to 31 yrs	Head dome: NaCl ≈500 $\mu g/m^3$ H_2SO_4 ≈500 $\mu g/m^3$	10.3 10.9		1 h	20 min	22 to 25	99	No pollutant effects	No pollutant effects	Tracheal clearance increased (4/4 subjects). Outer zone clearance increased (6/7 subjects). No effects on airway responsiveness	
Linn et al. (1989)	22 healthy 19 asthmatic 18 to 48 yrs	H_2O H_2SO_4 ≈2,000 $\mu g/m^3$	20 10 1		1 h	40 to 45 L/min	≈10	74 to 100	Increased total score with larger acid particles.	No pollutant effects	No effects on airway reactivity	4 asthmatic subjects unable to complete exposures because of symptoms.
Linn et al. (1994)	15 healthy 30 asthmatic 18 to 50 yrs	Air; ozone 0.12 ppm; H_2SO_4 100 $\mu g/m^3$: ozone+H_2SO_4	≈0.5	~2	6.5 h/dX 2 d	50 min X 6 29 L/min	21	50	Symptoms unrelated to atmosphere	↓FEV_1 & FVC in ozone, similar for healthy & asthmatic subjects. Greater fall in FEV_1 for acid+ozone than ozone alone, marginally significant interaction.	Increased airway responsiveness with ozone, marginal further increase with ozone+acid	Average subject lost 100 ml FEV_1 with ozone, 189 ml with ozone+acid Original findings replicated in 13 subjects

TABLE 11-2 (CONT'D). CONTROLLED HUMAN EXPOSURES TO ACID AEROSOLS AND OTHER PARTICLES

Ref.	Subjects	Exposures[1]	MMAD[2]	GSD[3]	Duration	Exercise	Temp °C	RH[4]	Symptoms	Lung Function	Other Effects	Comments
Morrow et al. (1994)	17 asthmatic 20 to 57 yrs 17 COPD 52 to 70 yrs	NaCl ≈ 100 $\mu g/m^3$ H_2SO_4 ≈ 90 $\mu g/m^3$			2 h	Asthmatics: 10 min X 4 COPD: 7 min X 1	21	30	No pollutant effects.	Asthmatics: ↓FEV_1 slightly greater after acid than after NaCl. COPD: No effects.		
Utell et al (1989)	15 asthmatic 19 to 50 yrs	Mouthpiece: NaCl 350 $\mu g/m^3$; H_2SO_4 350 $\mu g/m^3$, high NH_3; H_2SO_4, low NH_3	0.80	1.7	30 min	10 min $\dot{V}_E$ 3X resting		20 to 25		Greater fall in FEV_1 with low NH_3 (19%) than with high NH_3 (8%).		
Yang and Yang (1994)	30 healthy 25 asthmatic 23 to 48 yrs	Mouthpiece: Bagged polluted air, TSP = 202 $\mu g/m^3$			30 min	At rest				Healthy subjects: no change Asthmatics: ↓FEV_1 ≈7%	Increased airway responsiveness in asthmatics reported; no allowance for change in airway caliber	No control exposure

[1]Exposures in environmental chamber unless otherwise stated.
[2]Mass median aerodynamic diameter. In some studies expressed as volume median diameter; see text.
[3]Geometric standard deviation.
[4]Relative humidity.
[5]Hydroxymethanesulfonic acid.
[6]Specific airways resistance.

TABLE 5-2. POSSIBLE RESPONSES TO PARTICLE DEPOSITION IN THE RESPIRATORY TRACT

Principal Region of Deposition	Potential Mechanisms	Potential Consequences/Observations
Extrathoracic (ET)	Chemical and mechanical irritation/stimulation of receptors by deposited material	Symptomatic Effects: -Dryness in nose, mouth, and throat (polymerized dust containing carbon black, Andersen et al., 1979) -Sneezing, rhinitis (pollen), (Michel et al., 1977)
	Slowed, stopped mucociliary clearance by wood dust (Schlesinger and Lippmann, 1970)	Nasal cancer (wood workers, NAS, 1977a, p. 158)
	Enhanced deposition at larynx (Schlesinger and Lippmann, 1976)	Laryngeal cancer (cigarette smoke, NAS, 1977a, p. 150)
Tracheobronchial (TB)	Chemical and mechanical irritation/stimulation resulting in bronchoconstriction (Widdicombe et al., 1962; Nadel, 1973) by: ."Inert" dusts (granulated charcoal, coal dust, carbon dust, calcium carbonate, carbon impregnated plastic, iron hydroxide; Widdicombe et al., 1962; Dubois and Dautrebande, 1958; Andersen et al., 1979; Constantine et al., 1959) .Resuspended urban dust (crustal materials, sulfates, volatiles; Toyama, 1964) .Coarse organic dusts, aeroallergens (grain dusts, pollens, mold, etc. e.g., Dosman, 1980) .Fine acid aerosols (sulfuric acid, ammonium bisulfate; Utell et al., 1981) .Community air pollution with moderately high PM (Lebowitz et al., 1974) .Fine particles in combination with pollutant gases (SO_2; Koenig et al., 1981; McJilton et al., 1976)	Reduced respiratory function Enhanced breathing difficulties or other acute aggravation of heart and lung disease, including: -Asthma (Smith and Paulus, 1971) -Bronchitis (Lawther et al., 1970) -Emphysema and cardiovascular disease (Martin and Bradley, 1960) -Influenza (sodium nitrate, Utell et al., 1980) Enhanced deposition of fine and coarse particles (Albert et al., 1973)
	Enhanced sensitivity to subsequent bronchoconstrictive agents by sulfuric acid (Utell et al., 1981)	As above
	Altered clearance rates, increased mucous production by deposited material (cigarette smoke, sulfuric acid, dusts) (Lippmann et al., 1981; Camner et al., 1973)	Possible promotion of bronchitis by repeated exposure to sulfuric acid (Lippmann et al., 1981) -Increased bronchitis prevalence in people exposed to community air pollution (Holland et al., 1969; Holland and Reid, 1965) -Increased bronchitis prevalence in workers exposed to coal dust, other dusts (Morgan, 1978)
	Direct damage to tissues by acid aerosols	Bronchial lesions (Alarie et al., 1975)
	Increased deposition at bronchial bifurcations (Bell and Friedlander, 1973), slower clearance (Hilding, 1957)	Sites of enhanced deposition are most frequent sites of bronchogenic carcinoma (Schlesinger and Lippmann, 1978)
	Interaction of carcinogens and ambient particles	Intratracheal BaP tumor production potentiated by carbon, iron ore, asbestos (Pylev and Shabad, 1973; Stenback et al., 1976; Saffioti et al., 1968) Increased lung cancer among urban vs. rural smokers

TABLE 5-2. POSSIBLE RESPONSES TO PARTICLE DEPOSITION IN THE RESPIRATORY TRACT (CONTINUED)

TABLE 5-2. POSSIBLE RESPONSES TO PARTICLE DEPOSITION IN THE RESPIRATORY TRACT (CONTINUED)

Principal Region of Deposition	Potential Mechanisms	Potential Consequences/Observations
Pulmonary (Alveolar, AL)	Decreased oxygen transport and probable increased resistance of blood flow through pulmonary capillaries	Aggravation of cardio-pulmonary disease associated with -London air pollution (Ministry of Health, 1954; Martin, 1964)
	Death of macrophages resulting in release of proteolytic enzymes that damage alveolar tissues, by silica, other coarse dusts (Ziskind et al., 1976)	Pneumoconiosis -Sand in desert dwellers (Bar-Ziv and Goldberg, 1974) -Agricultural dust in farm workers (Sherwin et al., 1979 -Dust in zoo animals (Brambilla et al., 1979) -Silica in granite workers (Craighead and Vallyathan, 1980) -Slate dust in miners/quarrymen (Glover et al., 1980) -Coal dust in miners (Morgan, 1978) -Fibrosis followed exposure to chrysotile asbestos (NIOSH, 1976)
	Damage to macrophages, other host defense mechanisms by surface coating of toxic materials (Camner et al., 1974a; Aranyi et al., 1979) Damage to tissues by acid aerosols	Increased susceptibility to infection -Increased mortality in infected mice exposed to Cd, Ni, Mn aerosols (Gardner 1981; Graham et al., 1978; Adkins et al., 1979, 1980) -Loss of alveolar surface area ("emphysema") in dogs exposed to H_2SO_4 (Hyde et al., 1978)
	Combined effect of exposure and slow clearance of particles (Pratt and Kilburn, 1971)	Accumulation of pigment in lungs from inhaled particulate matter (Pratt and Kilburn, 1971; Sweet et al., 1978) Possible role of community air pollution in emphysema (Ishikawa et al., 1969)
Thoracic, not specific to TB, AL	Possible effects on host mechanisms (clearance, immunology) promoting infection	Increased lower respiratory tract infection in children with increased BS+SO_x exposure (Douglas and Waller, 1966; Lunn et al., 1967) Increased influenza rates ("Dust"; Kalpazanov et al., 1976)
	Successive measurements of respiratory function suggest damage to the lung during childhood may be produced by infection (Speizer et al., 1980)	Persistent changes in airways in those children with higher infection rates previously exposed to particulate matter (Colley et al., 1973; Kiernan et al., 1976)
	Absorption of systemic toxicants (e.g. pesticides, trace elements, carcinogens) resulting in extra-respiratory effects. Absorption efficiency greatest for alveolar deposition	Hematological and nervous system effects of lead (EPA, 1977) Gastro-intestinal cancer (Winklestein and Kantor, 1967)

TABLE 5-3. SENSITIVE POPULATION SUBGROUPS

Subgroup	Population Estimates	Rationale (or Criteria)	Observational/Associations Supporting Increased Sensitivity
Individuals with chronic obstructive pulmonary diseases . Bronchitis . Bronchiectasis . Emphysema	7,800,000 (DHEW, 1973)	-Mucus hypersecretion and blocked airways may predispose individuals to bronchospasm -Enlarged airspaces increase blood flow resistance through the pulmonary capillary network, increasing cardiac stress	Many of the deaths and illnesses during and after air pollution episodes were among people with pre-existing obstructive diseases (Ministry of Health, 1954; Martin, 1964; Lawther et al., 1970; Martin and Bradley, 1960)
Individuals with cardiovascular disease	16,100,000 (DOC, 1980)	-Enhanced sensitivity to difficulties in breathing	Many deaths and hospitalizations during pollution episodes among cardiovascular patients (Ministry of Health, U.K., 1954; Martin, 1964)
Individuals with influenza	Unknown	-Increased sensitivity of respiratory epithelium (Utell et al., 1980)	Influenza patients were more sensitive to $NaNO_3$ during their period of sickness (Utell et al., 1980). Highest mortality during influenza epidemic on days with highest PM. (Martin and Bradley, 1960).
Asthmatics	6,000,000 (DHEW, 1973)	-Hyperreactive airways (Boushey et al., 1980)	Sulfuric acid enhanced response to bronchoconstrictive agent in asthmatics, not in normals (Utell et al., 1981)
Elderly	24,658,000 >65 years old (DOC, 1980)	-Reduced lung elasticity (Cotes, 1979) -Immunologically deficient	-Many of the deaths and illnesses during air pollution episodes were among elderly (Ministry of Health, 1954; Martin and Bradley, 1960; Greenburg et al., 1962).
Children	46,300,000 >14 years old (DOC, 1980)	-Immunological immaturity implies diminished protection (Eisen, 1976) -Childhood respiratory infection might prevent the lungs from reaching their full size at maturity (Bouhuys, 1977; Speizer et al., 1980) -Children likely to spend a greater amount of time outdoors and to be more active. Probably higher ventilation rates and thus, increased inhalation of pollutants.	-Increased acute respiratory disease with high particles, SO_x (Lebowitz et al., 1972; Douglas and Waller, 1966) -Effects of acute respiratory disease acquired during childhood persisted until adolescence or young adulthood (Colley et al., 1973; Kiernan et al., 1976).
Smokers	50,000,000 (DHEW, 1977)	-Urban lung cancer in smokers greater (Doll, 1978) -Combinations of PM and carcinogens may enhance response -Increased tracheobronchial deposition (Albert et al., 1973)	-Frequency of respiratory symptoms and diseases greater in smokers exposed to same occupational or community pollution as non-smokers (Lambert and Reid, 1970; NIOSH, 1976).
Mouth or oronasal breathers	15% of population (Niinimaa et al., 1981; Saibene et al., 1978)	-Increased particle penetration (CD, p. 11-20)	