California Environmental Protection Agency

Air Resources Board

Staff Report:

Public Hearing to Consider Amendments to the Ambient Air Quality Standards for Particulate Matter and Sulfates

Prepared by the Staff of
the Air Resources Board and
the Office of Environmental Health Hazard Assessment

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State of California
California Environmental Protection Agency
AIR RESOURCES BOARD

STAFF REPORT: INITIAL STATEMENT OF REASONS FOR PROPOSED RULEMAKING

PUBLIC HEARING TO CONSIDER AMENDMENTS TO THE AMBIENT AIR QUALITY STANDARDS FOR PARTICULATE MATTER AND SULFATES

To be considered by the Air Resources Board on June 20, 2002, at:

California Environmental Protection Agency
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Disclaimer

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Table of Contents

Appendices

Glossary and Abbreviations

AAQS	ambient air quality standard(s)
Aethelometer	an instrument to measure light absorption
AL	alveolar
ARB	Air Resources Board
BAM	beta attenuation monitor
CAAM	continuous ambient air monitor
CAC	correlated acceptable continuous
CAS	California approved sampler
CFR	Code of Federal Regulations
C.I.	confidence interval, a statistical measure of the interval in which the true value of an estimate is likely to be found
Coarse Particles	particles with an aerodynamic diameter between 2.5 and 10 micrometers (microns), also referred to as the coarse fraction, or PM10-PM2.5
COH	coefficient of haze, a measurement of particle light absorption that was historically used as a surrogate for suspended particle mass. A COH instrument draws a known volume of air through a paper filter, then reports the change in light transmittance between a clean filter and the filter with aerosol deposit as though it were a transmittance measurement over a path, equal to the filtered volume divided by the filter area. COH / 1000 ft = $(\log_{10} (I_0/I_1) * 10{,}000) / L$ where I_0 is the clean filter transmittance, I_1 is the transmittance of the filter with aerosol deposit, and L equals the filtered volume divided by the filter area expressed in feet.
COPD	chronic obstructive pulmonary disease
DEP	diesel exhaust particle
DOP	dioctyl phthalate
ESP	electrostatic precipitator
ETS	environmental tobacco smoke
ET	extrathoracic, referring to the upper respiratory tract
Extinction	the reduction of the intensity of a beam of light as it propagates through a transmitting medium: $(I_0-I_1)/I_0$ where I_0 and I_1 are the beam intensity at the beginning and end, respectively, of the transmittance path
Extinction Coefficient	natural logarithm of extinction per unit distance. The Extinction Coefficient is defined as B_{ext} in the following equation: $B_{ext} = - \ln ((I_0-I_1)/I_0) / d$ where I_0 and I_1 are beam intensity at the beginning and end, respectively, of the transmittance path and d is the length of the path
FDMS	filter dynamics measurement system
FEM	federal equivalent method
FEV_1	forced expiratory volume in one second, a measure of lung function

Fine Particles	PM2.5, or particulate matter with a mean aerodynamic diameter of 2.5 micrometers (microns) or less
FRM	federal reference method
FVC	forced vital capacity, a measure of lung function
HRV	heart rate variability, a measure of the heart's ability to respond to stress
ICAM	intercellular adhesion molecule, involved in directing movement of immune cells to the site of injury or inflammation
lpm	liters per minute
LRS	lower respiratory symptoms
Mie Scattering	light scattering by particles with diameters near the wavelength of the light (0.1 μm to 10 μm). Mie scattering is the dominant cause of visible atmospheric haze.
MMEF	mid-maximal expiratory flow, a measure of lung function
NAAQS	National Ambient Air Quality Standard
Nephelometer	an instrument to measure light scattering in air.
nm	nanometer, or one billionth of a meter
NO_2	nitrogen dioxide
NO_x	oxides of nitrogen, which includes nitric oxide (NO), nitrogen dioxide (NO_2), and other oxides of nitrogen
NO_y	total reactive nitrogen
Odds Ratio (OR)	a measure of association between an exposure and disease. An odds ratio of one indicates no association, while odds ratios greater than one or less than one indicate positive and negative associations between the exposure and disease, respectively
OEHHA	California Office of Environmental Health Hazard Assessment
PEF	peak expiratory flow, a measure of lung function
PM	particulate matter
PM10	particulate matter with an aerodynamic diameter of 10 micrometers (microns) or less
PM2.5	particulate matter with an aerodynamic diameter of 2.5 micrometers (microns) or less, also referred to as fine particles
PMNs	polymorphonuclear cells, a class of white blood cells involved in acute inflammatory response
RAAS	reference ambient air monitor
Rayleigh Scattering	light scattering by atmospheric gases. Rayleigh scattering decreases as the fourth power of wavelength. In pure air, blue light (λ = 400 nm) is scattered 9 times more efficiently than red light (λ = 700 nm).
Relative Risk (RR)	a measure of association between an exposure and disease. A relative risk of one indicates no association, while relative risks greater than one

	or less than one indicate positive and negative associations between the exposure and disease, respectively.
REM	regional equivalent monitor
RMSSD	root mean square successive differences, a measure of heart rate variability. More specifically, this measure is the square root of the mean of the sum of squares of differences between adjacent normal beats over the whole electrocardiographic recording.
SBP	systolic blood pressure
scc	sharp cut cyclone
SDNN	standard deviation of all normal R-R intervals, a measure of heart rate variability
SDANN	standard deviation of all normal R-R intervals of successive 5-minute periods, a measure of heart rate variability
SES	sampler equilibration system
SO_2	sulfur dioxide
SO_x	oxides of sulfur, which includes sulfur dioxide (SO_2) and sulfur trioxide (SO_3)
SSI	size selective inlet
TB	tracheobronchial, referring to the conducting airways from the trachea through the bronchioles
TEOM	tapered element oscillating microbalance
Transmissometer	an instrument to measure light extinction in air
TSP	total suspended particles, a measure of airborne particles of all sizes
Ultrafine Particles	particles with an aerodynamic diameter less than 0.1 micrometer (100 nanometers)
Visual Range (V_r)	the greatest distance at which a black target can be distinguished from the background sky around the majority of the horizon circle
vscc	very sharp cut cyclone
U.S. EPA	United States Environmental Protection Agency
µg	microgram, or one millionth of a gram
$\mu g/m^3$	micrograms per cubic meter
µm	micrometer (micron), or one millionth of a meter

1. Executive Summary

In this report, the staff of the Air Resources Board proposes amendments to the state Ambient Air Quality Standards (AAQS) for particulate matter. The potential health impacts from exposure to particulate matter (PM) air pollution are significant. Health effects associated with PM exposure include: premature mortality, increased hospital admissions for cardiopulmonary causes, acute and chronic bronchitis, asthma attacks and emergency room visits, respiratory symptoms, and days with some restriction in activity. These adverse health effects have been reported primarily in infants, children, the elderly, and those with pre-existing cardiopulmonary disease.

The Children's Environmental Health Protection Act (Senate Bill 25, Senator Martha Escutia; Stats. 1999, Ch. 731, Sec. 3) requires the Air Resources Board (ARB or Board), in consultation with the Office of Environmental Health Hazard Assessment (OEHHA), to "review all existing health-based ambient air quality standards to determine whether, based on public health, scientific literature, and exposure pattern data, these standards adequately protect the health of the public, including infants and children, with an adequate margin of safety" (Health & Safety Code section 39606(d)(1)). In December 2000, as a result of that requirement, the ARB approved a joint ARB/OEHHA staff report (ARB and OEHHA, 2000) that contained preliminary reviews of all of the health-based California ambient air quality standards. These reviews were not exhaustive, but were narrowly targeted to two purposes: (1) to determine whether the existing ambient air quality standards adequately protect the health of the public, including infants and children, with an adequate margin of safety; and (2) to prioritize for full review those standards determined not to adequately protect public health (Health & Safety Code section 39606(d)(1) and (2)).

The staff recommended, and the Board concurred, that among several standards deemed possibly inadequate, the existing standards for particulate matter less than 10 micrometers in aerodynamic diameter (PM10) should be the first to undergo full review. This recommendation was based on the assessment that almost everyone in California is exposed to levels at or above the current State PM10 standards during some parts of the year, and that the statewide potential for significant health impacts associated with PM exposure was determined to be large and wide-ranging. Finally, the staff recommended, and the Board concurred, that the standard for sulfates be reviewed concurrently with the PM10 standards since sulfates are a component of particulate matter.

This report presents the findings and recommendations of a joint ARB/OEHHA review of the health and scientific literature on PM and sulfates, as well as exposure pattern data for PM and sulfates in California. The proposed amendments to the AAQS for particulate matter are based on a health effects review and recommendations from OEHHA. The scientific review suggests the need for separate standards for PM2.5 (particulate matter less than 2.5 micrometers in aerodynamic diameter) in addition to revising the standards for PM10 to make them more health protective. The review also concluded that the standard for sulfates should be retained.

In accordance with Health & Safety Code section 57004, the proposed amendments were peer reviewed by the Air Quality Advisory Committee (AQAC), an external scientific peer review committee, comprised of world-class scientists in the PM field and appointed by the Office of the President of the University of California.

As part of the review process, a joint ARB/OEHHA staff report entitled "Review of the California Ambient Air Quality Standards for Particulate Matter and Sulfates" was submitted to the AQAC for their review. This report, containing recommendations for revising the PM

standards, was released to the AQAC and the public on November 30, 2001. Public workshops to receive community input on the proposal to review the standards were held during December 2001 in Sacramento, Oakland, Bakersfield, El Monte, Mira Loma, and Huntington Park.

The AQAC met on January 23 and 24, 2002, to review the scientific basis of the recommendations and comments received from the public. The AQAC's major findings were that the recommendations for amending the PM standards in the November 30, 2001 report were based upon sound scientific knowledge, methods, and practices and supported by the scientific literature. However, the AQAC did not concur with the lack of a recommendation for a 24-hour standard for PM2.5. The AQAC concluded that there was adequate information in the scientific literature and in the studies reviewed in the November 30, 2001 report to support a 24-hour standard for PM2.5. The AQAC requested staff to develop a proposal to establish a 24-hour PM2.5 standard and to incorporate it into the overall staff recommendation. In response, staff from ARB and OEHHA developed a proposal entitled "Draft Proposal to Establish a 24-hour Standard for PM2.5, Report to the Air Quality Advisory Committee." This draft proposal and associated public comments were reviewed and approved by the AQAC at its meeting on April 3, 2002. Following that AQAC meeting, the staff report was revised to incorporate the proposal to establish a 24-hour PM2.5 standard along with written and oral comments received from the AQAC and the public.

Proposed Amendments to the Ambient Air Quality Standards for Particulate Matter:

The proposed amendments to the standards are largely based on results from epidemiological studies in hundreds of cities. These studies indicate strong associations between both long- and short-term exposure to PM and a variety of adverse health effects, as described above. California ambient air quality standards have four elements (see Health and Safety Code section 39014, and title 17, California Code of Regulations, sections 70100 and 70200): (1) definition of the air pollutant, (2) an averaging time, (3) a pollutant concentration, and (4) a monitoring method to determine attainment of the standard. Staff's recommendations for amending the ambient air quality standards for PM and sulfates are summarized below.

Pollutant, Concentrations and Averaging Times:

- PM10 Annual-Average Standard – Lower the annual-average standard for PM10 from 30 micrograms per cubic meter ($\mu g/m^3$) to **20 $\mu g/m^3$, not to be exceeded**. Revise the averaging method from an annual geometric mean to an annual arithmetic mean. This recommendation is based on the results of numerous epidemiological studies which have found associations between long-term PM10 exposure and adverse health effects, such as mortality and morbidity from cardiopulmonary causes.
- PM10 24-hour-Average Standard – Retain the 24-hour-average standard for PM10 at **50 $\mu g/m^3$, not to be exceeded.**
- PM2.5 Annual-Average Standard – Establish a new annual-average standard for PM2.5 at **12 $\mu g/m^3$, not to be exceeded**. Establish the new PM2.5 standard as an annual arithmetic mean. This recommendation is based on a growing body of epidemiological and toxicological studies showing significant toxicity (resulting in mortality and morbidity) related to exposure to fine particles.
- PM2.5 24-hour-Average Standard – Establish a new 24-hour-average standard for PM2.5 at **25 $\mu g/m^3$, not to be exceeded**. This recommendation is based on epidemiological

studies showing associations between ambient PM2.5 levels and mortality and morbidity resulting from cardiopulmonary causes.

- Sulfates 24-hour-Average Standard – Retain the 24-hour-average standard for sulfates at **25 µg/m^3**.

Monitoring Methods, Samplers, and Instruments:

- PM10 Monitoring Method – Adopt the Federal Reference Method (FRM) for PM10 as the method for California.
- PM2.5 Monitoring Method – Adopt the Federal Reference Method (FRM) for PM2.5 as the method for California.
- Continuous PM Samplers – Adopt those continuous PM samplers which have been found to be suitable for determining compliance with the state PM10 and PM2.5 AAQS, and designate them as California approved samplers (CAS).
- Sulfates Monitoring Method – Revise the sulfates monitoring method by deleting the current total suspended particle (TSP) sulfates method, ARB method MLD 033, and replacing it with the existing ARB method for PM10 sulfates, ARB method MLD 007.

Health Benefits:

The health benefits from attaining the proposed standards are substantial. For example, a quantitative risk assessment estimated that attainment of the proposed annual PM10 standard from current ambient levels would result in a reduction of approximately 6,500 cases (3,200 – 9,800 for a 95 percent confidence interval (95% CI)) of premature mortality per year. This estimate is based on the assumption that mortality is primarily associated with exposure to PM2.5 rather than with the coarse PM fraction. Estimated annual reductions in hospitalizations related to attaining the proposed PM10 standards are 1,200 (66-2,300, 95% CI) for chronic obstructive pulmonary disease, 1,700 (760-2,600, 95% CI) for pneumonia, 3,100 (2,500-3,600, 95% CI) for cardiovascular causes, and 960 (400-1,500, 95% CI) for asthma. Among children ages 7 to 14, attainment of the PM10 standard is estimated to result in about 389,000 (161,000 –573,000, 95% CI) fewer days of lower respiratory symptoms per year. Of these, approximately half of the days of lower respiratory symptoms may be associated with attainment of the proposed PM2.5 standard.

Other Recommendations:

- Staff recommends that the standards for PM and sulfates be revisited within five years, to evaluate new evidence regarding the health effects associated with averaging time, particle size, chemistry, and concentration.
- Staff also recommends that further scientific information be gathered and research be conducted into the health effects of short-term exposures to PM, especially effects from less than 24-hour exposures. This information should be considered when staff revisits the PM standards to determine if AAQS with averaging times of less than 24 hours would be appropriate.

Environmental and Economic Impacts:

The proposed ambient air quality standards will in and of themselves have no environmental or economic impacts. Standards simply define acceptable air quality. Local air pollution control or air quality management districts (Districts) are responsible for the adoption of rules and regulations to control emissions from stationary sources, while the Board is responsible for controls related to mobile sources. A number of different control measures are possible,

and each will have its own environmental and economic impacts. These impacts will be evaluated when specific control measures are proposed by the ARB or the Districts.

Environmental Justice Concerns:

State law defines environmental justice as the fair treatment of people of all races, cultures, and incomes with respect to the development, adoption, implementation, and enforcement of environmental laws, regulations, and policies. Ambient air quality standards define clean air, therefore, all of California's communities will benefit from the proposed health-based standards.

Comment Period and Board Hearing:

Release of this staff report opens the official 45-day comment period required by the Administrative Procedure Act. Please direct all comments to either the following postal or electronic mail address:

Clerk of the Board
Air Resources Board
1001 "I" Street, 23rd Floor
Sacramento, California 95814
aaqspm@listserv.arb.ca.gov

To be considered by the Board, written submissions not physically submitted at the hearing must be received at the ARB no later than 12:00 noon, June 19, 2002.

Public workshops are scheduled for June 2002 to present the recommendations and receive public input on the Report. Information on these workshops, as well as summaries of the presentations from past workshops and meetings are available by calling (916) 445-0753 or at the following ARB website: www.arb.ca.gov/research/aaqs/std-rs/std-rs.htm.

The final recommendations for revising the PM and sulfate standards will be presented to the Board at a public hearing scheduled for June 20, 2002.

The staff recommends that the Board adopt the proposed amendments to the Ambient Air Quality Standards for Particulate Matter and Sulfates. The proposed amendments and their basis are described in detail in this staff report.

1.1 References

Air Resources Board and Office of Environmental Health Hazard Assessment (2000). Adequacy of California Ambient Air Quality Standards: Children's Environmental Health Protection Act. Staff Report. Sacramento, CA. Available at http://www.arb.ca.gov/ch/ceh/airstandards.htm.

2. Introduction and Overview

Particulate matter (PM) is a complex mixture of suspended particles and aerosols composed of small droplets of liquid, dry solid fragments, and solid cores with liquid coatings. Particles vary widely in size, shape and chemical composition, and may contain inorganic ions, metallic compounds, elemental carbon, organic compounds, and compounds from the earth's crust. PM may be either directly emitted into the atmosphere (primary particles) or formed there by chemical reactions of gases (secondary particles) from natural or man-made (anthropogenic) sources such as SO_2, NO_X, and certain organic compounds. PM is a public health concern because it can be inhaled into the upper airways and lungs, with the amount inhaled directly related to size and shape. Detailed discussions on exposure and associated adverse human health effects are presented in Sections 6 and 7, respectively.

To protect public health, the Air Resources Board (ARB or Board) previously adopted three ambient air quality standards for particulate matter: an annual-average standard for particulate matter less than 10 micrometers in diameter (PM10), a PM10 24-hour-average standard, and a sulfates 24-hour-average standard. This report presents the findings and recommendations of a joint review by the ARB and the Office of Environmental Health Hazard Assessment (OEHHA) of the health and scientific literature on PM and sulfates, as well as exposure pattern data for PM and sulfates in California. Based on the results of that review, staff proposes amendments to the PM standards to ensure that they continue to adequately protect public health. The proposed amendments to the PM standards are based on recommendations from OEHHA. The scientific review suggests the need for separate annual and 24 hour standards for PM2.5 (particulate matter 2.5 micrometers or less in aerodynamic diameter) in addition to revising the annual standard for PM10 to ensurepublic health protection. The review concludes that the standard for sulfates should be retained, although staff recommends a change in the monitoring method to expand monitoring capabilities in the State.

2.1 Setting California Ambient Air Quality Standards

Section 39606(a)(2) of the Health and Safety Code authorizes the ARB to adopt standards for ambient air quality "in consideration of public health, safety, and welfare, including, but not limited to, health, illness, irritation to the senses, aesthetic value, interference with visibility, and effects on the economy".

Ambient air quality standards (AAQS) represent the legal definition of clean air. They specify concentrations and durations of exposure to air pollutants that reflect the relationships between the intensity and composition of air pollution and undesirable effects (Health and Safety Code section 39014). The objective of an AAQS is to provide a basis for preventing or abating adverse health or welfare effects of air pollution (title 17, California Code of Regulations, section 70101).

Ambient air quality standards should not be interpreted as permitting, encouraging, or condoning degradation of present air quality that is superior to that stipulated in the standards. Rather, standards represent the minimum acceptable air quality. An AAQS adopted by the Board is implemented, achieved, and maintained by the adoption and implementation of control measures through rules and regulations that are separate from the standard itself. These rules and regulations are primarily, though not exclusively, emissions limitations that apply to specific source categories of pollutants established by the regional and local air pollution control and air quality management districts for stationary sources, and

by the Board for vehicular sources (see generally, Health and Safety Code sections 39002, 40000, and 40001).

California law specifies that standards be health based, although welfare effects are also considered. Health-based standards are predicated on a review of health science literature, and are to be based on the recommendation of OEHHA (Health and Safety Code section 39606(a)(2)). Standards are set to ensure that sensitive population sub-groups are protected from exposure to levels of pollutants that may cause adverse health effects. In addition, OEHHA is to assess the following considerations for infants and children in its recommendation (Health and Safety Code section 39606(b)):

- Exposure patterns among infants and children that are likely to result in disproportionately high exposure to ambient air pollutants in comparison to the general population.
- Special susceptibility of infants and children to ambient air pollutants in comparison to the general population.
- The effects on infants and children of exposure to ambient air pollutants and other substances that have a common mechanism of toxicity.
- The interaction of multiple air pollutants on infants and children, including the interaction between criteria air pollutants and toxic air contaminants. OEHHA's assessment of these considerations is to follow current principles, practices, and methods used by public health professionals.

In accordance with Health & Safety Code section 57004, the proposed amendments were peer reviewed by the Air Quality Advisory Committee (AQAC), an external scientific peer review committee, comprised of world-class scientists in the PM field and appointed by the Office of the President of the University of California. Under Health and Safety Code section 57004(d)(2), the committee prepares a written evaluation of the staff report that describes the scientific basis of the proposed ambient air quality standard. A description of the AQAC review of the proposed standards for particulate matter and sulfates follows later in this chapter. The findings of the Air Quality Advisory Committee can be found in Appendix 2.

2.2 Current California Ambient Air Quality Standards for Particulate Matter and Sulfates

2.2.1 Particulate Matter, 24-hour and Annual Averages

The current California ambient air quality standards for PM10 are 50 micrograms per cubic meter ($\mu g/m^3$) for a 24-hour average and 30 $\mu g/m^3$ for an annual geometric mean. Both values are not to be exceeded. Both standards were adopted by the ARB in 1982 (ARB 1982). They were based on recommendations from the Department of Health Services (at the time, the Department of Health Services fulfilled the role in ambient air quality standard setting now assigned to the OEHHA). The standards were based on studies indicating a significant association between particulate pollution and excess mortality, increased symptoms of respiratory disease in persons with chronic bronchitis and asthma, respiratory functional impairment, and increases in respiratory illness among school children. Evidence from short-term exposure studies indicated that effects were evident at concentrations as low as 70 $\mu g/m^3$ total suspended particulate (TSP) and at 60 $\mu g/m^3$ British smoke. These concentrations are equivalent to PM10 concentrations of approximately 41 to 60 $\mu g/m^3$, respectively. The Department recommended a 24-hour standard of 50 $\mu g/m^3$, which was approximately the mid-point of the range of values noted above. It was also essentially neither

a relaxation nor tightening of the previous 24-hour TSP standard when converted to an equivalent PM10 concentration.

The range of values at which long-term effects (effects on pulmonary function and increased respiratory illness) were observed was approximately 50 to 177 $\mu g/m^3$ when TSP was converted to PM10. Another chronic health effect of concern was cancer. The epidemiological studies reviewed did not establish a relationship between cancer and community air pollution, although known carcinogens were recognized in community air at that time. The Department of Health Services concluded that a particle standard should not only protect the public against pulmonary function health effects, but also to some degree serve as a surrogate measure for protection against cancer. Until more substantial evidence concerning cancer was available, the Department of Health Services believed that the long-term standard should not be a relaxation of the TSP standard. An annual geometric mean of 30 $\mu g/m^3$ (10 μm diameter) was approximately equivalent to the former annual TSP standard when corrected to PM10.

2.2.2 Sulfates, 24-hour Average

The current California ambient air quality standard for sulfates was established in 1976 at 25 $\mu g/m^3$ as a 24-hour average (ARB, 1976). The need for a sulfates standard was based on concern that a natural gas shortage would lead to greater use of fuel oil containing higher levels of sulfur, which would result in increases in ambient sulfate levels, particularly in the South Coast Air Basin. The small body of scientific literature available suggested that the projected concentrations of sulfates posed health risks, further raising concerns.

The 1976 sulfates standard was based on a critical harm value methodology so that public health could be protected, even though there was insufficient information available at the time to set a standard according to the usual threshold model. The concentration selected, 25 $\mu g/m^3$, was the midpoint between an upper bound of 33 $\mu g/m^3$ based on analysis of industrial exposures, and a lower bound of 10 $\mu g/m^3$ derived from the few epidemiological studies available. The midpoint of the range was selected as opposed to the lower bound because of uncertainties in the epidemiological data related to the adequacy of the statistical models used for the analyses, and whether potential confounding factors had been adequately controlled.

At the time the sulfates standard was promulgated, in 1976, it was known that there were differences in the sulfate concentrations reported from collocated samplers that used different methods of collection and analysis (ARB, 1976). The Board decided the use of glass filters to collect 24-hour high-volume total suspended particle samples was the most practical method to use. They were also unable to identify a suitable size-segregating collection device.

In 1977, the ARB conducted a subsequent review of the sulfate standard and monitoring methodology (ARB 1977). The review indicated that the variability of sulfate data between different types of glass-fiber filters may be due in part to a sulfate artifact which ranged from 1 to 8 $\mu g/m^3$, depending on which filter types were used. After the review in 1977, and because other methodologies based on respirable particles (e.g. PM10) were not yet developed, no changes were recommended to the monitoring methods and the level of the standard was also reaffirmed (ARB, 1977).It should be noted that the uncertainty of the exposure estimates does not impact the sulfate standard. The 1976 standard recommendation, affirmed by the 1977 review, was neither directly based on industrial health nor epidemiologic studies. Rather, since the standard was based on a critical harm level methodology, the uncertainties in the monitoring data did not enter into selection of the concentration for the standard.

2.3 Review of the California Ambient Air Quality Standards

2.3.1 Review Schedule

The Children's Environmental Health Protection Act (Senate Bill 25, Escutia, Stats. 1999, Ch. 731 section B; Health and Safety Code section 39606) required the Board, in consultation with the OEHHA, to evaluate all health-based standards by December 31, 2000, to determine whether the standards were adequately protective of the health of the public, including infants and children (Health and Safety Code section 39606(d) and (e)). Standards deemed possibly not protective were prioritized for full review. If the standard is found during the full review to be inadequate, the standard will be revised. The Act requires that the highest priority standard be reviewed and, if necessary, revised no later than December 31, 2002. Additional standards where health protection, particularly for infants and children, may not be sufficient are to be reviewed, and revised as necessary, at the rate of at least one standard per year (Health and Safety Code section 39606(d)(2)). Regulations also require the review of standards whenever substantial new information becomes adopted by the ARB pertaining to ambient air quality standards available, and at least once every five years (title 17, California Code of Regulations, section 70101).

In the report on the adequacy of the standards (ARB and OEHHA, 2000), the Board found that health effects may occur in infants, children, and other groups of the population exposed to several pollutants at or near levels corresponding to current standards. The standard with the highest priority for review is PM10 including sulfates. Other standards with a high priority for review include ozone and nitrogen dioxide. Standards with a lower priority for review are carbon monoxide, sulfur dioxide, hydrogen sulfide, and lead.

After extensive review of the scientific literature, ARB and OEHHA staff developed the staff report titled “Review of the California Ambient Air Quality Standards for Particulate Matter and Sulfates, Report to the Air Quality Advisory Committee” (ARB, 2001a). This report, which was released November 30, 2001, contained the proposed PM and sulfate standards. As described in the following section, the public was afforded an opportunity to comment on and participate in the standard setting process.

2.3.2 Public Outreach

Public outreach for the standard review involved dissemination of information through various outlets to include the public in the regulatory process. In an ongoing effort to include the public in the review of the PM standards, the ARB and OEHHA integrated outreach into public meetings, workshop presentations, electronic “list serv” notification systems, and various web pages. Notification of release of the staff report, the schedule for public meetings and workshops, and invitations to submit comments on the staff report were made through the “list serv” notification system. The notices gave information on where, when and how materials relating to the PM and sulfates standards reviews was available, and how interested persons could participate in the standards review process. Public workshops on the proposed PM and sulfates standards were held in December 2001 in Oakland, Sacramento, Bakersfield, Mira Loma, El Monte, and Huntington Park. Additional public workshops on the proposed standards are scheduled for June 2002.

In addition, public meetings of the Air Quality Advisory Committee (AQAC) were held in Berkeley on January 23 and 24, 2002, and in Oakland on April 3, 2002 (described below). The public was invited to submit comments to the committee before and during these meetings.

Individuals or parties interested in signing up for an electronic e-mail “list-serv” notification on the PM standards, as well as any air quality-related issue, may self-enroll at the following

location: www.arb.ca.gov/listserv/aaqs/aaqs.htm. Additional information on the standards review process is also available at the PM standards review schedule website at: www.arb.ca.gov/research/aaqs/std-rs/std-rs.htm.

2.3.3 Air Quality Advisory Committee Review and Public Comments

The Air Quality Advisory Committee, an external scientific peer review committee that was appointed by the President of the University of California, met January 23 and 24, 2002 to review the initial staff report and public comments, and to ensure that the scientific basis of the recommendations for the annual PM10 and PM2.5 standards and the 24-hour PM10 standard are based upon sound scientific knowledge, methods, and practices. Although the AQAC approved the scientific underpinning of the recommendations, finding that the changes proposed for the AAQS were appropriate, the AQAC also concluded that the staff report, which lacked a recommendation for a 24-hour PM2.5 standard, needed to be revised to incorporate such a recommendation. In response, staff from ARB and OEHHA released an update to the staff report titled "Draft Proposal to Establish a 24-hour Standard for PM2.5, Report to the Air Quality Advisory Committee." This proposed recommendation and associated public comments were reviewed and approved by AQAC on April 3, 2002.

Following the April 3rd AQAC meeting, the draft report was revised to reflect comments received from AQAC and to address comments made by the public. These comments, both written and oral, have been summarized, responded to and incorporated when appropriate into this Staff Report. A summary of the comments, and ARB/OEHHA responses is provided in Appendices 2 and 3. The comments ranged in scope and detail, and included procedural issues related to the standards-setting process, editorial issues, and requests that a particular reference be included. Other concerns related to control issues, natural PM background, the statistical form of the standards, and attainment designations. Another group of questions addressed the epidemiological models used, and the interpretation and application of the scientific literature. Each comment was considered in the process of revision of the draft report, and a response to the comment has been prepared (see Appendices 2 and 3). The comments were accommodated in the revised draft report in various ways, including correction of errors, expanded discussion, clarification of explanation, consideration and inclusion of additional material, and addition of references, as described in the responses to the public comments. Comments that staff disagreed with or which addressed issues that were not part of the standards or the standard setting process were not incorporated into the report. In these cases, an explanation for not incorporating the comment is provided in the responses to comments (Appendices 2 and 3).

2.4 Recommendations

The proposed amendments to the standards are largely based on results from epidemiological studies in hundreds of cities. These studies indicate strong associations between both long- and short-term exposure to PM and a variety of adverse health effects, as described above. California Ambient Air Quality Standards have four elements (Health and Safety Code section 39014, and title 17, California Code of Regulations, Article 2, section 70200): (1) definition of the air pollutant, (2) an averaging time, (3) a pollutant concentration, and (4) a monitoring method to determine attainment of the standard. A summary of staff's proposed recommendations for amending the PM and sulfates standards is listed below.

2.4.1 Pollutant, Concentrations and Averaging Times

- PM10 Annual-Average Standard – Lower the annual-average standard for PM10 from 30 micrograms per cubic meter (µg/m^3) to **20 µg/m^3, not to be exceeded**. Revise the averaging method from an annual geometric mean to an annual arithmetic mean.

This recommendation is based on the results of numerous epidemiological studies of mortality and morbidity, which have found associations between adverse health effects and PM10 when the long-term (i.e., months to years) study mean concentrations are at or below the current annual average standard of 30 $\mu g/m^3$. The recommendation is primarily based on the Harvard Six-Cities data (Dockery et al., 1993) and the American Cancer Society (ACS) study (Pope et al., 1995), both reanalyzed by Krewski et al. (2000). Other investigations, including the Children's Health Study (McConnell et al., 1999) and the Harvard Six-Cities Study (Dockery et al., 1989), have also reported associations between long-term PM exposures and morbidity outcomes, including bronchitis, exacerbation of asthma, and reductions in lung function. In these studies, the long-term (one- or multi-year) mean PM10 concentrations ranged from about 21 to 35 $\mu g/m^3$.

- PM10 24-hour-Average Standard – Retain the 24-hour-average standard for PM10 at **50 $\mu g/m^3$, not to be exceeded.**

  Staff recommends that the 24-hr standard for PM10 at 50 $\mu g/m^3$, not to be exceeded, be retained. The recommendation is based on time series studies of daily mortality and morbidity.

- PM2.5 Annual-Average Standard – Establish a new annual-average standard for PM2.5 at **12 $\mu g/m^3$, not to be exceeded**. Establish the new PM2.5 standard as an annual arithmetic mean.

  This recommendation is based on a growing body of epidemiological and toxicological studies showing significant toxicity related to exposure to fine particles. The ACS and Harvard Six-Cities long-term exposure studies (Dockery et al., 1993; Pope et al., 1995; Krewski et al., 2000) reported robust associations between long-term exposure to PM2.5 and mortality. The mean PM2.5 concentrations for all the cities studied were 18 and 20 $\mu g/m^3$ in the Six-Cities and the ACS studies, respectively. In the ACS study, the relative risks are similar in cities at the lowest long-term PM2.5 concentrations of 11 and 12.5 $\mu g/m^3$. Larger increases in risk do not occur until the long-term PM2.5 mean equals 14.9 $\mu g/m^3$. Therefore, an annual standard of 12 $\mu g/m^3$ would be below the mean of the most likely effects level and would provide a margin of safety. Additional evidence comes from other epidemiological studies that examined the relationships between multiple daily exposures of PM2.5 and adverse health outcomes. These studies have long-term (three- to four-year) means in the range of 13 to 18 $\mu g/m^3$.

- PM2.5 24-hour-Average Standard – Establish a new 24-hour-average standard for PM2.5 at **25 $\mu g/m^3$, not to be exceeded**.

  This recommendation is based on studies showing associations between ambient PM2.5 levels and mortality and morbidity when the 98th percentile of the study PM2.5 concentration ranged between 28 and 55 $\mu g/m^3$. The methodology used to derive the standard is based on setting the level of the standard at a concentration below the 98th percentile observed in studies consistently associated with adverse health effects. The underlying principle is to reduce not only the mean concentration (represented by the annual average), but specifically the upper tail of the distribution, described by the 98th percentile of the distributions of published studies. For this standard staff has relied primarily on studies relating fine particle concentrations with daily mortality, the most serious irreversible health impact. Ultimately, additional protection will be provided by expressing the standard in a "not to be exceeded" form.

- Sulfates 24-hour-Average Standard – Retain the 24-hour-average standard for sulfates at **25 µg/m^3**.

  Exposure to ambient sulfates has been associated with mortality and the same range of morbidity effects as PM10 and PM2.5, although the associations have not been as consistent as with PM10 and PM2.5. These effects have been particularly noted in areas rich in strongly acidic sulfates, such as the eastern United States and Canada. In contrast, controlled exposure studies involving high levels (up to 1,000 µg/m^3) of strongly acidic sulfates have demonstrated little, if any, effect on volunteer subjects, (e.g., Aris et al., 1991). Furthermore, in California, acidic sulfates (principally sulfuric acid and ammonium sulfate) constitute a small fraction of the PM mass relative to the areas in which sulfates have been found to be associated with adverse health impacts. Also, sulfate concentrations in California have been far lower during the past few years than the level of the existing standard. In view of the mixed evidence on sulfates and health in California, the low likelihood of health risks in relation to ongoing reduction trends in sulfate emissions and ambient levels, staff recommends that the current standard be retained until the next review of the PM standard, if not earlier. However, staff is making recommendations to change the monitoring method for sulfates.

2.4.2 Monitoring Methods, Samplers, and Instruments

- PM10 Monitoring Method – Adopt the Federal Reference Method (FRM) for PM10 as the method for California. This proposal allows for alignment of the State method for PM monitoring with all federal high-volume and low-volume samplers, and thereby will eliminate confusion of having two methods (State and federal) for the same parameter.

- PM2.5 Monitoring Method – Adopt the Federal Reference Method (FRM) for PM2.5 as the method for California. This proposal allows for alignment of the State method for PM monitoring with all federal high-volume and low-volume samplers, and thereby will eliminate confusion of having two methods (State and federal) for the same parameter.

- Continuous PM Samplers – Adopt those continuous PM samplers which have been found to be suitable for determining compliance with the state PM10 and PM2.5 AAQS, and designate them as California approved samplers (CAS). This proposal allows for the use of continuous PM sampler technology. Continuous monitoring for either PM10 or PM2.5 has many advantages over traditional filter based sampling techniques. A continuous method is an in-situ, automatic measurement method of suspended particle mass with varied averaging time (minutes to hours) that provides an instantaneous result. Their 24 hour/day, 7day/week sampling schedule will further our understanding of PM emission patterns and exposure, and can be used to enhance public health research into short-term peak exposure. They can provide more data for model validation, to aid in identifying air pollution source(s), and to reflect dispersion patterns. Official approval of continuous instruments/methods will promote further development of continuous samplers and potentially reduce the cost of the air monitoring network.

- Sulfates Monitoring Method – Revise the sulfates monitoring method by deleting the current total suspended particle (TSP) sulfates method, ARB method MLD 033, and replacing it with the existing ARB method for PM10 sulfates, MLD 007. This proposal allows the ARB to use its existing PM10 network to greatly expand its monitoring network capabilities for sulfates. By doing so, the ARB greatly expands its ability to better understand sulfate air quality in the state. This method changes allows for the minimization of any artifact-forming potential through the use of alkalinity-controlled filters.

The staff also proposes to maintain the regulatory language that permits other samplers deemed to give equivalent results to be approved by the ARB at a subsequent time.

This action is intended to eliminate the ambiguity that currently exists between the acceptable use of samplers for State and federal programs and to respond to the need for continuous samplers to meet a variety of needs. It will also greatly expand the database of information that will be available to decision-makers. Adopting the specific samplers into the regulation will make information about appropriate monitoring methods and samplers accessible, standard and enforceable.

2.4.3 Other Recommendations:

Further, in light of the adverse health effects observed at current ambient concentrations and the lack of a demonstrated threshold, staff makes the following recommendations for Board approval:

- Staff recommends that the standards for PM and sulfates be revisited within five years, to evaluate new evidence regarding the health effects associated with, particle size, chemistry, concentration,and averaging time.
- Staff also recommends that further scientific information be gathered and research be conducted into the health effects of short-term exposures of PM, especially effects from less than 24-hour exposures. This information should be considered when staff revisits the PM standards to determine if AAQS with averaging times of less than 24 hours would be appropriate.

2.5 Health Benefits

Although a precise measure of risk is difficult to determine, staff performed a quantitative risk assessment based on attainment of the recommended annual average standards of 12 $\mu g/m^3$ and 20 $\mu g/m^3$ for PM2.5 and PM10, respectively. The results of this assessment are summarized in Tables 9.4 and 9.6, respectively. The assessment applied concentration – response functions from available epidemiologic studies to California by using California-specific PM, mortality and morbidity data (see Chapter 9 for a full discussion).

The quantitative risk assessment estimated that attainment of the proposed annual PM10 standards would result in a reduction of approximately 6,500 cases of premature mortality per year (3,200 – 9,800, 95 percent confidence interval (CI)). This estimate is based on the assumption that mortality is primarily associated with exposure to PM2.5 rather than with the coarse PM fraction. Estimated mean annual reductions in hospitalizations related to attaining the proposed PM10 standards are 1,200 cases for Chronic Obstructive Pulmonary Disease (COPD) (66 – 2,300, CI), 1,700 cases for pneumonia (760 – 2,600, CI), 3,100 cases for cardiovascular causes (2,500 – 3,600, CI), and 960 cases for asthma (400 – 1,500, CI). Among children ages 7 to 14, attainment of the PM10 standard is estimated to result in about 390,000 fewer days of lower respiratory symptoms per year (160,000 – 570,000, CI). Of these, approximately half of the days of lower respiratory symptoms may be associated with attainment of the proposed PM2.5 standard.

Use of the concentration-response functions from short-term exposure studies, which only capture part of the total effects on mortality, generates an estimate of 1,900 fewer premature deaths per year (2,200 – 3,100, CI) based on attainment of a standard of 12 $\mu g/m^3$ for PM2.5. Attainment of the recommended PM2.5 standards is estimated to result in up to about 11,000 fewer cases of chronic bronchitis among people over age 27. Estimated reductions in hospitalizations are 600 (33 – 1,200, CI) for COPD, 860 (390 – 1,300, CI) for pneumonia, and 470 (86 – 850, CI) for asthma.

In summary, the epidemiologic evidence and risk assessment support the likelihood of significant reductions in mortality and morbidity effects with attainment of the recommended annual and 24-hour PM standards.

2.6 Environmental and Economic Impacts

The proposed ambient air quality standards will in and of themselves have no environmental or economic impacts. Standards simply define clean air. Once adopted, local air pollution control or air quality management districts are responsible for the adoption of rules and regulations to control emissions from stationary sources to assure their achievement and maintenance. The Board is responsible for adoption of emission standards for mobile sources. A number of different control measures are possible, and each will have its own environmental and economic impact. These impacts must be evaluated when any control measure is proposed. Environmental or economic impacts associated with the imposition of future control measures will be considered when specific measures are proposed.

2.7 Environmental Justice

State law defines environmental justice as the fair treatment of people of all races, cultures, and incomes with respect to the development, adoption, implementation, and enforcement of environmental laws, regulations, and policies (Senate Bill 115, Solis; Stats 1999, Ch. 690; Government Code § 65040.12(c)). The Board recently established a framework for incorporating environmental justice into the ARB's programs consistent with the directives of State law (ARB, 2001b). The policies developed apply to all communities in California, but recognize that environmental justice issues have been raised more in the context of low-income and minority communities, which sometimes experience higher exposures to some pollutants as a result of the cumulative impacts of air pollution from multiple mobile, commercial, industrial, areawide, other sources. Because ambient air quality standards simply define clean air, all of California's communities will benefit from the proposed health-based standards, as progress is made to attain the standards. Over the past twenty years, the ARB, local air districts, and federal air pollution control programs have made substantial progress towards improving the air quality in California. However, some communities continue to experience higher exposures than others as a result of the cumulative impacts of air pollution from multiple mobile and stationary sources and thus may suffer a disproportionate level of adverse health effects (see section 7.7.2 of this report). Since the same ambient air quality standards apply to all regions of the State, these communities will benefit by a wider margin and receive a greater degree of health improvement from the revised standards than less affected communities, as progress is made to attain the standards. Moreover, just as all communities would benefit from new, stricter standards, alternatives to the proposed recommendations, such as recommending no change to the PM10 standards, or not proposing standards for PM2.5, would adversely affect all communities. Once ambient air quality standards are adopted, the ARB and the local air districts will propose emission standards and other control measures to reduce emissions from various sources of PM. The environmental justice aspects of each proposed control measure will be evaluated in a public forum at this time.

As additional relevant scientific evidence becomes available, the PM standards will be reviewed again to make certain that the health of the public is protected with an adequate margin of safety. To ensure that everyone has an opportunity to stay informed and participate fully in the development of the PM standards, ARB and OEHHA staff have held (and will continue to conduct) workshops in a number of communities across the State and have distributed information by mail and through the internet, as described in section 2.3.2 in this chapter.

2.8 Research Needs

Available evidence indicates that significant adverse health effects may occur among both children and adults when ambient PM concentrations exceed current State standards or become elevated above those proposed in this report. The foundation for revising California PM standards is based primarily on numerous epidemiological studies conducted throughout the world which yielded remarkably consistent results, despite local differences in PM sources and types of co-pollutants. Although this consistency was sufficient to guide staff in proposing new, more stringent standards, several data gaps were identified during the preparation of this document. Moreover, many questions about the mechanisms by which particles adversely affect health remained unanswered. Results from research designed to address these questions would refine knowledge and reduce uncertainties in various aspects of the PM literature and should be ongoing at the State, federal, and international level. Specific areas of research that would assist the Board with subsequent revisions of the standards include:

- health impacts of short-term exposures to PM and sulfates
- health impacts of long-term exposures to PM and sulfates
- health impacts of ultrafine PM
- relationship between community and individual exposures to PM
- factors contributing to sensitivity in individuals and groups
- health effects of PM related to physical properties and/or chemical constituents
- physiological mechanisms of PM and sulfates effects
- how PM interacts with other air pollutants to harm health
- health impacts of PM at low concentrations
- role of PM in causing new disease
- impacts of PM and sulfates on children including neonates
- environmental justice and its relationship to PM health effects

Development and application of improved study methodologies will require research in several areas, including improvements in air monitoring and exposure assessment methodologies. As ambient air monitoring for PM expands to include time-resolved data reporting, it would be useful to incorporate this new data into community health investigations. Further, studies are needed to determine how community and indoor levels of PM relate to actual human exposures.

Development and application of improved statistical methodologies, particularly for epidemiological studies, are needed to improve the analytical tools available to health investigators as they evaluate the health impacts of daily or multi-day observations collected over prolonged study periods. Improved identification of and control for potentially confounding factors in epidemiological studies are critically needed.

Review of the health effects literature undertaken for this document presented staff with a major challenge in determining safe levels of PM for short- or longer-term exposure. The epidemiological studies reviewed reported adverse effects even at the lowest levels of ambient PM present. The statistical methods available, as well as the sources and types of air quality and health data available for use in these studies, impose substantial limitations to identifying truly safe levels of these pollutants.

Crucial to answering the questions outlined above is an improved physical characterization (particle shape and aerodynamic diameter) and chemical speciation of PM and sulfate samples which will allow identification of the toxic components of the ambient mixture. Physical and chemical characterization data for sulfates and PM will likely become increasingly important in designing hypothesis-driven animal and controlled human exposure studies. Comparisons of the toxicity of different sized particles of the same chemical species are also needed (ultrafine vs. fine vs. coarse).

More information is needed to identify the physiological, genetic, medical and other factors that contribute to susceptibility to PM and sulfates health effects. Age appears to be one factor in susceptibility to adverse effects resulting from exposure to PM and sulfates. Studies on children and neonates are critically needed. Subjects at risk of PM and sulfates-induced health effects need to be incorporated into research on the health impacts of these pollutants. Hypothesis-driven animal toxicological experimental studies, as well as human clinical studies, offer especially valuable opportunities to investigate issues that are related to biological sensitivity. This information will be very useful in optimizing research protocols and refining subject selection criteria so that future research targets the most significant endpoints and most at-risk subpopulations.

2.9 References

Air Resources Board (1976). Regulations Concerning a 24-hour Sulfate Ambient Air Quality Standard or Significant Harm Level. Staff Report 76-4-5. Sacramento, CA.

Air Resources Board (1977). Review of the 24-Hour Sulfate Ambient Air Quality Standard. Staff Report 77-20-3. Sacramento, CA.

Air Resources Board (1982). California Ambient Air Quality Standard for Particulate Matter (PM10). Staff Report. Sacramento, CA.

Air Resources Board and Office of Environmental Health Hazard Assessment (2000). Adequacy of California Ambient Air Quality Standards: Children's Environmental Health Protection Act. Staff Report. Sacramento, CA. Available at http://www.arb.ca.gov/ch/ceh/airstandards.htm.

Air Resources Board (2001a). Review of the California Ambient Air Quality Standards for Particulate Matter and Sulfates, Report to the Air Quality Advisory Committee. Staff Report. Sacramento, CA.

Air Resources Board (2001b). Policies and Actions for Environmental Justice, December 13, 2001.

Aris R, Christian D, Sheppard D, Balmes JR (1991). Lack of bronchoconstrictor response to sulfuric acid aerosols and fogs. Am Rev Respir Dis 143(4 Pt 1):744-50.

Dockery DW, Speizer FH, Stram DO, Ware JH, Spengler JD, Ferris BG Jr (1989). Effects of inhalable particles on respiratory health of children. Am Rev Respir Dis 139:587-94.

Dockery DW, Pope CA III, Xu X, Spengler JD, Ware JH, Fay ME *et al*. (1993). An association between air pollution and mortality in six U.S. cities. N Engl J Med 329:1753-9.

Krewski D, Burnett R, Goldberg MS, Koover K, Siemiatycki J, Jerrett M *et al*. (2000). Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Res Rep Health Eff Inst (A special report of the Institute's Particle Epidemiology Reanalysis Project).

McConnell R, Berhane K, Gilliland F, London S, Vora H, Avol E *et al*. (1999). Air pollution and bronchitic symptoms in southern California children with asthma. Environ Health Perspect 107:757-60.

Pope CA III, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE *et al*. (1995). Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Respir Crit Care Med 151:669-74.

3. Physics and Chemistry of Particles

3.1 Introduction

Airborne particulate matter (PM) is not a single pollutant, but rather a mixture of many subclasses of pollutants with each subclass potentially containing many different chemical species. Particles may be either directly emitted into the atmosphere (primary particles) or formed there by chemical reactions of gases (secondary particles) from natural and anthropogenic sources such as SO_2, NO_X, and certain organic compounds. The relative importance of primary and secondary particles generally depends on the geographical location with precursor emissions, atmospheric chemistry, and meteorology all playing a role. Examples of PM include combustion-generated particles, such as those from automobiles or wood burning; photochemically-produced particles, such as those found in urban haze; salt particles formed from sea spray; and soil-like particles from resuspended dust.

In California, the proximity of a location to a variety of sources, in addition to the diurnal and seasonal variations in meteorological conditions, causes the size, composition, and concentration of particulate matter to vary in space and time. PM pollution is the most serious and complex air pollution problem facing both scientific communities and regulatory agencies, and reducing particulate pollution is one of the most difficult environmental challenges facing California because of the great diversity of sources and chemical species involved.

Atmospheric particles contain inorganic ions, metallic compounds, elemental carbon, organic compounds, and crustal compounds. Some atmospheric particles are hygroscopic and contain particle-bound water. The organic fraction is especially complex, containing hundreds of organic compounds. The particle formation process includes nucleation of particles from low-vapor-pressure gases emitted from sources or formed in the atmosphere by chemical reactions; condensation of low vapor pressure gases on existing particles; and coagulation of particles. Thus, any given particle may contain PM from many sources. The composition and behavior of airborne particles are fundamentally linked with those of the surrounding gas. An aerosol may be defined as a suspension of solid or liquid particles in air. The term aerosol includes both the particles and all vapor or gas-phase components of air. However, while this is the rigorous definition of aerosols, the term is often used in the atmospheric chemistry literature to denote just the particles.

A complete description of the atmospheric aerosol would include an accounting of the chemical composition, optical properties, morphology, and size of each particle, and the relative abundance of each particle type as a function of particle size. However, most often the physical and chemical characteristics of particles are measured separately. Size distributions by particle number, from which surface area and volume distributions are calculated, often are determined by physical means, such as electrical mobility or light scattering of suspended particles. Chemical composition usually is determined by analysis of collected samples. The mass and average chemical composition of particles, segregated according to aerodynamic diameter by cyclones or impactors, can also be determined. This chapter provides general information on the physics and chemistry of atmospheric particles that may be useful in reading subsequent sections. For a more extensive review of the physics and chemistry of PM, the reader is referred to Finlayson-Pitts and Pitts (1999), Warneck (1999), and Seinfeld and Pandis (1998).

3.2 Physical Properties

3.2.1 Definition

Particulate matter can exist in the liquid or solid phase and its size can span several orders of magnitude, from a molecular cluster of 0.002 µm in aerodynamic diameter to coarse particles on the order of 100 µm. The lower end of the size range is not sharply defined because there is no accepted criterion at which a cluster of molecules becomes a particle. The upper end corresponds to the size of fine drizzle or very fine sand; these particles are so large that they quickly fall out of the atmosphere and hence do not remain suspended for significant periods of time. The most important particles with respect to atmospheric chemistry and physics are generally in the 0.002 to 10 µm range.

Atmospheric particles are usually referred to as having a radius or diameter, implying they are spherical. However, many particles in the atmosphere have quite irregular shapes for which geometrical radii and diameters are not meaningful. Hence, the size of such irregularly shaped particles is expressed in terms of equivalent diameter that depends on a physical, rather than a geometrical, property. One of the most commonly used term is the aerodynamic diameter, which is defined as the diameter of a sphere of unit density (1 g/cm^3) that has the same terminal falling speed in air as the particle under consideration. The aerodynamic diameter of particles is important because it determines the residence time in the air, and it reflects the various regions of the respiratory system in which particles of different sizes become deposited.

3.2.2 Particle Size Distributions

The atmosphere, whether in urban or remote areas, contains significant concentrations of aerosol particles, sometimes as high as 10^7 to 10^8 particles/cm^3. The aerodynamic diameter of these particles span over four orders of magnitude, from a few nanometers to around 100 µm. Because the size of the atmospheric particles plays such an important role in both their chemistry and physics in the atmosphere, as well as their effects, it is important to know the distribution of particle sizes.

Urban aerosols are mixtures of both primary and secondary particles. The number distribution is dominated by particles smaller than 0.1 µm, while most of the surface area is in the 0.1 to 0.5 µm size range. The aerosol size distribution is quite variable in an urban area. Extremely high concentrations of very fine particles (less than 0.1 µm) are found close to sources such as highways, but their concentrations decrease rapidly with distance from their source. Figure 3.1 (Seinfeld and Pandis, 1998) describes the number of particles as a function of their diameter for rural, urban-influenced rural, urban, and freeway-influenced urban aerosols. There is roughly an order of magnitude more particles close to the freeway compared to the average urban concentration.

An important feature of atmospheric aerosol size distribution is the tri-modal character: (1) nuclei, (2) accumulation, and (3) coarse. As the technology for measuring small particles has improved, ultrafine particles (with diameters less than .01 µm, i.e., <100 nm) have also been increasingly studied. Particles in the atmosphere are now frequently treated in terms of the four modes summarized in Figure 3.2 (Finlayson-Pitts and Pitts 1999). This figure shows the mechanisms such as condensation and coagulation that transfer aerosol mass from one size range to another, and also shows the major sources and removal processes for each one. The number distribution is dominated by particles smaller than 0.1 µm, while most of the surface area is in the 0.1 to 0.5 µm size range. The mass distribution has usually two distinct modes, one in the submicrometer regime (referred to as accumulation mode) and the other in the coarse particle regime.

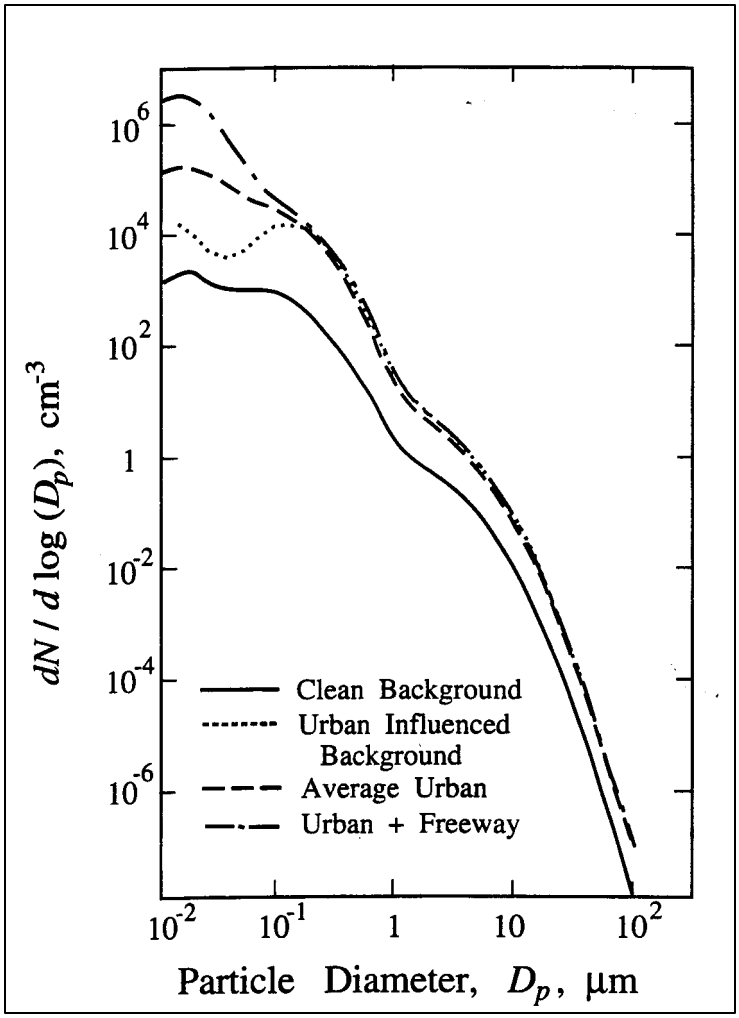


Figure 3.1. Aerosol number distribution for the average urban, for urban influenced by background, and for background (adapted from Seinfeld and Pandis, 1998). Number concentrations are shown on logarithmic scale to display the wide range by site and size, where N is the number concentration and D_p is the mean diameter.

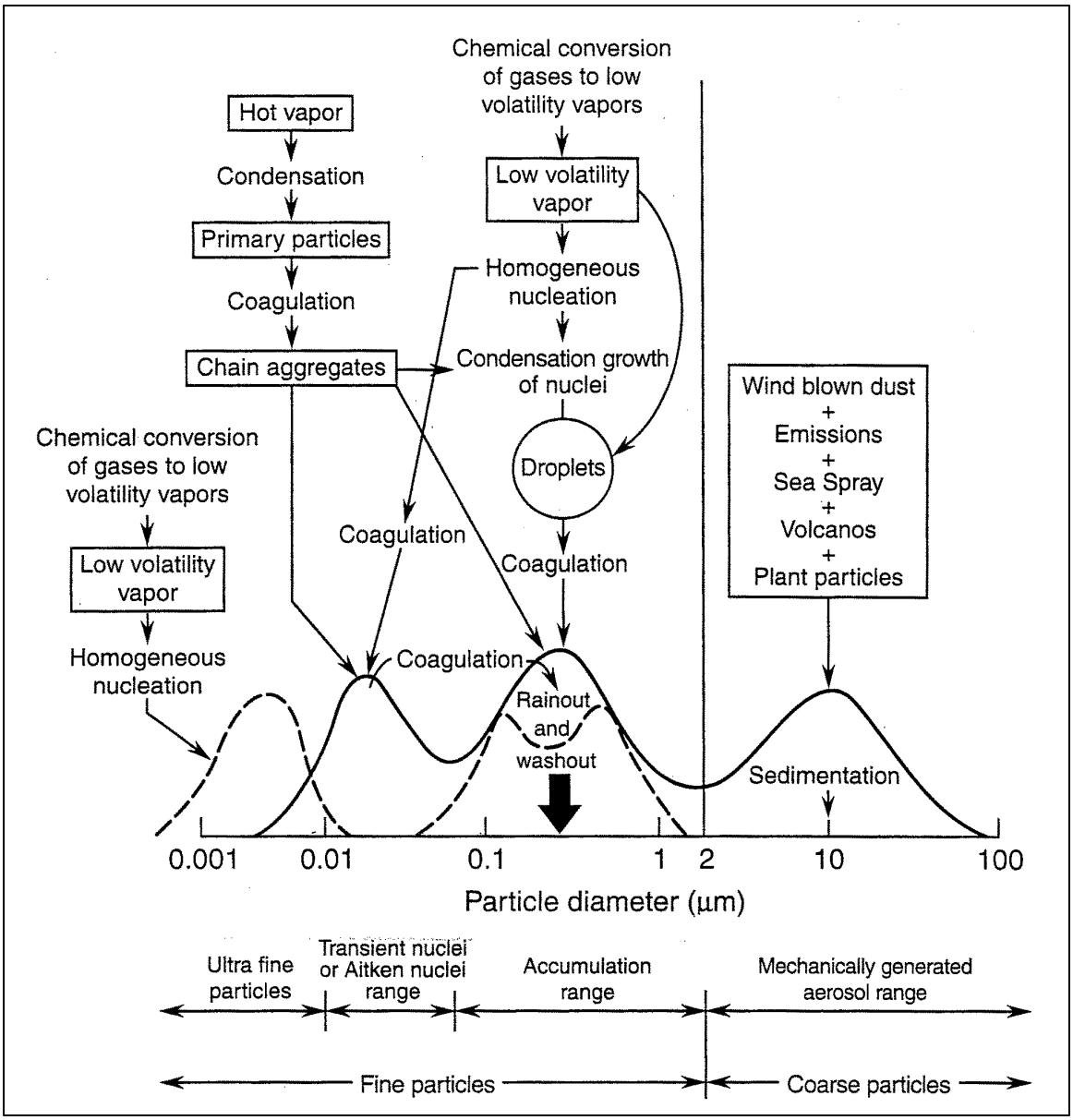


Figure 3.2. Schematic of an atmospheric aerosol size distribution showing four modes (adapted from Finlayson-Pitts and Pitts, 1999).

The nuclei mode, corresponding to particles below about 0.1 μm, may not be noticeable in volume or mass distributions. Nuclei mode particles are the result of nucleation of gas phase species to form condensed phase species with very low equilibrium vapor pressure. As an example, metallic ultrafine particles may be formed from metals in lubricating oil or fuel additives that are vaporized during combustion of gasoline or diesel fuels (Kittelson 1998). Recent smog chamber studies and indoor experiments show that atmospheric oxidation of certain organic compounds found in the atmosphere can produce highly oxidized organic compounds with an equilibrium vapor pressure sufficiently low to result in nucleation (Kamens et al. 1999; Weschler and Shields 1999). Some scientists argue that ultrafine particles pose potential health problems and that some health effects may be more closely associated with particle number or particle surface area than particle mass. Because nuclei-mode particles

contribute the major portion of particle number and a significant portion of particle surface area, further attention to nuclei-mode particles is justified.

The size range from 0.1 to 2.5 µm, is the accumulation mode. Fine particles include both the accumulation and the nuclei modes. Nuclei-mode particles may be removed by dry deposition or by growth into the accumulation mode. This growth takes place as other low vapor pressure material condenses on the particles or as nuclei-mode particles coagulate with themselves or with accumulation mode particles. The coagulation rates for particles in the nuclei range with the larger particles in the accumulation range are usually larger than for self-coagulation of the small particles. This occurs because of the high mobility of the small particles combined with the larger target area of the bigger particles.

Particles in accumulation mode tend to represent only a small fraction of the total particle number, but a significant portion of the aerosol mass. Because they are too small to settle out rapidly, they have much longer lifetimes than coarse particles. This long lifetime, combined with their effects on visibility, cloud formation, and health, makes them of great importance in atmospheric physics and chemistry. Because of the nature of their sources, particles in the accumulation mode generally contain organic compounds as well as soluble inorganic compounds such ammonium nitrate and ammonium sulfate.

The third mode, containing particles larger than 2.5 µm, is known as the coarse particle mode. Coarse particles are usually produced by mechanical processes such as grinding, wind, or erosion. As a result, they are relatively large and hence settle out of the atmosphere by sedimentation in a reasonably short time, except on windy days, where fallout is balanced by reentrainment. Chemically, their composition reflects their source, and hence it is predominantly inorganic such as sand and sea salt, although significant amounts of organic compounds have also been reported associated with them (Boon et al. 1998). Because the sources and sinks are different from those of the smaller modes, the occurrence of particles in this mode tends to be only weakly associated with the fine particle mode. The majority of biological particles, such as spores and pollens, tend to be in the coarse particle range.

While particles in the coarse particle mode are generally sufficiently large that they are removed relatively rapidly by gravitational settling, there are large-scale mechanisms of transport that can carry them long distances during some episodes. The results of several studies indicate the transport of dust in larger particles from the Sahara Desert to the northwestern Mediterranean, Atlantic Ocean, and the United States (Gatz and Prospero 1996). Similarly, dust transported from Asia has been reported on a regular basis over the Pacific (Zhang et al. 1997). Asian dust has been observed during the spring at the Mauna Loa Observatory in Hawaii (Zieman et al. 1995; Holmes et al. 1997). At this location, the elemental signaure (in terms of silica to iron or titanium to iron ratios) in particles in the size range 0.5 to 3.5 µm is very similar to those measured during dust storms in Beijing, consistent with long-range transport of these particles.

The literature includes references to fine, coarse, suspended, respirable, inhalable, thoracic and other adjectives to indicate a size segregation of PM. Uniform criteria are not always employed in the application of these designations. Particles less than 2.5 µm in aerodynamic diameter are generally referred to as “fine” and those greater than 2.5 µm diameters as “coarse”. The selection of PM10 as an indicator was based on health considerations and was intended to focus regulatory concern on those particles small enough to enter the thoracic region. Detailed definitions of the various sizes and their relationships are given in standard aerosol textbooks (e.g., Seinfeld and Pandis 1998, Finlayson-Pitts and Pitts, 1999, Friedlander, 2000).

3.2.3 Particle Formation and Growth

The formation of particles in various size ranges in the atmosphere may occur by a number of mechanisms. These include reaction of gases to form low-vapor-pressure products followed by nucleation to form new particles or condensation on preexisting particles, along with some coagulation between particles. An important parameter in particle nucleation and in particle growth by condensation is the saturation ratio, which is defined as the ratio of the partial pressure of a species to its equilibrium vapor pressure above a flat surface. For either condensation or nucleation to occur, the species vapor pressure must exceed its equilibrium vapor pressure.

Nucleation can occur both in the absence or presence of foreign material (pre-existing particles, such as primary particles emitted by sources). Homogeneous nucleation is the nucleation of vapor on embryos comprised of vapor molecules only, in the absence of foreign substances. Heterogeneous nucleation is the nucleation on a foreign substance or surface, such as an ion or a solid particle. In addition, nucleation processes can be homomolecular (involving a single species) or heteromolecular (involving two or more species). Once the initial nucleation step has occurred, the nuclei of the new phase tend to grow rapidly. Nucleation theory attempts to describe the rate at which the first step in the phase transformation process occurs – the rate at which the initial very small nuclei appear. For a review of nucleation in the atmosphere, the reader is referred to literature on nucleation and atmospheric aerosols (Fukura and Wagner 1992; Seinfeld and Pandis 1998).

Condensation occurs when the vapor concentration of a species exceeds its equilibrium concentration (expressed as its equilibrium vapor pressure). Condensable species can either condense on the surface of existing particles or can form new particles. The relative importance of nucleation versus condensation depends on the rate of formation of the condensable species and on the surface or cross-sectional area of existing particles (McMurry and Friedlander 1979). In ambient urban environments, the available particle surface area is sufficient to rapidly scavenge the newly formed condensable species. Formation of new particles (nuclei mode) is usually not important except near sources of condensable species. The results of several studies report observations of the nuclei mode in traffic (Hildemann et al. 1991; Abdul-Khalek et al. 1998). New particle formation also can be observed in cleaner, remote regions. Bursts of new particle formation in the atmosphere under clean conditions usually occur when aerosol surface area concentrations are low (Covert et al. 1992). High concentrations of nuclei mode particles have been observed in regions with low particle mass concentrations, indicating that new particle formation is inversely related to the available aerosol surface area (Clarke 1992). For more detailed discussions of the quantitative treatment of condensation processes in the atmosphere, the reader is referred to articles by Pandis et al. 1995, and Kerminen and Wexler 1995.

Coagulation refers to the formation of a single particle via collision and adhesion of two smaller particles. Small particles undergo relatively rapid Brownian motion (i.e., constant random movement along an irregular path caused by the bombardment of surrounding air molecules), that leads to sufficient particle-particle collisions to cause such coagulation. Coagulation of smaller particles with much larger ones is similar to condensation of a gas on the larger particles and acts primarily to reduce the number of small particles, adding relatively little to the mass or size of the larger particles. Hence the larger mode will not show significant growth by such a mechanism. The rate of such processes depends on the diameter of the large particle, how rapidly the smaller particle is carried to it (i.e., the diffusion of the smaller particle), and the concentrations of the particles. Self-coagulation, where the particles are approximately the same size, can, however, lead to changes in the size

distribution of the aerosol particles. The rate of this process is a strong function of the particle concentration as well as the particle size (Pandis et al. 1995).

3.2.4 Removal Processes

Once particles are in the atmosphere, their size, number, and chemical composition are changed by several mechanisms until they are ultimately removed by natural processes. Some of the physical and chemical processes that affect the "aging" of atmospheric particles are more effective in one regime of particle size than another. The lifetimes of particles vary with size. Coarse particles can settle rapidly from the atmosphere within hours, and normally travel only short distances. However, when mixed high into the atmosphere, as in dust storms, the smaller-sized coarse-mode particles may have longer lives and travel distances. Nuclei mode particles rapidly grow into the accumulation mode. However, the accumulation mode does not grow into the coarse mode. Accumulation-mode fine particles are kept suspended by normal air motions and have very low deposition rates to surfaces. They can be transported thousands of kilometers and remain in the atmosphere for a number of days.

Atmospheric species removal processes can be grouped into two categories: dry deposition and wet deposition. Dry deposition denotes the direct transfer of species, both gaseous and particulate, to surfaces and proceeds without the aid of precipitation. Wet deposition, on the other hand, encompasses all processes by which airborne species are transferred to surfaces in aqueous form (i.e., rain, snow, or fog). Wet deposition include processes such as dissolution of atmospheric gases in airborne droplets (cloud droplets, rain, or fog), removal of atmospheric particles when they serve as nuclei for the condensation of atmospheric water to form a cloud or fog droplet, and removal of atmospheric particles when the particle collides with a droplet both within and below clouds.

Dry deposition rates are expressed in terms of a deposition velocity that varies with particle size, reaching a minimum between 0.1 and 1.0 µm aerodynamic diameter. The wide ranges of reported dry deposition velocities for any given pollutant reflect a combination of experimental uncertainties as well as real differences due to meteorology, nature of the surface, diurnal variation, etc. The overall uncertainty in the appropriate value of the deposition velocity to use under a given set of circumstance can thus be quite large. A discussion of these issues can be found in articles by Gao and Wesley (1995) and Wesley and Hicks (1999).

Accumulation-mode particles are removed from the atmosphere primarily by cloud processes. Fine particles, especially particles with a hygroscopic component, grow as the relative humidity increases, serve as cloud condensation nuclei, and grow into cloud droplets. If the cloud droplets grow large enough to form rain, the particles are removed in the rain. Falling rain drops impact coarse particles and remove them. Ultrafine or nuclei mode particles are small enough to diffuse to the falling drop, be captured, and removed in rain.

3.2.5 Meteorology and Particles

Meteorological conditions are, generally, the biggest factor influencing the temporal variation in pollutant concentrations. Weather plays a major role in what primary particles are emitted, and to what degree. "Background" aerosol (e.g., sea spray, volcanic dust) concentrations are affected by wind transporting material or by "stirring up" local natural aerosols. Rain suppresses dust from both natural and manmade sources. Seasonal and daily variations in weather influence the production of biogenic pollutants (gases, pollen, etc.). Primary emissions from human activities will be similarly influenced, both directly, as with wind and rain on dust, and indirectly through changes in human activity (e.g., residential wood burning increases in colder weather, and agricultural activity peaks during planting and harvesting).

Secondary particle formation is influenced by a combination of precursor pollutant concentrations and weather conditions. Conversion of SO_X to sulfate aerosols is accelerated by the presence of oxidants and OH radicals in the air (as during ozone episodes) and is accelerated even more under humid conditions when the conversion can occur inside water droplets. NO_X conversion to nitrate is even more sensitive to weather conditions, as formation rates must compete with dissociation back to gases, so that nitrate is generally a cool-wet (e.g., winter) weather phenomenon. Figure 3.3 represents a flowchart of actual linkages between particulate matter air pollution and controlling factors of weather and source activity. Due to the influences of these links, the same emissions can result in high PM concentrations on one occasion, and low concentrations on another. The purpose of detailed analysis is to refine our understanding of how the linkages shown in this chart act on pollutants so that we can accurately determine what portions of the measured concentrations are due to each of the various sources.

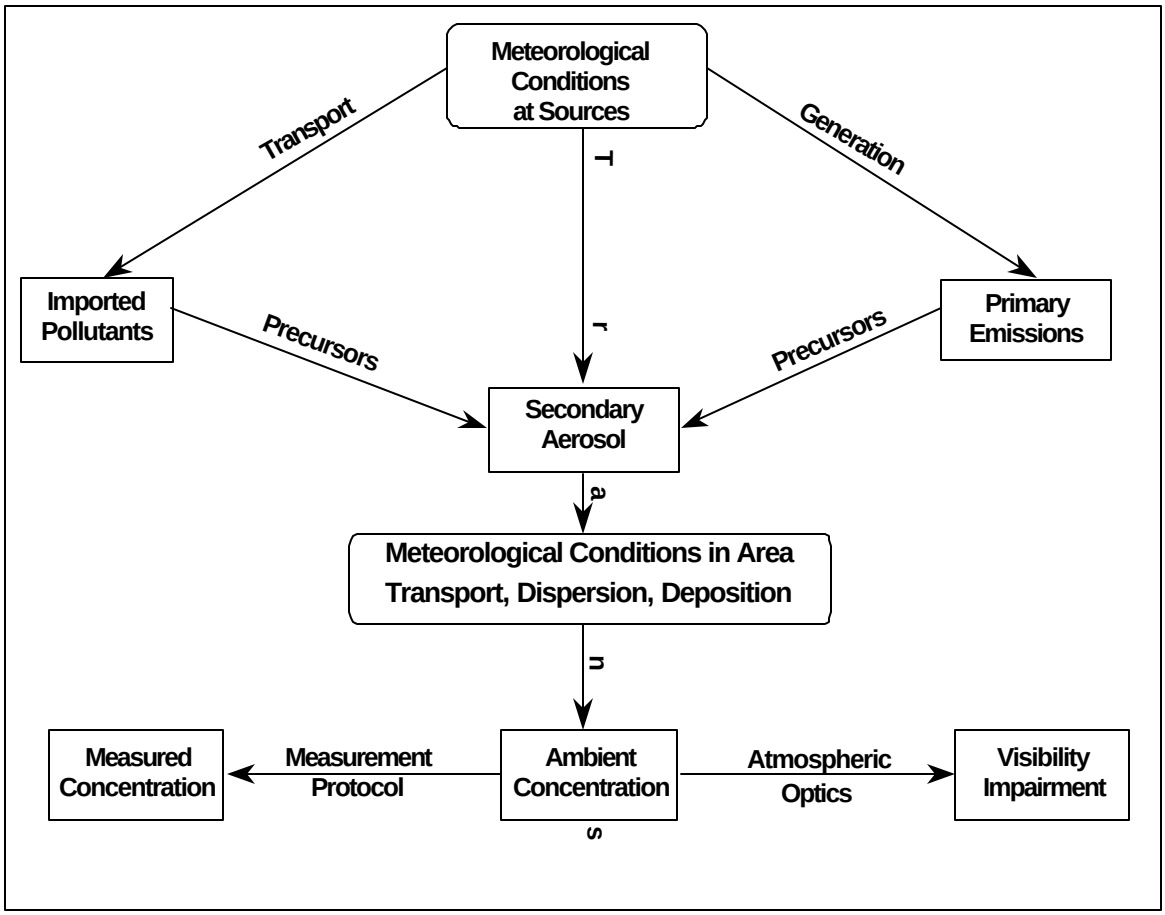


Figure 3.3. Flowchart of actual linkages between particulate matter air pollution and controlling factors of weather and source activity

Pollutant concentrations at measurement sites vary not only due to the various influences on local pollutants, but also due to the transport of material from upwind areas. In addition to variable local influences, occasional transport of PM can significantly influence concentrations, particularly at sites downwind of major urban centers. Different conditions not only cause different concentrations, they can also alter the mix of responsible sources; in other words, the sources identified for appropriate control can vary not only temporally but

also among monitoring sites. For example, in the San Joaquin Valley, PM10 and PM2.5 episodes in the winter-time are often accompanied by light and variable winds, thus limiting horizontal transport. As a result, pollutants tend to accumulate in local areas; however, a uniform gradient of secondary aerosols was seen valley wide (nitrates in particular). Results of several data analyses, as to the cause of this smooth gradient in secondary particulate concentrations, revealed a shallow mixing layer near the surface with nearly calm winds, but winds of 4 to 8 m/s were observed about 100 meters above the surface. Thus, pollutants trapped near the surface when mixed into this fast moving upper layer, were transported large distances and reacted with sources such as ammonia to form the secondary aerosols.

3.2.6 Fine Mass and Aerosol Light Scattering Relationship

The aerosol parameter to be monitored must be a suitable causal measure of health effects, as well as effects on visibility, climate, etc. It can be presumed that, for health effects, penetration into the lung and toxicity of the aerosol chemical species are relevant. On the other hand, visibility effects are determined by the light extinction under atmospheric conditions. The direct aerosol effect on climate is due to scattering and absorption of sunlight while the indirect aerosol effect on climate is due to the aerosol interaction with cloud processes. Because each of the aerosol effects is associated with a specific size and/or chemical composition, it is not likely that a single monitoring variable would be equally suitable as a surrogate for all of the effects. Thus, a choice in the measurement technique requires a value judgment as to which effect (health, visibility, or climate) matches most closely with exposure.

Depending on their size and composition, particles can scatter or absorb light. Coefficient of haze (COH) and nephelometer ($B_{scat,}$ or scattering coefficient) measurements provide an indication of the relative contributions of light absorption and light scattering. The COH is a direct measure of the light-absorbing ability of the particles. Light absorption is primarily due to elemental carbon from combustion. The nephelometer roughly measures all scattering by fine particles. The characteristics of scattering light are extremely sensitive to the size of the scattering particles. Light scattering by the large particles (>10 µm diameter) is generally not significant. As particle sizes approach the range of light wavelengths (0.1-1 µm) they become significantly more efficient in light scattering. COH units are defined as the quantity of particulate matter that produces an optical density of 0.01 on a paper filter tape. A photometer detects the change in the quantity of light transmitted through the spot as the particulate matter collects on the paper filter tape and produces an electrical signal proportional to the optical density. A COH of less than 1.0 represents relatively clean air while a COH of greater than 2.0 represents air with a relatively high concentration of primary combustion-generated particles and/or secondary aerosols formed in the atmosphere.

As was noted earlier in this chapter, the aerosol population is a mixture of different particle sizes, and each size class is composed of an internal and/or external mixture of chemically diverse particles. Hence, it is not possible to express the aerosol concentration as a single number, as is the case for gaseous pollutants. On the other hand, practical considerations dictate that the number of aerosol parameters to be monitored has to be limited. Routine monitoring of aerosol chemical composition in many size classes does not appear to be practical for regulatory purposes. Rather, the aerosol size - chemical composition distribution function needs to be monitored using integral measures such as fine mass concentration (PM2.5) and/or total (or size segregated) light scattering coefficient. PM2.5 is the integral of the aerosol mass - size distribution up to about 2.5 µm. The total light scattering is also an integral of the aerosol mass size distribution but also weighted by the size-dependent scattering efficiency factor.

Numerous field investigations have been performed on the correlation between scattering coefficient and particulate volume and mass concentration. Most of the earlier studies (1970s) were based on “high volume” (non-particle size selective sampler) total suspended particle (TSP) mass concentration measurements whose uncertainties and ill-defined upper particle size limits resulted in questionable data. As attention focussed on fine particle monitoring during the 1980s, similar comparison field tests restricted to smaller particles were conducted. It is well established that the fine particle mass concentration measured by size segregated filter sampling has a strong statistical correlation with total aerosol light scattering. The main reason for this relationship is that both the fine particle mass as well as the light scattering efficiency factor have a peak in the size range 0.3 - 0.6 µm. Exception to this relationship occurs when the characteristic aerosol size is either smaller (e.g., primary automobile exhaust) or larger (wind blown dust) than the above size range.

Husar and Falke (1996) conducted a comparative study of the aerosol light scattering and fine particle mass data. A comparison of the light scattering coefficient and PM2.5 was performed for fourteen different sites in the western U.S. (including six sites in California). The scatter charts of daily PM2.5 and scattering data included the slope (m^2/g) of the relationship as well as the correlation, R^2. The data for the fourteen sites indicate a good correlation, with half of the sites exhibiting R^2 above 0.8. A notable exception is Azusa, CA, (R^2 = 0.61). The slope, i.e., the light scattering PM2.5 ratio, ranges between 4.1 and 11.9 with an average of 7.4 m^2/g.

Groblicki et al. (1981) presented the light scattering coefficient observed in studies in Denver, Colorado as a function of the observed mass in the fine and coarse particle ranges, respectively. It has been seen that a good linear relationship exists between scattering coefficient and the fine mass, but not between scattering coefficient and coarse particle mass. A good linear relationship has been observed in a number of areas ranging from pristine to urban sites with scattering coefficient to fine particle mass concentration ratio of approximately 3 (Waggoner et al. 1981; Conner et al. 1991).

Light scattering dominates light absorption except where there are light absorbing particles or gases present. Graphitic or elemental carbon (commonly known as soot) is very efficient at absorbing light. Particle light absorption is about 10% of particle scattering in rural areas, but can be nearly equal to particle light scattering in urban areas where elemental carbon is present (Waggoner and Weiss 1981). Because of the nature of its sources, the elemental carbon contribution to light extinction varies geographically and temporally. For example, wood-burning fireplaces and diesel engines are major sources of elemental carbon, and areas with large numbers of these sources generally have more elemental carbon in the atmospheric aerosol, hence more light absorption.

The results of several studies of the contribution of various particle components to light scattering and light absorption suggest that sulfate and organic species are major contributors to light scattering, with the contribution of nitrate being more variable. Relative humidity influences particle light extinction strongly when relative humidity exceeds 70%. The effect of humidity on light scattering properties is also very dependent on chemical and microphysical variables, as components of fine particles (hygroscopic fraction of aerosol) will vary in their ability to absorb water.

Finally, although results of several studies are strongly suggestive of common optical properties for the fine particle fraction, it would be disingenuous to claim that PM2.5 mass and light scattering coefficient are always equivalent, either temporally and spatially. The high-time resolution (i.e., hourly measurements) light scattering data clearly indicate that aerosol variation is significant in both seasonal and monthly time scales. There is also a measurable

diurnal variation of up to 50% of the daily average values where primary particle emissions are significant. The light scattering-humidity relationship depends on the particle composition, microstructure (i.e., internally or externally mixed aerosols) as well as the history of relative humidity values previously experienced by the particles. Hence the relationship between fine particle mass and light scattering can be obscured by many physical/ chemical factors and sampling errors. All of these factors should be examined carefully before the use of any scattering data for estimating fine mass concentration.

3.3 Chemical Properties of Particles

Generally, atmospheric PM can be divided into fine (<2.5 µm) and coarse particles (>2.5 µm). Fine and coarse particles differ in formation mechanisms, chemical composition, sources, and exposure relationships. Figure 3.4 represents a schematic diagram of both primary and secondary particles formation.

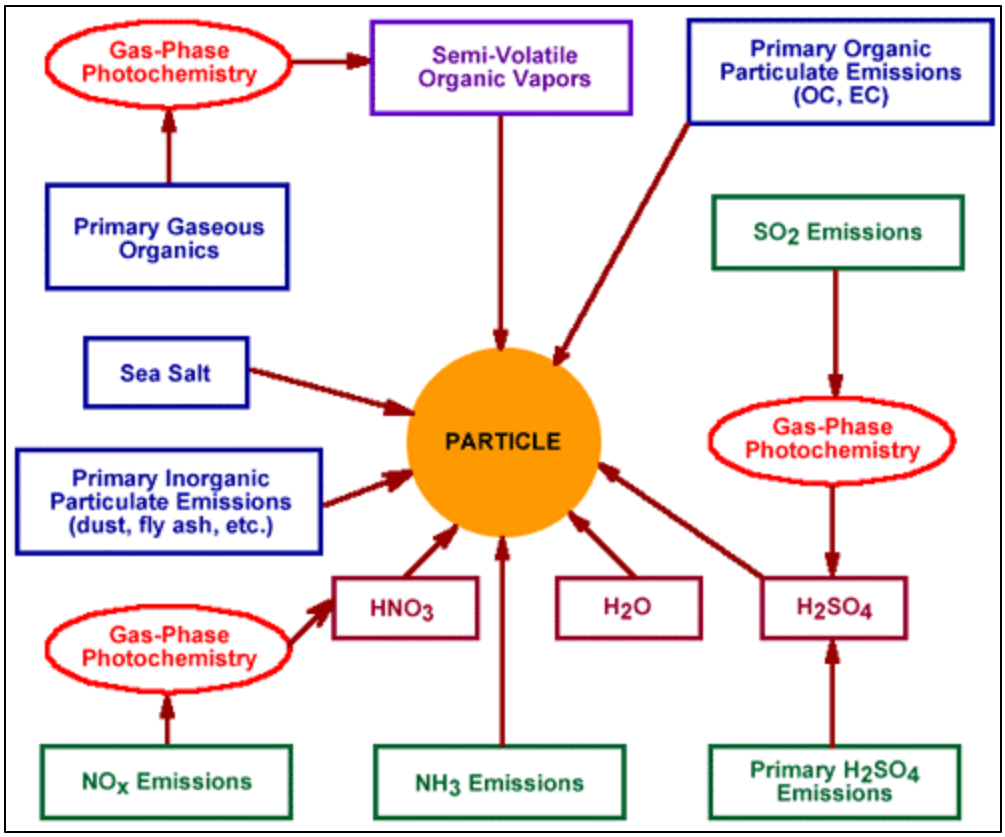


Figure 3.4. Schematic diagram of particle formation (adapted from Meng et al 1997).

Fine PM is derived from combustion material that has volatilized and then condensed to form primary PM, or from precursor gases (such as sulfur dioxide, nitrogen oxides, and certain organic compounds) reacting in the atmosphere to form secondary PM. Fine particles typically are comprised of sulfate, nitrate, ammonium, elemental carbon, organic compounds, and a variety of other compounds.

Coarse particles, in contrast, are formed by crushing, grinding, and abrasion of surfaces, which breaks large pieces of material into smaller pieces. These particles are then suspended by wind or by anthropogenic activity such as construction, mining, and agricultural activities.

As the particles respond to conditions in their atmospheric environment, their chemical and physical properties - and hence their characteristics, such as light scattering and toxicity - can change by accumulation of atmospheric gas-phase chemical reaction products or through heterogeneous reactions with gas-phase species.

3.3.1 Nitrate Chemistry

The atmospheric chemistry leading to formation of particulate nitrate is fairly complicated. Fresh NO_X emissions, which consist primarily of nitric oxide (NO) undergo reactions with ozone and peroxy radicals to form nitrogen dioxide (NO_2), via the reactions shown below.

$$NO + O_3 \rightarrow NO_2 + O_2 \quad (3.1)$$

$$NO + HO_2 \rightarrow NO_2 + OH \quad (3.2)$$

The NO_2 can be directly converted to nitric acid via the homogenous gas phase reaction with the hydroxyl radical (OH).

$$NO_2 + OH \rightarrow HNO_3 \quad (3.3)$$

This is the principal formation mechanism for nitric acid in the daytime (Finlayson-Pitts and Pitts, 1999). Modeling calculations suggest that more than 90% of the daylight HNO_3 formation occurs via this reaction. It involves the OH radical, which is the key species in the photochemical oxidation cycle. The OH radical concentration is controlled by the amount of sunlight and the ambient concentrations of ozone, water vapor, NO, NO_2, and reactive organic compounds.

NO_2 reacts with O_3 forming nitrate radical (NO_3). An important reaction of NO_3 is with NO_2 to form N_2O_5. The second major formation pathway for nitric acid is the reaction of N_2O_5 with water vapor and liquid water.

$$N_2O_5 + H_2O \rightarrow 2\ HNO_3 \quad (3.4)$$

The rate of reaction will only be significant when the liquid water content of the atmosphere is high, i.e., when clouds and fog are present.

There is a wide range of conversion rates for nitrogen dioxide to nitric acid, ranging from less than 1 percent per hour to 90 percent per hour. Although they vary throughout a 24-hour period, these rates are significant during both daytime and nighttime hours. This is in contrast to the gas-phase sulfate chemistry, which is most active during daylight hours.

The principal chemical loss process for gas-phase nitric acid is its reaction with gaseous ammonia to form ammonium nitrate (NH_4NO_3).

$$NH_3 + HNO_3 \rightarrow NH_4NO_3 \quad \text{(reversible)} \quad (3.5)$$

This reversible reaction is believed to be the primary source of fine (<2.5 µm diameter) nitrate aerosol in California's urban air. The equilibrium constant for the reaction is both temperature- and relative humidity-dependent. High humidity and low temperature favor NH_4NO_3 formation. Aqueous NH_4NO_3 is formed at relative humidities above the relative humidity of deliquescence (62%).

Another pathway for the formation of nitrate aerosol is a heterogeneous chemical reaction between sea-salt particles and gas-phase nitric acid, leading to thermally stable sodium nitrate production in the particle phase accompanied by liberation of gaseous hydrochloric acid (HCl) from the particles. Gard et al. (1998) focussed their study on the replacement of chloride by nitrate in sea-salt particles (reaction 3.6) at Long Beach.

$$HNO_3 + NaCl \rightarrow NaNO_3 + HCl \quad (3.6)$$

Reaction (3.6) may be the principal source of coarse (2.5 to 10 µm) nitrate, and plays an important role in atmospheric chemistry because it is a permanent sink for gas-phase nitrogen oxide species. This reaction is one of the most extensively studied heterogeneous chemical reactions in the laboratory, and the extent to which this occurs is affected by many factors, including gas-phase and particle-phase concentrations, temperature, relative humidity, and reaction time.

Significant amounts of NO_X can be converted to organic nitrates, such as peroxyacetyl nitrate (PAN) which is the most abundant organic nitrate in urban air. The thermal decomposition of PAN is very temperature sensitive. As temperature rises, PAN decomposes back to NO_2 and methyl peroxyacetyl. A deficit exists in observable NO_Y species in ambient air, and it is thought that PAN-analog compounds could comprise a significant part of the missing nitrogen species. Nitric acid and ammonia are believed to be deposited on surfaces very rapidly, while sulfate deposits relatively slowly. NO_X, ammonium, and nitrate aerosol deposit at rates in between these two extremes.

The atmospheric chemistry leading to formation of particulate nitrate is complicated. The rate of formation depends on the concentrations of many intermediate species (including ammonia and radical species) involved in the reactive organic gases and NO_X photochemical system. Figure 3.5 summarizes chemical pathways involving nitrogen oxides in the atmosphere (Warneck 1999). Photochemically induced reaction pathways are indicated by bold arrows. These processes are active only during the day, whereas the others occur at all times.

Until recently it was assumed that the end product of tropospheric NO_X was nitric acid. However, a recent research project conducted under ARB sponsorship (Mochida and Finlayson-Pitts 2000) has shown that nitric acid on a surface can react with NO to regenerate NO_2 which can then form ozone and particulate nitrate. Preliminary modeling studies suggest that this reaction may increase the formation of particulate nitrate and that existing models underestimate the benefit of NO_X controls for reducing PM and ozone. This finding may have very serious implications as to the effectiveness of control strategies for both ozone and PM. An additional research contract is continuing with a focus on providing a more complete understanding of the effect of heterogeneous nitrogen chemistry on ozone and particle formation. The information gained in this project may have very serious implications as to the effectiveness of control strategies for both ozone and PM.

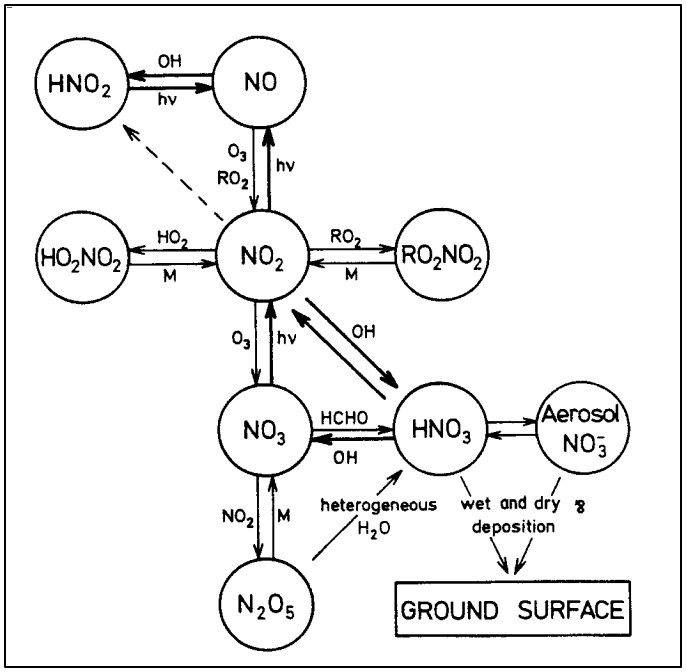


Figure 3.5. Oxidation scheme for nitrogen oxides and related compounds (adapted from Warneck 1999).

Ambient concentrations of secondary particles are not necessarily proportional to the quantities of their precursor emissions, since the rates at which they form and their gas/particle equilibria may be controlled by factors other than the concentration of the precursor gases. The rate of NO_X oxidation and the branching ratio between inorganic and organic nitrates depends on the specific environmental conditions in addition to reactant concentrations (Seinfeld and Pandis 1998). The partitioning of inorganic nitrate between gaseous nitric acid, ammonium nitrate, and nonvolatile nitrate is known to depend on a number of factors, such as relative humidity, temperature, and ammonia, in a nonlinear manner.

Secondary ammonium nitrate is generally the largest contributor to the PM2.5 mass during the winter at most of the urban sites in California. The results of several studies (Magliano et al.,1999; Kim, et al. 2000) indicate that during some episodes of high particle concentrations in California, ammonium nitrate – formed secondarily from NO_X and ammonia emissions – can account for over half of the PM2.5 mass. The formation of secondary particles, which are a major contributor to the fine PM levels in California, from gas-phase precursors is a complex, nonlinear process. Consequently, a one-to-one relationship between precursor emissions and ambient secondary PM concentrations is not expected. Understanding how particulate ammonium nitrate is formed and how to effectively reduce it through controls on NO_X and/or ammonia sources is a critical part of California's PM2.5 program.

3.3.2 Sulfate Chemistry

Sulfur dioxide emissions result almost exclusively from the combustion of sulfur-containing fuels. Other sulfur compounds, such as sulfur trioxide (SO_3), sulfuric acid (H_2SO_4), and sulfates (SO_4^{2-}), may also be directly emitted during combustion of sulfur-containing fuels, although usually only in small amounts. In the atmosphere, sulfur dioxide is chemically transformed to sulfuric acid, which can be partially or completely neutralized by ammonia and

other alkaline substances in the air to form sulfate salts (Warneck 1999; Seinfeld and Pandis 1998).

The oxidation of sulfur dioxide to sulfuric acid can occur in the gas phase, in or on particles, and in the aqueous phase (i.e., in droplets of rain, clouds, or fogs). Sunlight intensity, the presence of oxidants and oxidant precursors, relative humidity, and the presence of fogs and clouds all appear to be related to the observed high oxidation rates. Results of several studies show that aqueous-phase oxidation of SO_2 is a significant pathway for the total transformation of SO_2.

3.3.2.1 Aqueous-Phase Sulfur Dioxide Reactions

Oxidation of sulfur dioxide can also occur in the aqueous phase via reactions of dissolved sulfur constituents (hydrated SO_2, sulfite, and bisulfite; collectively called S(IV)) with hydrogen peroxide (H_2O_2), ozone, and oxygen catalyzed by iron and manganese (Kleinman 1984; Seigneur et al. 1984). Ozone is an important oxidant for sulfur dioxide at high pH, but its effect becomes negligible at pH levels less than 4. The extent of S(IV) oxidation is primarily limited by the availability of H_2O_2 and the low solubility of sulfur dioxide at low pH. When fog droplets form on acidic nuclei, the low initial pH prevents oxidation of S(IV) other than by H_2O_2.

The effects of season and time of day suggest the importance of photochemistry, and perhaps temperature, in the oxidation rate of SO_2. This does not necessarily imply that oxidation reactions themselves are photochemical in nature, but rather they may involve oxidants such as H_2O_2 which are formed through photochemical processes.

The fastest atmospheric reactions of SO_2 believed to be with H_2O_2, and with O_3 at higher pH values. Under extreme conditions of large droplets (>10 µm) and very high oxidant concentrations, the chemical reaction times may approach those of diffusion, particularly in the aqueous phase. However, it is believed that under most conditions typical of the troposphere, this will not be the case and the chemical reaction rate will be rate determining in the S(IV) aqueous phase oxidation.

In heavily polluted atmospheric water droplets, such as those found in urban fogs, metal-catalyzed S(IV) oxidation is a significant contributor to formation of S(VI) in the liquid phase, and apparently is more important than oxidation by H_2O_2.

3.3.2.2 Gas-Phase Sulfur Dioxide Reactions

Sulfur dioxide is converted to sulfuric acid in the gas phase during daylight hours, primarily by reaction with hydroxyl radical (OH). (See reaction sequence below.)

$$OH + SO_2 \rightarrow HOSO_2 \quad (3.7)$$

$$HOSO_2 + O_2 \rightarrow HO_2 + SO_3 \quad (3.8)$$

$$SO_3 + H_2O \rightarrow H_2SO_4 \quad (3.9)$$

The SO_3-H_2O adduct may dissociate back to reactants with about the same probability as it rearranges to sulfuric acid. Thus, the kinetics of sulfuric acid formation in reaction (3.9) may be considerably more complex than if it were a simple bimolecular reaction as written above.

Because of its extremely low vapor pressure ($<10^{-7}$ atmospheres), sulfuric acid quickly adheres to existing particles. Sulfuric acid reacts irreversibly with ammonia to form ammonium bisulfate, NH_4HSO_4 and ammonium sulfate, $(NH_4)_2SO_4$. Since the sedimentation velocity of these submicrometer particles is very low, sulfate can be transported long distances. In the absence of precipitation or fog, the typical atmospheric lifetime of fine particulate sulfate is on the order of several days. Washout by precipitation and accelerated

sedimentation resulting from incorporation of sulfate particles into fog droplets are important sinks.

In power-plant or smelter plumes containing SO_2 and NO_X, the gas-phase chemistry depends on plume dilution, sunlight, and volatile organic compounds, either in the plume or in the ambient air mixing into and diluting the plume. For the conversion of SO_2 to H_2SO_4, the gas-phase rate in such plumes during summer midday conditions in the eastern United States typically varies between 1 and 3% h^{-1} but in the cleaner western United States rarely exceeds 1% h^{-1}. For the conversion of NO_X to HNO_3, the gas-phase rates appear to be approximately three times faster than the SO_2 conversion rates. During the winter, rates for SO_2 conversion are approximately an order of magnitude lower than during the summer.

The contribution of aqueous-phase chemistry to particle formation in point-source plumes is highly variable, depending on the availability of the aqueous phase (wetted aerosols, clouds, fog, and light rain) and the photochemically generated gas-phase oxidizing agents, especially H_2O_2 for SO_2 chemistry. The in-cloud conversion rates of SO_2 to SO_4^{2-} can be several times larger than the gas-phase rates. Overall, it appears that SO_2 oxidation rates to SO_4^{2-} by gas-phase and aqueous-phase mechanisms may be comparable in summer, but aqueous phase chemistry may dominate in winter.

Nationwide, large reductions in ambient SO_2 concentrations have resulted in reductions in sulfate formation that would have been manifest in PM2.5 concentrations on the regional scale in the eastern and central United States, where sulfate has historically constituted a larger fraction of PM2.5 than in the west. Likewise, reductions in NO_2 concentrations would have had a more noticeable impact on PM2.5 concentrations in the western United States than in the eastern United States because nitrate is a larger component of the aerosol in the western United States. Trends in aerosol components (i.e., nitrate, sulfate, carbon, etc.) are needed for a more quantitative assessment of the effects of changes in emissions of precursors. Measurements of aerosol nitrate and sulfate concentrations have been obtained at North Long Beach and Riverside, CA, since 1978 (Dolislager and Motallebi, 1999). Downward trends in aerosol nitrate have tracked downward trends in NO_x concentrations, and SO_2 and sulfate concentrations have both decreased. However, the rate of decline of sulfate has been smaller than that of SO_2, indicating that long-range transport of sulfate from outside the air shed may be an important source in addition to the oxidation of locally generated SO_2. There are a number of reasons why pollutant concentrations do not track estimated reductions in emissions. Some of these reasons are related to atmospheric effects, such as meteorological variability and changes in the rates of photochemical transformations and deposition. Other reasons are related to uncertainties in ambient measurements and in emissions inventories.

3.3.3 Organic Particles

Atmospheric particulate carbon consists of both elemental carbon (EC) and organic carbon (OC). Elemental carbon has a chemical structure similar to impure graphite and is emitted directly by sources. Organic carbon can either be emitted directly by sources (primary OC) or can be the result of the condensation of low-vapor-pressure products of the gas-phase reactions of hydrocarbons onto the existing aerosol (secondary OC). Atmospheric carbon particles are emitted from more than 70 different types of air pollution sources (Gray and Cass 1998). Obvious sources include gasoline-powered motor vehicles, heavy-duty diesel vehicles, railroad engines, boilers, aircraft and many other combustors that burn fossil fuel. To the emissions from fuel combustion are added carbon particles from woodsmoke, food cooking operations, and even an ambient concentration increment from such minor sources

as cigarette smoke. In addition, there are fugitive sources including the organic carbon content of paved road dust, tire dust and vehicular brake wear particles.

Although the mechanisms and pathways for forming inorganic secondary particulate matter are fairly well known, those for forming secondary organic PM are not as well understood. Ozone and the hydroxyl radical are thought to be the major initiating reactants. Pandis et al. (1992) identified three mechanisms for formation of secondary organic PM: (1) condensation of oxidized end-products of photochemical reactions (e.g., ketones, aldehydes, organic acids, hydroperoxides), (2) adsorption of organic gases onto existing solid particles (e.g., polycyclic aromatic hydrocarbons), and (3) dissolution of soluble gases that can undergo reactions in particles (e.g., aldehydes). The first and third mechanisms are expected to be of major importance during the summertime when photochemistry is at its peak. The second pathway can be driven by diurnal and seasonal temperature and humidity variations at any time of the year. With regard to the first mechanism, Odum et al. (1996) suggested that the products of the photochemical oxidation of reactive organic gases are semivolatile and can partition themselves onto existing organic carbon at concentrations below their saturation concentrations. Thus, the yield of secondary organic PM depends not only on the identity of the precursor organic gas but also on the ambient levels of organic carbon capable of absorbing the oxidation product.

The formation of atmospheric aerosols from biogenic emissions has been of interest for many years. Recent laboratory and field studies support the concept that nonvolatile and semivolatile oxidation products from the photo-oxidation of biogenic hydrocarbons could contribute significantly to ambient PM concentrations in both urban and rural environments. A number of multifunctional oxidation products have been identified in laboratory studies (Yu et al. 1998; Glasius et al. 2000; Koch et al. 2000). Many of these compounds have subsequently been identified in field investigations (Kavouras et al. 1998, 1999b). However, further investigations are needed to accurately assess their overall contributions to fine PM concentrations.

Generally, organic PM concentrations, composition, and formation mechanisms are poorly understood. Particulate organic matter is an aggregate of hundreds of individual compounds spanning a wide range of chemical and thermodynamic properties (Saxena and Hildemann, 1996). Some of the organic compounds are “semivolatile” such that both gaseous and condensed phases exist in equilibrium in the atmosphere. The presence of semivolatile or multiphase organic compounds complicates the sampling process. Understanding the mechanisms of formation of secondary organic PM is important because secondary organic PM can contribute in a significant way to ambient PM levels, especially during photochemical smog episodes. Experimental studies of the production of secondary organic PM in ambient air have focused on the Los Angeles Basin. Turpin and Huntzicker (1994, 1995) provided strong evidence that secondary PM formation occurs during periods of photochemical ozone formation in Los Angeles and that as much as 70% of the organic carbon in ambient PM was secondary in origin during a smog episode in 1987. Schauer et al. (1996) estimated that on an annually averaged basis, 20 to 30% of the total organic carbon PM in the <2.1µm size range in the Los Angeles airshed was secondary in origin.

A high degree of uncertainty is associated with all aspects of the calculation of secondary organic PM concentrations. Currently, it is not possible to fully quantify the concentration, composition, or sources of the organic components. Many of the secondary organic aerosol components are highly oxidized, difficult to measure, multifunctional compounds. This is compounded by the volatilization of organic carbon from filter substrates during and after sampling as well as potential positive artifact formation from the absorption of gaseous hydrocarbon on quartz filters. In addition, no single analytical technique is currently capable of

analyzing the entire range of organic compounds present in the atmosphere in PM. Even rigorous analytical methods are able to identify only 10 to 20% of the organic PM mass on the molecular level (Rogge et al. 1993a; Schauer et al. 1996).

Environmental smog chambers can be useful in elucidating the chemical mechanisms associated with the formation of compounds found in organic PM; however, significant uncertainties always arise in the interpretation of smog chamber data because of wall reactions. Limitations also exist in extrapolating the results of smog chamber studies to ambient conditions found in urban airsheds. Additional laboratory studies are needed to comprehensively identify organic compounds, strategies need to be developed to sample and measure such compounds in the atmosphere, and models of secondary organic aerosol formation need to be improved and added to air quality models in order to address compliance issues related to reducing PM mass concentrations that affect human exposure.

3.3.4 Particle-Vapor Partitioning

Several atmospheric aerosol species, such as ammonium nitrate and certain organic compounds, are semivolatile and are found in both gas and particle phases. A variety of thermodynamic models have been developed to predict the temperature and relative humidity dependence of the ammonium nitrate equilibria with gaseous nitric acid and ammonia. The gas-particle distribution of semivolatile organic compounds depends on the equilibrium vapor pressure of the compound, total particle surface area, particle composition, atmospheric temperature, and relative humidity. Although it generally is assumed that the gas-particle partitioning of semivolatile organics is in equilibrium in the atmosphere, neither the equilibria nor the kinetics of redistribution are well understood. Diurnal temperature fluctuations, which cause gas-particle partitioning to be dynamic on a time scale of a few hours, can cause semivolatile compounds to evaporate during the sampling process. The pressure drop across the filter can also contribute to loss of semivolatile compounds. The dynamic changes in gas-particle partitioning, caused by changes in temperature, pressure, and gas-phase concentration, both in the atmosphere and after collection, cause serious sampling problems.

A recent ARB-funded final research report (Ashbaugh et al. 1998) describes analysis of three data sets to evaluate the extent of mass loss on polytetrafluoroethylene (PTFE or Teflon®) filters due to ammonium nitrate volatilization. The results indicated that the effect on measured mass is site-dependent, and depends on the meteorological conditions and the fraction of PM mass that consists of ammonium nitrate particles. There is no straightforward method to correct for the mass loss without measuring it. The highest mass loss occurred during summer daytime in southern California, amounting to 30-50% of the gravimetric mass. This study of ammonium nitrate suggests potentially significant nitrate or semivolatile organic compounds loss using the Federal Reference Method sampler for fine particle sampling because it uses PTFE filters for mass concentrations. This may lead to control strategies that are biased toward sources of fugitive dust and other primary particle emission sources.

3.4 Summary

Atmospheric particles originate from a variety of sources and possess a range of morphological, chemical, physical, and thermodynamic properties. Atmospheric size distributions show that most atmospheric particles are quite small, below 0.1 µm, whereas most of the particle volume (and therefore most of the mass) is found in particles greater than 0.1 µm. Several processes influence the formation and growth of particles. New particles may be formed by nucleation from gas phase material. Existing particles may grow by condensation as gas phase material condenses onto existing particles. Particles may also grow by coagulation as two particles combine to form one. Gas phase material condenses preferentially on smaller particles and the rate constant for coagulation of two particles

decreases as the particle size increases. Therefore, nuclei mode particles grow into the accumulation mode but accumulation mode particles do not grow into the coarse mode.

The lifetimes of particles vary with particle size. Coarse particles can settle rapidly from the atmosphere within minutes or hours, and normally travel only short distances. However, when mixed high into the atmosphere, as in dust storms, the smaller-sized, coarse-mode particles may have longer lives and travel greater distances. Accumulation-mode fine particles are kept suspended by normal air motions and have very low deposition rates to surfaces. They can be transported thousands of kilometers and remain in the atmosphere for a number of days. Accumulation-mode particles are removed from the atmosphere primarily by cloud processes. Coarse mode particles of less than 10 µm diameter as well as accumulation-mode and nuclei-mode (or ultrafine) particles all have the ability to penetrate deep into the lungs and be removed by deposition in the lungs.

The major constituents of atmospheric PM are sulfate, nitrate, ammonium, and hydrogen ions; particle-bound water; elemental carbon; a great variety of organic compounds; and crustal material. Particulate material can be primary or secondary. PM is called primary if it is in the same chemical form in which it was emitted into the atmosphere. PM is called secondary if it is formed by chemical reactions in the atmosphere. Primary coarse particles are usually formed by mechanical processes. Primary fine particles are emitted, either directly as particles or as vapors that rapidly condense to form particles.

Most of the sulfate and nitrate and a portion of the organic compounds in atmospheric particles are secondary. Secondary aerosol formation depends on numerous factors including the concentrations of precursors; the concentrations of other gaseous reactive species such as ozone, hydroxyl radical, peroxy radicals, or hydrogen peroxide; atmospheric conditions, including solar radiation and relative humidity; and the interactions of precursors and preexisting particles within cloud or fog droplets, or on or in the liquid film on solid particles. As a result, it is considerably more difficult to relate ambient concentrations of secondary species to sources of precursor emissions than it is to identify the sources of primary particles.

Finally, current filter-based mass measurements lead to significant evaporative losses, during and possibly after collection, of a variety of semivolatile components (i.e., species that exist in the atmosphere in dynamic equilibrium between the condensed phase and gas phase). Important examples include ammonium nitrate and semivolatile organic compounds. Loss of these components may significantly impact the quality of the measurement, and can lead to both positive and negative sampling artifacts. The systematic bias in the sampling method is likely to result in a bias in recommended control strategies. If the measured mass is under-represented by the semivolatile compounds in the atmosphere, other sources of particulate matter will be over-represented. Thus, control strategies developed from the biased data will tend to overemphasize controls on nonvolatile species.

3.5 References

Abdul-Khalek IS, Kittelson DB, Graskow BR, Wei Q, Brear F. Diesel exhaust particle size: Measurement issues and trends, SAE 1998; Technical Paper No. 980525.

Ashbaugh LL, Eldred RA, Hering S. Loss of particle nitrate from Teflon sampling filters: effects on measured gravimetric mass. Final Report, California Air Resources Baord; Contract No. 96-305, 1998.

Boon KF, Kiefert L, McTainsh GH. Organic matter content of rural dust in Australia. Atmos Environ 1998; 32: 2817-2823.

Clarke AD. Atmospheric nuclei in the remote free-troposphere. J Atmos Chem 1992; 14: 479-488.

Conner WD, Bennett RL, Weathers WS, Wilson WE. Particulate characteristics and visul effects of the atmosphere at Research Triangle Park. J Air Waste Manage Assoc 1991; 41: 154-160.

Covert DS, Kapustin VN, Quinn PK, Bates TS. New particle formation in the marine boundary layer. J Geophys Res 1992; 97: 20,581-20,589.

Dolislager LJ, Motallebi N. Characterization of particulate matter in California. Journal of the Air & Waste Management Association 1999; 49: PM-45-56.

Dreher DB Harley RA. A fuel-based inventory for heavy duty diesel truck emissions. Journal of the Air & Waste Management Association 1998; 48, 352-358.

Finlayson-Pitts BJ, Pitts NJ. Chemistry of the upper and lower atmosphere. Academic Press, San Diego, CA 1999.

Friedlander, S.K., Smoke, dust and haze: fundamentals of aerosol dynamics, 2nd edition, New York, NY: Oxford University Press, 2000.

Gao W, Wesely ML. Modeling gaseous dry deposition over regional scales with satellite observation: model development. Atmos Environ 1995; 29: 727-737.

Gatz DF, Prospero JM. A large silicon-aluminum aerosol plume in central Illinois: north Africa desert dust. Atmos Environ 1996; 30: 3789-3799.

Glasius M, Lahaniati M, Calogirou A, Di Bella D, Jensen NR, Hjorth J, Kotzias D, Larsen BR. Carboxylic acids in secondary aerosols from oxidation of cyclic monoterpenes by ozone. Environ Sci Technol 2000; 34: 1001-1010.

Gray HA, Cass GR. Source contributions to atmospheric fine carbon particle concentrations. Atmos Environ 1998; 32: 3805-3825.

Groblicki PJ, Wolff GT, Countess RJ. Visibility reducing species in the Denver brown cloud. Atmos Environ 1981; 15, 2473-2484.

Hildemann LM, Markowski GR, Jones MC, Cass GR. Submicrometer aerosol mass distributions of emissions from boilers, fireplaces, automobiles, diesel trucks, and meat-cooking operations. Aerosol Sci Technol 1991; 14: 138-152.

Holmes JT, Samberg L, McInnes J, Zieman W, Zoller, Harris J. Long-term aerosol and trace acidic gas collection at Mauna Loa Observatory 1979-1991. J Geophs Res 1997; 102: 19007-19019.

Hughes LS, et al. The size and composition distribution of atmospheric particles in southern California. Environ Sci Technol 1999, 33: 3506-3515.

Husar RB, Falke R. The relationship between aerosol light scattering and fine mass. Report No. CX 824179-01; prepared for Office of Air Quality Planning and Standards, U.S.-EPA 1996.

Kamens R, Jang M, Chien C.-J, Leach K. Aerosol formation from the reaction of "alpha"-pinene and ozone using a gas-phase kinetics-aerosol partitioning model. Environ Sci Technol 1999; 33: 1430-1438.

Kavouras IG, Mihalopoulos N, Stephanou EG. Formation of atmospheric particles from organic acids produced by forests. Nature 1998; 395: 683-686.

Kavouras IG, Mihalopoulus N, Stephanou EG. Secondary organic aerosol formation vs primary organic aerosol emission: in situ evidence for the chemical coupling between monoterpene acidic photooxidation products and new particle formation over forests. Environ Sci Technol 1999b; 33: 1028-1037.

Kerminen VM, Wexler AS. Growth laws for atmospheric aerosol particles: an examination of the bimodality of accumulation mode. Atmos Environ 1995; 29: 3263-3275.

Kim BM, Teffera S, Zeldin MD. Characterization of PM2.5 and PM10 in the South Coast Air Basin of Southern California: Part 1- Spatial variations. J Air & Waste Mange Assoc 2000; 50: 2034-2044.

Kittelson DB, Engines and nanoparticles: A review. J. Aerosol Sci. 1998, 29: 575-588.

Kleinman LI. Oxidant requirements for the acidification of precipitation. Atmos Environ 1984; 18: 1453-1457.

Koch S, Winterhalter R, Uherek E, Kolloff A, Neeb P, Moortgat GK. Formation of new particles in the gas-phase ozonolysis of monoterpenes. Atmos Environ 2000; 34: 4031-4042.

Magliano KL, Hughes VM, Chinkin LR, Coe, DL, Haste TL, Kumar N, Lurmann FW. Spatial and temporal variations in PM10 and PM2.5 source contributions and comparison to emissions during the 1995 integrated monitoring study. Atmos Environ 1999; 33: 29 4757-4773.

McMurry PH, Friedlander SK. New particle formation in the presence of an aerosol. Atmos Environ 1979; 13: 1635-1651.

Meng Z, Dabdub D, Seinfeld JH. Chemical coupling between atmospheric ozone and particulate matter. Science 1997; 277: 116.

Mochida M, Finlayson-Pitts BJ. FTIR study of the reaction of gaseous NO with HNO_3 on silica surfaces: Implications for conversion of HNO_3 to photochemically active NO_X in the atmosphere. J Phy Chem A 2000; 104-9705.

Odum, JR, Hoffmann, T, Bowman, F, Collins, D, Flagan, RC, Seinfeld, JH. Gas/particle partitioning and secondary organic aerosol yields. Environ Sci Technol 1996; 30: 2580-2585.

Pandis SN, Harley RA, Cass GR, Seinfeld JH. Secondary organic aerosol formation and transport. Atmos Environ 1992; Part A 26: 2269-2282.

Pandis SN, Wexler AS, Seinfeld JH. Dynamics of tropospheric aerosols. J Phy Chem 1995; 99:9646-9659.

Rogge, W. F, Mazurek, MA, Hildemann, LM, Cass, GR, Simoneit, BR. Quantification of urban organic aerosols at a molecular level: identification, abundance and seasonal variation. Atmos Environ 1993a; 27: 1309-1330.

Saxena P, Hildemann LM. Water-soluble organics in atmospheric particles: a critical review of the literature and applications of thermodynamics to identify candidate compounds. J Atmos Chem 1996; 24: 57-109.

Schauer, JJ, Rogge, WF, Hildemann, LM, Mazurek, MA, Cass, GR. Source apportionment of airborne particulate matter using organic compounds as tracers. Atmos Environ 1996; 30: 3837-3855.

Seigneur C, Saxena P, Roth PM. Computer simulation of the atmospheric chemistry of sulfate and nitrate formation. Science 1984; 225: 1028-1029.

Seinfeld JH, Pandis SN. Atmospheric chemistry and physics – From air pollution to climate change". John Wiley and Sons, Inc, New York; 1998.

Turpin BJ, Huntzicker JJ. Investigation of organic aerosol sampling artifacts in the Los Angeles. Atmos Environ 1994; 28" 3061-3071.

Turpin BJ, Huntzicker JJ. Identification of secondary organic aerosol episodes and quantitation of primary and secondary organic aerosol concentrations during SCAQS. Atmos Environ 1995; 29: 3527-3544.

Waggoner AP, Weiss RE, Ahlquist NC, Covert DS, Will S, Charlson RJ. Optical characteristics of atmopsheric Aerosols. Atmos Environ 1981; 15: 1891-1909.

Weschler CJ, Shields HC. Indoor ozone/terpene reactions as a source of indoor particles. Atmos Environ 1999; 33: 2301-2312.

Yu J, Cocker DR, III, Griffin RJ, Flagan RC, Seinfeld JH. Gas-phase ozone oxidation of monoterpenes: gaseous and particulate products. J Atmos Chem 1998; 34: 207-258.

Zhang XY, Arimoto R, An ZS. Dust emission from Chinese Desert sources linked to variations in atmospheric circulation. J Geophys Res 1997; 102:28041-28047.

Zieman JJ, Holmes JL, Connor D, Jensen CR, Zoller WH. Atmospheric aerosol trace element chemistry at Mauna Loa Observatory. J Geophys Res 1995; 100: 25979-25994.

4. Sources and Emissions of Particles

Particulate matter is produced by emission sources either directly in particle form (primary PM), or as gases that react in the atmosphere to produce particulates (secondary PM). The emissions are produced by stationary, mobile, area-wide, and natural sources. For air pollution, the particulates of concern are those that are 10 micrometers or less in size (PM10), and, those that are 2.5 micrometers or less in size (PM2.5, which is a subset of PM10). This section discusses the characteristics of the major particulate matter sources.

PM emission levels are either measured, using monitoring equipment, or estimated, using emission inventory methods. Most of the information provided in this section is from estimated emission inventory data. This is currently the most reliable and comprehensive method of comparing PM emissions between sources and for evaluating regional emission sources.

4.1 Primary Particulate Sources

Primary particulate emission sources emit particulate matter directly to the air. Primary sources include stationary, mobile, area-wide, and natural particulate generating processes. Figure 4.1 summarizes the statewide directly emitted PM10 emission sources for California. Each of the major source categories depicted in the chart is discussed more fully below.

4.1.1 Stationary Sources

Stationary sources are generally small contributors to overall statewide primary particulate levels. The stationary source PM contribution is small because most major stationary source facilities have incorporated control equipment for decades and therefore are not large PM emitters. Some stationary sources of PM include industrial sources such as petroleum refining, wood and paper processing, food and agricultural processing, and sand, rock, and gravel mining and handling. Most stationary source facilities submit emission inventory reports to their air districts, so PM from these sources is typically well quantified. Most of the PM generated by combustion from stationary sources is PM2.5. Other stationary sources, such as those handling mineral products, emit relative greater proportions of PM10.

4.1.2 Mobile Sources

The contributions of directly emitted PM from mobile sources vary substantially within California. Sources of mobile emissions include gasoline and diesel powered vehicle exhaust emissions, tire wear, and break wear. Types of mobile sources include trucks, busses, heavy equipment, ships, trains, and aircraft. Like most combustion sources, the particulate emissions from mobile sources are nearly all in the PM2.5 size fraction. (This category does not include the road or soil dust created by car, truck, or equipment operations, which are included in the area-wide source category.)

4.1.3 Area-Wide Sources

Based on ambient measurements and emission inventory data developed by the ARB, area-wide sources contribute to a large fraction of the primary particulate emissions inventoried for the State. Area-wide sources are generally defined as sources that lack a definitive emissions point such as a stack or exhaust pipe, or sources which are relatively small, numerous, and geographically spread out.

For PM10, some of the most significant area-wide sources of directly emitted PM are geologic dust, such as windblown dust from disturbed lands, paved road dust, unpaved road dust, construction activities, and agricultural land preparation. Typical area-wide combustion sources, which predominantly produce particulates in the sub-2.5 micrometer size range,

include burning of agricultural debris, open burning, forest and range management burning, wildfires, fireplaces and wood stoves. The major PM sources vary from region to region in California, as well as by season.

4.1.4 Natural Sources

Most natural sources of PM are not currently included in the statewide emission inventory. These sources include marine-derived airborne salts, windblown dust from undisturbed lands, and biogenic emissions from plants. However, wildfires are currently included in emission inventory estimates.

4.2 Secondary Particulate Sources

Secondary particulate matter is typically 2.5 micrometers or less in size. Secondary PM is formed via atmospheric reactions of primary gaseous emissions. The gases that are the most significant contributors to secondary particulates in California are nitrogen oxides, ammonia, sulfur oxides, and certain organic gases.

The primary sources of nitrogen oxides and sulfur oxides include motor vehicle exhaust and stationary combustion sources such as boilers and other industrial equipment. Sources of ammonia include livestock operations such as dairies and feedlots, fertilizer application, some industrial sources, and biogenic sources. Organic gases are produced by both anthropogenic and natural sources.

Unlike direct emissions, it is not possible to develop an emission inventory for secondary particulates. This is because the particles form through various chemical pathways when gaseous emissions react in the atmosphere. So instead, the precursor gases are inventoried, and then location- and time-specific modeling is performed to estimate how much of each gas converts to particles.

Because a significant component of PM2.5 can be due to gaseous precursors, a pie chart that includes only the directly emitted PM2.5 emissions can be misleading, and is not included in this document. Such a chart would not give an accurate representation of which sources contribute to PM2.5 levels, especially in regions with high secondary particulate levels. For PM2.5, chemically speciated air quality monitoring data often provides a more meaningful portrayal of the sources contributing to PM2.5 in the air.

4.3 Regional Dependence of Source Contributions

The contributors to primary PM vary regionally in the State. Urban areas are typically dominated by paved road dust and construction-related emissions for directly emitted PM. More rural regions include paved and unpaved road dust, farming operations, and windblown dust as major contributors. Desert regions often have some of the cleanest air in the State, but, when episodic windstorms occur, they also experience some of the most dramatic exceedances of the PM10 standards. Unpaved road dust, paved road dust, and construction activities also contribute to PM10 in the desert.

Regional meteorology also plays a part in PM concentrations in the State. As mentioned, high winds can contribute to PM in the drier areas of the State. In other areas, stagnant air can exacerbate PM levels. Moist, colder weather in the San Joaquin Valley during winter contributes to the formation of secondary nitrates, and nitrates also contribute to high particulate levels in Southern California. In regions that are relatively cold, wood burning can substantially increase regional PM10 concentrations during the winter. In summary, the sources and quantities of PM emissions throughout the state are strongly affected by regional meteorology, geography, population, and land use.

4.4 Temporal Dependence of Source Contributions

Regional monitoring shows that many areas in the state show seasonal trends in ambient PM concentrations. For example, in the Sacramento Valley, PM values peak during October to January, and July to August. In the San Joaquin Valley, there are both winter and late fall peaks. The winter PM is predominantly fine particulate (PM2.5 and smaller secondary particulates), while the fall season PM has a more significant PM10 emission component due to directly emitted geologic dust. In Southern California, high PM levels occur at several times of the year based on meteorological conditions. In the Owens Valley, windstorms create short-term episodic high PM concentrations. And in places like Mammoth Lakes and Lake Tahoe, high particulate levels typically occur in the winter due to woodstove emissions and application of anti-skid materials to icy roads.

There are substantial regional and seasonal variations in the quantities and types of PM emitted to the air. These variations are not fully captured through emission estimates, especially when trying to include secondary particulates. Therefore, chemically speciated air quality monitoring data is a more effective means to identify the sources and levels of particulate matter for specific regions and locations.

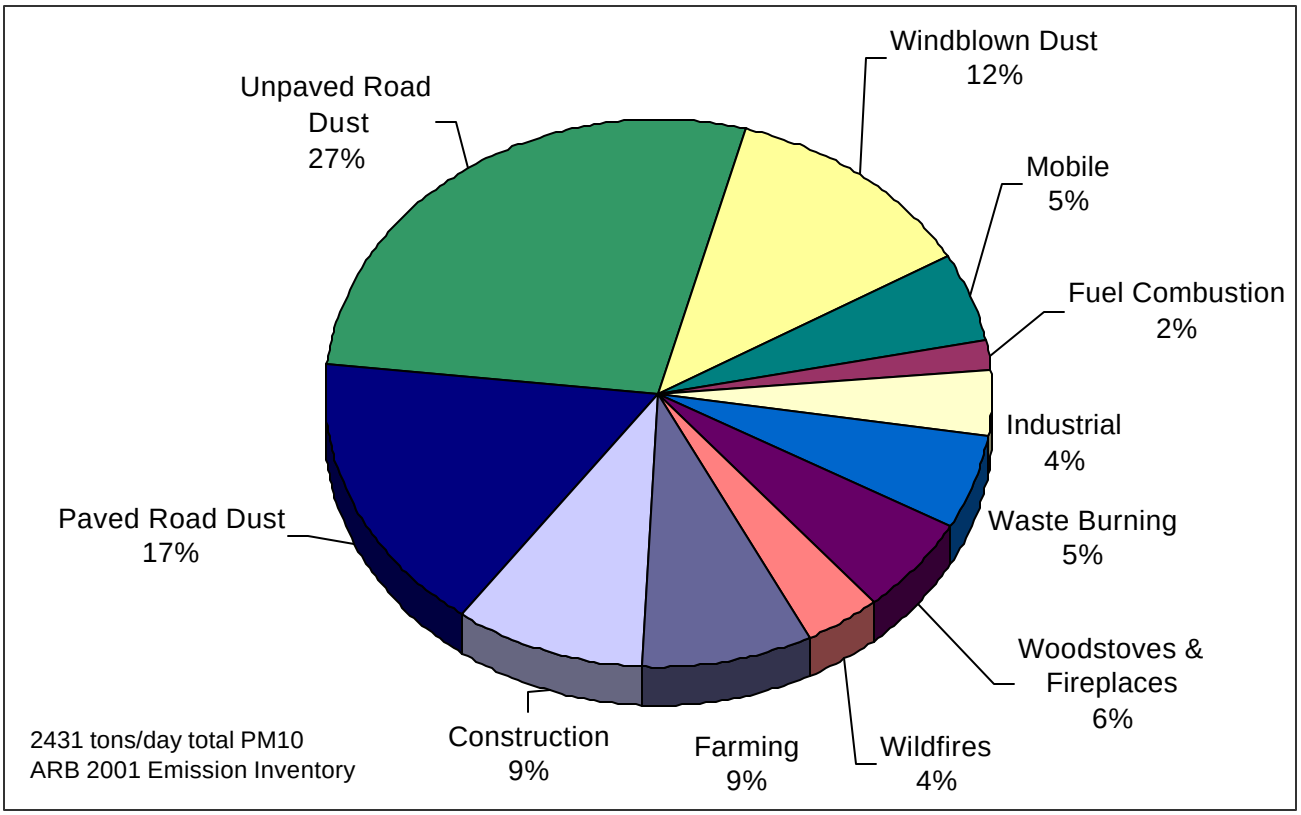


Figure 4.1. California Statewide PM10 Emission Inventory, Direct Particulate Emissions, 2001

4.5 References

Emission Inventory, 2001. California Air Resources Board. August 2001. Available via website at: http://www.arb.ca.gov/emisinv/eib.htm

Emission Inventory Procedure Manual, Volume III, Methods for Assessing Area Source Emissions. Air Resources Board. October 1997. Available via website at http://www.arb.ca.gov/emisinv/areasrc/areameth.htm

5. Measurement of Particulate Matter

5.1 Introduction

On December 9, 1982, the California Air Resources Board (Board or ARB) replaced the total particulate matter ambient air quality standard with a standard that focused on particles of a smaller diameter. The Board approved amendments to the California Code of Regulations, title 17, section 70200, which modified the definition of suspended particulate matter (PM) to specifically include particulate matter with an aerodynamic diameter of 10 microns or less (PM10), and established PM10 ambient air quality standards. The Board included general reference to a PM10 measurement method in the standard and directed staff to establish more specific criteria for PM10 sampling equipment. Method P, adopted by the Board in 1985, established the State method for ambient PM10 measurement. In 1986, the State identified the size selective inlet (SSI) high volume (hi-vol) PM10 sampler as the PM10 sampler satisfying the requirements of Method P.

In December 2000, the Board determined that the state ambient air quality standards for PM10 and particulate sulfates should be reviewed to ensure they are protective of public health. The Board asked staff to provide this review and any recommendations for changes to the standards by 2002.

This chapter addresses the measurement methods that are required to be used to determine compliance with the newly proposed PM standards. At this time, the ARB proposes to align the state and federal requirements for PM samplers by adopting the Federal Reference Methods (FRMs) for PM10 and PM2.5 as the state's method. The FRMs specify performance characteristics and operational requirements applicable to PM10 and PM2.5 monitoring methods, and for PM2.5, specify sampler design characteristics. The associated samplers meet the requirements specified in the methods. The method and associated sampler are designated as a reference method. We are not proposing to adopt Federal Equivalent Methods, per se, given their history of poor performance in California. Continuous samplers will be addressed, however, and will be incorporated as California Approved Samplers based on their performance in a recently concluded study in Bakersfield, CA.

The reference methods (FRMs) are traditional, filter-based sampling methods with laboratory weighing of the filters before and after sampling. The sampling and analytical methods are both labor-intensive. Each sample is collected during a 24-hour period, and one sample typically is taken every six days throughout the year at each monitoring station. There is a time-lag of days to weeks from the date of sampling to the time results are available.

Continuous monitoring for either PM10 or PM2.5 has many advantages over traditional filter based sampling techniques. A continuous method is an in-situ, automatic measurement method of suspended particle mass with varied averaging time (minutes to hours) that provides an instantaneous result. Their 24/7 sampling schedule will further our understanding of PM emission patterns and exposure, and can be used to enhance public health research into short-term peak exposure. They can provide more data for model validation, to aid in identifying air pollution source(s), and to reflect dispersion patterns. Official approval of continuous instruments/methods will promote further development of continuous samplers and potentially reduce the cost of the air monitoring network.

5.2 Existing Monitoring Requirements

5.2.1 State Method P for PM10

Method P (cited in title 17, California Code of Regulations, sections 70100 and 70200) describes the design and performance requirements for the PM10 sampler to be used to determine compliance with the state ambient air quality standards. Method P is contained in Appendix 5 part A of this document and describes the operating principle and design of the samplers, which are in turn specified in proposed section 70100.1.

An "ideal" sampler should be designed to determine the mass concentration of ambient particulate matter of a mean aerodynamic diameter of 10 micrometer (μm) or less (PM10) to simulate particle penetration of the human respiratory system as described by the Chan-Lippmann model (1980). According to this model, PM10 particles are small enough to enter the thoracic region of the human respiratory tract. An ideal sampler is the one that collects 50 percent (referred as D50) of all particles of 10 ± 1 μm aerodynamic diameter, and which collects a rapidly declining fraction of particles as their diameter increases and rapidly increasing fraction of particles of smaller particle diameters. Aerodynamic diameter is defined as the diameter of a spherical particle of a unit density with settling velocity equal to that of the particle in question. Particles with the same size and shape but with different densities will have different aerodynamic diameters.

Suspended particulate matter refers to atmospheric particles, solids, or liquids, except uncombined water. Dry, free-flowing particles should be sampled with the same efficiency as liquid, sticky particles. The expected mass concentrations of liquid particles should be within the limits of that predicted by the ideal sampler. For solid particles, the expected mass concentration should be no more than 5 percent above that obtained for liquid particles of the same size. The sampler must have less than 15 percent variation in the measurements produced by three collocated samplers.

Meteorology is one of several factors that can effect sampling efficiency. The performance of a PM10 sampler should be independent of wind speed to simulate human respiration. The inlet design and its internal configuration should be such that it shows no dependency on wind direction and wind speed when operated within 2 to 24 kilometers-per-hour wind speeds. To do this, the inlet should be omnidirectional, that is, the inlet should be symmetrical about the vertical axis.

A sampler must possess a sampling medium (filter) upon which the PM is collected without spattering and falling off. The sampler should be designed to hold and seal the filter in a horizontal direction so that the sample air is drawn uniformly downward through the filter to allow a uniform distribution of PM10 collected so as to permit subdivision of the filter for qualitative and quantitative analysis. Filters shall have a collection efficiency of more than 99 percent as measured by the dioctyl phthalate (DOP) test (ASTM-2986), with 0.3 μm particles at flow rates equal to the sampler's operating face velocity. Filters must have mechanical and chemical stability and be stable in a wide temperature range to allow a variety of qualitative and quantitative analyses. Filters must minimize artifacts, that is, should not react with the deposit and must not absorb contaminant gases. They must be non-hygroscopic, and have high chemical purity with alkalinity of <5 microequivalents/gram. The filters must be equilibrated prior to use at constant temperature and humidity conditions.

The sampler must possess an automatic flow control device which maintains a constant flow rate to within ± 10 percent of the recommended range for the sampler inlet over normal variations in line voltage and filter pressure drop during the sampling period. Change in flow velocity will result in change in nominal particle size collected. Therefore, it is important that

the flow rate through the inlet be maintained at a constant value that is as close as possible to the inlet design flow rate.

A timing/control device should be capable of starting and stopping the sampler during a sample collection period of 24 ± 1 hr (1,440 ± 60 min). An elapsed time meter, accurate to within 15 minutes, shall be used to measure sampling time. This meter is optional for samplers with continuous flow recorders if the sampling time measurement obtained by means of the recorder meets the ± 15 minutes accuracy specification. Using the total sampling time, the total volume of air sampled is determined. PM concentration is computed as the total mass of collected particles in PM10 size range divided by the volume of air sampled. The particulate matter concentration is expressed as micrograms per cubic meter ($\mu g/m^3$) corrected to standard conditions (760 torr and 25 C).

5.2.2 State Method for Sulfates

State regulations (section 70200 of title 17, California Code of Regulations) contain an independent ambient air quality standard for particulate sulfates. The measurement method for sulfates described in this standard is high-volume TSP sampling, with filter analysis by Turbidimetric Barium Sulfate Spectrometry, AIHL Method 61, or "[a]ny equivalent procedure which can be shown to the satisfaction of the Air Resources Board to give equivalent results at or near the level of the air quality standard."

The ARB's current measurement method for sulfates is MLD Method 033, which uses TSP sampling followed by ion chromatography. However, adsorption of SO_2 with subsequent chemical reactions on the filter surface, can, at sufficient concentrations, lead to gas-to-particle conversion, and can create positive artifacts on the filter. These can be minimized with the use of alkalinity-controlled filters which are provided for in the current PM10 network and PM10 Federal Reference Methods.

5.2.3 Federal Methods for PM10

Federal ambient measurement methods must be used to determine the attainment status of air basins nationwide. Federal Reference Methods (FRMs) use the measurement principles and specifications defined in U.S. EPA regulations. Provisions also allow for a Federal Equivalent Method (FEM) to be approved by the U.S. EPA and used for the same purpose. The requirements for an FRM for PM10 are described in Appendix M, Title 40, Code of Federal Regulations Part 50, 1997, and are provided here as Appendix 5 part B to this document. These requirements are, for the most part, the same as the California Method P. This is understandable, as Method P was adopted by the ARB in consultation with the U.S. EPA as that agency was preparing to propose methods for the NAAQS for PM10. A comparison of the two methods, item-by-item, is given in Appendix 5 part C. There are differences in a few specific requirements, which are highlighted in the table. The discussion below focuses only on those differences. The purpose of presenting this information is to highlight the need to update Method P.

- The FRM requires PM10 sampler to simulate particle penetration of the human respiratory system as described by the Chan-Lippmann (1980), penetration model. The D50 cut-point of the sampler is 10 μm with a tolerance of ± 0.5 μm, compared to the tolerance of ± 1.0 μm for Method P.
- The alkalinity of filter medium should be less than 2.5 microequivalents/gram for FRM as opposed to less than 5 microequivalents/gram for Method P. The filters should be equilibrated at constant relative humidity of between 20 percent and 45 percent ±5 percent for FRM instead of <50 percent relative humidity for Method P before weighing.

- The precision of collocated FRM samplers must be 5 $\mu g/m^3$ for PM concentration below 80 $\mu g/m^3$ and 7 percent for PM10 concentration above 80 $\mu g/m^3$ for FRM, as opposed to 15 percent for all concentrations for Method P.
- The FRM requires the air flow rate through the sampler remain stable over a 24-hour period, regardless of filter loading; the specific requirements are ±5 percent of the initial reading for the average flow, and ±10 percent of the initial flow rate for any instantaneous flow measurement. For Method P, the flow rate should be within 10 percent at all times.
- Typically, an analytical balance with a sensitivity of 0.1 mg is required for hi-vol samplers (flow rates >0.5 m^3/min, large filters). Lo-vol samplers (flow rates <0.5 m^3/min, smaller filters) require a more sensitive balance, which is not indicated in Method P.
- The particulate matter concentration is expressed as micrograms per cubic meter ($\mu g/m^3$) at local temperature and pressure (LTP) as opposed to standard temperature and pressure (STP, 760 torr and 25 C) for Method P.

The other major difference between the State and federal method is the designation of FEM test protocols, not included in Method P, although method equivalency is referred to and accepted in general terms in State regulations.

The differences between the FRMs and state-approved samplers can generally be attributed to advancements and improvements in sampler flow control and filter medium technology that occurred since Method P was established. The PM10 air monitoring network in California meets FRM requirements. Consequently, changing Method P to be consistent with the FRM will bring the criteria into line with both equipment and material specifications, and field and laboratory practices.

5.2.4 Federal Methods for PM2.5

The U.S. EPA promulgated rigorous design and performance specifications for its PM2.5 FRM samplers (40 CFR part 50, Appendix L; 40 CFR part 53, Subpart E; and 40 CFR part 58, Appendix A, all dated July 18, 1997. These are set forth in Appendix 5, parts D, E and F, respectively, of this document). Only measurements made using U.S. EPA-designated FRM samplers may be used to determine an area's compliance status with the PM2.5 NAAQS. The PM2.5 sampler is an adaptation of the PM10 lo-vol sampler that initially removes, by impaction, particles larger than PM10. Downstream lies a second impactor (Well Impactor Ninety-Six [WINS]) that reduces the cut point to 2.5 μ m.

The dimensions and materials of sampler components that come in contact with the sampled air stream (the first stage inlet, the downtube, the second stage separator [WINS], the upper filter holder, the filter cassette, and the filter support screen) are specified by design (40 CFR part 50, Appendix L, July 18, 1997). The design of the other components of the FRM sampler is left to manufacturers, as long as resulting samplers meet all the prescribed performance specifications.

Performance specifications include active monitoring of a number of operational characteristics of the samplers, including sampler volumetric flow, temperature, and pressure. The performance criteria specify strict requirements for controls that must be observed for sampler operations. These include sampling efficiency, accuracy, precision, sampling medium, flow controller, laboratory, calibration, and measurement procedures. The details of the performance criteria are given in Appendix L of 40 CFR part 50 (see Appendix 5 part D to this document).

The current network of PM2.5 samplers in California (more than 80) was funded almost entirely by the U.S. EPA following the adoption of the federal PM2.5 standard. The U.S. EPA

continues to provide operating funds for the network. The samplers' operation is governed by federal regulation.

The network of PM2.5 samplers has been operating in California since 1999. Additional speciation samplers will eventually supplement the FRM samplers and provide information about the composition of the particulate matter in the sample. Staff recommends adopting both the FRM sampler and the performance and operational requirements of the methods for the proposed State PM2.5 standard. Staff is not proposing to adopt the U.S. EPA's FEM criteria for PM2.5.

5.3 Available PM Sampling Methods

There are two fundamental methods commonly used to measure atmospheric PM10 and PM2.5 that are potentially useable in California. The first is a laboratory-based, gravimetric, or filter method, in which particles segregated by size are collected on a pre-weighed filter medium and weighed after sampling to determine PM mass. PM concentration is calculated by dividing the mass increase of the filter by the 24-hour total volume of air (at ambient conditions) that passed through the filter.

The second fundamental technique employs in-situ field samplers that are based on different operational principles, but that all operate continuously and produce real-time, hourly average concentrations. There are pros and cons to either type of sampler. Over the years, the staff has heard reports of the need for both types of samplers. Consequently, staff is proposing to incorporate the leaders in both types of samplers as part of this regulation as California Approved Samplers for PM.

New technology samplers may be added in future years, and others perhaps deleted, from the list of approved sampler as the situation warrants. The samplers proposed in this action have been demonstrated to have wide applicability, and have good agreement with standard methods. The intent of this rulemaking is to incorporate recent advances in sampler technology, align the state with federal samplers where possible, and incorporate samplers that can be used widely in California in areas with persistent high PM levels. The proposal does not support approving different samplers for every air basin or approving samplers that respond differently based on season of the year unless absolutely necessary.

The staff carefully considered the limitations of a wide variety of samplers in making the method proposals. Filter based methods can result in loss of PM during or after sampling, or formation of PM on the filter medium during sampling. Loss of semi-volatile chemical species such as atmospheric ammonium nitrate and organics may occur during and after sampling as particles move from the particle to the gas phase. The amount of particulate matter lost is dependent on the concentration and composition of the semivolatile components, and the handling and retention time on the sampler at the conclusion of the sample run. Loss of volatile chemical species can underestimate PM mass. PTFE (polytetrafluoroehtylene or Teflon®) filters can easily lose semi-volatile materials (a so-called negative artifact) (Eatough et al., 1993; Gundel et al., 1995). Therefore, regular sampling procedures now include steps to minimize these losses, including rapid removal of filters from the sampler, prompt storage in Petri dishes, transport in cool environments, expedited transport of filters from the field to the weigh rooms, and prompt extractions once the filters are weighed (Achtelik and Omand, 1998).

Adsorption of gases (such as SO_2) with subsequent chemical reactions with other gases on the filter surface, can, at sufficient concentrations, lead to gas-to-particle conversion, and can create positive artifacts on the filter. These can be reduced with the use of alkalinity-controlled filters and possibly the use of low-volume samplers.

Formation particles from gaseous species can lead to overestimation of PM mass (i.e., a positive artifact). Quartz filters can adsorb some gas-phase organics producing positive artifacts (Gundel et al., 1995; Turpin et al., 1994).

Operation of conventional filter-based samplers with laboratory gravimetric analysis is extremely time-consuming and labor intensive to produce a single mass measurement compared to real time, continuous samplers. Moreover, data are available only on a 24-hour average basis from conventional filter-based techniques. This limits using the data to investigate sub-24-hour health effects. The time lag inherent in data availability in conventional filter-based methods also precludes their use to provide the general public with timely warnings about episodic air pollution hazards. Filter-based systems can also have problems with particle loss during handling and transport, particularly when mass loading on the filter is high. These "sloughing" effects have been minimized by having strict operational protocols.

5.3.1 General Description of Gravimetric Methods

5.3.1.1 High-Volume Size Selective Inlet Sampler

The size selective inlet (SSI) sampler is described in Method P and is recognized by the U.S.EPA as an FRM. California identified the SSI in May 1986 as the PM10 sampler to be used for the State AAQS. The U.S. EPA identified it in 1987 as an FRM (U.S. EPA/ORD, 2000).

The high-volume (hi-vol) SSI sampler used in the State and federal PM10 networks consists basically of a PM10 inlet, an impactor, a flow control system recorder, and a pump. The automatic flow control system consists of either a mass flow controller or a volumetric flow controller, which controls the flow to 40 ft^3/min (at standard temperature and pressure). The flow rate through the impactor is used with the elapsed time to determine size of particle collected and the volume of air sampled. According to one manufacturer, its inlet has a cut-point of 9.7 μm in winds up to 22 miles per hour (36 kilometers per hour) (Thermo Andersen, Inc.).

PM10 hi-vol samples are collected on an 8x10 inch (20x25 cm) quartz filter that offers high collection efficiencies and is resistant to absorbing artifacts related to the collection of sulfates and nitrates. However, the quartz filter can under some circumstances adsorb organic vapor more readily than a PTFE filter leading to an overestimation of PM mass (Turpin et al., 1994). Volatile constituent losses during sampling and transport are known to exist; however, prompt sample removal can minimize these losses.

There are three versions of the SSI samplers currently designated as FRMs. The unit widely used in California, the SA-1200 (Sierra-Anderson 1200), is a single-stage fractionator with hinged design to facilitate oiling and cleaning of the impaction shim.

The SSI sampler provides a direct measurement of PM10 mass concentration. The large filter size provides two benefits. First, it increases the precision and accuracy of mass measurement, and second, it provides sufficient PM that can be analyzed for many of the primary constituents of interest.

5.3.1.2 Low-Volume Sampler

5.3.1.2.1 PM10

Low-volume (lo-vol) PM10 FRM samplers collect PM of a specific size range on a filter at a flow rate considerably less than for the hi-vol samplers. A lo-vol sampler consists of a PM10 inlet, an impactor, a pump, a flow rate controller, and a timer. Fundamentally, the operational

principles of the lo-vol and the hi-vol (SSI) samplers are the same. The differences occur with features such as the inlet size, flow rate, and filter size. These differences are discussed below. The flow rate in both cases is a critical feature of the instrument's ability to segregate particle sizes. Lo-vol samplers were not available at the time Method P was promulgated.

Federally approved lo-vol samplers (FRM) are equipped with either a flat or tilted PM10 inlet, as specified in Appendix L of 40 CFR Part 50 (Figures L-2 through L-19). They use small PTFE-coated fiber filters. The filters are chemically stable, and suitable for a wide temperature range. The sampler may have a manual or an automatic filter-changing mechanism. It must be able to measure 24-hour PM10 mass concentrations of at least 300 $\mu g/m^3$, while maintaining the operating flow rate within the specified limits.

FRM lo-vol samplers operate at a flow rate of 16.67 lpm. They use gravimetric means to determine ambient PM mass concentrations. The PTFE filters can be analyzed for elements, but are incompatible for analysis of some elements such as carbon, and do not provide a large enough sample to analyze all chemical constituents. The FRM lo-vol samplers have the same labor-intensive limitations of the hi-vol samplers which, in air monitoring networks, allows for only intermittent, 24-hour sampling.

5.3.1.2.2 PM2.5

PM2.5 FRM samplers are updated versions of the PM10 lo-vol FRM samplers. Sampler operation is controlled by a microprocessor. Downstream of the PM10 inlet is a Well Impactor Ninety Six (WINS) impactor, a filter medium, a timer, and a flow controller.

The WINS is a particle separator, where suspended PM2.5 is separated from the PM10. The WINS impactor is a single jet impactor, which impacts into a "well" holding a 37 mm glass fiber substrate impregnated with 1 mL of tetramethyltetraphenyltrisiloxane (silicone oil) single-compound diffusion oil. The WINS impactor inertially separates fine particles of an aerodynamic diameter of 2.5 μm or less from PM10. Larger particles are captured in the oil-impregnated substrate in the wells of the impactor, and the PM2.5 and smaller particles are collected on a 47-mm filter.

The filter is made of PTFE, and has a particle collection efficiency of greater than 99.7 percent. Prior to its use, the filter is equilibrated for 24 hours at a temperature range of 20 to 23 C and at a relative humidity in the range of 20 to 40 percent, and preweighed in a laboratory. The well filter needs to be cleaned regularly or the resulting deposited material can begin to affect the cut point of the inlet.

The sampler flow rate is 16.67 lpm (1.000 m^3/hr), measured by volumetric flow rate at the temperature and pressure of the sample air entering the inlet.

5.3.1.2.3 Dichotomous Sampler

The dichotomous sampler (dichot) is another low-volume sampler (draws air at 16.67 lpm). Ninety percent of the air (15.00 lpm) flows through the fine particulate filter, and the remaining 10 percent (1.67 lpm) flows through the coarse particulate filter.

The dichotomous sampler uses a virtual impactor (region of stagnant air) to segregate the air sample into two fractions. The virtual impactor particle separator accelerates the air sample through a nozzle and then deflects the air at a right angle. Most particles smaller than 2.5 micrometers (fine fraction) will follow the higher air flow path and collect on a fine particulate filter. Particles between 2.5 and 10 micrometers (coarse fraction) have sufficient inertia to impact into the chamber below the nozzle and are collected on a coarse particulate filter. Ten

percent of the sample air flows through the coarse particulate filter and because of this, approximately 1/10 of the fine particulate are collected on the coarse particulate filter.

The coarse and fine particulate filters are 37 mm in diameter and are mounted in plastic rings. The filters are weighed to calculate mass concentrations and, where appropriate, analyzed to determine the concentration of selected chemical elements.

PM2.5 measurements made using the dichot with its virtual separation technique do not meet U.S. EPA requirements for PM2.5 sampling in terms of the impactor type, filter size, and flow rate. The dichot utilizes a PM10 inlet similar to that in a a lo-vol sampler, but the flow rate is only 10 percent of the total flow rate, hence introducing a potential source of difference from the lo-vol PM10 sampler. Therefore, the use of this method for PM2.5 produces data that are not usable for compliance designation with the NAAQS for PM2.5. It is a reference sampler, however, for PM10.

5.3.2 General Description of Continuous Methods

Continuous methods produce hourly average PM concentration measurements in real time on a daily basis. In contrast to the intermittent sampling frequency of filter-based methods, continuous monitoring of atmospheric PM concentration has many advantages over periodic sampling; principally, the ability to assess air quality on those days missed by periodic samplers and finer time resolution. Arnold et al., (1992) collected daily 24-hour PM10 samples with an automated monitor and noted that 80 percent of the highest 10 daily concentrations in 1989 and 1990 were not encountered by the commonly used every-sixth-day sampling schedule.

Continuous methods provide data that can be accessed remotely in real time, and fill many needs for information that are very impractical, if not impossible, for typical filter-based methods. These include timely warnings about episodic air pollution hazards, enhanced public health research, air quality indexing, investigating diurnal variation and short term peak exposure, model evaluation, complaint investigation, data analyses, and specifying source impacts.

Several brands of continuous measurement technologies are commercially available. These include the tapered element oscillating microbalance (TEOM) including the SES, FDMS and differential configurations, beta attenuation monitors (BAM), and continuous ambient mass monitors (CAMM).

The CAMM is based on a measure of increasing pressure drop across a membrane filter with increasing particle loading on the filter. The analyzer consists of a diffusion dryer to remove particle-bound water and a filter tape to collect PM. Babich et al. (2000), using a Fluoropore membrane filter, measured PM using the CAMM in seven U.S. cities. Results of comparison to the Harvard Impactor (HI) yielded a good correlation ($r = 0.95$) and the average of CAMM-to-HI of 1.07. When the same sampler was tested by the ARB in 1998/1999 at the Bakersfield monitoring station during the winter months, it yielded a slope of 0.74 and correlation of 0.98 (Chung et al. (2001). Although well correlated, the sampler showed a bias of 25% compared to the FRM.

The BAM and the TEOM are the two most commonly used, commercially available, continuous PM analyzers in California. Both have been used to measure ambient PM10 and PM2.5 mass concentrations. These two technologies are designated FEMs for PM10. Because of their widespread use, a discussion of each is provided below.

5.3.2.1 Beta Attenuation Monitor

Several researchers (Jaklevic et al., 1981 and Kim et al., 1999) have used the measurement principle of absorption of beta radiation by PM on a filter as an indicator of particulate matter mass to provide real-time measurement of atmospheric PM. A Beta Attenuation Monitor (BAM) uses a lo-vol size selective inlet, a filter tape, a beta attenuation source and detector, a lo-vol flow controller, and a timer. The sampler contains a source of beta radiation (^{14}C or ^{85}Kr) and a detector to measure the beta absorption of PM accumulated on a filter. The filter material is a roll or cassette, which advances automatically on a time sequence. When particles are placed between the beta source and the detector, the beta rays are attenuated or absorbed by particles in their path. The difference in attenuation before and after the segment of the tape used to collect PM is attributed to the PM deposited on the filter. The reduction in beta ray intensity passing through the collected PM is assumed to be a function of the mass of material between the source and the detector. The degree of beta radiation attenuation is converted to PM concentration.

5.3.2.1.1 PM2.5 Results

Instrument inter-comparison studies of BAM PM2.5 units (a Met One model 1020) were conducted at Bakersfield (1998 – 1999) (Chung et al., 2001) and Fresno (1999 – 2000) (Appendix 5 part G). The results at Fresno were good (regression coefficient [R^2] of 0.97, slope of 1.07, intercept of 7.06). At Bakersfield (1998-1999), the PM2.5 BAM study compared one BAM equipped with a standard PM2.5 WINS inlet and one with sharp cut cyclone with the PM2.5 FRM. The comparison showed very good agreement (R^2 = 0.99 each, slopes of 0.91 and 0.97, and intercepts of 0.8 and 3.25, respectively). A minimum of 20 data pairs were gathered at each location (Chung et al., 2001). These studies were surveyed and considered in designing the 2001/2002 California Approved Sampler Study in Bakersfield, CA. The sampler comparison study is summarized in Appendix 5 part H. It included collocated Thermo Andersen BAMs (model FH 64 C14, here-after named And-BAM) and Met One BAMs (model 1020, here-after named Met-BAM) were configured to measure PM10 and PM2.5, and were operated in parallel with PM10 (SSI and Partisol) and PM2.5 (RAAS) FRMs. Using the results of the collocated samplers, precision was determined. The accuracy was evaluated using the lo-vol FRM as the reference. Two of each of the continuous PM2.5 samplers were fitted with sharp-cut cyclones to isolate the PM2.5 fraction from PM10.

The accuracy of the PM2.5 sampler data was determined by comparing 24-hr average data with the RAAS FRM (Table 4, Appendix 5 part H). There was excellent agreement between the continuous methods and the FRM. Regression analysis of the And-BAM and Met-BAM against the RAAS produced slopes of 1.03 and 1.03, respectively, correlations of 0.98 and 1.0 respectively, and intercepts of -1.32 and -1.58 respectively. A minimum of 102 data pairs was used in the analysis.

5.3.2.1.2 PM10 Results

Data comparing the SSI to the Met One BAM PM10 in Bakersfield in 1998-99 yielded limited but encouraging results (R^2 = 0.99 with slope of 1.01 and intercept of 1.90 $\mu g/m^3$ for eight data pairs (Chung et al., 2001). A study in Fresno in 2000, however, showed a weaker relationship (R^2 = 0.76 with slope of 1.11 and intercept of 23.24 $\mu g/m^3$ for 10 data pairs).

California Approved Sampler Study: In the ARB's 2001/2002 sampler comparison study in Bakersfield, (Appendix 5 part H) the And-BAM and Met-BAM PM10's compared favorably to the Partisol PM10 yielding slopes of 1.04 and 1.13 respectively, correlation values of 0.99 and 1.0 respectively, and intercepts of –2.50 and -1.65 respectively. Thirty data pairs or more were used for comparison.

The results of comparison of continuous samplers with each other and with PM2.5 and PM10 FRMs were compared with U.S. EPA PM10 class II test specifications (slope 1 ± 0.1, intercept ± 5 $\mu g/m^3$, correlation ≥ 0.97, and precision of 7%, Table 5, Appendix 5 part H). The rationale for selecting this test is provided in Appendix 5 part H.

Accuracy and precision values of these continuous samplers for both size cuts meet or exceed the proposed test specifications. Consequently, staff recommend that both the Thermo Andersen BAM (FH 64 C14 model), and the Met One BAM (1020 model) be approved for use to determine compliance with the State AAQS for PM2.5 and PM10.

5.3.2.1.3 Tapered Element Oscillating Microbalance

The Tapered Element Oscillating Microbalance (TEOM) is a low-volume sampler (16.67 lpm) that uses a mass sensor to measure airborne particle mass in real time. A TEOM consists of a size-selective inlet, flow splitter, sample filter, microbalance, flow controller timer, and software that makes the operation of the instrument fully automatic. In practice, the TEOM collects PM on a filter located on the top of a hollow, oscillating tapered tube. A small portion of the incoming air flow is drawn through the filter and through the tube. The oscillation frequency of the tapered inlet tube is inversely proportional to the mass of the sample that is deposited on the collection filter. The frequency decreases as mass accumulates on the filter, providing a direct measure of inertial mass. The typical measurement is collected over a period of ten minutes. The sample chamber is maintained above ambient temperatures (30-50C) to minimize the effect of temperature changes and thermal expansion of the tapered element that may affect the oscillation frequency, and to reduce particle-bound water (Patashnick et al., 1991).

Several studies (Allen et al. 1997, Chung et al. 2001, Cook et al. 1995) have shown that the concentration of PM10 or PM2.5 mass using the TEOM are often lower than PM measurements produced by other methods. Analysis of the constituents typically found in such cases indicates that this is caused by loss in the heated sample chamber of semi-volatile PM, such as ammonium nitrate and/or organic compounds. While most of the volatile components are found in the fine PM fraction (PM2.5), discrepancies between PM10 TEOM and reference samplers have also been observed.

Cook et al. (1995) studied the performance of the PM2.5 TEOM with a candidate FRM PM2.5 sampler at the Bakersfield monitoring station. The TEOM was operated at two temperatures, 30C and 50C, to determine the effect of temperature on measurement of PM mass. At both temperatures, the TEOM measured lower PM2.5 mass than the dichot or a single-stage lo-vol gravimetric sampler. PM concentrations from the TEOM at 50C were much lower (negative bias) compared to those at 30C, confirming the effect of temperature at that location on semi-volatile organics and nitrates from the filter.

In another study in Bakersfield, a PM10 SSI (an FRM) and TEOM sampler operated in parallel from November 1998 to February 1999 (Appendix 5 part G). This is a period when PM concentrations, and in particular volatile components, are high. The samplers correlated well, but again, the TEOM showed a significant negative bias (R^2 = 0.95 with slope of 0.37). At the Fresno Supersite, for the 1999 - 2000 sampling period, similar samplers correlated as well, and less bias relative to the FRM was seen (R^2 = 0.95 and slope of 0.83).

When PM2.5 was evaluated at the Fresno Supersite, the TEOM showed poor correlation and a very large negative bias with respect to the PM2.5 (R^2 = 0.31 with a slope of 0.42). At both sites, the TEOM underestimated PM mass concentration where semi-volatile components of PM are a significant component in both PM fractions.

In general, EPA-approved TEOMs have not performed well in the two areas of the State with the most persistent PM problem, the San Joaquin Valley Air Basin and the South Coast Air Basin. The TEOM's disadvantage is that the temperature necessary for the proper operation of the microbalance volatilizes a substantial component of the PM as part of the measurement process. This is more pronounced for measurement of fine PM fraction where volatile components make up a large part of the PM. The instrument manufacturer acknowledges this situation, and has developed several enhancements in recent years that address this problem.

A Sampler Equilibration System (SES) conditions the incoming sample air to lower humidity and temperature, to reduce losses of volatile species for the TEOM (Meyer et al., 2000). The SES utilizes a Nafion dryer which fits between the flow splitter that follows the size-selective inlet and the sensor unit. This unit was evaluated by the U.S. EPA as part of the Environmental Technology Verification program. The results were encouraging; however, the negative bias still existed.

Patashnick et al. (2001) subsequently developed a differential TEOM, which is a matched pair of TEOM sensors that operate at ambient temperature. The intent of the design is to measure continuous PM mass including volatile components. Downstream flow from a common size selective inlet is passed through a dryer. At the dryer exit, the flow is split with each branch passing through an electrostatic precipitator (ESP), each of which alternately turns off and on, out of phase with the other. The flow through each ESP is directed to a separate microbalance. The difference in the effective mass measured by the two microbalances is the sum of PM masses due to non-volatile and volatile component, from which filter artifacts and effective mass due to instrument sensitivity due to temperature changes is subtracted. However, this instrument is very expensive and is not applicable for routine field use.

In 2001, Rupprecht & Patashnick developed the commercial version of the differential system, the filter dynamics measurement system (FDMS). The FDMS uses the TEOM-SES system, a dryer, a switch, a purge filter conditioning unit, and a microbalance configured in a way to measure both volatile and non-volatile PM mass.

The sampling process consists of alternate sampling and purge cycles of 5-mintes each. During the first five minutes a PM is collected on the microbalance filter and mass is determined. The next five minutes, the collected PM is purged by air from the purge filter control unit from which PM is removed. Any decrease in filter mass observed during the purging cycle is attributed to the loss of volatile PM. The mass lost is added back to the mass measured during the first cycle (before purging) thus compensating for any loss during sampling. The reported mass concentration approximates the sum of nonvolatile and volatile PM.

California Approved Sampler Study: In the latest Bakersfield sampler study (ARB, 2001 – 2002) (Appendix 5 part H), comparison of the Partisol PM10 and the FDMS produced a slope (1.05), correlation (0.97), and an intercept (1.08) that agree with the proposed test specifications (Table 5, Appendix 5 part H). Thirty data pairs were used in the comparison.

Comparison of PM2.5 FDMS with the RAAS PM2.5 produced a slope (1.03), correlation (0.99), and an intercept (2.92) that are within the acceptable ranges described in the test specifications. At least 100 data pairs were used for the comparison.

Because of its performance, the staff recommends that the FDMS be approved for use to determine compliance with the State AAQS for PM10 and PM2.5.

5.4 Recommendations

The staff recommends the following monitoring methods:

- PM10 Monitoring Method – Adopt the Federal Reference Method (FRM) for PM10 as the method for California.
- PM2.5 Monitoring Method – Adopt the Federal Reference Method (FRM) for PM2.5 as the method for California.
- Continuous PM Samplers – Adopt those continuous PM samplers which have been found to be suitable for determining compliance with the state PM10 and PM2.5 AAQS, and designate them as California approved samplers (CAS).
- Sulfates Monitoring Method – Revise the sulfate monitoring method by deleting the current total suspended particle (TSP) sulfate method, ARB method MLD 033, and replacing it with the existing ARB method for PM10 sulfates, MLD 007.

A summary of measurement methods recommended by staff is provided below.

(a) Measurement of PM10 shall be accomplished by one of the two following techniques:

(1) A sampler that meets the requirements of the U.S. EPA Federal Reference Method(FRM) sampler for PM10, as specified in 40 CFR Part 50, Appendix M (1997), and which employs an inertial impactor; or,

(2) A sampler that has been demonstrated to the satisfaction of the Air Resources Board to produce measurements equivalent to the FRM.

At the time of Board consideration of this regulation, the following samplers are deemed to satisfy section (2) above, and staff is recommending their adoption by the Board: A continuous PM10 sampler as specified in Appendix 5 part H, Thermo Andersen BAM (model FH 62 C14), Met One BAM (model 1020), and Rupprecht & Patashnick FDMS (series 8500).

(b) Measurement of PM2.5 shall be accomplished by one of the following two techniques:

(1) A sampler which meets the requirements of the U.S. EPA Federal Reference Method (FRM) sampler for PM2.5, as specified in the 40 CFR Part 50, Appendix L (1997); with either a WINS impactor or a U.S. EPA approved very sharp cut cyclone (Federal Register, 2002) to isolate PM2.5 from PM10, or

(2) A sampler which has been demonstrated to the satisfaction of the Air Resources Board to produce measurements equivalent to the FRM.

At the time this regulation is considered by the Board, the following samplers are deemed to satisfy section (2) above, and staff is recommending their adoption by the Board: A continuous PM2.5 sampler as specified in Appendix 5 part H, with either a very sharp cut cyclone or a sharp cut cyclone: Thermo Andersen BAM (model FH 62 C14), Met One BAM (model 1020), and the Rupprecht & Patashnick FDMS (series 8500).

The rationale for these recommendations is given below

5.4.1 PM10

The Board needs to update the State PM10 method to reflect advancements and improvements in sampler technology. The FRM for PM10 is quite similar to Method P and includes requirements that are more up-to-date. The FRM sampler operation requirements

are currently used in the State and local air monitoring network. Incorporating the FRM into the AAQS will simply change legal requirements to reflect practice.

State methods have not been updated since the mid-1980's and need to recognize advances in both hi-vol and lo-vol measurement technology. Expanding the State method for PM monitoring to include all federal high-volume and low-volume samplers will allow the FRM samplers that perform well, but had not been approved for use in California, to be used for both State and federal regulatory activities. This will eliminate confusion of having two methods (State and federal) for the same parameter.

Allowing the use of the lo-vol PM10 method for the State PM10 standard offers the advantage of having one PM10 sampler produce data for both the State PM10 standard and as a possible element for a the potential federal PM coarse standard.

5.4.2 PM2.5

There are more than 80 PM2.5 FRM samplers currently in operation in California. Approval of the staff's proposal by the Board will incorporate these samplers into the State network and enhance their usefulness in the State. Adopting a continuous sampler technology that corresponds to a high degree with the FRM for PM2.5 has many programmatic and public health benefits to the State.

Staff proposes to maintain the provision in regulation for it to evaluate new measurement technologies and approve them pending a determination they are consistent with the applicable FRM.

5.4.3 Sulfates

The current sulfates standard uses MLD Method 033 for the sampling and analysis of sulfate by TSP. The ARB also has MLD Method 007 (latest version dated April 22, 2002) for measuring sulfates on PM10 filters, which uses PM10 sampling with sulfates analysis by ion chromatography. Staff recommends replacing the existing MLD Method 033 with MLD Method 007 as the proposed measurement method. This will provide the capability of using the PM10 sampling network for measuring sulfates, as well as allow for the minimization of any artifact-forming potential through the use of alkalinity-controlled filters.

5.5 Estimated Costs and Impacts

A substantial PM2.5 monitoring network, largely funded by the U.S. EPA, is now in place in California, and the U.S. EPA is in the process of implementing the last stages of the network build-up. A state-funded portion of the PM2.5 network also exists, primarily as continuous samplers. It was funded by the legislature in the late 1990's in response to Health & Safety Code section 39619.5, that requires the Board to conduct "monitoring of airborne fine particles smaller than 2.5 microns in diameter (PM 2.5)." The methods, samplers, and instruments proposed by staff include those used in, and so take full advantage of, these existing networks. Little extra expense is anticipated. However, approval of continuous samplers may result in requests for additional samplers, given their economies of operation.

The Board has had in place an extensive PM10 network for years. This proposal reaffirms the past samplers and incorporates the more recent federal samplers into the list of approved samplers for the State AAQS. The State's PM10 sampling method (Method P) has been the State ambient air monitoring method since 1985. The sampler of choice at that time was the hi-vol SSI. Implementing the proposed changes in Method P to explicitly acknowledge all FRM samplers would not incur any cost to either government entities or private businesses, because it would allow the operators of the PM samplers the option of retaining the current

sampling method. In fact, it would expand the number of acceptable samplers now in use to include heretofore unrecognized methods.

The recommended changes to Method P may result in cost saving to the extent that continuous PM10 monitoring methods are used in place of conventional filter-based methods. Continuous methods are less labor intensive than Method P and generate substantially more data. The staff cannot quantify any cost saving since it is unknown to what extent local agencies would choose to use to continuous samplers, instead of the conventional filter-based samplers used now.

Appendix 5 part I lists PM10 and PM2.5 monitoring sites and includes the various types of samplers that were being used at these sites in June 2001.

5.6 References

Achtelik, GH and Omand, J. Effects of environmental conditions on particulate nitrate stability during post sampling phase. 1998. MLD, CARB, PO Box 2815, Sacramento, CA 95812.

Allen G, Sioutas C. Koutrakis P, Reiss R, Lurmann FW, Roberts PT. Evaluation of TEOM method for measurement of ambient particulate mass in urban areas. J of Air Waste Management Assoc. 1997; 47:682-689.

Arnold S, Hague W, Pierce G, Sheetz R. The use of beta gauge monitors for PSI and every day SIP monitoring: an overview of the Denver experience. In: Chow JC; Ono DM. editors. PM10 standards and nontraditional particulate source controls, A&WMA/EPA international specialty conference. V. I; January; Scottsdale, AZ. Pittsburgh, PA, Air Waste Management Assoc.; 1992. p 12-23. (A&WMA transaction series no. 22)

Babich P, Wang P-Y, Allen G, Sioutas C, Koutrakis P. 2000. Development and evaluation of continuous ambient PM2.5 mass monitor. Aerosol Science and Tecnol. 32:309-324.

Chan TC and Lippman M. Experimental measurements and empirical modeling of the regional deposition of inhaled particles in humans. Amer Ind Hyg Assoc. J. 1980; 41:390-408.

Chung A, Chang DPY, Kleeman MJ, Perry KD, Cahill TA, Dutcher D, McDougall EM, Stroud K. Comparison of real-time instruments used to monitor airborne particulate matter. J. Air Waste Management Assoc., 2001, 51:109-120.

Cook JP, Oslund WE, Frank N. Evaluation of fine particulate (PM2.5) in an area of volatile constituents. In: Chow, J, Ono D, editor. Particulate matter: health and regulatory issues: proceedings of an international specialty conference; April; Pittsburgh, PA. Pittsburgh, PA: Air & Waste Management Assoc., 1995; 277- 296. (A&WMA publication VIP-49).

Eatough DJ, Wadsworth, A, Eatough DA, Crawford JW, Hansen LD, Lewis EA. 1993. A multi-system multi-channel diffusion denuder sampler for the determination of fine-particulate organic material in the atmosphere. Atmos. Environ. Part A 27:1213-1219.

Federal Register, Vol 67, No. 63, April 2, 2002, Page 15566.

Gundel LA, Lee VC, Mahanama KRR, Stevens RK, Daisey JM. 1995. Direct determination of the phase distribution of semivolatile polycyclic aromatic hydrocarbons using annular denuders. Atmos. Environ. 29:1719-1733.

Jaklevic JM, Gatti RC, Goulding FS, Loo BW. A beta gauge method applied to aerosol samples. Environ Sci Technology 1981; 15:680-684.

Kim YJ, Park SS, Lee KW, Chun KJ, Lee JY, Lim YS, Han JS. Development and testing of an automated beta gauge particulate sampler with filter cassette mechanism Air & Waste Management Assoc. 92nd Annual meeting. June 1999; 20-24. St Louis, Missouri.

Meyer MB, Patashnick H, Ambs JL, Rupprecht EG. Development of a sample equilibration system for the TEOM continuous PM monitor. J. Air Waste Management Assoc. 2000; 50:1345-1349.

Patashnick H, Rupprecht EG. 1991. Continuous PM10 measurements using the tapered element oscillating microbalance. J. Air Waste Management Assoc. 41(8):1079-1083.

Patashnick, H, Rupprecht, G, Ambs, JL, Meyer, MB. Development of reference standard for particulate matter in ambient air. . Aerosol Sci. and Technology. 2001; 34:42-45.

Thermo Andersen, Inc. Reference method PM10 size selective hi-vol air sampler; 500 Technology Court, Smyrna, GA 30082-9211.

Turpin BJ, Huntzicker JJ, Hering SV. 1994. Investigation of organic aerosol sampling artifacts in the Los Angeles Basin. Atmo. Environ. 28:3061-3071.

U.S. EPA/ORD. List of designated reference and equivalent methods; May 9, 2000.

6. Exposure to Particles

6.1 Area Designations

California has two ambient air quality standards for inhalable particulate matter (PM10), one with a 24-hour averaging time and a level of 50 $\mu g/m^3$, and an annual standard with a level of 30 $\mu g/m^3$. Health & Safety Code (H&SC) section 39607(e) requires the Air Resources Board (ARB) to establish and periodically review criteria for designating areas as nonattainment, attainment or unclassifiable. The last review was completed in November 2000 (ARB 2000).

The Board designates areas based on recent ambient air quality data. The data must satisfy specific siting and quality assurance procedures established by the U. S. Environmental Protection Agency (U.S. EPA) and adopted by the ARB. An area is designated nonattainment if ambient PM10 concentrations in that area violate either of the State standards at least once during the previous three calendar years.

The Board designates an area as attainment if air quality data show PM10 concentrations have not violated the standards during the three previous years. Regions without adequate PM10 monitoring data are designated unclassified.

Since highly irregular or infrequent events can lead to ambient PM10 concentrations over the 24-hour State standard level, such exceedances are not considered violations. The area designation criteria define three types of highly irregular or infrequent events: extreme concentration, exceptional concentration, and unusual concentration.

An extreme concentration event is identified through a statistical procedure that calculates the PM10 concentration that is expected to occur no more than once per year. This calculated PM10 concentration is the Expected Peak Day Concentration (EPDC). The EPDC is calculated for each monitoring site using PM10 concentration data collected during a three-year period. Unusual meteorology can cause an extreme concentration event. PM10 concentrations measuring higher than the EPDC are identified as extreme concentrations and are not considered violations of the standard. Included in these criteria is a once per year expected rate of exceedances (on average).

An exceptional concentration event is an identifiable event that causes an exceedance of the State standard, but that is beyond reasonable regulatory control. Examples include wildfires, severe windstorms, and seismic activity.

An unusual concentration event is an anomalous exceedance of the State standard that cannot be identified as an extreme concentration or an exceptional event. Unusual concentration events apply only to areas designated attainment or unclassified.

As specified in the California Code of Regulations, title 17, section 70302, the geographical extent of an area designated for PM10 usually is an entire air basin. However, the Board may designate smaller areas, based on a review of topography and meteorology, population density, location of emission sources, and existing political boundary lines.

As shown in Figure 6.1, virtually all of California violates the current State PM10 air quality standards. Only Lake County is designated attainment for the State standards. In the Mountain Counties Air Basin, Amador County and portions of Mariposa and Tuolumne Counties are unclassified. The Yosemite National Park, located in Tuolumne and Mariposa Counties is designated nonattainment.

Figure 6.1. **Area Designations for the State PM10 Ambient Air Quality Standards (Reference: Air Resources Board. Proposed area designations and maps. Staff report: Initial statement of reasons for proposed rulemaking, Sacramento, 2000).**

Air districts with areas designated nonattainment for the State PM10 standards are not required by State law to develop plans for attaining the State PM10 standards. However, H&SC sections 40001 and 40913 require such districts to adopt and enforce rules and regulations to expeditiously attain the PM10 standards.

6.2 Monitoring Network

California has a PM10 monitoring network with over 130 monitors statewide (Figure 6.2). At each monitoring site, High Volume Size Selective Inlet samplers collect 24-hour average PM10 samples, usually once every six days. The network is described in further detail in the State and Local Air Monitoring Network Plan (ARB 2000a).

To assess the nature and extent of PM2.5 pollution in the State, ARB and local air districts began deploying PM2.5 samplers in 1998. Currently we have placed federally-approved PM2.5 mass monitoring equipment (Federal Reference Method, FRM monitors) at 81 sites across California (Figure 6.3). FRM monitors collect 24-hour average PM2.5 samples, usually once every three days. More information about the PM2.5 network is contained in ARB's 2000 California Particulate Matter Monitoring Network Description (ARB 2000b).

California's dichotomous (dichot) sampler network has been in operation since 1983. Until recently the network comprised 20 sites collecting 24-hour samples every sixth day (Figure 6.4). The dichot sampler, or virtual impactor, uses a low-volume PM10 inlet followed by a virtual impactor which splits the air stream in two, separating particles into two fractions: fine particles (PM2.5) and coarse particles (PM2.5-10). The sum of the fine and coarse fractions provides a measure of total PM10. With the implementation of the federally required PM2.5 network, a number of dichot monitoring sites were closed by early 2000. With the exception of the dichot site in Fresno, the complete phase out of the dichot network occurred in December 2000.

Figure 6.2. PM10 Mass Monitoring Sites

Figure 6.3. PM2.5 FRM Mass Monitoring Sites

Figure 6.4. Dichotomous Sampler Sites

6.3 Characterization of Ambient Air Quality

6.3.1 Overview

This section describes the characteristics of PM10 and PM2.5 by each air basin in California, including: ambient concentrations; seasonal variations; identification of sources leading to the observed ambient particle concentrations; and the frequency distribution of the observed concentrations. To assess the spatial and temporal characteristics of PM10 and PM2.5 concentrations, we analyzed the following ambient air quality data:

- PM10 observations from Size Selective Inlet (SSI) monitors (from 1998 to 2000) (ARB 1998, ARB 2000a);
- PM2.5 information from the newly deployed Federal Reference Method (FRM) monitors, available only for two years (1999 and 2000) (ARB 2000b); and
- PM10-2.5 and PM2.5 data from dichotomous (dichot) samplers (from 1988 to 1999) (ARB 1998).

The data were extracted from the U.S. Environmental Protection Agency (U.S. EPA) Aerometric Information Retrieval System (AIRS) on November 15, 2001 (on May 18, 2001 for frequency distribution analysis).

For assessing the chemical composition of ambient PM10 and PM2.5, we reviewed information available from:

- The State's PM10 and PM2.5 monitoring networks;
- Two-Week Samplers (TWS) used in the California Children's Health Study (Taylor et al. 1998);
- The Interagency Monitoring of Protected Visual Environments (IMPROVE) program; and
- From special studies conducted in Imperial Valley, Sacramento, San Francisco Bay Area, San Joaquin Valley (1995 Integrated Monitoring Study, IMS95), Santa Barbara County, and South Coast Air Basin (1995 PM10 Enhancement Program, PTEP95).

6.3.1.1 PM10 and PM2.5 Ambient Concentrations

Table 6.1 lists maximum 24-hour and annual average PM10 concentrations in micrograms per cubic meter ($\mu g/m^3$) recorded in each air basin from 1998 through 2000 and PM2.5 concentrations from 1999 and 2000 – since federally approved PM2.5 monitors have been in operation in California. The revised State PM10 and the new State PM2.5 standards are proposed to be based on an annual arithmetic mean. The average of quarters is the preferred method for determining the arithmetic mean. For PM10 Table 6.1 includes two annual average statistics. The first is the annual geometric mean for the site with the highest value within the air basin. The annual geometric mean is used for the current State standard. The second statistic is the annual average of quarters with the highest annual average of quarters within the air basin, which is also the site with the highest annual geometric mean. We applied the data completeness criteria specified for the current State annual PM10 standard for estimating both annual means. For PM2.5 the table lists the annual average of quarters for the site with the highest value. This is also the annual averaging used for the current federal standard. In addition, Table 6.1 shows the number of days with measured PM10 concentrations over the current PM10 State standard and the number of days with concentrations over the proposed State PM10 standard. For PM2.5 the table lists the number of days with measured concentrations over the federal PM2.5 standard and the number of days with concentrations over the proposed State PM2.5 standard. No conclusions on attainment for the federal PM2.5 standards should be drawn from these data, since attainment designations will be based on three years of air quality data. No conclusions on attainment for the revised State PM10 and new PM2.5 standards should be drawn from these data either, since attainment designations are part of a separate regulatory process. Detailed data by monitoring station for each air basin are presented in Appendices 6-A and 6-B. We used SSI data for PM10 and FRM data for PM2.5 to generate these tables. Monitoring data are presently being evaluated for occurrences of exceptional events, consequently the data listed in Table 6.1 and Appendices 6-A and 6-B include data that in the future may be removed from AIRS.

Table 6.1. Maximum PM10 and PM2.5 Statistics per Air Basin

Air Basin	Year	PM10 (µg/m^3)[1,2]				PM2.5 (µg/m^3)[1,2,3]			
		Sampled Days over State Std.	Max. 24-hour Conc. (Std.=50)	Annual Average Concentrations[4]		Sampled Days over Federal Std.	Sampled Days over New State Std.	Max. 24-hour Conc. (Federal Std.=65) (New State Std.=25)	Annual Avg. Concentration
				Max. Annual Geometric Mean[5] (Current Std.=30)	Max. Annual Arithmetic Average of Quarters[7] (Revised Std.=20)				Max. Annual Average of Quarters[6,7] (Federal Std.=15) (New State Std.=12)
Great Basin Valleys	1998	29	1116	20	51				
	1999	7	514	14	15		2	41	Incomplete data
	2000	28	3059	19	39	2	9	68	Incomplete data
Lake County	1998		35	Incomplete data	Incomplete data				
	1999		43	Incomplete data	Incomplete data			15	Incomplete data
	2000		22	10	11			9	Incomplete data
Lake Tahoe	1998	2	59	20	23				
	1999		41	17	20			21	8
	2000		50	18	20			23	8
Mojave Desert	1998	8	165	14	16				
	1999	12	109	28	32		2	48	12
	2000	11	90	19	23		4	39	12
Mountain Counties	1998	11	92	23	25				
	1999	13	125	23	25	4	13	92	11
	2000	10	98	16	18		10	48	9
North Central Coast	1998	5	76	26	28				
	1999	9	103	28	31		2	31	Incomplete data
	2000	4	74	24	26		1	26	Incomplete data
North Coast	1998		50	20	21				
	1999	11	100	21	25		4	37	9
	2000	2	51	20	22			24	9
Northeast Plateau	1998	4	66	Incomplete data	Incomplete data				
	1999	12	100	22	26		2	40	8
	2000	10	80	18	23		2	38	9

Table 6.1. Maximum PM10 and PM2.5 Statistics per Air Basin (continuation)

Air Basin	Year	PM10 (µg/m^3)[1,2]				PM2.5 (µg/m^3)[1,2,3]			
		Sampled Days over State Std.	Max. 24-hour Conc. (Std.=50)	Annual Average Concentrations[4]		Sampled Days over Federal Std.	Sampled Days over New State Std.	Max. 24-hour Conc. (Federal Std.=65) (New State Std.=25)	Annual Avg. Concentration
				Max. Annual Geometric Mean[5] (Current Std.=30)	Max. Annual Arithmetic Average of Quarters[7] (Revised Std.=20)				Max. Annual Average of Quarters[6,7] (Federal Std.=15) (New State Std.=12)
Sacramento Valley	1998	17	130	23	29				
	1999	27	179	30	38	11	64	108	18
	2000	17	86	25	28	5	42	98	16
Salton Sea[8]	1998	53	176	59	66				
	1999	63	227	66	78		15	53	15
	2000	92	268	73	85	1	16	84	17
San Diego County	1998	18	89	39	43				
	1999	24	121	48	52		70	64	18
	2000	25	139	32	34	2	55	66	16
San Francisco Bay Area	1998	5	92	23	25				
	1999	12	114	25	29	4	48	91	Incomplete data
	2000	7	76	24	27	1	47	67	14
San Joaquin Valley	1998	51	160	32	40				
	1999	62	183	50	60	42	109	136	28
	2000	64	145	45	53	32	106	160	23
South Central Coast	1998	18	110	24	25				
	1999	18	90	28	31		11	65	14
	2000	24	113	26	31		14	55	15
South Coast	1998	59	116	43	50				
	1999	55	183	65	72	12	108	121	31
	2000	83	139	55	59	20	189	120	28

Notes for Table 6.1.

(1) Monitoring data are presently being evaluated for occurrences of exceptional events, consequently the table includes data that in the future may be identified as recorded during an exceptional event and be removed from consideration.

(2) No conclusions on attainment for the revised State PM10 and new PM2.5 standards should be drawn from these data, since attainment designations are part of a separate regulatory process.

(3) No conclusions on attainment for the federal PM2.5 standard should be drawn from these data, since attainment designations will be based on three years of data.

(4) The same number of ambient PM10 observations is used for estimating each annual average for PM10; the only difference among the annual averages is the averaging method used.

(5) For PM10 the table lists the monitoring site with the highest estimated annual geometric mean in the corresponding air basin. The same site also has the highest estimated annual arithmetic average of quarters.

(6) For PM2.5 the table lists the monitoring site with the highest estimated annual average of quarters in the corresponding air basin.

(7) The annual average of quarters for each monitor is estimated by first averaging the ambient 24-hour PM measurements to obtain quarterly means and then averaging the estimated quarterly means (as generally described in Appendix N to 40 CFR Part 50: National Ambient Air Quality Standards for Particulate Matter; Final Rule. July 18, 1997).

(8) Salton Sea PM10 statistics exclude data from the Calexico-East Site, because data from this site do not represent widespread exposure.

As shown in Table 6.1, with the exception of Lake County, all air basins exceed the State 24-hour PM10 standard of 50 $\mu g/m^3$. The Great Basin Valleys Air Basin recorded the three highest 24-hour PM10 levels in the State, 3059 $\mu g/m^3$ in 2000, 1116 $\mu g/m^3$ in 1998, and 514 $\mu g/m^3$ in 1999. Four air basins exceeded the current annual PM10 State standard of 30 $\mu g/m^3$: Salton Sea, San Diego, San Joaquin Valley, and South Coast. The Salton Sea Air Basin had the highest PM10 annual geometric means - 73 $\mu g/m^3$ in 2000 and 66 $\mu g/m^3$ in 1999 - followed by the South Coast Air Basin - 65 $\mu g/m^3$ in 1999. In air basins exceeding both current State PM10 standards, the ratios of maximum 24-hour and annual concentrations compared to the respective standards suggest that the 24-hour State standard is controlling (Table 6.2). As shown in Table 6.1, with the exception of Lake County, all air basins exceeded the proposed annual PM10 State standard of 20 $\mu g/m^3$. Currently, eight air basins (Great Basin Valleys, Mountain Counties, Sacramento Valley, Salton Sea, San Diego County, San Francisco Bay Area, San Joaquin Valley, and South Coast) recorded 24-hour concentrations over the federal PM2.5 standard. Values over the 24-hour standard in Mountain Counties in 1999 may have been caused by extensive wildfires. With the exception of Great Basin Valleys, Mountain Counties and San Francisco Bay Area, the other five air basins also recorded maximum annual averages above the federal annual PM2.5 standard. In comparison, all air basins - with the exception of Lake County and Lake Tahoe - recorded 24-hour concentrations over the proposed State PM2.5 standard of 25 $\mu g/m^3$ and seven air basins (Sacramento Valley, Salton Sea, San Diego County, San Francisco Bay Area, San Joaquin Valley, South Central Coast and South Coast) also had maximum annual averages above the proposed State annual PM2.5 standard.

Table 6.2. Ratios of yearly maximum 24-hour PM10 and annual average concentrations compared to the respective State standards.

Air Basin	Year	Max. 24-hour/Std. (Std. = 50 $\mu g/m^3$)	Max. Annual Avg[(1)]/Std. (Std. = 30 $\mu g/m^3$)
Salton Sea	1998	3.5	1.8
	1999	4.5	2.1
	2000	5.4	3.1
San Diego	1998	1.8	1.3
	1999	2.4	1.6
	2000	2.8	1.1
San Joaquin Valley	1998	3.2	1.1
	1999	3.7	1.7
	2000	2.9	1.5
South Coast	1998	2.3	1.6
	1999	3.7	2.2
	2000	2.8	1.8

(1) For the air quality monitoring site with the highest estimated annual geometric mean for PM10 in the corresponding air basin.

Background sites are intended to quantify regionally representative PM concentrations for sites located away from populated areas and other significant emission sources. Background concentrations are defined as concentrations that would be observed in the absence of anthropogenic emissions of PM and the aerosol particles formed from anthropogenic precursor emissions of VOC, NO_X and SO_X. However, it is very difficult to find true background sites. Depending on the season and meteorological conditions, even the monitoring sites located in pristine areas can be influenced by anthropogenic emissions and transport. This in turn may lead to higher annual average PM concentrations. Annual average PM concentrations from the IMPROVE network are presented in Table 6.3 (aggregated over a three year period, March 1996 to February 1999) (Malm et al. 2000).

Table 6.3. Annual Average PM10 and PM2.5 Concentrations at IMPROVE Sites

Site	Annual Average PM10 (ug/m^3)	Annual Average PM2.5 (ug/m^3)
Lassen Volcanic National Park	5.06	2.68
Pinnacles National Monument	10.97	4.55
Point Reyes National Seashore	12.42	4.01
Redwood National Park	7.45	2.44
San Gorgonio Wilderness Area	13.72	7.20
Sequoia National Park	18.64	8.86
Yosemite	8.52	4.33

As part of California's PM2.5 program, three locations have been selected to measure background PM2.5 concentrations: Point Reyes National Seashore in Northern California, and San Rafael Wilderness and San Nicholas Island in Southern California. These sites are located away from populated areas and other significant sources of particulate and particulate precursor emissions. The sites have been in operation since December 2000. Data from these sites are not yet available. However, data obtained from the IMPROVE program for Point Reyes from March 1996 through February 1999 indicate that the annual average concentrations were 4.01 $\mu g/m^3$ for PM2.5 and 12.42 $\mu g/m^3$ for PM10. PM10 and PM2.5 data collected at San Nicolas Island as part of PTEP95 program show a PM10 annual average of 18.7 $\mu g/m^3$ and a PM2.5 annual average of 6.82 $\mu g/m^3$ (Kim et al. 2000). In addition, as part of California Regional PM10/PM2.5 Study (CRPAQS), we will be evaluating data from several sites, including one site in a desert locality and one site in a forested area for determining regional background PM concentrations.

6.3.1.2 Historical Trends

We determined PM concentration trends using dichot PM2.5, PM10-2.5, and PM10 data collected from 1988 through 1999 at selected urban sites. The dichot sampler uses a low-volume PM10 inlet followed by a virtual impactor, which splits ambient air samples into fine (PM2.5) and coarse (PM10-2.5) particle fractions. The sum of these two fractions provides a measure of total PM10. We estimated annual arithmetic mean concentrations, by averaging quarterly (January through March, April through June, July through September, and October through December) arithmetic means. Data illustrated in Figure 6.5 indicate that, overall, the annual means of PM2.5 decreased until 1998, increasing in 1999 at most sites. Monthly rainfall

data obtained from National Weather Service stations indicate 1999 was a much drier year than 1997 and 1998, contributing to higher particulate matter concentrations in 1999. As shown in Figures 6.6 and 6.7, the coarse PM10-2.5 and the PM10 annual means exhibited similar trends, with a slightly less pronounced decrease in coarse fraction concentrations in the 1994 to 1999 period.

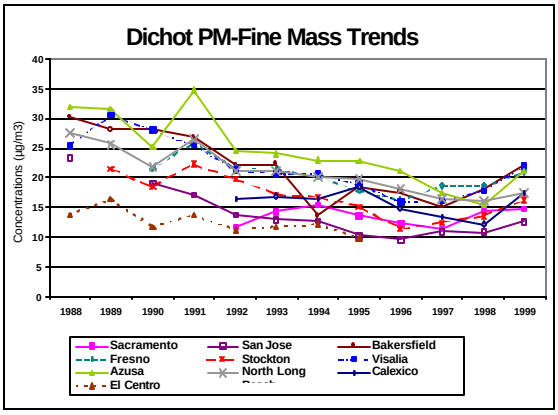


Figure 6.5. Annual trends in PM2.5 concentrations

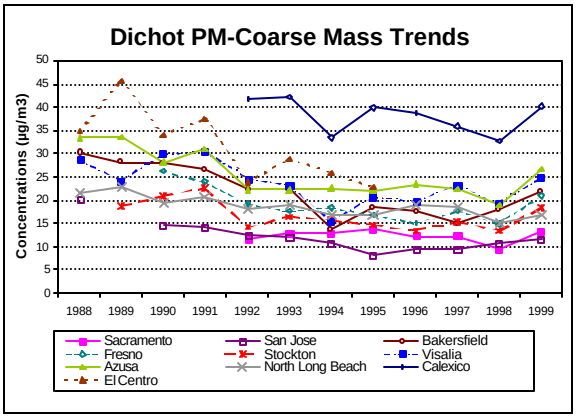


Figure 6.6. Annual trends in Coarse PM concentrations

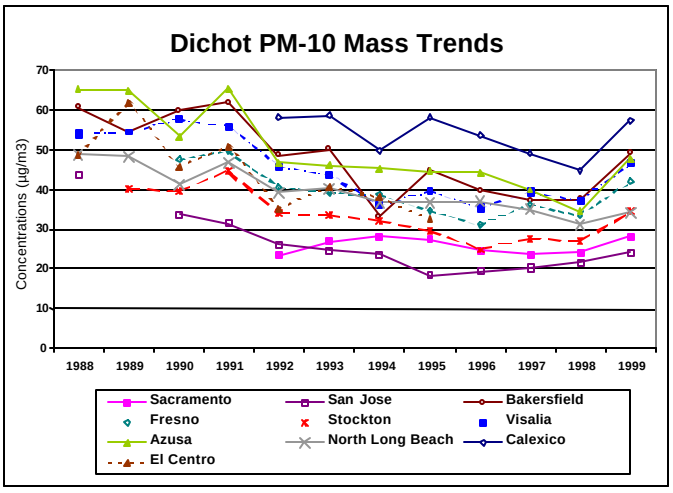


Figure 6.7. Annual trends in PM10 concentrations

6.3.1.3 Seasonality

Plots showing seasonal variation in ambient particulate matter concentrations were generated using FRM data for PM2.5 and SSI data for PM10. These seasonality plots are included in the subsections of this chapter describing particulate matter air quality in each air basin. The data represent the peak 24-hour PM concentration per month for each size fraction. In some cases PM2.5 is higher than PM10. This can occur for two reasons. First, the measurements are made on two different sampling systems and therefore have different levels of accuracy, precision,

and uncertainty. Second, in some cases peak PM10 and PM2.5 concentrations do not occur on the same day. The plots were generated to provide an understanding of the seasonality of peak concentrations, not to compare specific PM10/PM2.5 concentrations to each other.

In general, there are a number of air basins which exhibit strong seasonal patterns. Areas such as Sacramento, the San Joaquin Valley, and the San Francisco Bay Area record much higher PM2.5 and PM10 concentrations in the winter months (Figure 6.8). During this time of year, the PM2.5 size fraction drives the particulate matter concentrations.

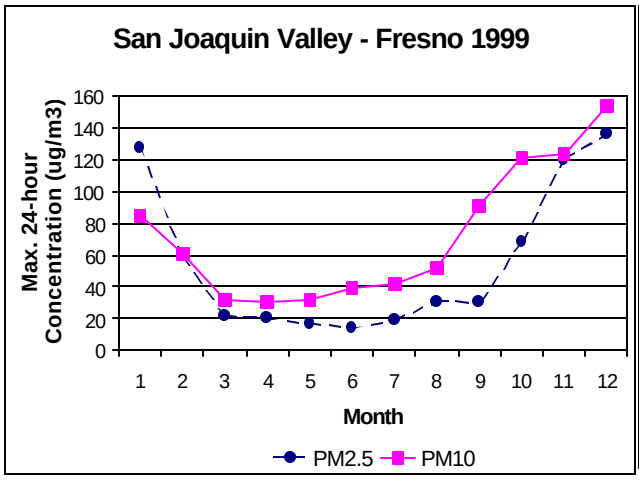


Figure 6.8. Monthly variation in maximum 24-hour PM10 and PM2.5 concentrations

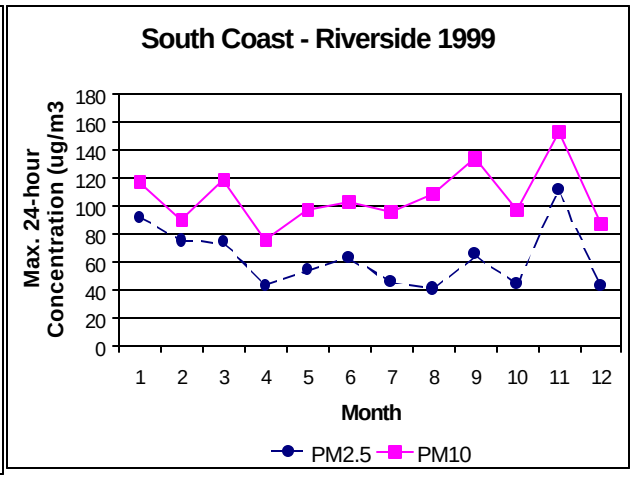


Figure 6.9. Monthly variation in maximum 24-hour PM10 and PM2.5 concentrations

Other areas such as the South Coast have a much more uniform distribution (Figure 6.9). In the South Coast, PM10 and PM2.5 concentrations remain high throughout the year.

In yet other areas there are specific episodic exceedances due to fugitive dust events (Great Basin Valleys, Salton Sea), or fires (Mountain Counties).

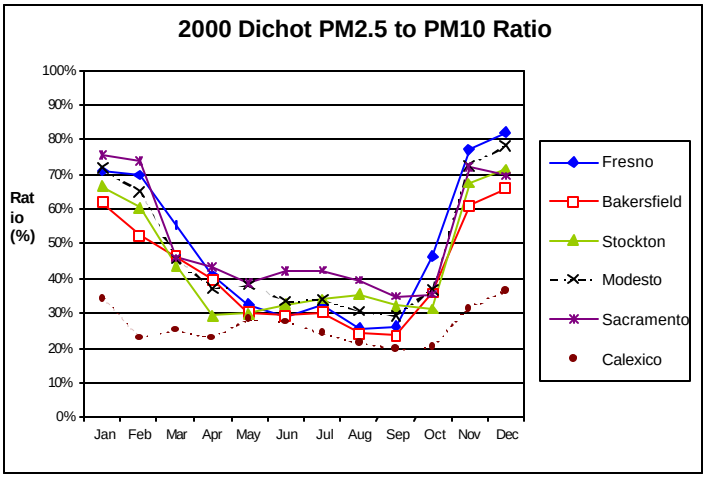


Figure 6.10. Monthly variation in the PM2.5 to PM10 ratio

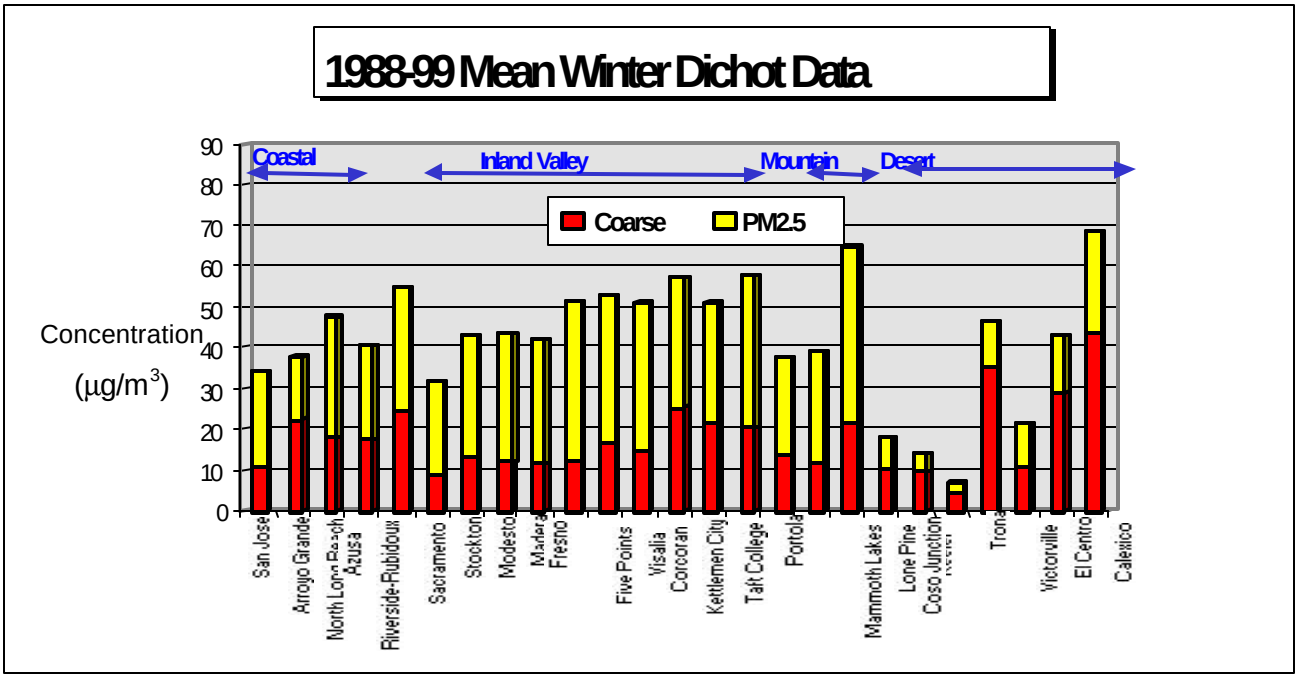
We used data collected with dichot samplers from 1998 to 2000 to estimate the ratios of PM2.5 to PM10 concentrations (e.g., Table 6.4). Figure 6.10 shows that in general, the average PM2.5 portion of PM10 was higher during the winter (November to February) than during the rest of the year (March to October). These seasonal differences were most pronounced in the San Joaquin Valley (75% in the winter and 38% during the rest of the year) and least prominent in the Mojave Desert (46% in winter and 39% during the rest of the year). No seasonal differences were apparent in the Great Basin Valleys Air Basin (see data for the Coso Junction monitoring site in Table 6.4)

Table 6.4. Ratios of monthly average PM2.5 to PM10 mass with a reported minimum and maximum at each location, 1999. Ratios were estimated using dichot data.

1999	Portola	Sacramento	San Jose	Modesto	Fresno	Bakersfield	Coso Jct	Victorville	Long Beach	Calexico
Jan	0.71	0.78	0.65	0.73	0.82	0.71	0.27	0.49	0.62	0.41
Feb	0.59	0.60	0.51	0.68	0.68	0.58	0.43	0.48	0.54	0.41
Mar	0.54	0.61	0.39	0.51	0.53	0.44	0.30	0.38	0.45	0.31
Apr	0.49	0.49	0.42	0.40	0.47	0.39	0.36	0.56	0.45	--
May	0.32	0.39	0.34	0.31	0.33	0.28	0.38	0.36	0.39	--
Jun	0.30	0.38	0.39	0.32	0.35	0.27	0.34	0.37	0.47	0.30
Jul	0.28	0.44	0.52	0.34	0.35	0.26	0.39	0.39	0.45	0.24
Aug	0.43	0.48	0.52	0.38	0.31	0.28	0.30	0.34	0.46	0.24
Sep	0.32	0.41	0.52	0.37	0.28	0.31	0.38	0.34	0.45	0.21
Oct	0.40	0.42	0.51	0.34	0.38	0.34	0.30	0.34	0.49	0.26
Nov	0.74	0.66	0.60	0.63	0.68	0.58	0.36	0.43	0.61	0.26
Dec	0.71	0.45	0.59	0.72	0.67	0.60	0.32	0.45	0.58	0.32
Max Ratio	0.91	0.86	0.80	0.85	0.88	0.82	0.73	0.75	0.96	0.59
Min Ratio	0.19	0.13	0.30	0.11	0.19	0.16	0.14	0.20	0.30	0.17

Seasonal variations in meteorological conditions and in the activity of emissions sources cause the size, composition, and concentration of particulate matter to vary by region and by season. Because air typically flows inland from the Pacific Ocean, the percentage of days exceeding the California 24-hour standard is generally lower along the coast than in inland areas. As the air parcel moves downwind across areas with significant anthropogenic activities, fresh emissions and gas-to particle conversion cause PM concentrations to increase with distance, for example, along the North Long Beach, Azusa, Riverside-Rubidoux corridor. PM2.5 concentrations are highest during the winter months (November to February). Cool temperatures, low inversion layers, and humid conditions favor the formation of secondary nitrate and sulfate particles, which are found predominantly in the fine fraction. Residential wood combustion also leads to higher PM2.5 concentrations during the winter. From 1988 to 1999, in the San Joaquin Valley, 97% of the four highest 24-hour PM2.5 concentrations and 68% of the four highest PM10 concentrations occurred during the winter. In the South Coast 53% of the four highest PM2.5 and 58% of the highest PM10 levels occurred in the winter season. Soil dust is the dominant contributor to PM10 in the summer. A desert environment generally has low PM concentrations, but on occasion high winds cause significant increases in dust.

Figure 6.11. Winter average PM10 and PM2.5 concentrations by region



6.3.1.4 Source Apportionment

Chemical Mass Balance (CMB) models are used to establish which sources and how much of their emissions contribute to ambient particulate matter concentrations and composition. CMB models use chemical composition data from ambient PM samples and from emission sources. These data are often collected during special source attribution studies. The quality of source apportionment results depends on the adequacy of the chemical markers used for each potential source and of the ambient chemical composition data used in the analysis, as well as the inclusion of appropriate sources. The source attribution data presented in this report was derived from a variety of studies with differing degrees of chemical speciation. In general, however, the source categories can be interpreted in the following manner. The road and other dust, wood smoke, cooking, vehicle exhaust, and construction categories represent sources which directly emit particles. Road and other dust represents the combination of mechanically disturbed soil (paved and unpaved roads, agricultural activities) and wind-blown dust. Wood smoke generally represents residential wood combustion, but may also include combustion from other biomass burning such as agricultural or prescribed burning and cooking. The vehicle exhaust category represents direct motor vehicle exhaust particles from both gasoline and diesel vehicles. Construction reflects construction and demolition activities. Ammonium nitrate and ammonium sulfate represent secondary species (i.e., they form in the atmosphere from the emissions of nitrogen oxides (NO_X), sulfur oxides (SO_X), and ammonia). Combustion sources such as motor vehicles and stationary sources contribute to the NO_X that forms ammonium nitrate. Mobile sources such as diesel vehicles, locomotives, and ships and stationary combustion sources emit the SO_x that forms ammonium sulfate. Ammonia sources include animal feedlots, fertilizers, and motor vehicles. The other carbon sources category reflects organic sources not included in the source attribution models, such as natural gas combustion, as well as secondary organic carbon formation. The unidentified category represents the mass that cannot be accounted for by the identified source categories. It can include particle-bound water, as well as other unidentified sources. Figure 6.12 illustrates the results of the PM2.5

source apportionment analysis conducted for Fresno using ambient air samples collected January 1st through 4th, of 1996.

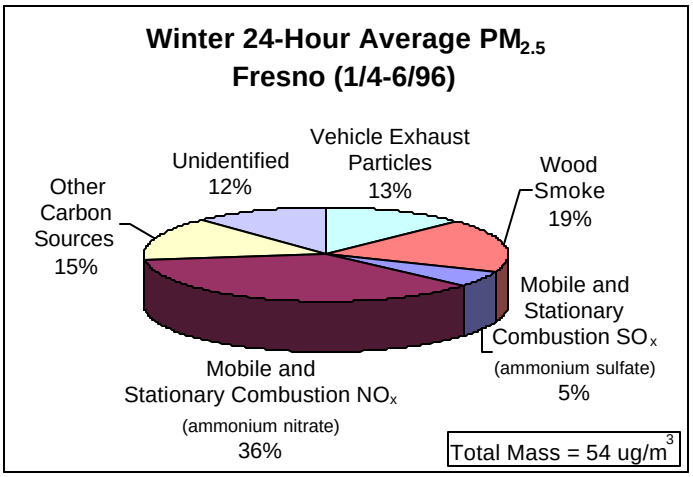


Figure 6.12. Source categories contributing to PM2.5 in Fresno during the winter.

For the area represented by the Children's Health Study and by the IMPROVE network, specific source apportionment analysis has not been conducted. Instead, the primary chemical components of ambient PM2.5 are shown. As discussed above, nitrate and sulfate are secondary species. Soil, elemental carbon, as well as much of the organic carbon are primary species.

New data that is becoming available will allow for better, and more consistent source apportionment. For example, the PM2.5 speciation samplers measure the species needed for source apportionment analysis on the same sampler. Previously, ions and carbon were measured on the SSI, and elements on the dichotomous samplers, requiring data from different samplers to be combined for a complete picture. Data from special studies such as the California Regional PM10/PM2.5 Air Quality Study (CRPAQS) and the 1997 Southern California Ozone Study (SCOS97) will also provide more detailed speciation data for source apportionment analysis.

As mentioned earlier, the size, concentration, and chemical composition of PM vary by region and by season. A number of areas exhibit strong seasonal patterns. Other areas have a much more uniform distribution – PM concentrations remain high throughout the year.

In the San Joaquin Valley, the San Francisco Bay Area, and the Sacramento area, there is a strong seasonal variation in PM, with higher PM10 and PM2.5 concentrations in the fall and winter months. The higher concentrations are due to increased activity for some emissions sources and meteorological conditions that are conducive to the build-up of PM. During the winter, the PM2.5 size fraction drives the PM concentrations, and the major contributor to high levels of ambient PM2.5 is the secondary formation of PM caused by the reaction of NOx and ammonia form ammonium nitrate. Emissions from wood smoke – mostly from fireplaces and wood stoves – vehicle exhaust particles, and other carbon sources also contribute significantly to PM2.5 levels. The San Joaquin Valley also records high PM10 levels during the fall. During this season, the coarse fraction (PM10-2.5) – mostly road and agricultural dust - drives the PM concentrations.

In the South Coast region, PM10 and PM2.5 concentrations remain high throughout the year. The more uniform activity patterns of emission sources, as well as less variable weather patterns, leads to this more uniform concentration pattern. On an annual basis, dust from roads and construction is the major contributor to ambient PM10. NOx emitted from mobile and

stationary combustion sources, combined with ammonia, contributes significantly. Vehicle exhaust particles and other carbon sources also contribute.

In other areas, high PM can be more episodic than seasonal. For example, in Owens Lake in the Great Basin Valleys Air Basin, episodic fugitive dust events lead to very high PM10 levels, with soil dust as the major contributor to ambient PM10. In Imperial Valley in the Salton Sea Air Basin, fugitive dust and dust from roads and farming operations lead to high PM10 levels.

Background sites often exhibit very different profiles. In national parks like Redwoods, Lake Tahoe, and Pinnacles, organic carbon is the major component of annual average fine particulate matter (Sisler 1996). Figures 6.13 and 6.14 show the PM2.5 chemical composition at two of the PM2.5 program background sites. Data for Point Reyes are from analysis of ambient air collected in 1995 as part of the IMPROVE program. Composition data for San Nicholas Island were collected as part of the PTEP95 study. The data show sea salt, sulfate, and organic carbon are the largest contributors to PM2.5 at both sites. Organic carbon particles in background sites originate from natural combustion processes such as wild fires and organic aerosols formed from VOC emissions from vegetation. In addition, natural emissions of gaseous sulfur compounds contribute to the background sulfate component.

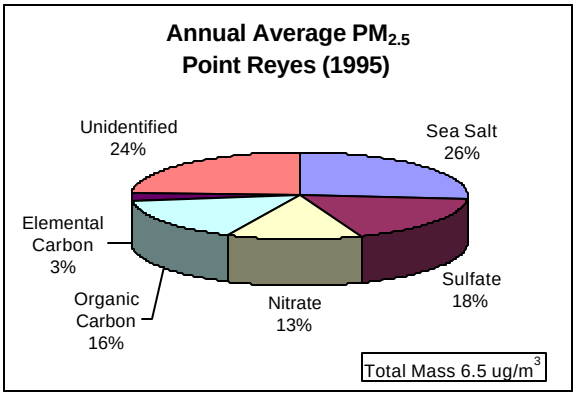


Figure 6.13. Chemical composition of ambient PM2.5 in the Point Reyes National Seashore

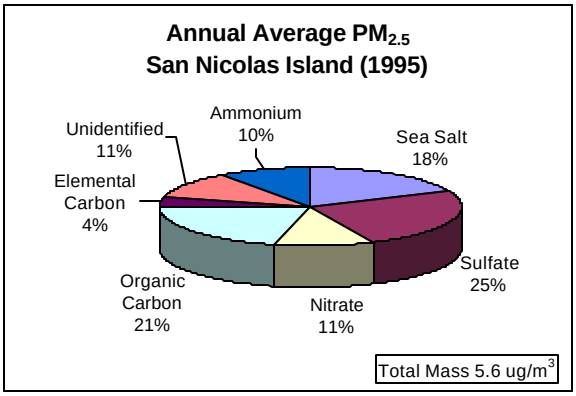


Figure 6.14. Chemical composition of ambient PM2.5 in San Nicolas Island

6.3.1.5 Frequency of Measured PM10 and PM2.5 Concentrations

We generated histograms that represent the frequency distribution of observed particulate matter concentrations at all sites within an air basin. Separate histograms were plotted for 1998-2000 for PM10 (Appendices 6-C1 to 6-C3) and 1999-2000 for PM2.5 observations (Appendices 6-D1 and 6-D2). As with previous analyses, the PM10 data is derived from the SSI monitor and the PM2.5 data from the FRM monitor. These data were obtained from AIRS on May 18, 2001. Figures 6.15 and 6.16 show the PM2.5 histograms generated for the North Coast Air Basin and the South Coast Air Basin, respectively. Each bar represents the number of observations within the specified range. For example, for PM2.5 the first bar is the number of observations between 0and 5 $\mu g/m^3$, the second between 5and 10 $\mu g/m^3$ and so on. The histograms provide information on the frequency of high concentrations within each air basin, as well as the most frequent, or predominant concentration levels, and can provide insight into the impact of setting the standards at varying levels.

In many of the air basins, 80% of the PM10 observations are below 30 to 35 μg/m^3. However, other air basins, such as the San Joaquin Valley and the South Coast, have significant numbers of observations that are much higher. In these areas, the 80% cumulative frequency is not reached until about 70 μg/m^3. For PM2.5, in many of the air basins, most of the observations are below 10 to 20 μg/m^3 (in the North Coast Air Basin example shown on Figure 6.15, all observations are below 30 μg/m^3). However, as with PM10, areas such as the San Joaquin Valley and the South Coast (Figure 6.16), exhibit a distribution such that the 80% cumulative frequency is reached at 35 to 40 μg/m^3.

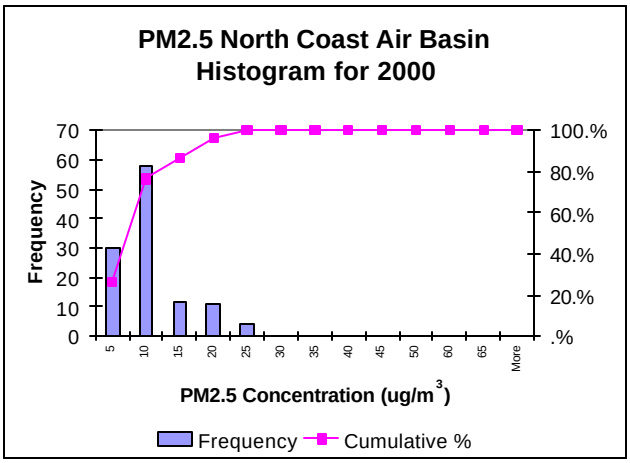


Figure 6.15. Frequency distribution of ambient PM2.5 levels in the North Coast Air Basin (measurements from all monitors in the air basin).

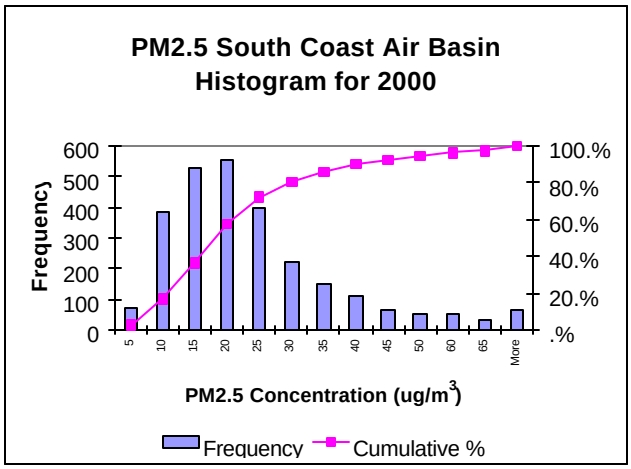


Figure 6.16. Frequency distribution of ambient PM2.5 levels in the South Coast Air Basin (measurements from all monitors in the air basin).

6.3.1.6 Diurnal Variation in PM10 levels

We used PM10 data collected with a Tapered Element Oscillating Monitor (TEOM) at two rural agricultural locations in the Sacramento Valley and filter-based samples collected at one urban and one rural site in the San Joaquin Valley to analyze hourly variations in PM10 levels. TEOM samplers collect PM10 samples continuously, while filter-based samples were collected every three hours. PM10 levels can vary significantly within a day and continuous monitoring data are most useful to study these variations. On a rice straw burning day, in the Sacramento Valley, PM10 concentrations reached 4 to 5 times the level of the State 24-hour standard for several hours, although the 24-hour average PM10 level was barely above the current State standard. In the San Joaquin Valley, PM10 levels varied significantly in urban Fresno during the course of a winter day, with the highest concentrations occurring at nighttime, while PM10 concentrations did not vary much throughout the day in rural SW Chowchilla. Chemical composition data indicate diurnal variations in ammonium nitrate were the primary cause of the PM10 variations in SW Chowchilla. The rise in PM10 concentrations in Fresno corresponded mostly to significant nighttime peaks in vegetative burning, mobile sources, and excess organic carbon.

6.3.1.7 Particle Size Distribution

Data on particle size distribution is limited. During the IMS95 winter study in San Joaquin Valley, air samples using a Micro-Orifice Uniform Deposit Impactor (MOUDI) sampler were collected at Bakersfield (Chow et al. 1997). The MOUDI partitions ambient PM samples into nine size cuts between 0.054 and 15 μm. We used these data to study the size distribution of PM10

components. Soil components were concentrated mainly in the larger size fractions (>3.16 μm), the coarse component of PM10. The size of nitrate particles peaked between 1 and 1.78 μm, while organic carbon particles appeared in both larger (peak between 0.37 and 1 μm) and smaller (<0.054 μm) size fractions.

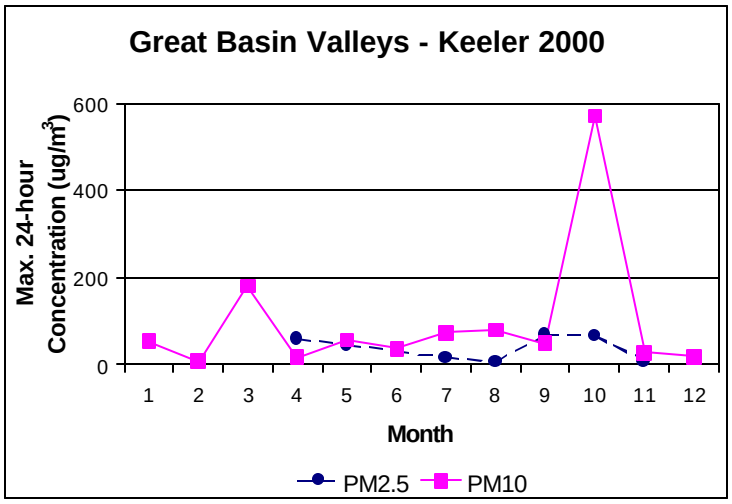
6.3.2 Characterization of Ambient Particulate Matter by Air Basin

This section describes the characteristics of ambient particulate matter for each of the fifteen air basins in the State. The information presented includes: maximum 24-hour and annual average PM10 and PM2.5 concentrations, seasonal variation of particulate matter levels; frequency of measured PM10 and PM2.5 concentrations, and ratios of PM2.5 to PM10 levels. Where available, source attribution information is also included. For areas where no source attribution analyses are available, the primary chemical composition of ambient PM10 or PM2.5 is illustrated. Based on the 2000 annual average PM10 emission inventory, we identify the main sources of directly emitted PM10.

On an annual basis, particulate levels are low in the Great Basin Valleys Air Basin (PM10 = 14 to 20 $\mu g/m^3$). Eighty percent of the 24-hour PM10 observations were below 25 to 30 $\mu g/m^3$ and 80% of the 24-hour PM2.5 observations were under 10 to 15 $\mu g/m^3$. However, on a short term, episodic basis, Great Basin Valleys may record some of the highest monitored levels in the State. During windy conditions, dust from the Owens and Mono dry lakebeds produce extremely high concentrations of particulate in the air, reaching 3059 $\mu g/m^3$ in Mono in 2000 and 1116 $\mu g/m^3$ in Owens in 1998. Particulate levels exceeded the 24-hour State PM10 standard 64 times in the 1998-2000 period and two observations over the federal PM2.5 standard were recorded in the 1999-2000 period. The Great Basin Valleys Air Basin did not exceed the PM10 annual standard.

Figure 6.17 illustrates the monthly variation of the maximum daily PM10 and PM2.5 concentrations at Keeler in 2000. Keeler is located near the Owens dry lakebed. High PM10 concentrations can occur at any time of the year, though more frequently in the spring and fall. PM2.5 concentrations are relatively uniform most of the year.

Figure 6.17. Monthly variation in maximum 24-hour PM10 and PM2.5 concentrations at Keeler



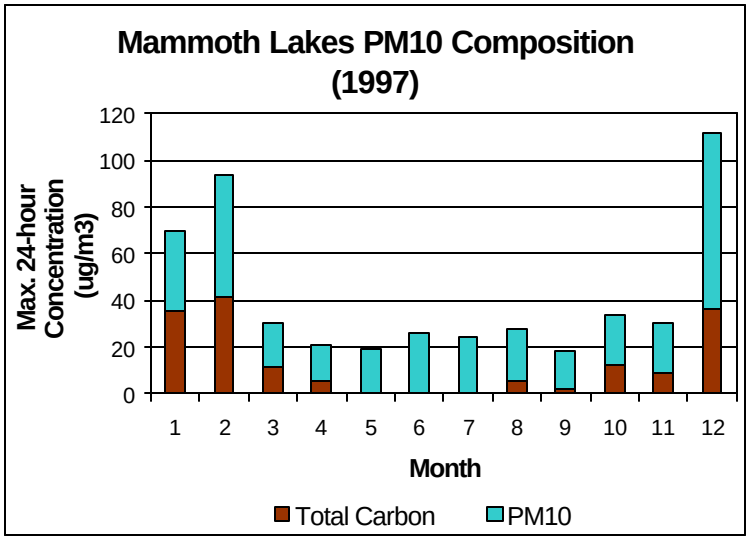
*The monitors used to measure PM10 and PM2.5 are different and occasionally recorded concentrations of PM2.5 which are greater than PM10.

Data obtained from the Keeler and Coso Junction dichotomous samplers in 1999 indicate the PM2.5 component of PM10 ranges from 14% to 89%, with an annual average of 33%.

Based on the 2000 annual PM10 emission inventory, the major sources of directly emitted particulate matter in the Great Basin Valleys Air Basin are unpaved road dust, windblown dust, residential wood burning, and wildfires.

In the town of Mammoth Lakes, high PM10 concentrations usually occur during the winter months (December – February). Figure 6.18 shows the monthly variation of the maximum daily PM10 concentrations in 1997. The chart also illustrates how much of the measured PM10 is total carbon. During the winter, total carbon comprises 30% to 50% of the measured PM10. Sources of carbon include residential wood combustion and motor vehicles.

Figure 6.18. Monthly variation in maximum 24-hour PM10 and total carbon levels at Mammoth.

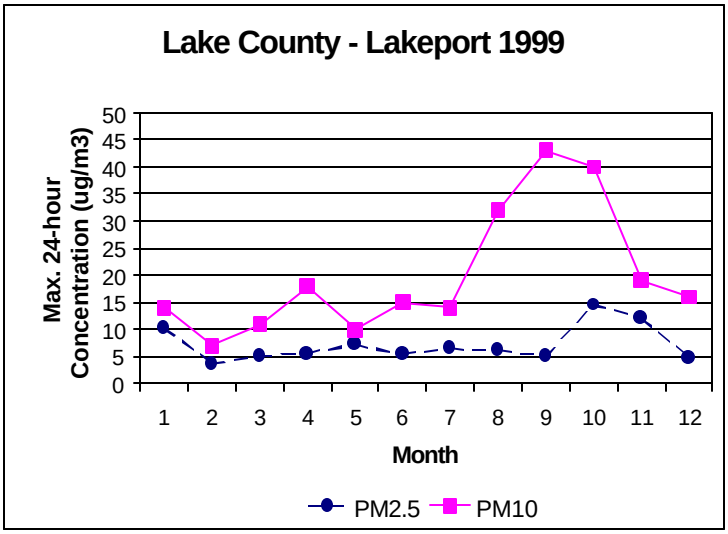


On average, Lake County has among the lowest particulate levels in the State. Maximum 24-hour PM10 ranges from 22 to 35 µg/m^3 and maximum 24-hour PM2.5 from 9 to 15 µg/m^3, with no exceedances of either standard.

Figure 6.19 illustrates the monthly variation of the maximum daily PM10 and PM2.5 concentrations at Lakeport in 1999. PM10 levels are highest from August through October and are low the rest of the year. PM2.5 concentrations peak in October and November.

Based on estimated 2000 annual average PM10 emission inventory data, the principal sources of directly emitted particulate matter in Lake County are unpaved road dust and residential wood burning. Occasionally, Lake County also has significant levels of particulates from wildfires.

Figure 6.19. Monthly variation in maximum 24-hour PM10 and PM2.5 concentrations at Lakeport

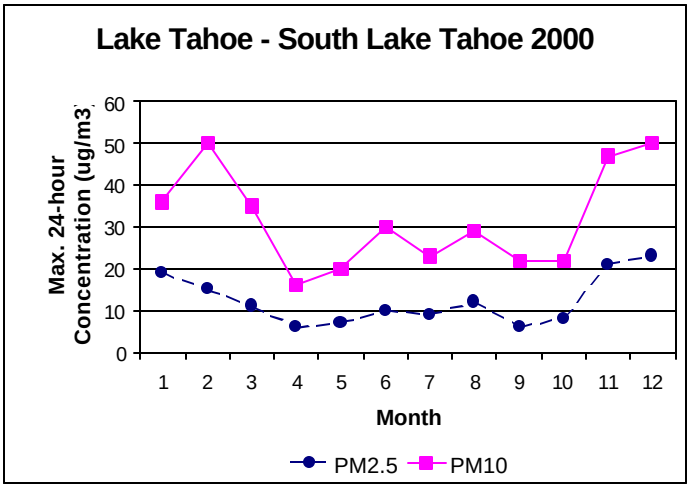


*The monitors used to measure PM10 and PM2.5 are different and occasionally recorded concentrations of PM2.5 which are greater than PM10.

In the Lake Tahoe Air Basin, particulate levels exceeded the 24-hour State PM10 standard two times in the 1998-2000 period, but fine particulate levels were well below the federal PM2.5 standards. The State annual PM10 standard was also not exceeded. In 1998, 80% of the PM10 observations were below 45 to 50 µg/m^3. In the last two years, 80% of the PM2.5 observations were below 10 to 15 µg/m^3.

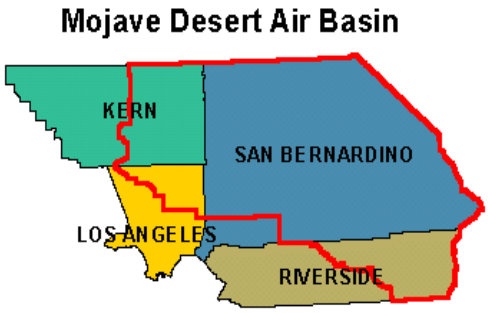
Figure 6.20 illustrates the monthly variation of the maximum daily PM10 and PM2.5 concentrations in South Lake Tahoe in 2000. PM10 as well as PM2.5 levels are highest during the late fall and winter (November through February), and are lowest in the in spring and summer.

Figure 6.20. Monthly variation in maximum 24-hour PM10 and PM2.5 concentrations at South Lake Tahoe



*The monitors used to measure PM10 and PM2.5 are different and occasionally recorded concentrations of PM2.5 which are greater than PM10.

Based on the 2000 annual PM10 emission inventory, the major sources of directly emitted particulate matter are unpaved road dust and residential wood burning.



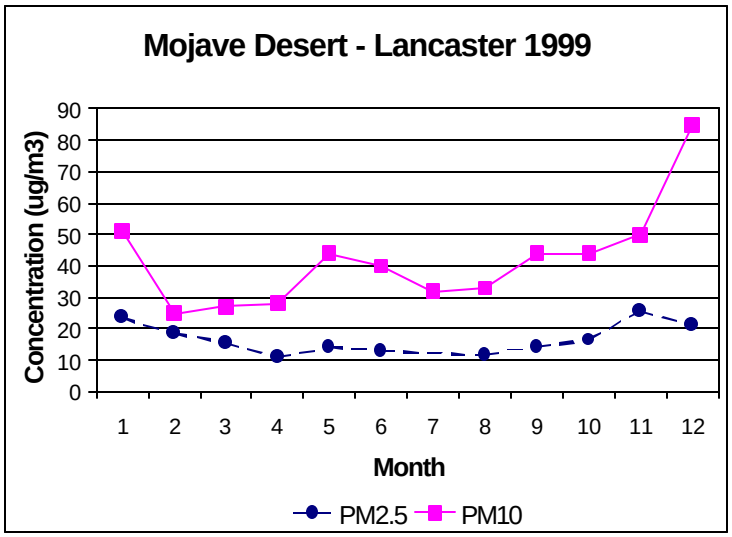
In the Mojave Desert Air Basin, particulate levels exceeded the 24-hour State PM10 standard 31 times in the 1998-2000 period, but fine particulate levels were below the federal PM2.5 standards. The State annual PM10 standard was also not exceeded. Eighty percent of the PM10 observations were below 30 to 35 $\mu g/m^3$ and 80% of the PM2.5 observations were below 20 to 25 $\mu g/m^3$.

Figure 6.21 illustrates the monthly variation of the maximum daily PM10 and PM2.5 concentrations in Lancaster in 1999. PM10 as well as PM2.5 levels are highest during the winter months - December and January. During the rest of the year, PM2.5 levels are quite low, while PM10 levels fluctuate with no distinct pattern.

Data from the dichotomous sampler at Victorville in 1999 indicate the PM2.5 component of PM10 ranges from 19% to 75%. The average PM2.5 fraction of PM10 is 46% from November to February and 39% from March to October.

Ambient air quality data from 1997 through 1999 show low levels of secondary nitrate and sulfate particulate in the Mojave Desert, indicating that most of the particulate matter is primary in origin.

Figure 6.21. Monthly variation in maximum 24-hour PM10 and PM2.5 concentrations at Lancaster



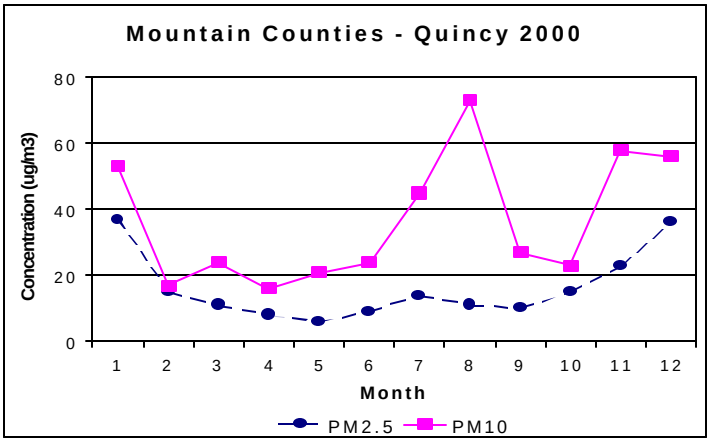
*The monitors used to measure PM10 and PM2.5 are different and occasionally recorded concentrations of PM2.5 which are greater than PM10.

Based on the 2000 annual PM10 emission inventory, the major contributors to primary particulates in the Mojave Desert Air Basin are unpaved road dust, windblown dust, paved road dust, and construction related dust. A few point source categories, such as mineral processing facilities, also contribute significant emissions.

In the Mountain Counties Air Basin, particulate levels exceeded the 24-hour State PM10 standard 34 times in the 1998-2000 period and four observations over the federal 24-hour PM2.5 standard were recorded in 1999. Fine particulate exceedances in 1999 were most probably due to wild fires which occurred in the late summer and early fall. Neither the State PM10 nor the federal PM2.5 annual standards were exceeded. In the Mountain Counties 80% of the PM10 observations were below 30 to 35 $\mu g/m^3$ and 80% of the PM2.5 readings were below 10 to 15 $\mu g/m^3$.

Figure 6.22 illustrates the monthly variation of the maximum daily PM10 and PM2.5 concentrations in Quincy in 2000. Highest ambient concentrations of PM10 occur during the summer and winter months, while fine particulate matter levels are highest in the late fall and early winter months of November through January.

Figure 6.22. Monthly variation in maximum 24-hour PM10 and PM2.5 concentrations at Quincy



*The monitors used to measure PM10 and PM2.5 are different and occasionally recorded concentrations of PM2.5 which are greater than PM10.

Data obtained from the Portola dichotomous sampler in 1999 show that the PM2.5 portion of PM10 ranged from 19% to 91%. The average PM2.5 fraction of PM10 was 72% from November through January and 41% during the rest of the year.

Based on the 2000 annual PM10 emission inventory, directly emitted particulate sources are unpaved road dust, wood burning stoves and fireplaces, and open burning.

As shown on Figure 6.23 , substantial levels of organic carbon are observed in the late fall and winter months, most likely due to residential burning and motor vehicles. There may also be episodic particulate emission impacts when forest management burning takes place.

Figure 6.23. Monthly variation in maximum 24-hour PM10 and total carbon concentrations at Quincy

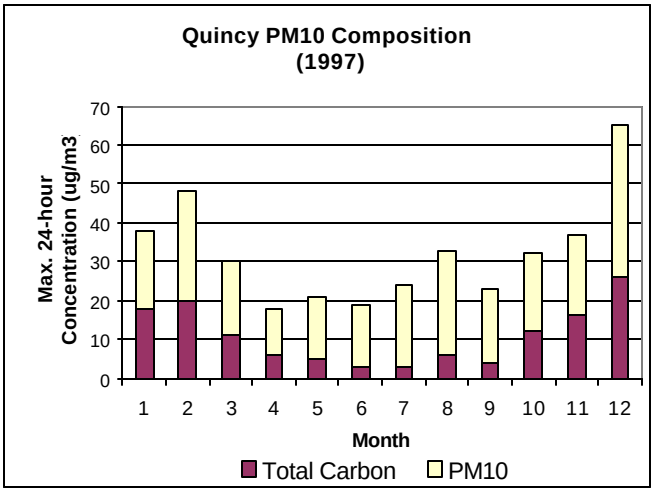
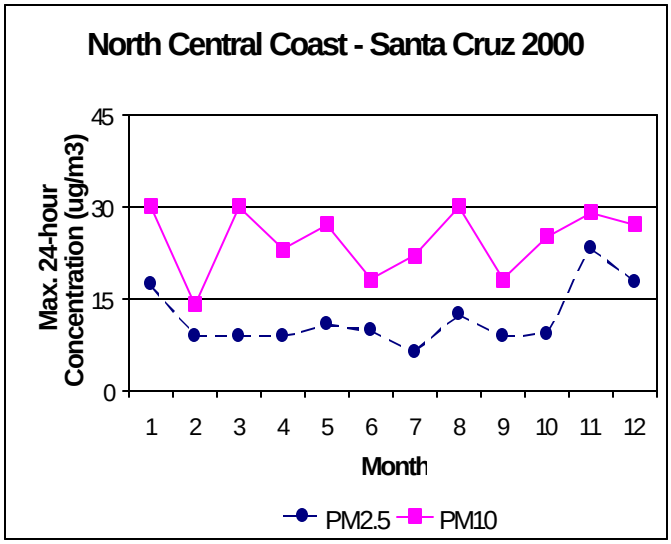


Figure 6.24 illustrates the monthly variation of the maximum daily PM10 and PM2.5 concentrations in Santa Cruz in 2000. Fine particulate levels are highest from November through January and are very low the rest of the year. PM10 levels fluctuate throughout the year, with no distinct seasonal pattern.

Based on the 2000 annual PM10 emission inventory, the major sources of directly emitted particulates in the North Central Coast Air Basin are unpaved roads, windblown dust, dust from farming operations, paved road dust, and residential wood burning.

In the North Central Coast Air Basin, particulate levels exceeded the 24-hour State PM10 standard 18 times in the 1998-2000 period, but fine particulate levels were below the federal PM2.5 standards. The State annual PM10 standard was also not exceeded. In the North Central Coast, 80% of the PM10 observations were below 30 to 35 $\mu g/m^3$ and 80% of the PM2.5 measurements were below 10 to 15 $\mu g/m^3$.

Figure 6.24. Monthly variation in maximum 24-hour PM10 and PM2.5 concentrations at Santa Cruz

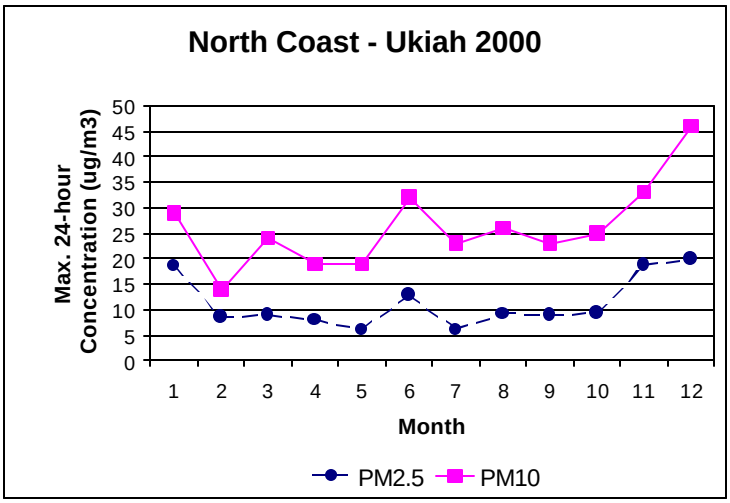


*The monitors used to measure PM10 and PM2.5 are different and occasionally recorded concentrations of PM2.5 which are greater than PM10.

In the North Coast Air Basin, particulate levels exceeded the 24-hour State PM10 standard 13 times in the 1998-2000 period, but fine particulate levels were below the federal PM2.5 standards. The State annual PM10 standard was also not exceeded. In the North Coast Air Basin, 80% of the PM10 observations were below 30 to 35 $\mu g/m^3$ and 80% of the PM2.5 measurements fell below 10 to 15 $\mu g/m^3$.

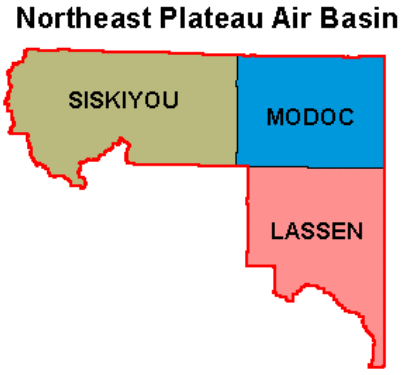
Figure 6.25 illustrates the monthly variation of the maximum daily PM10 and PM2.5 concentrations in Ukiah in 2000. PM10 as well as PM2.5 levels are highest during the months of November through January, with a smaller peak in June.

Figure 6.25. Monthly variation in maximum 24-hour PM10 and PM2.5 concentrations at Ukiah



*The monitors used to measure PM10 and PM2.5 are different and occasionally recorded concentrations of PM2.5 which are greater than PM10.

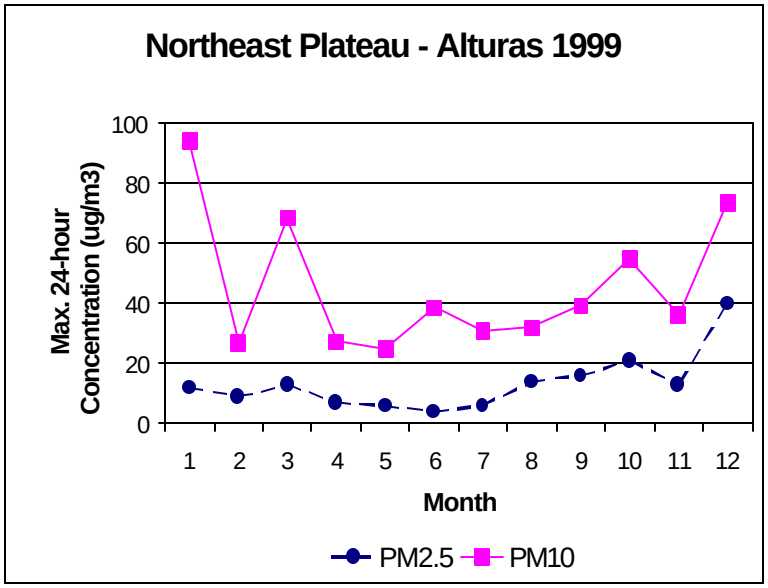
Based on the 2000 annual PM10 emission inventory, the principal source of directly emitted particulate matter is unpaved road dust. Other significant sources are residential wood burning and waste burning, which could include forest management burning.



In the Northeast Plateau Air Basin, particulate levels exceeded the 24-hour State PM10 standard 26 times in the 1998-2000 period, but fine particulate levels were below the federal PM2.5 standards. The State annual PM10 standard was also not exceeded. In this air basin, 80% of the PM10 measures were below 30 to 35 $\mu g/m^3$ and 80% of the PM2.5 observations were below 15 to 20 $\mu g/m^3$.

Figure 6.26 illustrates the monthly variation of the maximum daily PM10 and PM2.5 concentrations in Alturas in 2000. PM10 levels are highest during the winter months of December through March with lower concentrations during the spring and summer. PM2.5 levels are highest in December.

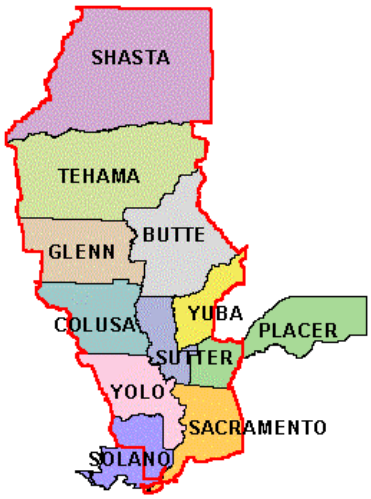
Figure 6.26. Monthly variation in maximum 24-hour PM10 and PM2.5 concentrations at Alturas



*The monitors used to measure PM10 and PM2.5 are different and occasionally recorded concentrations of PM2.5 which are greater than PM10.

The 2000 annual PM10 emission inventory shows that unpaved road dust is the predominant source of directly emitted particulates. The Northeast Plateau Air Basin may also have occasional high emissions from wildfires and forest management burning.

Sacramento Valley Air Basin



In the Sacramento Valley Air Basin, particulate levels exceeded the 24-hour State PM10 standard 61 times in the 1998-2000 period and PM2.5 concentrations over the federal PM2.5 standard were recorded 16 times in the 1999-2000 period. Particulate levels also exceeded both the State PM10 and federal PM2.5 annual standards. In the Sacramento Valley Air Basin, 80% of the PM10 observations are below 45 to 50 $\mu g/m^3$ and 80% of the PM2.5 measurements are below 20 to 25 $\mu g/m^3$.

Figures 6.27 and 6.28 illustrate the monthly variation of the maximum daily PM10 and PM2.5 concentrations in Colusa, a rural community in the central portion of the Valley, and the city of Sacramento, in the southern portion of the Valley for 2000, respectively. In Colusa, PM10 levels oscillate throughout the year with no distinct seasonal pattern. PM2.5 levels are highest in the fall and winter. In contrast, in Sacramento, both PM10 and PM2.5 levels are low during the spring and summer, with PM10 reaching peak values in the fall and early winter and PM2.5 reaching highest values in the winter. Data obtained from the Sacramento dichotomous sampler show that in 1999 and 2000 the PM2.5 portion of PM10 ranged from 13% to 86%. The two-year average PM2.5 portion of PM10 from November through February was 68% dropping to 43% from March through October.

Figure 6.27. Monthly variation in maximum 24-hour PM10 and PM2.5 concentrations at Colusa

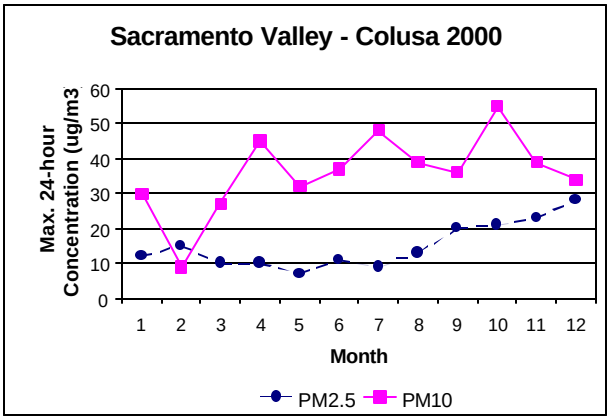
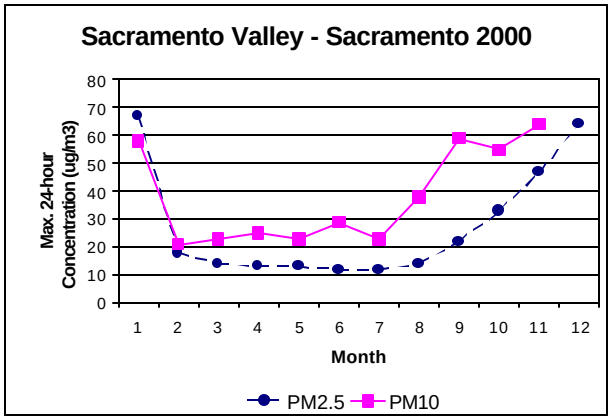


Figure 6.28. Monthly variation in maximum 24-hour PM10 and PM2.5 concentrations at Sacramento



*The monitors used to measure PM10 and PM2.5 are different and occasionally recorded concentrations of PM2.5 which are greater than PM10.

Based on the 2000 annual PM10 emissions inventory, the major sources of directly emitted particulates in the Sacramento Valley include soil from farming, construction dust, paved road dust, smoke from residential wood combustion, and exhaust from mobile sources such as cars and trucks.

Figures 6.29 and 6.30 illustrate source contributions to ambient PM10 and PM2.5 during the winter in Sacramento. The data are from the analysis of ambient air samples collected from November through January, during six years - 1991 through 1996 (Motallebi 1999, Motallebi 2001). The constituents shown can vary based on a variety of factors such as meteorology and which particulate sources are most active.

Figure 6.29. Source contribution to PM10 during the winter

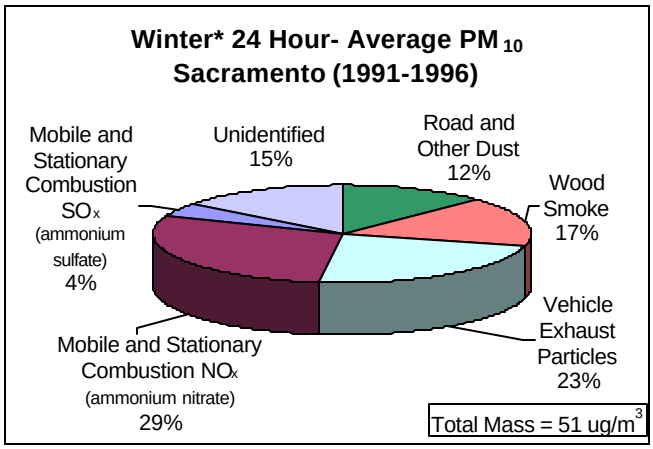
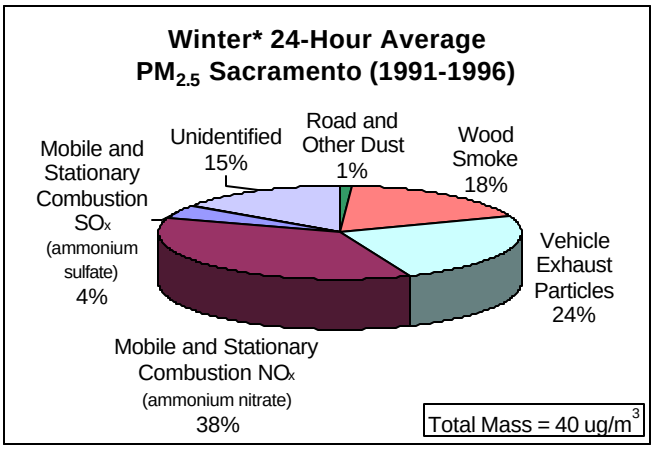


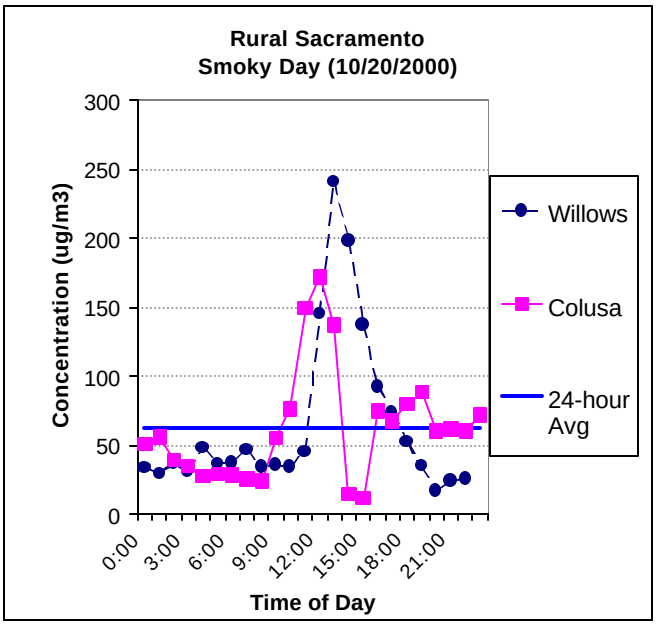
Figure 6.30. Source contribution to PM2.5 during the winter



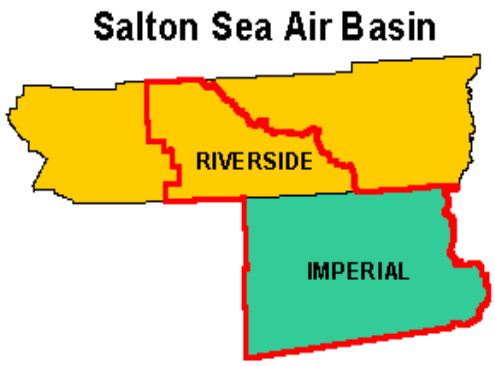
*Average of days with PM10 > 40 µg/m^3.

NOx emissions from mobile and stationary combustion sources, combined with ammonium, contribute the most to ambient PM levels. Vehicle exhaust particle emissions and wood smoke from residential wood combustion also contribute significantly. While road and other dust is a significant component of ambient PM10, its contribution to PM2.5 is minor.

Figure 6.31. Hourly PM10 levels on a smoky day in rural Sacramento



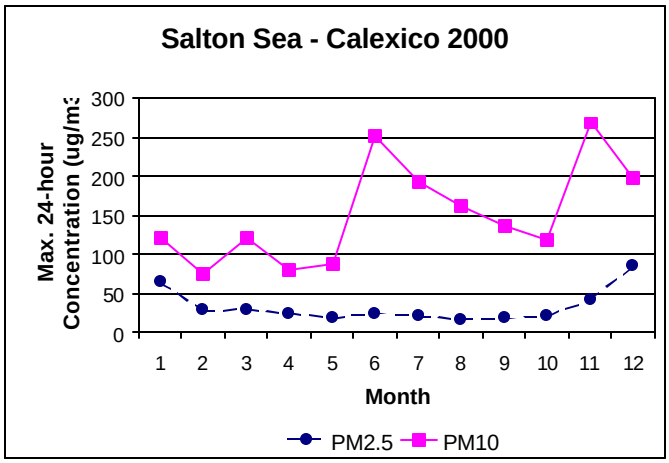
The northern Sacramento Valley can be impacted by seasonal agricultural burning, mostly during the fall. Figure 6.31 illustrates the hourly variation in PM10 levels on a rice straw burning day in Willows and Colusa in 2000. PM10 levels reached 4 to 5 times the level of the State 24-hour PM10 standard for two hours in Willows and an average of 3 times the level of the standard for three hours in Colusa.



In the Salton Sea Air Basin particulate levels exceeded the 24-hour State PM10 standard 208 times in the 1998-2000 period, but only one observation over the 24-hour federal PM2.5 standard was recorded in 2000. Particulate levels also exceeded both the State PM10 and federal PM2.5 annual standards. Eighty percent of the PM10 observations were below 100 to 120 $\mu g/m^3$, while 80% of the PM2.5 measurements fell below 20 to 25 $\mu g/m^3$.

Figure 6.32 illustrates the monthly variation of the maximum daily PM10 and PM2.5 concentrations in Calexico in 2000. PM10 levels peak in the summer and fall. Fine particulates show a small increase in the fall and winter.

Figure 6.32. Monthly variation in maximum 24-hour PM10 and PM2.5 in Calexico



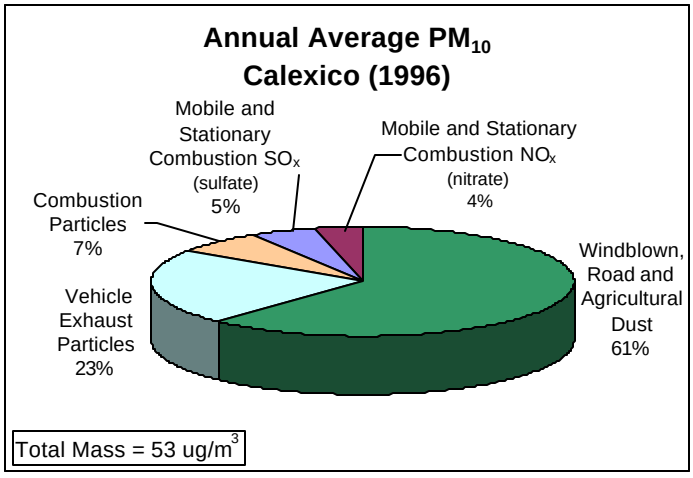
*The monitors used to measure PM10 and PM2.5 are different and occasionally recorded concentrations of PM2.5 greater than PM10.

Data obtained from the Calexico dichotomous sampler in 2000 indicate the PM2.5 component of PM10 ranges from 13% to 49%. The average PM2.5 fraction of PM10 from November to January is 34% and from February to October is 24%.

Based on the 2000 annual PM10 emission inventory, the major contributor of directly emitted particulates in the Salton Sea is windblown dust. Unpaved road dust and farming related dust also contribute.

Data for Figures 6.33 and 6.34 are from the source apportionment analysis of ambient samples collected during 1996 in Calexico. The source profiles developed by Chow and Watson (1997) were used in the analysis (Woodhouse, 2001).

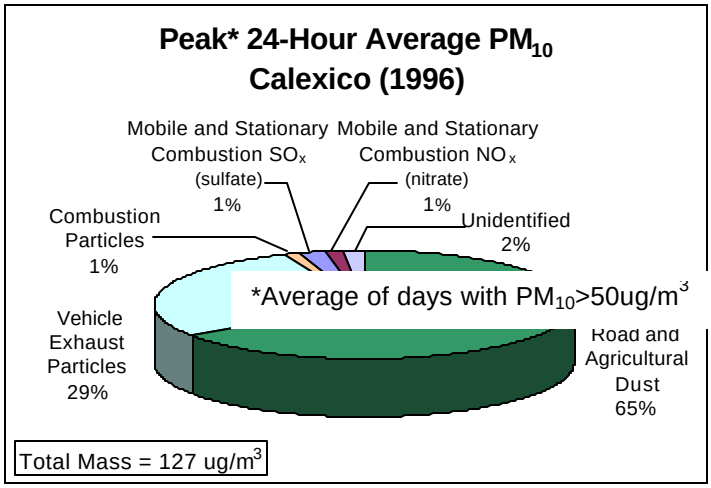
Figure 6.33. Sources contributing to annual average PM10 levels in Calexico



In both cases, dust – windblown, road and agricultural – is the major contributor to PM10. Vehicle exhaust particle emissions also contribute significantly. Combustion emissions from industrial processes and agricultural burns contribute noticeably to PM10 on an annual basis, but are a minor contributor to the peak 24-hour average PM10 levels.

The observed results could partially be due to transported pollutants from the neighboring city of Mexicali, which has high traffic. Secondary sulfate and nitrate formed from reactions in the atmosphere of nitrogen oxides and sulfur oxides from motor vehicle exhaust and other combustion processes also are small contributors to particulate matter levels in the air basin.

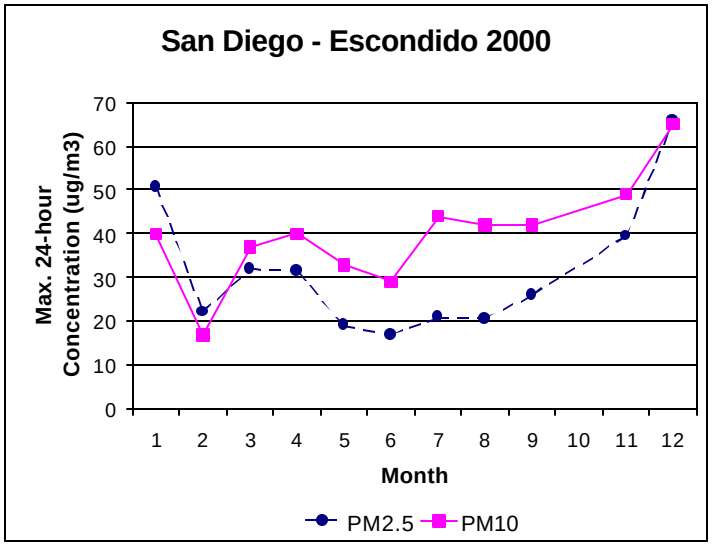
Figure 6.34. Sources contributing to peak 24-hour PM10 levels in Calexico



In the San Diego Air Basin, particulate levels are high year-round, exceeding both the annual State PM10 and federal PM2.5 standards over the 1998-2000 period. Ambient particulate levels also exceeded the State 24-hour PM10 standard 67 times in these three years and two PM2.5 observations over the federal PM2.5 standard were recorded in the 1999-2000 period. In San Diego County, 80% of the PM10 measurements were below 40 to 50 $\mu g/m^3$ and 80% of the PM2.5 observations were below 20 to 25 $\mu g/m^3$.

Figure 6.35 illustrates the monthly variation of the maximum daily PM10 and PM2.5 concentrations in Escondido in 2000. PM10 concentrations exhibit no distinct seasonal pattern, while PM2.5 concentrations are highest during the fall and winter.

Figure 6.35. Monthly variation of maximum 24-hour PM10 and PM2.5 levels in Escondido

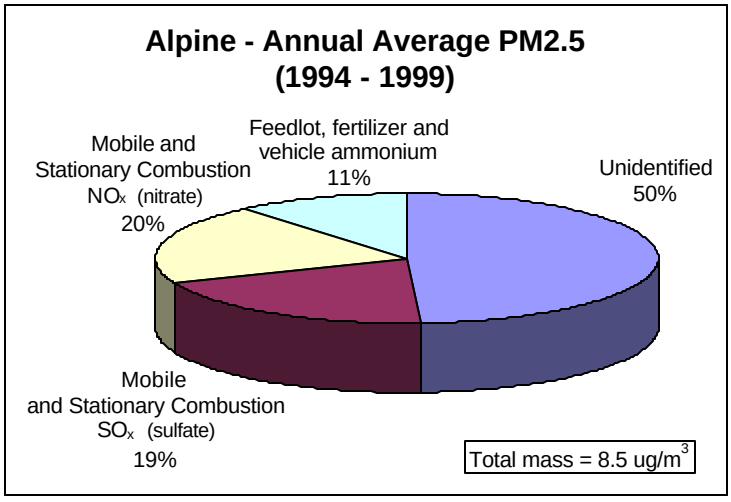


*The monitors used to measure PM10 and PM2.5 are different and occasionally recorded concentrations of PM2.5 which are greater than PM10.

Based on the 2000 annual PM10 emission inventory, the major contributors to directly emitted particulates in the San Diego Air Basin are construction dust, paved road dust, and unpaved road dust. Other sources are fireplaces and woodstoves, mobile sources, and mineral processes.

Data for Figure 6.36 are from the chemical analysis of ambient data collected in Alpine from 1994 through 1999 as part of the Southern California Children's Health Study. The data show substantial contributions from secondary nitrate and sulfate formed from reactions in the atmosphere of nitrogen oxides and sulfate oxides from motor vehicle exhaust and other combustion processes. The unidentified category represents emissions from dust sources and from total carbon. Carbon sources include wood smoke, other combustion sources, and motor vehicles (Salmon et al. 2001).

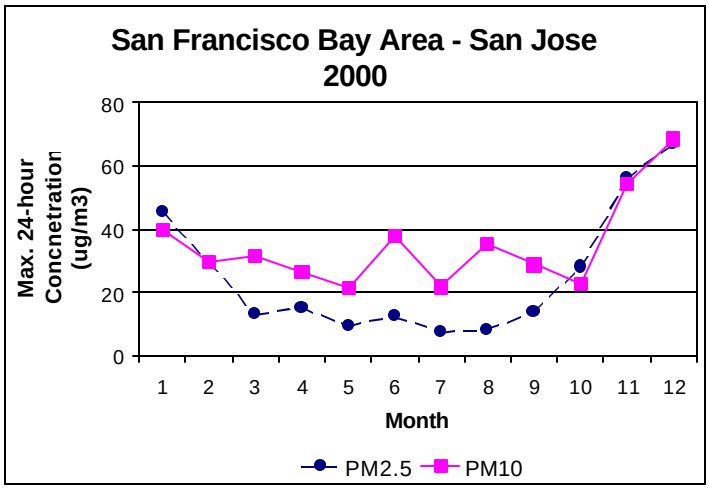
Figure 6.36. Sources contributing to annual average PM2.5 levels in Alpine



In the San Francisco Bay Area Air Basin, particulate levels exceeded the 24-hour State PM10 standard 24 times in the 1998-2000 period and five PM2.5 observations over the 24-hour federal PM2.5 standard were recorded in the 1999-2000 period. Eighty percent of the 24-hour PM10 observations were below 25 to 30 $\mu g/m^3$ and 80% of the 24-hour PM2.5 measurements were below 20 to 25 $\mu g/m^3$.

Figure 6.37 illustrates the monthly variation of the maximum daily PM10 and PM2.5 concentrations in San Jose in 2000. Highest concentrations of both PM10 and PM2.5 occur during the winter months of November through January. PM2.5 drives PM10 concentrations during the winter, while smaller summer peaks are driven by PM10.

Figure 6.37. Monthly variation of maximum 24-hour PM10 and PM2.5 levels in San Jose



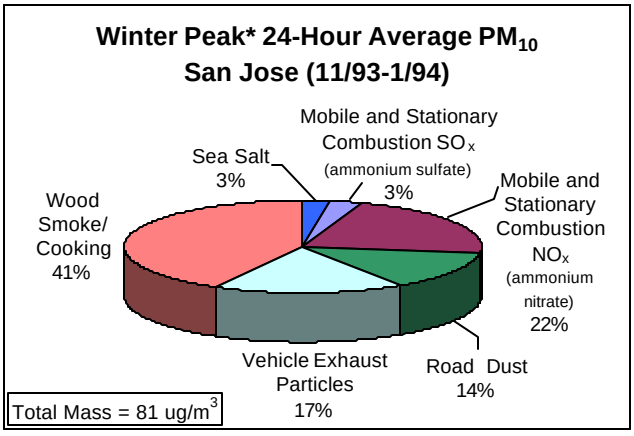
*The monitors used to measure PM10 and PM2.5 are different and occasionally recorded concentrations of PM2.5 which are greater than PM10.

Data obtained from the San Jose dichotomous sampler in 1999 indicate the PM2.5 portion of PM10 ranges from 30% to 80%. The average PM2.5 portion of PM10 from November to January is 61%, dropping to 46% from February to October.

Based on the 2000 annual PM10 emission inventory of directly emitted particulate matter, major sources include smoke from residential wood combustion, dust from construction operations, and the dust created by vehicles traveling on paved roads. There are also significant emissions from unpaved road dust in some counties and motor vehicle exhaust from cars and trucks.

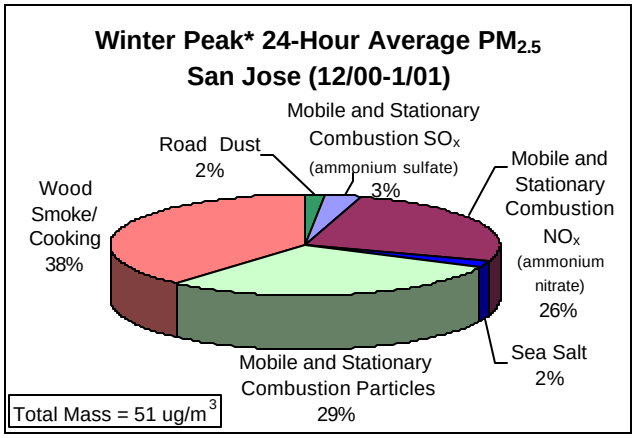
Figures 6.38 and 6.39 illustrate the sources of PM during the winter in the San Franciso Bay Area. The data are from the source apportionment analysis conducted by the Bay Area Air Quality Management District using samples collected during two special studies (Fairley, 1996, 2001).

Figure 6.38. Sources contributing to winter peak 24-hour levels of PM10 in San Jose



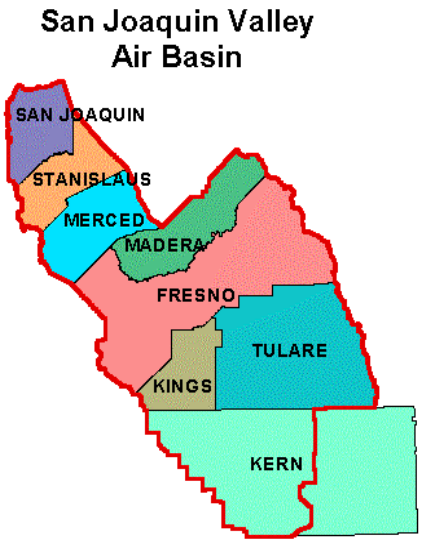
*Average of days with PM_{10} > 50 ug/m^3.

Figure 6.39. Sources contributing to winter peak 24-hour levels of PM2.5 in San Jose



*Average of days with $PM_{2.5}$ > 40 ug/m^3.

During the winter in San Jose, high PM concentrations are associated with high levels of wood smoke - primarily from residential wood combustion, and cooking. NO_x emitted from mobile and stationary combustion sources, in combination with ammonia, contributes about one-fourth of the PM levels. Particle emissions from mobile and stationary combustion sources are also a major contributor to PM2.5. Road dust is a significant contributor to PM10, but not PM2.5.



In 1999 and 2000, the San Joaquin Valley Air Basin recorded the highest PM2.5 levels in the State – more than twice the federal standard - and 74 PM2.5 observations over the federal standard were recorded. Particulate levels exceeded the 24-hour State PM10 standard 177 times in the 1998-2000 period. Particulate concentrations also exceeded both the State PM10 and federal PM2.5 annual standards. In the San Joaquin Valley Air Basin, 80% of the PM10 observations were below 60 to 65 µg/m^3 and 80% of the PM2.5 measurements were below 35 to 40 µg/m^3.

Figures 6.40 and 6.41 illustrate the monthly variation of the maximum daily PM10 and PM2.5 concentrations in Corcoran in 2000 and in Fresno in 1999. In Corcoran, PM10 levels are highest in October and November, with PM2.5 peaking in November. In Fresno, PM10 and PM2.5 are highest from October through January. PM2.5 drives PM10 concentrations during the wintertime in Fresno. The PM2.5 fraction of PM10 is smaller in Corcoran with fall peaks driven by PM10. Data obtained from the Fresno dichotomous sampler from 1998 through 1999 indicate

Figure 6.40. Monthly variation of maximum 24-hour PM10 and PM2.5 levels in Fresno

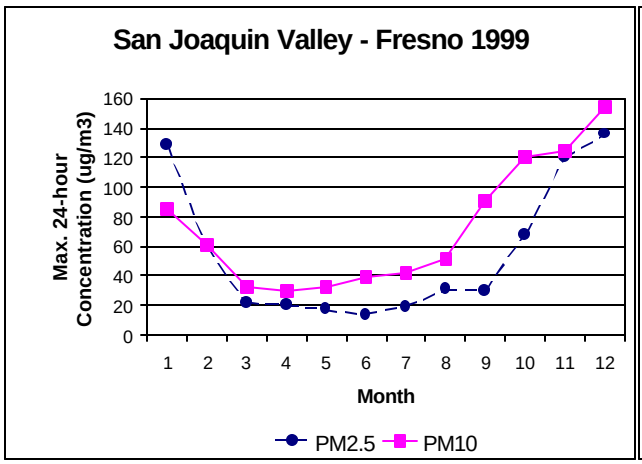
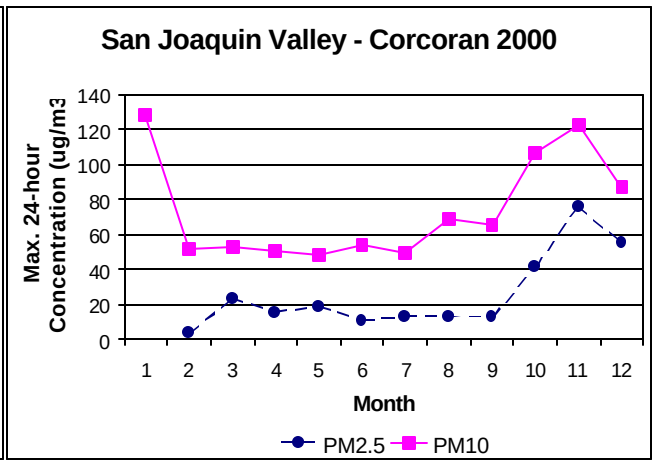


Figure 6.41. Monthly variation of maximum 24-hour PM10 and PM2.5 levels in Corcoran



*The monitors used to measure PM10 and PM2.5 are different and occasionally recorded concentrations of PM2.5 which are greater than PM10.

the PM2.5 component of PM10 ranges from 19% to 88%. The November through February average PM2.5 fraction is 75% of PM10 and the March through October average is 38%. Data obtained from the Corcoran dichotomous sampler from 1998 and 1999 show that the PM2.5 component ranges from 12% to 90%. The November through February average PM2.5 portion of PM10 is 62% and the March through October average is 28%.

Figure 6.42 shows the daily variations in PM2.5 levels in Fresno during the winter of 2000 to 2001. The data were obtained as part of the CRPAQS study. PM2.5 concentrations were over the federal 24-hour PM2.5 standard close to 40% of the time.

Based on the 2000 annual PM10 emission inventory, the major sources of directly emitted particulates in the San Joaquin Valley are agricultural and unpaved road dust, paved road dust, and windblown dust. Other sources include stationary industrial activities, residential wood combustion, and particulates emitted by mobile sources such as cars and trucks.

Figures 6.43 and 6.44 illustrate source contributions to ambient PM in the San Joaquin Valley during the fall and winter. These are the results from a detailed chemical analysis of samples collected during the 1995-Integrated Monitoring Study (Magliano et al. 1999).

Figure 6.42. Daily variations in winter PM2.5 levels in Fresno

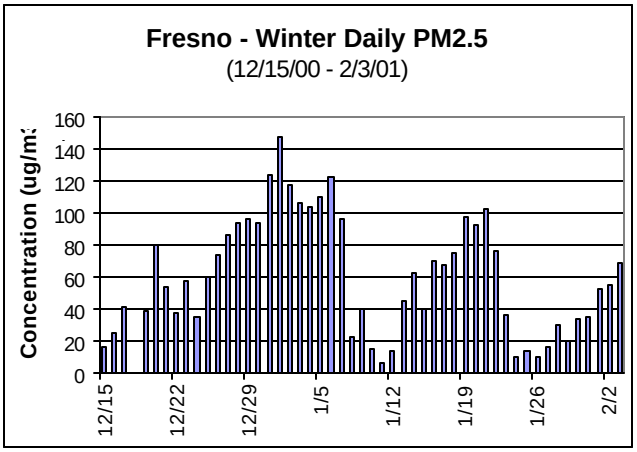


Figure 6.43. Sources contributing to PM10 levels in the fall in Corcoran

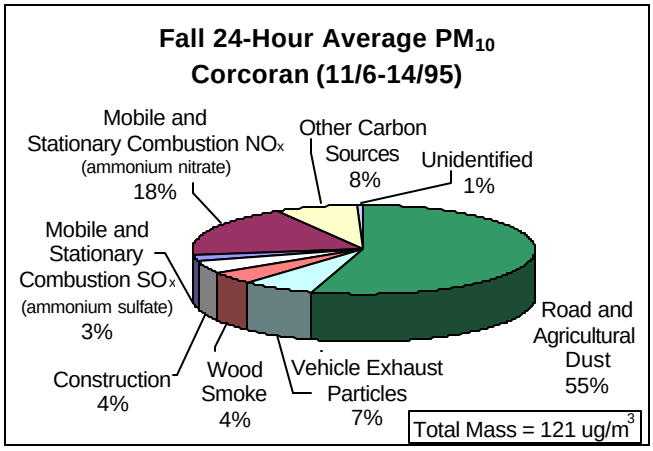
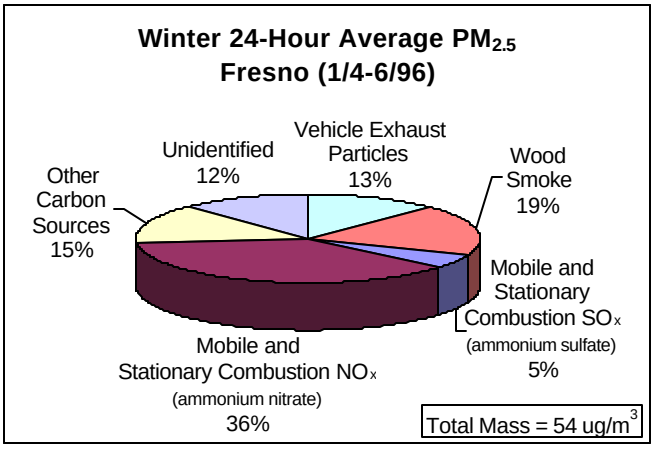


Figure 6.44. Sources contributing to PM2.5 levels in the winter in Fresno



In the fall at Corcoran, elevated concentrations of PM10 were associated with high levels of road and agricultural dust. NOx emissions from mobile and stationary combustion sources, combined with ammonia, led to significant secondary ammonium nitrate contributions to PM10. During the winter, in Fresno, secondary ammonium nitrate was the major contributor to PM2.5 and PM10. Emissions from wood smoke, vehicle exhaust particles, and other carbon sources also contributed significantly to PM2.5. levels.

Figure 6.45 illustrates the source contributions to winter PM2.5 levels at two urban and two rural areas in the valley. The Bakersfield and Fresno sites were located in large urban areas; the Kern Wildlife Refuge site was located amidst natural vegetation, while the SW Chowchillla site was in a rural area, surrounded by agricultural fields. At the peak of a winter PM2.5 episode, PM2.5 concentrations at the two rural sites were about half of the PM2.5 levels at the two urban sites. Secondary ammonium nitrate was the largest contributor at all four sites. Vegetative burning and direct mobile source exhaust contributed 19% and 12% of the PM2.5 mass in the urban areas, but only an average of 8% and 9% at the rural sites. The excess organic carbon resulting from combustion sources other than vegetative burning and mobile sources as well as secondary organic carbon – was significant at the urban, but not at the rural sites.

Figure 6.45. Sources contributing to the PM2.5 levels at the peak of a winter episode.in two urban and two rural areas

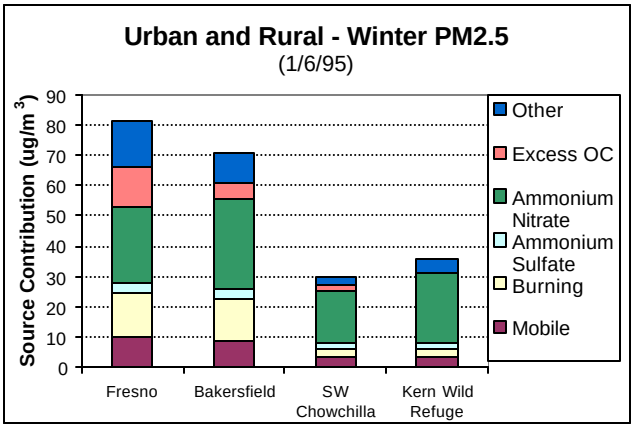


Figure 6.46. Comparison of hourly variations in winter PM2.5 levels at urban Fresno and rural SW Chowchilla

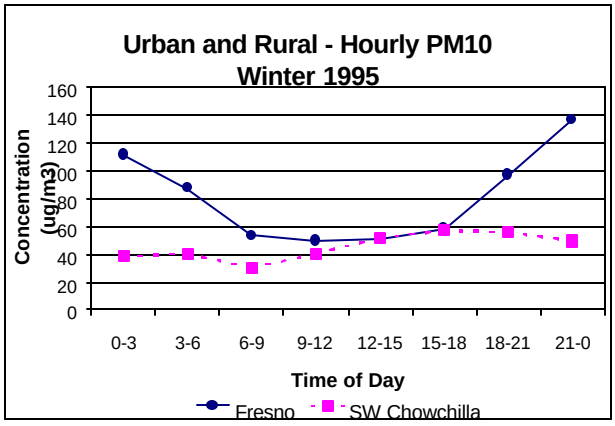
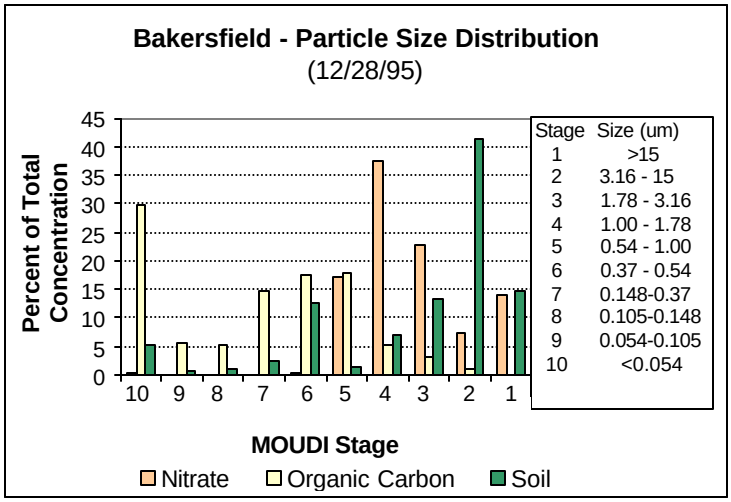


Figure 6.46 illustrates that in the winter in Fresno, PM10 levels varied significantly during the course of the day, with the highest concentrations occurring during the nighttime. In contrast, in rural SW Chowchilla PM10 levels did not vary much within a day. Chemical composition data indicate diurnal variations in ammonium nitrate were the primary cause of the PM10 variations in SW Chowchilla. The rise in PM10 concentration in Fresno corresponded mostly to significant nighttime peaks in vegetative burning, mobile sources, and excess organic carbon (Magliano et al. 1999).

Figure 6.47. Particle size distributions for nitrate, organic carbon and soil during a winter episode in Bakersfield



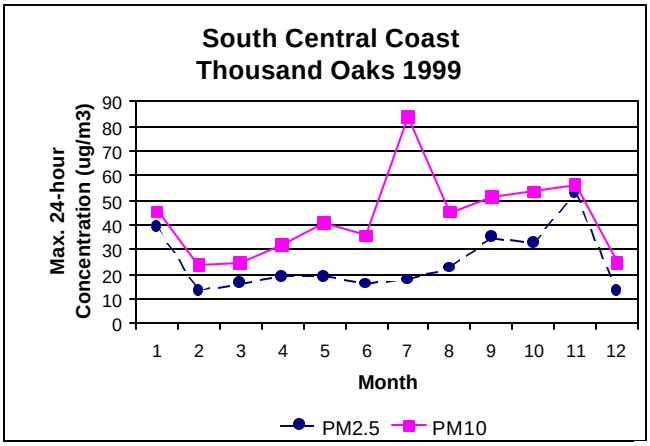
Data for Figure 6.47 are from air samples collected with a Micro-Orifice Uniform Deposit Impactor (MOUDI) sampler at Bakersfield during IMS95 (Chow et al. 1997). The size distribution of nitrate particles peaked between 1 and 1.78 um. Organic carbon particles appeared in both smaller (<0.054 μm) and larger (peak between 0.37 and 1 um) stages. The ultrafine carbon particles (< 0.08 μm) result from direct emissions from combustion sources or from the condensation of gases cooled down soon after they are emitted. The soil components were concentrated mainly on the larger particle size fractions (>3.16 μm), the coarse fraction of PM10.

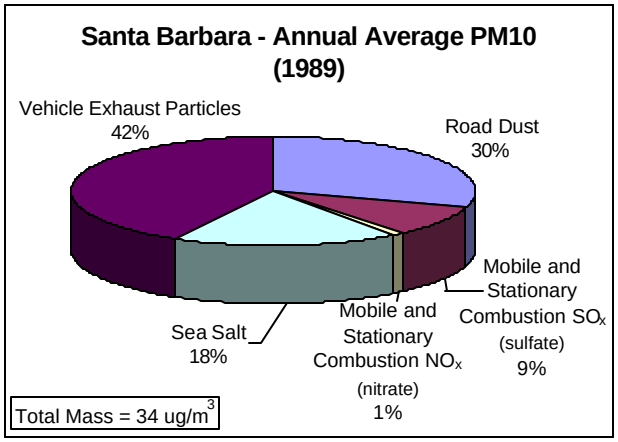
In the South Central Coast Air Basin, particulate levels exceeded the 24-hour State PM10 standard 60 times in the1998-2000 period. Neither of the federal PM2.5 standards or the State annual PM10 were exceeded in the last few years. Eighty percent of the 24-hour PM10 observations were below 30 to 35 $\mu g/m^3$ and 80% of the 24-hour PM2.5 measurements were under 10 to 15 $\mu g/m^3$.

Figure 6.48 illustrates the monthly variation of the maximum daily PM10 and PM2.5 concentrations at Thousand Oaks in 1999. PM10 concentrations tend to peak in the summer, while highest PM2.5 levels occur in November and January.

Figure 6.48. Monthly variation of maximum 24-hour PM10 and PM2.5 levels in Thousand Oaks

Figure 6.49. Sources contributing to annual average PM10 levels in Santa Barbara

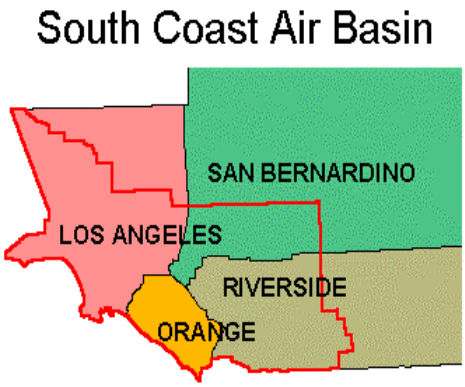




*The monitors used to measure PM10 and PM2.5 are different and occasionally recorded concentrations of PM2.5 which are greater than PM10.

Based on the 2000 annual PM10 emission inventory, the major contributors of directly emitted particles in the South Central Coast Air Basin are paved and unpaved road dust, dust from farming operations, and residential and waste burning. This region can also have significant seasonal wildfire emissions

Data for Figure 6.49 are form source apportionment analysis performed for the 1989 Santa Barbara County PM10 Study (Chow et al. 1996). The constituents shown can vary daily and from year to year depending on factors such as meteorology and which particulate sources are most active. On an annual basis, in the city of Santa Barbara, vehicle exhaust is the major contributor to PM10 levels. Sea salt and road dust also contribute significantly. Secondary ammonium nitrate and sulfate are relatively small contributors.



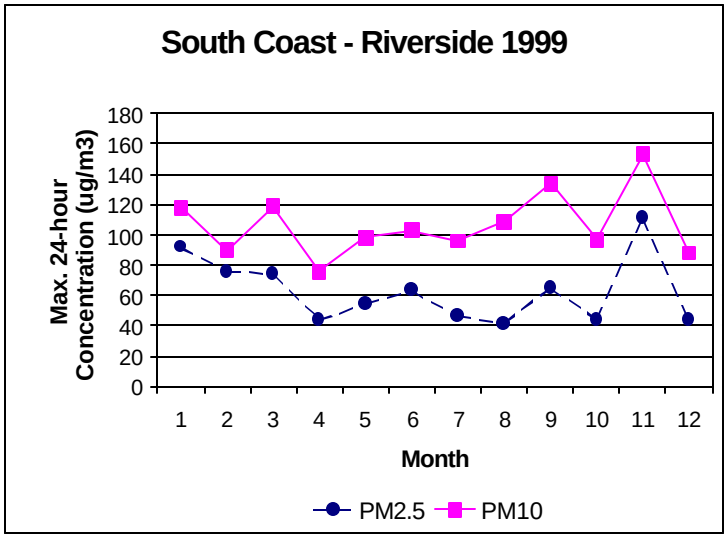
In the South Coast Air Basin, particulate levels exceeded the 24-hour State PM10 standard 197 times in the 1998-2000 period, and 38 PM2.5 observations over the 24-hour federal PM2.5 standard were recorded in the 1999-2000 period. Particulate levels also exceeded both the State PM10 and federal PM2.5 annual standards. The South Coast recorded some of the highest levels of PM2.5 in the State – almost twice the level of the standard. Eighty percent of the 24-hour PM10 observations were below 65 to 80 $\mu g/m^3$ and 80% of the 24-hour PM2.5 measurements were below 35 to 40 $\mu g/m^3$.

Figure 6.50 illustrates the monthly variation of the maximum daily PM10 and PM2.5 concentrations in Riverside in 1999. Both PM10 and PM2.5 concentrations exhibit no distinct seasonal pattern, with high concentrations throughout the year.

Data obtained from the Long Beach dichotomous sampler in 1999 indicate the PM2.5 portion of PM10 ranges from 30% to 96%. The average PM2.5 portion of PM10 from November to February is 59% dropping to 45% from February to October.

The 2000 annual PM10 emission inventory shows that the major sources of directly emitted particulates in the South Coast Air Basin are paved road dust, unpaved road dust, construction related dust, and the general categories of motor vehicle emissions and industrial emissions.

Figure 6.50. Monthly variation of maximum 24-hour PM10 and PM2.5 levels in Riverside



*The monitors used to measure PM10 and PM2.5 are different and occasionally recorded concentrations of PM2.5 which are greater than PM10.

Data for the illustrations below are from the source apportionment analysis that the South Coast Air Quality Management District (SCAQMD) performed for the 1997 Air Quality Management Plan. SCAQMD collected samples during a one-year special study from January 1995 to February 1996 as part of the PM10 Technical Enhancement Program (SCAQMD, 1996).

Figure 6.51 shows that on an annual basis, in Central Los Angeles, dust from roads and construction is the major contributor to ambient PM10. This is not the case for the episode on November 17, 1995 (Figure 6.52). In both cases, NOx and SOx emitted from mobile and stationary combustion sources, combined with ammonia, contribute significantly. Vehicle exhaust particles and emissions from other carbon sources also contribute to both annual and episodic ambient PM10 levels.

On an annual basis, in Rubidoux, dust from roads and construction is the major contributor to ambient PM10 (Figure 6.53). In contrast, as shown in Figure 6.54, dust was a minor contributor to the PM10 episode on November 17, 1995. In both cases, NOx emitted from mobile and stationary combustion sources, combined with ammonia, contributes significantly. Vehicle exhaust particles and emissions from other carbon sources also contribute to both annual and episodic ambient PM10 levels.

Figure 6.51. Sources contributing to annual average PM10 levels in Central Los Angeles

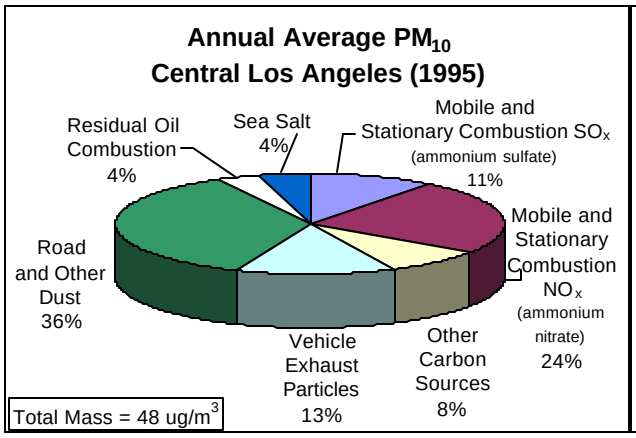


Figure 6.52. Sources contributing to PM10 levels during a November episode in Central Los Angeles

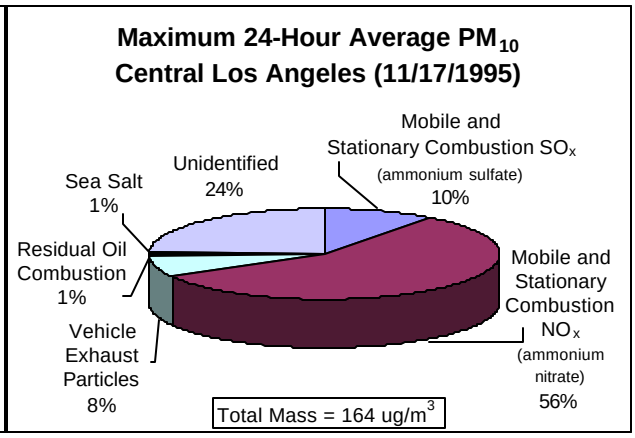


Figure 6.53. Sources contributing to annual average PM10 levels in Rubidoux

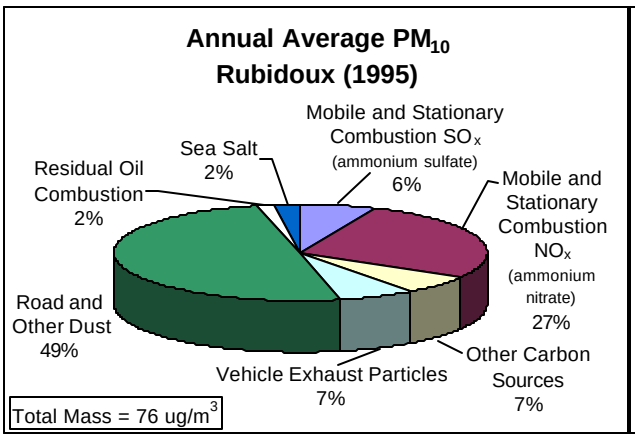
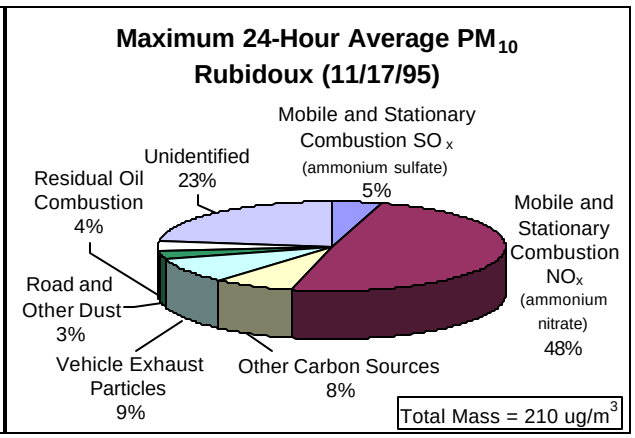


Figure 6.54. Sources contributing to a PM10 levels during a November episode in Rubidoux



6.4 Ambient Air Quality Population Exposure

6.4.1 Introduction

This section addresses two main questions: 1) what percent of the population in each air basin is exposed to ambient concentrations over the levels of the current and revised State PM10 standards, the current federal PM2.5 standards and the new State PM2.5 standards. 2) what is the population weighted average PM10 and PM2.5 concentration in each air basin?

We recognize that PM can vary in a small spatial scale. Our exposure analysis is limited by the number of PM monitors in the State and their geographic distribution in relation with location to the population. In addition, research on which PM sizes (e.g., coarse, fine, ultrafine) and which compounds of PM are the most toxic is an on-going effort at laboratories in the U.S. and abroad. Studies on the spatial distribution of specific PM compounds and particle size number are ongoing at the Fresno and Los Angeles Supersites. Samples to study these variables in the San Joaquin Valley were collected as part of the 2000-2001 California Regional PM10/PM2.5 Study, and are now being analyzed. We realize that these points need to be considered in future PM standard reviews.

6.4.2 Ambient Air Quality Exposure Model Details and Assumptions

The basic procedure for determining exposure was first adopted by the ARB in 1993 to fulfill the requirements of section 39607(f) of the Health and Safety Code. Full details are provided in *Guidance for Using Air Quality-Related Indicators in Reporting Progress in Attaining the State Ambient Air Quality Standards* (ARB 1993). For this application, ambient PM concentrations and population counts were associated by census tract and merged to assemble a distribution of exposures to different concentrations of PM.

Concentrations of many air pollutants including particulate matter change significantly from one place to another. PM10 concentrations may be well under the State standard in one location but above the standard less than 10 kilometers away. Accordingly, population exposures tend to be more accurate when the population data used to estimate them are highly resolved geographically.

Population counts by census tract provide a convenient basis for determining population exposures to air pollutants. A typical census tract contains several thousand people. Densely populated areas have many census tracts, while sparsely populated regions have very few.

Air pollutant data from a network of air quality monitors are used to determine appropriate values at census tracts that lie between them. The concentration for a census tract is a weighted average of the concentrations at all monitors within a maximum allowed distance. For the present analyses of PM10 and PM2.5, the maximum distance was 50 kilometers (75 km in the Great Basin Valleys Air Basin). A small number of census tracts are more than 50 km from any PM monitor, so their populations were not included in the analyses. The population numbers will be affected slightly by different choices for the maximum distance.

The weight assigned to each monitor is the inverse square of its distance from the census tract. In this way, close monitors are more influential than distant monitors are. Geographical features, such as mountain ranges, were not used in the model

6.4.3 Data Used

Ambient PM air quality data were extracted from AIRS on May 18, 2001. Exposure calculations were performed for three metrics: for PM10: annual arithmetic mean (AAM), annual geometric mean (AGM), and peak 24-hour concentration, represented by the Expected Peak Day Concentration (EPDC). For PM2.5 we performed calculations for the annual arithmetic mean and the EPDC. PM10 data from 1998 through 2000 and PM2.5 data from 1999 through 2000 were obtained from all monitors in the State meeting quality assurance criteria for valid data. For each metric, different numbers of monitors were available which met the specified validity criteria. Therefore the population represented for each metric is slightly different. For PM10 the population used in the analysis represented 99 percent of the 1990 statewide total population, while for PM2.5 it ranged from 62% to 66%, due to smaller number of monitors available. For variations among air basins see Appendices 6-G1 to 6-G3 and 6-H1 to 6-H2.

As mentioned in section 6.1, the EPDC for a monitoring site is the peak 24-hour PM10 (or PM2.5) concentration expected to occur no more than once per year. The EPDC is a highly precise estimate of the 99.7^{th} percentile ($364/365^{th}$ percentile) of the 24-hour PM10 (or PM2.5) concentrations measured at the monitoring site. Since the sampling frequency for PM10 concentrations is usually once every six days and for PM2.5 it varies by monitoring site (once every six days, once every three days or daily), the method used for calculating the EPDC automatically compensates for sampling frequencies that are less than daily. To calculate the EPDC, we use the highest twenty percent of all measurements during the last three years. An "exponential-tail" model is used for this purpose (Larsen and Nystrom, 1992; Breiman et al., 1978). The computer program to determine the EPDC is available to the public upon request (Contact: Larry Larsen, ARB).

1990 census data reported by census tract were used as the 2000 data were not yet available in the census tract format. The census data contains the shape, size, and centroid of each census tract, as well as the population count.

6.4.4 Discussion of the Ambient Air Quality Exposure Model Results

The detailed output of the exposure model for each of the three PM10 metrics is provided in Appendices 6-E1 to 6-E6 and for the PM2.5 metrics in Appendices 6-F1 to 6-F6. For each metric there is a statewide summary as well as a summary by air basin. For the PM2.5 AAM, the concentration data are shown in 2 $\mu g/m^3$ and in 5 $\mu g/m^3$ increments with the associated population exposed to concentrations within that range. For the PM2.5 EPDC and the three PM10 statistics, the concentration data are shown in 5 $\mu g/m^3$ increments. An additional column is provided to indicate the percent of the population that is above the relevant standards. Table 6.5 summarizes the results of the PM10 statewide assessment.

Table 6.5. Population in the State Exposed to PM10 Levels above the Current State Standards.

Area	Above Current PM10 Standards				Above Revised Annual PM10 Standard		Pop. Exposed to Revised Annual Std. – Pop. Exposed to Current Annual Std.	
	24-hour EPDC > 50 μg/m^3		Annual Geometric Mean > 30 μg/m^3		Annual Arithmetic Mean > 20 μg/m^3			
	Percent of Population Exposed	1990 Population. Exposed[(1)] (x100,000)	Percent of Population Exposed[(1)]	1990 Population. Exposed[(1)] (x100,000)	Percent of Population Exposed[(1)]	1990 Population. Exposed[(1)] (x100,000)	Percent of Population	1990 Population (x100,000)
Great Basin Valleys	100	0.29	0	0	22.5	0.03	22.5	.03
Lake County	0	0	0	0	0	0	0	00
Lake Tahoe	100	0.39	0	0	66.7	0.26	66.7	.26
Mojave Desert	100	3.29	7.9	0.23	51.5	1.35	43.6	1.12
Mountain Counties	100	3.27	0	0	70	2.17	70	2.17
North Central Coast	92.4	5.75	0	0	78.7	4.90	78.7	4.90
North Coast	84.8	1.72	0	0	24.7	0.69	24.7	0.69
Northeast Plateau	51.8	0.19	0	0	24.3	0.09	24.3	0.09
Sacramento Valley	100	20.12	2.3	0.47	93.4	18.78	91.1	18.31
Salton Sea	100	3.45,	91.8	3.21,	100	3.36	8.2	0.15
San Diego County	100	24.67	36.1	8.90	100	24.67	63.9	15.77
San Francisco Bay Area	100	58.77	0	0	68.3	40.14	68.3	40.14
San Joaquin Valley	100	25.60	68.9	18.28	100	25.60	31.1	7.32
South Central Coast	86.6	10.87	0	0	74	9.34	74	9.34
South Coast	100	128.62	90.6	116.48	100	128.61	9.4	12.13
Statewide	98.9	287.00	50.7	147.57	89.5	259.99	38.8	112.42

(1) This represents the percent of the 1990 population used in the exposure analysis. The total population used in the analysis varied by metric, since the number of monitors with data meeting specified validity criteria was different for each metric (Appendices VI-G1 to VI-G3).

Figures 6.55, 6.56 and 6.57 show the distribution of the statewide population exposed to different ambient PM10 concentration ranges.

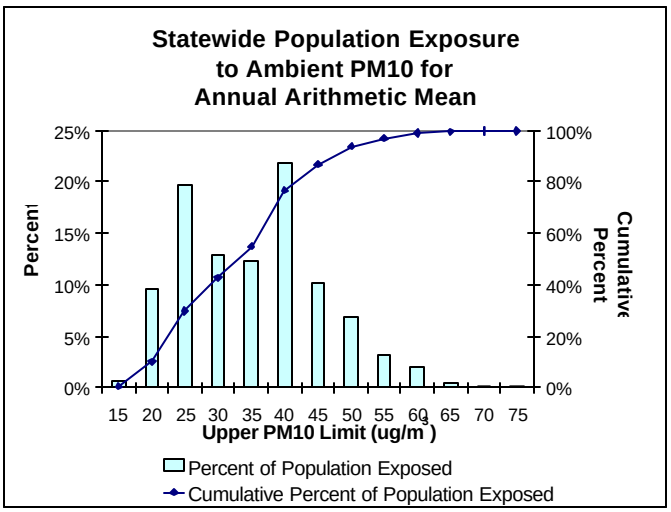


Figure 6.55. Estimated percentages of the statewide population exposed to various annual arithmetic mean levels of ambient (outdoor) PM10.

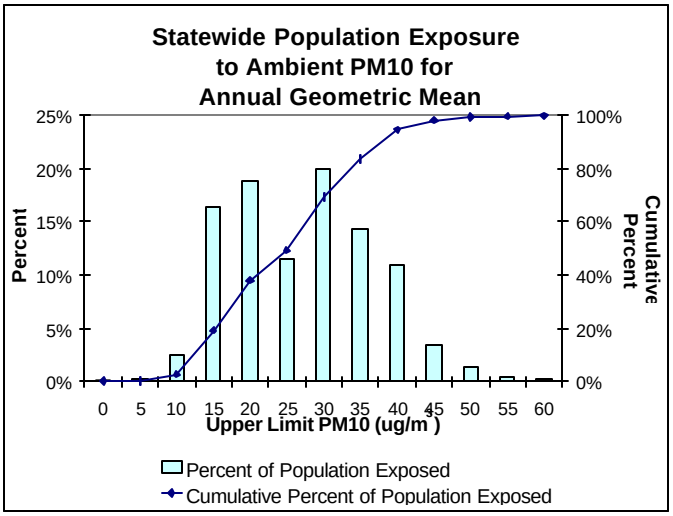


Figure 6.56. Estimated percentages of the statewide population exposed to various annual geometric mean levels of ambient (outdoor) PM10.

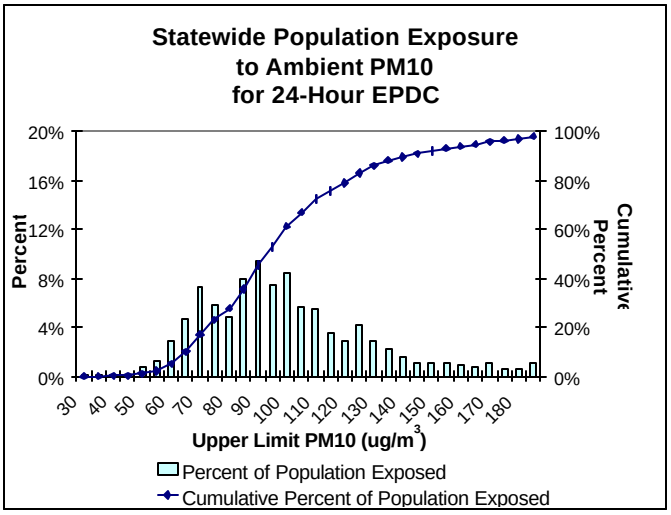


Figure 6.57. Estimated percentages of the statewide population exposed to various 24-hour average levels of ambient (outdoor) PM10.

The AAM statistics show that 57% of the statewide population is exposed to ambient PM10 concentrations over 30 $\mu g/m^3$, while 33% is exposed between 20 and 30 $\mu g/m^3$. The AGM statistics indicate 47% of the people in the State are exposed to annual ambient PM10 levels between 15 and 30 $\mu g/m^3$. Based on the EPDCs, essentially the whole State has PM10 levels exceeding the 24-hour State PM10 standard.

The air basin statistics in Appendices 6-E1 to 6-E3 show that for the two forms of the PM10 annual average, the percent of the population exposed to concentrations of 30 $\mu g/m^3$ or more is highest in the South Coast (over 90%), Salton Sea (over 90%), and San Joaquin Valley (over 68%). San Diego follows with over 36% of its population exposed to annual PM10 levels exceeding the State annual PM10 standard. Based on AAMs, less than 12% of the population in the Great Basin Valleys, Mojave Desert, North Central Coast, South Central Coast, and Sacramento Valley are exposed to concentrations equal to or above 30 $\mu g/m^3$. But, based on the AGMs, less than 8% of the population living in the Mojave Desert and San Joaquin Valley are exposed to PM10 levels over 30 $\mu g/m^3$, while no one in Great Basin Valleys, North and South Central Coast is exposed over these levels.

The 24-hour EPDC statistics show that essentially the entire State has PM10 levels exceeding the 24-hour State PM10 standard. Lake County is an exception, with its entire population exposed to concentrations below the current standard. Annual Geometric Mean values, show that about half of the State's population is exposed to annual average PM10 concentrations over the current annual PM10 State standard of 30 $\mu g/m^3$. A much larger fraction (90%) of the State's population is exposed to annual average levels over the revised annual PM10 standard of 20 $\mu g/m^3$, as shown by the Annual Arithmetic Mean statistics. A sizable portion of the population (36% to 92%) in four air basins (Salton Sea, San Diego, San Joaquin Valley, and South Coast), 2% of the population in Sacramento, and 8% of the population in Mojave Desert is exposed to annual average PM10 concentrations over the current State annual PM10 standard. In contrast, a significant fraction (23%-100%) of the total number of inhabitants in all air basins, with the exception of Lake County, are exposed to ambient PM10 levels over the revised annual State standard. The revised State annual PM10 standard would protect an additional 39% of the State population included in the study or 11 million persons over the current annual standard.

Table 6.6 summarizes the results of the PM2.5 assessment.

Table 6.6. Population in the State Exposed to Ambient PM2.5 Levels above the Current Federal and New State Standards.

Area	Above Current Federal PM2.5 Standards				Above New State PM2.5 Standards				Pop. Exp. to. New State Std – Pop. Exp. to. Federal Std			
	24-hour EPDC > 65 μg/m^3		Annual Mean > 15 μg/m^3		24-hour EPDC > 25 μg/m^3		Annual Mean > 12 μg/m^3		24-hour		Annual Mean	
	Pop. Exp.[(1)] (%)	1990 Pop. Exp.[(1)] (x10^5)	Pop. Exp.[(1)] (%)	1990 Pop. Exp.[(1)] (x10^5)	Pop. Exp.[(1)] (%)	1990 Pop. Exp.[(1)] (x10^5)	Pop. Exp.[(1)] (%)	1990 Pop. Exp.[(1)] (x10^5)	Pop. Exp.[(1)] (%)	1990 Pop. Exp.[(1)] (x10^5)	Pop. Exp.[(1)] (%)	1990 Pop. Exp.[(1)] (x10^5)
Great Basin Valleys	20.6	0.03	0	0	20.6	0.03	0	0	0	0	0	0
Lake County	0	0	0	0	0	0	0	0	0	0	0	0
Lake Tahoe	0	0	0	0	100	0.26	0	0	100	0.26	0	0
Mojave Desert	0	0	0	0	100	1.34	0	0	100	1.34	0	0
Mountain Counties	17.2	0.13	0	0	100	0.76	12.5	0.27	82.8	0.63	12.5	0.27
North Central Coast	0	0	0	0	69.5	2.75	0	0	69.5	2.75	0	0
North Coast	0	0	0	0	100	1.21	0	0	100	1.21	0	0
Northeast Plateau	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sacramento Valley	99.0	13.24	30.5	0.41	100	13.37	49.3	6.68	0.1	0.13	15.8	2.66
Salton Sea	7.4	0.17	21.4	0.50	100	2.31	51.3	1.20	92.6	2.14	29.9	0.70
San Diego County	0	0	63.4	10.43	100	16.44	100	16.46	100	16.44	36.6	6.03
San Francisco Bay Area	90.7	35.53	8.6	0.34	100	39.18	58.4	22.88	9.3	3.65	49.8	22.54
San Joaquin Valley	100	17.12	89.3	14.43	100	17.12	98.9	17.49	0	0	9.6	3.06
South Central Coast	0	0	0	0	100	7.18	59.2	4.96	100	7.18	0	0
South Coast	99.9	85.24	98.4	83.95	100	85.33	98.8	84.74	0.1	0.11	0.4	0.79
Statewide	80.7	151.30	60.6	113.67	96.5	180.93	82.5	154.60	15.8	29.63	21.8	40.93

(1) This represents the percent of the 1990 population used in the exposure analysis. The total population used in the analysis varied by metric, since the number of monitors with data meeting specified validity criteria was different for each metric (Appendices VI-H1 to VI-H2).

Figures 6.58 and 6.59 show the distribution of the statewide population exposed to different PM2.5 concentration ranges.

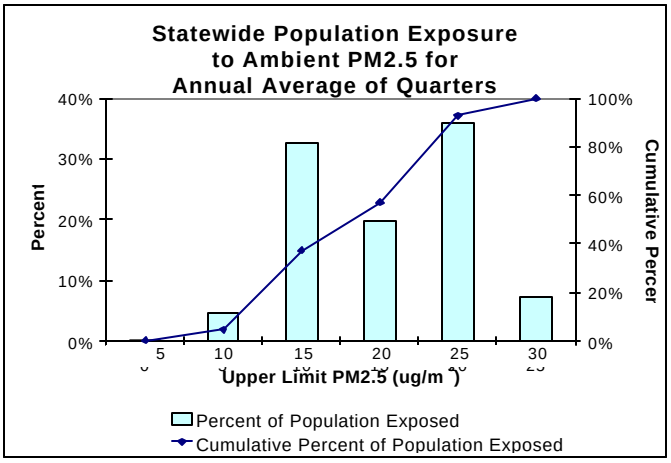


Figure 6.58. Estimated percentages of the statewide population exposed to various annual average of quarter mean levels of ambient (outdoor) PM2.5.

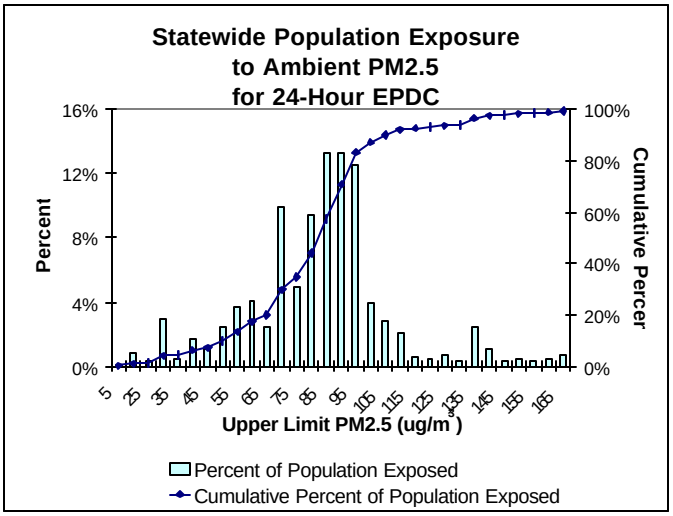


Figure 6.59. Estimated percentages of the statewide population exposed to various 24-hour average levels of ambient (outdoor) PM2.5.

The 24-hour EPDC statistics show that 80% of the Statewide population is exposed to PM2.5 levels above the current federal 24-hour PM2.5 standard of 65 $\mu g/m^3$, while 97% of the Statewide population is exposed to ambient PM2.5 concentrations over 25 $\mu g/m^3$, the level of the new State 24-hour PM2.5 standard. The fraction of the population in the seven air basins with 24-hour EPDCs over 65 $\mu g/m^3$ ranges from 7% in the Salton Sea to 100% in the South Coast. With the exception of Lake County and Northeast Plateau, all air basins have at least 20% of their population exposed to 24-hour concentrations over the new State 24-hour PM2.5 standard. The new State 24-hour standard would protect an additional 16% of the population in the State (close to 3 million persons) over the protection offered by the current federal standard.

Annual Mean statistics show that 61% of the statewide population corresponding to 11 million inhabitants (21%-98% in six air basins) is exposed to annual average PM2.5 concentrations over the current federal standard of 15 $\mu g/m^3$. In contrast, 83% of the State's population (15 million persons) is exposed to PM2.5 levels over the new State annual PM2.5 standard of 12 $\mu g/m^3$. In addition to the protection rendered by the current federal annual PM2.5 standard the new State annual standard would provide significant additional protection to people living in

the Mountain Counties, Salton Sea, San Francisco Bay Area, and the South Central Coast air basins.

From the data presented in Appendices 6-E1 to 6-E3 and 6-F1 to 6-F2, we estimated PM10 and PM2.5 annual and 24-hour population weighted concentrations for each air basin. We assumed that the population in a specific concentration bin is exposed to the midpoint concentration in that bin. Table 6.7 lists the results of the analysis for PM10.

The annual statistics show that in three air basins - South Coast, San Joaquin Valley, and Salton Sea - the basinwide population weighted annual geometric mean PM10 concentrations are above 30 $\mu g/m^3$. The Salton Sea has the highest PM10 annual averages in the State (58 $\mu g/m^3$). The South Coast (with an average of 37 $\mu g/m^3$) and the San Joaquin Valley (with 34.3 $\mu g/m^3$) follow. With the exception of Great Basin Valleys and Lake County, all air basins have population weighted annual arithmetic means over 20 g/m^3.

The results of the 24-hour PM10 concentration analysis show that, with the exception of Lake County, the rest of the State has basinwide population weighted average EPDCs over 50 $\mu g/m^3$. The desert areas - Great Basin Valleys and Salton Sea - have the highest EPDCs, around 300 $\mu g/m^3$. The South Coast and San Joaquin Air Basins follow, with EPDCs above 100 $\mu g/m^3$. The rest of the air basins have EPDCs between 50 and 100 $\mu g/m^3$.

Table 6.7. Estimated Population Weighted Annual Means and 24-hour EPDC for PM10

Population Weighted Metrics for PM10			
Air Basin	Annual Arithmetic Mean (ug/m^3)	Annual Geometric Mean (ug/m^3)	Expected Peak Day Concentration (ug/m^3)
Great Basin Valleys	16.7	11.1	337.1
Lake County	10.8	9.2	40.8
Lake Tahoe	20.8	17.5	69.2
Mountain Counties	23.0	15.8	86.4
Mojave Desert	21.6	23.4	69.4
North Coast	17.5	15.9	59.6
North Central Coast	24.2	22.7	73.0
Northeast Plateau	13.0	9.7	61.2
South Coast	40.7	37.0	105.3
South Central Coast	23.0	21.4	62.4
San Diego	28.8	28.8	72.8
San Francisco Bay Area	21.7	19.4	79.4
San Joaquin Valley	39.5	34.3	158.8
Salton Sea	70.2	58.0	299.9
Sacramento Valley	24.5	21.0	100.6
Statewide	33.1	30.0	100.4

Table 6.8 lists the population weighted statistics calculated for PM2.5. The PM2.5 annual means show that three air basins – South Coast , San Diego and San Joaquin Valley - have basinwide population weighted averages above 15 $\mu g/m^3$, while three additional air basins – San Francisco Bay Area, Salton Sea and Sacramento Valley – have population weighted annual measn over 12 $\mu g/m^3$.

Four air basins have population weighted average 24-hour EPDCs over 65 $\mu g/m^3$. The San Joaquin Valley has the highest EPDC (132 $\mu g/m^3$) of about twice the level of the standard, followed by the Sacramento Valley (93.1 $\mu g/m^3$), South Coast (87 $\mu g/m^3$), and San Francisco Bay Area (71 $\mu g/m^3$). Seven additional air basins have population weighted EPDCs over 25 $\mu g/m^3$

Table 6.8. Estimated Population Weighted Annual Means and 24-hour EPDC for PM2.5

Population Weighted Metrics for PM2.5		
Air Basin	Annual Arithmetic Mean (ug/m^3)	Expected Peak Day Concentration (ug/m^3)
Great Basin Valleys	7.5	18
Lake County	2.5	17.5
Lake Tahoe	7.5	27.5
Mountain Counties	9.8	44.9
Mojave Desert	12.3	27.6
North Coast	7.5	35.6
North Central Coast	7.5	23.8
Northeast Plateau	NA	NA
South Coast	22.3	87
South Central Coast	11.8	43.1
San Diego	15.7	54.3
San Francisco Bay Area	12.9	71
San Joaquin Valley	20.3	132
Salton Sea	13.6	45.3
Sacramento Valley	12.8	93.1
Statewide	18.2	81.4

6.5 Characterization of Personal and Indoor Exposures

6.5.1 Personal PM Exposures

Peoples' actual exposures to PM, or their "personal exposures," have been shown in numerous studies to differ notably from outdoor PM concentrations measured at ambient monitoring stations, and often are much higher than outdoor PM levels. This is primarily due to people's close proximity to sources of PM throughout the day, especially PM sources inside of buildings, where people spend the large majority of their time. Personal PM exposures are estimated by

measuring pollutant concentrations in a person's breathing zone, the area near their nose and mouth, using portable instruments worn by the individuals. Because people often spend time in enclosed environments close to PM sources such as smoking, cooking, and cleaning activities, personal concentrations also are often higher than indoor PM concentrations measured at fixed locations in the indoor environment. The results of recent studies of personal and indoor concentrations of PM most relevant to understanding Californians' exposures are presented in Table 6.9. and discussed below.

The first major study to demonstrate personal PM concentrations above indoor and outdoor concentrations was the Particle Total Exposure Assessment Methodology (PTEAM) Study, conducted in 1990 in Riverside, California. Investigators measured PM10 and PM2.5 for 12-hour daytime and nighttime periods in 178 homes during September to November. They found 12-hour daytime personal PM10 concentrations to be about 50% higher than simultaneously measured daytime residential indoor or outdoor concentrations. Daytime personal concentrations averaged 150 $\mu g/m^3$, while indoor and outdoor concentrations both averaged about 95 $\mu g/m^3$ (Clayton et al. 1993, Ozkaynak et al. 1996*a*,*b*). Most importantly, 12-hour daytime personal PM10 concentrations exceeded the California 24-hour ambient air quality standard of 50 $\mu g/m^3$ for about 90 percent of the monitoring days and exceeded the federal PM10 standard of 150 $\mu g/m^3$ for 25 percent of the monitoring days. During nighttime, personal PM10 concentrations decreased and were similar to concurrent indoor and outdoor concentrations (roughly 80 $\mu g/m^3$), reflecting the importance of the proximity of people to PM sources during normal activities in determining personal exposure concentrations.

The PTEAM study used a probability sampling design, in which study subjects were carefully chosen to ensure that the sampled population represented the city of Riverside as a whole. These types of studies are large and expensive, and therefore not frequently performed. Three other probability studies of personal PM levels have been performed since PTEAM; in two, investigators found higher personal concentrations than corresponding outdoor concentrations, while outdoor concentrations were not measured in the third study. In Toronto, the investigators found average personal and outdoor PM2.5 concentrations of 28 $\mu g/m^3$ and 15 $\mu g/m^3$, respectively (Pellizzari et al. 1999). For PM10, average personal and outdoor concentrations were 68 $\mu g/m^3$ and 24 $\mu g/m^3$, respectively. In Basel, Switzerland, average personal and residential outdoor PM2.5 concentrations were measured at 24 $\mu g/m^3$ and 19 $\mu g/m^3$, respectively; for nonsmokers, average personal and residential outdoor concentrations were both 18 $\mu g/m^3$, showing the large impact smoking can have on personal PM2.5 exposures (Oglesby et al. 2000). In Mexico City, personal PM10 concentrations, averaged 97 $\mu g/m^3$, but no outdoor measurements were available for comparison (Santos-Burgoa et al. 1998).

Many smaller-scale particle exposure studies that have not used probability sampling design have been performed, in both the general population and in populations sensitive to PM such as the elderly or patients with chronic obstructive pulmonary disease (COPD). Recent U.S. studies of personal PM10 and PM2.5 in which all or most of the study subjects were elderly and/or COPD patients include Evans et al. 2000, Linn et al. 1999, Rojas-Bracho et al. 2000, Sarnat et al. 2000, and Williams et al. 2000*a,b,c*. Like PTEAM, these smaller studies have also shown that personal exposures can be higher than simultaneously measured residential indoor and outdoor concentrations.

Table 6.9. Recent Personal-Ambient Air Particulate Matter Exposure Studies

Reference	Location and population	No. of subjects	Study period	Age range	PM Size[a]	N[b]	Concentration: arithmetic mean (SD); all in μg/m³			Personal-ambient correlation[d]	
							Personal	Indoor	Ambient	Value	Type
Probability-based studies											
Clayton et al., 1993	Riverside, CA PTEAM	178	08-11/90	10-70	PM10-day	171	150 (84)	95 (61)	91 (48)	0.37[c]	P
					PM10-night	168	77 (40)	63 (37)	77 (48)	0.54[c]	P
Pellizzari et al., 1999	Toronto, Canada	732	11/95-10/96	>16	PM2.5	922	28	21	15	0.23	P
					PM10	141	68	30	24	NA[f]	
Oglesby et al., 2000	Switzerland EXPOLIS	50	01/97-12/97	25-55	PM2.5	44	24 (17)	NA[f]	19 (12)	0.07	P
	Subset with no ETS exposure	20	01/97-12/97	25-55	PM2.5	20	18 (13)	NA[f]	18 (7)	0.21	P
Not probability-based, California studies											
Linn et al., 1999	Los Angeles; Elderly COPD	30	10/96-02/97	56-83	PM2.5	60	24 (15)	24 (15)	25 (15)[c]	0.26[e]	P
					PM10	59	35 (15)	33 (16)	40 (18)	0.22	P
Evans et al., 2000	Fresno , CA; Elderly	5	02/99	>60	PM2.5	56	13	9.7	22	0.41	P
	Fresno, CA; Elderly	16	04/99-05/99	>60	PM2.5	190	11	8.0	8.6	0.84	P
Not probability-based, recent United States studies											
Rojas-Bracho et al., 2000	Boston, MA; COPD	18	01-09/96, 01-02/97	38-60	PM2.5	224	22 (14)	18 (14)	14 (11)	0.61[g]	L
					PM10	225	37 (23)	32 (25)	22 (19)	0.35[g]	L
Sarnat et al., 2000	Baltimore, MD; Elderly	15	06-08/97	62-82	PM2.5	37	27 (14)	NA[f]	25 (12)	0.76	L
			06-08/97		PM10	37	34 (12)	NA[f]	34 (13)	0.64	L
			02-03/99		PM2.5	36	19 (11)	NA[f]	5.6 (49)	0.25	L
			02-03/99		PM10	36	28 (17)	NA[f]	7.5 (73)	0.53	L
Williams et al., 2000*a,b,c*	Baltimore, MD; Elderly, healthy and COPD	21	07/98-08/98	72-93	PM2.5	23	13	9.4	22	0.80	L

a-Refer to publication for measurement averaging times; most are 24 hr, 'day' refers to 12 hr daytime, 'night' refers to 12 hr nighttime.
b-Listed sample size for personal samples; see reference publication for sample size information for indoor and ambient samples.
c-Outdoor residential measurements substituted as ambient concentration data from a nearby monitoring site were unavailable.
d-Values are Pearson correlation coefficients unless otherwise noted; types are Pooled (P) or median Longitudinal (L).
e-Values were calculated as the square root of R^2 from mixed model regression
f-NA indicates information was not available
g-Spearman rank correlations

However, these studies of sensitive populations have generally shown smaller personal-outdoor or personal-ambient station differences than those found in the PTEAM study, and stronger correlations with ambient levels. This appears to be in part due to the reduced activity levels of many of the study subjects who have pre-existing lung disease (for example, fewer cleaning and cooking activities), as well as the use of longitudinal study designs (multi-day monitoring) which are more likely to reflect personal to outdoor relationships. These studies have also generally found smaller differences between personal and ambient levels for PM2.5 as compared to PM10, and that correlations between ambient and personal levels are generally higher for PM2.5 than for PM10. Notable exceptions exist, however, such as the results from two recent studies of elderly subjects in Fresno, California and Baltimore, Maryland (Evans et al. 2000, Williams et al. 2000*a,b,c*) where personal levels were lower than ambient levels, on average. Also, another study of elderly subjects in Baltimore found a lower correlation between personal and ambient levels of PM2.5 than for PM10 (Sarnat et al. 2000). These results are likely explained by the reduced activity level of the study participants; seasonal differences in ambient levels, ventilation practices, local variability, and the presence or use of fewer indoor PM sources; and multi-day monitoring.

There are few data available on personal PM2.5 concentrations in California, although non-smoking elderly subjects in Fresno (Howard-Reed, et al., 2000, Evans et al. 2000) and COPD patients in Los Angeles (Linn et al. 1999) have been studied. Because none of the PM2.5 studies have used a probability-based design, and although much information is currently being gathered about PM2.5 in California, the extent to which Californians' personal exposures to PM2.5 are elevated above ambient concentrations is largely unknown.

Because measured personal exposures to PM are often greater than estimates based on time-weighted averages of concurrent indoor and outdoor PM levels, researchers have identified a "personal cloud" of PM. It is thought that this "personal cloud" is due to an individual's activities (which can generate or resuspend particles), their proximity to other activities that generate PM emissions, and their visits to non-monitored environments with elevated PM levels. Examples of activities that generate PM likely to contribute to elevated personal PM include smoking, cooking, cleaning, travel, some types of work, and playing on a carpeted floor.

In several PM exposure studies, researchers have estimated the magnitude of the "personal cloud." Wallace (2000*a*) reviewed several recent studies, and found that the personal cloud for PM10 for healthy persons, from children to the elderly, was often about 30 $\mu g/m^3$, but it ranged from 3-67 $\mu g/m^3$ among individuals. The personal cloud for PM2.5 was smaller, ranging from 6-27 $\mu g/m^3$. The personal cloud for COPD patients in two studies was considerably smaller than that for the general population: 6-11 $\mu g/m^3$ for PM10, and about 6 $\mu g/m^3$ for PM2.5; this reduction is probably attributable to the reduced level of personal activities of the study subjects, and the lack of significant indoor PM sources in their homes.

The sources and composition of the personal clouds were not identified in these studies. Personal activities that resuspend particles from clothes, furnishings, and other surfaces may be an important source, in addition to the activities listed above. Recent studies have identified other factors that could affect the personal cloud composition and size fractions, such as the use of cosmetics and antiperspirants (Conner, et al., 2001), and the proximity and type of combustion sources such as incense burning and cooking (McBride et al. 1999; Fortmann et al. 2001).

In summary, in spite of the many studies cited, the ability to accurately estimate PM exposure concentrations for general populations, especially PM2.5 exposures, is still limited by the small number of probability design studies, the large amount of individual variability, and the limited seasonal coverage of the probability studies that have been conducted (which did not include

the important seasonal variations in air exchange rates; U.S. EPA 2001). The PTEAM study remains the only major probability sampling PM exposure study conducted in the U.S. and still provides the most relevant California PM10 exposure data, although it essentially covered just one season in one city. Representative PM2.5 data for all Californians are lacking.

6.5.2 Sources of Indoor PM

Indoor PM sources often increase particle concentrations inside a building above ambient concentrations, due to the trapping effect of the building shell. A key factor in the effectiveness of this trapping is the air exchange rate of the building, which tends to vary by season and is strongly affected by open windows and doors, mechanical ventilation, and building construction characteristics.

Outdoor air infiltration and indoor combustion sources such as smoking and cooking are typically the greatest sources of indoor PM (Wallace 1996; Ozkaynak et al. 1996*a*,*b;* Brauer et al. 2000; Abt et al. 2000; Fortmann et al. 2001). For example, through source apportionment the PTEAM investigators estimated that, on average, about 76% of the indoor PM2.5 mass and 66% of the indoor PM10 mass originated outdoors. They also estimated that, on average, 5% of PM2.5 and 4% of PM10 was attributed to tobacco smoking; 4% of PM2.5 and 5% of PM10 was attributed to cooking; and 14% of PM2.5 and 26% of PM10 were from unexplained sources (Ozkaynak et al. 1996*a*). Abt et al. (2000) and Long et al. (2000) found that the relative contribution of outdoor PM to indoor levels varied by particle size, with outdoor air generally contributing a majority of the smaller particles (less than 0.5 micrometers) measured indoors, while indoor sources contributed more to the larger (2-10 micrometers) size fraction. Thus, reductions in outdoor PM levels can have a major effect on the indoor concentrations.

For PTEAM homes with smokers, it was estimated that 30% of the PM2.5 mass and 24% of the PM10 mass came from smoking. For homes in which cooking occurred during the monitoring period, 25% of the PM2.5 and PM10 was estimated to come from the cooking activity (Ozkaynak 1996*b*). These results are consistent with those found in many previous indoor studies that have examined the impact of cigarette smoking on indoor PM levels, and led to subsequent studies of indoor cooking emissions that have confirmed the high impact that some cooking methods can have on indoor and personal PM levels (Abt et al. 2000; Wallace 2000*b*; Brauer et al. 2000; Fortmann et al. 2001). In a study of a variety of cooking activities using gas and electric stoves in a test home in northern California, kitchen PM10 levels ranged to more than 1400 $\mu g/m^3$ during frying, broiling, and baking activities (Fortmann et al., 2001). During use of the self-cleaning feature, oven cleaning resulted in kitchen PM10 levels up to 3661 $\mu g/m^3$, and indoor PM2.5 ranged to 2032 $\mu g/m^3$, while concurrent outdoor levels ranged only to 20 $\mu g/m^3$. The burning of wood, incense, and mosquito coils can also be important combustion sources of residential indoor PM, especially in the 2.5 µm size range and below (Brauer et al. 2000; Lofroth et al. 1991).

Physical generation or re-suspension of particles also can be an important PM source. Indoor surfaces such as carpets and draperies can attract and re-emit particles (Thatcher and Layton, 1995, Kamens et al. 1991). Particle concentrations from carpets can be high even in homes where good cleaning practices are used, and the particles can become re-entrained in the indoor air when people walk or play on the carpeted surface (Wallace 2000*a*; Roberts and Dickey 1995; Abt et al. 2000, Vette et al. 2001). Track-in of particles on shoes and by children and pets has also been shown to contribute significantly to indoor particle concentrations in residences (Roberts and Dickey 1995, Thatcher and Layton 1995). House dust particles have been found to include vapors, metals, and semi-volatile chemicals of intermediate vapor pressures, such as pesticides and polycyclic aromatic hydrocarbons (Rothenberg et al. 1989; Roberts and Dickey 1995; Lewis et al. 1999; U.S. EPA, 1999) that have their own toxic

properties. These contaminants are often adsorbed onto the surfaces of house dust particles, and are available for re-emission to the air and subsequent inhalation, and for dermal absorption and/or ingestion by children through floor contact and hand-to-mouth behavior (Lewis et al, 1994; Zartarian et al. 1998; Zartarian and Leckie, 1998). For toxics such as lead, floor dust levels can be a major determinant of exposure.

Biological contaminants such as fungi, bacteria, house dust mites, and pollen also can contribute to indoor particle concentrations, especially in buildings with moisture problems from flooding or roof leaks that have not been properly repaired. Many biological contaminants can trigger asthma attacks in sensitive individuals and cause other adverse health effects such as allergy symptoms, sinus and respiratory infections, headaches and irritant effects (NAS 1993; NAS 2000). Bioallergens, such as pollen, in outdoor air can also penetrate indoor spaces. Re-entrained road dust may be a particularly important source of bioallergens in both indoor and outdoor air (Miguel et al. 1998).

6.5.3 Relation of Personal PM Concentrations with Ambient Concentrations

Although much effort has been made to determine the relationship between outdoor and personal PM concentrations, no consistent predictive relationship has been found. Complicating factors include varying degrees of particle infiltration from outdoors, varying particle removal rates indoors, and the wide variety of peoples' activities and proximity to sources.

The complex relationships between personal exposures and outdoor concentrations are reflected in the variable correlations found between personal PM10 concentrations and ambient concentrations. Correlations (r) of personal PM10 concentrations with ambient concentrations in studies utilizing a cross-sectional study design (each individual monitored for one day), including PTEAM, have been low, ranging from 0 to about 0.3 (Dockery and Spengler 1981, Sexton et al. 1984*a*,*b*, Spengler et al. 1985, Lioy et al. 1990, Clayton et al. 1993, Ozkaynak et al. 1996*b*). In these studies, investigators have generally collected personal exposure samples over durations of 12 or 24 hours.

However, for longitudinal studies with seven or more repeated measurements, correlations for a given subject between personal and outdoor concentrations are greater than for a cross section of subjects with a single measurement period (Wallace 1996, Wallace 2000*a*). Additionally, recent studies for PM2.5 have found stronger correlations for personal PM2.5 concentrations with outdoor particle concentrations than were found for PM10 in earlier PM10 studies. Rojas-Bracho et al. (2000) found that the median longitudinal Spearman correlation coefficient (r) between personal and outdoor PM2.5 concentrations for each individual over multiple days was 0.61 in Boston. Median longitudinal Pearson correlations (r) were 0.25 and 0.76 for winter and summer, respectively, in Baltimore (Sarnat et al. 2000). Average Pearson correlation coefficients (r) between personal and outdoor PM2.5 concentrations were 0.41 and 0.84 for the winter and spring phases of study in Fresno, respectively (Evans et al. 2000), 0.26 during the fall and winter in Los Angeles (Linn et al. 1999), and 0.89 in Baltimore (Williams et al. 2000*a*,*b*). However, because most of these studies used elderly and/or ill subjects, the correlations may be greater than would be seen for healthy individuals. This appears to be due to the participants' reduced rates of activities and mobility relative to the general population (see Table 6.9 for a description of the demographic group observed in each study), the absence of major indoor PM sources, and increased operation of heating, cooling, and ventilation systems (which usually have air filtration) (Rodes et al. 2001; Williams et al. 2000*a*).

Strong correlations between personal and outdoor concentrations have also been observed in two European studies. In their longitudinal study of 13 children in the Netherlands, Janssen et al. (1999) found longitudinal correlation coefficients between personal and outdoor PM10 of 0.75 for all children and 0.84 for children not exposed to environmental tobacco smoke.

Correlation coefficients for PM2.5 were 0.86 for all children and 0.92 when environmental tobacco smoke exposures were excluded. Personal concentrations averaged 28 $\mu g/m^3$, while outdoor concentrations measured 17 $\mu g/m^3$. In a study of elderly subjects with cardiovascular disease, the median Pearson correlation (r) for personal and outdoor PM2.5 was 0.79 in Amsterdam and 0.76 in Helsinki (Janssen et al. 2000).

In spite of the complex relationship between personal and outdoor PM concentrations, studies have shown outdoor PM to be a consistent and important contributor to overall PM exposure. Analysis of the results of personal exposure studies have estimated average outdoor contributions to personal PM mass exposures ranging from about 50% to 64% for PM10 (Ozkaynak et al. 1996*a*, Mage 1998) and to 75% or more for PM2.5 (Koutrakis et al. 1992, Mage 1998). Mage (1999) also found that variations in personal exposures of persons with similar lifestyles and no exposure to tobacco smoke were driven by variations in ambient PM concentrations. The work of Mage et al. (1999) and Wilson et al. (2000) attempts to show that indoor and personal PM concentrations reflect the "superposition" of an ambient-derived indoor PM component, which tracks outdoor concentrations, and a more variable indoor-derived PM component, which does not. Sarnat et al. (2000) showed that personal-to-ambient concentration correlations improve greatly with increasing air exchange rates. Findings such as these help explain why mortality and morbidity effects seen in epidemiology studies have been linked to ambient PM concentrations despite the sometimes poor correlations between personal and outdoor concentrations for a given population on a given day, such as is reflected in cross-sectional studies.

6.5.4 Contributions of Outdoor Sources of PM to Indoor Concentrations

Outdoor particles enter buildings and contribute to indoor concentrations. The rate at which particles infiltrate into indoor environments and the ratio of indoor to outdoor concentrations are dependent on many factors, especially the air exchange rate of the building, the use of operable windows and doors, and the aerodynamic size of the particles. In addition, outdoor concentrations measured outside of or near the building where indoor measurements are taken can vary considerably in relation to corresponding ambient levels measured at stationary ambient monitoring stations, especially for PM10 at higher concentrations (Ozkaynak et al. 1996*b*) .

Indoor PM10 concentrations in PTEAM were similar to nearby outdoor PM10 concentrations during the daytime, but slightly lower during the nighttime (Clayton et al. 1993). Indoor PM2.5 concentrations were similar to outdoor concentrations during the daytime, and lower during the nighttime. However, through source apportionment techniques, the PTEAM investigators estimated that, of the total indoor mass of particles, outdoor particles contributed 66% of the PM10 mass and 76% of the PM2.5 mass (Ozkaynak et al. 1996*a*,*b*).

Correlations (r^2) between indoor PM2.5 and nearby outdoor PM2.5 were estimated in studies of elderly subjects to be 0.93 (winter) and 0.75 (spring) in Fresno (Evans et al. 2000), and 0.96 in Baltimore (Williams et al. 2000a). In a study of four Boston homes with air exchange rates below 1.0 hr^{-1}, Abt et al. (2000) estimated that only 20-43 percent of indoor particles from 2-10 µm were from outdoors, while 63-92 percent of indoor PM from 0.02-0.3 µm were from the outdoors.

The outdoor-derived fraction of indoor PM is determined by several factors (e.g., air exchange rate, particle penetration, and deposition) and, under steady-state conditions, can be calculated from the following equation, assuming no indoor sources are present:

$$C_{out\text{-}in} = C_{out} [P\, a/(a + k)]$$

where P is the particle penetration factor, *a* is the air exchange rate of the building, k is the particle deposition rate, $C_{out\text{-}in}$ is the concentration of particles of outdoor origin in indoor air (i.e., those that have infiltrated indoors), and C_{out} is the concentration of particles in outdoor air. Both P and k are in large part dependent on particle size, making the solution to this equation dependent on the particle size fraction considered, except when *a* is high. The steady state modeling approach is shown here for simplicity, but dynamic modeling is generally needed because pollutant source emissions and ventilation are episodic and not constant.

Air exchange rate is the rate at which the air in an indoor air space is exchanged with the same volume of outdoor air. In residential buildings, air exchange rates vary widely depending upon building construction, opening of windows and doors, wind-and fan-induced pressure changes, and seasonal changes. A number of investigators have reported air exchange rates for homes in California (Ozkaynak et al. 1996*a*,*b*, Sheldon et al. 1993, Wilson et al. 1993, Pellizzari et al. 1999, Wilson et al. 1986). Representative values for the mean and standard deviation of air exchange rates in residential buildings in California have been estimated at 1.2 and 1.0, respectively, with a log normal distribution (Air Resources Board, 1998*b*), and have been measured as high as 5 or more air changes per hour.

The penetration factor denotes, for a given volume of air that enters the building, the fraction of the outdoor contaminant mass that moves through the building shell to the indoor space without interception. For residential buildings, the main entry routes of outdoor air are open windows and doors, cracks in the building shell, and mechanical ventilation systems such as swamp coolers, whole house fans, and central systems with substantial duct leakage. Penetration factors are calculated based on measurements of other parameters, mainly indoor and outdoor particle mass concentrations and air exchange rates, and can vary depending on the size fraction of PM being considered. The values of the penetration factor for PM2.5 in residences have generally been estimated in the range of 0.5 to 1.0 (Long et al. 2001; Suh et al. 1994, Koutrakis et al. 1992, Dockery and Spengler 1981, Ozkaynak et al. 1996*b*), with California studies showing penetration factors for PM2.5 and PM10 close to 1.0 (Ozkaynak et al. 1996*b*; Thatcher and Layton 1995). More recent field studies have found differences in penetration efficiencies among particles of different sizes, with larger sizes showing reduced penetration (Abt et al. 2000; Vette et al. 2001; Long et al. 2001), especially under conditions of low air exchange. Laboratory studies with simulated penetration and infiltration scenarios have generally supported and complemented the field results, although they are limited to leakage measurements (Mosley et al. 2001; Liu and Nazaroff 2001; Thornburg et al. 2001).

In public and commercial buildings, penetration depends on the size of the building, whether operable windows are present, and the presence or absence of a central HVAC system with filtration. A large, multi-story building with a central system and high efficiency filtration would generally have very low penetration and infiltration of particles of all sizes. At the other extreme, a small grocery or retail store with no central system and open windows and doors would be similar to many homes and have high penetration and infiltration due to the high air exchange rate with little interception of particles (Air Resources Board, 1998*b*).

In the process of entering an indoor environment, particle concentrations may be reduced by various mechanisms, including deposition, transformation, decay, decomposition, and adsorption. The cumulative effect of these processes is reflected in the particle deposition (removal) rate. Typically, particles of larger aerodynamic diameter have higher deposition rates. Values for the particle deposition rate for California homes, estimated as part of the PTEAM study, were 0.39 hr^{-1} for PM2.5 and 0.65 hr^{-1} for PM10 (Ozkaynak et al. 1996*b*). Other investigators have found a wider range of deposition rates for particles of different aerodynamic size, with the lowest deposition rates shown by particles in the 0.1 to 1.0 micrometer range (Thatcher and Layton 1995; Long et al. 2001; and others). Additionally, the indoor furnishings

and material surfaces can affect deposition, with rough "fleecy" materials collecting particles more than smooth, slick surfaces.

In summary, the contribution of outdoor PM to indoor PM concentrations can be substantial but highly variable. The transport of outdoor PM into a building's air volume and surfaces is very complex and varies greatly, depending on many factors through time for different particle size ranges. The indoor-outdoor relationships for PM also vary with outdoor PM concentrations, so that simple indoor-outdoor ratios do not reflect the true interrelationships. Using the PTEAM source apportionment results as an example from a large, population-based study in California, indoor PM can be expected to be comprised of about and ¾ outdoor PM10 and PM2.5, respectively. For elderly and ill persons in nursing homes, hospitals, or apartments, the outdoor PM contribution appears to be much less. The contribution of outdoor PM2.5 is generally greater than that for PM10 due to increased penetration. During cold weather periods, the outdoor PM contribution appears to be less due to reduced air exchange rates. The contribution of outdoor PM to indoor PM in public, commercial and multi-family buildings would be expected to be somewhat less than that for single-family residences due to outdoor air filtration by mechanical ventilation systems; however, representative data are lacking in this area.

6.5.5 Indoor Concentrations in Public and Commercial Buildings

Because adults and children in California typically spend about 62% and 75% of their time in their residence, respectively (Jenkins et al. 1992, Phillips et al. 1991), residences are the most important locations for overall PM exposure for most people. However, significant time—about 25% on average--is also spent in other buildings, such as at work and school, so PM concentrations in these buildings are also important in estimating exposure to PM.

PM concentrations in public and commercial buildings appear to often be lower than ambient concentrations, but far fewer studies have been conducted for public buildings than residences. Reasons for lower indoor PM concentrations in public and commercial buildings include the use of particle filters in mechanical ventilation systems, inoperable windows, reduced exterior surface to volume ratios, and the lack of many indoor sources typically present in residences. However, as with residences, the presence of indoor sources in public and commercial buildings can produce indoor concentrations that exceed concurrent ambient concentrations, especially if smoking is allowed in the building. The largest public and commercial building PM study to date was conducted in the Pacific Northwest for 38 commercial buildings (Turk et al. 1987). Buildings where smoking was prohibited averaged 19 $\mu g/m^3$ PM3.5 indoors, the same as the outdoor level, while buildings where smoking was permitted averaged 70 $\mu g/m^3$, notably higher than the outdoor level. (PM3.5 was measured as respirable PM or "RSP"). Sheldon et al. (1988) measured PM in six buildings in the eastern U.S., and found indoor PM concentrations generally lower than outdoors where there was no smoking, but much higher indoor concentrations where smoking was allowed (14 to 56 $\mu g/m^3$ versus 13 to 17 $\mu g/m^3$ outdoors).

Elevated PM concentrations can occur in other enclosed environments such as inside motor vehicles, but few studies have been conducted to examine such exposures. The most comprehensive study to date has been that of Rodes et al. (1998) conducted in Sacramento and Los Angeles. Real-time fine particle count concentrations and black carbon concentrations inside vehicles increased up to ten times the average roadway concentrations when following certain diesel vehicles. However, average PM mass concentrations inside the vehicle were similar to outdoor concentrations measured at the nearest ambient monitor, while roadway PM concentrations were somewhat higher. Average in-vehicle PM10 concentrations were about 27 $\mu g/m^3$ for Sacramento runs and 61 $\mu g/m^3$ for Los Angeles as compared to 29 $\mu g/m^3$ and 73 $\mu g/m^3$ at the nearest ambient stations, respectively. In-vehicle PM concentrations averaged 60 to 80% of those concentrations measured just outside the vehicle, which reflected the elevated

roadway concentrations. In summary, it appeared the impact of traffic on PM exposures inside vehicles was small with regard to total mass, although significant differences in traffic PM chemical composition and PM size distribution are probably present compared to ambient PM. Using carpool lanes appeared to reduce in-vehicle PM concentrations significantly, although carpool lanes were used in only two of the 29 two-hour runs.

6.5.6 PM Exposures in Sensitive Subgroups

Individuals with pre-existing respiratory disease, such as COPD and asthma, and pre-existing cardiovascular disease can be more susceptible to adverse effects from exposure to particulate pollutants. Until recently, personal exposures of such groups to particles had not been measured. Only a few small, recent studies have been conducted to examine the PM2.5 exposures of such groups. Elderly healthy persons and young children may also be more susceptible; the PM exposures of these subpopulations have been discussed earlier in this chapter.

Rojas-Bracho et al. (2000) and Linn et al. (1999) have reported on PM10 and PM2.5 exposures for individuals with COPD. Rojas-Bracho et al. found that mean personal PM10 and PM2.5 concentrations were 67% and 52% above outdoor PM10 and PM2.5 concentrations, respectively, for 18 COPD patients in Boston. (PM10 and PM2.5 concentrations were 22 and 14 $\mu g/m^3$ outdoors, 32 and 18 $\mu g/m^3$ indoors, and 37 and 22 $\mu g/m^3$ personal, respectively). Personal-to-ambient concentration median longitudinal correlations were moderate with better correlation observed for PM2.5 ($r=0.61$) than for PM10 ($r=0.35$) or for PM2.5-10 ($r=0.35$). The authors attribute this to the higher deposition rate of PM2.5-10 compared to that for PM2.5. The authors also found personal-to-outdoor concentration ratios to be high (i.e., greater than 3) when air exchange rates were low (less than one exchange per hour). Overall, this study found similar results to those of other studies for healthy adults except PM concentrations were lower.

Linn et al. monitored 15 COPD patients for PM2.5 exposures and 15 for PM10 exposures in Los Angeles during the fall and winter. PM10 and PM2.5 mean concentrations were 40 and 25 $\mu g/m^3$ for outdoors, 33 and 24 $\mu g/m^3$ for indoors, and 35 and 24 $\mu g/m^3$ for personal. The personal and indoor PM levels are similar to those discussed above for the Rojas-Bracho et al. study of COPD, although the outdoor PM levels in Los Angeles were higher. Unlike other studies, this study did not find personal concentrations to be significantly higher than indoors or outdoors. The authors suggest that the lack of increased personal PM in these subjects with severe COPD may be due to less personal activity, less time spent outside of the home, less personal particle generation, and failure to keep the personal monitor in their personal environment at all times. The pooled correlation of personal PM concentrations to ambient PM concentrations at a monitoring station (some of which were distant from the subject's home) were quite low ($r^2 < 0.1$). Daily indoor PM levels tracked PM levels outside the home more closely ($r^2 = 0.27$ for PM10, $r^2 = 0.19$ for PM2.5); presumably personal PM levels had similar correlations, because they correlated very closely with indoor PM levels.

Lillquist et al. (1998) reported indoor and outdoor PM10 measurements in three Utah hospitals over one winter season. Significant variability in indoor PM levels was found both among room types and among hospitals, and the relationship between indoor PM10 levels and outdoor levels was highly variable. The ICUs had significantly lower PM10 levels than other types of rooms, after adjusting for hospital differences. Thus, the most critically ill individuals may experience some protection in hospitals from ambient PM; however, in general, hospitals do not offer regular protection from ambient PM.

Two studies nearing completion will add significantly to our understanding of Californians' exposures to PM in both sensitive subpopulations and healthy persons. Conducted by the Harvard School of Public Health, both studies are designed to longitudinally examine the

relationships between outdoor and indoor concentrations and personal exposures across different seasons. The first of the two studies is examining PM exposures of a group of 15 individuals with COPD in Los Angeles. This study will provide information on how 24-hour average PM10 and PM2.5 mass, elemental carbon, particulate nitrate, and elemental concentrations vary by season, individual, and subject activity. The second study uses a similar design for a group of healthy persons, though it involves even more detailed characterizations of the PM levels and ventilation characteristics of the subject's homes, and focuses on determining the contribution of outdoor concentrations to personal exposures. The results from these two studies are expected to provide important new information on the relationships between outdoor and indoor concentrations as well as personal exposures for several components of PM, for both COPD patients and healthy persons in California.

6.5.7 Summary

Ambient PM is usually the major contributor to indoor and personal PM exposure, especially when few indoor sources are present. However, the relationships between indoor and ambient concentrations and personal and ambient PM concentrations are complex, and correlations between total PM mass concentrations in different microenvironments are sometimes low. People's use of, or proximity to, sources of PM, such as indoor cooking and cigarette smoke, typically results in higher personal exposure levels than indoor and ambient levels measured concurrently by stationary monitors. Indoor sources of PM such as cooking, tobacco smoke, and cleaning activities such as vacuuming often contribute to elevated indoor concentrations as well. Investigators have generally found somewhat greater correlations between personal and ambient PM concentrations for single individuals studied over several days as compared to single day analyses for more individuals, and for elderly or ill individuals with more limited activities and few indoor sources. Correlations also tend to be greater for PM2.5 than for PM10, in part because of increased penetration and reduced deposition rates indoors for smaller particles. However, there remains much uncertainty in the current understanding of these relationships.

6.6 References

Abt E, Suh H, Catalano P, and Koutrakis P. Relative Contribution of Outdoor and Indoor Particle Sources to Indoor Concentrations. Environmental Science and Technology 2000, 34: 3579-3587.

Air Resources Board. Guidance for using air quality-related indicators in reporting progress in attaining the State ambient air quality standards, Sacramento; 1993.

Air Resources Board. State and Local Air Monitoring Network Plan, Sacramento; 1998a.

Air Resources Board. Proposed identification of diesel exhaust as a toxic air contaminant, Appendix D to Appendix III, Part A, Exposure assessment. Sacramento, CA, 1998b.

Air Resources Board. Proposed area designations and maps. Staff report: Initial statement of reasons for proposed rulemaking, Sacramento; 2000.Air Resources Board. State and Local Air Monitoring Network Plan, Sacramento; 2000a.

Air Resources Board. 2000 Particulate Matter Monitoring Network Description, Sacramento; 2000b

Brauer M, Hirtle R, Lang B, Ott W. Assessment of indoor fine aerosol contributions from environmental tobacco smoke and cooking with a portable nephelometer. J Exposure Anal Environ Epidemiol 2000; 10:136-144.

Breiman, L., Gins, J., Stone, C. Statistical Analysis and Interpretation of Peak Pollution Measurements. TSC-PD-A190-10. Technology Service Corporation. 1978.

Chow JC, Watson JG. Imperial Valley/Mexicali Cross border PM10 transport study, Final Report to the U.S. Environmental Protection Agency, Region 9, DRI Document No. 8623.2F Reno, Nevada 1997.

Chow JC, Egami RT. San Joaquin Valley 1995 Integrated Monitoring Study: Documentation, evaluation, and descriptive data analysis of PM10, PM2.5, and precursor gas measurements. Final Report to the California Regional Particulate Air Quality Study, Sacramento, Sacramento CA. 1997: p.6-1to 6-23.

Chow JC, Watson JG, Lowenthal DH. Sources and chemistry of PM10 aerosol in Santa Barbara County, CA. Atmos Environ 1996; 30: 1489-1499.

Clayton CA, Perritt RL, Pellizzari ED, Thomas KM, Whitmore RW, Wallace LA, Ozkaynak H, Spengler JD. Particle Total Exposure Assessment Methodology (PTEAM) Study: distribution of aerosol and elemental concentrations in personal, indoor, and outdoor air samples in a Southern California community. J Exposure Anal Environ Epidemiol 1993; 3(2): 227-250.

Conner TL, Norris GA, Landis MS, Williams RW. Individual particle analysis of indoor, outdoor, and community samples from the 1998 Baltimore particulate matter study. Atmos Environ 2001; 35:3935-3946.

Dockery DW, and Spengler JD. Indoor-outdoor relationships of respirable sulfates and particles. Atmos Environ 1981; 15: 335-343.

Evans GF, Highsmith RV, Sheldon LS, Suggs JC, Williams RW, Zweidinger RB, Creason JP, Walsh D, Rodes CE, Lawless PA. The 1999 Fresno particulate matter exposure studies: comparison of community, outdoor, and residential PM mass measurements. J Air Waste Manage Assoc 2000; 50:1887-1896.

Fairley D. Bay Area Air Quality Management District. 2001; Personal communication.

Federal Register, Vol. 62, No. 138, 40 CFR Part 50.

Fortmann R, Kariher P, Clayton R. Indoor Air Quality: Residential Cooking Exposures. ARCADIS Geraghty & Miller, Inc. Final Report to the California Air Resources Board, Contract No. 97-330, November 2001.

Howard-Reed C, Rea A W, Zufall M J, Burke JM, Williams RW, Suggs JC, Sheldon LS, Walsh D, Kwock R. Use of a continuous nephelometer to measure personal exposure to particles during the U.S. Environmental Protection Agency Baltimore and Fresno panel studies. J Air Waste Manage Assoc 2000; 42:1125-1132.

Janssen N, Hoek G, Harssema H, Brunekreef B. Personal Exposure to Fine Particles in Children Correlates Closely with Ambient Fine Particles. Archives of Environmental Health, 1999; 54 (2): 95-101.

Janssen N, de Hartog J, Hoek G, Brunekreef B, Lanki T, Timonen K, Pekkanen J. Personal exposure to fine particulate matter in elderly subjects: relation between personal, indoor, and outdoor concentrations. J Air Waste Manage Assoc 2000; 50:1133-1143.

Jenkins P, Phillips T, Mulberg E, Hui S. Activity patterns of Californians: use of and proximity to indoor pollutant sources. Atmos Environ 1992; Vol. 26A, No.12:2141-2148.

Kamens R, Lee C, Wiener R, Leith D. A study to characterize indoor particles in three non-smoking homes. Atmos Environ 1991; Vol. 25, No. 5/6:939-948.

Kim BM, Teffera S, Zeldin MD. Characterization of PM2.5 and PM10 in the South Coast Air Basin of Southern California: Part 1 – Spatial variations. J Air & Waste Manage Assoc 2000; 50: 2034-2044.

Koutrakis P, Briggs S, and Leaderer B. Source apportionment of indoor aerosols in Suffolk and Onondaga Counties, New York. Environ Sci Tech1992; 26:521-527.

Larsen, L. C. and Nystrom, N. Technical Support Document for Proposed Amendments to the Criteria for Designating Areas of California as Nonattainment, Attainment, or Unclassified for State Ambient Air Quality Standards. Air Resources Board. Sacramento; 1992.

Lewis R, Fortmann R, Camann D. Evaluation of Methods for Monitoring the Potential Exposure of Small Children to Pesticides in the Residential Environment. Arch Environ Contamination and Toxicology 1994; 26, 37-46.

Lewis R, Fortune C, Willis R, Camann D, Antley J. Distribution of Pesticides and Polycyclic Aromatic Hydrocarbons in House Dust as a Function of Particle Size. Environ Health Perspect 1999; 107:721-726.

Lillquist Dr, Lee Js, Ramsay Jr, Boucher KM, Walton ZL, and Jl Lyon. A comparison of indoor/outdoor PM10 concentrations measured at three hospitals and a centrally located monitor in Utah. Applied Occup. Environ. Hyg. 13(6), June 1998, p.409-415.

Linn WS, Gong, Jr H, Clark KW, Anderson KR. Day-to-day particulate exposure and health changes in Los Angeles area residents with severe lung disease. J Air Waste Manage Assoc 1999; Special Issue on PM; 49: PM108-115.

Lioy PJ, Waldman JM, Buckley T, Butler J, Pietarinen C. The personal, indoor, and outdoor concentrations of PM10 measured in an industrial community during the winter. *Atmos Environ* 1990; 24B(1):57-66.

Liu D and Nazaroff W. Modeling pollutant penetration across building envelopes. *Atmos Environ* 2001, 35 (26): 4451-4462.

Lofroth G, Stensma C, Brandhorst Saatzkorn M. Indoor sources of mutagenic aerosol particulate matter : smoking, cooking and incense burning. *Mutation Research* 1991, 261 (1), 21-28.

Long C, Suh H, Catalano P, Koutrakis P. Using Time- and Size-Resolved Particulate Data to Quantify Indoor Penetration and Deposition Behavior. Environ Sci Technol 2001, 35: 2089-2099.

Mage D. How Much PM Of Ambient Origin Are People Exposed To?, in *Measurement of Toxic and Related Air Pollutants. Proceedings of a Specialty Conference, September 1-3, 1998, Cary, NC*; Vol. II: 666-681. Air & Waste Management Association.

Mage D, Wilson W, Hasselblad V, Grant L. Assessment of human exposure to ambient particulate matter. J Air Waste Manage Assoc 1999; 49:174-185.

Magliano KL, Hughes VM, Chinkin LR, Coe DL, Haste TL, Kumar N, Lurmann FW. Spatial and temporal variations of PM10 and PM2.5 source contribution and comparison to emissions during the 1995 integrated monitoring study. Atmos Environ 1999; 33: 4757-4773.

Malm, WC. Spatial and seasonal patterns and temporal variability of haze and its constituents in the United States: Report III. Cooperative Institute for Research in the Atmosphere, Colorado State University, ISSN 0737-5352-47, 2000.

McBride SJ, Ferro AR, Ott WR, Switzer P, Hildemann LM. Investigations of the proximity effect for pollutants in the indoor environment. J Expo Anal Environ Epidemiol 1999, Nov-Dec; 9(6): 602-21.

Miguel AG, Cass GR, Glovsky MM, Weiss J. Allergens in Paved Road Dust and Airborne Particles, Final Report to the California Air Resources Board contract No. 95-312, August 1998.

Mosley R, Greenwelt D, Sparks L, Guo Z, Tucker G, Fortmann R, and Whitfield C. Penetration of Ambient Fine Particles into the Indoor Environment. Aerosol Science and Technol 2001, 34: 127-136.

Motallebi N. Wintertime PM2.5 and PM10 source apportionment at Sacramento, California. J. Air & Waste Manage Assoc 1999; 49: 25-34.

Motallebi N. Air Resources Board. Personal Communication.

NAS National Academy of Science. *Indoor Allergens: Assessing and Controlling Adverse Health Effects..* Institute of Medicine, 1993. National Academy Press, Washington, DC.

NAS National Academy of Science. *Clearing the Air: Asthma and Indoor Air Exposures.* Institute of Medicine. 2000. National Academy Press, Washington, DC.

Oglesby L, Kunzli N, Roosli M, Braun-Fahrlander C, Mathys P, Stern W, Jantunen M, Kosua, A. Validity of ambient levels of fine particles as surrogate for personal exposure to outdoor air pollution-results of the European EXPOLIS-EAS study (Swiss Center Basel). J Air Waste Manage Assoc 2000; 42:1125-1132.

Ozkaynak H, Xue J, Weker R, Butler D, Koutrakis P, Spengler J. The Particle TEAM (PTEAM) Study: analysis of the data. Report to the U.S. EPA, Volume III of Final Report, 1996*a*.

Ozkaynak H, Xue J, Spengler J, Wallace L, Pellizzari E, and Jenkins P. Personal exposure to airborne particles and metals: results from the Particle TEAM Study in Riverside, California. J Exposure Anal Environ Epidemiol 1996*b*; Vol. 6, No. 1, 57-77.

Pellizzari ED, Clayton CA, Rodes CE, Mason RE, Piper LL. Particulate matter and manganese exposures in Toronto, Canada, Atmos Environ 1999 33: 721-734.

PhillipsTJ, Jenkins PL, Mulberg EJ. Children in California: Activity Patterns and Presence of Pollutant Sources, in *Proceedings of the 84th Annual Meeting and Exhibition*, Air & Waste Management Association, Vol. 17, Paper No. 91-172.5, Vancouver, British Columbia, June 1991.

Roberts JW, Dickey P, Exposure of Children to Pollutants in House Dust and Indoor Air. Reviews of Environmental Contamination and Toxicology 1995, Vol. 143: 60-77.

Rodes C, Sheldon L, Whitaker D, Clayton A, Fitzgerald K, Flanagan J, DiGenova F, Hering S, Frazier C. Measuring concentrations of selected air pollutants inside California vehicles. Final Report to the California Air Resources Board, Contract No. 95-339, Sacramento CA. 1998.

Rodes CE, Lawless PA, Evans GF, Sheldon LS, Williams RW, Vette AF, Creason JP, Walsh D. The relationships between personal PM exposures for elderly populations and indoor and outdoor concentrations for three retirement center scenarios. J Expo Anal Environ Epidemiol 2001 Mar-Apr;11(2):103-15.

Rojas-Bracho L, Suh HH, Koutrakis P. Relationships among personal, indoor, and outdoor fine and coarse particle concentrations for individuals with COPD. J Exposure Anal Environ Epidemiol 2000; 10:294-306.

Rothenberg S, Nagy P, Pickrell J, Hobbs C. Surface Area, Adsorption, and Desorption Studies on Indoor Dust Samples. J Am Ind Hyg Assoc 1989; 50(1):15-23.

Salmon LG, Mertz KA, Mayo PR, Cass GR. Determination of the elemental carbon, organic compounds, and source contributions to atmospheric particles during the Southern California Children's Health Study. Final Report to the California Air Resources Board, Contract No. 98-320, Sacramento CA. 2001.

Santos-Burgoa C, Rojas-Bracho L, Rosas-Perez I, Ramirez-Sanchez A, Sanchez-Rico G, Mejia-Hernandez S. Modelaje de exposición a partículas en población general y riesgo de enfermedad respiratoria. Gac Med Mex 1998; 134:407-417.

Sarnat JA, Koutrakis P, Suh HH. Assessing the relationship between personal particulate and gaseous exposures of senior citizens living in Baltimore, MD. J Air Waste Manage Assoc 2000; 50:1184-1198.

Schauer JJ, Cass GR. Source contributions to airborne particles in the San Joaquin Valley during the IMS95 study. Draft Final Report to the California Air Resources Board, Contract No. 97-6PM, Sacramento CA. 1998

Sexton K, Spengler JD, Treitman RD. Effects of residential wood combustion on indoor air quality: a case study in Waterbury, Vermont. Atmos Environ 1984*a*; 18(7): 1371-1383.

Sexton K, Spengler JD, Treitman RD. Personal exposure to respirable particles: a case study in Waterbury, Vermont. Atmos Environ 1984*b*; 18(7): 1385-1398.

Sheldon LS, Handy RW, Hartwell TD, Whitmore RW, Zelon HS, Pellizzari ED. Indoor air quality in public buildings: volume 1. US EPA, Washington DC, 1988; EPA 600/6-88/009a. NTIS PB 89-102503/AS.

Sheldon L, Clayton A, Keever J, Perritt R, and Whitaker D. Indoor concentrations of polycyclic aromatic hydrocarbons in California residences. Draft final report to the Air Resources Board, Contract No. A033-132, 1993.

Sisler JF. Spatial and seasonal patterns and long term variability of the composition of the haze in the United States: An analysis of data from the IMPROVE network, Cooperative Institute for Research in the Atmosphere, Colorado State University, ISSN 0737-5352-32. 1996.

Spengler, JD, Treitman RD, Tosteson TD, Mage DT, Soczek ML. Personal exposures to respirable particulates and implications for air pollution epidemiology. Environ Sci Tech 1985; 19:700-707.

Suh HH, Koutrakis P, Spengler JD. The relationship between airborne acidity and ammonia in indoor environments. J Exposure Anal Environ Epidemiol 1994; 4: 1-22.

Taylor CA Jr., Stover C.A., Westerdahl F.D. Speciated fine particle (<2.5 μm aerodynamic diameter) and vapor-phase acid concentrations in Southern California. A&WMA 91st Annual Meeting and Exhibition. San Diego CA June 14-18, 1998.

Thatcher TL, Layton DW. Deposition, resuspension, and penetration of particles within a residence. Atmos Environ 1995; 29(13): 1487-1497.

Thornburg J, Ensor D, Rodes C, Lawless P, Sparks L, and Mosley R. Penetration of Particles into Buildings and Associated Physical Factors. Part I: Model Development and Computer Simulations. Aerosol Science and Technol 2001, 34 284-296.

Turk BH, Brown JT, Geisling-Sobotka K, Froehlich DA, Grimsrud DT, Harrison J, Koonce JF, Prill RJ, Revsan KL. Indoor air quality and ventilation measurements in 38 Pacific Northwest commercial buildings. Volume I: measurement results and interpretation. Lawrence Berkeley Laboratory, 1987; Final report, LBL-22315.

United States Environmental Protection Agency (US EPA). Transport of Lawn-Applied 2,4-D from Turf to Home: Assessing the Relative Importance of Transport Mechanisms and Exposure Pathways. EPA 600-R-99-040. Research Triangle Park, NC, 1999.

U.S. EPA, Air Quality Criteria for Particulate Matter, Volume 1. EPA 600/P-99/002aB. March 2001, Second External Review Draft.

Vette A, Rea A, Lawless P, Rodes C, Evans G, Highsmith R, and Sheldon L. Characterization of Indoor-Outdoor Aerosol Concentration Relationships during the Fresno PM Exposure Studies, Aerosol Science and Technology 2001, 34: 118-126.

Wallace L. Indoor particles: a review. J Air Waste Manage Assoc 1996; 46:98-126.

Wallace L. Correlations of Personal Exposure to Particles with Outdoor Air Measurements: A Review of Recent Studies. Aeorosol Science and Technol 2000*a*: 32:15-25

Wallace L. Real-time monitoring of particles, PAH, and CO in an occupied townhouse. Appl Occup Environ Hyg 2000*b*; 15:39-47.

Williams R, Suggs J, Zweidinger R, Evans G, Creason J, Kwock R, Rodes C, Lawless P, Sheldon L. The 1998 Baltimore Particulate Matter Epidemiology-Exposure Study part 1. Comparison of ambient, residential outdoor, indoor and apartment particulate matter monitoring. J Exposure Anal Environ Epidemiol 2000*a*; 46, 10: 518-532.

Williams R, Suggs J, Creason J, Rodes C, Lawless P, Kwock R, Zweidinger R, Sheldon L . The 1998 Baltimore particulate matter epidemiology-exposure study part 2. Personal exposure assessment associated with an elderly study population. J Exposure Anal Environ Epidemiol 2000*b*; 10:533-543.

Williams R, Creason J, Zweidinger R, Watts R, Sheldon L, Shy C. Indoor, outdoor, and personal exposure monitoring of particulate air pollution: the Baltimore elderly epidemiology-exposure pilot study. Atmos Environ 2000*c*; 34:4193-4204.

Wilson AL, Colome SD, Baker PE, Becker EW. Residential Indoor Air Quality Characterization Study of Nitrogen Dioxide. Phase I final report for Southern California Gas Company. 1986.

Wilson AL, Colome SD, Tian Y. California Residential Indoor Air Quality Study, Volume 1: Methodology and descriptive statistics. Prepared for Gas Research Institute, Pacific Gas and Electric Company and Southern California Gas Company by Integrated Environmental Services, Irvine, CA, 1993.

Wilson WE, Mage DT, Grant LD. Estimating separately personal exposure to ambient and non-ambient particulate matter for epidemiology and risk assessment: why and how. J Air Waste Manage Assoc 2000; 50:1167-1183.

Woodhouse LF. Air Resources Board. Personal communication.

Zartarian VG, Ferguson AC, and Leckie JO (1998). "Quantified Dermal Activity Data from a Four-Child Pilot Field Study," *J. Exposure Analysis and Environ. Epidemiology* 8(4): 543-553.

Zartarian VG and Leckie JO (1998). "Feature Article–Dermal Exposure: The Missing Link," *Environmental Science and Technology* 3(3): 134A-137A.

7. Health Effects of Particulate Matter

The Children's Environmental Health Protection Act (Senate Bill 25, Senator Martha Escutia, Stats. 1999, Ch. 731) required the ARB, in consultation with OEHHA, to "review all existing health-based ambient air quality standards (AAQS) to determine whether, based on public health, scientific literature, and exposure pattern data, these standards adequately protect the health of the public, including infants and children, with an adequate margin of safety." Of those AAQS identified as providing insufficient public health protection, SB 25 requires the ARB to revise the highest priority standard by December 31, 2002. Last year OEHHA staff, assisted by six academic air pollution researchers, undertook a critical review of the health impacts of exposure to the regulated pollutants, and categorized the latter into two tiers, with the first representing greater potential risks to public health at the concentrations of the current AAQS. Of the first-tier standards, OEHHA identified the AAQS for particulate matter as the highest priority pollutant, and recommended to the ARB that this standard be the first to consider for a more thorough evaluation and possible revision. This decision was based on the evidence in the literature of health effects, including mortality and morbidity in infants, children, the elderly and other potentially sensitive subgroups, associated with particulate matter at or below the current state standards. The ARB accepted the recommendation by OEHHA staff at the Board Meeting held in December 2000.

This chapter contains a targeted, critical review by OEHHA staff of the research relevant to setting the standard(s) for the particulate matter AAQS for California. Beginning with deposition, clearance and dosimetry of particles (section 7.1), the review focuses primarily on epidemiological studies of mortality associated with both acute and chronic exposure to PM (sections 7.2, 7.3 and 7.4), as well as morbidity outcomes (sections 7.5 and 7.6). This review of the most pertinent literature is followed by discussions of susceptible subpopulations (section 7.7), plausible biological and toxicological mechanisms underlying the epidemiological observations (section 7.8), and causal inference regarding the associations between ambient PM concentrations and increased morbidity and mortality (section 7.9). The OEHHA staff recommendations for revision of California's AAQS for PM are provided in section 7.10).

In brief, OEHHA staff recommends that the current PM10 standards be revised. There are compelling reasons to be concerned about significant adverse health effects associated with ongoing exposures occurring at or below concentrations prescribed by the existing standards. Recommended changes include:

- Revise the annual average standard for PM10 from 30 to 20 $\mu g/m^3$.
- Retain the 24-hour standard for PM10 of 50 $\mu g/m^3$, not to be exceeded.
- Add an annual average standard for PM2.5 of 12 $\mu g/m^3$, given growing evidence from epidemiological and toxicological studies of significant toxicity related to this size fraction of PM.
- Establish a 24-hour standard for PM2.5 of 25 $\mu g/m^3$, not to be exceeded.
- Retain the current 24-hour average standard of 25 $\mu g/m^3$ for sulfates.
- Prevent degradation of current ambient air concentrations, measured as PM10 or PM2.5.

7.1 Particle Deposition, Clearance and Dosimetry

For particles to exert any biological effect, they must first come into contact with the target organ tissue: for purposes of this document, the initial target organ of concern is the

respiratory tract. In general, particles 10 μm or less in diameter are considered respirable by humans. The depth of penetration into the lung and extent of deposition are determined by a particle's aerodynamic diameter, its ability to attract water (hygroscopicity), electrostatic charge, and by host characteristics, including airway structure and geometry, as well as depth, rate and mode of breathing (e.g., nasal vs. oronasal). Many inhaled particles are exhaled without depositing in the respiratory tract; the theoretical particle diameter for minimal deposition is about 0.5 μm. In general, for particles with diameters greater than 0.5 and less than 10 μm, increasing size is associated with greater total lung deposition, while for particles with diameter less than 0.5 μm deposition is inversely related to particle size. Soluble particles can be cleared by dissolution into the extracellular fluid lining the airways, with subsequent transport into epithelial or other cells of the respiratory tract, and then into the circulation. Insoluble particles are cleared by more complex mechanisms, as described below.

7.1.1 Deposition

The respiratory tract is often considered to consist of three anatomically and functionally distinct units: (a) the extra-thoracic (ET - from the mouth and nose to the larynx); (b) the tracheo-bronchial (TB – from the larynx through the conducting airways; and (c) the alveolar (AL – the gas exchange zone). In general, more serious pollution-related health outcomes are related to effects in the TB and AL regions. The patterns of particle deposition in the respiratory tract do not, however, correspond well to the categories used to classify particles for regulatory purposes (PM10, fine (PM2.5) and coarse (PM10 – PM2.5) fractions). Generally, larger particles demonstrate a greater fractional deposition in the ET and upper TB areas, while smaller particles show greater deposition in the deep lung (lower TB and AL). These regional patterns reflect principally the mechanisms of deposition that differentially influence particles by size.

Mechanisms of nonfibrous particle deposition include: (i) gravitational settling, for particles more dense than air; (ii) impaction on the wall of a bronchus or bronchiole, due to inertia maintained when the airstream changes direction at an anatomical bend or bifurcation; (iii) diffusion related to Brownian motion; and (iv) electrostatic attraction, which is generally considered of lesser importance than the other three. Settling and diffusion are more important for particles less than about 3 μm, while inertial impaction generally affects larger particles, particularly in the ET and upper TB area (Foster, 1999). For ultrafine particles (with diameters <0.1 μm in diameter), diffusion represents the dominant mode of deposition.

The ET region and especially the nose effectively filter out a large fraction of inhaled particles, mainly those above 1 μm in diameter, but also including ultrafine particles. In general, inertial impaction predominates in the ET region, so increasing particle size and increasing flow rates will tend to increase particle deposition. However, fractional deposition of ultrafine particles (inhaled at flow rates between 5.9 and 22 liters/min) in the nose has also been reported to be very high (in excess of 93%) (Swift and Strong, 1996).

In the TB and AL areas, increased depth of breathing tends to enhance the deposition of fine particles, while an increased respiratory rate has the opposite effect (Foster, 1999). Exercise and increased respiratory rates also tend to result in greater deposition in larger, central airways, and less in the AL region (Foster, 1999). Using inert particles 1, 3, and 5 μm in diameter, Kim et al. (1996) showed that, even in healthy adults, there is striking heterogeneity of deposition patterns, with airway surface doses 2 to 16.6 times greater in large airways and up to 4.5 times greater in small airways than in the alveolar region for larger (3 and 5 μm) particles. A similar, but less pronounced, pattern was also observed for particles of 1 μm diameter. Heterogeneous local particle dose enhancement may also be important among individuals with obstructive lung disease (see below).

Among healthy adults, airway caliber (measured by specific airway resistance) appears to be an important determinant of particle deposition, with a generally inverse relationship between airway diameter and deposition efficiency (Bennett et al., 1996). This may result from the decreased cross-sectional distance that particles have to traverse (by inertial velocity, gravitational settling, or diffusion) before depositing. Women tended to display a greater deposition fraction than men (perhaps because of a smaller respiratory tract anatomy overall). Nevertheless, because men breathed more rapidly than women, they showed a greater deposition of particles per unit time, though the difference was slight when normalized to lung surface area. However, under controlled breathing conditions, women tend to display greater deposition of coarse particles, defined here as those with 3 and 5 μm in diameter, throughout the lung, particularly in the ET and TB regions (Kim and Hu, 1998). Bennett and colleagues (1996) also found that the deposition fraction of inert fine particles, defined here as those with 2 μm in diameter, was independent of age among 62 healthy adults (ages 18 – 80), which suggests that among elderly individuals, pre-existing lung disease may be more important than age *per se* with respect to respiratory tract deposition (see below).

Individuals with asthma and chronic obstructive lung disease experience greater fractional deposition of fine particles, defined here as those with 1 μm in diameter, than individuals with healthy, normal lungs, with the degree of particle retention roughly proportionate to the severity of airway obstruction (Kim and Kang, 1997). Adult subjects with asthma or COPD showed approximately 1.6- and 2.0-fold greater fractional deposition, respectively, of fine particles than healthy subjects (Kim and Kang, 1997). Anderson et al. (1990) showed a similar increase in deposition efficiency of fine and ultrafine particles, defined here as those with 0.02 – 0.24 μm in diameter, in several individuals with asthma and COPD relative to healthy subjects. This study also included 3 individuals with restrictive lung disease (characterized by lung fibrosis or scarring); these subjects demonstrated particle deposition patterns similar to healthy individuals. The enhanced deposition of particles in individuals with chronic obstructive lung disease is likely to have at least four physiological bases: (1) narrowed airways result in increased deposition by inertial impaction; (2) relatively low expiratory flow rates and even airway collapse during expiration allow for longer particle residence time in the lung, favoring deposition of fine and ultrafine particles by diffusion; (3) mucus hypersecretion may cause airflow irregularities that can enhance particle deposition; and (4) uneven ventilation related to airway obstruction may result in deeper particle penetration into those areas of the lung that are still ventilated and functional (Kim and Kang, 1997).

In such individuals, one can observe focal hyperdeposition of particles, often in sites of airflow limitation in central airways, even when nominal ambient particle concentrations are relatively low (Foster, 1999). Airway hyperresponsiveness, which is one of the hallmarks of asthma but can also occur in otherwise healthy individuals, is likewise associated with enhanced regionalization of deposition to the central airways (Foster, 1999). This may exaggerate the patterns of local deposition enhancement observed in healthy individuals (Kim et al., 1996, see discussion above). The work of Kim and Kang (1997) indicates that such dose amplification can occur because individuals with obstructive lung disease: (1) ventilate only a portion of their lungs, (2) experience increased deposition compared with healthy individuals, and (3) if symptomatic, tend to have increased minute ventilation. Assessing these factors together, Kim and Kang (1997) estimate that such individuals may have more than three-fold greater total lung deposition than healthy subjects, with this enhanced deposition concentrated in small areas of the lung.

One group of investigators modeled short-term particle deposition in various regions of the respiratory tract using a dosimetry model developed by the International Committee on

Radiological Protection (Snipes et al., 1997). They identified large differences in deposition between the ET, TB and AL regions. Daily deposition of all particle sizes was estimated to be greater (by one to three orders of magnitude) in the TB compared with the AL region. For instance, using aerosol size distributions corresponding to those of Philadelphia (favoring the fine mode) and Phoenix (favoring the coarse mode), and assuming inhalation for 24 hr/day, 7 days/week of 50 $\mu g/m^3$, they predicted that the daily mass of particles deposited per gram of epithelial tissue/day to range from 0.47 to 1.8 μg in the TB region and 1 to 34 ng in the AL (or in their model the alveolar-interstitial or AI region). The predicted mass deposited per unit of epithelial tissue surface area under similar simulated exposure conditions was much lower, ranging from 0.9 to 3.5 ng/cm^2 in the TB area and 0.78 to 25 pg/cm^2 in the AI region. Examining predicted dose/unit surface area in terms of particle number suggested daily deposition of up to 100,000 particles/cm^2 (in the fine mode) in the TB region.

Results of the deposition modeling forming the basis for the report by Snipes et al. (1997) are presented in slightly different form in the 1996 U.S. EPA Criteria Document for particulate matter (U.S. EPA, 1996; vol II, chapter 10). For instance, using the same inhalational assumptions as noted in the previous paragraph for normal adult males in the general population exposed to the Phoenix aerosol, the model predicted daily deposition of 2 and 6 μg/day of fine and coarse mode particles, respectively, in the bronchi, 3 (fine) and 4 (coarse) μg/day in the bronchioles, and 17 (fine) and 12 (coarse) in the alveolar region. Particle doses were estimated to increase substantially in all zones of the lower respiratory tract among "mouth breathers": 5, 5, and 27 for fine mode and 31,12, and 30 μg/day for coarse mode doses in the bronchi, bronchioles, and alveolar regions, respectively (U.S. EPA, 1996). Higher doses were also predicted to occur as a result of light or heavy work (involving increased breathing rates). Somewhat lower doses were estimated to result from exposure to a Philadelphia-like aerosol, which is characterized by a particle distribution favoring smaller particles. The model employed in these deposition exercises is based on average doses and does not take into account the potential impacts of age, gender, disease states or inter-individual variations in anatomy, ventilation patterns, short-term peak exposures, and so forth. Nonetheless, this report suggests the likelihood of significant particle deposition in the lung from ambient PM exposures, especially within the TB region. While many particles will be cleared from the lung, some remain in the airways, interstitium and lymph nodes for prolonged periods of time, as discussed below.

7.1.2 Clearance

The localization of deposition in the lung will affect the rate, mode, and completeness of clearance. Soluble particles are cleared from the respiratory tract by absorption into extracellular fluids or mucus, then to epithelial cells, from which they can pass into the circulation (Foster, 1999). Insoluble ultrafine particles can also be taken up into the respiratory epithelium and have recently been reported to enter the blood of humans within minutes of inhalation, suggesting a potential route for the rapid initiation of systemic particle-related effects (Ferin el al., 1992; Nemmar et al., 2001). However, in general, insoluble particles have been considered to be cleared in two phases: (1) a faster TB phase considered to be more or less complete within 24 - 36 hours, which is effected by mucociliary activity; (2) a more prolonged phase, which can continue for days to months, which is considered to be mediated via engulfment by alveolar macrophages for particles depositing in the deep lung (Foster, 1999).

The ciliated airways in the TB region are covered by a thin two-fluid liquid; the upper mucous layer traps particles and transports them up to the throat, propelled by ciliary beating in the lower layer. Upon reaching the oropharynx, the mucus containing the particles is usually swallowed or expectorated. Carriage on the mucociliary "escalator" is the principal

mechanism of the “fast” phase; mucociliary transport rates are generally fastest in the trachea and large bronchi. Some particles may be engulfed by macrophages in the airways, which can then be transported on the mucociliary escalator. However, these processes are not universally successful; some insoluble particles cross into the airway epithelium and enter the lung interstitium (Ferin et al., 1992; Churg and Brauer, 1997).

The slow clearance phase has traditionally been considered to affect particles that deposit deep in the lung, beyond the ciliated epithelium. Recent evidence, however, indicates that a substantial fraction of particles depositing in the TB region, particularly the bronchioles, are not cleared for days (Falk et al., 1999; U.S. EPA, 1996). Falk et al. (1999) followed the long-term clearance (over a 6-month period) of 6 μm radiolabelled Teflon particles inhaled at 0.5 or 0.05 l/s by human volunteers. The slow inhalation rate facilitates particle deposition that is nearly independent of airway resistance, allowing for greater deposition in the bronchioles. About half the deposited particles remained in the lungs after 24 hr. At inhalation rate of 0.5 l/s, 14% of the particles that had not cleared by 24 hr showed a clearance half-time of 3.7 days, while the remaining 86% demonstrated a clearance half-time of 217 days. Of the particles retained at 24 hr after slow inhalation (0.05 l/s), 35% cleared with a half-time of 3.6 days, while the remaining 65% showed a half-time of 170 days (Falk et al., 1999). Thus, for both slow and normal modes of inhalation, there appear to be three phases of clearance: an initial fast phase ($\leq$ 24 hr), an intermediate phase ($t_{½} \approx$ 4 days), and a slow phase ($t_{½} \approx$ 200 days). These investigators assumed that the intermediate phase represented clearance from the bronchiolar region, while the slow phase represented clearance from the AL region.

Alveolar macrophages are the principal clearance vehicle in the AL region. Particle-containing macrophages can make their way to the mucociliary escalator, move to a lymphatic channel within the interstitium to regional lymph nodes, or cross into the circulation, either after passing through the lymph node or possibly by direct entry into the blood across the alveolar capillary endothelium. However, as noted above, clearance processes are not 100% effective: numerous particles are translocated into the epithelium and interstitium (often within hours of deposition), where they may become aggregated in specific sites around the airways or blood vessels. Lymph nodes can become storage depots for particles, as well.

Once in the interstitium, particles tend to stay there; clearance is extremely slow, on the order of months to decades. Particle access to the lung interstitium increases as particle size decreases and particle numbers increase (Ferin et al., 1992). In an examination of autopsy lung tissues of elderly, never-smoking residents of Vancouver (a city with relatively low levels of particulate air pollution; mean PM10 from 1984 - 1993 = 20 - 25 $\mu g/m^3$), Churg and Brauer (1997) found that 96% of particles retained in the lung parenchyma had (calculated) aerodynamic diameters < 2.5 μm, with a geometric mean of 0.41 μ, while coarse and ultrafine particles comprised 4.0 and 4.8%, respectively of the total. Investigating the size and composition of particles retained in the airways among residents of Mexico City as well as Vancouver, Churg and Brauer (2000) found strikingly large numbers of particles (roughly 10^7/g dry lung tissue), with generally increasing quantities proceeding from the mainstem bronchus to the deep lung. The highest concentrations, with particle numbers 25-100 times higher than along the mainstem bronchus, were in the respiratory bronchioles (at the junction between the conducting airways and the alveoli) and at large airway carinas (anatomic bifurcations of the airways). In addition, there were enormous differences (up to several hundred-fold) in particle retention among the study subjects, probably reflecting inter-individual variability in clearance rates.

Exposure to respiratory irritants can stimulate epithelial, sensory neural, and other airway cells to release cytokines and other chemical messengers, and can result in local inflammation, altered epithelial permeability, increased mucus secretion, and

bronchoconstriction. Disease states characterized by mucus hypersecretion and disruption of the normal epithelial architecture (e.g., asthma and chronic bronchitis) can produce mucus stasis and adversely affect particle clearance (Foster, 1999). As alveolar macrophages engulf substantial quantities of particles, their viability and functional integrity can be adversely affected by PM exposures, which have been attributed in part to soluble metal-induced oxidative stress (Soukup and Becker, 2001). Effects on alveolar macrophages may not be limited to fine and ultrafine particles. Kleinman et al. (1995) demonstrated that essential alveolar macrophage functions (phagocytosis and oxidant generation) can be inhibited by coarse particles in re-suspended road dust. In vitro experiments suggest that, in addition to decreasing alveolar macrophage phagocytosis, PM10 exposure appears to reduce resistance to infection with respiratory syncytial virus (Becker and Soukup, 1999). Recent work suggests also that ultrafine particle uptake by human alveolar macrophages is common (observed in macrophages obtained from all 14 subjects), and that there may be an inverse relationship between lung function and the extent of ultrafine particle content of alveolar macrophages (Hauser et al., 2001).

Mucociliary clearance can be affected by exposure to acidic aerosols (Schlesinger et al., 1992). In humans, mucociliary clearance has been shown to be depressed following exposures to approximately 100 $\mu g/m^3$ sulfuric acid particles for one to two hours (Spektor et al., 1989). In contrast, depression of mucociliary clearance in animals requires concentrations greater than 100 $\mu g/m^3$ delivered over several hours or even months (U.S. EPA, 1989; Mautz et al., 1996; Kleinman et al., 1999). Altered mucociliary clearance in humans has the potential to affect the incidence of respiratory infection in healthy, as well as compromised, subjects.

7.1.3 Differences between Children and Adults

There are significant anatomic and physiological differences between the developing lungs of children and those of mature adults (Snodgrass, 1992). These include differences in the size and shape of the conducting airways, the number and orientation of physiologically active gas exchange regions, and ventilation rates. Though the basic structure of the airways is established *in utero*, most of the alveoli ($\approx$ 85%) develop in infancy and early childhood. Alveolar multiplication coincides with incorporation of elastin and collagen in the lung, which are responsible for the mature lung's mechanical properties (Lipsett, 1995). With growth and development other patterns of anatomical differences emerge. For instance, TB airways increase in diameter and length until adulthood. Lung volume expands disproportionately in relation to the increasing number of alveoli during somatic growth, indicating enlargement of individual alveoli (Murray, 1986). Repeated episodes of PM-related injury and inflammation may therefore have long-term consequences on the lung's functional abilities (see section 7.6, below).

Because of differences in anatomy, activity, and ventilation patterns, children are likely to inhale and retain larger quantities of pollutants per unit body weight than adults (Adams, 1993). Phalen et al. (1985) developed a model incorporating airway dimensions measured in lung casts of people (aged 11 days to 21 years) to predict that particle deposition efficiency would be inversely related to body size, which would tend to accentuate differences in exposure related to activity and ventilation patterns. Corroborative evidence for this was provided by Oldham et al. (1997), who found that in models of the proximal TB airways (i.e., the trachea and the first two bronchial bifurcations) of 4- and 7-year-old children and an adult, deposition efficiencies for radiolabelled particles 1.2, 4.5, 9.7 and 15.4 μm in median aerodynamic diameter were greater in the child models in almost all cases. As expected, particle deposition efficiency increased markedly with increasing particle size in this model system. For instance, in the model of the four-year-old child, the deposition efficiency

increased from 0.3% to 10.7% when the smallest and largest particle sizes were used, respectively.

Inhalation experiments comparing particle deposition patterns in children and adults have produced somewhat inconsistent results. Schiller-Scotland et al. (1994) reported greater fractional deposition in healthy children, aged 3 – 14 years, compared with adults, when breathing 1, 2 or 3 μm particles spontaneously through a mouthpiece. The differences were greater with the larger particles. However, as noted by the authors, these children were breathing more deeply than expected, which is a common tendency when breathing through a mouthpiece. This propensity may result in greater time-dependent deposition of fine particles (by sedimentation and diffusion). Schiller-Scotland et al. (1994) also noted that, among the older children (mean age = 10.9 years) who were capable of controlled breathing in time with a metronome, particle deposition was inversely related to body height, so that the shorter children demonstrated greater fractional deposition (for 1 and 2 μm particles, the only categories analyzed in this manner). In contrast, Bennett and Zeman (1998) found no significant differences between children (7 – 14 yr), adolescents (14 to 18 yr), and young adults (19 – 35 yr) in deposition (measured as deposition fraction or rate) of 2 μm particles during spontaneous breathing at rest. Unlike the study by Schiller-Scotland et al. (1994), this investigation tailored the participants' mouthpiece breathing patterns to those measured during unencumbered breathing, in order to control for the tendency to breathe more deeply through a mouthpiece. Another difference between the study by Bennett and Zeman (1998) and that by Schiller-Scotland et al. (1994) is that the former did not include very young children, who would have had difficulty in mimicking their normal breathing patterns while using a mouthpiece. However, Schiller Scotland et al. (1994) found that older children (mean age = 10.9 years) as well as the younger ones (mean age = 5.3 years) also showed increased fractional particle deposition relative to adults.

Children demonstrate lower absolute minute ventilation at rest than adults, despite having higher breathing rates. Relative to lung volume, however, children demonstrate a higher minute ventilation than adults. Thus, Bennett and Zeman (1998) noted that children tended to have a somewhat greater normalized deposition rate (by about 35%) than the combined group of adolescents and adults, suggesting that children at rest would receive higher doses of particles per unit of lung surface area than adults. This tendency might be additionally enhanced by activity patterns, as children spend more time than adults in activities requiring elevated ventilation rates. However, it is unknown whether flow-dependent deposition mechanisms operative at higher ventilation rates in children would offset the decreases that would occur in time-dependent mechanisms (sedimentation and diffusion). If this offset does occur, then particle deposition would likely be shifted more towards the larger, more central airways, which would tend to increase the dose per surface area in children versus adults (Bennett and Zeman, 1998).

The above studies suggest that children may experience proportionately greater particle deposition than adults. It is also possible that, especially in very young children, immature respiratory defenses may result in lower clearance rates in relation to those observed in adults. For instance, Sherman et al. (1977) reported that alveolar macrophages of neonatal rabbits (1 day old) ingested significantly fewer bacteria than older animals (7 days). To the extent that this phenomenon may also apply across species and to nonbiological particles, the immaturity of the neonatal human lung may result in slower and less complete particle clearance.

7.2 Overview: Epidemiological Studies of Airborne Particulate Matter

Particulate matter (PM) is a heterogeneous, complex mixture of liquid and solid particle sizes and chemicals; thus, it has been difficult to conduct animal or human clinical studies using mixtures found in ambient air. Until the recent development of ambient air particle concentrators, toxicological and controlled human experiments involving PM have generally used simple model particles (e.g., sulfuric acid) or mixtures taken from a single source (e.g., diesel exhaust or residual oil fly ash). In contrast, some health effects of gaseous pollutants can be studied directly using controlled concentrations in chamber experiments. Therefore, most of the health evidence on PM has been derived from observational epidemiological studies of human populations in a variety of geographic (principally urban) locations. Most of the studies have examined short-term or acute health consequences of PM exposure on health (i.e., those occurring on the same day as or within a few days of the exposures of interest), including both mortality and morbidity. Studies of the acute effects of PM exposure typically involve daily time-series observations collected over several months or years. The studies often examine whether daily counts of mortality or cause-specific hospitalizations are correlated with daily concentrations of PM, after controlling for effects of other covariates and potential confounders. Such factors may include temporal and meteorological variables, e.g., day-of-the-week, extremes in temperature, humidity or dewpoint, co-pollutants, and longer-term trends represented by seasonal changes or population growth. Well designed time-series studies can have several methodological strengths, including: (1) a large sample size (sometimes up to 4 to 8 years of daily data), conferring substantial statistical power to detect effects; (2) implicit incorporation of a wide range of population demographics, baseline health characteristics, and human behaviors, enhancing the generalizability of the results; (3) real-world exposures, avoiding the need to extrapolate to lower concentrations or across species; (4) the ability to examine effects in potentially sensitive individuals, children and infants; and (5) a limited number of covariates or potential confounders, particularly other pollutants and weather factors. Limitations of or potential uncertainties associated with time-series studies include: (1) difficulty in determining actual pollutant concentrations to which people are exposed; (2) the potential for misclassification of exposure; (3) the potential for omission of important explanatory factors or inappropriate control of potential confounding factors; (4) difficulty in measuring or observing all potential health effects; (5) covariation among pollutants, making it difficult to attribute an effect to a single pollutant. Moreover, the average daily PM10 concentration in a given location will be similar to the annual average PM10 concentration. While relationships between health outcomes and acute exposures have frequently been identified through time-series analysis, it may be difficult to determine the effect of a single 24-hour exposure independent of the influence of low-level chronic exposures. Nevertheless, the epidemiological studies of PM provide a major body of evidence regarding the associated health effects, and serve as a basis for many of the conclusions and recommendations that follow.

7.3 Daily Exposure – Mortality

Over the past two decades, several dozen time-series studies spanning five continents have demonstrated associations between daily counts of mortality and daily or multi-day changes in the concentrations of several common air pollutants. Among these pollutants, various particulate matter metrics – including PM10 (particulate matter with a median aerodynamic diameter equal to or less than 10 microns), PM2.5 (particulate matter with a median aerodynamic diameter equal to or less than 2.5 microns), black smoke, and sulfates – appear

to show the most consistent associations with mortality, although some associations have also been reported for ozone, sulfur dioxide, carbon monoxide, and nitrogen dioxide.

Time-series studies examine daily changes in air pollution, typically based on 24-hour average concentrations, in relation to daily counts of mortality. The analysis typically uses multivariate regression models that control for potential confounding factors other than a specific pollutant that may vary over time and may also be associated with mortality. Such factors include day of the week, season, weather, time, and co-pollutants. For example, there is evidence that meteorological factors, such as extremes in temperature and humidity, are associated with mortality. Similarly, there have been consistent observations of cause-specific mortality patterns related to the day of the week. Failure to control for such effects could bias the estimated effects of air pollution. All of the mortality studies associated with short-term exposure reviewed below incorporated statistical control for the effects of weather. In addition, two studies (Samet et al., 1998; Pope and Kalkstein, 1996) involved very detailed modeling of weather patterns with the aid of a meteorologist. These studies found that the estimated effects of PM were not affected by the more complex consideration of weather factors. Likewise, population increases over time must be taken into account since they could, by themselves, explain some increases in daily mortality. In addition, in cities with temperate climates throughout the world, colder winter seasons are associated with more respiratory disease and mortality. Again, failure to adjust for seasonal patterns in mortality could lead to a false attribution of these effects to air pollution.

Most of the air pollution-mortality studies published over the last decade employ statistical techniques that control for these potentially confounding influences. In particular, recent, higher-quality studies are characterized by: (1) use of Poisson regression models, since mortality is a rare event and can be described by a Poisson distribution; (2) three or more years of daily data in a given city or metropolitan area; (3) examination of the effect of day-of-the-week and daily changes in the weather; and (4) use of locally weighted smoothing (loess). The latter is a technique that can account for both time trends and seasonal patterns (due to variations in weather and population susceptibility) in daily mortality data. The loess smoothing technique can accommodate nonlinear and nonmonotonic patterns between time and other factors and the health outcome, offering a flexible nonparametric modeling tool. Including a smoothed variable in the model does not explain the underlying reason for the pattern over time, but controls for it statistically, allowing one to observe the relationship between daily mortality and environmental factors after the underlying trend in daily mortality is controlled for. In addition, adding a locally weighted smooth of time diminishes short-term fluctuations in the data, thereby helping to reduce the degree of serial correlation. Serial correlation exists when the errors of the regression model are related over time, producing biased estimates of the variance of the explanatory variable coefficients, which may in turn result in spurious tests of statistical significance.

With increasing statistical sophistication, these studies have shown that either one-day or multi-day PM average concentrations are associated with both total and cardiopulmonary mortality. However, although acute exposures have repeatedly been reported to exert an independent effect on mortality, the influence of a single 24-hour exposure at a concentration relevant to the PM standards, absent any other exposure to PM, has not been (and probably cannot be) determined epidemiologically. Our review focuses primarily on those studies that used PM10 or PM2.5 as the exposure metric. Other measures of PM include black smoke (BS), coefficient of haze (COH), and sulfates.

7.3.1 General Results

There are now many studies linking short-term (i.e., daily) changes in PM10 with premature mortality. This includes not only studies from throughout the U.S., including several from California, but also those from a diverse group of cities throughout the world: such as Santiago, Chile (Ostro et al., 1996), Mexico City (Castillejos et al., 2000), Sao Paulo, Brazil (Saldiva et al., 1995), Amsterdam (Verhoeff et al., 1996), Bangkok (Ostro et al., 1999a) and Sydney (Morgan et al., 1998). Such cities span a wide range of environmental and population characteristics, including temperature–air pollution relationships, housing stock, transportation systems, industrial emissions, population age distributions, typical activity patterns, and baseline health conditions. Meta-analyses of earlier mortality studies suggest that, after converting the alternative measures of particulate matter used in the original studies to an equivalent PM10 concentration, the effects on mortality are fairly consistent (Ostro, 1993; Dockery and Pope, 1994; Schwartz, 1994a). Specifically, the mean estimated change in daily mortality associated with a one-day 10 $\mu g/m^3$ change in PM10 implied by these studies is approximately 0.8 percent, with a range of 0.5 percent to 1.6 percent. Since these meta-analyses were published, many more studies of acute exposure-mortality have been completed. All include control for weather and other potential confounding factors and most use sophisticated smoothing techniques as well. Table 7.1 summarizes the acute exposure mortality studies that have directly measured PM10. The table provides information for single-pollutant models of all-cause mortality, using the lags demonstrating the strongest associations with mortality, based on t-statistics. When necessary, the authors were contacted to complete the information provided in the tables and figures in section 7.3.

Table 7.1 Summary of Cities Included in Short-term PM10 Studies for All Age Groups (except where noted), Sorted by Mean* Concentration

ID	City/Region	Country	Reference	Time Period	Mean* in µg/m^3 (BOLD=Median)	% Increase (95% CI) per 10 µg/m3	# of obs
1	Stockholm	Sweden	Katsouyanni et al., 2001	Jan 1990-Dec 1996	**14**	0.39 (-1.30, 2.08)	2555
2	Portage, WI	US	Schwartz et al., 1996	Mar 1979-Dec 1987	18	0.7 (-0.4, 1.7)	1436
3	Sydney	Australia	Morgan et al., 1998	Jan 1989-Nov 1993	18	0.95 (0.32, 1.60)	1795
4	Ottawa	Canada	Burnett et al., 2000	1986-1996	20	1.45 (-0.88, 3.78)	433
5	Edinburgh	Scotland	Prescott et al., 1998	1992-1995	21	0.1 (-2.8, 3.0)	1460
6	Birmingham	England	Katsouyanni et al., 2001	Jan 1992-Dec 1996	**21**	0.28 (-0.23, 0.80)	1825
7	Vancouver	Canada	Burnett et al., 2000	1986-1996	22	1.46 (-0.28, 3.20)	565
8	Paris	France	Katsouyanni et al., 2001	Jan 1991-Dec 1996	**22**	0.43 (-0.02, 0.88)	2190
9	Helsinki	Finland	Katsouyanni et al., 2001	Jan 1993-Dec 1996	**23**	0.32 (-0.51, 1.16)	1460
10	Edmonton	Canada	Burnett et al., 2000	1986-1996	23	1.28 (-0.90, 3.46)	508
11	Buffalo-Rochester, NY	US	Gwynn et al., 2000	May 1988-Oct 1990	24	2.33 (0.49, 4.16)	175
12	Boston, MA	US	Schwartz et al., 1996	May 1979-Jan 1986	25	1.2 (0.7, 1.7)	1140
13	London	England	Katsouyanni et al., 2001	Jan 1992-Dec 1996	**25**	0.69 (0.35, 1.03)	1825
14	Calgary	Canada	Burnett et al., 2000	1986-1996	26	1.47 (-0.49, 3.43)	598
15	Birmingham	England	Wordley et al., 1997	Apr 1992-Mar 1994	26	1.1 (0.1, 2.1)	730
16	Winnipeg	Canada	Burnett et al., 2000	1986-1996	26	0.35 (-1.18, 1.88)	538
17	Toronto	Canada	Burnett et al., 2000	1986-1996	26	0.67 (-0.02, 1.36)	889
18	Topeka, KS	US	Schwartz et al., 1996	Sep 1979-Oct 1988	27	-0.5 (-2.0, 0.9)	1432
19	Montreal	Canada	Burnett et al., 2000	1986-1996	27	0.51 (-0.25, 1.27)	853
20	Basel	Germany	Katsouyanni et al., 2001	Jan 1990-Dec 1995	**28**	0.41 (-0.44, 1.27)	2190
21	Helsinki (Mortality for under 65 years)	Finland	Ponka et al., 1998	1987-1993	**28**	3.45 (1.08, 5.88)	2555
22	Minneapolis, MN	US	Braga et al., 2000	1986-1993	28	1.34 (0.78, 1.90)	2920

ID	City/Region	Country	Reference	Time Period	Mean* in µg/m^3 (BOLD=Median)	% Increase (95% CI) per 10 µg/m3	# of obs
23	St. Louis, MO	US	Dockery et al., 1992	Sep 1985-Aug 1986	28	1.50 (0.15, 2.85)	311
24	Zurich	Switzerland	Katsouyanni et al., 2001	Jan 1990-Dec 1995	**28**	0.42 (-0.30, 1.15)	2190
25	London	England	Bremner et al., 1999	Jan 1992-Dec 1994	29	0.26 (-0.20, 0.72)	1095
26	Kingston/Knoxville, TN	US	Dockery et al., 1992	Sep 1985-Aug 1986	30	1.60 (-1.32, 4.52)	330
27	St Louis, MO	US	Schwartz et al., 1996	Sep 1979-Jan 1987	31	0.6 (0.1, 1.0)	1375
28	Detroit, MI	US	Lippmann et al., 2000	1992-1994	31	0.86 (-0.22, 2.00)	490
29	Windsor	Canada	Burnett et al., 2000	1986-1996	31	2.88 (0.88, 4.88)	850
30	Knoxville, TN	US	Schwartz et al., 1996	Jan 1980-Dec 1987	32	0.9 (0.1, 1.8)	1481
31	Montreal	Canada	Goldberg et al., 2001a	1984-1993	32	0.67 (-0.16, 1.49)	3650
32	Seattle, WA	US	Braga et al., 2000	1986-1993	32	0.52 (0.11, 0.94)	2920
33	Ogden, UT	US	Pope et al., 1999a	1985-1995	32	1.62 (0.30, 2.90)	2308
34	Geneva	Switzerland	Katsouyanni et al., 2001	Jan 1990-Dec 1995	**33**	-0.10 (-1.02, 0.81)	2190
35	Madrid	Spain	Katsouyanni et al., 2001	Jan 1992-Dec 1995	**33**	0.53 (0.07, 1.00)	1460
36	San Jose, CA	US	Fairley, 1999	1989-1996	34	1.54 (0.74, 2.34)	823
37	Chicago, IL	US	Braga et al., 2000	1986-1993	36	0.81 (0.54, 1.09)	2920
38	Chicago, IL	US	Schwartz, 2001a	1988-1993	**36**	0.89 (0.61, 1.16)	2190
39	Detroit, MI	US	Braga et al., 2000	1986-1993	36	0.87 (0.60, 1.15)	2920
40	Pittsburgh, PA	US	Braga et al., 2000	1986-1993	36	0.84 (0.51, 1.18)	2920
41	Provo/Orem, UT	US	Pope et al., 1999a	1985-1995	38	0.95 (0.15, 1.75)	3687
42	Lyon	France	Katsouyanni et al., 2001	Jan 1993-Dec 1997	**39**	1.35 (0.31, 2.39)	1825
43	Athens	Greece	Katsouyanni et al., 2001	Jan 1992-Dec 1996	**40**	1.53 (0.98, 2.09)	1825
44	Budapest	Hungary	Katsouyanni et al., 2001	Jan 1992-Dec 1995	**40**	0.29 (-0.62, 1.19)	1460
45	Chicago, IL	US	Ito and Thurston, 1996	1985-1990	41	0.5 (0.3, 0.8)	1529
46	Salt Lake City, UT	US	Pope et al., 1999a	1985-1995	41	0.77 (0.30, 1.30)	3700
47	Teplice	Slovakia	Katsouyanni et al., 2001	Jan 1990-Dec 1997	**42**	0.64 (-0.03, 1.32)	2920

ID	City/Region	Country	Reference	Time Period	Mean* in µg/m^3 (BOLD=Median)	% Increase (95% CI) per 10 µg/m3	# of obs
48	Tel Aviv	Israel	Katsouyanni et al., 2001	Jan 1991-Dec 1996	**43**	0.64 (0.13, 1.15)	2190
49	Mexico City	Mexico	Castillejos et al., 2000	1993-1995	45	1.83 (0.98, 2.68)	866
50	Detroit, MI	US	Lippmann et al., 2000	1985-1990	45	0.34 (0.04, 0.64)	1565
51	Steubenville, OH	US	Schwartz et al., 1996	Apr 1979-Sep 1987	46	0.9 (0.1, 1.6)	1520
52	Phoenix, AZ (Mortality for 65 years and older)	US	Mar et al., 2000	1995-1997	46	1.06 (0.01, 2.11)	1095
53	Coachella Valley, CA	US	Ostro et al., 2000	1989-1998	47	0.41 (-0.41, 0.81)	3011
54	Milano	Italy	Katsouyanni et al., 2001	Jan 1990-Dec 1996	**47**	1.16 (0.79, 1.53)	2555
55	Utah Valley, UT	US	Pope et al., 1992	Apr 1985-Dec 1989	47	1.47 (0.86, 2.08)	1706
56	Birmingham, AL	US	Schwartz, 1993	Aug 1985-1988	48	1.1 (0.2, 2.0)	1248
57	Erfurt	Germany	Katsouyanni et al., 2001	Jan 1991-Dec 1995	**48**	-0.56 (-1.34, 0.21)	1825
58	Cracow	Poland	Katsouyanni et al., 2001	Jan 1990-Dec 1996	**54**	0.13 (-0.54, 0.81)	2555
59	Rome	Italy	Katsouyanni et al., 2001	Jan 1992-Dec 1996	**57**	1.28 (0.75, 1.81)	1825
60	Los Angeles, CA	US	Kinney et al., 1995	Jan 1985-Dec 1990	58	0.5 (0.0, 1.1)	364
61	Barcelona	Spain	Katsouyanni et al., 2001	Jan 1991-Dec 1996	**60**	0.93 (0.57, 1.29)	2190
62	Bangkok	Thailand	Ostro et al., 1999a	Jan 1992-Nov 1995	65	1.70 (1.11, 2.29)	1431
63	Torino	Italy	Katsouyanni et al., 2001	Jan 1990-Dec 1996	**65**	1.05 (0.71, 1.38)	2555
64	Prague	Czech	Katsouyanni et al., 2001	Feb 1992-Dec 1996	**66**	0.12 (-0.24, 0.48)	1795
65	Sao Paulo (Mortality for 65 years and older)	Brazil	Saldiva and Bohm, 1995	May 1990-Apr 1991	82	1.31 (0.28, 2.33)	365
66	Rome	Italy	Michelozzi et al., 1998	Jan 1992-Jun 1995	84	0.66 (0.31, 1.02)	1278
67	Santiago	Chile	Ostro et al., 1996	1989-1991	115	1.13 (0.87, 1.39)	779

* Average of 24-hour measurements over time period.

Among the first of the multi-city studies on mortality, Schwartz et al. (1996) examined data from the Harvard Six-Cities investigation. This database included monitors sited specifically to support ongoing epidemiological studies and be representative of local population exposures. Consistent associations were reported between daily mortality and daily exposures to both PM10 and PM2.5. The mean concentrations of PM10 among the six cities ranged from 18 to 47 $\mu g/m^3$ (overall mean of 30 $\mu g/m^3$) with a joint effect estimate indicating a 0.8% (95%CI = 0.5 – 1.1) increase in daily total mortality per 10 $\mu g/m^3$ of PM10.

Samet et al. (2000a) applied a wide range of statistical tools and sensitivity analyses to a database consisting of the 88 largest cities in the United States (NMMAPS), while Samet et al. (2000b) focused on the 20 largest cities. For both of these studies, the combined effect of all of the cities indicated an association consistently within but near the lower end of the range reported by earlier researchers (approximately 0.5% per 10 $\mu g/m^3$ of PM10). Among these cities, the long-term mean PM10 concentrations ranged from 24 to 46 $\mu g/m^3$. The authors examined pollution and sociodemographic factors that might modify the estimated effects of PM10. They reported no association, in univariate models, between the effect estimates for each of the cities and the mean level of PM or other pollutants (ozone, nitrogen dioxide, sulfur dioxide or carbon monoxide) in the city. This suggests a constant slope or effect per $\mu g/m^3$ of PM regardless of the average concentration of PM or other pollutants. In addition, city-wide estimates of sociodemographics such as median income, percent unemployed, and percent below poverty level did not modify the estimated effect of PM. However, there may have been insufficient statistical power to detect any effect of such socioeconomic factors on the PM-mortality relationship.

Samet et al. (2000a) indicated that their estimates may be at the lower end of the range because their database included a wide range of cities and incorporated findings in some cities where no effects were observed. There may be other explanations for the lower effects, however. For example, the studies only considered lags (or delayed effects) of zero, one or two days, or an average of zero and one day lags, though other studies have reported greater effects with longer lags or multi-day moving averages. Since many of the cities in the study collected PM10 data on an every-sixth-day basis, cumulative averaging times could not be examined. Another possible reason for the lower effect estimates in the study by Samet et al. (2000a) relates to the number of covariates used in the regression model. Besides PM10, day of week, and a smooth of time using 7 degrees of freedom (or cycles of about 7 weeks), two variables were included for temperature and two for dewpoint (same day and an average of the three previous days). Most previous mortality studies used fewer controls for weather factors or, more appropriately, modeled extreme weather events (e.g., binary variables indicating a day with temperature above 80 degrees or below 32 degrees). To the extent that PM may be causally related to mortality and correlated as well with these meteorological variables, these multiple statistical controls could result in an underestimate of the effects of PM, though residual confounding by weather factors might also bias the PM effects away from the null hypothesis of no effect. Thurston and Ito (2001) demonstrated that the modeling of weather factors had a significant impact on the estimated effect of ozone, and postulated that it could impact the estimated effects of secondary aerosols, as well.

The largest and most significant regional effects were found for the Northeast U.S. and for Southern California, with modest heterogeneity in the PM-mortality relationships from region to region. The regional heterogeneity may have resulted from differences in: (1) the particle composition and size distributions; (2) the underlying distributions of age, chronic disease, and other determinants of susceptibility among the local populations, including behaviors, activity patterns, and exposures; or (3) the density of pollutant monitors and relative exposure measurement errors. Moreover, the application of a similar statistical model to all 90 cities

may have contributed to the inter-city and inter-regional variability observed by these researchers. Similar loess smoothers of time and temperature were used throughout the country, despite the diversity of climate, PM sources, and population characteristics. By not tailoring the model to each locale, they may have had varying degrees of "goodness-of-fit" of the models to the mortality patterns in the individual cities, which might either exaggerate or underestimate the magnitude of the associations between ambient PM and daily mortality in different locations. In the Samet et al. (2000a) analysis, the averaged effect for the six California counties studied (Los Angeles, San Diego, Orange, Santa Clara, San Bernardino and Alameda) was 0.9% per 10 $\mu g/m^3$ (with a range of 0.3% to 2.0%) versus 0.5% for all 90 cities together. The same data set was used to address issues relating to potential exposure measurement error bias and confounding by co-pollutants. They found that measurement error would likely underestimate the effect of PM (Zeger et al., 2000) and that co-pollutants such as ozone, nitrogen dioxide, sulfur dioxide, and carbon monoxide did not significantly affect or confound the estimated effect of PM (Samet et al., 2000a).

In studies of 10 U.S. cities, Schwartz (2000a,b) examined the effects of PM10 for all age groups, and for a more limited subset of individuals above age 65. For the group of all ages, a 10 $\mu g/m^3$ change in PM10 (measured as a two-day average of lag 0 and lag 1) was associated with a 0.7% increase in daily mortality. For the elderly age group, the same change in PM10 was associated with a 1.1% increase in mortality. For these 10 cities, the arithmetic mean of PM10 ranged from 27 to 41 $\mu g/m^3$.

In another multi-city study, Burnett et al. (2000) analyzed mortality data for 1986 - 1996 from the eight largest Canadian cities. This study found that both PM10 and PM2.5 were associated with daily mortality. For PM10, a 10 $\mu g/m^3$ increase was associated with a 0.7% (95%CI = 0.2 – 1.2) increase in daily mortality, with a mean PM10 concentration of 26 $\mu g/m^3$. For PM2.5, a similar 10 $\mu g/m^3$ increase was associated with a 1.2% increase in daily mortality (95% C.I. = 0.44 – 1.96). Moolgavkar (2000a) examined the association between air pollution and mortality in three large U.S. counties: Cook (including Chicago), Maricopa (including Phoenix), and Los Angeles, for 1987 through 1995. For the latter two counties, only every sixth day measures of PM10 were available, unlike most of the other studies which had daily data (except Samet et al., 2000a, b). PM10 was significantly associated with mortality in all three counties but with a lower effect estimate (approximately 0.2 to 0.4% per 10 $\mu g/m^3$) than found in most other studies. In addition, the author concluded that it was difficult to assign the effect to any single pollutant because of the high correlation among pollutant measurements.

Another multi-city study involved 29 European cities that measured PM10 (although in some of the cities PM10 was estimated from observations collected from a subset of days using co-located TSP or Black Smoke monitors) lKatsouyanni et al., 2001). Using a methodology similar to the U.S. studies cited above, an association between daily mortality and PM10 was reported, with an overall effect estimate of 0.6% per 10 $\mu g/m^3$. The study reports heterogeneity in the effect estimates, which was likely due to real differences in PM sources and exposures among the cities. In this regard, cities that had higher concentrations of nitrogen dioxide, indicating the likelihood of a greater contribution of ambient pollution from mobile sources, especially diesel, demonstrated greater PM10-associated effects. For example, for cities in the lowest quartile for nitrogen dioxide, the estimated PM10 effect was 0.2% per 10 $\mu g/m^3$, while for cities in the highest quartile for nitrogen dioxide the effect estimate was 0.8% per 10 $\mu g/m^3$.

In addition to these multi-city investigations, studies examining the effect on mortality of short-term exposure to PM have been conducted in over 100 cities. Those studies that specifically use PM10 (as opposed to Black Smoke, Coefficient of Haze (COH), nephelometry data or

other measures of PM) as their exposure metric are summarized in Table 7.1, which displays the estimated effect and ambient concentration of PM10 for each city. As in the studies conducted in the early 1990s, these studies indicate a generally consistent mortality effect of around 1% per 10 $\mu g/m^3$ of PM10. Taken together and combined with the evidence of morbidity effects described below, these studies provide compelling evidence of a significant impact of PM on mortality. Although the relative risk per unit is low, the large number of people exposed suggests the existence of a major impact on public health.

Many of the above studies reported that lags in exposure to PM10 of one to four days exhibited stronger associations with mortality than did same-day exposures to PM10. In addition, cumulative exposures of three or five days, when tested, often had stronger associations than single-day lags. Recent analyses demonstrate that effect estimates increase when a longer-term average of exposure is used. For example, Schwartz (2000b) examined mortality for those above age 65 in 10 U.S. cities. A regression model that allowed for an air pollution effect to persist over several days using a distributed lag was incorporated, resulting in a doubling of the relative risk, to approximately 2% per 10 $\mu g/m^3$ of PM10.

In a separate study restricted to out-of-hospital deaths (i.e., excluding those due to homicide or trauma), the effect size increased four-fold (Schwartz, 2001a). Schwartz (1994b) had previously found a much greater likelihood of deaths occurring outside of hospitals or clinics on days with high versus low concentrations of PM (measured as Total Suspended Particles or TSP). These findings suggest that particulate air pollution may have had a greater impact among individuals who were not in the hospital when exposed and who were not admitted to the hospital before expiring. Sudden death may therefore be a factor in air pollution-related mortality, which suggests that the average impact on loss of life is likely to be more than just a few days, since it need not include only those already chronically ill and hospitalized. The recent paper by Peters et al. (2000a), demonstrating associations between serious cardiac arrhythmias and several pollutants, including PM2.5 and black carbon, supports this notion. However, deaths occurring among those outside of a hospital may represent individuals who are frail or without health insurance, or both. In contrast to the results reported by Schwartz (1994b), Levy et al. (2001) did not find any association between PM10 and the incidence of primary cardiac arrest using a case-crossover analysis. This study, though, involved a small number of cases in Seattle, where relatively low levels of PM occurred during the study period [1988-1994, mean PM10 = 31.9 $\mu g/m^3$, mean PM2.5=18.4 $\mu g/m^3$].

The results of these studies also indicate that the associations between PM and mortality are not significantly confounded by weather patterns, longer-term seasonality, or day of week. This evidence is provided by careful modeling and controlling for these factors in the individual studies, as well as by the heterogeneous nature of the cities examined. Specifically, consistent evidence of an effect of PM has been observed in cities in both cold (e.g., Detroit and Montreal) and warm (e.g., Mexico City and Bangkok) climates, in some cities where PM peaks in the summer (Steubenville, Philadelphia) and in others with peaks in winter (e.g., Utah Valley) or spring (Helsinki), and in cities with substantial seasonal changes in mortality (e.g., Chicago) and in others with little seasonality (e.g., Coachella Valley, Birmingham, Bangkok). These factors are carefully modeled and controlled for in the studies, and the mortality results are consistent throughout, thereby providing compelling evidence of an effect. Furthermore, factors such as smoking, exposure to secondhand smoke or occupational irritants, and personal characteristics are not confounders in these studies since they do not vary with air pollution on a daily basis.

A related issue is whether there is independent evidence of an effect of PM, or whether confounding by co-pollutants makes it impossible to implicate PM as a pollutant of concern. One method for examining such potential confounding involves including multiple pollutants in

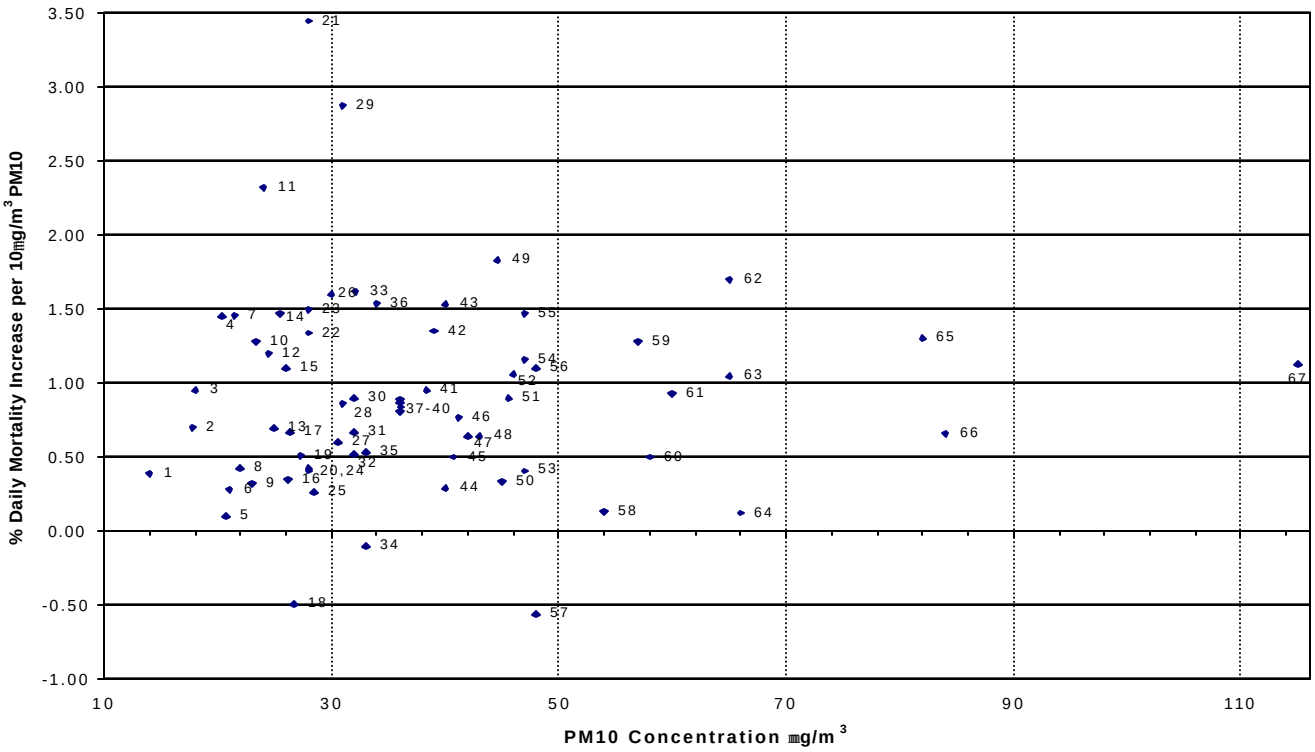
the explanatory regression model. While this method can help rule out confounding effects if the effect of PM10 is unchanged when other pollutants are included in the model (assuming non-differential measurement error), the reverse is not true. If the estimated effect of PM10 is altered after inclusion of other pollutants, this may be a predictable result of statistical collinearity. It is well established that regression estimates can vary widely with the inclusion/exclusion of highly correlated covariates. It may also be the result of differential pollutant measurement errors or monitor performance. However, single-pollutant models may incorporate the effects of other highly correlated pollutants not included in the model, so that any health impact attributed to the pollutant in the model may be overestimated.

Despite these potential limitations, there is substantial evidence from the available literature that PM effects are, in general, not substantially affected by co-pollutants. In many of the time-series mortality studies, inclusion of additional pollutants into the regression model does not alter the estimated impact of PM. Samet et al. (2000a) provide a recent and comprehensive test of this theory using the data set consisting of 90 U.S. cites, as described earlier. The authors sequentially tested the estimated effect of PM10 after gaseous pollutants (ozone, nitrogen dioxide, sulfur dioxide, and carbon monoxide) were each added to the regression model. The authors report minimal change in the estimated PM10 coefficient after these inclusions. Similar results have been reported in most studies that have examined PM10 and mortality, with few exceptions (e.g., Moolgavkar, 2000a). In a different approach to the issue, Schwartz (2000a) examined the sensitivity of the PM10 coefficient to different amounts of co-pollutant covariation among 10 U.S. cities. Theoretically, if the PM10 effect were really a result of confounding by another pollutant, the estimated PM10 effect per $\mu g/m^3$ would be greater in those cities where PM10 was highly correlated with other pollutants, indicating that PM10 was taking on some of the explanatory power of the "true" causal co-pollutant. However, Schwartz (2000a) did not find any evidence consistent with this hypothesis, suggesting that confounding of the effects of PM10 by other pollutants was unlikely. Similarly, in the study of 29 European cities, Katsouyanni et al. (2001) report no effect modification or confounding associated with either ozone or sulfur dioxide. PM effects were higher in cities with higher concentrations of nitrogen dioxide, but the effects of PM were not attenuated.

The recent findings of Sarnat et al. (2001) are important in assessing the usefulness of multi-pollutant models. In a study of 56 subjects in Baltimore, studied over both the summer and winter seasons, ambient and personal exposure to PM2.5, ozone, sulfur dioxide, nitrogen dioxide and carbon monoxide were measured for 12 consecutive days. Ambient concentrations of PM2.5 were correlated with the ambient concentrations of the gaseous pollutants. Personal and ambient PM2.5 exposures were also correlated, but personal and ambient concentrations were not related for any of the gases. In fact, ambient measures of the gases were associated with personal exposure to PM2.5. The authors concluded that this indicates that ambient PM2.5 is a suitable surrogate for personal PM2.5 and that ambient gaseous concentrations are surrogates, not confounders, for PM2.5. Therefore, multi-pollutant models may not be a suitable method of evaluating the effects of gaseous pollutants, and the health effects attributable to ambient gases may result from exposure to PM2.5. This important finding needs to be replicated in other settings.

We have attempted to provide a context for both the average ambient concentrations and the statistical level of uncertainty in these studies. Figure 7.1 and Table 7.1 summarize the estimated effect levels and the associated average concentrations for the available studies that used PM10. (Unpublished data for individual city results within multi-city studies were graciously supplied by the authors.) This obviates the need to adjust from some other PM

Figure 7.1 Daily Mortality Estimates and PM10 Concentration



Note: Both median and mean are used to indicate average study concentration. Number in the figure refers to city identifier; see Table 7.1 for study details.

measure (such as black smoke to PM10), and thereby reduces one source of uncertainty. The figure indicates that many studies in which the average PM10 concentrations are in the range of 20 to 30 $\mu g/m^3$ show associations between daily exposure to PM10 and mortality. However, all of the published studies at the lower end of the range have been conducted outside of California, and several are from outside the U.S. The cities are sorted by PM10 concentration in Table 7.1 and show, for example, that the 10 lowest concentrations occur in Stockholm, Portage (Wisconsin), Sydney, Ottawa, Edinburgh, Vancouver, Paris, Helsinki, and Edmonton. Factors that may affect the PM-mortality relationships, including sources of PM, different distributions of PM size and chemical compositions, time spent outdoors, proximity to roadways, climate, population age distribution and health status, smoking characteristics, and use of medical care, may all affect extrapolations to California. Figure 7.2 demonstrates that the studies themselves may involve greater uncertainty at lower mean PM10 ambient concentrations. As the average PM10 level decreases, the confidence intervals of the estimated effect on mortality tend to increase. The associated t-statistic (which equals the regression coefficient divided by the standard error of the estimate) is a unit-free measure of the association in each of the regressions. The larger the t-statistic, the stronger the

association and the smaller the 95% confidence interval associated with the estimated effect. The larger the t-statistic, the stronger the association and the smaller the 95% confidence interval associated with the estimated effect. Therefore, Figure 7.2 also indicates that at lower ambient concentrations, the t-statistic tends to be lower as well. This simple figure, however, does not account for other factors that may be confounding this relationship. For example, studies conducted in generally less polluted cities may involve other factors that affect the association, such as weather, particle composition, or housing stock (i.e., with different levels of "tightness" and infiltration rates). In addition, lower variation in the pollution exposure, with everything else the same, will result in greater variance in the estimated pollution regression coefficient. Therefore, Figure 7.2 can only be considered suggestive regarding the reasons for the greater degree of uncertainty at lower concentrations. It should be noted that many studies have found statistically significant associations between PM10 and mortality at low ambient concentrations and that analyses explicitly conducted to determine thresholds have failed to detect any (see section 7.3.5 below). Therefore, Figure 7.2 should not be construed as demonstrating a threshold level of zero risk. It also should be noted that the large (n = 88) multi-city study of short-term exposure and mortality by Samet et al. (2000a) found that although the magnitude of the estimated mortality effect varied across all of cities (and tended to be associated with PM within each city), the effect estimate was independent of the mean PM10 in any given city. Thus, cities with higher average concentrations of PM10 tended to have the same general effect per microgram of PM10 as cities with lower averages. The t-statistic associated with the estimated coefficient of PM10 will be affected by both the strength of the association between PM10 and mortality, and the number of observations used in the regression model. Theoretically, the t-statistic should increase with the square root of the number of observations. In order to control for this factor and still determine whether the concentrations of PM10 were associated with greater uncertainty, we conducted a simple statistical analysis of the 62 single-city studies for which we had complete data for all-cause mortality for all age groups together (see Table 7.1 for details of the studies). Only all-age, all-cause mortality results are included, using the lag with the highest association with mortality, based on the t-statistic. In the analysis, we used ordinary least squares multiple regression to explain variations in the t-statistic as a function of both the number of study observations (days) and the average concentration of PM10. We also used locally weighted smoothing analysis (Cleveland and Devlin, 1988) to examine the shape of the possible associations. Both the concentration of PM and the square root of the number of days in the study appear to have linear associations with the t-statistic. Specifically, we found the following relation:

$$\text{Tstat} = -0.39 + 0.025\ \text{SRN} + 0.0528\ \text{PM}$$

$$(0.019) \qquad (0.0129)$$

$$p = 0.18 \qquad p < 0.0001$$

$$R^2 = 0.25$$

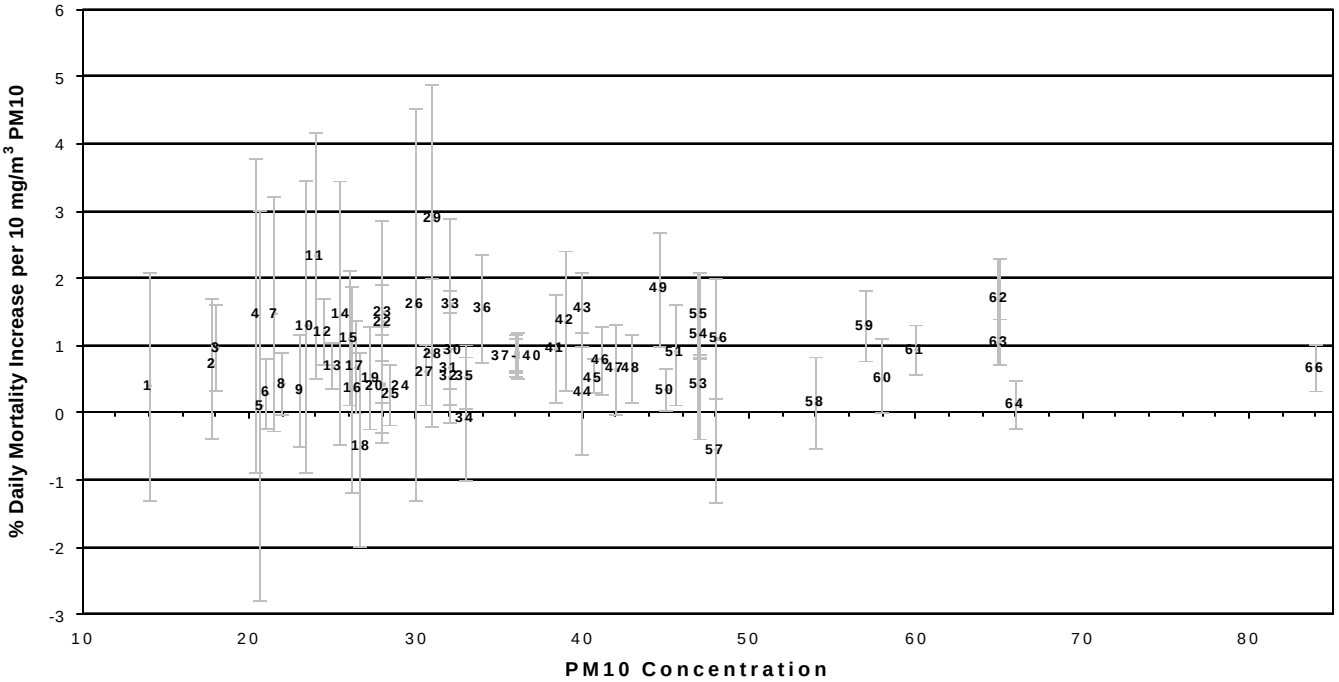
where Tstat = t-statistic of the association between PM10 and mortality,

SRN = square root of the number of days of the study,

PM = average study concentration of PM10

(standard errors in parentheses).

Figure 7.2 Uncertainty in Daily All-Age, All-Cause Mortality Studies



Note: Bars represent 95% CI of estimated PM10 effect; number in the figure refers to city identifier. The city identifier is placed at the point estimate location. Santiago, Chile does not appear in this graph. See Table 7.1 for study details.

The estimated coefficients indicate that uncertainty (the inverse of the t-statistic) decreases with increasing sample size and PM concentration. The coefficient of SRN had a p-value of 0.18, while the coefficient of PM had a p-value < 0.0001, indicating that the mean PM concentration may be an important determinant of the level of uncertainty in these studies. About 25% of the variation in the dependent variable Tstat was explained by the two terms. The lack of statistical significance of the study day coefficient suggests confounding by one or more omitted variables, which might include other time-variant factors such as co-pollutants or meteorological factors, or other variables such as population size, which would determine the number of deaths/day. The statistical significance of the study PM10 concentration coefficient may also be influenced by unmeasured covariates. However, the high precision of that estimate suggests that PM10 concentrations would still be an important predictor even with the inclusion of other covariates in the model.

Figure 7.3 displays a plot of the linear fit for the predicted value of Tstat versus average PM10 concentration, after controlling for number of observations. While this simplistic analysis does not control for a wide range of other factors that may affect the strength of the association, it does suggest greater uncertainty at lower concentrations. The plot also indicates that there are at least two influential data points: the observations associated with the highest and lowest t-statistics. Therefore, as a sensitivity analysis, the model was rerun after deleting these two points. The resulting model produced a slightly lower coefficient for PM10 of 0.046 (s.e. = 0.015, $p < 0.01$), a higher coefficient for SRN of 0.031 (s.e. = 0.018, $p < 0.10$) with an $R^2 = 0.20$. Thus, both the number of observations and the study average concentration of

PM10 were associated with the t-statistic of the estimated effect of pollution when these influential observations were deleted.

7.3.2 Effects by Size Cuts: Fine and Coarse Particles

In the last several years, several daily exposure-mortality studies have examined associations using different particle cut sizes, especially PM2.5 and coarse (PM10 – PM2.5) (abbreviated below as CP). The ability of these epidemiological studies to differentiate between the effects of different PM size cuts, however, is limited by two factors. First, PM metrics in a given region are often highly correlated. For example, in many urban areas, PM2.5 and PM10 are highly correlated ($r > 0.7$) on a daily basis. On the other hand, in areas where crustal PM predominates, daily concentrations of PM10 are correlated with CP. The second factor that limits the interpretation of the epidemiological studies is the relative degree of exposure measurement error. Since PM2.5 tends to be more uniformly spatially distributed than CP, it is likely that a fixed-site monitor will be less precise in measuring the latter. Since misclassification of exposures would normally result in biasing the estimated effect downwards, the relative difference in measurement error could lead to relatively lower (and less certain) effect estimates for CP.

Earlier studies of PM2.5 used measures of PM2.5 components, such as sulfates (Bates and Sizto, 1987), or estimates of PM2.5 based on airport visibility (Ostro, 1995). Schwartz et al. (1996) was among the first studies using actual measures of PM2.5 in the Harvard Six-Cities data set, and then determining CP using the difference between PM10 and PM2.5. Based on both the individual-city analyses and a meta-analysis of all six cities, an association was demonstrated between daily mortality and PM2.5, but not CP. An effect of CP was observed in only one of the six eastern and mid-western cities included in the database (Steubenville, Ohio). In this study, the mean PM2.5 among the cities ranged from 11 to 30 $\mu g/m^3$ with a mean of 18 $\mu g/m^3$, while CP ranged from 7 to 16 $\mu g/m^3$, with a mean of 11.5 $\mu g/m^3$. These findings were validated in an independent replication of the six-Cities data by Klemm et al. (2000).

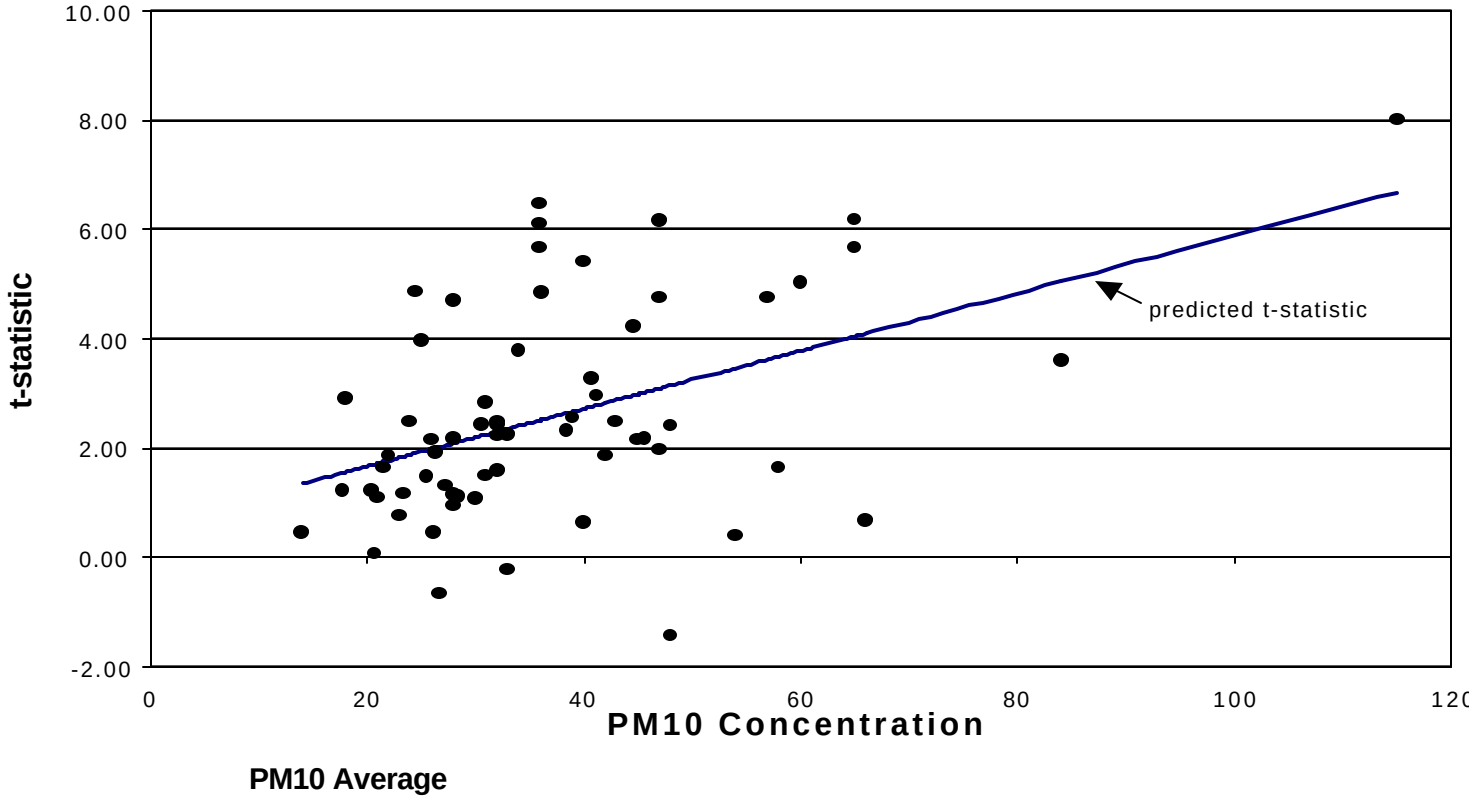
Among more recent studies (summarized in Table 7.2 and Figure 7.4) examining the relative impacts of coarse and fine particles, however, the results have been mixed. The estimated effects of PM appear to depend on: (1) the cities being studied; (2) the lags in exposure used in the statistical models; (3) the mortality endpoint(s) under study (i.e., all-cause versus cardiovascular or respiratory); and (4) the season(s) under study. In some cities, only a PM2.5 effect is found. In other cities, both PM2.5 and CP are associated with mortality, while in a third set of cities, an association is found only for CP. Table 7.2 provides a summary of these findings. For example, support for a dominant PM2.5 effect is provided by the Fairley (1999) study of Santa Clara County, California. In this study, PM10 (mean = 34) and PM2.5 (mean = 13) were associated with all-cause daily mortality, whereas no effect was observed for CP. When cardiovascular mortality was examined in relation to the three different PM cut sizes, associations were found for only PM10. A similar result was reported for all-cause mortality in a study of eight Canadian cities (Burnett et al., 2000). The effect of PM2.5 on mortality was stronger than that of CP, although the latter did demonstrate a positive, though weaker, association with mortality.

In contrast, results from Coachella Valley, CA (which includes Palm Springs), Detroit and Mexico City suggest effects of CP greater than those of PM2.5. In PM data from Coachella Valley, Ostro et al. (2000) found very high correlations between CP and PM10 ($R \sim 0.95$) with the ratio of CP/PM10 of approximately 0.60. This is the reverse of most urban areas, particularly in the eastern part of the U.S., where PM2.5 is more highly correlated with PM10 and the PM2.5/PM10 ratio is typically between 0.55 and 0.75 (U.S. EPA, 1996). Using 2.5

years of data of PM10 and PM2.5, both CP and PM10, but not PM2.5, were associated with cardiovascular mortality (Ostro et al., 2000). For all-cause mortality, no associations were found for the alternative measures of PM, with the exception of a 4-day lag in PM2.5, which was the only single-day lag demonstrating a positive association. In a previous study conducted in the same geographic location using data from 1989 - 1992, there was also an association between PM10 and cardiovascular mortality, although no measures were available for PM2.5 or CP (Ostro et al., 1999b).

The more recent Coachella Valley study (Ostro et al., 2000), as well as analyses by Pope et al. (1999a) and Schwartz et al. (1999), all indicate, however, that high PM days dominated by windblown dust were not associated with excess mortality. It is not clear whether these findings are due to lower toxicity of crustal particles (relative to those generated by combustion processes) or because people change their behavior and reduce exposure on windy days. Lippmann et al. (2000) examined the effects of different size cuts of PM using mortality data from Detroit and pollution data from the adjacent city of Windsor, Canada. For this study, daily data were collected from May to September with every third- or sixth-day data during the rest of the year, over a two-year period. No associations were reported between all-cause mortality and any PM metrics. However, for cardiovascular mortality, associations were reported for CP, but not PM2.5. Finally, in a study of four years of data from Mexico City, CP had a larger impact and stronger association than PM2.5 for all-cause, cardiovascular and respiratory mortality (Castillejos et al., 2000).

Figure 7.3 Comparison of t-statistic from Daily All-Age Mortality Studies and Study



Note: Regression fit after controlling for number of observations in study.

Three separate studies of PM-mortality relationships in Phoenix also demonstrate effects from exposure to CP. Mar et al. (2000) found stronger associations of all-cause mortality with CP than with PM2.5 for individuals 65 and older. Equally strong associations were reported linking both PM2.5 and CP with cardiovascular mortality. Using a different statistical model, Smith et al. (2000) also found stronger associations and estimated effects between all-cause mortality and CP, relative to PM2.5. Similarly, Clyde et al (2000) also reported stronger effects for CP in their analysis of data from Phoenix. Finally, Wichmann et al. (2000) analyzed several years of mortality data from Erfurt, Germany. Most of the analysis was focused on PM data using a mobile aerosol spectrometer, which provided size-specific number and mass concentration data in several size classes. However, filter-based impactor data on PM10 and PM2.5 were collected at the same time. Analyses of these data indicated associations between daily cardiovascular or respiratory mortality and PM10, PM2.5 and ultrafine particles.

More mixed results were generated from an analysis of PM2.5 and CP data from Santiago, Chile (Cifuentes et al., 2000). The authors reported that the results were season-dependent. PM2.5 had a stronger association with mortality for the year as a whole and in the winter, whereas CP had a stronger effect during summer. Lipfert et al. (2000a) analyzed data from Philadelphia and the surrounding metropolitan area. For all-cause mortality in Philadelphia, stronger associations (based on t-statistics) were reported for PM2.5 than CP, but the effects per $\mu g/m^3$ were of similar magnitude for the two measures. For cardiovascular mortality in the seven-county region, PM2.5 had a stronger association and effect size than CP, while for respiratory mortality, the effect size for CP was greater. Finally, in a relatively small data set from Pittsburgh, Pennsylvania, Chock et al. (2000) report no clear association between mortality and either PM2.5 or CP for individuals under 75 years old.

There are several issues related to the interpretation of these studies. For example, in some cities, PM2.5 are likely to be correlated with ozone. In Detroit the correlation was 0.49 (Lippmann et al., 2000), while the correlations between these pollutants in Montreal and Toronto were likely to have been higher (Burnett et al., 2000 - only the joint correlation across all eight cities was reported). Although several measures of PM were associated with mortality and morbidity in Detroit, the effects were less consistent than those observed in other studies. Also, when cardiovascular mortality was examined (in a subset of the above studies) instead of all-cause mortality, another mixed pattern emerged. For instance, in Santa Clara County, Fairley (1999) reported strong associations between cardiovascular mortality and PM10, but not PM2.5 or CP. In Coachella Valley (Ostro et al., 2000), Mexico City (Castillejos et al., 2000) and Detroit (Lippmann et al., 2000), associations were found for PM10 and CP, but not PM2.5. In contrast, Mar et al. (2000) reported associations between cardiovascular mortality and both PM2.5 and CP in Phoenix.

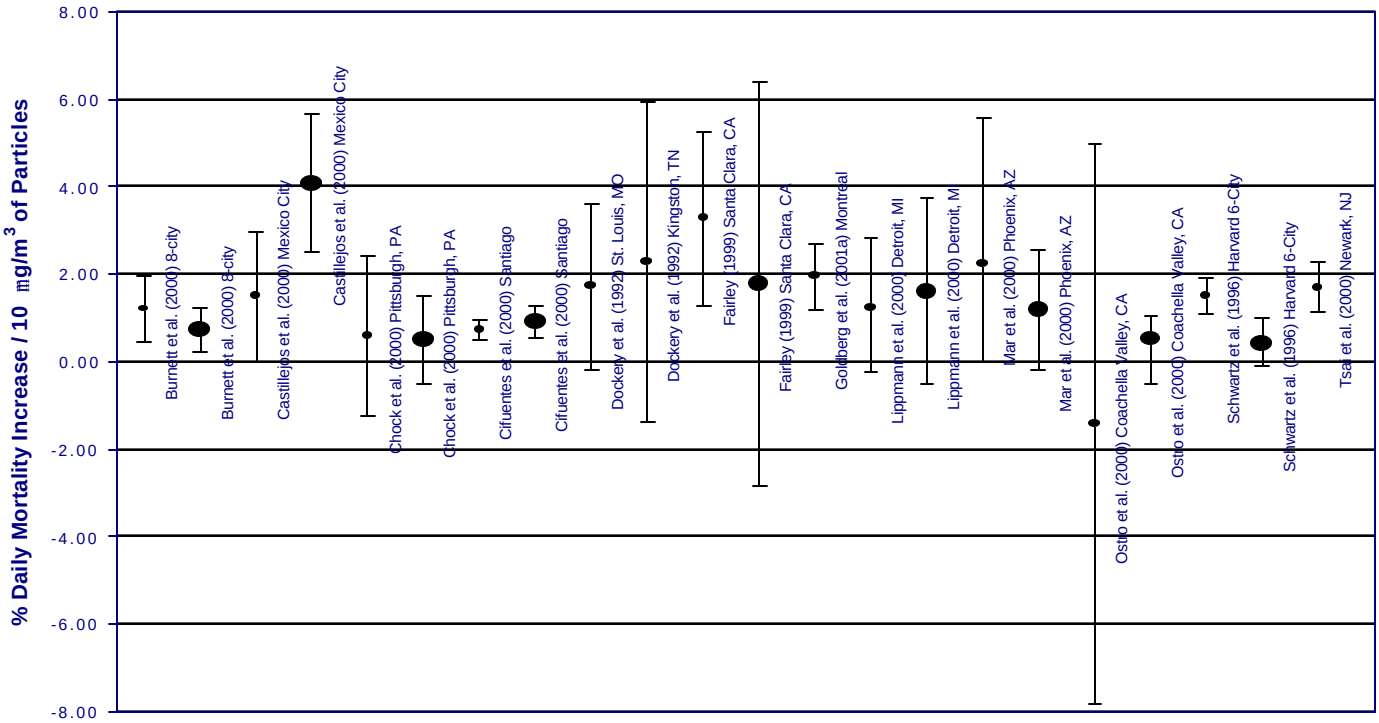
In summary, the relative results of PM2.5 versus CP, as summarized in Table 7.2 and Figure 7.4, are mixed. In some of the mortality studies, primarily those undertaken in cities on the East Coast, PM2.5 effects appear to predominate. In other studies, CP has a stronger association with mortality, while in a third set of studies, the effects of PM2.5 and CP are similar. However, on average, the effect of a unit mass increase in PM2.5 appears to be greater than a comparable increase in CP mass. For the studies summarized in Table 7.2 and Figure 7.4, the average effect of PM2.5 is about 1.7% per 10 $\mu g/m^3$, with a range of around 0.6 to 5.2%. For CP, the mean effect of the summarized studies is around 1% per $\mu g/m^3$ with a range from less than zero at the low end to 2 to 4% on the high end. Thus, the PM2.5 effect generally appears stronger per unit mass, due perhaps to greater intrinsic toxicity, greater indoor infiltration rates, lower exposure measurement error, or other factors.

Table 7.2. Cities Included in Studies of Short-term Exposure and Daily Mortality for All Age Groups (except where noted) Associated with PM2.5 (FP) and Coarse Particles (CP)

City	Country	Reference	Particle Type	Time Period	Mean* in $\mu g/m^3$ (except where noted)	% Increase (95% CI) per $10 \mu g/m^3$
8-Cities (Calgary, Edmonton, Montreal, Ottawa, Toronto, Vancouver, Windsor, Winnipeg)	Canada	Burnett et al., 2000	FP CP	1986-1996	13 13	1.20(0.45, 1.96) 0.71(-0.28, 1.71)
Mexico City	Mexico	Castillejos et al., 2000	FP CP	1993-1995	27 17	1.48(-0.01, 2.96) 4.07(2.49, 5.66)
Pittsburgh, PA (Mortality for 75 years and older)	US	Chock et al., 2000	FP CP	1989-1991	NA NA	0.59(-1.25, 2.43) 0.50(-0.51, 1.51)
Santiago	Chile	Cifuentes et al., 2000	FP CP	1988-1996	64 47	0.73(0.52, 0.94) 0.91(0.55, 1.27)
St. Louis, MO	US	Dockery et al., 1992	FP	1985-1986	18	1.71(-0.17, 3.59)
Kingston, TN	US	Dockery et al., 1992	FP	1985-1986	21	2.28(-1.37, 5.93)
Santa Clara, CA	US	Fairley, 1999	FP CP	1990-1996	13 11	3.26(1.27, 5.24) 1.77(-2.86, 6.41)
Montreal	Canada	Goldberg et al., 2001a	FP	1984-1993	18	1.93(1.16, 2.71)
Detroit, MI	US	Lippmann et al., 2000	FP CP	1992-1994	18 13	1.24(-0.26, 2.83) 1.58(-0.49, 3.74)
Phoenix, AZ (Mortality for 65 years and older)	US	Mar et al., 2000	FP CP	1995-1997	13 34	2.22(0.00, 5.56) 1.17(-0.20, 2.54)
Coachella Valley, CA	US	Ostro et al., 2000	FP CP	1995-1998	17 31	-1.42(-7.81, 4.97) 0.51(-0.51, 1.02)
Harvard 6-Cities (Boston, Knoxville, Portage, St. Louis, Steubenville, Topeka,)	US	Schwartz et al., 1996	FP CP	1979-1988	15 9 (medians)	1.5(1.1, 1.9) 0.4(-0.1, 1.0)
Newark, NJ	US	Tsai et al., 2000	FP	Summers & Winters 1981 & 1982	42	1.70(1.11, 2.29)

* Average of 24-hour measurements over time period.

Figure 7.4 Daily Mortality Increases Associated with Fine and Coarse Particles



Note: Bar represents 95% confidence interval; small and large dots represent fine and coarse particles respectively.

7.3.3 Effects by Chemical-specific or Source-oriented Analysis and by Other Size Cuts

Besides examining the relative impacts of PM2.5 and CP, several studies have examined the effects of chemical-specific constituents, including sulfates and a wide range of elements, especially metals. For example, in a study in Santa Clara County, Fairley (1999) examined the impacts of nitrates, sulfates, and COH (coefficient of haze). The latter is highly correlated with elemental carbon, and is likely to be a good marker of pollution from motor vehicles (especially diesel exhaust) and of wood smoke. All three of these constituents of PM2.5 were associated with all-cause mortality, while nitrates were also associated with cardiovascular mortality. These findings were consistent with those in the Netherlands, where associations were reported for sulfates, nitrates, and black smoke (Hoek et al., 2000). In a study in Buffalo, Gwynn et al. (2000) reported effects on total mortality for COH, sulfates and hydrogen ion, a measure of aerosol acidity. Lippmann et al. (2000) did not find associations of mortality with sulfate or hydrogen ion in Detroit, although only limited data for these pollutants were available. In their study of the eight largest Canadian cities, Burnett et al. (2000) examined the impact of 47 separate elements within PM2.5 and CP. Among the constituents in the fine fraction, sulfates, zinc, nickel and iron were all associated with mortality, as was COH. These elements are associated with a wide range of sources, including, among those relevant to California, oil combustion, road dust, tire wear, and incinerators (Burnett et al., 2000).

Several studies also examined source-oriented combinations of pollutants. For example, Ozkaynak and Thurston (1987), used 1980 U.S. vital statistics data in a cross-sectional analysis of air pollution and mortality. Applying fine particle source apportionment techniques, particles from industrial sources (e.g., iron and steel emissions) and from coal combustion were more significant contributors to mortality than were soil-derived particles. Laden et al., (2000) examined PM2.5 data from the Harvard Six-Cities study, and characterized the pollutants into three different factors: motor vehicle emissions, coal combustion, and soil and crustal material. Generally, both the motor vehicle and coal factors were associated with mortality, with the strongest effect from the former. The crustal material in PM2.5 was not associated with mortality. In a study with a limited number of days in three New Jersey cities, Tsai et al. (2000) examined the effects of source-type components on mortality. Using factor analysis, this study reported associations of sulfates and motor vehicle tracers with both all-cause and cardiopulmonary mortality. Ozkaynak et al. (1996a) also reported associations between pollutants linked with motor vehicles and total, cardiovascular and respiratory mortality.

Finally, Wichmann et al. (2000) examined the effects of PM2.5 mass as well as ultrafine particles (0.01 to 0.1 μm) for the small German city of Erfurt. The number rather than the mass of ultrafine particles was used as the exposure measure. For this study, three different size classes of ultrafines were measured, including 0.01 to 0.03 μm, 0.03 to 0.05 μm, and 0.05 to 0.1 μm. The authors reported that both PM2.5 mass and several measures of ultrafines were associated with daily mortality.

Several studies have indicated a potential role for high concentrations of acidic sulfates in excess human mortality, particularly in London in the 1950s and 1960s (Thurston et al., 1989; Ito et al., 1993). More recent studies of cities in North America with lower acidic sulfate levels have been inconsistent (Dockery et al., 1992; Burnett et al., 2000; Lippmann et al., 2000, Gwynn et al., 2000). For instance, Dockery et al. (1992) found that PM10 concentrations showed a stronger relationship with daily mortality in St. Louis than did ambient sulfate levels. As noted above, Lippmann et al. (2000) did not find an association of sulfates with mortality in Detroit. In contrast, Gwynn et al. (2000), in a time-series analysis in Buffalo, NY, found stronger relationships between both acid particles and sulfates and respiratory mortality than

that observed for PM10. However, she and her colleagues found no relationship between sulfates and circulatory (cardiovascular) mortality. Burnett et al. (2000) found associations between sulfates and mortality in eight Canadian cities. Thus, strong acid sulfates may play a role in the observed PM-mortality associations, particularly in urban areas with elevated levels of these sulfate classes. However, it should be noted that in California, strong acidic sulfates (particularly sulfuric acid) constitute but a small fraction of PM mass (Chapter VI).

7.3.4 Mortality Displacement

Additional support for pollution-related mortality occurring outside of the hospital and for the likelihood of significant shortening of life is provided by recent studies reporting associations between ambient PM and increased heart rate, decreased heart rate variability, and the incidence of arrhythmias (Liao et al., 1999; Pope et al., 1999b, c; Peters et al., 2000a; Gold et al., 2000; see section 7.7). These outcomes are considered reliable predictors of the risk of death from heart disease (See, e.g., Tsuji et al., 1996; Nolan et al., 2000). Direct evidence for a nontrivial reduction in life expectancy is provided by studies that statistically control for the phenomenon of mortality displacement; i.e., in which the time of death might be delayed by only a few days. If all pollution-related deaths were associated with such mortality displacement, the total life shortening would likely be very small. However, both Schwartz (2000c) and Zeger et al. (1999) have shown, using both frequency- and time-domain methods, that most air pollution-associated mortality is not due to such displacement. For cardiovascular deaths, mortality displacement does not appear to be a major factor, as the average life-shortening appears to be greater than two to three months. In contrast, deaths from chronic obstructive pulmonary disease (COPD, which consists mainly of emphysema and chronic bronchitis) appeared to be more consistent with a mortality displacement hypothesis (Schwartz, 2001a, 2000c).

The possibility of significant loss in life expectancy is suggested by studies indicating that death occurring outside of a hospital had larger (two- to four-fold) and stronger associations with PM than did deaths occurring inside hospitals (Schwartz 2001a, 2000c). This suggests that some of the impacts of PM occur among a subgroup that is not under intensive medical care, and may not be at the end-stage of their disease. However, it is possible that some out-of-hospital deaths may have occurred among the large contingent of uninsured people in the U.S., who perhaps should have been under medical care.

Finally, evidence of a significant loss in life-years from air pollution is provided by studies of infants and children (see section 7.7.3). Several recent studies suggest that exposure to PM may result in neonatal or infant mortality (for example, Woodruff et al., 1997; Ostro et al., 1999a; Bobak and Leon, 1998). These studies indicate that infants and children, possibly those with pre-existing respiratory illness, may represent an additional subgroup especially sensitive to effects of exposure to ambient PM pollution.

7.3.5 Analysis of Thresholds

For short-term exposure to PM, two general methods are available to address the issue of the existence of a threshold, or an ambient PM level below which there would be no risk of a significant adverse health outcome. First, it can be examined indirectly, by considering data sets with very low mean ambient concentrations. Second, it can be examined directly by developing statistical tests that carefully model the shape of the concentration-response function. Both of these approaches appear to indicate the lack of an observable population threshold. Several studies have been conducted in cities with low ambient concentrations of PM10, including Morgan et al. (1998) for Sydney, Australia (mean = 18 $\mu g/m^3$, based on conversion from co-located nephelometry data), Wordley et al. (1997) for Birmingham, UK (mean = 26 $\mu g/m^3$), Schwartz et al. (1996) for the Harvard Six-Cities (mean = 25 $\mu g/m^3$),

Burnett et al. (2000) for the eight largest Canadian cities (mean =26 μg/m^3), and Gwynn et al. (2000) for Buffalo and Rochester (mean = 24 μg/m^3). In addition, several cities in the data set used by Samet et al. (2000a) have mean concentrations in the low 20s. Examination of these data indicates that the concentration-response functions are not driven by the high concentrations and that the slopes of these functions do not appear to increase significantly at higher concentrations.

Among the statistical approaches, Schwartz et al. (2000a) examined the concentration-response relationship in 10 U.S. cities, restricting the data to days on which the PM10 concentration was less than 50 μg/m^3. The resulting risk estimates were statistically significant and greater than for that for the entire data set. Two other papers first addressed the issue of whether existing statistical techniques could identify a threshold, assuming one existed. Cakmak et al. (1999) simulated data with varying amounts of exposure measurement error, based on actual data from Toronto. They examined whether statistical models used in most air pollution epidemiology (including locally weighted smoothing techniques in Poisson regression models) would be able to detect thresholds in the PM-mortality association. They concluded that, if a threshold existed, it is highly likely that the existing statistical modeling would detect it. Many mortality papers have, in fact, examined the shape of the concentration-response function and indicated that a linear (non-threshold) model fit the data well (Pope, 2000).

A different statistical approach was used by Schwartz and Zanobetti (2000) in their analysis of 10 U.S. cities. The authors combined concentration-response curves across the cities, after demonstrating that this approach produced unbiased estimates. Predicted values of the response function were estimated at 2 μg/m^3 intervals. Results from this approach did not provide any evidence for a threshold effect. Finally, Daniels et al. (2000) used an alternative approach to test for the existence of a threshold using the 20 largest cities in the U.S. The authors considered three different log-linear regression models. One used a simple linear term for PM10, which could then be used as a basis for comparison with the other models. A second model used a cubic spline model that would allow for nonlinearity in PM10 that could represent a threshold function. The third model presumed a threshold, in which a grid search was used to test for a concentration that would support a threshold. The results indicated that for the second model, which can allow for a threshold if the underlying data suggest one, a linear specification provided the best fit to the data. Analysis using the grid search model suggested that no threshold was apparent for either total mortality or cardiopulmonary mortality. Finally, using a goodness-of-fit test (Akaike's information criterion) to compare the simple linear nonthreshold model with models that would allow for a threshold concentration, the authors reported that there was no evidence to prefer the threshold models to the linear model.

Schwartz et al. (1996) examined the relationship of PM2.5 concentrations and daily mortality in the Harvard Six Cities dataset. When they restricted the analysis to days on which the PM2.5 24-hour average concentrations equalled or exceeded 30 or 25 μg/m^3, Schwartz et al. (1996) reported that the strong association persisted, suggesting that, if there is a threshold of effect, it cannot be found at concentrations in excess of 25 μg/m^3. On the other hand, Smith et al. (2000) statistically examined the threshold issue in data on mortality and ambient PM2.5 from Phoenix, AZ. They reported evidence of a significant change in the regression slope at a concentration of around 20 to 25 μg/m^3 PM2.5, suggesting the possibility of a threshold in this range. However, to our knowledge, this is a (nearly) unique publication reporting such a finding. Staff from OEHHA and the Bay Area Air Quality Management District (BAAQMD) analyzed data from the two published California studies involving 24-hour measurements of PM2.5 and daily mortality counts (in Coachella Valley [Ostro et al., 2000] and Santa Clara

County [Fairley, 1999]). The modeling techniques used for the exposure-response functions included piecewise linear regression (e.g., utilizing several "hockey-stick" models), locally weighted smoothing in generalized additive models, trimming analysis (selectively deleting days with high PM2.5 values), and Bayesian models (comparing the likelihoods of various thresholds) to explore the evidence for a nonlinear exposure-response at low PM2.5 concentrations. In general, staff found that a linear, nonthreshold model within the concentration range of interest for PM2.5 provided an adequate fit to the data, while threshold (or other nonlinear) models provided no better fit. Except for the report of Smith et al. (2000), it appears that the relationship between daily mortality and PM2.5 can be well characterized by a nonthreshold model, consistent with the findings reported by others for PM10 (see above).

7.3.6 Summary

Staff concludes the following from the above results:

- The associations observed between daily changes in PM10 and mortality appear to be independent of the effect of weather factors, seasonality, time, and day of week – all of which are typically controlled for in the analyses. The studies include a wide range of environments, pollution-temperature conditions, population age distributions, background health conditions, socioeconomic status, and health care systems. The range of the association is approximately 0.5% to 1.6% increase in mortality per 10 $\mu g/m^3$ increment of PM10. However, when longer exposure averaging times are examined, using distributed lags of several days or cumulative exposures of up to several months, the estimated effects may be approximately 2% per 10 $\mu g/m^3$. Although the relative risk per unit is low, the large number of people exposed suggests the existence of a potentially major impact on public health.
- The effects of PM cannot be explained by exposure to other pollutants. As might be expected, examining several correlated pollutants in the same model often increases the variation of and attenuates the estimated PM effect. However, the estimated PM impact is generally consistent regardless of the concentration of, or degree of co-variation with, other pollutants, giving strong support to an independent effect of PM.
- The elderly, those with chronic heart or lung disease, and infants appear to be at significantly greater risk of PM-associated mortality. Study results suggest that some, and perhaps a large fraction of, mortality associated with acute exposure is not the result of just a few days of life shortening. Rather, for cardiovascular mortality, there is evidence that significant reductions in life expectancy may be involved. In addition, if the associations between PM and infant mortality represent causal relationships, large reductions of life expectancy could result, as well.
- The effects associated with short-term exposure to PM appear to occur at current ambient concentrations, including cities or counties where the long-term mean PM10 concentration is around 25 to 35 $\mu g/m^3$ (Figure 7.1). As suggested by Figure 7.2, greater uncertainty is apparent with decreasing concentrations, particularly those below about 25 $\mu g/m^3$.
- No threshold of response has been observed in the PM-mortality studies. Several direct and indirect approaches have consistently found that non-threshold, linear models provide the best fit to the data.
- Premature mortality appears to be associated not only with PM10, but also with both fine and coarse particles, as well as sulfates (a subset of PM2.5). The effects per unit mass appear to be greater for PM2.5 than CP; this may be due to intrinsically greater toxicity of PM2.5 versus CP, but may also be attributable to differential measurement error in

monitoring for CP than for PM2.5, or greater indoor infiltration rates of PM2.5 versus CP (and therefore greater overall exposure to PM2.5), or to some combination of these three. In addition, there is preliminary evidence that pollutants from mobile sources, oil burning, steel industry emissions, and coal combustion are associated with mortality. Crustal materials, particularly those entrained on windy days, have been reported by several investigators to be less strongly associated with premature mortality.

7.4 Chronic Exposure – Mortality

7.4.1 Study Design and Methods

Several air pollution studies examine the effects of long-term exposure to PM using a prospective cohort design. In this type of study, a sample of individuals are selected and followed over time. For example, Dockery et al. (1993) published results for a 15-year prospective study based on approximately 8,000 individuals in six cities in the eastern United States. Pope et al. (1995) published results of a 7-year prospective study of the mortality experience of approximately 550,000 individuals in 151 cities in the United States using a cohort participating in a long-term investigation sponsored by the American Cancer Society (ACS). These studies used individual-level data so that other factors that affect mortality can be characterized and adjusted for in the analysis. Specifically, these studies were able to control for mortality risks associated with differences in body mass, occupational exposures, smoking (current and past), alcohol use, age, and gender. Once the effects of individual-level factors were determined, the models examined whether longer-term city-wide averages in PM (measured as PM10, PM2.5 or sulfates) were associated with different risks of mortality and life expectancies. Several different cause-specific categories of mortality were examined, including lung cancer, cardiopulmonary, and all other causes. These studies incorporate much, but not all, of the impact associated with short-term exposures (Kunzli et al., 2001). An effect that would tend not to be included in the long-term studies is mortality displacement of a very short-term nature, such as a few days. These effects would not alter the differences in overall life expectancy predicted by the longer-term studies.

Statistical analysis used proportional hazards regression modeling with time since enrollment as the underlying time variable. The study samples were stratified by combinations of age (5-year groups), gender and race. Additional analyses were undertaken after stratifying the samples by smoking habit and gender. The greatest uncertainties in these studies involve the disease-relevant times, durations, and intensities of exposure. Both studies assigned city-wide, multi-year averages that occurred when the study participants were young to middle-aged adults (between ages 20 and 50, approximately). Thus, early childhood exposure was not estimated and no within-city differences in exposure were incorporated into the analysis. These errors in exposure assessment would tend to make it more difficult to detect an effect of pollution and would bias the analysis towards the null hypothesis of no effect. Therefore, it is unlikely that bias or misclassification of exposure could explain the results.

7.4.2 Summary

Both the ACS and Harvard Six-Cities studies report robust and statistically significant associations between several years of exposure to PM and various measures of mortality. Smoking was the dominant factor in explaining mortality patterns, overall and for each of the cause-of-death categories. Regarding air pollution effects, Dockery et al. (1993) reported associations between total mortality and PM10, PM2.5, and sulfates. An association with CP is also apparent (U.S. EPA, 1996). Smaller associations were found with total suspended particles, sulfur dioxide, nitrogen dioxide, and aerosol acidity, but no association was found with ozone. Using a model that included smoking and other non-pollution explanatory variables, all-cause mortality and cardiopulmonary deaths (but not “all other causes”) were

both related to sulfates and PM2.5. In additional analyses, PM2.5 was associated with cardiopulmonary mortality but not with "all other" mortality. In this study, PM2.5 concentrations ranged from 11 to 29.6 $\mu g/m^3$ (with a mean of 18 $\mu g/m^3$) and PM10 ranged from 18 to 46.5 $\mu g/m^3$ (with a mean of 30 $\mu g/m^3$). It should be noted that these pollutants were measured only for part of the follow-up time for this cohort: while the mortality experience in the Six Cities covered the years 1974 – 1991, PM2.5 and PM10 were measured from 1979 through 1985, while sulfates were measured from 1979 through 1984. During these pollutant measurement periods, the concentrations of PM2.5 and sulfates remained relatively stable; nevertheless, the effects of exposures prior to the study could not be evaluated with this data set.

In the study using the ACS cohort, Pope et al. (1995) reported associations between fine particles and sulfates with both all-cause mortality and cardiopulmonary mortality. Across the 50 cities with PM2.5 data, PM2.5 ranged from 9 to 33.5 $\mu g/m^3$, with a median of 18 and a mean of 20 $\mu g/m^3$. For the 151 cities with sulfate data, sulfates ranged from 3.5 to 23.5 $\mu g/m^3$ with a mean of 11 $\mu g/m^3$. Exposure data collection was not concurrent with the mortality incidence data: annual arithmetic mean sulfate data were obtained for the year 1980, while for PM2.5 the investigators used the city-specific medians of data collected from 1979 to 1983. Mortality among the cohort, meanwhile, was assessed from September 1982 through 1989. The relative risk estimates for this study were smaller than those reported from the Six-Cities study but the confidence intervals around the relative risk estimates overlapped enough that the results were statistically indistinguishable. The estimated mortality effects of approximately 4 to 7% per 10 $\mu g/m^3$ of long-term exposure to PM10 are much larger than those effects associated with daily exposure (approximately 1% per 10 $\mu g/m^3$). These studies also provide a basis for calculating reductions in life expectancy associated with PM exposure. The results suggest that the 24 $\mu g/m^3$ difference in PM2.5 between the cleanest and dirtiest cities is associated with an almost 1.5-year difference in life expectancy (Pope, 2000). Brunekreef (1997) used a life-table for men in the Netherlands and estimated a difference of 1.1 years in life expectancy between the two extreme cities in the ACS study. In addition, the difference in life expectancy of a person who actually died from diseases associated with air pollution was estimated to be about 10 years. This is because air pollution-related deaths only make a small fraction of the total deaths in a given city. Subsequent analysis by Pope and colleagues (reported in Krewski et al., 2000) demonstrated an association between mortality and PM, when PM2.5 was used as the metric of exposure. No association was found, however, when either PM15 or the coarse particle fraction measured as PM15 – PM2.5 was used.

Krewski et al. (2000) completed an independent validation and reanalysis of both the Six-Cities and the ACS cohort studies. The first task of this study was to recreate the data sets and validate the original results. Krewski et al. (2000) reported few errors in the coding and data merging in the original studies and basically replicated the results of both studies. The second task was to conduct an exhaustive sensitivity analysis of the original studies to determine whether the results were robust. Specifically, the authors examined the effects of: (1) alternative statistical models; (2) potential individual-level interactions and confounders such as physical activity, education, body mass, smoking status, marital status, alcohol consumption and occupational exposure; (3) potential city-wide confounders such as population growth, income, weather, number of hospital beds and water hardness; (4) consideration of various subgroups; (5) non-linear specifications in the dose-response function that would allow for the possibility of a threshold; (5) co-pollutants, including ozone, sulfur dioxide and nitrogen dioxide; (6) alternative PM exposure estimates, including different years and particle sizes; (7) underlying variation from city to city; (8) spatial correlation between cities; and (9) time-dependent variables such as air pollution exposure and individual risk factors that change over time. In general, the re-analysis confirmed the original results of

associations between mortality and long-term exposure to PM. However, in some cases, the adjustment for spatial correlation led to an attenuation of the effect of PM2.5 or sulfates. Also, inclusion of SO_2, which was likely to be highly correlated with PM2.5 and sulfates, into the model reduced the estimated effect of both PM2.5 and sulfates. Among the more important new findings were: (1) education (possibly serving as a marker for socioeconomic status, health care or lifestyle factors) appears to be a significant effect modifier (see section 7.7.2 below); (2) PM2.5 was more strongly associated with mortality than was either PM10 or CP; (3) the results were not confounded by either individual-level or city-wide (ecological) covariates; (4) the associations between sulfate and PM2.5 and both all-cause and cardiopulmonary mortality were near linear within the relevant ranges, with no apparent threshold; (5) the PM effects were not confounded by and were independent of effects of other pollutants, (6) the effects were robust with respect to alternative functional forms, alternative air pollution data, and detailed spatial analysis; and (7) the results of the original investigators were confirmed.

Chronic exposure to PM was also examined using a smaller and younger nonsmoking cohort participating in the Seventh Day Adventist Health Study (Abbey et al., 1999). For the years prior to 1987, PM10 data were unavailable and were estimated from TSP concentrations. In this study, neither mean PM10 nor sulfate concentrations were associated with mortality. However, using the particle exposure metric of the number of days when PM10 levels were above 100 $\mu g/m^3$, an interquartile range of 43 such days was associated with both all-cause and nonmalignant respiratory mortality in males, but not females. In a follow-up study using a subset of the cohort living near airports, estimates of PM2.5 were developed from data on airport visibility (McDonnell et al., 2000). PM10 was again estimated from season- and city-specific regressions using TSP data. Positive but nonstatistically significant associations were found between all three measures of PM (PM10, CP and PM2.5) and both all-cause and respiratory mortality in males. Although the mean of the estimated value of PM10 was relatively high in these studies (i.e., 51 $\mu g/m^3$ in Abbey et al,, 1999 and 59 $\mu g/m^3$ in McDonnell et al,, 2000), most of the measures of PM10 were estimated from either TSP or from airport visibility. This process added errors in the measurement of exposure which would likely lead to a lowered effect estimate.

Finally, preliminary results of a study of long-term exposure among a cohort of 50,000 men assembled by the U.S. Veterans Administration (VA) from 32 clinics in the mid 1970s has been reported by Lipfert et al. (2000b). The study cohort included a larger proportion of African-Americans (35%) than the U.S. population as a whole, and a large percentage of current or former smokers (81%). The cohort was selected at the time of recruitment based on their having mild to moderate hypertension and on their receiving care at VA facilities. Many individual-level risk factors were not ascertained, so socioeconomic variables were assembled at the zip-code, census tract and county levels. County-wide pollutant levels based on county of residence at the time of entry into the study were obtained for TSP, PM10, PM2.5, PM15, PM15-2.5, SO_4, O_3, CO, and NO_2. PM2.5 data were available for only 26,000 men. Four different exposure periods were examined (1974 and earlier, 1975-81, 1982-88, 1989-96) with three sequential mortality follow-up periods (1975-81, 1982-88, 1989-96). In addition, analysis was conducted using the entire follow-up period, not disaggregated into separate periods. The final proportional hazards regression model involved 233 terms, including age, systolic and diastolic blood pressures, body mass index, height, race, age and race interaction terms, present or former smoking, average zip-code education, and poverty, and a clinic-specific variable. The most consistently positive effects were found for ozone and nitrogen dioxide exposures in the years immediately preceding death. When the PM analyses used segmented (shorter) time periods, the results were highly variable, including significantly negative mortality coefficients for some PM metrics. However, when methods

consistent with previous long-term exposure studies were used (i.e., the entire follow-up period of mortality as a function of average PM concentrations over several years), results for sulfates, PM2.5 and PM15 were similar to those reported in previous studies.

7.5 Daily Exposure – Morbidity

Over the last decade, several hundred epidemiological studies have reported associations between alternative measures of PM and a range of morbidity outcomes. The PM measures have included PM10, black smoke (BS), COH, sulfates , more recently, PM2.5 and CP. The health outcomes associated with PM include, but are not limited to, hospitalization for cardiovascular or respiratory disease, emergency room and urgent care visits, asthma exacerbation, acute and chronic bronchitis, restrictions in activity, work loss, school absenteeism, respiratory symptoms, and decrements in lung function. Typically, these studies have involved either of two analytic methods. First, many of the outcomes use a methodology similar to that described above for mortality related to short-term exposure -- time-series analysis of daily count data. Specifically, daily counts of an endpoint such as hospitalization for cardiovascular disease are examined in response to single- and multi-day average concentrations of PM. As in the case of mortality, these models also control for potential confounders, such as season, meteorology, day of week, and time trends. A second approach involves the use of panel data, in which a cohort of subjects (e.g., asthmatic children) is followed prospectively over a period of several months or years while daily health outcomes and pollution measures are recorded and then compared. In the following subsections, we briefly review some of the important health outcomes, with particular attention given to studies undertaken in California. The review is not meant to be exhaustive, but rather to illustrate the range and consistency of morbidity effects associated with PM10 or its components.

7.5.1 Cardiovascular Hospital Admissions

Associations between daily concentrations of PM10 and daily hospital admissions for cardiovascular disease have been reported for close to a hundred cities in the U.S, Canada and Europe (Table 7.3). As is the case for the mortality studies related to short-term exposure, there are several multi-city efforts (Schwartz et al., 1999; Samet et al., 2000a; Zanobetti et al., 2000a). For example, Schwartz et al. (1999) examined daily hospital admissions for cardiovascular disease (ICD9 codes 390 – 429) from 1988 to 1990 among persons above age 65 for eight metropolitan areas, including Chicago, Colorado Springs, Minneapolis, New Haven, St. Paul, Seattle, Spokane, and Tacoma. For five of the cities and for the effect estimate pooled across all eight cities, statistically significant associations were reported with PM10. Across the cities, a 10 $\mu g/m^3$ change in PM10 was associated with about a 1% change in hospitalization for cardiovascular disease. The median PM10 concentration in these cities ranged from 23 to 37 $\mu g/m^3$.

Samet et al. (2000a) examined data on hospitalizations for cardiovascular disease among people 65 and older in 14 U.S. cities from 1985 to 1994. The cities were located throughout the U.S., though none was in California. Again, a statistically significant association was reported across the cities with a pooled effect estimate of 1.1% per 10 $\mu g/m^3$. The estimate increased to 1.5% per 10 $\mu g/m^3$ when a two-day average of PM10 was used and PM10 was restricted to concentrations less than 50 $\mu g/m^3$. For these cities, the long-term mean PM10 ranged from 24 to 45 $\mu g/m^3$, with a group mean of 33 $\mu g/m^3$. Zanobetti et al. (2000a) essentially confirmed the Samet et al. (2000a) results and also demonstrated that other pollutants such as carbon monoxide, ozone and sulfur dioxide were not confounding or modifying the estimated effects of PM10. Burnett et al. (1997a) also reported an association between PM, measured as COH, and congestive heart failure (ICD9 = 427) for those ages 65

and above living in Canada's 10 largest cities, from 1981 to 1994. The effect size was similar to that reported for PM10 in the U.S. studies. Similar results have been reported between PM and either total cardiovascular disease or subsets thereof (e.g., heart failure or ischemic heart disease) in a disparate range of cities including, but not limited to: Detroit (Lippman et al., 2000), Tucson (Schwartz, 1997), Toronto (Burnett et al., 1997b), London (Atkinson et al., 1999), Edinburgh (Prescott et al., 1998), Sydney (Morgan et al., 1998), Chicago (Morris and Naumova, 1998) and Hong Kong (Wong et al., 1999). In addition, Stieb et al. (2000) reported associations between emergency department visits for angina or myocardial infarction and both PM10 and PM2.5.

Among California cities, associations have been reported between PM10 and hospitalization for total cardiovascular disease, myocardial infarction, congestive heart failure and cardiac arrhythmia among individuals above age 30 in Los Angeles (Linn et al., 2000). Daily gravimetric measures of PM10 were estimated from TEOMs and averaged 37 $\mu g/m^3$ in the winters to 54 $\mu g/m^3$ during autumn. In another study of Los Angeles' hospitals, Moolgavkar (2000b) reported associations between PM10 and total cardiovascular admissions among people ages 20 to 64, and among those 65 and above. Mean PM10 was 44 $\mu g/m^3$. The effect magnitudes of PM10 estimated for Los Angeles were generally similar to those reported for other studies in the U.S. -- a 0.6 to 2% increase in cardiovascular hospitalizations per 10 $\mu g/m^3$ of PM10.

Only a few cardiovascular admissions studies have measured PM2.5 and CP concentrations. However, among those that measured different particle sizes, Lippmann et al. (2000) reported associations of hospitalizations for heart failure and ischemic heart disease with both PM2.5 and CP in Detroit. Likewise, Burnett et al. (1997b) found associations between hospitalizations for total cardiovascular conditions, heart failure, dysrhythmias, and ischemic heart disease and both PM2.5 and CP in Toronto. Finally, in the Moolgavkar (2000b) study in Los Angeles, an association was reported between PM2.5 and cardiovascular hospital admissions for the 20 to 64 age group, and for the group aged 65 and above. Estimates for CP were not provided. Gwynn et al. (2000) found little evidence of a relationship between PM10 or sulfates and circulatory (cardiovascular) hospital admissions.

In summary, studies over the past several years consistently report associations between PM10 and hospitalization for total cardiovascular disease and several of its specific components, such as congestive heart failure and ischemic heart disease. These effects have been mostly reported among people above age 65, a group that dominates the prevalence of cardiovascular diseases. For many of these studies, the long-term mean PM10 ranged from 25 $\mu g/m^3$ to 40 $\mu g/m^3$, although studies of cities with reported means below and above this range exist, as well. Most of the studies carefully controlled for the potential confounding of weather, season, time, and co-pollutants. Overall, PM10 is consistently associated with these clinically significant cardiovascular endpoints, with a general effect estimate of between 0.6 to 2% per 10 $\mu g/m^3$. These relatively low risk estimates, however, are shared over a large segment of the population regularly exposed to PM who has pre-existing cardiovascular disease. Based on the few studies that have measured both fine and coarse particles, associations are apparent between hospital admissions for cardiovascular diseases and both of these exposure measures. In these studies, mean PM2.5 ranged from 17 $\mu g/m^3$ to 22 $\mu g/m^3$. Finally, as indicated in section 7.3., associations between daily or multi-day exposure to PM10 and cardiovascular-related mortality have also been reported. In addition, section 7.8 includes a summary and discussion of several of the other cardiovascular outcomes associated with PM such as changes in heart rate, heart rate variability, arrhythmia, heart attacks, and blood viscosity. The coherence of the mortality and morbidity results provides compelling evidence of an effect of PM.

Table 7.3. Summary of Cities Included in Studies of Short-term Exposure & Hospital Admissions for PM10, PM2.5 (FP) and Coarse Particles (CP)

City/Region	Time Period	Reference	Age Group	Endpoint* and % Increase (95% CI) per 10μg/m^3	Particle Type	Mean** in μg/m^3 (BOLD=Median)
London	1992-94	Atkinson et al., 1999	all ages	CV: 0.64(0.18, 1.10)	PM10	29
Toronto metro area	1992-94	Burnett et al., 1997b	all ages	CV: 5.40(2.20, 8.80) Resp: 5.00(2.08, 8.00)	CP	12
Toronto metro area	1992-94	Burnett et al., 1997b	all ages	CV: 2.36(0.72, 4.08) Resp: 3.40(1.36, 5.52)	FP	17
Toronto metro area	1992-94	Burnett et al., 1997b	all ages	Resp: 2.12(0.90, 3.42)	PM10	28
Montreal	Summers 1992-93	Delfino et al., 1997a	under 65	Resp-ED: 9.56(1.96, 17.12)	FP	12
Montreal	Summers 1992-93	Delfino et al., 1997a	under 65	Resp-ED: 7.32(2.00, 12.64)	PM10	22
Buffalo	May 1988-Oct 1990	Gwynn et al., 2000	all ages	CV: 1.14(-0.66, 3.10) Resp: 2.20(0.80, 3.60)	PM10	24
London	1992-94	Hajat et al., 2001	0-14	Resp-Allergic Rhinitis Dr Visits:5.67(2.21, 9.45)	PM10	29
London	1992-94	Hajat et al., 2001	15-64	Resp-Allergic Rhinitis Dr Visits:6.85(4.59, 8.66)	PM10	29
Los Angeles, CA	1992-95	Linn et al., 2000	30 & older	CV: 0.65(0.41, 0.89) Resp: 0.66(0.34, 1.00)	PM10	46
Detroit, MI	1992-94	Lippmann et al., 2000	65 & older	CV-Dys: 0.08(-4.88, 5.76); HF: 2.08(-1.32, 5.80); IHD: 4.20(1.08, 7.56); Stroke: 1.96(-1.88, 6.20) Resp-COPD: 3.72(-1.76, 10.00); Pneu:4.80(0.32, 9.60)	CP	12
Detroit, MI	1992-94	Lippmann et al., 2000	65 & older	CV-Dys: 1.28(-2.60, 5.60); HF: 3.64(0.96, 6.48); IHD: 1.72(-0.56, 4.16); Stroke: 0.72(-2.12, 3.76) Resp-COPD: 2.20(-1.88, 6.80); Pneu: 5.20(1.48, 8.80)	FP	18

City/Region	Time Period	Reference	Age Group	Endpoint* and % Increase (95% CI) per 10μg/m^3	Particle Type	Mean** in μg/m^3 (BOLD=Median)
Detroit, MI	1992-94	Lippmann et al., 2000	65 & older	CV-Dys: 0.58(-1.63, 2.74); HF: 1.94(0.04, 4.02); IHD: 1.78(0.10, 3.60); Stroke: 0.96(-1.10, 3.24) Resp-COPD: 1.92(-1.02, 5.40); Pneu: 4.40(1.66, 7.20)	PM10	31
Santa Clara Co, CA	Winters 1988-92	Lipsett et al., 1997	all ages	Resp-Asthma ED Visits: 6.94(3.20, 11.30)	PM10	61
Paris	1991-95	Medina et al., 1997	all ages	Resp-Asthma House Visits: 2.54(0.82, 4.38)	PM13	25
Cook Co, IL	1987-95	Moolgavkar, 2000b	65 & older	CV: 0.84(0.60, 1.10) Resp-COPD:0.40(-0.04, 0.86)	PM10	**35**
Los Angeles Co, CA	1987-95	Moolgavkar, 2000c	0-19	Resp-COPD: 6.84(3.56, 10.32)	CP	NA
Los Angeles Co, CA	1987-95	Moolgavkar, 2000c	20-64	Resp-COPD: 3.60(1.20, 6.12)	CP	NA
Los Angeles Co, CA	1987-95	Moolgavkar, 2000c	65 & older	Resp-COPD: 2.04(-0.16, 4.36)	CP	NA
Los Angeles Co, CA	1987-95	Moolgavkar, 2000c	0-19	Resp-COPD: 1.72(-0.04, 3.56)	FP	**22**
Los Angeles Co, CA	1987-95	Moolgavkar, 2000b	20-64	CV: 1.40(0.72, 2.12) Resp-COPD: 2.24(0.76, 3.76)	FP	**22**
Los Angeles Co, CA	1987-95	Moolgavkar, 2000b, c	65 & older	CV: 1.72(1.00, 2.44) Resp-COPD: 2.04(0.36, 3.76)	FP	**22**
Los Angeles Co, CA	1987-95	Moolgavkar, 2000c	0-19	Resp-COPD: 2.14(0.88, 3.46)	PM10	**44**
Los Angeles Co, CA	1987-95	Moolgavkar, 2000b, c	20-64	CV: 0.88(0.44, 1.34) Resp-COPD: 1.30(0.34, 2.30)	PM10	**44**
Los Angeles Co, CA	1987-95	Moolgavkar, 2000b, c	65 & older	CV: 0.64(0.24, 1.06) Resp-COPD: 1.22(0.22, 2.26)	PM10	**44**
Maricopa Co, AZ	1987-95	Moolgavkar, 2000b	65 & older	CV: -0.48(-1.38, 0.46)	PM10	**44**
Minneapolis/St. Paul, MN	1986-91	Moolgavkar et al., 1997	65 & older	Resp-COPD+Pneu: 1.74(0.92, 2.60)	PM10	34
Birmingham, AL	1986-91	Moolgavkar et al., 1997	65 & older	Resp-COPD+Pneu: 0.30(-0.30, 0.92)	PM10	43

City/Region	Time Period	Reference	Age Group	Endpoint* and % Increase (95% CI) per 10μg/m^3	Particle Type	Mean** in μg/m^3 (BOLD=Median)
King County, WA	1987-95	Moolgavkar et al., 2000	all ages	Resp-COPD: 2.56(0.36, 4.84)	FP	18
King County, WA	1987-95	Moolgavkar et al., 2000	all ages	Resp-COPD: 1.02(0.00, 2.08)	PM10	30
Sydney	1990-94	Morgan et al., 1998	all ages	CV: 1.56(0.44, 2.72)	FP (bscat)	10
Chicago, IL	1986-89	Morris & Naumova, 1998	over 65	CV: 0.78(0.20, 1.38)	PM10	41
Los Angeles, CA	Wet seasons 1991-94	Nauenberg & Basu, 1999	all ages	Resp-Asthma: 3.24(0.40, 6.00)	PM10	45
Spokane, WA	1995-97	Norris et al., 2000	under 65	Resp-Asthma ED Visits: 0.48(-2.18, 3.52)	PM10	28
Seattle, WA	1995-96	Norris et al., 2000	under 18	Resp-Asthma ED Visits: 11.24(2.08, 24.22)	PM10	22
Santiago	1992-93	Ostro et al., 1999b	under 2	Resp-Lower Resp Clinic Visits: 0.50(0.04, 0.96)	PM10	109
Santiago	1992-93	Ostro et al., 1999b	2-14	Resp-Lower Resp Clinic Visits: 0.74(0.16, 1.34)	PM10	109
Edinburgh	1992-95	Prescott et al., 1998	under 65	CV: 0.40(-2.50, 3.80)	PM10	21
Edinburgh	1992-95	Prescott et al., 1998	65 & older	CV: 2.48(0.92, 4.18)	PM10	21
14 cities, US	1985-94, range varies by city	Samet et al., 2000a	65 & older	CV: 1.10(0.94, 1.24) Resp-COPD: 1.50(1.06, 1.96); Pneu: 1.34(1.06, 1.64)	PM10	24-45
14 cities, US	1985-94, range varies	Samet et al., 2000a	65 & older	CV: 1.52(1.20, 1.82)	PM10 < 50μg/m^3	24-45
Minneapolis/ St. Paul, MN	1986-89	Schwartz, 1994c	65 & older	Resp-Pneu: 0.12(0.10, 0.13); COPD: 0.16(0.12, 0.21)	PM10	36
Tacoma, WA	1988-90	Schwartz, 1995	65 & older	Resp: 2.00(0.64, 3.45)	PM10	37
Cleveland, OH	1988-90	Schwartz, 1996	65 & older	Resp: 1.16(0.10, 2.28)	PM10	43
Tuscon, AZ	1988-90	Schwartz, 1997	over 65	CV: 1.21(0.22, 2.19)	PM10	42
8 metro counties, US	1988-90	Schwartz et al., 1999	65 & older	CV: 1.00(0.74, 1.28)	PM10	**23-37**
Seattle, WA	1987-94	Shepard et al., 1999	under 65	Resp-Asthma: 4.44(1.12, 8.04)	CP	16
Seattle, WA	1987-94	Shepard et al., 1999	under 65	Resp-Asthma: 3.48(1.32, 5.72)	FP	17
Seattle, WA	1987-94	Shepard et al., 1999	under 65	Resp-Asthma: 2.74(1.10, 4.52)	PM10	32

City/Region	Time Period	Reference	Age Group	Endpoint* and % Increase (95% CI) per 10μg/m^3	Particle Type	Mean** in μg/m^3 (BOLD=Median)
Saint John	1992-96	Stieb et al., 2000	all ages	CV: 6.04(-0.12, 13.12)	FP	9
Saint John	1992-96	Stieb et al., 2000	all ages	CV: 6.50(2.04, 11.86)	PM10	14
Atlanta, GA	Summers 1992-94	Tolbert et al., 2000	under 17	Resp-Asthma ED Visits: 2.64(0.24, 5.34)	PM10	39
Hong Kong	1994-95	Wong et al., 1999	all ages	CV: 0.60(0.16, 1.08)	PM10	**45**

*Endpoint Abbreviations:
COPD=Chronic Obstructive Pulmonary Disease
CV=Cardiovascular
Dys=Dysrhythmia
ED=Emergency Department
HF=Heart Failure
IHD=Ischemic Heart Disease
Pneu=Pneumonia
Resp=Respiratory

**Average of 24-hour measurements over time period.

7.5.2 Respiratory Hospital Admissions (RHA)

Many studies have also used time-series analysis to examine associations between daily PM and hospitalization for respiratory diseases (Table 7.3). Such endpoints have included total respiratory admissions (ICD9 = 460-519) for all age groups, as well as for those greater than age 65, and admissions for chronic obstructive pulmonary disease (COPD), pneumonia and asthma. For example, the recent NMMAPS multi-city study (Samet et al, 2000a) examined associations between PM10 and several specific respiratory diseases among a group of individuals aged 65 and above. Associations were reported between PM10 and both COPD and pneumonia. Among the 14 cities in the analysis, the long-term mean PM10 concentration ranged from 24 to 45 μg/m^3. The NMMAPS results suggest a range of 1.5 to 3% increase in the risk of RHA per 10 μg/m^3 of PM10.

Similar findings of an association of PM10 and hospital admissions for total respiratory diseases or its components such as COPD, asthma or pneumonia have been reported for many other cities throughout the U.S., including Minneapolis (Schwartz, 1994c; Moolgavkar et al., 1997), Tacoma (Schwartz, 1995), Cleveland (Schwartz, 1996), Buffalo (Gwynn et al., 2000), Chicago (Zanobetti et al., 2000a), Detroit (Lippmann et al., 2000) and Seattle (Sheppard et al., 1999).

Three separate studies have reported similar associations using data from Los Angeles (Linn et al., 2000; Moolgavkar, 2000b; Nauenberg and Basu, 1999). The Linn et al. (2000) study used pulmonary hospital admissions data from 1992 to 1995 and found positive associations with PM10 (mean = 45 μg/m^3) throughout the year, but especially in the winter. Moolgavkar (2000c) used data on COPD for 1987 through 1995, and reported associations with PM10 (median = 44μg/m^3), and both PM2.5 (median = 22 μg/m^3) and CP for three different age

groups: 0 to 19, 20 to 64, and 65 and above. Finally, Nauenberg and Basu (1999) used data on hospital admissions for asthma from 1991 through 1994. Associations were reported with PM10 (mean = 45 $\mu g/m^3$) in the "wet season" (Jan 1 to March 1) but not the "dry season". The wet season effect was also stronger among MediCal claimants, suggesting an effect modification by income. Gwynn and Thurston (2001) also reported stronger effects from PM10 and other pollutants on respiratory hospital admissions among those without insurance or on Medicaid versus those with private insurance or Medicare.

Besides Moolgavkar (2000b), a few other studies have reported findings using PM2.5 and CP. For example, Lippman et al. (2000) found an association between pneumonia admissions for those aged 65 and above and both PM2.5 and CP in Detroit. For COPD, an association was also reported with PM2.5, and less so with CP. Likewise, Burnett et al. (1997b) found associations between hospital admissions for respiratory diseases and both PM2.5 and CP in Toronto. Sheppard et al. (1999) also found associations between both PM2.5 and CP and asthma hospital admissions. Finally, Moolgavkar et al. (2000) found an association between PM2.5 and hospital admissions for COPD in King County (Seattle). No results were reported for CP.

Associations have also been reported between PM10 and emergency department and urgent care visits, which may or may not result in hospital admissions. For example, in a study conducted in Santa Clara County, California, Lipsett et al. (1997) reported associations between PM10 (mean = 61 $\mu g/m^3$) and emergency room visits for asthma during the winters, particularly on colder days. Using limited data (only one year), Delfino et al. (1997a) found associations between respiratory emergency department visits and PM10, PM2.5, sulfates and hydrogen ion in Montreal. Norris et al. (2000) analyzed emergency room visits for asthma in Spokane and Seattle, using two years of data on patients of all ages in Spokane, and 16 months of data for asthma cases below the age of 18 in Seattle. Besides PM10, a stagnation index was created, which reflected days with relatively low windspeed. Factor analysis indicated that days characterized by greater stagnation were likely to involve higher concentrations of products of incomplete combustion (including fine particulate elemental carbon) and sulfates. In Spokane, associations were found between emergency asthma visits and the stagnation index, but not with PM10. However, for Seattle both of these metrics were associated with pediatric emergency room visits.

Several other studies have also reported effects among children. For example, Tolbert et al. (2000) examined the effects of air pollution on roughly 6,000 pediatric emergency room visits for asthma during the summers of 1993-1995 in Atlanta. Several different statistical models were used to explore the sensitivity of the results to the model selection. PM10 concentrations (mean = 39 $\mu g/m^3$) were highly correlated with 1-hour maximum ozone (r = 0.75). Associations between daily visits and PM10 were reported, with consistent results across all of the models. Medina et al. (1997) analyzed doctors' house calls for asthma in Paris, France for the years 1991 to 1995. Black smoke (BS) was used as a measure of particulate matter. House calls for asthma were divided into three age groups: all ages, 0 to 14 years, and 15 to 64 years. Associations were reported for the full age group (0 to 64 years), but especially for children below age 14. The effect estimate for children, based on a 4-day moving average of BS was 8 times higher than that of the older population. Hajat et al. (1999) reported a similar association in London, England between PM10 and doctor visits for asthma for children below age 14. While the effect size was not as greatas in the Medina et al. (1997) study, the strongest effect was found from a multi-day average of exposure to PM10. In examining allergic rhinitis, Hajat et al. (2001) reported stronger associations for adults than for children. The associations were also stronger for multi-day averages of PM10. Ostro et al. (1999c) analyzed associations between PM and daily visits to primary health care clinics in Santiago, Chile among children under age 2, and ages 2 to 14. This area is characterized by very high

levels of ambient PM10, especially during the winter, when inversions are common. For this study, several public clinics around the city were organized to undertake a specific study of urgent care visits for lower and upper respiratory symptoms. Associations were found between PM10 and visits for lower respiratory symptoms for both age groups.

Several studies suggest relationships between strong acid sulfates and respiratory hospital admissions. In a time-series study in Buffalo, NY, Gwynn et al. (2000) reported stronger associations between both H+ aerosol and sulfates and respiratory hospital admissions than those observed for either PM10 or COH. Burnett et al. (1994), in an analysis of urgent daily admissions at 168 acute care hospitals in Ontario, Canada, found significant associations of sulfates (lagged 0 to 3 days) with several respiratory diseases, but not with nonrespiratory conditions. These associations were not significant during the winter, when the sulfate levels tend to be lower. However, during the summer months, sulfates were strongly correlated with both PM2.5 and with H+ ($r>0.8$), so it is difficult to ascribe a "causal" role to any one of these PM constituents.

In summary, studies over the past several years consistently report associations between PM10 and several different measures of hospitalization or urgent care for respiratory diseases. The outcomes include hospitalization for total respiratory diseases, as well as for specific diagnoses, including COPD, asthma and pneumonia. In addition, associations have been reported between PM10 and the need for urgent care including emergency department visits, doctor visits, and public clinic visits. These effects have been reported primarily among elderly individuals, but effects have been also reported among all age groups, including children under age 18, and children under age 2. For many of these studies, the long-term mean PM10 ranged from 25 $\mu g/m^3$ to 40 $\mu g/m^3$, although studies of cities with reported means below and above this range exist as well. Most of the studies carefully control for potential confounding by weather, season, time, and co-pollutants. Overall, PM10 consistently was associated with these clinically significant respiratory endpoints, with a general effect estimate of between 1.25 and 5% increase in risk per 10 $\mu g/m^3$. Based on the few studies that have measured both fine and coarse particles, associations have been reported between hospital admissions for respiratory diseases and both of these exposure measures. In these latter studies, long-term mean PM2.5 ranged from 17 $\mu g/m^3$ to 22 $\mu g/m^3$. Finally, as indicated in section 7.3, associations between daily or multi-day exposure to PM10 and respiratory-related mortality have also been reported.

7.5.3 Asthma Exacerbation

Asthma affects more than 15 million Americans, including almost 5 million children, making it the most common childhood illness in the U.S. Asthma prevalence increased 75% from 1980 to 1994 in the United States (Mannino et al., 1998). In a recent analysis of data from the National Health Interview Survey, the prevalence of asthma among children aged 5 – 14 was about 67% higher than for adults aged 35 and above (74.4/1000 vs. 44.6/1000, respectively; Mannino et al., 1998). Asthma surveillance data developed by the U.S. Centers for Disease Control and Prevention (CDC) and recent reports on asthma hospitalization by the California Department of Health Services (CDHS, 2000), and King County, Washington, indicate that children, especially young children, may experience severe exacerbations at a greater rate than older children or adults (Mannino et al., 1998; CDHS, 2000). Hospitalization rates for children 0 to 4 years are greater than for all others (49.7/10,000/year for ages 0 – 4 versus a range of 18.0 to 25.5/10,000/year for all other age groups) and is four-fold higher among black children versus white children (CDHS, 2000). While hospitalization rate data are influenced by a number of factors, including access to health care, these data support the notion that asthma may generally affect young children more than adults.

In the last few years, many studies have been published on the effects of PM exposure on symptoms and lung function changes in asthmatics (Table 7.4). These studies typically follow a panel of subjects who record daily health outcomes over several months. Several outcomes have been measured, including specific symptoms (e.g., cough, shortness of breath, wheeze, chest tightness, phlegm), medication use, and lung function changes [e.g., peak expiratory flow rate (PEF), forced expiratory volume (FEV), and forced vital capacity (FVC)]. Concurrent air pollution is recorded along with potential confounders that also change on a daily basis and might be associated with the health outcome such as weather factors, environmental tobacco smoke (ETS) or wood smoke exposure, activity patterns, time spent outdoors, use of air conditioning, and day of week. Generally, the study of air pollution and asthma is analytically challenging since the disease and its triggers are complex. Several of the studies combine individuals with different levels of asthma severity and medication use, or combine asthmatics and non-asthmatics. Nevertheless, evidence for a fairly consistent (but not universal) effect of PM has emerged over the last several years, including several studies conducted in California.

For example, Ostro et al. (2001) examined the effect of PM10 and PM2.5 on 138 African-American children with current, physician-diagnosed asthma living in Los Angeles from August through October, 1993. Daily reporting of cough, shortness of breath and wheeze, and asthma episodes (i.e., the start of several consecutive days with symptoms) were associated with PM10 (24-hour mean = 52 $\mu g/m^3$) and PM2.5 (12-hour mean = 41 $\mu g/m^3$), but not with ozone. The PM10 effects were slightly stronger than those from PM2.5, with a 10 $\mu g/m^3$ change in PM10 associated with an approximate change in onset of symptom rates of from 5 to 15%. In addition, an association was reported between PM10 and the use of extra asthma medication. These findings supported an earlier study of 83 African-American children with asthma in Los Angeles that indicated an association between PM10 and shortness of breath (Ostro et al., 1995).

Delfino et al. (1997b, 1998) examined panels of asthmatics living in California. In a summer study, 22 asthmatics, ages 9 to 46, from the semi-rural town of Alpine were followed (Delfino et al., 1997b). Symptoms were not related to PM10 (24-hour mean = 26 $\mu g/m^3$) or any of the other pollutants or bioaerosols measured. However, there was an association between PM10 and inhaler use. Delfino et al. (1998) followed a panel of 24 asthmatics, ages 9 to 17 from August to October, 1995 in Alpine. “Bothersome” asthma symptoms (either cough, wheeze, sputum production, shortness of breath, or chest tightness) were associated with both PM10 (24-hour mean = 31 $\mu g/m^3$) and ozone, with a greater relative effect from PM10. The largest effects of PM10 were on those children not currently on anti-inflammatory medication.

In studies outside of California, Yu et al. (2000) followed 133 asthmatics, ages 5 to 13, living in Seattle. A strong association was reported between asthma symptoms and ambient particles monitored using nephelometry, which measures primarily PM1.0, or particles below one micron in diameter. Vedal et al. (1998) examined 75 physician-diagnosed asthmatic children, ages 6 to 13, living in Port Alberni, British Columbia. Several other groups of non-asthmatics were analyzed as well. For the entire group (n = 206), PM10 (median = 22 $\mu g/m^3$) was associated with increases in both cough and phlegm (7% increase in each per 10 $\mu g/m^3$ PM10), and decreased PEF. Stratified analysis indicated effects only among asthmatic children. No consistent effects were found in the other groups of children. Thurston et al (1997) examined children with asthma at a summer camp in Connecticut. Associations were reported between both sulfates and ozone (which were highly correlated) and asthma

Table 7.4. Summary of Cities Included in Studies of Short-term Exposure and Respiratory Morbidity for PM10, PM2.5 (FP) and Coarse Particles (CP)

City/ Region	Time Period	Reference	Age Group, Other Demographics	Particle Type	General Results	Mean* ($\mu g/m^3$)
Rural and Urban areas, Holland	Winters 1992/93-94/95	Boezen et al., 1999	7-11, N=632	PM10	Association with lower respiratory symptoms among children with both bronchial hyperresponsiveness and high total serum IgE.	Urban: 55, 42, and 31. Rural : 45, 44, 27
Alpine (rural southern CA)	Aug-Oct 1995	Delfino et al., 1998	9-17, N=24, asthmatics	PM10	Association with asthma symptoms, especially children less frequently symptomatic for asthma on anti-inflammatory medication	31
Alpine (rural southern CA)	Summer 1994	Delfino et al., 1997b	9-46, N=22, asthmatics	PM10	Association with inhaler use	26
Amsterdam, Holland	Early Summer 1995	Gielen et al., 1997	7-13, N=61, majority asthmatics	PM10	Association with acute respiratory symptoms	31
Leiden University Medical Center, Holland	Summer 1995	Hiltermann et al., 1998	18-55, N=60	PM10	Association with shortness of breath and bronchodilator use	40
Reanalysis of several studies including Utah Valley, UT; Bennekom, Holland; Uniontown, PA; State College, PA	varies by study	Hoek et al., 1998	children	PM10	Significant decreases in PEF	varies by study
Los Angeles, CA	Summer 1992	Ostro et al., 1995	7-12, N=83, african-american, asthmatics	PM10	Association with shortness of breath, particularly moderate and severe asthmatics.	56
Los Angeles, CA	Aug-Oct 1993	Ostro et al., 2001	8-13, N=138, african-american asthmatics	PM10, FP	Association with new episodes of cough and extra asthma medication.	PM10=52, FP=41

City/ Region	Time Period	Reference	Age Group, Other Demographics	Particle Type	General Results	Mean* ($\mu g/m^3$)
Ausburg, Germany	Oct 1994-June 1995; severe episode Jan 7-19, 1985	Peters et al., 1997	25-64, N=3256	TSP	Association with increased plasma viscosity in both men and women when comparing severe pollution episode to the remainder of study.	TSP=47; severe episode TSP=98
Utah Valley, UT	Winter 1990/91	Pope and Dockery, 1992	10-12, N=79, split between those asymptomatic for asthma and those symptomatic for asthma but not on medications	PM10	Particularly symptomatic children, associations with respiratory symptoms and significant association with small decreases in PEF.	76
Mexico City	Apr 1991-Feb 1992; 2 months	Romieu et al., 1996	5-13, N=71, mild asthmatics	PM10	Association with increased lower respiratory illness and decreased PEF.	167
New Haven, CT and Tacoma, WA	1988-1990	Schwartz et al., 1994	65 and older, all hospital admissions	PM10	Association with respiratory hospital admissions	41-New Haven; 37-Tacoma
Reanalysis of Harvard Six City Study, Uniontown and State College, PA	varies by study	Schwartz and Neas, 2000	children grades 2-5,	FP and CP	Association with lower respiratory symptoms; stronger effect with FP. Association with decreased PEF for FP.	varies by study
Kuopio, Finland	Spring 1995; six weeks	Tiittanen et al., 1999	8-13, N=49, children with chronic respiratory disease	PM10, FP, CP	Association with morning PEF and cough; strongest association for FP and CP.	PM10=28, FP=15

City/ Region	Time Period	Reference	Age Group, Other Demographics	Particle Type	General Results	Mean* ($\mu g/m^3$)
urban and nonurban areas, Holland	Winters 1992/93-94/95	van der Zee et al., 1999	7-11, N=795	PM10	Significant association with decreases in PEF and lower respiratory symptoms in symptomatic children.	ranged 24-53
Port Alberni, British Columbia, Canada	May 1990-Mar 1992	Vedal et al., 1998	6-13, N=206 including 75 asthmatics	PM10	Associations with cough and phlegm and decreased PEF, particularly among asthmatics.	22 (median)
Seattle, WA	Nov 1993-Aug 1995; 28-112 days	Yu et al., 2000	5-13, N=133, mild/moderate asthmatics	PM10, PM1.0 (nephel-ometry)	Association with asthma symptoms; strong association for PM1.0	PM10=25, PM1.0=10
Vinton, VA	Summer 1995	Zhang et al., 2000	adult, N=673, mothers	PM10, FP, CP	Association with new episodes of rhinitis for CP.	NA

*Average of 24-hour measurements over time period.

symptoms, PEF and bronchodilator use. Data on PM10 were not available. Pope and Dockery (1992) studied two different cohorts of fifth- and sixth-grade students in Utah Valley. One group had symptoms of asthma or had been diagnosed by a physician as having asthma, but was not currently on medication. The other group had no history or symptoms of asthma. Associations were found for both groups between PM10 and both PEF and respiratory symptoms. The symptomatic group demonstrated a greater effect from exposure to PM10.

Several studies on asthma have also been completed outside of the U.S. and Canada. For example, Gielen et al. (1997) reported associations between PM10 and both asthma symptoms and PEF among children in Amsterdam. Hilterman et al. (1998) reported associations between PM10 and symptoms, but not PEF, in asthmatic adults living in Leiden, the Netherlands, while Peters et al. (1997) reported associations between various measures of PM and both symptoms and PEF among adults in Erfurt, Germany. Finally, Romieu et al. (1996) also reported associations between PM10 and asthma exacerbation among a panel of children living in Mexico City.

Overall, the effects of PM on asthma exacerbation are not as consistent as those found with hospitalizations for cardiovascular or respiratory disease. This is likely due to the complexity and multi-dimensional aspects of the disease itself, and the subsequent difficulties in estimating the impact of air pollution. Nevertheless, several well-conducted prospective cohort studies, often involving over 100 children with asthma, have found associations between PM10 and a range of asthma symptoms or medication use. Most of the studies have

controlled for potentially confounding factors such as weather and other pollutants, such as ozone. Given the findings reported above, of an association between PM10 and hospitalizations and urgent care for asthma, it is reasonable to expect an impact on less severe asthma outcomes as well.

7.5.4 Respiratory Symptoms and Other Adverse Outcomes

Panel studies and other analytical study designs have also been used to examine the effect of air pollution on the general population (including both asthmatic and non-asthmatic individuals) (summarized in Table 7.4). A wide range of outcomes has been studied including upper and lower respiratory symptoms (in aggregate form and separated out by specific symptoms), lung function changes, restrictions in activity due to respiratory illness, school absenteeism and work loss. Although these effects are clearly not as significant as mortality and hospitalization, they may have an important effect on public health since they impact a greater proportion of the population. Some of these studies are summarized below to provide a sense of the range of impacts associated with exposure to PM.

In a study in three cities in Southern California (Azusa, Glendora and Covina), Ostro et al. (1993) examined the daily effects of air pollution on 321 nonsmoking adults. Associations were reported between both sulfates and ozone on lower respiratory symptoms. Schwartz and Neas (2000) reanalyzed three different panel studies to examine the relative impact of PM2.5 and CP on respiratory symptoms and peak flow in young children. First, daily respiratory data from 1,844 children in second through fifth grade from six eastern cities (the Harvard Six-Cities) were used. The second and third data sets involved daily data collected from June through August from fourth and fifth grade children living in Uniontown and State College, PA. In both of these studies, twice daily PEF measures were recorded. The analysis of the Six City data suggested that, using single pollutant models, lower respiratory symptoms (any day with at least two of the following: cough, phlegm, chest pain or wheeze) were associated with both PM2.5 and CP, as well as sulfates. The stronger effects were observed for PM2.5 and sulfates. When considering only cough as the outcome, associations were again found with all of the measures of PM, but the strongest effect was with CP. In the analysis of PEF in the two other cities, an association was found with PM2.5 and sulfates but not with CP. Zhang et al. (2000) examined respiratory symptoms among 673 mothers living in Vinton, VA during the summer of 1995. Of all the pollutants considered, only CP were associated with a new episode of rhinitis.

Tiittanen et al. (1999) examined the association between PM and PEF and cough among 49 children with chronic respiratory symptoms living in Kuopio, Finland. Several different measures of PM were available, including PM10, PM2.5, CP, black carbon, resuspended road dust, and ultrafines. Associations were reported between morning PEF and all of the measures of PM. In addition, incidence of cough was also associated with all of the PM measures. For cough, however, the strongest association was with a 4-day cumulative average of both PM2.5 and ultrafines. Since the PM measures were highly correlated, it is difficult to attribute the effect to any single constituent. Schwartz et al. (1994) examined the respiratory symptoms of 300 elementary school children from April to August in each of six eastern cities. Several different endpoints were considered, including lower respiratory symptoms (reports of at least two among cough, chest pain, phlegm, and wheeze), upper respiratory symptoms (reports of at least two among hoarseness, sore throat, and fever), and cough alone. An association was reported between both PM10 and PM2.5 and lower respiratory symptoms, cough, and to a lesser extent, upper respiratory symptoms.

Two studies in the Netherlands examined the impact of wintertime PM10 on symptoms in two panels of children. Boezen et al. (1999) studied a panel of children ages 7 to 11 to determine if those with bronchial hyperresponsiveness (BHR) and high serum concentrations of IgE

were more responsive to air pollution. Based on data from three winters, an association was found between PM10 and lower respiratory symptoms among children with BHR and high total IgE. No associations between PM10 and respiratory symptoms were found among children who did not have both of these factors. The wintertime PM10 averages for the three years were 55, 42 and 31 $\mu g/m^3$. In a related study, van der Zee et al. (1999) examined PEF and respiratory symptoms among children in urban and rural areas with and without asthma, chronic cough, or wheeze (classified as symptomatic). In both the urban and rural areas, associations were found between PM10 and both lower respiratory symptoms and decrements in PEF among the symptomatic children. However, stronger effects were observed in the urban areas. Among the non-symptomatic children, no association between PM10 and symptoms was found. In the urban area, PM10 averaged 48, 37 and 29 $\mu g/m^3$ during the three winters that were studied, versus 35, 35 and 24 $\mu g/m^3$ in the rural area.

Regarding changes in lung function, Hoek et al. (1998) reanalyzed data on PEF from four other studies conducted in Utah, the Netherlands, and Uniontown and State College, PA. This paper focused on explaining significant decrements in PEF, defined as a daily change greater than 10% below a person's mean. This change was found to be associated with changes in PM10.

Besides respiratory symptoms and changes in lung function, other less severe symptoms have been reported for the general population. For example, Ostro (1987) and Ostro and Rothschild (1989) used data from six years of the annual Health Interview Survey conducted by the National Center for Health Statistics. Based on a two-week recall period, the endpoint used in these studies was restricted activity days, which includes days spent in bed, days missed from work, or days when activities were partially restricted due to illness. In Ostro (1987), which included 49 metropolitan areas, an association was reported between PM2.5, estimated from airport visibility and restricted activity in adults. Ostro and Rothschild (1989) reported an association between PM2.5 and both respiratory-related restrictions in activity and minor restrictions (days where activity was restricted but not resulting in work loss) in adults. These studies imply about a 10 to 15% change in reduced activity per 10 $\mu g/m^3$ of PM10. Finally, Ransom and Pope (1992) examined PM10 and weekly absenteeism in an elementary school in Utah. An association was reported with PM10, with about a 4% increase in absenteeism per 10 $\mu g/m^3$.

7.6 Chronic Exposure – Morbidity

Data from the past quarter century suggest that long-term PM exposures are associated with chronic respiratory symptoms or disease, and possibly with decreased lung function. Much of this evidence derives from cross-sectional analyses, which compare symptom or disease prevalence, or lung function, during a given time period (e.g., one year) among communities with different average pollution levels (e.g., Ferris et al., 1973, Hodgkin et al., 1984; Mullahy and Portney, 1990). Cross-sectional studies, however, while suggestive of potentially meaningful associations, are generally not considered good evidence of causal relationships because inter-city differences may be due to unmeasured factors other than air pollution. Also, chronic health effects are thought to occur as a result of long-term or repeated exposures, but cross-sectional investigations generally present a snapshot in time and are not informative regarding the critical exposure averaging time (e.g., 1 year, 10 years, or even the number of times a given level is exceeded during a specified period). Moreover, in cross-sectional studies people who may have died from exposure-related illness are not included in the analysis. This "survivor bias" tends to underestimate effects of exposures (assuming that such effects exist). Nevertheless, several large cross-sectional investigations in the U.S. and Europe, in which individual-level data on a variety of other relevant factors have been collected (e.g., smoking status, exposure to environmental tobacco smoke, occupational

exposures), provide reasonably consistent evidence for effects of long-term exposure to PM on chronic respiratory health outcomes.

Several large-scale cohort studies provide prospective evidence related to long-term effects of PM exposure. These studies have collected information on individual participants, and therefore can statistically control for most of the potentially relevant confounding variables, including cigarette smoking, exposure to environmental tobacco smoke, occupational exposures (for adults), weight, alcohol consumption, and so forth. The most important of the relevant cross-sectional and cohort studies are summarized in the following paragraphs. Most have been conducted in the United States, and several have been undertaken (at least in part) in California. One large cohort study undertaken in four cities in the Los Angeles basin (the Chronic Obstructive Respiratory Disease or CORD study) is not included in the discussion, because inter-city differences in participants' lung function were not presented by pollutant (e.g., Tashkin et al., 1994; Detels et al., 1991).

7.6.1 The Adventist Health Study

In 1977, a cohort of 6,338 nonsmoking non-Hispanic white Seventh Day Adventists, aged 25 years and older and residing principally in three large metropolitan areas of California (San Francisco, San Diego, and the South Coast Air Basin), were enrolled in a long-term study of the effects of air pollution on respiratory health (AHSMOG). Approximately 10% of the study population lived in other areas of California. One criterion for enrollment was residential stability: all participants had to have lived within 5 miles of their 1977 address for 10 years or longer. Participants completed questionnaires in 1977, 1987, and 1992 regarding residential and work location histories, past smoking, exposure to ETS, occupational exposures, presence of various respiratory symptoms, and physician diagnoses of respiratory disease. Cumulative air pollution exposure was assessed by interpolation of fixed-site monitoring data in relation to the subjects' residences and worksites during the study period. Numerous reports describing the morbidity and mortality of this cohort have been published: earlier reports focused on total suspended particulates (TSP) as the PM metric (e.g., Abbey et al., 1993) and will therefore not be discussed. Several of the more recent articles are described below, while the mortality results are described in section 7.3.

Abbey and colleagues (1995a, b) analyzed the incidence of chronic respiratory disease in relation to several particle metrics for the 10-year period 1977 through 1987 for a subset of 3,914 study participants. PM10 concentrations were estimated using site- and season-specific regressions on TSP data during this period. They reported that long-term exposures to estimated PM10 concentrations exceeding 80 or 100 $\mu g/m^3$ for at least 250 hours/year produced statistically significant increases in risk of newly reported symptoms of overall airway obstructive disease (AOD, consisting of a triad of asthma, chronic bronchitis and emphysema) and of chronic bronchitis alone, but not asthma (Abbey et al., 1995a). Although point estimates of risk associated with lower concentrations of estimated PM10 were all greater than one, none was statistically significant. For a subset of the cohort living near airports (n=1,868), PM2.5 concentrations were also estimated using visibility data (Abbey et al., 1995b). In this group, PM2.5, PM10 and sulfates were all significantly related to worsening severity of AOD (relative risks of 2.20, 2.64, and 3.04, respectively) or asthma alone (relative risks of 2.05, 2.82, and 2.75, respectively), while sulfates and PM10, but not fine particles, were both associated with significantly increased risks of AOD, and PM10 with chronic bronchitis. All of the long-term studies in this document involve exposure measurement error, which generally would tend to impede researchers' ability to detect any relationship between air pollution and health. In these reports this situation is exaggerated because neither PM2.5 nor PM10 were directly measured, suggesting that these results, though perhaps reliable qualitatively, should not be considered quantitatively accurate.

Beeson et al. (1998) examined associations between several air pollutants and lung cancer incidence (n=36 incident cases, 16 in men and 20 in women) from 1977-1992), adjusting for several covariates (attained age, pack-years of past cigarette smoking, years of education, and consumption of alcohol at baseline), though a variety of other variables were also examined as potential confounders. The estimated annual mean concentration of PM10 from 1973-1992 was 51 $\mu g/m^3$ (SD=16.52). As in prior reports on this cohort, PM10 concentrations from 1977-87 were estimated from TSP measurements, while after 1987 PM10 was measured directly. Incident lung cancer in men was significantly associated with the average annual mean concentration of PM10 (RR = 5.21, 95% C.I.=1.94-13.99, for an interquartile range or IQR of 24 $\mu g/m^3$), with somewhat lower estimates for ozone and SO_2. For women, lung cancer incidence was associated with PM10 (including the annual mean concentration and several exceedance frequencies), but these relationships were not statistically significant. In multi-pollutant models, the coefficients for PM10 and SO_2, but not ozone, remained stable. Although these RR estimates for men were stable in a variety of sensitivity analyses, they are substantially higher than those observed in other investigations, and may be due to a lower baseline lung cancer rate in the nonsmoking Seventh Day Adventist source population. However, the relatively small number of cases on which these are based suggests a need for cautious interpretation.

In 1993, 1,391 of the study participants who had completed all three questionnaires and met several other criteria successfully completed lung function testing. For this analysis, mean PM10 levels averaged over monthly values from 1973-1993 were 54.1 $\mu g/m^3$ for male subjects (range 20.0 – 80.6) and 52.7 $\mu g/m^3$ (range 21.3 – 80.6) for female subjects. An inter-quartile difference of 54 days/yr in excess of 100 $\mu g/m^3$ PM10 was associated with significant decreases in FEV_1 of –7.2% (95% C.I = -11.5 - -2.7) in men whose parents had a history of obstructive lung disease or hay fever, and of –1.5% (95% C.I. –2.7 - -0.4) FEV1/FVC in male never-smokers. No such effects were seen in women or in other strata of men. These results should be viewed with caution because: (1) these results are essentially cross-sectional and represent only about 1/5 of the original AHSMOG cohort members; who may differ from those who did not participate in this part of the study in ways that may affect estimation of the PM-lung function relationship; and (2) about 2/3 of the PM10 data were estimated from TSP.

7.6.2 The Six-Cities and 24-Cities Studies

In the mid-1970s a cohort of white first- and second-grade school children (n = 10,106) in six cities in the eastern U.S. were enrolled in a study to examine both cross-sectional and longitudinal relationships between air pollution and respiratory disease and lung function growth. The mean annual TSP concentrations ranged from 39.3 (Portage, WI) to 114.1 (Steubenville, OH) $\mu g/m^3$, while the corresponding range for sulfates was 5.4 to 18.8 $\mu g/m^3$. Exploring the relationships between pollutant levels in the year preceding the second annual health examination of the children, Ware and colleagues (1986) reported significant relationships between both average PM concentrations (measured as TSP) and sulfates (i.e., the sulfate fraction of TSP) and cough frequency, bronchitis and a composite index of lower respiratory illness. For a 10 $\mu g/m^3$ increase in sulfates, the odds ratios for these three health outcomes were 1.60, 1.68, and 1.57, respectively. Sulfate levels in the 6 cities ranged from 4.4 to 19.3 $\mu g/m^3$. These air pollution – health outcome relationships were observed when the analysis focused on inter-city pollutant differences, but were not supported by analyses within each city over time. No relationship was observed between any of the air pollution metrics and lung function, even when the analysis was restricted to lifetime residents of the six cities.

In a subsequent analysis involving several highly correlated PM metrics (TSP, PM15, PM2.5, and sulfates, measured during 1980-81), all were found to be related to chronic cough, bronchitis, and chest illness reported on health questionnaires (Dockery et al., 1989).

Comparing the least and most polluted cities for PM15 (Portage, Wisconsin, and Steubenville, Ohio, respectively), the annual mean concentrations were 20.1 $\mu g/m^3$ and 58.8 $\mu g/m^3$. For PM2.5 the range was 11.8 – 36.7 $\mu g/m^3$, represented by Topeka, Kansas and Steubenville, respectively. Across the range of PM15, the odds ratios for these three health outcomes for all children were 3.7 (95% C.I. = 1.0 – 13.5) for chronic cough, 2.5 (95% CI = 1.1 - 6.1) for bronchitis, and 2.3 (95% CI = 0.8 – 6.7) for chest illness. For sulfates and PM2.5, the odds ratios for these outcomes were approximately doubled; however, unlike the results for chronic cough and bronchitis in relation to PM15, these effect estimates were not statistically significant. There was no association between any pollutant and asthma or persistent wheeze. However, when the analysis was stratified by the presence of asthma or persistent wheezing, the fine particle-related odds ratios for bronchitis and chest illness among those with these conditions were about 60% higher than for the group as a whole, but nevertheless were still not significant. Among the asthmatic and wheezy children, odds ratios for these symptoms in relation to PM15 were at least as high as those for the fine particle metrics, and also were significant for chest illness, and remained so for the nonasthmatic children for the other symptoms. While these results suggest that the combined coarse and fine fractions (measured as PM15) were likely more influential than PM2.5 or sulfates alone in relation to chronic respiratory symptom reporting, the estimates were not statistically distinguishable (i.e., there was substantial overlap between the confidence intervals around the odds ratios for each metric). Finally, as in the earlier report on this cohort, there was no relationship between any PM metric and lung function.

Subsequently, the same group of investigators evaluated the relationships of several air pollutants, including PM10, PM2.1, fine particle sulfate and strong acidity, to respiratory symptoms and lung function in 13,369 white children, aged 8 to 12, in 24 suburban communities throughout the U.S. and Canada (Dockery et al., 1996; Raizenne et al., 1996). Three of the 24 communities were located in California (Livermore, Monterey, and Simi Valley). Particle measurements in each city took place every other day over a one-year period, based on the assumption that this would serve as a reasonably representative surrogate for longer-term exposures; nevertheless this study is essentially cross-sectional in design. Mean PM concentrations over all 24 cities in this study were as follows: PM10 = 23.8 $\mu g/m^3$ (SD=5, range 15.4 – 32.7), PM2.1 = 14.5 $\mu g/m^3$ (SD = 4.2, range 5.8 – 20.7), and sulfates = 4.7 $\mu g/m^3$ (SD = 2.2, range 0.7 – 7.4). Neither PM10 nor PM2.1, *per se*, was associated with any chronic respiratory symptoms. Comparing cities with the highest and lowest annual concentrations, sulfates were associated with at least one episode of bronchitis (OR = 1.65, 95% C.I. = 1.12-2.42) and with any bronchitic symptom (OR = 1.27, 95% C.I. = 1.01-1.61); fine particle strong acidity (which includes sulfates) was linked with bronchitis (OR = 1.66, 95% C.I. = 1.11-2.48). There were no obvious susceptible subgroups within this study population.

Acceptable lung function data were obtained from a subset of 10,251 children in 22 of the 24 communities. All measures of particles were reported to be associated with small, but statistically significant, decrements in several measures of lung function across the ranges of each pollutant. The greatest point estimates of effect were observed for particle strong acidity. For instance, a change in particle strong acidity of 52 $nmol/m^3$ was associated with the following percentage decrements: forced vital capacity (FVC) = -3.45 (95% C.I. = -4.87 - -2.01), forced expiratory volume in one second (FEV_1) = -3.11 (95% C.I. = -4.62 - -1.58), and peak expiratory flow rate (PEFR) = -3.71 (95% C.I. = -7.10 - -0.20). Still, the estimated lung function differences associated with the range of strong particle acidity could not be statistically distinguished from those related to the other particle metrics. More importantly, because of the cross-sectional nature of this investigation, it is not possible to postulate a causal relationship between any particle metric and long-term decrements in the growth and

development of children's respiratory function. This would require a prospective design, such as that employed in the Children's Health Study.

7.6.3 Children's Health Study

Children may be at greater risk from long-term exposures to particles or other air pollutants because the growth and development of the respiratory system may be permanently affected by early environmental insults. Funded by the California Air Resources Board, the Children's Health Study was designed as a 10-year investigation of the impacts of southern California air pollution on lung growth and development and other indices of respiratory health among 3,676 fourth-, seventh-, and tenth-graders in 12 communities, which were chosen to emphasize different long-term air pollution conditions. For data collected in 1986-90, prior to the health data collection efforts, the 24-hr average PM concentration ranged from 28.0 $\mu g/m^3$ in Atascadero and Santa Maria to 84.9 $\mu g/m^3$ in Mira Loma and Riverside. In 1994, the mean 24-hr average PM10 concentration across the 12 communities was 34.8 $\mu g/m^3$ (range = 13.0 $\mu g/m^3$ in Lompoc to 70.7 $\mu g/m^3$ in Mira Loma) (McConnell et al., 1999; Peters et al., 1999a). Although the full 10 years of follow-up data have not been analyzed yet, the initial cross-sectional analysis and some longitudinal results have been published. At enrollment, neither PM10 nor PM2.5 were associated with respiratory illness among the total cohort (ever or current asthma, bronchitis, cough, or wheeze) assessed by questionnaire (Peters et al., 1999a). In contrast, among children with asthma, respiratory symptoms increased with increasing particle levels (McConnell et al., 1999). Specifically, there was about a 40% increase in the odds of bronchitis among asthmatics per 19 $\mu g/m^3$ change in PM10 measured over 2-week intervals (OR=1.4, 95% C.I. = 1.1-1.8). Exposure to a 15 $\mu g/m^3$ increment in fine particles resulted in about the same magnitude of increase in risk, which was not statistically significant. Both measures of PM were also associated with at least a doubling of risk of phlegm in asthmatic children. Acid vapors and NO_2 were also associated with respiratory symptoms in asthmatic children. However, because all four (PM10, PM2.5, NO_2, and acid vapor) were highly correlated, it is not possible to definitively attribute these effects to any single pollutant (McConnell et al., 1999).

In another cross-sectional analysis of the Children's Health Study, both PM10 and PM2.5, as well as NO_2, were significantly associated with decreased lung function (forced vital capacity [FVC], forced expiratory volume in one second [FEV_1], and maximal mid-expiratory flow [MMEF]), especially in girls who spent more time outdoors (Peters et al., 1999b). Recently these results were supported in an analysis of lung function growth over a four-year period (Gauderman et al., 2000). Examining the data from a sample of children who were fourth-graders at enrollment, the investigators found statistically significant effects on lung function growth associated with PM10, PM2.5, PM10-2.5 (coarse particles), NO_2, and inorganic acid vapors. The effects were more pronounced for tests measuring airflow at low lung volumes, especially for children spending more time outdoors. However, unlike the cross-sectional results, there were no differences observed by gender. Although the effects on the children who were seventh- and tenth-graders at enrollment were generally also negative, these were not statistically significant, in part because the sample sizes in the higher grades were markedly smaller. As with the cross-sectional symptom data, the independent effects of the different pollutants cannot be assessed because of high inter-pollutant correlations.

Although data on sulfate concentrations have been collected as part of the Children's Health Study, no analyses examining potential independent effects of this component of PM2.5 have been published. According to ARB staff, such analyses will be conducted during the next few years.

7.6.4 The SAPALDIA Study

The Swiss Study on Air Pollution and Lung Disease in Adults (SAPALDIA) examined the long-term effects of air pollution exposure in a cross-sectional study of 9,651 adults residing in eight areas in Switzerland in 1991. Eligibility for the study was conditional on having lived in the same area for at least three years. PM measurements used in the analysis were taken over a 1-year period (1991 for TSP, and 1993 for PM10), on the assumption that air pollution concentrations had not changed significantly over the proceeding several years. Significant associations were observed between chronic symptoms (chronic phlegm, chronic cough, breathlessness at rest during the day or at night, and dyspnea on exertion) and the pollutant metrics TSP, PM10 and NO_2 (Zemp et al., 1999). These associations were strongest for PM10; the investigators estimated that an increase of 10 $\mu g/m^3$ PM10 (within the observed range across cities of 10.1 – 33.4 $\mu g/m^3$), would correspond to increases in risk among never-smokers of 30% for chronic phlegm (OR=1.30, 95% C.I. = 1.04-1.63), 41% for breathlessness during the day (OR=1.41, 95% C.I. = 1.13-1.76), and 23% for dyspnea on exertion (OR = 1.23, 95% C.I. = 1.09-1.39). Nevertheless, the roles of PM10 versus NO_2 in the observed associations could not be ascertained, as NO_2 concentrations were strongly correlated with PM10 levels (r = 0.91).

The SAPALDIA investigators also examined lung function (FEV_1 and FVC) in study participants in relation to several air pollutants, controlling for age, sex, height, weight, atopy, educational level, nationality, smoking status (never, ever, and current), workplace exposures, residential gas stove, serious respiratory infection before age 5, and other potentially influential covariates (Ackermann-Liebrich et al., 1997). Statistically significant decrements in both indices of lung function were found in relation to annual mean levels of PM10, sulfur dioxide, and nitrogen dioxide, with the strongest effects being related to PM10 (-3.4% for FVC and –1.6% for FEV1 in healthy never-smokers, per 10 $\mu g/m^3$ annual average PM10, $p<0.001$ for both estimates). The mean PM10 concentration in this study (measured only in 1993) was 21.2 $\mu g/m^3$ (SD = 7.4), with a range of 10.1 – 33.4. Similar, but slightly smaller, estimates were found for past and current smokers. As with the respiratory symptom analysis, however, the strong pollutant inter-correlations made it impossible to disentangle the effects of the various pollutants ($r_{PM10,SO2} = 0.93$; $r_{PM10,\ NO2} = .91$, $r_{SO2,NO2} = 0.86$). Thus, they concluded that the principal source of all three pollutants, fossil fuel combustion, was associated with the decrements in lung function.

7.6.5 Summary

In summary, the evidence of PM effects in these studies of morbidity in relation to chronic exposures is not as consistent as for mortality. In several studies, the various PM metrics are highly inter-correlated, or co-varied with gaseous pollutants, so that it was not possible to attribute the effects observed to any single pollutant or to a specific mix of pollutants (e.g., the Six-Cities, Children's Health, and SAPALDIA studies). In studies examining effects of exposure to different PM metrics, in some cases the point estimates of effect were greater for those metrics encompassing the coarse fraction (e.g., Dockery et al., 1989), and in some cases the reverse was true. Overall, there is some, albeit weak, evidence of a PM-related effect on chronic morbidity, as measured by chronic respiratory symptoms and lung function. However, it is not possible, based on current evidence, to identify which size cuts or specific constituents are likely to be most influential.

7.7 Susceptible Subgroups at Risk for Mortality

7.7.1 By Disease Status

Pre-existing cardiovascular disease is clearly a risk factor for PM-related mortality. Many of the time-series studies, and both the ACS and Harvard Six-City chronic exposure studies, report statistically significant associations for cardiovascular-specific mortality (for example, Samet et al., 2000a; Ostro et al., 2000; Fairley, 1999; Schwartz, 1993). When compared with all-cause mortality, the cardiovascular-specific mortality typically (but not always) generates larger and more precise effect estimates for PM. These conditions might be further exacerbated by pre-existing respiratory disease. Several mortality studies of acute air pollution exposure provide evidence to identify the most likely sensitive subgroups among adults. For example, Schwartz (1994b) reported that respiratory conditions were more likely to be contributing causes of death on high versus low PM days. Thus, air pollution was associated with increased deaths from respiratory conditions and increased deaths from other causes with respiratory conditions as a contributing factor. In a study of hospital admissions in Cook County, Zanobetti et al. (2000b) found that acute bronchitis and pneumonia increased the risk for admission to hospital with cardiovascular disease, while Zanobetti and Schwartz (2001) found that diabetics were also at greater risk. Finally, in a daily mortality study in Montreal (Canada), Goldberg et al. (2001b) found that the association between PM and mortality was elevated among those with acute lower respiratory disease, coronary artery disease, congestive heart failure, and any cardiovascular disease. No risk elevation was observed for those with acute upper respiratory disease, airways disease (which was defined to include chronic bronchitis, emphysema, asthma, and bronchiectasis), acute coronary artery disease (i.e., acute myocardial infarction, and other acute and subacute forms of chronic ischemic heart disease), hypertension or cerebrovascular disease (i.e., stroke). Taken together, these studies suggest that concurrent lower respiratory infections and subsets of cardiovascular disease may be precursors to death associated with PM.

7.7.2 By Socioeconomic Status

Several mortality studies have examined whether socioeconomic status (SES) and related factors such as education and race/ethnicity affect the magnitude of PM-mortality associations. These studies help address the question of whether factors linked with poverty or educational attainment render individuals more susceptible to the adverse effects of exposure to air pollution. To date the findings have been mixed. The prospective cohort studies investigating the potential impacts of longer-term exposure appear to find consistent effect modification by education, whereas the acute exposure studies do not demonstrate much, if any, modification of these relationships.

In their re-examination of the American Cancer Society (ACS) data set originally analyzed by Pope et al. (1995), Krewski et al. (2000) conducted an exhaustive set of sensitivity analyses. They considered a wide range of alternative specifications, ecological variables, corrections for spatial autocorrelation, interactions, adjustment for time-varying parameters, and measures of occupational exposure, smoking, and physical activity. Their findings corroborated those of the original study.

However, the relative risk (RR) estimates from the prospective cohort studies vary significantly when the analysis was stratified by educational attainment (Table 7.5). For those with a less than high school education, the relative risk (RR) associated with an inter-quartile change in the annual average fine particle concentration was 1.35 (95% C.I. = 1.17 – 1.56), while for those with more than a high school education, the RR = 1.06 (95% C.I. = 0.95- 1.17). This lower risk associated with more education was also observed in the education-stratified re-analysis of the Dockery et al. (1993) study (Krewski et al., 2000). The lack of an

association among more well-educated individuals may indicate that better nutrition and access to health care (or some other variables correlated with educational attainment) may be important co-factors in air pollution-associated mortality. The effect of SES did not appear to be confounded by occupational exposures in these cohorts. For example, among the groups with either low or high occupational exposures, higher educational attainment was associated with lower risks from air pollution. Among individuals with lower educational attainment, poverty, poor nutrition, and less access to medical resources are all more common. Anecdotally, lower SES is also likely to be associated with residences closer to mobile and stationary sources of pollution. Therefore, it is possible that SES is simply associated with higher exposure to existing sources, rather than an effect modifier.

In a third prospective cohort study (of Seventh Day Adventists in California), McDonnell et al. (2000) analyzed a subset located close to airports, in order to utilize airport visibility as a surrogate measures of PM2.5. For the population as a whole, no association was observed between alternative measures of PM (fine, coarse or PM10) and either all cause mortality or non-cancer respiratory mortality. Similarly, no association was apparent for the male cohort. This group was then further disaggregated by other subsets including individuals who were: past smokers, exposed occupationally, exposed to ETS, with a history of cardiovascular or respiratory disease, using antioxidant pills, living in high-density areas, and not using alcohol. The largest observed effect, which was statistically significant, was among those living in high housing density, which is often associated with low SES.

There is some, albeit fragmentary, evidence of effect modification of the PM-mortality relationship by income or education. For example, Zanobetti and Schwartz (2000) tested for effect modification in the four largest cities with daily PM10 data during the study period of 1986 – 1993 (Chicago, Detroit, Minneapolis-St. Paul, Pittsburgh). They used individual-level educational status from the death records of the National Center for Health Statistics. In three of the four cities, the PM10 effect for the cohort members with less than 12 years of education was larger than that for those with more than 12 years of education. In two of the cities, the PM effect for those in the low-education group was more than twice the other cohort. Thus, there was weak evidence of effect modification by education. In contrast, in a study of air pollution and mortality in 10 U.S. cities, Schwartz (2000a) examined whether the city-specific mortality effect was modified by several city-wide factors. No effect modification of the pollution effect was found from unemployment, living in poverty, college degree or the proportion of the population that is nonwhite, although sample size limited the ability for detection. Samet et al. (2000a) tested for effect modification of the PM10-mortality association among the 90 cities used in the study. Using aggregate (city-wide) statistics, they tested for potential modification using local SES-related variables, including household income, percent of the population having less than a high school education, percent using public transit, and percent unemployed. None of these factors helped explain the city-specific pollution effects. However, the variable representing the percent of the population having less than a high school education had a moderate (but still not statistically significant) association with the regression coefficients.

The evidence to date, therefore, suggests that there may be a greater effect of PM among individuals from lower SES groups, although the actual risk factors are unknown. Candidate risk factors include poor nutrition, lower access to and use of health care, and higher air pollution exposures due to location of residences near PM sources such as freeways and industrial facilities.

7.7.3 By Age

7.7.3.1 The Elderly

Existing evidence suggests that most of the more severe effects of PM are likely to be experienced by elderly people with pre-existing heart or lung disease. For example, when the acute exposure mortality studies have disaggregated the sample by age, the elderly subsample typically exhibits stronger associations and larger effect sizes. In some extreme cases, statistically significant effects are observed only for the elderly subset (Goldberg et al., 2001b; Kelsall et al., 1997). As summarized in Table 7.6, PM has, in general, a disproportionate effect on the elderly. For example, a study in Brisbane, Australia (Simpson et al., 1997) found that 81% of all mortality occurred in the age group above 65, but 90% of the PM-related mortality occurred in this group. Likewise, in Santiago, Chile (Ostro et al., 1996) the rates are 65 and 79%, respectively. Thus, a large share, but not all, of the acute-exposure mortality occurs within the elderly population.

7.7.3.2 Infants and Children

While the elderly may dominate the potential population at risk, several recent cross-sectional and time-series studies have reported associations between ambient PM and neonatal or infant mortality, low birth weight or higher rates of prematurity. For example, in Rio de Janeiro (Penna and Duchiade, 1991) and the United States (Woodruff et al., 1997), cross-sectional associations have been reported between measures of PM and neonatal or infant mortality. Woodruff et al. (1997) studied a cohort of four million infants born between 1989 and 1991, who were studied using data from the National Center for Health Statistics. Infants were assigned three different PM10 exposure intervals based on metropolitan area-wide data averaged over the first 2 postnatal months. The mean PM10 was 31 $\mu g/m^3$. Logistic regression was used to examine whether there was an association between early neonatal exposure and total or cause-specific mortality, after controlling for other demographic and environmental factors. Associations were found between higher PM10 exposure and both all-cause and respiratory-specific mortality.

Another study (Dejmek et al., 1999) evaluated the impacts of PM2.5 and PM10 on intrauterine growth retardation (IUGR) in the highly polluted Teplice District in the Czech Republic. Again, three different exposure intervals were determined for several pollutants (PM, nitrogen dioxide and sulfur dioxide) for each month of gestation. Data analysis found no effect from nitrogen dioxide, but PM10 and sulfur dioxide in early pregnancy were associated with IUGR, after controlling for several potential confounders. Both PM10 and PM2.5 (which were highly correlated in this study) were associated with the likelihood of an IUGR birth, defined as one where the birth weight fell below the 10^{th} percentile by gender and age for live births in the Czech Republic. These results suggest that exposure to PM in Teplice (which includes PM2.5, PM10, sulfates, acid aerosols and PAHs) early in pregnancy may impact subsequent fetal growth and development.

Bobak and Leon (1998) conducted a matched case-control study of all births registered in the Czech Republic from 1989 to 1991, which were linked to death records. A logistic model was used to estimate the effects of PM on the risk of death, after controlling for socioeconomic status, birth weight and length, and gestational age. An association was found between PM and post-neonatal respiratory mortality. Bobak (2000) used a somewhat similar database of live births registered in the Czech Republic in 1990-1991 to examine associations between air pollution and both low birth rate and prematurity. The birth outcomes were linked with pollution data on TSP, sulfur dioxide and nitrogen dioxide in the 67 of 85 districts (about 85% of all births) for which data were available. Outcomes studied included the likelihood of lower birth weight (<2,500 g), prematurity (< 37 weeks of gestation) and IUGR (< 10^{th} percentile of

birth weight for gestational age and sex). The analysis controlled for sex, parity, maternal age group, education, marital status, nationality and month of birth. Associations were found between TSP (median concentration = 72 $\mu g/m^3$) and both low birth weight and prematurity, but not with IUGR. The association with TSP and low birth weight appeared to be explained by low gestational age.

In both the cross-sectional and case-control study designs, it may be difficult to separate the effects of pollution from other factors such as poverty, exposure patterns (e.g., in the higher pollution areas people may spend more time outside or live closer to highways), and diet. However, daily time-series studies have also reported associations between changes in PM and infant or child mortality in Mexico City (Loomis et al., 1999) and Bangkok (Ostro et al., 1999a). The statistical models used in these studies were similar to those used in the adult mortality studies of acute exposure – general additive Poisson models, controlling for time, season and weather. In Mexico City, 3- to 5-day lags in PM2.5 (mean = 27 $\mu g/m^3$) were associated with infant (< 1 year) mortality. Likewise, in Bangkok, lags of 2 or 3 days of PM10 (mean = 65 $\mu g/m^3$) were associated with child (< 5 years) mortality. These two studies suggest about a 2 to 4% increase in daily infant mortality per 10 $\mu g/m^3$ PM10. In both of the cities, however, the personal exposure to PM is likely to be much greater than in the U.S. due to factors such as weather, poverty, time spent outdoors, and housing ventilation. In addition, differences in prenatal maternal health status and early postnatal infant diet may make it difficult to extrapolate these findings to California.

Finally, Ritz et al. (2000) reported associations between PM and both low birth weight and premature delivery among a cohort of 98,000 neonates born in Southern California between 1989 and 1993. Prematurity was defined as a birth occurring at less than 37 weeks of gestation. Seventeen monitoring stations throughout the Los Angeles air basin had data for at least four pollutants of interest, including PM10, carbon monoxide, nitrogen dioxide and ozone; only 8 of the stations had PM10 data. Only births for women living within 2 miles of a monitoring station were included in the analysis. Pollution exposures were averaged over several distinct periods, such as 1, 2, 4, 6, 8, 12, and 26 weeks before birth and the entire pregnancy, as well as averages over the first and second months of pregnancy. Several known risk factors were controlled for, including maternal age, race, education, parity, sex of the infant. However, data were not available for maternal smoking or exposure to ETS, marital status, maternal height, and pregnancy weight gain. Ultimately, the strongest association was found between PM10 averaged over the 6 weeks prior to birth and the likelihood of pre-term birth.

7.7.4 Summary

Taken together, the evidence to date suggests that exposure to PM is likely to have a disproportionate effect on the elderly, and possibly on children and infants. The impacts on the elderly have been observed in both the mortality and the hospitalization studies. In the latter, associations were found between PM10 and hospitalization for both cardiovascular disease and respiratory diseases, including COPD and bronchitis. These outcomes are observed primarily in the elderly, and many of the studies restricted the sample to those above age 65. It may be premature to generalize the findings of the effects of PM exposure on infants. Many of the studies were cross-sectional in nature, making it more difficult to attribute the effect to a single factor. On the other hand, the time-series studies were undertaken outside of the U.S., where the pollution concentrations, exposure conditions and underlying socioeconomic factors may be very different from that in the U.S. Besides predicting mortality, several studies have reported associations between exposure to PM and low birth weight, prematurity, and IUGR.

Table 7.5. Relative Risk Estimates for Mortality Related to Average Annual PM2.5 -- Effect Modification by Education in Two Prospective Cohort Mortality Studies*

Study (Δ PM2.5)**	Less than High School Education	High School Graduates	Post-high school education
ACS (24.5)	1.27 (1.13 – 1.42)	1.20 (1.08 – 1.33)	1.05 (0.96 – 1.23)
Six-cities (18.6)	1.45 (1.13 – 1.85)	1.30 (0.98 – 1.73)	0.98 (0.72 – 1.36)

* - Pope et al. (1995); Dockery et al. (1993)

** - Δ PM2.5 = inter-quartile range of PM2.5 (annual average)

Table 7.6. Pollution-related mortality versus all-cause mortality in the elderly population.

City	First Author	% Share of Total Mortality for Elderly	% Share of Pollution-related Mortality for Elderly
Santiago, CH	Ostro (1996)	65	79
Mexico City	Loomis (1999)	57	68
London	Bremner (1999)	82	62
Bangkok	Ostro (1999)	66	73
Brisbane, AU	Simpson (1997)	81	90
Philadelphia	Kelsall (1997)	41	33

7.8 Biological Mechanisms

7.8.1 Overview

Until recently, there was no clear mechanistic explanation for the observed epidemiological findings of mortality and morbidity following acute or subacute exposure to ambient particles, especially those findings referable to the cardiovascular system. However, within the past few years epidemiological and controlled exposure studies in human subjects, as well as some toxicological investigations, have provided evidence of several biologically plausible mechanisms that may underlie some of the serious adverse effects observed in the time-series investigations. The initial target organ affected by exposure to particles is the lung, though small particles have been reported to penetrate into the blood and be detected in the systemic circulation within minutes of inhalation (Nemmar et al., 2001a, b). Within the lung,

effects have been observed in both the conducting airways and the gas-exchange zone, both of which may result in local and systemic effects. In epidemiological studies examining the relationships between PM pollution and mortality, in particular, effects have often involved the cardiovascular system. Much of this section will focus on recent research suggesting mechanisms by which systemic effects, particularly those affecting the heart, may occur.

The basic pathophysiological models of PM-related health impacts begin with deposition of PM in the airways and the alveoli, eliciting an inflammatory response, and potentially affecting pulmonary defenses against infection. Inflammation is a stereotyped biological response to injury or infection and, although necessary in principle for the defense of the organism's physiological integrity, can also result in amplification of injury, both locally and systemically. A variety of cell types in the lung (e.g., alveolar macrophages and epithelial cells) may respond to the presence of particles by secreting chemical messengers (cytokines and chemokines), which in turn can attract inflammatory cells to the lungs from the circulation. Particles may also adversely affect the ability of macrophages to protect the lung against inhaled micro-organisms, which could result in enhanced susceptibility to infection. Programmed cell death (apoptosis) may be induced in both epithelial cells and macrophages by particles, further reducing native defenses against environmental stresses. Inflammation of the bronchi and bronchioles is associated with airway hyperresponsiveness, accompanied by an increased propensity of smooth muscle cells of the airways to constrict in response to irritants, cold air, pharmacological spasmogens, and other agents.

Acute responses to PM may also involve effects on the autonomic nervous system and the composition of the blood. Chronic lung diseases, including asthma, emphysema, and chronic bronchitis, all involve ongoing, unresolved inflammation in the lung. Additional inflammatory stimuli in the lungs could exacerbate chronic lung disease, resulting in bronchoconstriction and respiratory symptoms, as well as reduced blood oxygenation. In addition, chronic inflammation may facilitate PM-induced release of pro-inflammatory mediators, resulting in additional pulmonary inflammation and systemic (including cardiac) effects.

7.8.2 Pulmonary and Systemic Inflammation

Reports in humans and experimental animals suggest that inhalation of particles from diverse sources can cause pulmonary and systemic inflammatory responses. Many of these experiments involved exposure concentrations well above ambient levels, as well as nonphysiologic modes of administration (such as intratracheal administration), both of which must temper extrapolation of the results to general population exposures. In a variety of *in vivo* animal and *in vitro* experimental models (Costa and Dreher, 1997; Kennedy, et al., 1998; Brain, et al., 1998; Li et al., 1996), exposures to high concentrations of PM have been found to cause lung inflammation, cell and tissue injury, and changes in cell populations. In many cases, toxicological studies involving high-level exposures *in vitro* or by intra-tracheal instillation or inhalation indicate that the presence of soluble transition metals (e.g., iron, vanadium, nickel) enhances inflammatory responses (Kodavanti et al., 1997, 1999; Monn and Becker, 1999; Costa and Dreher, 1997; Li et al., 1997). These metals may generate localized oxidative stress through the formation of oxygen-based free radicals, such as the potent hydroxyl radical (Donaldson et al., 1997). The injury caused by oxidative stress may lead to a decrease in epithelial integrity, resulting in enhanced transfer of particles into the lung interstitium. The presence of particle-associated metals is not, however, a *sine qua non* for inflammation to take place. Ultrafine carbon black particles (i.e., particles of aerodynamic diameter less than 100 nm or 0.1 μm) appear to cause markedly greater inflammation than fine particles in experimental settings; these effects of ultrafine particles are not mediated by soluble metals or iron at the particle surface (Brown et al., 2000). Moreover, on a mass basis, ultrafine carbon black particles exert a greater effect than fine particles *in vitro* on alveolar

macrophage function, which could, in theory, affect the host's ability to clear other particles, including infectious micro-organisms (Renwick et al., 2001).

PM-associated organic compounds such as polycyclic aromatic hydrocarbons (PAHs) may also exert toxic effects in the lung via oxidative stress. A series of experiments using diesel exhaust particles (DEP) demonstrates the induction of reactive oxygen species (ROS, including hydrogen peroxide and superoxide) by both lung macrophages and epithelial cells (Nel et al., 2001). Generation of such oxidant stress can activate specific transcription factors, including nuclear factor κB and activator protein-1, which can upregulate the expression of genes for cytokines, chemokines, and other pro-inflammatory mediators. DEPs or organic extracts of DEPs may also, through oxidant effects on mitochondria, induce programmed cell death (apoptosis) or necrosis of macrophages and respiratory epithelial cells (Nel et al., 2001). Moribund macrophages release additional ROS in their immediate environments, amplifying the oxidative stress and, in addition, would be unable to engulf and kill infectious micro-organisms. Apoptosis of respiratory epithelial cells could lead to a loss of integrity of the lining of the airways, which may facilitate airway hyperresponsiveness and exacerbation of asthma or other conditions involving airway inflammation. Generation of oxidant stress has also been demonstrated both *in vivo* and *in vitro* after exposure to concentrated, resuspended PM2.5 and ultrafine carbon black (Shukla et al., 2000). While these experiments are suggestive of potential effects in humans, the exposure modes and concentrations used constrain the general applicability of the results.

Although there has been little toxicological work examining potential impacts of coarse versus fine particles, some recent literature indicates that, *in vitro*, the coarse fraction can elicit greater pro-inflammatory effects than the fine fraction, due at least to metals and endotoxin in the coarse fraction (Monn and Becker, 1999; Soukoup and Becker, 2001). Endotoxin is a generic name for an essential component of gram-negative bacterial cell walls, and is nearly ubiquitous in soils. Exposure of humans to endotoxin in largely occupational settings has resulted in increased lung inflammation, enhanced airway responsiveness, increases in systemic immune cell populations, and decrements in lung function (Michel et al., 1997; Vogelzang et al., 1998; Zock et al., 1998). Monn and Becker (1999) demonstrated the importance of endotoxin associated with the coarse particle fraction (PM10-PM2.5) in the induction of pro-inflammatory cytokines, such as interleukin-6. In these *in vitro* studies, coarse fraction PM induced cytokine levels 50 times higher than those seen with the soluble fractions of coarse PM or fine-mode particles. Kleinman et al. (1995) demonstrated that lung permeability, a measure of cell damage and inflammation, was increased by coarse fraction road dust exposure in a dose-dependent fashion. While the relevance of such work to human responses to ambient PM remains to be established, it is clear that different size cuts of PM (coarse, fine and ultrafine) of PM10 can deposit throughout the airways (see Section 7.1), and have the potential to elicit intrapulmonary inflammation and compromise the functional abilities of alveolar macrophages.

The intrapulmonary responses elicited by PM may be due in part to neurogenic inflammation. Sensory neurons in contact with irritant particles (e.g., within the conducting airways) can be stimulated to release neuropeptides (e.g., substance P, calcitonin gene related peptide, neurokinin A and others), which can initiate airway inflammatory events, including release of cytokines, vasodilation, and mucus secretion. Neuropeptides act on a variety of cell types within the lung, including epithelial and smooth muscle cells (resulting in modulation of inflammation and airway hyperresponsiveness), as well as immune cells (polymorphonuclear cells or PMNs, lymphocytes, eosinophils, and others), which can amplify the inflammatory response. Recent *in vitro* experiments indicate that specific irritant (capsaicin or vanilloid) receptors on neurons are necessary for PM-related neurogenic inflammation to occur, as evidenced by responses to several types of particles, including ambient particles collected

from St. Louis and Ottawa, coal fly ash, residual oil fly ash, and particles from the eruption of Mt. St. Helens (Veronesi et al., 2000).

Several controlled exposure studies in humans clearly demonstrate that particle inhalation evokes an inflammatory response. Salvi et al. (1999) exposed 15 healthy human adult volunteers to either air or diesel exhaust (PM10 concentration = 300 $\mu g/m^3$) for an hour each, at least 3 weeks apart, and examined inflammatory responses 6-hr post-exposure in bronchial washings, bronchoalveolar lavage fluid, bronchial biopsies, and in peripheral blood samples. They observed a vigorous inflammatory response in the samples obtained from the lung, including significantly increased numbers of PMNs, T- and B-lymphocytes, mast cells, inflammatory mediators (histamine and fibronectin), as well as several adhesion molecules that facilitate the passage of inflammatory cells from the circulation into the airways. In the blood samples, they reported increased platelets (cells involved in the initial formation of blood clots) and PMNs, suggesting that the diesel exposure stimulated the bone marrow to release these cells into the circulation and then to the airways.

Subsequently, the same group of investigators reported that this diesel exposure protocol also resulted in increased intra-airway transcription of messenger RNA for interleukin-8 (IL-8), a protein that attracts PMNs to sites of injury (Salvi et al., 2000). In addition, they detected increased production of IL-8 and another protein (GRO-α), both of which promote inflammation, in the subjects' airways. Another laboratory (Nightingale et al., 2000) also reported evidence of airway inflammation following a different experimental protocol in 10 healthy adult volunteers (involving 2-hr exposures to 200 $\mu g/m^3$ of re-suspended diesel exhaust particles, with different timing and methods of obtaining intra-airway specimens). Though this group found no increases in three mediators of inflammation in the subjects' blood, they did report an increase in exhaled carbon monoxide after diesel exhaust exposure, suggesting the presence of oxidative stress in the lung (Nightingale et al., 2000). The increased exhaled carbon monoxide levels are thought to be due to the oxidant-related induction of the enzyme heme oxygenase, which catalyzes the first step in the degradation of heme (the principal structure of hemoglobin) to bilirubin, producing carbon monoxide as a by-product (Otterbein et al., 2000). Though the findings of Nightingale et al. (2000) are somewhat inconsistent with those of Salvi et al. (1999, 2000), some of the discrepancies may be due to differences in the study designs and methods. For instance, some of the discordance may be due to differences in doses or in the timing of sample collection; inflammatory responses follow a consistent succession of events, with increases in different cytokines and cell types occurring sequentially. These events begin within hours of the initial exposure, which could help explain the short time lag between exposure and outcome observed in some time-series studies (Nordenhäll et al., 2000).

Taken together, these publications suggest a potential pathway by which particles might increase airway inflammation and provoke exacerbations of chronic respiratory disease such as asthma. However, these data should be interpreted cautiously. First, the exposure concentrations were relatively high: ambient particle levels rarely reach 200 - 300 $\mu g/m^3$ in the U.S., though this range is not uncommon in some of the larger cities in the developing world. In addition, diesel exhaust exposures may not be representative of PM generally, and are well recognized to enhance allergic inflammation (Nel et al., 1998). However, in some cities outside the U.S. (such as London, UK, or Santiago, Chile), diesel exhaust particles comprise the majority of small particles (QUARG, 1993; Cifuentes et al., 2000). Moreover, other particles administered in high doses (e.g., residual oil fly ash) are capable of amplification of the allergic response in experimental animals (Gavett et al., 1999). Finally, the Salvi et al. studies involved whole diesel exhaust, which also contains oxidant gases known to enhance intra-pulmonary inflammation. While additional controlled studies using lower exposure concentrations might be useful to confirm these results, these kinds of investigations have

other limitations on interpretability, including the typically small number of subjects, ethical and practical constraints on subject eligibility (i.e., those at either end of the age spectrum, people who are seriously ill would routinely be excluded), and other factors that might bias the results towards the null hypothesis of no effect.

In a controlled study using particles potentially more representative of those to which the general population is exposed, Ghio et al. (2000) reported evidence of mild airway inflammation, without concomitant lung injury. In this study the investigators used concentrated ambient particles (CAPs) collected in the immediate vicinity of the Human Studies Facility of the U.S. EPA in Chapel Hill, NC. The investigators exposed 38 healthy adults once to either clean air (n = 8) or CAPs (n = 30) for 2 hours, with intermittent exercise. The CAPs exposures ranged from 23.1 to 311.1 $\mu g/m^3$ of PM2.5, with a mean concentration of 120.5 $\mu g/m^3$. Technical limitations of the concentrator restricted the range of particles collected to those with diameters between 0.1 and 2.5 μm. As reported in the controlled diesel exposure studies (discussed above), they found an influx of PMNs into the airways (an approximately 3.7-fold increase in bronchial washings and 6.2-fold increase in bronchoalveolar washings obtained 18 hr post-exposure), comparable to what has been observed among individuals exposed to low concentrations of ozone for several hours. However, they found no increase in indicators of lung injury or in the concentrations of a variety of pro-inflammatory mediators (IL-8, IL-6, fibronectin, and others) in the lung lavage fluid. Moreover, most of the blood parameters analyzed showed no exposure-related changes (hemoglobin, hematocrit, red blood cell count, PMNs, lymphocytes, other white blood cells [monocytes], platelets, ferritin [an iron transport protein that can increase during the early phases of an inflammatory reaction], or blood viscosity). However, fibrinogen, a key constituent involved in blood clotting, was elevated by the CAPs exposures relative to clean air ($p = 0.009$), with no obvious exposure-dependence. Thus, while not entirely consistent with the diesel exhaust controlled exposure studies discussed in preceding paragraphs (which may be due in part to differences in experimental protocol), this CAPs study suggests that exposures to ambient particles in healthy humans can result in a mild pulmonary inflammatory response. Though the exposure concentration was higher than what would ordinarily be encountered in the U.S., the cumulative particle exposure experienced by most of the subjects in this experiment would be lower than 24-hr PM exposures in many urban areas.

Tan et al. (2000) obtained venous blood samples at weekly intervals from 30 military recruits in Singapore who followed standardized outdoor activities throughout the Southeast Asia haze episode of 1997 resulting from wildfires in Indonesia. Measures of immature PMNs in the subjects' blood were analyzed in relation to daily measures of several pollutants (including 24-hr PM10) monitored during and after the haze episode, which lasted for about 5 weeks. During the episode the mean PM10 concentration was 125.4 $\mu g/m^3$, while afterwards it was about 40.0 $\mu g/m^3$. Tan et al. (2000) found the strongest relationship between same-day PM10 and increased immature PMNs in the circulation, though there was also a statistically significant relationship with a one-day lag. Although not sufficient to establish a cause-and-effect relationship, these results suggest an immediate stimulation of the bone marrow from inhalation of smoke containing high levels of particles, resulting in the early ejection of immature PMNs into the circulation.

In a subsequent experiment in which rabbits had 5 mg of PM10 (previously collected in Ottawa, Canada) instilled intrapharyngeally twice a week for three weeks, the same laboratory found that repeated PM exposure increased the production of PMNs in the bone marrow and accelerated their release into the circulation (Mukae et al., 2001). The PM10 exposure resulted in diffuse inflammation of the lungs, with particles present in alveolar macrophages, lung epithelial cells (Type II pneumocytes), and in the airway walls. The effects on PMN production in bone marrow and release of immature cells into the blood were

associated with the numbers of particles ingested by alveolar macrophages. Also, for purposes of comparison, the investigators found that a higher percentage of human alveolar macrophages, obtained from lung sections removed from both smokers and nonsmokers with small lung tumors, contained fewer particles than those taken from the experimental rabbits (Mukae et al., 2001).

For individuals with chronic lung disease, such as asthma or COPD, such pro-inflammatory effects may result in exacerbation of disease. PM effects on alveolar macrophage function may also compromise one of the principal pulmonary defenses against infection (Renwick et al., 2001). The latter may also represent an important pathway for worsening of both asthma and COPD, as serious exacerbation of both conditions is often related to respiratory infection.

Taken together, these data suggest that inhalation of different sources of particles may initiate inflammatory events in human lungs, with some (albeit sparse) evidence of systemic impacts, including stimulation of bone marrow to accelerate production of inflammatory cells to respond to the pulmonary insult. However, these observations are subject to the caveat that the results observed in the high-dose animal and *in vitro* experiments, as well as in the controlled human exposures, may or may not be directly applicable to humans exposed to ambient PM.

7.8.3 Effects on the Circulation and Cardiac Events

Changes in the composition of the blood may result from PM exposure, with potentially serious effects on individuals with cardiovascular disease. Several years ago, Seaton et al. (1995) proposed that exposure to ultrafine particles might induce alveolar inflammation, which could lead to exacerbation of pre-existing lung disease and increased blood coagulability. Increased blood coagulability could in turn lead to acute cardiovascular events, notably myocardial infarctions, by the formation of blood clots (thrombi) in compromised coronary arteries, or through the formation of such thrombi in other sites, which subsequently travel through the circulation to the coronary arteries. Research during the past decade has demonstrated that thrombus formation is the critical event in many patients suffering an acute coronary event (Rosito and Tofler, 1996). As described above, several studies of controlled exposures to particles demonstrate increases in both cellular and biochemical markers of inflammation in the lung (Salvi et al. 1999, 2000; Nightingale et al., 2000; Ghio et al., 2000). This observation is subject to the caveat that three of these four studies involved exposures to high concentrations of diesel exhaust particles, which may not necessarily be representative of ambient PM generally. The Ghio et al. (2000) study also noted a PM-related increase in fibrinogen, a key component in blood coagulation). Fibrinogen concentrations have been reported to be elevated in cigarette smokers and individuals exposed to cigarette smoke, which is well recognized as a risk factor for cardiovascular disease (Sato et al., 1996; Iso et al., 1996). At least one study of rats exposed to residual oil fly ash particles at a high dose level (8.3 mg/kg by intratracheal instillation) also found an increase in the animals' blood fibrinogen levels (Gardner et al., 2000). Plasma viscosity was also elevated in these animals, but not significantly so. Some recent epidemiological data suggest potential effects of particulate air pollution on blood coagulation (Peters et al., 1997; Seaton et al., 1999). Recently, PM pollution has also been linked with the onset of myocardial infarction (Peters et al., 2001a). While the existing evidence is still somewhat sparse and is not completely consistent, plausible mechanisms for the time-series results regarding cardiovascular morbidity and mortality are beginning to emerge.

Using data collected as part of a large cross-sectional study of cardiovascular risk factors in southern Germany (MONICA -- MONItoring of trends and determinants in CArdiovascular disease), Peters et al. (1997) analyzed blood viscosity in relation to a 13-day air pollution episode that occurred in January 1985. During the episode, TSP and sulfur dioxide were

markedly elevated. The investigators found that, although the distributions of viscosity had not shifted during the episode, there was a tendency (among some of the participants) towards higher values on episode days. During the air pollution episode, the risk of having blood (or strictly speaking, plasma) viscosity above the 95th percentile [determined for the whole study, including before and after the episode] was increased in both genders (OR = 3.62, 95% CI = 1.61-8.13 for men, and OR = 2.26, 95% CI = 0.97-5.26 for women). Odds ratios for increased plasma viscosity related to a 100 $\mu g/m^3$ increment in TSP concentration were also elevated for both men and women, but were not statistically significant. Blood viscosity has been associated with severity of cardiovascular disease (Junker et al., 1998). Moreover, subjects with elevated plasma viscosity also tended to have increased heart rates as well, suggesting multiple pathways of elevated cardiovascular risk (Peters et al. 2000b). Fibrinogen, one of the principal proteins involved in the determination of blood viscosity, is well established as an important independent risk factor for myocardial infarction and stroke (Yarnell et al., 1991; Ernst et al., 1993). However, fibrinogen was not specifically assayed in this investigation.

In a subset of the German MONICA study population, consisting of 631 randomly selected healthy men aged 45 to 64 years, the investigators examined C-reactive protein concentrations in blood obtained during the initial cross-sectional study (1984-85) and again three years later (Peters et al., 2001b). C-reactive protein (CRP) is a sensitive indicator of infection, injury, and inflammation, and has been linked with increased risks of both incidence and exacerbation of cardiovascular disease (Haverkate et al., 1997; Rifai and Ridker, 2001). CRP levels were elevated during the 1985 air pollution episode, with the strongest effects related to TSP. In multivariate regression analyses, the odds of having elevated CRP above the 95th percentile (for the entire study) were increased by $\approx$ 50% for same-day TSP (31 $\mu g/m^3$ inter-quartile range), to $\approx$ 75% for a five-day TSP average (26 $\mu g/m^3$ inter-quartile range). These increases were unchanged even after deletion of the 1985 episode days, indicating that acute and subacute effects could be observed even at normal ambient PM levels: the mean TSP concentrations during the two study periods were 54 $\mu g/m^3$ in 1984/85 and 47.8 $\mu g/m^3$ in 1987-88.

In a large, representative cross-sectional sample of the United States population, Schwartz (2001b) found that ambient PM10 was associated with elevated blood levels of several cardiovascular risk factors. Schwartz (2001b) examined local PM10 concentrations either the same day or the day before an extensive questionnaire and physical examination (including obtaining venous blood samples) were administered to approximately 20,000 individuals in 44 communities as part of the Third National Health and Nutrition Examination Survey. In single pollutant models, controlling for age, race, sex, body mass index, and cigarette smoking, PM10 concentrations were significantly associated with serum fibrinogen levels, platelet counts, and white blood cell counts. Platelets and fibrinogen were also associated with NO_2, while WBC counts were associated with SO_2: none of the three blood markers were associated with ozone. In multi-pollutant models, only the coefficients linking PM10 and these cardiovascular risk factors remained significant. Schwartz undertook extensive sensitivity analyses, examining the potential impacts of social factors (poverty, educational attainment, household size), other exposures (environmental tobacco smoke, serum cotinine [a biomarker of exposure to tobacco smoke], use of a wood stove, fireplace, or gas stove), dietary influences (serum vitamin C, intake of fish, shellfish, saturated fat, caffeine, and alcohol), as well as other cardiovascular risk factors (systolic blood pressure, total serum cholesterol and high density lipoprotein levels). The associations between PM10 and fibrinogen, platelet counts, and WBC counts remained robust to the inclusion of all of these potential confounders and effect modifiers. The estimated odds ratios for being in the top 90th percentile of the distribution of these blood markers for the entire NHANES population associated with an

interquartile change in PM10 (26 $\mu g/m^3$) were 1.77 (95% CI = 1.26-2.49) for fibrinogen, 1.27 (95% CI = 0.97-1.67) for platelet counts, and 1.64 (95% CI = 1.17-2.30) for WBC counts.

Seaton et al. (1999) obtained monthly blood samples from 112 elderly individuals in two cities in the United Kingdom, and investigated relationships between several blood constituents and 3-day PM10 concentrations (including modeled personal exposure and central city real-time measurements). While there was no relationship between personal PM exposure and fibrinogen, the investigators found an unanticipated pattern of PM-associated changes in blood components suggesting a sequestration of red blood cells, specifically decreased levels of hemoglobin, RBCs, and packed cell volume. In addition, there was a significant decrease in platelets in relation to personal PM exposure and a decrease in fibrinogen associated with central-city PM measurements (both of these blood components are involved in the formation of blood clots). Finally, they observed a significant increase in CRP, consistent with the recent Peters et al. (2001b) study discussed above. Seaton et al. (1999) speculated that these results might be explained by particle-associated effects on RBC adhesive properties, making these cells more likely to be involved in thrombus formation in the circulation. The findings related to decreased RBCs, hemoglobin, platelets and fibrinogen are not entirely consistent with the results of the controlled exposure study by Ghio et al. (2000) or the cross-sectional data from Schwartz (2001b), discussed above.

If indeed PM pollution might be causally linked with increased formation of blood clots, one might also expect to see a relationship with the incidence of myocardial infarctions. One mechanism by which myocardial infarction may develop is through disruption of an atherosclerotic plaque in one of the coronary arteries; the extent to which this becomes a site of thrombus formation depends in part on the balance of forces affecting blood coagulation in the individual's circulation. Recently, Peters et al. (2001a) examined potential associations between PM concentrations and the timing of symptom onset in 772 patients with myocardial infarction in the greater Boston area. They found significant associations between symptom onset and both acute (within 2 hr prior to symptom onset) and subacute (24-average PM2.5 in the previous day) exposures, after adjusting for season, weather, and day of the week. Moreover, they found increasing risks with increasing PM2.5 concentrations. Adjusted odds ratios for increases in PM2.5 from the 5^{th} to the 95^{th} percentiles in 2-hr (25 $\mu g/m^3$, representing the range of the 2-hr average PM2.5 distribution between the 5^{th} and 95^{th} percentiles) and 24-hr (20 $\mu g/m^3$, representing the range of the 24-hr average PM2.5 distribution between the 5^{th} and 95^{th} percentiles) exposures were 1.48 (95% CI=1.09-2.02) and 1.62 (95% CI=1.13-2.34), respectively. For PM10 the comparable odds ratios for 2-hr (40 $\mu g/m^3$) and 24-hr (30 $\mu g/m^3$) averaging times were 1.51 (95% CI=1.06-2.15) and 1.66 (95% CI=1.11-2.49), respectively. In this study the mean levels of 2-hr and 24-hr average PM2.5 were both 12.1, and for PM10 the corresponding mean values were both 19.4, though in both instances the shorter averaging intervals showed greater variability. Interestingly, the entire range of 24-hr PM2.5 concentrations in this study was lower than the U.S. EPA's ambient air quality standard for fine particles of 65 $\mu g/m^3$.

7.8.4 Disturbances of the Cardiac Autonomic Nervous System

PM-associated mortality may be explained, at least in part, by alterations in autonomic nervous system balance. Heart rate variability (HRV – a measure of the heart's ability to respond to stress), resting heart rate, blood pressure, and cardiac arrhythmias are all intimately connected with the balance between the two principal components of the autonomic nervous system – i.e., sympathetic and parasympathetic nervous systems. Numerous studies have demonstrated an association between cardiac autonomic balance and all-cause mortality (Tsuji et al., 1994), sudden cardiac death (Algra et al., 1993), and death due to congestive heart failure (Szabó et al., 1997).

HRV refers to oscillations both in the intervals between consecutive heart-beats and in consecutive instantaneous heart rates as observed on an electrocardiogram. Reduced HRV is considered a good predictor of increased risk of cardiovascular morbidity and mortality (Tsugi et al., 1994; 1996; Nolan et al., 1998). HRV can be used to stratify the risk of sudden death following myocardial infarction (Kleiger et al., 1987; Copie, 1996) and in congestive heart failure (Szabó et al., 1997). A marked decrease in HRV is observed immediately preceding EKG changes precipitating ischemic sudden death; fatal arrhythmias may be triggered by such sudden autonomic dysfunction (Corbalan et al., 1974; Pozzati et al., 1996). Although decreased HRV clearly indicates a worse prognosis for individuals with heart disease, it is unknown whether this relationship is causal or whether decreased HRV represents only an epiphenomenon of more fundamental pathophysiological changes. Moreover, though several studies (described in the following paragraphs) demonstrate associations between PM exposure and HRV, the mechanistic linkage (if any) between these phenomena is unknown.

Several recent publications have linked exposure to ambient PM with decreased HRV (Liao et al., 1999; Gold et al., 2000; Pope et al., 1999c). There are at least a half dozen ways of measuring changes in HRV discussed in these papers, and there are some differences in results between studies. However, they are all consistent in demonstrating an inverse relationship between particulate air pollution and at least one measure of HRV. Of particular interest in these studies is the observation that these HRV changes could be observed shortly after exposure to PM (i.e., within hours).

The first published study examining the relationship between air quality and heart rate variability involved seven individuals with heart disease (congestive heart failure, angina, history of myocardial infarction, coronary artery bypass graft surgery, and arrhythmias), whose heart rates and rhythms were monitored on several occasions with and without elevated levels of particulate air pollution (Pope et al., 1999c). In this small study, PM10 was associated with decreased measures of total HRV (SDNN) and long-term HRV (SDANN), but an increase in one of the short-term measures of parasympathetic tone (r-MSSD). While parasympathetic tone is generally considered to have a beneficial or protective effect, there is at least one study suggesting that increases in parasympathetic stimulation of the heart may be linked to serious arrhythmias (Kasanuki et al., 1997).

Liao and colleagues (1999) undertook standardized cardiac monitoring in 26 elderly residents of a retirement home in Baltimore over a three-week period, examining changes in HRV in relation to several concurrently measured indoor and outdoor particulate metrics. Among the 18 subjects with pre-existing cardiovascular disease, the investigators reported statistically significant, decreased HRV in relation to several indoor and outdoor measures of PM2.5 measured the same day or one day previously. Minimal, nonsignificant effects were observed among the subjects with no documented cardiovascular disease, though the number of individuals in this group was small (n=8). One aspect of the analysis included dividing each individual's HRV (specifically, the high-frequency power, an indicator of parasympathetic tone) into tertiles, and evaluating the relationships between PM2.5 levels and the position of the high-frequency power on any given day within that individual's distribution for the whole study. The investigators reported that, when the 24-hour PM2.5 concentration exceeded 15 $\mu g/m^3$, the risk of having an individual's HRV in the lowest third of his or her HRV distribution increased by three-fold, compared to days when the PM2.5 concentration was lower (OR = 3.08, 95% C.I. = 1.43 – 6.59). The clinical significance of this report is unclear; however, as cardiac parasympathetic activity is generally considered beneficial, acute decreases in this index of HRV may indicate an increased risk of an adverse cardiac event.

Gold and colleagues (2000) conducted 163 brief (25 minutes) electrocardiographic measurements in 21 ambulatory Boston residents (aged 53 to 87), once a week over a three-month period. Ambient PM2.5 and PM10 were measured in real-time with TEOMs located about 6 km from the study site. They reported a variety of statistically significant effects on two measures of HRV related to PM2.5, measured during the hour of EKG monitoring and during the three hours prior to such monitoring. No associations between PM2.5 and HRV were seen at a lag period longer than 24 hours, nor was any association noted for coarse particles. Although different metrics were used in this study than in the Liao et al. (1999) investigation, these investigators also found a relationship between PM2.5 and decreased parasympathetic cardiac activity for a short interval preceding the measurement of HRV.

Another recent publication by Pope et al. (2001) reinforces the observations that changes in HRV can occur quite rapidly after exposure to air pollution. Sixteen volunteers were monitored electrocardiographically over the course of a day when they spent alternating 2-hour periods outside and inside a smoking lounge at a major airport. Several measures of HRV were significantly decreased in relation to several measures of exposure during the 2-hr periods in the smoking lounges. In contrast to the Liao et al. (1999) and Gold et al. (2000) reports, the measures reflecting parasympathetic tone appeared to be less strongly affected than the other measures relative to measured particles. While cigarette smoke contributes little to ambient air pollution, the rapidity of the changes observed in HRV is consistent with the findings of the studies discussed above.

Exposure to particulate air pollution has also been associated with another potentially adverse disturbance of the cardiac autonomic nervous system, as manifested by increased heart rate. Increased resting heart rate is considered an independent risk factor for cardiovascular mortality (Goldberg et al., 1996, Mensink and Hoffmeister, 1997). This phenomenon has not been extensively investigated in epidemiological studies. Pope et al. (1999b) found that, among 90 elderly but healthy individuals in Utah, PM10 levels were related to small, but significantly increased resting heart rates. For instance, a 100 $\mu g/m^3$ increase in PM10 (same-day) was associated with about a 50% increased risk of having at least a 10-beats/min elevation in heart rate or pulse (OR =1.51, 95% C.I. = 1.00-2.29), while PM10 lagged by one day was associated with a near-doubling of the risk of the pulse increasing by at least 10 beats/min (OR =1.95, 95% C.I. = 1.35-2.82).

In another analysis of the German participants in the MONICA study (discussed above), Peters et al. (1999c) assessed whether resting heart rates increased in relation to air pollution among a subset of 2,681 men and women who had valid electrocardiographic tracings during both the 1984-85 and 1987-88 parts of the study. During the 1985 episode, resting heart rates were increased, more so in women than in men, relative to non-episode days of the study. In addition, mean heart rates were slightly, but significantly, elevated in relation to same-day and five-day averages of TSP, sulfur dioxide, and carbon monoxide. Even excluding the episode days from the analyses, both TSP and sulfur dioxide were still both related to small, but significant changes in mean heart rates (between 1 and 2 beats/min). Though the overall mean elevations in heart rate were small, they provide support for the notion that PM air pollution is associated with altered autonomic control of the heart.

In contrast to these studies, Gold et al. (2000), in a study of elderly Boston residents, found that PM2.5 levels were associated with decreased resting heart rate. However, this finding appears to be physiologically inconsistent with the finding of decreased PM-associated short-term HRV in this panel, as described above. The investigators speculated that this inconsistency may be due to autonomic dysregulation, in which both HR and HRV might decrease in concert. In any case, there is limited evidence that ambient PM is associated with changes in heart rate in humans.

Control of blood pressure is another manifestation of the influence of the autonomic nervous system, particularly the sympathetic nervous system. Elevated blood pressure (or hypertension) is the most common cardiovascular condition in the U.S., affecting over 60 million Americans (Oparil, 1992). Hypertension is a well recognized risk factor for cardiovascular disease, stroke, and renal disease. In an examination of a subset of 2,607 participants in the German MONICA study (discussed above), 5-day average TSP (70 $\mu g/m^3$) was associated with a 1.96 mm Hg increase in systolic blood pressure (SBP), adjusting for relevant confounders and effect modifiers, including temperature, barometric pressure, and individual cardiovascular risk factors (Ibald-Mulli et al., 2001). Although sulfur dioxide was also associated with increased SBP, inclusion of both pollutants in the same regression models indicated that the TSP effect dominated that of sulfur dioxide. Interestingly, the effects on SBP were magnified in individuals with other cardiovascular risk factors: for subjects with high levels of plasma viscosity, a 90 $\mu g/m^3$ same-day increase in TSP was associated with a 6.93 mm Hg increase in SBP (95% CI = 4.31-9.75); while among those with higher resting heart rates (>90^{th} percentile, or > 80 beats/min), the same increment in TSP was associated with a 7.76 mm Hg increase in SBP (95% CI = 5.70-9.82). These findings suggest that there may be persons with pre-existing cardiovascular disease who are especially susceptible to autonomic effects of exposure to ambient particles. How PM may affect SBP is unknown, but may be related to increased blood levels of endothelin-1, a protein involved with regulating vascular tone, which has been detected in the blood of experimental animals exposed by inhalation of very high levels (40 mg/m^3) of resuspended urban particles, even though these failed to produce obvious structural pathology in the animals' lungs (Bouthillier et al., 1998). Endothelin-1 is produced not only by lung capillary (endothelial) cells, but also by airway epithelial and neuroendocrine cells, as well as macrophages. A variety of potentially adverse cardiovascular effects have been associated with elevated levels of endothelin-1, including increased blood coagulability, worsening of congestive heart failure, and increased risk of mortality after myocardial infarction (Bouthillier et al., 1998).

Finally, the incidence of serious cardiac arrhythmias has been linked with exposure to PM2.5. Implanted cardioverter defibrillators (ICDs) can initiate pacemaker activity if required, or provide an electric shock to the heart in order to terminate potentially fatal arrhythmias (ventricular fibrillation or ventricular tachycardia). An ICD logs each such event electronically. Peters and colleagues (2000b) recorded the ICD data for 100 individuals for approximately 3 years, and compared the ICD events with air pollution over this period. Overall, NO_2 and CO appeared to provide the strongest associations with ICD discharges. In the most susceptible members of this population (i.e., those with 10 or more discharges [n = 6]), however, PM2.5 and PM10 were both associated with an increased risk of an ICD discharge (OR = 1.64, 95% CI = 1.03 – 2.62; and OR = 1.68, 95% CI = 0.98 – 2.86, respectively, with a 2-day lag for each). Though the effects for both PM2.5 and NO_2 were essentially linear, including both pollutants in the same regression model reduced the PM effect to zero, while the NO_2 estimate remained unchanged. Although this study is limited by the small number of patients at high risk, and by the lack of individual clinical data other than the ICD discharges, it does suggest another potential effect of PM (as well as gaseous pollutants) on cardiac autonomic balance. A recent mortality time-series study conducted in the Netherlands (Hoek et al., 2001) provides some consistency with these findings, with risks of mortality from arrhythmia in relation to 7-day means of black smoke (40 $\mu g/m^3$, RR= 1.071, 95% CI=1.001-1.146) and PM10 (80 $\mu g/m^3$, RR=1.041, 95% CI = 0.932-1.163).

Recent publications involving PM exposures of "sick" or compromised experimental animals provide evidence supportive of these findings in humans. The compromised animal models examined in these studies include monocrotaline (MCT) treated rats, which serve as a model for pulmonary hypertension, rodents with chronic bronchitis induced by high-level sulfur

dioxide exposure, spontaneously hypertensive rats, and aged rodent models. Effects observed under these exposure conditions include a variety of cardiac arrhythmias, bradycardia (slowing of the heart rate), increases in plasma fibrinogen (a protein integral to blood clotting discussed above), hypertension, increases in pulmonary inflammation and mortality (Costa and Dreher, 1997; Kodavanti et al., 1999, Watkinson et al., 1998, 2000; Campen et al., 2000; Gardner et al., 2000).

A series of experiments in spontaneously hypertensive (SH) rats is illustrative of the utility of compromised animal models. The pathophysiology of hypertension in the SH rats is similar to that observed in essential hypertension in humans. Kodavanti et al. (2000a) examined normotensive and spontaneously hypertensive rats, exposed to filtered air or to high-dose (15 mg/m^3) residual oil fly ash (ROFA – a source containing high levels of the soluble metals iron, vanadium, and nickel) particles by nose-only inhalation for six hours/day for three days. They found that, compared to normotensive rats, the SH rats had evidence of pulmonary inflammation, alveolar hemorrhage, cardiomyopathy, and evidence of ST-segment depression by electrocardiography (ECG), an indicator of insufficient oxygen delivery to the heart muscle. After ROFA exposures, the SH rats showed significantly greater pulmonary injury and inflammation, including alveolar hemorrhage, a compromised ability to increase anti-oxidant defensive responses, and exaggerated depression of the ST segment on ECG (Kodavanti et al., 2000a). In addition, both strains of rats exhibited similar adverse reactions to ROFA exposure, including increased airway reactivity, focal lesions in alveoli and airways, as well as around airways and blood vessels of the lung, pulmonary inflammation and production of inflammatory cytokines. Thus, although the dose levels were extremely high compared to ambient particles, this experiment suggests that compromised animals are potentially more vulnerable to pollutant-associated oxidative stress and pulmonary vascular leakage than healthy animals. Generally similar results were obtained with an experiment using one-time intratracheal administration of high-dose ROFA or nickel, but not vanadium (Kodavanti et al., 2001).

Several toxicological studies report cardiac arrhythmias in compromised animals exposed to high-dose ROFA. Investigators exposed Sprague-Dawley rats (one group with pulmonary inflammation and hypertension from MCT pre-treatment and one control group) intratracheally to large doses of ROFA (0.25, 1.0, and 2.5 mg) and observed a variety of cardiac arrhythmias in both groups (Watkinson et al., 1998; Campen et al., 2000). However, the compromised group had more severe arrhythmias, including patterns indicative of inadequate cardiac oxygenation (myocardial ischemia) and conduction abnormalities (2^{nd} degree heart block), accompanied by substantial mortality rate in all exposure levels (about half of the compromised animals died). In a study of rats exposed intratracheally to several different kinds of particles (ROFA, volcanic ash, and resuspended ambient particles from Ottawa, Canada), ROFA induced significant pulmonary inflammation, bradycardia and arrhythmias in healthy rats, which were exaggerated in MCT-treated rats. MCT and SH rats exposed by inhalation showed similar, but less severe, effects. Older SH rats exposed to high dose ambient particles (2.5 mg intratracheally) also exhibited significant bradycardia and cardiac arrhythmias. The volcanic dust administration had no cardiac effects in any animal group (Watkinson et al., 2000).

Rats exposed to concentrated ambient particles (whose composition can vary from day to day) were found to exhibit various degrees of pulmonary inflammation (Kodavanti et al., 2000b). In these whole-body inhalation studies, involving exposure concentrations of 475 – 907 $\mu g/m^3$, the pulmonary responses, when they occurred, were generally modest, and the animals with chronic bronchitis fared slightly worse than the control animals. Thus, although these exposure conditions were found to cause injury and inflammation, the results were inconsistent, which may have been due in part to the relatively low metal content of these

particles (collected in Research Triangle Park, NC, a nonurban area). These results suggest that the very high-dose intratracheal experiments using toxic ROFA particles, for instance, may have limited generalizability to environmental exposures.

In a similar vein, Gardner et al. (2000) found increased blood fibrinogen levels in rats exposed only to the highest dose of ROFA particles by intratracheal instillation (8.3 mg/kg), but not at lower concentrations (1.7 and 0.3 mg/kg). Recognizing the limited statistical power of this investigation (six rats per exposure group), these results suggest that although animal models may help illuminate potential toxicological mechanisms, the necessity of using extremely high-dose exposures warrants a cautious interpretation.

Thus, animal studies using high-dose exposures by intratracheal administration and inhalation provide ancillary support for observations of pulmonary inflammation and cardiopulmonary toxicity in epidemiological and controlled human exposures. Such investigations bolster the biological plausibility of the human studies, but are nevertheless limited by uncertainties related to cross-species extrapolation and high-level exposures used.

7.8.5 Summary

In summary, recent research provides mechanistic support for a causal relationship between ambient PM and the cardiopulmonary morbidity and mortality consistently observed in time-series studies. Such support derives from clinical, epidemiological, and toxicological studies of a variety of pathophysiological events that could result in adverse cardiovascular outcomes. Localized airway inflammation and absorption of particles not only into the lung interstitium, but into the circulation, may result in systemic impacts, including effects on factors influencing blood coagulation, altered cardiac autonomic control, and recruitment of inflammatory cells from the bone marrow. Interestingly, most if not all of these events have been reported to occur acutely (within a day or less of exposure), and at least in the German MONICA study, several were observed to occur in concert in a subgroup of potentially vulnerable individuals. While the evidence is still fragmentary, it represents a dramatic advance from a few years ago, and begins to sketch a framework of biological plausibility for the time-series studies.

7.9 Causal Inference

This section deals with the evidence that the associations between both acute and chronic exposures to ambient PM and human morbidity and mortality represent causal relationships. The following criteria for causal inference are considered: (1) the consistency of the findings; (2) the coherence of the study results; (3) the likelihood that findings are due to chance; (4) the possibility that findings are due to bias or confounding; (5) temporal sequence of the associations; (6) the specificity of the findings; (7) evidence for exposure-response relationships; (8) strength of the associations; and (9) the biological plausibility of a causal associations. These are based on informal guidelines for causal inference described by Sir Austin Bradford Hill, as modified by other epidemiologists (Hill, 1965; Rothman, 1982).

7.9.1 Consistency of Results Among Different Studies

The consistency of results among scores of epidemiological studies provides substantial evidentiary support for causality. Several hundred studies, conducted among different populations on five continents over multiple time periods, have reported small, but consistently elevated risks of daily mortality and diverse measures of morbidity (such as hospital admissions and emergency department visits for cardiac and respiratory causes, exacerbation of asthma, increased respiratory symptoms, restricted activity days, school absenteeism, and decreased lung function). Though the principal study design has been time-series analysis, modeling approaches have differed substantially among investigators;

moreover, similar estimates of effect have been obtained with other study designs, including case-crossover and panel studies. The ranges of risk estimated in all these studies have been remarkably similar, despite the different PM source mixtures and size distributions, co-pollutant distributions, weather patterns, population characteristics (distributions of age, baseline health status, and access to health care) (See section 7.3, for example). Daily mortality and morbidity have also been linked with different measures of PM, as well, including TSP, PM10, PM2.5, the coarse fraction (PM10-PM2.5), black smoke, and ultrafine particles. It can be seen in Table 7.1 and sections 7.3 through 7.6 that, with few exceptions, there is a consistent tendency for point estimates of relative risk to be greater than unity. If these findings were due to chance, one would expect a more nearly equal distribution of point estimates of risk above and below unity. In general, consistency of results across scores of investigations offers one of the strongest arguments favoring a causal relationship (Ostro, 1993

7.9.2 Coherence of Results

Referring in particular to the time-series studies of mortality, Bates (1992) has argued that, if the PM-mortality relationship is causal, there should also be evidence of relationships between PM and health outcomes of lesser severity, such as hospitalizations, changes in lung function, and so forth, suggesting an ensemble of coherence among possible outcomes. This phenomenon has been observed in a number of areas throughout the world; perhaps the best illustration of such coherence in a given area are the studies undertaken in the Utah Valley. In addition to increases in PM-associated mortality, studies in this area have demonstrated statistically significant relationships between ambient PM and respiratory hospitalizations, decrements in children's lung function, school absenteeism, respiratory symptoms, medication use among asthmatics, increased heart rate and decreased heart rate variability among elderly individuals (Pope, 1996; Pope et al., 1999a, b). Finally, there are over twenty cities in which associations between PM10 and both mortality and hospital admissions have been reported.

7.9.3 Likelihood That the Findings are Due to Chance

Almost all the studies described in the previous sections showed increased risks of PM-associated morbidity and mortality, though these results are not all statistically significant. While the informativeness of testing for statistical significance has been the subject of lively debate in epidemiology for at least the past decade, this process does represent one of the conventional approaches to assessing the likelihood that study results might be attributable to chance. The purpose of significance testing is to compare the results observed with what would be expected to occur by chance if the null hypothesis of no effect or no relationship (e.g., between ambient PM exposure and daily mortality) were true. This assessment is usually based on comparison with a pre-designated significance level (usually 5%), which indicates a traditional, convenient cut-off value for assessing the likelihood of the results that could be expected to occur by chance. Thus, finding that the results are statistically significant represents a judgment that the results are not likely to be due to chance. Moreover, it should be noted that many of the results cited above are highly statistically significant, indicating that they could be considered extremely unlikely to be due to chance. However, these are still probabilistic assessments, and it is still possible that the results could be due to random variation.

Assuming the existence of a causal relationship, a variety of factors influence the calculations underlying an assessment of significance, including the size of the subject population, the numbers of events observed over the duration of the study interval, an appropriate specification of the model relating pollution to mortality or morbidity events, the extent of exposure measurement error, the degree of covariation among the pollutants and

meteorological variables (see confounding below), and other potential biases. Thus, the absence of statistical significance in a given study may indicate the lack of a real causal relationship, but may also reflect the influence of one or more of these factors.

7.9.4 The Possibility That Findings are Due to Bias or Confounding

In evaluating these results, one needs to consider confounding, information bias and selection bias. In the time-series studies that are population-based, selection bias is not an important issue. Rather the principal concerns regarding the validity of the results would be confounding and information bias, specifically the potential impact of misclassification of exposure.

Confounding occurs when the estimates of effect are distorted by an extraneous variable that is associated with both the exposure and outcome of interest, where that extraneous variable is not part of the causal pathway between the exposure and the outcome. In daily time-series analyses, any confounder would have to vary in concert with both the daily fluctuations in pollutant concentrations and with the health outcome. Thus, variables that one might intuitively consider as potential confounders, such as cigarette smoking patterns, are not relevant in this context. The principal potential confounders of concern in such studies are meteorological variables and gaseous co-pollutants such as ozone and sulfur dioxide, and possibly the presence of respiratory epidemics such as influenza.

Of the meteorological variables, temperature is probably the most important, as it has been demonstrated to have independent effects on a variety of health outcomes, including mortality. All of the time-series studies of PM and mortality cited in this report have controlled for temperature, or have at least examined whether temperature could be a confounder. Investigators have employed a variety of modeling approaches to assess the impact of temperature; some studies have undertaken sensitivity analyses to assess the likelihood that weather-related impacts were being inappropriately ascribed to PM (Samet et al., 1998; Pope and Kalkstein, 1996). The weight of the evidence indicates that the PM-associated health outcomes are not the result of confounding by temperature or other meteorological variables. In addition, similar estimates of PM-related effects have been obtained in cities with diverse climates and different seasonal relationships between PM and temperature. This issue is discussed in greater detail in section 7.3.

Respiratory epidemics, such as influenza, regularly occur in specific seasons (e.g., influenza generally is a winter phenomenon in the United States). To the extent that there is adequate control of seasonal meteorological influences in any given study, this should address potential effects of confounding by infectious disease. In addition, if PM-associated mortality or morbidity is also observed in other seasons in a given locale, this would indicate that respiratory infectious disease epidemics could not explain the association. In some instances it would be methodologically inappropriate to control for influenza, for example, if this outcome itself represents either one of the health outcomes of interest or can be considered part of the causal pathway for one of the health outcomes, such as exacerbation of asthma. Several studies have explicitly modeled infectious respiratory illness outbreaks in examining PM-associated health effects; these also indicated that the relationships could not be explained by seasonally concurrent epidemics (Braga et al., 2000).

Finally, there is the issue of confounding by gaseous co-pollutants, including specifically ozone, nitrogen dioxide, sulfur dioxide, and carbon monoxide. All of these pollutants have also been associated in time-series studies with daily mortality and a variety of other adverse health outcomes. Therefore, in the presence of strong correlations between any one or more gaseous pollutants with a PM metric within a given study, it may be difficult to disentangle their relative impacts. In some instances, particularly in studies outside of North America, measurements of co-pollutants were limited, and therefore the potential impacts of these

gaseous pollutants could not be controlled for in the analysis. The two principal methods to address potential confounding by gaseous pollutants are: (1) to examine PM effects in multiple locations in which there are different correlations between PM and the various gases; and (2) to include multiple (measured) pollutants in the regression model. Using the first method, if the PM coefficients are consistent from place to place in the presence or absence of a putative co-pollutant confounder, this suggests that the associations between PM and mortality or morbidity indices are independent of, and not confounded by, the other pollutants. In view of the plethora of epidemiological studies in diverse locations, some with high ozone or sulfur dioxide levels, and some with low concentrations of these pollutants, the evidence is compelling that PM effects cannot be explained away due to confounding by co-pollutants.

In a recent, large-scale application of the second method involving 90 U.S. cities, Samet et al. (2000a) sequentially tested the estimated effects of PM10 on daily mortality after each of the principal gaseous pollutants (ozone, nitrogen dioxide, sulfur dioxide, and carbon monoxide) was added to the regression model. These authors reported trivial or no change in the estimated PM10 coefficients when the other pollutants were included in the model. Similar results have been obtained in most of the studies that have examined PM10 and mortality, with few exceptions (e.g., Moolgavkar, 2000a). Other recent examinations of the problem of confounding by co-pollutants have also found little evidence that confounding can explain the associations between PM concentrations and adverse health outcomes (Schwartz, 2000a; Katsouyanni et al., 2001).

One other potential threat to validity of the results of epidemiological studies is information bias, particularly in the form of exposure measurement error. In this instance we are concerned with errors in measurement of PM exposures. Such measurement error is an inherent feature of epidemiological studies: given that pollutant concentrations vary over space and time, as do individuals' activity patterns, it is not possible to measure personal exposures to the important components of PM for large numbers of individuals. This is a multi-dimensional problem that could consist of the following components: (1) use of a PM metric that includes some "nuisance" particles that do not really contribute to health effects rather than the "true" components that are biologically active; (2) errors in measurement between the values recorded by ambient monitors and the true ambient levels, due to either instrument error or temporal-spatial variation, or both; (3) differences between aggregate ambient measurements and individual personal exposures; (4) differences between average personal exposures and true ambient pollutant levels; and (5) differences in the accuracy of measurement of co-pollutants, so that in multivariate regression models, those pollutants measured with greater accuracy and precision may spuriously appear to have a greater effect than they would if all were measured with equivalent accuracy and precision.

Typically the effects of measurement error tend to bias the results towards the null hypothesis of no effect – that is, the effects of PM on morbidity and mortality tend to be underestimated. There may be exceptions to this generalization, however. Recently the issues of measurement error in air pollution time-series studies were systematically reviewed, characterizing the errors in measurement as either classical or Berksonian in nature (Zeger et al., 2000). Berkson-type errors, an example of which is using aggregate rather than individual exposure data, do not produce biased regression coefficients. Zeger and colleagues (2000) suggest that in the usual case, time-series studies will tend to underestimate, rather than overestimate, pollutant effects. In the case of multi-pollutant models, differences in the monitoring accuracy and precision of pollutants may result in confounding, with the effects of a more poorly measured pollutant being transferred to one measured more accurately, but only when the pollutants or their errors in measurement (particularly the latter) are strongly correlated. When pollutant levels are strongly correlated, they generally should not be included in the same regression model, as this produces unstable and biased estimates of

effect. Zeger et al. (2000) suggest that the largest potential source of bias in measurement error is likely to be due to differences between ambient measurements and average personal exposures, which could occur if indoor sources produce particles of similar size and toxicity as outdoor local and regional sources. Taking the "best" data set available that would allow an examination of the magnitude of this kind of error (from the P-TEAM study in Riverside, CA), they found again that standard regression analysis will tend to underestimate the strength of the association between fixed-site monitoring data and adverse health outcomes (mortality, in this case).

Based on the above, it is possible that, in limited circumstances, particularly when multiple pollutants are measured with error, that some of the PM effect may be due in part to differential measurement error. However, it is reasonable to infer that in most situations, the results of the numerous time-series studies of PM-associated morbidity and mortality cannot be explained by information bias.

7.9.5 Temporality of the Associations

That a putative cause precede its effect(s) is a *sine qua non* for causal inference (Rothman, 1982). It is in this sense that this guideline for causal inference is typically used in epidemiology, and is clearly met in the ensemble of PM studies. In the time-series studies of morbidity and mortality, one typically finds significant associations between PM concentrations and adverse health outcomes with lags of zero to four days, with moving average concentrations occasionally demonstrating a slightly stronger association. Several studies examining "reverse lags" (i.e., with the health effects preceding the pollution measurements) have found no relationship.

However, a number of investigations have found statistically significant associations between PM concentrations and adverse health outcomes on the same day. For certain health outcomes, such as exacerbation of asthma, this could be explained mechanistically without much difficulty. For cardiovascular outcomes, including mortality, such short lags between exposure and outcome might appear problematic. Nonetheless, recent evidence suggests relatively rapid systemic responses to PM pollution that are consistent with the observations in the time-series studies (Gold et al., 2000; Pope et al., 2001 – see section 7.8 above).

7.9.6 Specificity of Effect

In the original formulation of the guidelines for causal inference, Hill (1965) expressed the notion that the basis for causal inference would be strengthened if an exposure led specifically to a single effect. The absence of such specificity does not necessarily negate the existence of a causal relationship – witness the protean manifestations of disease engendered by exposure to cigarette smoke. Nevertheless, it is intuitive that the more specific an association between an exposure and an adverse health outcome, the more likely it is to represent a causal relationship. Although PM exposures have been linked to a variety of adverse effects, the latter are circumscribed to effects on the respiratory and cardiovascular systems. Given our current understanding of the pathophysiology of inflammation, with both local (respiratory) and systemic effects, these are the organ systems that one would expect to be most strongly affected by exposure to particles. While many of the mortality time-series studies have examined impacts on total mortality only, a few have done comparative analyses of relationships with cardiac- and respiratory mortality and with mortality from all other causes (section 7.3). The results of these studies suggest that the relationship between PM exposure and mortality is relatively specific to those organ systems expected to be affected by such exposures. A similar pattern can be observed with time-series studies of hospitalizations (section 7.5).

7.9.7 Evidence for Exposure-response Relationships

As noted above, the data from most of the time-series studies discussed in this document clearly demonstrate statistically significant exposure-response relationships. The range of the PM-mortality coefficients is surprisingly narrow over a wide range of PM concentrations over time and across locations, indicating that, at least within the observable range in most metropolitan areas examined, this relationship is more or less linear (section 7.3). Generally, for morbidity outcomes that are more common than daily deaths, the magnitude of the associations are slightly greater, as one would expect if the relationships were causal.

7.9.8 Strength of Association

The relative risk (RR) estimates obtained in the epidemiological studies of morbidity and mortality are generally low, with virtually all estimates of effect less than two. RR estimates of this magnitude may weaken the evidence of causality, due to the possibility of uncontrolled confounding or other sources of bias producing the findings. However, small estimates of relative risk do not, in themselves, nullify the existence of a causal relationship. As indicated above, the potential threats to the validity of any given study (i.e., bias and confounding) are not likely explanations of the consistent findings of increased PM-associated risks of morbidity and mortality.

In addition, when either the outcome measures or the exposure metric are given greater precision, the estimate of effect increases, which, everything else held equal, increases the plausibility of the association. For instance, as indicated in section 7.3 and Tables 7.1 and 7.2, the risks of mortality associated with PM10 range from 0.5% to 1.6% per 10 $\mu g/m^3$ of PM10, while the likely range for PM2.5 is 1% to 2.5% per 10 $\mu g/m^3$. For cardiac and respiratory causes of death, the corresponding ranges per 10 $\mu g/m^3$ increase in PM10 concentration are 0.8% to 1.8% and 1.3% to 3.7%, respectively (Ostro et al., 1999a). This was highlighted in a recent publication from the Netherlands, in which specific causes of cardiorespiratory mortality were found to be more strongly related to PM10, than was total mortality (Hoek et al., 2001).

Thus, although the estimates of effect are low, they are consistently highly statistically significant, and increase in magnitude and precision with better specification of either the outcome or the exposure metric.

7.9.9 Biological Plausibility of the Associations

Biological plausibility is not necessary for causal inference from epidemiological studies, since it depends on the state of knowledge of ancillary disciplines. When present, however, supporting evidence from other scientific fields such as toxicology can strengthen the case for a causal association between an exposure and a disease outcome. A decade ago biological plausibility for a causal linkage of ambient PM with mortality or with multiple indicators of morbidity would have been purely speculative. Major recent advances in toxicology, clinical exposure studies, and epidemiological studies with intermediate endpoints suggest that effects observed in the epidemiological studies are likely to be initiated with inflammatory responses in the lung, which can have both local and systemic effects. Focal hyperdeposition of particles at airway carinas and in the respiratory bronchioles, may lead to localized particle concentrations substantially greater than what might be anticipated based only on an assessment of ambient concentrations. Particle mass or constituents may generate oxidative stress and inflammation. Inflammatory reactions in the airways may exacerbate pre-existing pulmonary disease, such as asthma or COPD, and may also result in systemic impacts. Potential mechanisms of toxicity are discussed in detail in section 7.8, especially those that might bear on cardiovascular events, including effects on blood coagulability and viscosity, as well as disturbances of cardiovascular autonomic control. While the picture is far from

complete, plausible biological mechanisms have been proposed and are the subject matter of active research.

7.9.10 Summary

The scientific evidence linking PM exposure to premature mortality and a range of morbidity outcomes appears to meet the generally accepted guidelines for causal inference in epidemiology (Hill, 1965). Much current research is now focusing on biological mechanisms in order to provide a more complete understanding of the effects of PM.

7.10Recommendations for Standards

This chapter presents the staff recommendations for the Board to consider in promulgating the PM Ambient Air Quality Standards (AAQSs) for California. The section begins with findings on the overall adequacy of the current standards for PM with respect to protecting the health of the public, including infants and children. It continues with recommendations for the pollution indicators, averaging times, forms, and concentrations adequate to protect public health.

The recommended concentrations for the PM standards should be based on scientific information about the health risks associated with PM, recognizing the uncertainties in these data. With this in mind, the numerous studies of PM-associated morbidity and mortality indicate that, within the concentration ranges reported, there is no identifiable "bright line" or threshold PM concentration for either short- or long-term exposures, below which health effects would not occur However, the Children's Environmental Health Protection Act [Senate Bill 25, Escutia; Stats. 1999, Ch. 731, sec. 3; Health & Safety Code section 39606(d)(2)] does not require setting a given AAQS at a level that ensures zero risk. Given the current state of the science, which is limited by the uncertainties of existing data sets and methods available to analyze the impacts of low-level exposures, it is not possible to set such standards for particulate matter. Rather, the statute requires a standard that "*adequately* protects the health of the public, including infants and children, *with an adequate margin of safety*." (Emphasis added)

The governing statutory language indicates that California's ambient air quality standards should also protect other vulnerable populations, in addition to infants and children, and the general public [(H&SC sections 39606(d)(2) and 39606(d)(3)]. This legislative directive is consistent with historical practice in California, where ambient air quality standards have been formulated to protect identifiable susceptible subgroups, as well as the general population. For instance, the one-hour sulfur dioxide standard was developed in order to protect the most sensitive recognized subgroup, exercising asthmatics. Nonetheless, even with standards tailored to shield vulnerable populations, there may be exquisitely sensitive individuals remaining outside the ambit of protection.

Both the Health & Safety Code (section 39606) and the federal Clean Air Act (section 109) refer to an adequate margin of safety, but no specific legislative definition of this term is provided. The concept of a margin of safety derives from the field of structural engineering, in which such margins of safety (or safety factors) are applied to design and construction specifications in order to prevent structural failures, which might otherwise result from variability in design, materials, or workmanship. The science of predicting health outcomes resulting from PM exposures is considerably less developed than the design considerations of structural engineering, making the notion of a margin of safety even more appropriate in setting ambient PM standards. An "adequate margin of safety" in standard-setting is generally understood to account and compensate for scientific uncertainty, as well as the lack of precise predictions regarding the health impacts of air pollutants on a multiplicity of potentially susceptible subpopulations. Some of the relevant uncertainties in this instance

would include, among others, potential health hazards that have not been identified, factors determining variability in response to PM among susceptible subpopulations, micro-environmental variability in PM exposure related to indoor penetration of PM, activity patterns, and geographic proximity to point and area sources. The incorporation of a safety margin has been recognized by the California Supreme Court as integral to the process of promulgating ambient air quality standards [See Western Oil and Gas Association v. Air Resources Board, 37 Cal.3d, 502 (1984)].

As described in the preceding chapters, using the current epidemiological data and analytic techniques, researchers have been unable to detect a level of PM exposure below which no adverse health effects would ever be expected to occur, which creates substantial uncertainties in the prediction of health impacts of low-level PM exposure. To the extent that health effects associated with ambient PM have occurred at relatively low levels of exposure, and that there is substantial inter-individual variability in response to environmental insults, it is difficult to promulgate any PM standard that will provide universal protection for every individual against all possible PM-related effects.

Nevertheless, taking into account the current knowledge regarding the health impacts of PM, the limitations of the scientific data and the methods available to analyze this data, as well as variability in real-world exposures and human responses to PM, we have operationalized the concept of an adequate margin of safety by recommending multiple standards that, in combination, should protect nearly all of the California population, including infants and children, against PM-associated effects throughout the year. We have reviewed the available scientific literature and proposed standards that, when attained, will avoid exposures that have been reported to produce health effects in published studies.

7.10.1 Adequacy of Current California AAQS for PM in Protecting Public Health

The extensive epidemiologic data on the health effects of PM, supported by clinical and toxicological evidence, suggests that, in combination, the current annual average standard for PM10 of 30 $\mu g/m^3$ and the 24-hour average of 50 $\mu g/m^3$ do not offer sufficient protection of public health, including that of infants and children (ARB, 2000). Chronic exposures to ambient PM appear to be especially deleterious, and may influence responses to shorter-term (usually daily) exposures. Nonetheless, as reviewed in the above sections, there are strong and consistent associations between daily exposure to PM (measured as PM10, PM10-PM2.5, or PM2.5) and a range of adverse outcomes, including premature mortality, hospital admissions, emergency room and urgent care visits, asthma exacerbation, chronic and acute bronchitis, restrictions in activity, school absenteeism, respiratory symptoms, and reductions in lung function. These studies have been conducted in a wide range of cities on five continents, with differing PM sources, climates, seasonal patterns, co-pollutants, and population characteristics. The more severe outcomes are experienced primarily by the elderly and by people with pre-existing chronic heart or lung disease. However, several epidemiological studies suggest that children under age five may also experience serious adverse outcomes from exposure to PM10, including premature mortality and hospitalization for respiratory conditions (See section 7.7.3.2).

As indicated in section 7.3, many of the epidemiologic studies demonstrate associations between PM10 and the risk of premature mortality. The extent of early mortality or life shortening may be from days to years. Because the exposure-response relationship between ambient PM and daily mortality appears to be linear with no identifiable threshold, it is possible that associations between PM10 and adverse health effects may occur throughout the range of concentrations reported in each study. However, these occurrences are intuitively more likely when particle levels are elevated, especially in the upper portion of the PM distribution. Although we cannot know at what concentration health impacts of PM

exposures begin, for purposes of these recommendations, the staff has identified the mean PM10 concentration in any given study as representing a likely minimum effects level. This approach is consistent with that taken in the recommendation for the California 24-hour standard for sulfur dioxide. At higher mean concentrations however, the probability increases that adverse health outcomes will occur below the mean, in contrast, as concentrations decrease, the associated risks incorporate a larger range of uncertainty (see section 7.3). In view of the current state of the science, it is not possible to identify specific levels at which no PM-related adverse effects will occur; however, the strength of the association of interest in any given study is likely to be greatest at the mean PM concentration.

Analyses of mortality (summarized in sections 7.3 and 7.4, Tables 7.1 and 7.7, as well as Figure 7.1) and morbidity (summarized in sections 7.5 and 7.6) demonstrate that numerous epidemiological investigations have found associations of adverse health effects with PM10 when the long term (i.e., months to years) study mean concentrations are at or below the annual average standard of 30 $\mu g/m^3$. Both of the studies reporting associations between long-term exposure and mortality have mean concentrations of PM10 or its equivalent at or below the current annual average standard in California (Pope et al., 1995; Dockery et al., 1993). In the report by Dockery et al. (1993), the long-term average for PM10 ranged from 18 to 46.5 $\mu g/m^3$ in the six cities studied, with an overall mean of 30 $\mu g/m^3$. A stronger association was found for PM2.5, which ranged from 11 to 29.6 $\mu g/m^3$, in which the overall mean concentration was 18 $\mu g/m^3$. Likewise, Pope et al. (1995) reported associations of mortality with PM2.5 in the analysis of the American Cancer Society cohort, with an overall study mean of 20 $\mu g/m^3$. If the ratio of PM2.5 to PM10 is approximately 0.65, as it was in many urban areas included in the American Cancer Society study, this would convert to a PM10 average of about 28 $\mu g/m^3$. Therefore, it appears that the current annual ambient standard does not incorporate an adequate margin of safety against the occurrence of mortality associated with long-term exposures.

Numerous epidemiological studies have demonstrated small, but consistent, relationships between health outcomes and daily variations in PM concentrations. It should be noted, however, that the impacts associated with the underlying chronic exposure cannot be fully separated from the health effects attributed to daily peak PM10 or PM2.5 exposures. The notion that chronic exposures exert a major influence on health outcomes is reinforced when one examines the mortality risks associated with daily versus chronic exposure. Most of the time-series studies demonstrate a 0.5 to 1% increase in total mortality per 10 $\mu g/m^3$ change in PM10 (section 7.3). In contrast, based on the American Cancer Society cohort study, the estimated mortality effect of chronic PM10 exposure is in the range of four to seven percent per 10 $\mu g/m^3$ change in the long-term average of PM10 (Pope et al., 1995; section 7.4). These results suggest that longer-term exposures (i.e., several days to several years) account for a substantial fraction of PM10-related mortality.

While relationships between health outcomes and daily exposure measurements have been identified through time-series analysis, it is not possible to completely disentangle the influence of low-level chronic exposures. Nonetheless, recognizing the limitations of the existing epidemiological data, the literature suggests that, when long-term mean PM10 or PM2.5 concentrations are within the ranges reported in the published literature, it is possible to document a variety of adverse health outcomes in relation to day-to-day PM fluctuations.

Long-term mean PM10 levels near and below that of the current ambient California 24-hour standard have been consistently linked with respiratory symptoms and exacerbations of asthma in children. Although there are a few studies linking infant mortality to ambient PM, it is not clear, based on existing data, whether infants and children are more or less susceptible to PM-associated premature mortality than older adults with chronic heart and lung disease.

For example, it is possible that children who die of sudden infant death syndrome may have physiological abnormalities that render them unusually susceptible to the effects of PM; however, the database of published studies is too sparse for causal inference. As indicated in section 7.7.3.2, most studies of infant mortality consist of either: (I) cross-sectional study designs, in which statistical control for all potential confounders is difficult and causal inference problematic, or (ii) time-series studies conducted in cities outside of the United States in which the PM levels are much greater than in California. In the latter group of studies, factors related to infant nutrition, health care and exposures may not be generalizable to the United States. Given the current state of knowledge, it is uncertain whether infants and children represent an additional susceptible subpopulation with respect to air pollution-associated mortality at current ambient concentrations of PM. However, childhood respiratory morbidity does appear to be consistently linked with different measures of PM, within the same concentration ranges as those associated with mortality in adults with chronic heart and lung disease (See sections 7.3 and 7.5).

The voluminous published data suggest that, taken together, the current PM10 AAQSs are probably not adequately protective of public health, particularly for the elderly and individuals with pre-existing heart or lung disease. In addition, the available evidence suggests the need for new standards for PM2.5. From the perspective of public health protection, the principal shortcoming appears to be related to chronic PM exposures, though short-term effects on morbidity and mortality are also clearly important. The quantitative benefits assessment (section 9) suggests that significant mortality and morbidity benefits will result from reducing population exposures to PM.

7.10.2 Recommended Pollution Indicators

The scientific evidence suggests a need for standards to encompass fine particles as well as PM10. We therefore recommend that the PM10 indicator be retained and that both long- and short-term standards for PM2.5 be promulgated as well. These recommendations are predicated on the following rationale:

- PM10 and PM2.5 are both associated with a wide range of serious adverse health outcomes, including premature mortality, hospitalizations, and asthma exacerbation, among others.
- Dosimetry studies indicate that both fine and coarse particles deposit throughout the respiratory tract (see section 7.1). Fine particles are more likely to deposit in the alveolar region (or gas exchange zone) and may initiate inflammatory responses, with both local and systemic effects. Coarse particles (PM10 – PM2.5) can also deposit in significant quantities in the conducting airways and, to a lesser extent, in the gas exchange region of the lung. Moreover, multiple studies in which the health impacts of PM2.5 and coarse mode have been examined have reported adverse effects associated with both metrics.
- Particles larger than 10 μm in median aerodynamic diameter have limited deposition in either the alveolar or tracheobronchial region, but rather deposit preferentially in the nose and oropharynx. The health impacts related to particle deposition in the ET region have not been extensively explored. Therefore, staff does not recommend an ambient air quality standard for particles larger than 10 μm.
- Ultrafine particles (particles with aerodynamic diameters between 0.001 and 0.1 μm), which can deposit in significant quantities throughout the respiratory tract, have been linked with serious health impacts, including premature mortality and asthma exacerbation. There is a small but growing toxicological database suggesting that ultrafine particles may be more toxic, on a mass basis, than fine particles of similar composition. However, there are few epidemiologic studies of ultrafine particles and findings are mixed.

Therefore, there are insufficient data available to judge whether or not an ambient air quality standard for ultrafine particles is needed. Staff does not recommend an ambient air quality standard for ultrafine particles at this time.

- While recent toxicological research suggests potentially important roles for transition metals (e.g., iron, nickel, or vanadium) and PM-associated organic compounds in PM toxicity, there is insufficient evidence to develop ambient air quality standards for metals or any other specific chemical constituents of PM10 or PM2.5, with the exception of sulfates (see below). Therefore, staff does not recommend promulgating any other ambient air quality standard for any specific constituent of either PM10 or PM2.5. Ambient concentrations of most of the identified fine particulate constituents of potential concern, including sulfates, particulate acids, metals, and organic compounds, will be reduced by control strategies targeting PM10 and PM2.5 mass.

- Serious health effects have been associated with exposure to ambient sulfates, particularly in areas rich in strongly acidic sulfates, such as the eastern United States and Canada (See sections 7.3, 7.4, 7.5 and 7.6). The results of such studies, however, have not been as consistent as for PM10, PM2.5 or the coarse fraction. Some studies (Gwynn et al., 2000) suggest that particle-associated hydrogen ion (H^+) and strong acidic sulfates are associated more with respiratory effects than other particle metrics, including PM10. However, in other studies, sulfates are highly correlated with the fine mode in which they predominantly occur, such that independent effects of these correlated co-pollutants cannot be reliably estimated. In a third set of studies, no association was reported for sulfates or strong particle acidity, while associations were found for PM10 (for example, Lippmann et al., 2000, Schwartz et al., 1994). In contrast to the results of some of the epidemiological studies, controlled exposure studies involving high levels (up to 1,000 $\mu g/m^3$) of strongly acidic sulfates have demonstrated little, if any, effect on volunteer subjects, including those with asthma (e.g., Aris et al., 1991). Though daily sulfate excursions in epidemiological studies have been linked with a variety of adverse health events, the nature of the study data does not allow for segregation of outcomes related to chronic low-level exposure from those associated with acute (daily) elevations in sulfate concentrations. Thus, though the mean concentrations of some multi-year studies are lower than the current 24-hour sulfate standard in California (Burnett et al., 1994; Gwynn et al., 2000), these do not directly address the adequacy of the current 24-hour sulfate standard because it is difficult to separate the impact of a single 24-hour exposure. In this light, staff believes that the current scientific database is insufficient to use for revision of the existing sulfate standard.

  In California, acidic sulfates (principally sulfuric acid and ammonium sulfate) constitute a small fraction of the PM mass relative to the areas in which sulfates have been found to be associated with adverse health impacts. For instance, in Long Beach, where the fixed-site monitor consistently shows the highest sulfate levels in the South Coast Air Basin, sulfates constitute about 13% of PM10 mass and 22% of PM2.5 mass on an annual basis, and about 16% of the maximum 24-hr PM10 mass (15 $\mu g/m^3$ sulfates/93 g/m3 PM10) and 21% of the maximum PM2.5 mass (13 $\mu g/m^3$ sulfates/61 μg/m3 PM2.5), respectively. In the San Francisco Bay Area and in Bakersfield, the percentages are much lower (ARB, 1994). In the ongoing Children's Health Study in Southern California, data on sulfates have been collected, but not yet analyzed as predictors of children's respiratory morbidity or lung function growth and development. According to ARB staff, these data should be analyzed over the next couple years.

  In general, sulfates detected in California are less strongly acidic than those commonly found in the eastern United States and Canada. Though a time-series study linked sulfate

concentrations in 1978-79 in Azusa, California with respiratory symptom reporting in adults, ambient levels during that study period exceeded the standard (Ostro et al., 1993). Sulfate concentrations in California have been lower, typically far lower, during the past few years than the level of the existing standard. Although a mortality time-series study undertaken in Santa Clara County (1989-1996) involving very low 24-hour average sulfate values (mean = 1.8, range 0-7.9 g/m^3) suggests an association with daily respiratory mortality, staff believes this finding can be attributed principally to the strong covariation of sulfates with PM2.5 (Fairley, 1999). Based on an assessment of current scientific evidence and ambient air quality data, staff believes that exposures to sulfates in California do not appear to pose health risks distinct from or greater than those associated with exposures to particulate matter generally. In view of the mixed evidence in the sulfates health effects literature, the paucity of recent data examining sulfates and health in California, the low likelihood of health risks in relation to ongoing trends in sulfate emissions and ambient levels, staff recommends the current standard be retained until the next review of the PM standard.

In the review of the adequacy of the California AAQS to protect public health mandated by the Children's Environmental Health Protection Act (ARB, 2000), much of the evidence regarding the health impacts of sulfates was based on considerations of the PM epidemiology. Revisions of California's PM standards as recommended (below) will likely further reduce sulfate concentrations. In addition, based on discussions with ARB staff, the differences in sulfate composition and levels between California and the eastern United States are sufficient for OEHHA staff to recommend further studies in California prior to a full review of the sulfate standard. In particular, OEHHA staff recommends analysis of the sulfate data in relation to health indicators in the Children's Health Study, as well as time-series analyses of health outcomes and daily sulfate data being collected at the two California particulate matter Supersites in Los Angeles and Fresno. OEHHA recommends that ARB ensure that these analyses be conducted in such a manner as to provide optimally useful data for a full review of the sulfate standard.

- PM2.5 can infiltrate directly into residences, with greater penetration than the coarse fraction, and therefore individuals are likely to have more consistent indoor exposure to ambient PM2.5 than to the coarse fraction. Nevertheless, the coarse fraction also demonstrates substantial indoor infiltration, particularly in older buildings, or those in which windows or doors are kept open. Evidence from studies in California, indicate that 75% of indoor PM2.5 and 65% of indoor PM10 may originate outdoors (Ozkaynak et al., 1996b; see Chapter 6). Therefore, outdoor, ambient concentrations of PM2.5 and PM10 will play a significant role in total, personal exposure.

- Fine and coarse particles, in general, originate from different sources and have different lung penetration and deposition characteristics, but are both linked to adverse health effects. In most California cities, mobile sources are a significant source of PM10. In these cities, there are strong daily correlations between PM2.5 and PM10 throughout much of the year, such that a substantial fraction of PM10-associated health impacts can be reasonably ascribed to PM2.5. In some air basins, such as the San Joaquin Valley during the winter, PM10 concentrations are clearly dominated by the fine fraction.

- In contrast, PM2.5/PM10 ratios are lower in many parts of California than those observed nationally (Chapter 6). In some parts of the state, particularly in the inland air basins in Southern California, high PM10 concentrations are driven by the coarse mode. However, at this time, the current research database regarding coarse particles' health impacts is not as well developed as that for PM10 (or PM2.5). Therefore, staff recommends that PM10 standards be used as a basis for protection from exposure to coarse particles.

Taking into account all of the above factors, therefore, staff recommends the Air Resources Board promulgate new annual standards for PM10 and PM2.5, and a new 24-hour average standard for PM2.5, while retaining the existing 24-hour standards for PM10 and sulfates.

7.10.3 Averaging Times and Forms

The current PM10 AAQSs for California include both an annual standard based on the geometric mean concentration, and a 24-hour averaging time, not to be exceeded during the calendar year. These joint standards were developed to protect the public from both long-term and short-term exposures. Studies published since the California PM10 AAQSs were developed in the early 1980s support earlier findings and report associations between adverse health outcomes and both long-term (i.e., a year or longer) and short-term (i.e., from less than one day to several months) exposure to both PM10 and fine particles. Therefore, staff proposes standards using annual averages for PM10 and PM2.5, and 24-hr averages for PM10, PM2.5, and sulfates. The foundations for the annual averages are relatively straightforward, as explained in the subsections below. Identifying shorter-term average standards based on the existing epidemiological database is somewhat more difficult conceptually, due principally to the linear, nonthreshold nature of the relationship of ambient PM and adverse health effects, and somewhat less to the intermingling of effects related to chronic and acute exposure. While there is evidence of health effects associated with other averaging times (e.g., 4-hour and multi-year averages), staff believes that proposed averaging times will provide a satisfactory basis for setting PM standards and directing subsequent pollution control efforts.

Attainment of the annual standards described below will shift the current distributions of PM10, the coarse fraction, and PM2.5 to levels substantially lower than currently exist. Therefore, peak 24-hour averages of these particle measures will also decline. This implies that the current 24-hour average standard for PM10 should be exceeded in most air basins less frequently than today. However, data developed by ARB staff indicate that even if the proposed annual PM10 and PM2.5 standards are attained, some parts of California will sporadically experience significant elevations of one or both particle metrics into ranges associated with both morbidity and mortality. Therefore, short-term standards will function primarily to address intermittent short-term, seasonal exceedances (e.g., from residential wood combustion during the winter holiday season or prolonged summer temperature inversions) that might occur in air basins otherwise in attainment with the annual averages.

For the annual averages, OEHHA staff recommends using the arithmetic rather than the geometric mean because the former is: (1) more directly related to cumulative exposure; (2) more sensitive to repeated peak concentrations; and (3) more consistent with other annual standards. For the 24-hour standards, OEHHA staff recommends a “not to be exceeded” standard. The rationale for the latter is related to providing a margin of safety in the recommendations and is detailed below.

7.10.4 Recommended Concentrations

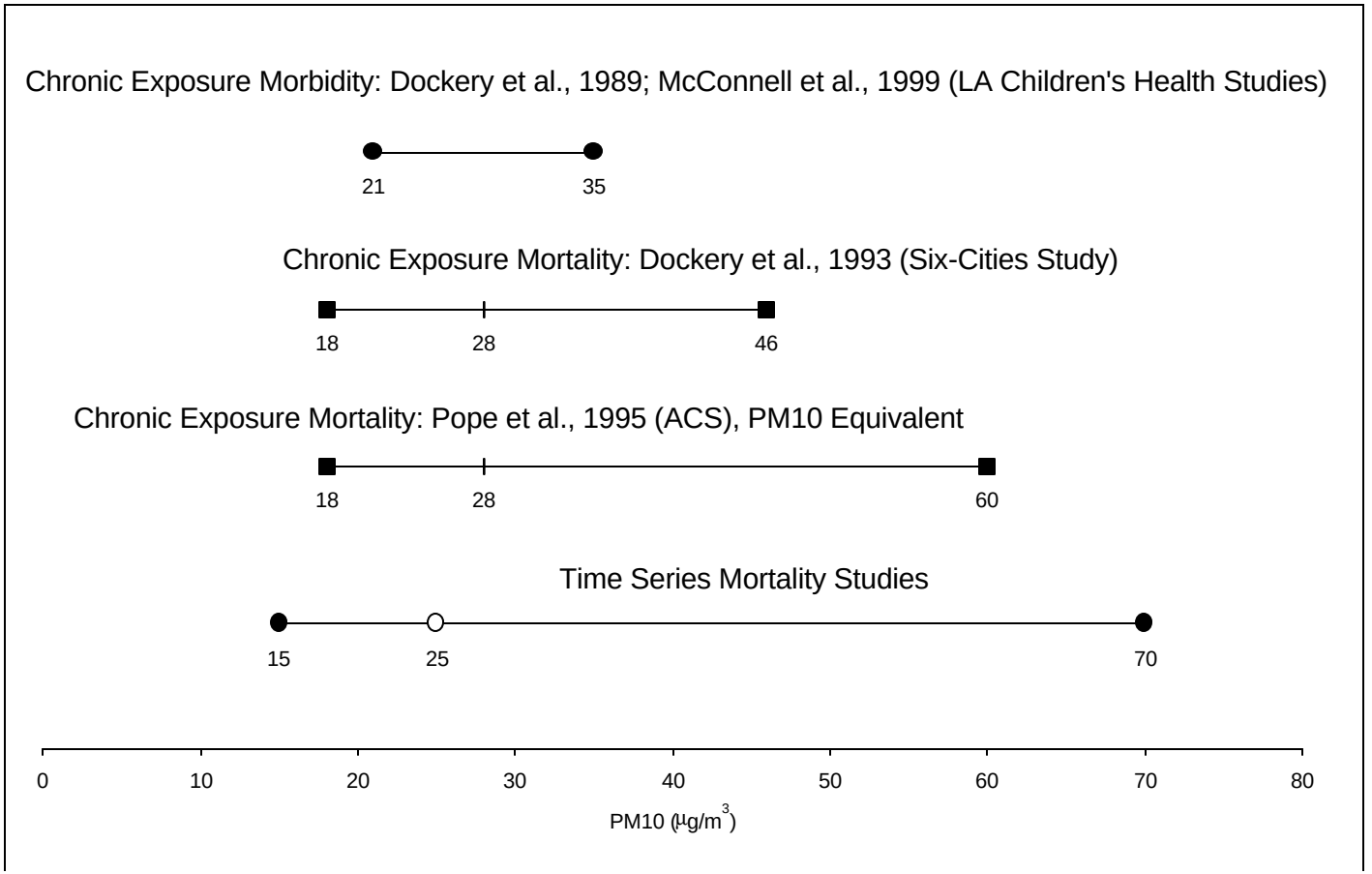
Although individual epidemiological studies are subject to some uncertainty, particularly with respect to exposure assessment, the overall body of evidence (including toxicologic, dosimetric and human clinical studies, in addition to the epidemiological investigations) particularly the consistency and coherence of results, provides compelling evidence of causal relationships between exposure to ambient PM and a variety of adverse health outcomes (See section 7.9). These studies provide a sound, scientific basis for the establishment of standards for both PM2.5 and PM10.

Multiple indicators of morbidity have been associated with exposures to several ambient PM, including hospital admissions, emergency room visits, exacerbation of asthma, work loss,

school absenteeism, bronchitis and respiratory symptoms, and changes in lung function. However, the choices of levels for the annual average standards described below are based primarily on studies of PM-associated mortality. The rationale for this choice is as follows: In the opinion of OEHHA staff, mortality is the most serious of all the health events associated with exposure to PM. PM-associated mortality has been observed at long-term average ambient concentrations comparable to those at which morbidity outcomes have been detected in other populations (See sections 7.3 – 7.6, Figure 7.5), which suggests that it would be reasonable to base the standards principally on studies involving mortality. To our knowledge, there is no evidence that morbidity effects would occur at PM concentrations lower than those associated with increased risks of mortality. This may be due to the different populations at risk examined in the various studies. That is, associations between 24-hour averages and mortality have been detected primarily in the elderly, who have a high prevalence of chronic cardiac and respiratory disease. In contrast, time-series or panel studies of children, who are not at high risk of mortality, have examined a variety of respiratory morbidity outcomes in relation to daily changes in PM. Though the initiation of biological reactions may overlap (e.g., airway and alveolar inflammation), the downstream pathophysiological consequences could vary by age, pre-existing genetic and acquired chronic conditions and co-morbidity, and so forth. Interestingly, there does not appear to be a gradient of exposure concentrations related to increasing health outcome severity. Thus, standards premised on providing protection against mortality should also, *a fortiori*, protect the public, including infants and children, against the occurrence of morbidity outcomes.

To the extent that the annual standards for PM10 and PM2.5 are attained, the distributions of 24-hour and other short-term averages of PM10 and PM2.5 will shift downward markedly throughout the year. The likelihood of adverse health events occurring after acute exposures will also therefore be substantially reduced. Nevertheless, there may well be areas that will attain the annual PM standards, yet still experience seasonally high PM excursions associated, for instance, with prolonged winter air stagnation combined with residential wood combustion or with summer temperature inversions. The plethora of time-series and panel studies cited in this document make it clear that short-term elevations of PM are associated with increased morbidity and mortality, though again, the impacts of the ongoing chronic PM exposure have not been identified. Therefore, though downward revisions to the annual PM standard will enhance protection of the health of the public, including infants and children, it is appropriate to limit shorter-term PM exposures, as well.

Figure 7.5 PM10 Range of Long-term Mean Concentrations Observed in Epidemiological Studies



= Range of concentrations in a given study

♦ = Study mean concentration

● = Range of study mean concentrations among multiple studies

= Lower end of concentrations that may be more relevant to California

7.10.4.1 Annual Standard for PM10

Considering the weight of evidence from the literature reviewed in prior sections, staff recommends that the annual average standard for PM10 be revised from 30 to 20 $\mu g/m^3$. Consideration of an annual standard at this level would place significant weight on the studies of mortality related to long-term PM exposure using the Harvard Six-Cities data (Dockery et al., 1993) and the American Cancer Society cohort (Pope et al., 1995), both reanalyzed by Krewski et al. (2000). In the study by Dockery et al. (1993), the long-term average for PM10 ranged from 18 to 46.5 $\mu g/m^3$ in the six cities, with an overall mean of 30 $\mu g/m^3$. Visual inspection of graphs of this study's results suggests a continuum of effects down to the lowest levels, with no evidence for a threshold (recognizing that it would be difficult to detect a threshold graphically in this set of six data points corresponding to the six cities). However, the city with the lowest long-term average PM10 concentration (Portage, WI) was, for purposes of analysis, designated as the reference category, against which the other cities were compared. In other words, it was assumed in the analysis that there was no increase in risk in this city. Thus, it would *not* be appropriate to infer, for standard-setting purposes, that PM-related effects on mortality occurred (or did not occur) at the long-term mean PM10 concentration of 18 $\mu g/m^3$ in Portage. In addition, while there appears to be a graphic exposure-response relationship by city, no clear increase in the risk of mortality is evident in Topeka, KS (which had a long-term annual PM10 concentration of 26.4 $\mu g/m^3$) relative to Portage. Finally, the relevant periods of exposure associated with long-term effects are unknown (other than those likely to be associated with short-term exposures within each year). In the absence of better information, it is reasonable to select the mean long-term PM10 level as a starting point for recommending the annual standard. In the Six-Cities study, the mean long-term PM10 level was 30 $\mu g/m^3$.

Likewise, Pope et al. (1995) reported effects on mortality associated with PM2.5, but not PM10, in the analysis of the American Cancer Society cohort, with an overall PM2.5 study mean of 20 $\mu g/m^3$. The recent re-analysis of the ACS study also suggests associations of mortality with long-term exposure to PM2.5, but not PM10 (Krewski et al., 2000). If one assumes that fine particles are driving the associations between PM and mortality in the ACS study, and that the ratio of PM2.5 to PM10 is about 0.65 for most of the urban areas included in that study (see Chapter 6), this would convert to an overall long-term average PM10 concentration of 28 $\mu g/m^3$.

Several investigations, including the Children's Health Study (McConnell et al., 1999) and the Harvard Six-Cities Study (Dockery et al., 1989), have also reported associations between long-term PM exposures and morbidity outcomes, including bronchitis, exacerbation of asthma, and reductions in lung function (see section 7.6). In these studies, the long-term (one- or multi-year) mean PM10 concentrations ranged from about 21 to 35 $\mu g/m^3$. Some of the morbidity studies, however, may be capturing the effects of exposure to multiple pollutants. For instance, in the Children's Health Study, the associations of adverse health outcomes with PM10 and PM2.5 could not be statistically disentangled from the co-pollutants NO_2 and acid vapors. Therefore, selection of a target concentration of 20 $\mu g/m^3$ puts greater likelihood on a PM-specific effect in these morbidity studies, and provides a margin of safety, assuming that there may be interactions among co-pollutants.

As noted above, the epidemiological studies of daily exposure and mortality have reported long-term mean or median PM10 concentrations from 14 to 115 $\mu g/m^3$ (see Table 7.1 and Figure 7.1). These studies examine short-term fluctuations in air quality in relation to daily changes in mortality over intervals ranging from months to years. The degree of uncertainty regarding the results generally decreases as the average or median concentration increases.

As can be seen in Figure 7.2, most of the studies that have long-term means or medians below 25 $\mu g/m^3$ have point estimates suggesting an association with PM10, but the confidence intervals tend to be wide and include the null value, indicating weaker, more uncertain associations. The annual averages of these short-term exposure studies are relevant, since associations are observed throughout a wide range of exposures and not only at the extreme values. In addition, some of the PM-associated mortality captured in the cohort studies described above would include the modest increments in short-term risks reported in the time-series studies, recognizing that larger long-term increments in risk appear to be related more to chronic than to short-term exposures. Finally, all of the time-series studies conducted at these lower concentrations were undertaken outside California and the United States. Studies more relevant to California (i.e., those conducted in California or other parts of the United States) reported long-term PM concentrations in the range of 25 to 35 $\mu g/m^3$ (see Table 7.1). Consideration of a standard of 20 $\mu g/m^3$ would, therefore, provide a margin of safety by placing significant weight on some of the time-series studies conducted outside of California and the U.S. This recognizes the generalizability of the results of these studies, although the sources and mix of PM constituents, the underlying population health characteristics, and the exposure patterns may differ from those in California. A standard set at 20 $\mu g/m^3$ would protect against mortality effects related to long-term exposure in adults and morbidity effects (such as acute bronchitis in children). The quantitative benefits assessment (section 9) suggests that attainment of this standard could result in the avoidance of an estimated 6,500 (95% CI=3,200-9,800) cases of premature mortality per year associated with the difference between this proposed level and the current annual averages of ambient PM10 concentrations throughout California (a population-weighted average exposure of 33.1 $\mu g/m^3$).

7.10.4.2 24-hour Average for PM10

Staff recommends that the 24-hour average for PM10 of 50 $\mu g/m^3$, not to be exceeded, be retained. If the recommendations for new 24-hour and annual standards for PM2.5 are adopted, this standard would offer protection primarily against peak concentrations of coarse particles in areas that otherwise attain the annual standard for PM10. For many urban areas in California, attainment of the annual standards will mean infrequent PM excursions, which would typically be associated with seasonal air stagnation or with wind events in desert or semi-arid areas. Thus, the 24-hour standard is intended to prevent occasional elevated PM10 levels. Staff believes that the existing 24-hour PM10 standard proscribing any single day concentration above 50 $\mu g/m^3$, in concert with attainment of the 24-hour standard for PM2.5 and the annual average standards for PM10 and PM2.5, would provide substantial protection of public health, including that of infants and children, as described below.

The 24-hour PM10 standard was first promulgated in California in 1983, based primarily on an analysis of daily mortality in London in relation to changes in PM. At that time, there were no epidemiological studies in which PM10 had actually been measured. Rather, critical PM10 concentrations had estimated from other PM metrics, including TSP and British Smoke. Since then, a voluminous literature has appeared linking fluctuations in short-term or daily measurements of PM10 with a variety of adverse health outcomes, as reviewed in sections 7.2, 7.3 and 7.5. Complemented by recent toxicological and controlled human exposure studies, the epidemiological foundation linking variations in ambient PM10 and daily morbidity and mortality has been firmly established.

Nonetheless, translating the results of these epidemiological studies into a short-term standard remains somewhat problematic. As noted in prior sections, multi-city analyses in Europe and the United States suggest exposure-response relationships between daily

variations in ambient PM10 and fluctuations in cardiopulmonary mortality and other health effects that are essentially linear and without an observable threshold. To the extent that this is an accurate characterization of PM10-mortality associations, and that the latter represent causal relationships, there is little guidance on where to draw a "bright line" in recommending a short-term standard. Moreover, in time-series studies segregation of the influence of chronic low-level exposures on individual susceptibility to daily PM elevations remains problematic. Cumulative exposures over several days or longer, rather than during a single 24-hour period, may represent a more relevant time frame of exposure. Consistent with this hypothesis, numerous epidemiological studies report morbidity or mortality effects of greater magnitude associated with multi-day moving averages compared with single-day lags (Hajat et al., 2001; Schwartz, 2000b; Schwartz et al., 1993; Pope et al., 1992). Nevertheless, as described above, 24-hour average PM10 concentrations have been consistently associated with increased daily mortality and morbidity.

Recognizing the limitations of the epidemiological data available for standard-setting purposes, OEHHA recommends retention of the 24-hour standard in consideration of the following factors: (1) the apparent linearity of dose-response; (2) the greater uncertainty of effects at the lower concentrations; (3) the paucity of epidemiological data documenting the impact of a single 24-hour exposure at low ambient (i.e., non-occupational) concentrations; (4) the dominance of the effects associated with chronic exposures and the impact of chronic exposure on the response to short-term elevations in PM concentration; (5) the likelihood of effects occurring at concentrations above 50 $\mu g/m^3$ and (6) the interrelationships of alternative averaging times.

7.10.4.2.1 Linearity of Dose-Response

As discussed above (section 7.3.5), time-series studies of morbidity and mortality indicate that the exposure-response relationships for 24-hour average PM exposures are linear and show no evidence of a threshold. The latter observation makes it difficult to identify where a "bright line" representing a single-day 24-hour PM10 standard should be drawn. The historic rationale for a 24-hour standard was the presumption that significant health effects occurred only on high concentration, "episodic" days or that high pollution days generated disproportionately greater and more severe adverse health outcomes. In general, the notion that episodic peaks alone are responsible for adverse effects ignores the potential role of chronic low-level exposures, which may predispose individuals towards greater susceptibility to elevated PM concentrations. In addition, there is little, if any, evidence that the exposure-response relationship becomes steeper at higher ambient concentrations; rather, the data generally indicate a linear exposure-response relationship.

7.10.4.2.2 Greater Uncertainty at Lower Concentrations

Epidemiological studies of short-term exposure and mortality have reported mean or median PM10 concentrations ranging from 14 to 115 $\mu g/m^3$ (see Table 7.1 and Figure 7.1). As can be seen in Figures 7.2 and 7.3, however, greater uncertainty about the effects exists as one moves to studies with lower concentrations. The greater uncertainty may be due to fewer health impacts associated with exposure to lower concentrations as well as other factors, including errors in exposure measurement, confounding by co-pollutants, and the chemistry of the particle mixture. Other uncertainties related to extrapolating the epidemiological findings from many of the daily exposure studies to California may result from differences in factors such as weather, housing stock, and population characteristics. Therefore, retention of the existing 24-hour standard acknowledges the uncertainty in applying the underlying studies with relatively low PM10 levels to urban and suburban populations in California.

7.10.4.2.3 Impact of Single 24-Hour Exposures at Low Concentrations

Exposures of 24-hours duration occur "on top of" consistent chronic low-level exposures to PM. The effects of long-term exposure to PM, as described in section 7.4, have been documented in several carefully conducted studies using a prospective cohort design. These studies incorporate effects associated with both short-and long-term exposures (although they may not include all of the impacts associated with mortality displacement). Basically, for these study effects to be observed, individuals must be continually moving into a "risk pool" from a non-risk or lower-risk status over time. Long-term exposure to PM subjects people to an increased risk (i.e., moves then into the "risk pool") of mortality from cardiovascular disease, whether or not their deaths are ultimately associated with a recent "acute" exposure to PM (Schwartz, 2001a; Kunzli et al., 2001). While acute daily exposures appear to exert an independent effect on mortality and morbidity, the influence of a single 24-hour exposure at a concentration relevant to the PM standards, absent any other exposure to PM, has not been (and probably cannot be) determined epidemiologically. This would require observance of weeks or months of exposure to very low background levels of PM followed by a single day peak exposure. Even for individuals exposed experimentally in chamber studies, prior exposure to ambient PM cannot be discounted. Therefore, it is difficult to completely isolate the impacts of short-term elevated PM levels from chronic background exposures. In addition, as reviewed above, there is evidence that multi-day PM10 exposures are, at least in some studies, associated with greater risks than single-day exposures.

7.10.4.2.4 Importance of Impacts of Chronic Exposure

Our quantitative benefits assessment (section 9) as well as similar efforts undertaken recently by the U.S. EPA (U.S. EPA, 2000) demonstrates the significant implications of long-term exposure on mortality. In addition, effects on adult cases of bronchitis and childhood acute bronchitis, both associated with longer-term exposure to PM, are significant as well. Therefore, from a public health perspective, one should focus on reducing the entire distribution of PM concentrations, which would also lower the number of peak days. Formulating a short-term index consistent with the annual average is a rational way to approach the issue of limiting peak exposures that might still occur even when the annual average PM standard is attained.

7.10.4.2.5 Relationship of Recommended 24-hour and Annual PM10 Standards

As discussed in Chapter 6, ARB uses the Expected Peak Day Concentration (EPDC) in determining the "design value" for the 24-hour standard. The development of the EPDC uses a statistical model of the highest 20% of the daily values from the previous three years, making it relatively robust with respect to fluctuations in daily meteorological conditions. Specifically, the index will not be unduly influenced by any single day, and exceptional events such as forest or urban fires can be excluded. We conducted an analysis to determine the relationship between the EPDC and the annual average of 20 $\mu g/m^3$, the most health-protective end of the range proposed above. This analysis identified the single day peak exposure concentration that is consistent, given the current statewide distributions of PM10, with an annual average of 20 $\mu g/m^3$.

Using data from 144 sites around the state, a linear regression model was run relating the EPDC to the annual average for each site. The regression model generated an r^2 of 0.72 and indicated that statewide, the EPDC associated with a 20 $\mu g/m^3$ annual average is 48 $\mu g/m^3$ which accords quiet closely with the existing standard. For the South Coast AQMD, representing the most populous air basin in the state, the predicted EPDC is 51 $\mu g/m^3$.

7.10.4.2.6 Likelihood of Effects Occurring at Single Exposures Above 50 μg/m^3

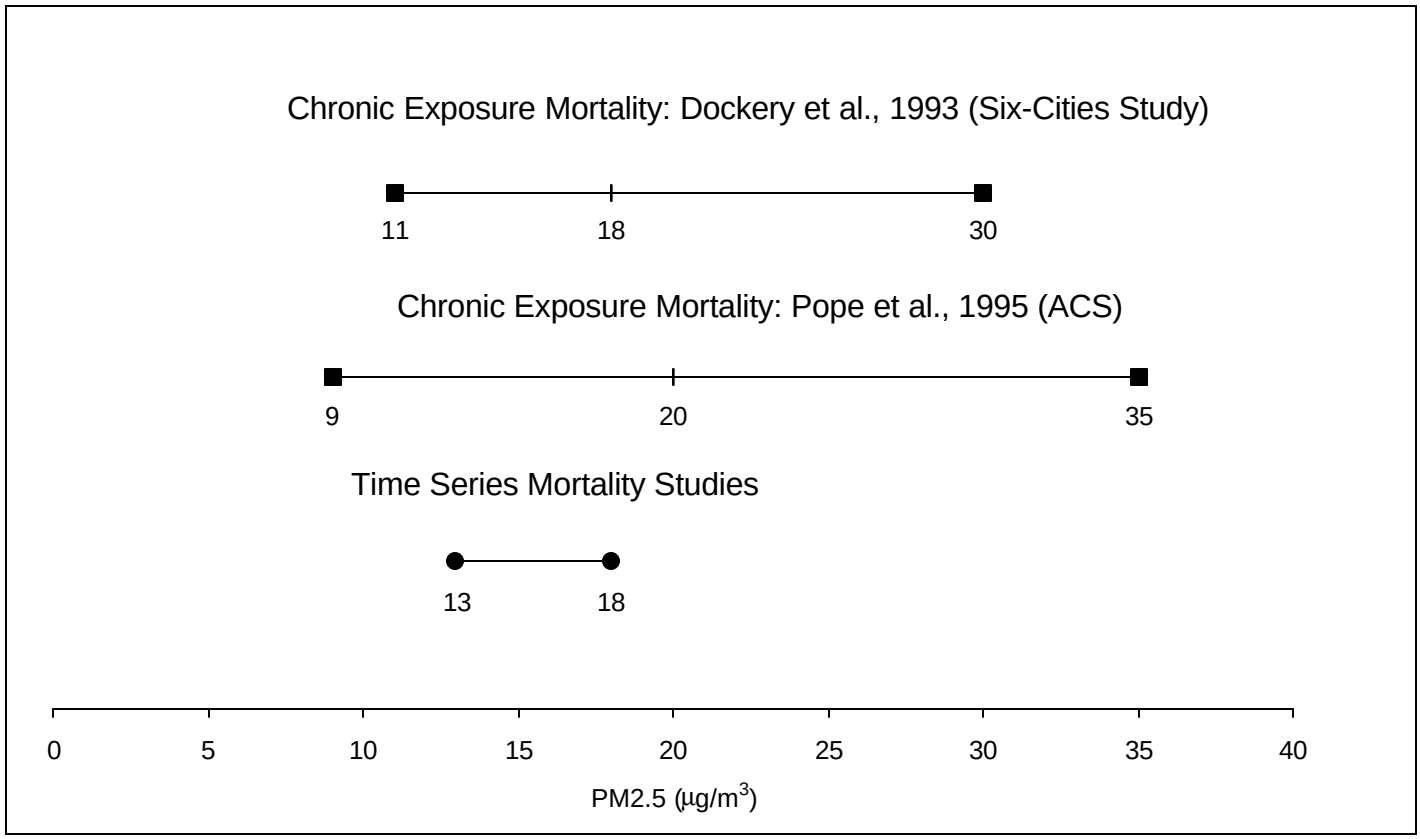
As indicated by Table 7.1, several studies with study means in the range of 15 to 30 μg/m^3 PM10 demonstrate associations between daily exposures and mortality. However, as indicated above, several studies at the lower concentration had wide confidence intervals that included the null value; that is, where the null hypothesis of no effect could not be rejected. OEHHA staff has examined the distribution of peak concentrations (i.e., 95th percentiles or maximum 24-hour concentrations) when they were provided in the time-series mortality studies reporting study mean concentrations of less than 30 μg/m^3. Many of these studies have peak values close to or above 50 μg/m^3. Given the linear, nonthreshold nature of the exposure-response relationship, keeping peak PM10 concentrations below 50 μg/m^3 will not categorically assure the absence of health impacts. However, combined with the recommended PM2.5 standard of 25 μg/m^3, attainment of a PM10 standard in which peak concentrations are kept below 50 μg/m^{3}is consistent with a distribution of PM10 in which the likelihood of mortality will be substantially reduced. Therefore, it is reasonable from a public health perspective to recommend a goal of preventing days when the 24-hour average concentration exceeds 50 μg/m^3.

In summary, while it is difficult to determine the effects of a single 24-hour exposure from available scientific studies, the evidence suggests that minimizing or eliminating days when the 24-hour PM10 average concentration exceeds 50 μg/m^3 represents a desirable public health goal. Bearing in mind that the attainment of the annual average PM10 standard will significantly depress the entire PM10 distribution, and attainment of the recommended 24-hour PM2.5 standard will in many instances also reduce peak PM10 levels, preventing single day PM10 concentrations above 50 μg/m^3 should afford additional public health protection. Therefore, we are proposing that the 24-hour standard be retained at 50 μg/m^3. Together, these standards should protect public health with an adequate margin of safety in the sense described in the introductory paragraphs of section 7.10.

7.10.4.3 Annual Standard for PM2.5

Staff recommends that the annual average for PM2.5 should be 12 μg/m^3, as explained below. Consideration of a standard at this level would place significant weight on the long-term exposure studies using the ACS and Harvard Six-Cities data (Dockery et al., 1993; Pope et al., 1995; Krewski et al., 2000). In both studies, robust associations were reported between long-term exposure to PM2.5 and mortality. The mean PM2.5 concentration was 18 μg/m^3 (range of 11.0 to 29.6 μg/m^3) in the Six-Cities study and 20 μg/m^3 (range of 9.0 to 33.5 μg/m^3) in the ACS study (see Figure 7.6). Thresholds were not apparent in either of these studies, although the precise period(s) and pattern(s) of relevant exposure could not be ascertained. If we assume, as in the PM10 standards considered above, that health effects are more likely to be observed when concentrations are at or above the mean or median PM2.5 levels, rather than at lower levels, then the most likely effects level for considering an annual PM2.5 standard would be 18 μg/m^3. Graphical analyses of these studies (Dockery et al., 1993, Figure 3 and Krewski et al., 2000, page 162) suggest a continuum of effects down to lower levels. In the case of the ACS study, uncertainty in the risk estimates becomes apparent at 13 μg/m^3. Around this level, the confidence bounds significantly widen since the concentrations are relatively far from the mean. In the Dockery et al. study, the relative risks are similar to the cities at the lowest long-term PM2.5 concentrations of 11 and12.5 μg/m^3. Larger increases in risk don't occur until the long-term PM2.5 mean equals 14.9 μg/m^3. Therefore, an annual standard of 12 μg/m^3 would be below the mean of the most likely effects level and would provide a margin of safety.

Figure 7.6 PM2.5 Range Of Long-term Mean Concentrations Observed In Epidemiological Studies



= Range of concentrations in a given study

♦ = Study mean concentration

● = Range of study mean concentrations among multiple studies

Targeting a long-term mean PM2.5 concentration of 12 μg/m^3 would also place some weight on the results of multiple daily exposure studies examining relationships between PM2.5 and adverse health outcomes (Table 7.2). These studies have long-term (three- to four-year) means in the range of 13 to 18 μg/m^3. It should be noted however, that many of these epidemiological investigations were conducted outside California, and may not be representative of exposures or population characteristics here. A standard set at 12 μg/m^3, well below the means of the major cohort mortality studies, would provide additional protection against mortality in adults associated with long-term exposure, as well as against a variety of morbidity effects in children (described in section 7.6, above). In the opinion of OEHHA staff, an annual PM2.5 standard of 12 μg/m^3 would be likely to provide adequate protection of public health, including that of infants and children, against adverse effects of long-term exposure.

The quantitative risk assessment examining the impacts of attainment of the annual PM2.5 standard (section 9) suggests that this could result in a reduction of 6,500 cases (95 percent CI 3,200 – 9,800) of premature mortality per year associated with the current annual average of ambient PM2.5 concentrations in California (approximately 18.5 μg/m^3, as reported in Chapter 9).

7.10.4.4 24-hour Standard for PM2.5

In the initial report to the Air Quality Advisory Committee (November 30, 2001), OEHHA staff did not propose a specific 24-hour standard for PM2.5. The Committee, however, unanimously recommended that OEHHA develop such a standard, and suggested several possible approaches. Responding to the Committee's concerns and suggestions, OEHHA staff members have formulated the following recommendation, in consultation with staff at the ARB.

As reviewed in prior sections, the epidemiological literature suggests the existence of impacts on both morbidity and mortality related to fluctuations in ambient PM2.5 on a daily basis. Morbidity outcomes associated with changes in 24-hour concentrations in PM2.5 include admissions to hospitals for respiratory and cardiac diseases (see sections 7.5.1 and 7.5.2). There is also a growing literature suggesting potential mechanistic linkages between ambient PM2.5 and exacerbations of cardiovascular disease that could result in hospitalization or death (see section 7.8). These include associations with serious cardiac arrhythmias, myocardial infarctions, and decreased heart rate variability (Peters et al., 2000a, 2001a, Liao et al., 1999; Gold et al., 2000; Pope et al., 1999c). As noted in prior sections, the entire spectrum of adverse health outcomes associated with ambient PM2.5, including exacerbations of asthma, emergency room visits, hospitalizations, as well as mortality, occurs within the same general concentration range and also seems to be best described by a linear, non-threshold model. Such a model implies that the level(s) at which adverse effects begin to occur cannot be identified and that there are no abrupt changes in the slope of the dose-response relationship to delineate a "bright line" or threshold.

Consistent observations of health effects associated with low ambient concentrations of fine particles, however, indicate that a short-term PM2.5 standard is required to protect public health. Moreover, while state-wide attainment of the proposed annual PM2.5 standard will result in a reduction of PM2.5 peak concentrations, some areas will be able to attain the annual standard and still experience periods during which 24-hour PM2.5 concentrations associated with increased morbidity and mortality can occur (e.g., during winter inversions accompanied by widespread residential wood combustion). This phenomenon also evidences the need for a short-term standard.

Development of a short-term standard for PM2.5, however, encompasses difficulties similar to those encountered with respect to the 24-hour standard for PM10, largely because the exposure-response relationships examined appear to be linear without clear evidence of a threshold. In order to address the lack of a "bright line" in the exposure-response curve, OEHHA staff members propose to reduce the entire distribution of fine particles below the reported levels of distributions consistently associated with adverse health effects. The underlying principle is to reduce not only the mean concentration (represented by the annual average), but also specifically the upper tail of the distribution, described by the 98th percentile of the distributions of published studies. In so doing, OEHHA has relied primarily on studies relating fine particle concentrations with daily mortality, the most serious irreversible health impact. As noted above and in section 7.5, associations of PM2.5 with morbidity, including effects such as exacerbation of asthma in children, have been observed to occur within the same concentration range as those linked with increased daily mortality in adults. We have therefore assumed that a standard intended to protect against the occurrence of mortality will also protect against these other important health outcomes.

7.10.4.4.1 Methodological Approaches

In developing this recommendation, OEHHA staff followed several approaches. Specifically, we have: (1) used statistical methods to examine the shape of the exposure-response relationships using two California datasets, and compared the results with those reported for other non-California datasets; (2) tabulated the results of all time-series studies published in English, for which direct PM2.5 monitoring data were available, that have explored associations between low levels of ambient PM2.5 and daily mortality; and (3) examined, with technical assistance from ARB staff, the upper tail of the PM2.5 distribution in California consistent with an annual average of 12 $\mu g/m^3$, based on data collected throughout California in 1999 and 2000. Based on the results of these analyses, OEHHA recommends that the 24-hour PM2.5 standard be established at a level of 25 $\mu g/m^3$, not to be exceeded. The adoption of the accompanying recommendation for an annual PM2.5 standard of 12 $\mu g/m^3$ is an integral component of this proposal. Attainment of the recommended annual standard will help shift the entire PM2.5 distribution to the left, and will influence peak concentrations, as well. However, in itself, the annual average will not fully address the issue of brief (i.e., one to several days) increases in PM2.5 levels. Thus, the 24-hour standard is intended to protect Californians against significant short-term elevations of PM2.5.

1. Statistical approaches

As discussed in section 7.3.5, staff from OEHHA and the Bay Area Air Quality Management District (BAAQMD) undertook a variety of detailed analyses of data from the two published California studies involving 24-hour measurements of PM2.5 and daily mortality counts (in Coachella Valley [Ostro et al., 2000] and Santa Clara County [Fairley, 1999]). In general, nonlinear models (and, in particular, models intended to identify possible thresholds) offered no improvement over a linear, nonthreshold model in fitting the data. These analyses, which

2. Distributions of PM2.5 in daily mortality studies.

OEHHA staff obtained data from the authors of all recently published studies examining ambient PM2.5 concentrations in relation to daily nonaccidental mortality. Table 7.7 provides information on the estimated percentage change in daily mortality associated with a 10 $\mu g/m^3$ change in PM2.5. All the point estimates of this relationship in Table 7.7 are positive, though not all are statistically significant. The upper tail of the PM2.5 distribution in each of these investigations is indicated by the 98th percentile, which is somewhat less subject to the factors determining the most extreme values. Examination of the PM2.5 levels in Table 7.7 indicates

Table 7.7 Distributions and Associations of 24-hour PM2.5 with Daily Total(T) and Cardiovascular(CV) Mortality for All Age Groups (except where noted) in U.S. and Canadian Cities with Mean* PM2.5 Concentrations < 25 μg/m³, Sorted by Reported 98th Percentile Concentrations**

City	Time Period	Reference	Mean* (μg/m³)	98th per-centile	% Increase (95% CI) per 10μg/m³
Edmonton	1986-1996	Burnett et al., 2000	10	28	T:2.18(-1.74, 6.10)
Calgary	1986-1996	Burnett et al., 2000	10	29	T:0.63(-3.58, 4.84)
Winnipeg	1986-1996	Burnett et al., 2000	10	29	T:0.38(-3.15, 3.91)
Vancouver	1986-1996	Burnett et al., 2000	13	30	T:2.56(0.23, 4.89)
Topeka, KS	1979-1988	Schwartz et al., 1996	12	31	T:0.80(-0.20, 3.60)
Phoenix, AZ (Mortality for 65 yrs & older)	1995-1997	Mar et al., 2000	13	32	T:2.22(0.00, 5.56) CV:6.85(2.22, 11.48)
Portage, WI	1979-1987	Schwartz et al., 1996	11	34	T:1.20(-0.30, 2.80)
Ottawa	1986-1996	Burnett et al., 2000	12	35	T:2.45(-0.53, 5.43)
Coachella Valley, CA	1995-1998	Ostro et al., 2000	17	38	T:-1.42(-7.81, 4.97) CV:3.73(-2.37, 9.84)
Toronto	1986-1996	Burnett et al., 2000	15	41	T:0.91(-0.05, 1.87)
Boston, MA	1979-1986	Schwartz et al., 1996	16	42	T:2.20(1.50, 2.90)
Windsor	1986-1996	Burnett et al., 2000	18	43	T:5.20(2.24, 8.16)
Montreal	1984-1993	Goldberg et al., 2001a	18	43	T:1.93(1.16, 2.71)
Kingston	1980-1987	Schwartz et al., 1996	21	44	T:1.40(0.20, 2.60)
St. Louis, MO	1979-1987	Schwartz et al., 1996	19	46	T:1.10(0.40, 1.70)
Santa Clara, CA	1990-1996	Fairley, 1999	13	51	T:3.26(1.27, 5.24) CV:2.48(-0.35, 6.02)
Montreal	1986-1996	Burnett et al., 2000	15	51	T:1.23(0.11, 2.35)
Detroit, MI	1992-1994	Lippmann et al., 2000	18	55	T:1.24(-0.26, 2.83) CV:1.28(-0.91,3.65)

* Mean of 24-hour measurements over time period.

** Some data in Table 7.7, particularly most of the 98th percentile values, were obtained directly from the authors of the published reports

that, when the 98th percentiles of the fine particle distributions are $\leq$32 $\mu g/m^3$, and the mean fine particle concentrations are <13 $\mu g/m^3$, the results are characterized by greater uncertainty, since the confidence intervals for the percent change in mortality include zero. These were studies conducted in Portage (WI), Topeka (KS), and in four Canadian cities (Calgary, Edmonton, Ottawa, and Winnipeg). One partial exception to this observation is Vancouver, British Columbia, which had a 98th percentile PM2.5 concentration of 30 $\mu g/m^3$, though the mean concentration was 13 $\mu g/m^3$. These results do not imply an absence of effects when peak PM2.5 concentrations are below 30 $\mu g/m^3$; rather, these estimates may be subject to greater uncertainty potentially ascribable to several factors, including fewer health impacts associated with exposure to lower concentrations, exposure measurement error, confounding by co-pollutants or meteorological factors, differences in the composition of particle mixtures, decreased statistical power, and reduced variance in the PM2.5 values in studies with lower means. The last explanation is unlikely, however, as we examined the coefficients of variation in the studies with relatively low PM2.5 mean concentrations and found that they were generally similar to those in the studies with higher mean levels. In contrast, statistical power (i.e., the ability to detect statistically a real relationship between two variables) is likely to be reduced at lower ambient pollutant concentrations. Based on model simulations conducted by staff at the BAAQMD, the increased uncertainty between lower-level PM2.5 concentrations and daily mortality may be attributable in part to insufficient statistical power.

Published studies provide some guidance for an appropriate reduction in the distribution of PM2.5. An annual PM2.5 standard of 12 $\mu g/m^3$ would represent a level lower than the long-term means of all the studies in which significant associations with changes in daily mortality have been identified (see Table 7.7 and section 7.3, above). Attainment of the annual average would, as previously noted, result in an across-the-board reduction of PM2.5, including peak concentrations. Setting a 24-hour standard level below 30 $\mu g/m^3$ would shift the upper extreme of the PM2.5 distribution to a level lower than those identified in the studies described above. Because the exposure-response relationship is characterized by a linear, nonthreshold model, such a 24-hour standard does not imply total elimination of health risks when this standard is attained. However, reduction of peak PM2.5 concentrations below those observed in studies reporting adverse effects represents a rational approach to reduce the risk of short-term PM2.5-associated mortality and morbidity and to position the entire distribution of PM2.5 below those for which there is current, published evidence of health effects.

3. Relationship of Recommended Annual PM2.5 Standards and 24-hour PM2.5 Concentrations in California

As discussed in Chapter 6, the ARB uses the Expected Peak Day Concentration (EPDC) to determine the "design value" for 24-hour standards. The development of the EPDC uses a statistical model of the highest 20% of the daily values from the previous three years, making it relatively robust with respect to fluctuations in daily meteorological conditions. Specifically, the index will not be unduly influenced by any single day, and exceptional events such as forest or urban fires can be excluded. We used a modified version of this process to examine the upper tail of the PM2.5 distribution (98th percentile) rather than the most extreme values within California. With assistance from ARB staff, we conducted an analysis to determine the relationship between the 98th percentile of the PM2.5 distribution in California and the proposed annual average of 12 $\mu g/m^3$. This analysis identified the 98th percentile concentrations consistent with an annual average of 12 $\mu g/m^3$, given recent statewide distributions of PM2.5.

Using data from 54 sites around the state, located principally in large urban areas, a linear regression model was performed (linear models fit the data better than non-linear models) relating the 98th percentile of the PM2.5 distribution to the annual average for the years 1999 and 2000 for each site. The regression model generated an r^2 of 0.79 and indicated that statewide, the 98th percentile for the distribution of PM2.5 associated with a 12 $\mu g/m^3$ annual average is approximately 39 $\mu g/m^3$. For sites within the jurisdiction of the South Coast Air Quality Management District, representing the most heavily populated air basin in the state, the predicted 98th percentile concentration is approximately 37 $\mu g/m^3$, while the corresponding value for three other major air basins (the San Francisco Bay Area, San Joaquin Valley, and Sacramento) is 45 $\mu g/m^3$, and that for the South Central Coast is 33 $\mu g/m^3$.

This approach to identify ambient PM2.5 98th percentile concentrations consistent with attainment of the proposed annual average indicates that, at least in some of the heavily populated air basins, predicted concentrations of PM2.5 could fall within ranges previously reported to be associated with increased daily mortality (Table 7.2) and morbidity. This modified EPDC exercise suggests the need for a lower short-term standard to limit excursions of PM2.5 to protect against increased risks of morbidity and mortality.

7.10.4.4.2 Recommendation for 24-hour PM2.5 Standard

Examining the evidence described above, OEHHA recommends that the 24-hour PM2.5 standard be 25 $\mu g/m^3$, not to be exceeded. The rationale for this recommendation is as follows:

(i) Multiple analyses of the exposure-response relationships between PM2.5 and mortality indicate that the data can be fitted most parsimoniously with linear, nonthreshold models. Given the apparent linearity of the exposure-response relationships in the epidemiological data, it is difficult to determine at what concentrations within the PM2.5 distributions in each study adverse health effects begin. Intuitively, one would expect greater biological responses and larger numbers of adverse events occurring at higher concentrations, everything else being equal. Nonetheless, in a linear exposure-response relationship, effects may be observed at lower levels as well (e.g., Schwartz et al., 1996).

The importance of the linear, nonthreshold exposure-response relationship cannot be overemphasized in light of legislation requiring that ambient air quality standards be "established at levels that adequately protect the health of the public, including infants and children, with an adequate margin of safety." (California Health & Safety Code section 39606(d)(2)) If a threshold in the exposure-response curve cannot be identified, then specification of an "adequate margin of safety" becomes challenging. The approach OEHHA staff members have adopted in pursuit of this objective has therefore been to: (1) identify indicators of the distribution of PM2.5 (specifically the means and 98th percentiles) in epidemiological studies that demonstrate the relationship of ambient fine particles with adverse health impacts, (2) recommend that the distribution of PM2.5 in California be reduced below the levels of these distributions, and (3) incorporate a margin of safety in the form of a standard "not to be exceeded", which will assure that the extreme values of the PM2.5 distribution in California will be lower (and in general substantially lower) than the 98th percentiles of PM2.5 distributions in published studies.

(ii) Without placing a short-term limitation on PM2.5 concentrations, recent experience in California indicates that even attainment of the recommended annual standard of 12 $\mu g/m^3$ will allow for excursions well into the range in which adverse effects, including mortality, have been identified in epidemiological studies. Notably, the modified EPDC analysis undertaken by the ARB staff indicates that for several large air basins, the estimated 98th percentile of the

PM2.5 distribution consistent with attainment of an annual standard of 12 $\mu g/m^3$ would be in excess of 40 $\mu g/m^3$. Thus, adoption of a 24-hour standard of 25 $\mu g/m^3$ would be intended to limit such excursions.

(iii) As with PM10, morbidity and mortality outcomes appear to occur within the same PM2.5 concentration ranges (see section 7.5). Therefore, we have focused on mortality as the most serious adverse health outcome. Changes in ambient air quality sufficient to protect against increases in mortality should, *a fortiori*, also protect against the occurrence of morbidity, in children as well as adults.

(iv) Among studies examining PM2.5 and mortality, the long-term mean concentrations of those finding a significant association varied from 13 to 21 $\mu g/m^3$, while the 98^{th} percentiles of the distributions ranged from 30 to 51 $\mu g/m^3$. Shifting the entire PM2.5 distribution downwards and limiting short-term excursions should reduce the likelihood of fine particle-associated mortality and morbidity. Recommending an annual average of 12 $\mu g/m^3$ addresses the issue of shifting the overall distribution downwards. By the same token, recommending a 24-hour PM2.5 limit of 25 $\mu g/m^3$ would place the upper extreme of the distribution lower than the 98^{th} percentile of those identified in studies finding significant associations with mortality, thereby incorporating a margin of safety. More specifically, except for the study of Vancouver (Burnett et al., 2000), all published investigations of PM2.5 and mortality in which statistically significant effects were detected had 98^{th} percentile PM2.5 concentrations of 32 $\mu g/m^3$ or greater. Positioning the upper extreme of the PM2.5 distribution in California at 25 $\mu g/m^3$ effectively incorporates a margin of safety into this recommendation, based on the best available scientific evidence.

7.10.4.5 24-hour Standard for Sulfates

Staff recommends that the 24-hour average standard for sulfate of 25 $\mu g/m^3$ be retained. Serious health effects have been associated with exposure to ambient sulfates, particularly in areas rich in strongly acidic sulfates such as the eastern United States and Canada. The results of such studies however, have not been as consistent as those for PM10, PM2.5, or the coarse fraction. In addition, though daily sulfate concentrations have been linked with a variety of adverse health events in epidemiological studies, the nature of the study data does not allow for segregation of outcomes related to chronic low-level exposure from those associated with daily elevations in sulfate concentrations.

In California, acidic sulfates (principally sulfuric acid and ammonium sulfate) constitute a small fraction of the PM mass relative to the areas in which sulfates have been found to be associated with adverse health impacts. Sulfate concentrations in California have been far lower during the past few years than the level of the existing standard. Based on an assessment of current scientific evidence and ambient air quality data, staff believes that exposures to sulfates in California do not appear to pose health risks distinct from or greater than those associated with exposures to particulate matter generally. In view of the mixed evidence in the sulfates and health in California, the low likelihood of health risks in relation to ongoing trends in sulfate emissions and ambient levels, staff recommends that the current standard be retained until the next review of the PM standard, if not earlier.

7.10.4.6 General Staff Conclusions

In light of the adverse health effects observed at current ambient concentrations and the lack of a demonstrated threshold, staff further concludes: (1) that in any air basin in California that currently attains the ambient air quality standards, for either PM10 or PM2.5, the air quality should not be degraded from present levels; and (2) that the ARB revisit the standards in five

years or less, in order to re-evaluate the evidence regarding the health effects associated with particle size, chemistry, and concentration.

7.10.5 Summary of Recommendations

- Revise the current PM10 annual average standard from 30 to 20 $\mu g/m^3$. Revise the averaging method to an annual arithmetic mean from the current annual geometric mean.
- Retain the 24-hour standard for PM10 at 50 $\mu g/m^3$, not to be exceeded.
- Establish an annual average standard for PM2.5 of 12 $\mu g/m^3$, given growing evidence from epidemiological and toxicological studies of significant toxicity related to this size fraction of PM. Establish the annual PM2.5 standard as an annual arithmetic mean.
- Establish a 24-hour standard for PM2.5 of 25 $\mu g/m^3$, not to be exceeded.
- Retain the current 24-hour average standard of 25 $\mu g/m^3$ for sulfates.

General Staff Conclusions Regarding Air Quality Degradation

- For any air basin in California that currently attains the ambient air quality standards, for either PM10 or PM2.5, that air quality should not be degraded from present levels.
- Revisit the standards in five years or less, in order to re-evaluate the evidence regarding the health effects associated with particle size, chemistry, and concentration.

7.11References

Abbey DE, Petersen FF, Mills PK, Kittle L (1993). Chronic respiratory disease associated with long-term ambient concentrations of sulfates and other air pollutants. J Expo Anal Environ Epidemiol 3(Suppl. 1):99-115.

Abbey DE, Ostro BE, Petersen F, Burchette RJ (1995a). Chronic respiratory symptoms associated with estimated long-term ambient concentrations of fine particulates less than 2.5 microns in aerodynamic diameter (PM2.5) and other air pollutants. J Expo Anal Environ Epidemiol 5(2):137-59.

Abbey DE, Ostro BE, Fraser G, Vancuren T, Burchette RJ (1995b). Estimating fine particulates less then 2.5 microns in aerodynamic diameter (PM 2.5) from airport visibility data in California. J Expo Anal Environ Epidemiol 5:161-80.

Abbey DE, Nishino N, McDonnell WF, Burchette RJ, Knusten SF, Beeson WL *et al*. (1999). Long-term inhalable particles and other air pollutants related to mortality in nonsmokers. Am J Respir Crit Care Med 159:373-82.

Ackermann-Liebrich U, Philippe L, Schwartz J, Schindler C, Monn C, Bolognini G *et al*. (1997). Lung function and long term exposure to air pollutants in Switzerland. Am J Respir Crit Care Med 155:122-9.

Adams WC (1993). Measurement of breathing rate and volume in routinely performed daily activities. Final Report. California Air Resources Board; Contract No. A033-205.

Air Resources Board (1994). Chapter II. Monitoring and Research Programs. The Atmospheric Acidity Protection Program: Annual Report to the Governor and the Legislature, 1993. Sacramento, CA: California Air Resources Board.

Air Resources Board (2000). Adequacy of California's Ambient Air Quality Standards: Children's Environmental Health Protection Act. Nov. 2, 2000. Sacramento, CA: California Environmental Protection Agency.

Algra A, Tijssen JG, Roelandt JR, Pool J, Lubsen J (1993). Heart rate variability from 24-hour electrocardiography and the 2-year risk for sudden death. Circulation 88(1):180-5.

Anderson PJ, Wilson JD, Hiller FC (1990). Respiratory tract deposition of ultrafine particles in subjects with obstructive or restrictive lung disease. Chest 97(5):1115-20.

Aris R, Christian D, Sheppard D, Balmes JR (1991). Lack of bronchoconstrictor response to sulfuric acid aerosols and fogs. Am Rev Respir Dis 143(4 Pt 1):744-50.

Atkinson RW, Anderson HR, Strachan DP, Bland JM, Bremner SA, Ponce de Leon A (1999). Short-term associations between outdoor air pollution and visits to accident and emergency departments in London for respiratory complaints. Eur Respir J 13:257-65.

Bates DV (1992). Health indices of the adverse effects of air pollution: the question of coherence. Environ Res 59(2):336-49.

Bates DV, Sizto R (1987). Air pollution and hospital admissions in Southern Ontario: the acid summer haze effect. Environ Res 43:317-31.

Becker S, Soukup JM (1999). Exposure to urban air particulates alters the macrophage-mediated inflammatory response to respiratory viral infection. J Toxicol Environ Health A 57(7):445-57.

Beeson WL, Abbey DE, Knutsen SF (1998). Long-term concentrations of ambient air pollutants and incident lung cancer in California adults: Results from the AHSMOG study. Environ Health Perspect 106:813-23.

Bennett WD, Zeman KL (1998). Deposition of fine particles in children spontaneously breathing at rest. Inhal Toxicol 10:831-42.

Bennett WD, Zeman KL, Kim C (1996). Variability of fine particle deposition in healthy adults: effect of age and gender. Am J Respir Crit Care Med 153(5):1641-7.

Bobak M (2000). Outdoor air pollution, low birth weight, and prematurity. Environ Health Perspect 108(2):173-6.

Bobak M, Leon DA (1998). Air pollution and infant mortality: the effects are specific for respiratory causes in the postneonatal period. Epidemiology 9:S58.

Boezen HM, van der Zee SC, Postma DS, *et al.* (1999). Effects of ambient air pollution on upper and lower respiratory symptoms and peak expiratory flow in children. Lancet 353:874-8.

Bouthillier L, Vincent R, Geogan P, *et al.* (1998). Acute effects of inhaled urban particles and ozone. Lung morphology, macrophage activity, and plasma endothelin-1. Am J Pathol 153:1873-84.

Braga AL, Zanobetti A, Schwartz J (2000). Do respiratory epidemics confound the association between air pollution and daily deaths? Eur Respir J 16(4):723-8.

Brain JD, Long NC, Wolfthal SF, Dumyahn T, Dockery DW (1998). Pulmonary toxicity in hamsters of smoke particles from Kuwaiti oil fires. Environ Health Perspect 106(3):141-6.

Bremner SA, Anderson HR, Atkinson RW, McMichael AJ, Strachan DP, Bland JM *et al*. (1999). Short-term associations between outdoor air pollution and mortality in London 1992-4. Occup Environ Med 56(4):237-44.

Brown DM, Stone V, Findlay P, MacNee W, Donaldson K (2000). Increased inflammation and intracellular calcium caused by ultrafine carbon black is independent of transition metals or other soluble components. Occup Environ Med 57(10):685-91.

Brunekreef B (1997). Air pollution and life expectancy: is there a relation? Occup Environ Med 54(11):781-4.

Burnett RT, Dales RE, Raizenne ME, Krewski D, Summers PW, Roberts GR *et al*. (1994). Effects of low ambient levels of ozone and sulphates on the frequency of respiratory admissions to Ontario hospitals. Environ Res 65:172-94.

Burnett RT, Dales RE, Brook JR, Raizenne ME, Krewski D (1997a). Association between ambient carbon monoxide levels and hospitalizations for congestive heart failure in the elderly in 10 Canadian cities. Epidemiology 8 :162-7.

Burnett RT, Cakmak S, Brook JR, Krewski D (1997b). The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. Environ Health Perspect 105:614-20.

Burnett RT, Brook JR, Dann T, Delocla C, Philips O, Calmak S *et al* . (2000). Association between particulate- and gas-phase components of urban air pollution and daily mortality in eight Canadian cities. In: Grant LD, ed. PM2000: Particulate Matter and Health. Inhal Toxicol 12(Suppl. 4):15-39.

Cakmak S, Burnett RT, Krewski D (1999). Methods for detecting and estimating population threshold concentrations for air pollution-related mortality with exposure measurement error. Risk Anal 19(3):487-96.

Campen MJ, Costa DL, Watkinson WP (2000). Cardiac and Thermoregulatory toxicity of residual oil fly ash in cardiopulmonary-compromised rats. Inhal Toxicol 12(Suppl. 2):7-22.

Castillejos M, Borja-Aburto VH, Dockery DW, Gold DR, Loomis D (2000). Airborne coarse particles and mortality. In: Inhalation Toxicology: proceedings of the third colloquium on particulate air pollution and human health; June, 1999; Durham, NC. Inhal Toxicol 12(Suppl. 1):67-72.

CDHS (2000). California County Asthma Mortality Chart Book: Data for 1990-1997. Oakland, California: Environmental Health Investigations Branch.

Chock DP, Winkler SL, Chen C (2000). A study of the association between daily mortality and ambient air pollutant concentrations in Pittsburgh, Pennsylvania. J Air Waste Manag Assoc 50(8):1481-500.

Churg A, Brauer M (1997). Human lung parenchyma retains PM2.5. Am J Respir Crit Care Med 155(6):2109-11.

Churg A, Brauer M (2000). Ambient atmospheric particles in the airways of human lungs. Ultrastruct Pathol 24(6):353-61.

Cifuentes LA, Vega J, Kopfer K, Lave LB (2000). Effect of the fine fraction of particulate matter versus the coarse mass and other pollutants on daily mortality in Santiago, Chile. J Air Waste Manag Assoc 50(8):1287-98.

Cleveland WS, Devlin SJ (1988). Robust locally-weighted regression and smoothing scatterplots. J Am Stat Assoc 74:829-36.

Clyde MA, Guttorp P, Sullivan E (2000). Effects of ambient fine and coarse particles on mortality in Phoenix, Arizona. J Expo Anal Environ Epidemiol :submitted.

Copie X, Hnatkova K, Staunton A, Fei L, Camm AJ, Malik M (1996). Predictive power of increased heart rate versus depressed left ventricular ejection fraction and heart rate variability for risk stratification after myocardial infarction. Results of a two-year follow-up study. J Am Coll Cardiol 27 (2):270-6.

Corbalan R, Verrier R, Lown B (1974). Psychological stress and ventricular arrhythmias during myocardial infarction in the conscious dog. Am J Cardiol 34(6):692-6.

Costa DL, Dreher KL (1997). Bioavailable transition metals in particulate matter mediate cardiopulmonary injury in healthy and compromised animal models. Environ Health Perspect 105(Suppl. 5):1053-60.

Daniels MJ, Dominici F, Samet JM, Zeger SL (2000). Estimating particulate matter-mortality dose-response curves and threshold levels: an analysis of daily time-series for the 20 largest US cities. Am J Epidemiol 152(5):397-406.

Dejmek J, Selevan SG, Benes I, Solansky I, Sram RJ (1999). Fetal growth and maternal exposure to particulate matter during pregnancy. Environ Health Perspect 107(6):475-80.

Delfino RJ, Becklake MR, Burnett RT, Brook JR, Becklake MR (1997a). Effects of air pollution on emergency room visits for respiratory illnesses in Montreal, Quebec. Am J Respir Crit Care Med 155:568-76.

Delfino RJ, Zeiger RS, Seltzer JM, Street DH, Matteucci RM, Anderson PR *et al*. (1997b). The effect of outdoor fungal spore concentrations on daily asthma severity. Environ Health Perspect 105(6):622-35.

Delfino RJ, Zeiger RS, Seltzer JM, Street DH (1998). Symptoms in pediatric asthmatics and air pollution: differences in effects by symptom severity, anti-inflammatory medication use and particulate averaging time. Environ Health Perspect 106(11):751-61.

Detels R, Tashkin DP, Sayre JW, Rokaw SN, Massey FJ Jr, Coulson AH *et al*. (1991). The UCLA population studies of CORD: X. A cohort study of changes in respiratory function associated with chronic exposure to SOx, NOx, and hydrocarbons. Am J Public Health 81(3):350-9.

Dockery DW, Speizer FH, Stram DO, Ware JH, Spengler JD, Ferris BG Jr (1989). Effects of inhalable particles on respiratory health of children. Am Rev Respir Dis 139:587-94.

Dockery DW, Schwartz J, Spengler JD (1992). Air pollution and daily mortality: associations with particulates and acid aerosols. Environ Res 59:362-73.

Dockery DW, Pope CA III, Xu X, Spengler JD, Ware JH, Fay ME *et al*. (1993). An association between air pollution and mortality in six U.S. cities. N Engl J Med 329:1753-9.

Dockery DW, Pope CA III (1994). Acute respiratory effects of particulate air pollution. Annu Rev Public Health 15:107-32.

Dockery DW, Cunningham J, Damokosh AI, Neas LM, Spengler JD, Koutrakis P *et al*. (1996). Health effects of acid aerosols on North American children: respiratory symptoms. Environ Health Perspect 104:500-5.

Dockery DW, Pope CA III, Kanner RE, Martin Villegas G, Schwartz J (1999). Daily changes in oxygen saturation and pulse rate associated with particulate air pollution and barometric pressure. Res Rep Health Eff Inst (83):1-19; discussion 21-8 .

Donaldson K, Brown DM, Mitchell C, Dineva M, Beswick PH, Gilmour P *et al*. (1997). Free radical activity of PM10: iron-mediated generation of hydroxyl radicals. Environ Health Perspect 105 Suppl 5:1285-9.

Ernst E, Resch KL (1993). Fibrinogen as a cardiovascular risk factor: a meta-analysis and review of the literature. Ann Intern Med 118(12):956-63.

Fairley D (1999). Daily mortality and air pollution in Santa Clara County, California: 1989-1996. Environ Health Perspect 107(8):637-41.

Falk R, Philipson K, Svartengren M, Bergmann R, Hofmann W, Jarvis N *et al*. (1999). Assessment of long-term bronchiolar clearance of particles from measurements of lung retention and theoretical estimates of regional deposition. Exp Lung Res 25(6):495-516.

Ferin J, Oberdorster G, Penney DP (1992). Pulmonary retention of ultrafine and fine particles in rats. Am J Respir Cell Mol Biol 6(5):535-42.

Ferris BG Jr, Higgins IT, Higgins MW, Peters JM (1973). Chronic nonspecific respiratory disease in Berlin, New Hampshire, 1961 to 1967. A follow-up study. Am Rev Respir Dis 107(1):110-22.

Foster WM (1999). Deposition and clearance of inhaled particles. In: Holgate ST, Samet JM, Koren HS, Maynard RL, eds. Air pollution and health. San Diego: Academic Press, 295-324.

Gardner SY, Lehmann JR, Costa DL (2000). Oil fly ash-induced elevation of plasma fibrinogen levels in rats. Toxicol Sci 56(1):175-80.

Gauderman WJ, McConnell R, Gilliland F, London S, Thomas D, Avol E *et al*. (2000). Association between air pollution and lung function growth in Southern California children. Am J Respir Crit Care Med 162:1383-90.

Gavett SH, Madison SL, Stevens MA, Costa DL (1999). Residual oil fly ash amplifies allergic cytokines, airway responsiveness, and inflammation in mice. Am J Respir Crit Care Med 160(6):1897-904.

Ghio AJ, Kim C, Devlin RB (2000). Concentrated ambient air particles induce mild pulmonary inflammation in healthy human volunteers. Am J Respir Crit Care Med 162(3 Pt 1):981-8.

Gielen MH, van der Zee SC, van Wijnen JH, van Steen CJ, Brunekreef B (1997). Acute effects of summer air pollution on respiratory health of asthmatic children. Am J Respir Crit Care Med 155:2105-8.

Gold DR, Litonjua A, Schwartz J, Lovett E, Larson A, Nearing B *et al*. (2000). Ambient pollution and heart rate variability. Circulation 101(11):1267-73.

Goldberg MS, Burnett RT, Bailar JC 3rd, Brook J, Bonvalot Y, Tamblyn R *et al*. (2001a). The association between daily mortality and ambient air particle pollution in Montreal, Quebec. 1. Nonaccidental mortality. Environ Res 86(1):12-25.

Goldberg MS, Burnett RT, Bailar JC III, Tamblyn R, Ernst P, Flegel K *et al*. (2001b). Identification of persons with cardiorespiratory conditions who are at risk of dying from the acute effects of ambient air particles. Environ Health Perspect 109(Suppl. 4):487-94.

Goldberg RJ, Larson M, Levy D (1996). Factors associated with survival to 75 years of age in middle-aged men and women. The Framingham Study. Arch Intern Med 156(5):505-9.

Gwynn RC, Thurston GD (2001). The Burden of Air Pollution: Impacts among Racial Minorities. Environ Health Perspect 109(Suppl. 4):501-6.

Gwynn RC, Burnett RT, Thurston GD (2000). A time-series analysis of acidic particulate matter and daily mortality and morbidity in the Buffalo, New York, region. Environ Health Perspect 108(2):125-33.

Hajat S, Haines A, Goubet SA, Atkinson RW, Anderson HR (1999). Association of air pollution with daily GP consultations for asthma and other lower respiratory conditions in London. Thorax 54:597-605.

Hajat S, Haines A, Atkinson RW, *et al.* (2001). Association between air pollution and daily consultations with general practitioners for allergic rhinitis in London, United Kingdom. Am J Epidemiol 153(7):704-14.

Hauser R, Godleski JJ, Hatch V, Christiani DC (2001). Ultrafine particles in human lung macrophages. Arch Environ Health 56(2):150-6.

Haverkate F, Thompson SG, Pyke SD, Gallimore JR, Pepys MB (1997). Production of C-reactive protein and risk of coronary events in stable and unstable angina. European Concerted Action on Thrombosis and Disabilities Angina Pectoris Study Group. Lancet 349(9050):462-6.

Hill AB (1965). The environment and disease: association or causation? Proc R Soc Med 58:295-300.

Hiltermann TJN, Stolk J, van der Zee SC, Brunekreef B, de Bruijne CR, Fischer PH *et al*. (1998). Asthma severity and susceptibility to air pollution. Eur Respir J 11:686-93.

Hodgkin JE, Abbey DE, Euler GL, Magie AR (1984). COPD prevalence in nonsmokers in high and low photochemical air pollution areas. Chest 86:830-8.

Hoek G, Dockery DW, Pope CA III, Neas LM, Roemer W, Brunekreef B (1998). Association between PM10 and decrements in peak expiratory flow rates in children: reanalysis of data from five panel studies. Eur Respir J 11:1307-11.

Hoek G, Brunekreef B, Verhoeff A, van Wijnen J, Fischer P (2000). Daily mortality and air pollution in The Netherlands. J Air Waste Manag Assoc 50(8):1380-9.

Hoek G, Brunekreef B, Fischer P, van Wijnen J (2001). The association between air pollution and heart failure, arrhythmia, embolism, thrombosis, and other cardiovascular causes of death in a time series study. Epidemiology 12(3):355-7.

Ibald-Mulli A, Stieber J, Wichmann HE, Koenig W, Peters A (2001). Effects of air pollution on blood pressure: a population-based approach. Am J Public Health 91(4):571-7.

Iso H, Shimamoto T, Sato S, Koike K, Iida M, Komachi Y (1996). Passive smoking and plasma fibrinogen concentrations. Am J Epidemiol 144(12):1151-4.

Ito K, Thurston GD (1996). Daily PM10/Mortality associations: An investigation of at-risk subpopulations. J Expo Anal Environ Epidemiol 6(1):79-95.

Ito K, Thurston GD, Hayes C, Lippmann M (1993). Associations of London, England, daily mortality with particulate matter, sulfur dioxide, and acidic aerosol pollution. Arch Environ Health 48(4):213-20.

Junker R, Heinrich J, Ulbrich H, Schulte H, Schonfeld R, Kohler E *et al*. (1998). Relationship between plasma viscosity and the severity of coronary heart disease. Arterioscler Thromb Vasc Biol 18(6):870-5.

Kasanuki H, Ohnishi S, Ohtuka M, Matsuda N, Nirei T, Isogai R *et al*. (1997). Idiopathic ventricular fibrillation induced with vagal activity in patients without obvious heart disease. Circulation 95(9):2277-85.

Katsouyanni K, Touloumi G, Samoli E, Gryparis A, Le Tertre A, Monopolis Y *et al*. (2001). Confounding and effect modification in the short-term effects of ambient particles on total

mortality: results from 29 European cities within the APHEA2 project. Epidemiology 12(5):521-31.

Kelsall JE, Samet JM, Zeger SL, Xu J (1997). Air pollution and mortality in Philadelphia, 1974-1988. Am J Epidemiol 146:750-62.

Kennedy T, Ghio AJ, Reed W, Samet J, Zagorski J, Quay J *et al*. (1998). Copper-dependent inflammation and nuclear factor-kappaB activation by particulate air pollution. Am J Respir Cell Mol Biol 19(3):366-78.

Kim CS, Kang TC (1997). Comparative measurement of lung deposition of inhaled fine particles in normal subjects and patients with obstructive airway disease. Am J Respir Crit Care Med 155(3):899-905.

Kim CS, Hu SC (1998). Regional deposition of inhaled particles in human lungs: comparison between men and women. J Appl Physiol 84(6): 1834-44.

Kim CS, Hu SC, DeWitt P, Gerrity TR (1996). Assessment of regional deposition of inhaled particles in human lungs by serial bolus delivery method. J Appl Physiol 81(5):2203-13.

Kinney PL, Kazuhiko I, Thurston GD (1995). A sensitivity analysis of mortality/PM10 associations in Los Angeles. Inhal Toxicol 9:59-69.

Kleiger RE, Miller JP, Bigger JT Jr, Moss AJ (1987). Decreased heart rate variability and its association with increased mortality after acute myocardial infarction. Am J Cardiol 59(4):256-62.

Kleinman MT, Bhalla KD, Phalen RF (1995). Cellular and immunologic injury with PM-10 inhalation. Inhal Toxicol 7:589-602.

Kleinman MT, Mautz WJ, Bjarnason S (1999). Adaptive and non-adaptive responses in rats exposed to ozone, alone and in mixtures, with acidic aerosols. Inhal Toxicol 11(3):249-64.

Klemm RJ, Mason RM Jr, Heilig CM, Neas LM, Dockery DW (2000). Is daily mortality associated specifically with fine particles? Data reconstruction and replication of analyses. J Air Waste Manag Assoc 50(7):1215-22.

Kodavanti UP, Jaskort RH, Costa DL, Dreher KL (1997). Pulmonary proinflammatory gene induction following acute exposure to residual oil fly ash : roles of particle-associated metal . Inhal Toxicol 9:679-701.

Kodavanti UP, Jackson MC, Ledbetter AD, Richards JR, Gardner SY, Watkinson WP *et al*. (1999). Lung injury from intratracheal and inhalation exposures to residual oil fly ash in a rat model of monocrotaline-induced pulmonary hypertension. J Toxicol Environ Health A 57(8):543-63.

Kodavanti UP, Schladweiler MCJ, Ledbetter AD, *et al.* (2000a). The spontaneously hypertensive rat as a model of human cardiovascular disease: evidence of exacerbated cardiopulmonary injury and oxidative stress from inhaled emission particulate matter. Toxicol Appl Pharmacol 164:250-63.

Kodavanti UP, Mebane R, Ledbetter A, Krantz T, McGee J, Jackson MC *et al*. (2000b). Variable pulmonary responses from exposure to concentrated ambient air particles in a rat model of bronchitis. Toxicol Sci 54(2):441-51.

Kodavanti UP, Schladweiler MCJ, Richards JR, Costa DL (2001). Acute Lung injury from intratracheal exposure to fugitive residual oil fly ash and its constituent metals in normo- and spontaneously hypertensive rats. Inhal Toxicol 13:37-54.

Krewski D, Burnett R, Goldberg MS, Koover K, Siemiatycki J, Jerrett M *et al*. (2000). Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Res Rep Health Eff Inst (A special report of the Institute's Particle Epidemiology Reanalysis Project).

Kunzli N, Medina S, Kaiser R, Quenel P, Horak F, Studnicka M (2001). Assessment of deaths attributable to air pollution: should we use risk estimates based on time series or on cohort studies? Am J Epidemiol 153:1050-5.

Laden F, Neas LM, Dockery DW, Schwartz J (2000). Association of fine particulate matter from different sources with daily mortality in six U.S. cities. Environ Health Perspect 108(10):941-7.

Levy D, Sheppard L, Checkoway H, Kaufman J, Lumley T, Koenig J *et al*. (2001). A case-crossover analysis of particulate matter air pollution and out- of-hospital primary cardiac arrest. Epidemiology 12(2):193-9.

Li XY, Gilmour PS, Donaldson K, MacNee W (1996). Free radical activity and pro-inflammatory effects of particulate air pollution (PM10) in vivo and in vitro. Thorax 51(12):1216-22.

Li XY, Gilmour PS, Donaldson K, MacNee W (1997). In vivo and in vitro proinflammatory effects of particulate air pollution (PM10). Environ Health Perspect 105(Suppl. 5):1279-83.

Liao D, Creason J, Shy C, Williams R, Watts R, Zweidinger R (1999). Daily variation of particulate air pollution and poor cardiac autonomic control in the elderly. Environ Health Perspect 107(7):521-5.

Linn WS, Szlachcic Y, Gong H Jr., Kinney PL, Zweidinger R (2000). Air pollution and daily hospital admissions in metropolitan Los Angeles. Environ Health Perspect 108:427-34.

Lipfert FW, Morris SC, Wyzga RE (2000a). Daily mortality in the Philadelphia metropolitan area and size-classified particulate matter. J Air Waste Manag Assoc 50(8):1501-13.

Lipfert FW, Perry HM Jr, Miller JP, Baty JD, Wyzga RE, Carmody SE (2000b). The Washington University-EPRI Veterans' Cohort Mortality Study: Preliminary Results. Inhal Toxicol 12(Suppl. 4):41-73.

Lippmann M, Ito K, Nadas A, Burnett RT (2000). Association of particulate matter components with daily mortality and morbidity in urban populations. Res Rep Health Eff Inst (95):5-72, discussion 73-82.

Lipsett MJ (1995). The Hazards of Air Pollution to Children. In: Brooks SM, Gochfeld M, Jackson R, et al., eds. Environmental Medicine. St. Louis, MO: Mosby-Year Book, Inc., 390-7.

Lipsett MJ, Hurley S, Ostro B (1997). Air pollution and emergency room visits for asthma in Santa Clara County, California. Environ Health Perspect 105:216-22.

Loomis D, Castillejos M, Gold DR, McDonnell W, Borja-Aburto VH (1999). Air pollution and infant mortality in Mexico City. Epidemiology 10:118-23.

Mannino DM, Homa DM, Pertowski CA, Ashizawa A, Nixon LL, Johnson CA *et al*. (1998). Surveillance for asthma--United States, 1960-1995. Morb Mortal Wkly Rep CDC Surveill Summ 47(1):1-27.

Mar TF, Norris GA, Koenig JQ, Larson TV (2000). Associations between air pollution and mortality in Phoenix, 1995-1997. Environ Health Perspect 108(4):347-53.

Martin GJ, Magid NM, Eckberg DL, *et al.* (1987). Heart rate variability and sudden cardiac death during ambulatory monitoring. Clin Research 35(3):302A.

Mautz WJ, Bhalla DK, Kikkawa Y, *et al.* (1996). Studies to determine long term effects of acidic atmospheres. Sacramento, CA: California Air Resources Board; Contract No. A033-088.

McConnell R, Berhane K, Gilliland F, London S, Vora H, Avol E *et al*. (1999). Air pollution and bronchitic symptoms in southern California children with asthma. Environ Health Perspect 107:757-60.

McDonnell WF, Nishino-Ishikawa N, Petersen FF, Chen LH, Abbey DE (2000). Relationships of mortality with the fine and coarse fractions of long-term ambient PM10 concentrations in nonsmokers. J Expo Anal Environ Epidemiol 10(5):427-36.

Medina S, Le Tertre A, Quenel P, Le Moullec Y, Lameloise P, Guzzo JC *et al*. (1997). Air pollution and doctors' house calls: results from the ERPURS system for monitoring the effects of air pollution on public health in Greater Paris, France, 1991-1995. Environ Res 75:73-84.

Mensink GB, Hoffmeister H (1997). The relationship between resting heart rate and all-cause, cardiovascular and cancer mortality. Eur Heart J 18(9):1404-10.

Michel O, Nagy AM, Schroeven M, Duchateau J, Neve J, Fondu P *et al*. (1997). Dose-response relationship to inhaled endotoxin in normal subjects. Am J Respir Crit Care Med 156(4 Pt 1):1157-64.

Michelozzi P, Forastiere F, Fusco D, Perucci CA, Ostro B, Ancona C *et al*. (1998). Air pollution and daily mortality in Rome, Italy. Occup Environ Med 55(9):605-10.

Monn C, Becker S (1999). Cytotoxicity and induction of proinflammatory cytokines from human monocytes exposed to fine (PM2.5) and coarse particles (PM10-2.5) in outdoor and indoor air. Toxicol Appl Pharmacol 155(3):245-52.

Moolgavkar SH (2000a). Air Pollution and Daily Mortality in Three U.S. Counties. Environ Health Perspect 108(8):777-84.

Moolgavkar SH (2000b). Air Pollution and Hospital Admissions for Diseases of the Circulatory System in Three U.S. Metropolitan Areas. J Air Waste Manag Assoc 50:1199-206.

Moolgavkar SH (2000c). Air Pollution and Hospital Admissions for Chronic Obstructive Pulmonary Disease in Three Metropolitan Areas in the United States. Inhal Toxicol 12(Suppl. 4):75-90.

Moolgavkar SH, Luebeck EG, Anderson EL (1997). Air pollution and hospital admissions for respiratory causes in Minneapolis-St. Paul and Birmingham. Epidemiology 8:364-70.

Moolgavkar SH, Hazelton W, Luebeck EG, *et al.* (2000). Air pollution, pollens, and admissions for chronic respiratory disease in King County, Washington. Inhal Toxicol 12(Suppl. 1):157-71.

Morgan G, Corbett S, Wlodarczyk J, Lewis P (1998). Air pollution and daily mortality in Sydney, Australia, 1989 through 1993. Am J Public Health 88:759-64.

Morris RD, Naumova EN (1998). Carbon monoxide and hospital admissions for congestive heart failure: evidence of an increased effect at low temperatures. Environ Health Perspect 106(10):649-53.

Mukae H, Vincent R, Quinlan K, English D, Hards J, Hogg JC *et al*. (2001). The effect of repeated exposure to particulate air pollution (PM10) on the bone marrow. Am J Respir Crit Care Med 163(1):201-9.

Mullahy J, Portney PR (1990). Air pollution, cigarette smoking, and the production of respiratory health. J Health Econ 9(2):193-205.

Murray JF (1986). The normal lung: the basis for diagnosis and treatment of pulmonary disease. 2nd edition. Philadelphia: Saunders, 46.

Nauenberg E, Basu K (1999). Effect of insurance coverage on the relationship between asthma hospitalizations and exposure to air pollution. Public Health Rep 114:135-48.

Nel AE, Diaz-Sanchez D, Ng D, Hiura T, Saxon A (1998). Enhancement of allergic inflammation by the interaction between diesel exhaust particles and the immune system. J Allergy Clin Immunol 102:539-54.

Nel AE, Diaz-Sanchez D, Li N (2001). The role of particulate pollutants in pulmonary inflammation and asthma: evidence for the involvement of organic chemicals and oxidative stress. Curr Opin Pulm Med 7(1):20-6.

Nemmar A, Vanbilloen H, Hoylaerts MF, Hoet PH, Verbruggen A, Nemery B (2001a). Passage of Intratracheally Instilled Ultrafine Particles from the Lung into the Systemic Circulation in Hamster. Am J Respir Crit Care Med 164(9):1665-8.

Nemmar A, Vanbilloen H, Verbruggen A, *et al.* (2001b). Evaluation of the passage of inhaled 99Tc-labelled ultrafine carbon particles into the systemic circulation in humans. Am J Respir Crit Care Med .

Nightingale JA, Maggs R, Cullinan P, Donnelly LE, Rogers DF , Kinnersley R *et al*. (2000). Airway inflammation after controlled exposure to diesel exhaust particulates. Am J Respir Crit Care Med 162(1):161-6.

Nolan J, Batin PD, Andrews R, Lindsay SJ, Brooksby P, Mullen M *et al*. (1998). Prospective study of heart rate variability and mortality in chronic heart failure: results of the United Kingdom heart failure evaluation and assessment of risk trial (UK-heart). Circulation 98(15):1510-6.

Nordenhäll C, Pourazar J, Blomberg A, Levin JO, Sandstrom T, Adelroth E (2000). Airway inflammation following exposure to diesel exhaust: a study of time kinetics using induced sputum. Eur Respir J 15(6):1046-51.

Norris G, Larson T, Koenig J, Claiborn C, Sheppard L, Finn D (2000). Asthma aggravation, combustion, and stagnant air. Thorax 55(6):466-70.

Oldham MJ, Mannix RC, Phalen RF (1997). Deposition of monodisperse particles in hollow models representing adult and child-size tracheobronchial airways. Health Phys 72(6):827-34.

Oparil S (1992). Arterial Hypertension. In: Wyngaarden JB, Smith LH Jr, Bennett JC, eds. Cecil Textbook of Medicine. Philadelphia, PA: W. B. Saunders Co, 253-69.

Ostro BD (1987). Air pollution and morbidity revisited: a specification test. Journal of Environmental Economics and Management 14:87-98.

Ostro BD (1993). The association of air pollution and mortality: examining the case for inference. Arch Environ Health 48(5):336-42.

Ostro BD (1995). Fine particulate air pollution and mortality in two Southern California counties. Environ Res 70:98-104.

Ostro BD, Rothschild S (1989). Air pollution and acute respiratory morbidity: an observational study of multiple pollutants. Environ Res 50 :238-47.

Ostro BD, Lipsett MJ, Mann JK, Krupnick A, Harrington W (1993). Air pollution and respiratory morbidity among adults in southern California. Am J Epidemiol 137(7):691-700.

Ostro BD, Lipsett MJ, Mann J, Braxton-Owens H, White M (1995). Air pollution and asthma exacerbations among African-American children in Los Angeles. Inhal Toxicol 7:711-22.

Ostro BD, Sanchez JM, Aranda C, Eskeland GS (1996). Air pollution and mortality: results from a study in Santiago, Chile. J Expo Anal Environ Epidemiol 6:97-114.

Ostro B, Chestnut L, Vichit-Vadakan N, Laixuthai A (1999a). The impact of particulate matter on daily mortality in Bangkok, Thailand. J Air Waste Manag Assoc 49(9 Spec No):100-7.

Ostro BD, Hurley S, Lipsett MJ (1999b). Air pollution and daily mortality in the Coachella Valley, California: a study of PM10 dominated by coarse particles. Environ Res 81(3):231-8.

Ostro BD, Eskeland GS, Sanchez JM, Feyzioglu T (1999c). Air pollution and health effects: a study of medical visits among children in Santiago, Chile. Environ Health Perspect 107:69-73.

Ostro BD, Broadwin R, Lipsett MJ (2000). Coarse and fine particles and daily mortality in the Coachella Valley, California: a follow-up study. J Expo Anal Environ Epidemiol 10(5):412-9.

Ostro BD, Lipsett MJ, Mann J, Braxton-Owens H, White M (2001). Air pollution and exacerbation of asthma in African-American children in Los Angeles. Epidemiology 12:200-8.

Otterbein LE, Choi AM (2000). Heme oxygenase: colors of defense against cellular stress. Am J Physiol Lung Cell Mol Physiol 279(6):L1029-37.

Ozkaynak H, Thurston GD (1987). Associations between 1980 U.S. mortality rates and alternative measures of airborne particle concentration. Risk Anal 7(4):449-61.

Ozkaynak H, Xue J, Zhou H, Raizenne M (1996a). Association between daily mortality and motor vehicle pollution in Toronto, Canada, March 25, 1996. Boston, MA: Harvard University, School of Public Health, Dept. of Environmental Health.

Ozkaynak H, Xue J, Spengler J, Wallace L, Pellizzari E, Jenkins P (1996b). Personal exposure to airborne particles and metals: results from the Particle TEAM study in Riverside, California. J Expo Anal Environ Epidemiol 6(1):57-78.

Penna MLF, Duchiade MP (1991). Air pollution and infant mortality from pneumonia in the Rio de Janeiro metropolitan area. Bulletin of PAHO 25:47-54.

Peters A, Doring A, Wichmann HE, Koenig W (1997). Increased plasma viscosity during an air pollution episode: a link to mortality? Lancet 349(9065):1582-7.

Peters A, Perz S, Doring A, Stieber J, Koenig W, Wichmann HE (1999c). Increases in heart rate during an air pollution episode. Am J Epidemiol 150(10):1094-8.

Peters A, Liu E, Verrier RL, Schwartz J, Gold DR, Mittleman M *et al*. (2000a). Air pollution and incidence of cardiac arrhythmia. Epidemiology 11(1):11-7.

Peters A, Perz S, Doring A, Stieber J, Koenig W, Wichmann HE (2000b). Activation of the autonomic nervous system and blood coagulation in association with an air pollution episode. Inhal Toxicol 12(Suppl. 2):51-61.

Peters A, Dockery DW, Muller JE, Mittleman MA (2001a). Increased particulate air pollution and the triggering of myocardial infarction. Circulation 103(23):2810-5.

Peters A, Frohlich M, Doring A, Immervoll T, Wichmann HE, Hutchinson WL *et al*. (2001b). Particulate air pollution is associated with an acute phase response in men; results from the MONICA-Augsburg Study. Eur Heart J 22(14):1198-204.

Peters JM, Avol E, Navidi W, et al. (1999a). A study of twelve southern California communities with differing levels and types of air pollution. I. Prevalence of respiratory morbidity. Am J Respir Crit Care Med 159:760-7.

Peters JM, Avol E, Gauderman WJ, et al. (1999b). A study of twelve southern California communities with differing levels and types of air pollution. II. Effects on pulmonary function. Am J Respir Crit Care Med 159: 768-75.

Phalen RF, Oldham MJ, Beaucage CB, Crocker TT, Mortensen JD (1985). Postnatal enlargement of human tracheobronchial airways and implications for particle deposition. Anat Rec 212(4):368-80.

Ponka A, Savela M, Virtanen M (1998). Mortality and air pollution in Helsinki. Arch Environ Health 53(4):281-6.

Pope CA III (1996). Particulate pollution and health: a review of the Utah valley experience. J Expo Anal Environ Epidemiol 6(1):23-34.

Pope CA III (2000). Epidemiology of fine particulate air pollution and human health: biologic mechanisms and who's at risk? Environ Health Perspect 108(Suppl. 4):713-23.

Pope CA III, Dockery DW (1992). Acute health effects of PM10 pollution on symptomatic and asymptomatic children. Am Rev Respir Dis 145:1123-8.

Pope CA III, Schwartz J, Ransom MR (1992). Daily Mortality and PM10 Pollution in Utah Valley. Arch Environ Health 47:211-7.

Pope CA III, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE *et al*. (1995). Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Respir Crit Care Med 151:669-74.

Pope CA III, Kalkstein LS (1996). Synoptic weather modeling and estimates of the exposure-response relationship between daily mortality and particulate air pollution. Environ Health Perspect 104:414-20.

Pope CA III, Hill RW, Villegas GM (1999a). Particulate air pollution and daily mortality on Utah's Wasatch Front. Environ Health Perspect 107(7):567-73 .

Pope CA III, Dockery DW, Kanner RE, Villegas GM, Schwartz J (1999b). Oxygen saturation, pulse rate, and particulate air pollution: a daily time-series panel study. Am J Respir Crit Care Med 159:365-72.

Pope CA III, Verrier RL, Lovett EG, Larson AC, Raizenne ME , Kanner RE *et al*. (1999c). Heart rate variability associated with particulate air pollution. Am Heart J 138(5 Pt 1):890-9.

Pope CA III, Eatough DJ, Gold DR, Pang Y, Nielsen KR, Nath P *et al*. (2001). Acute exposure to environmental tobacco smoke and heart rate variability. Environ Health Perspect 109(7):711-6.

Pozzati A, Pancaldi LG, Di Pasquale G, Pinelli G, Bugiardini R (1996). Transient sympathovagal imbalance triggers "ischemic" sudden death in patients undergoing electrocardiographic Holter monitoring. J Am Coll Cardiol 27(4):847-52.

Prescott GJ, Cohen GR, Elton RA, Fowkes FG, Agius RM (1998). Urban air pollution and cardiopulmonary ill health: a 14.5 year time series study. Occup Environ Med 55:697-704.

QUARG (1993). Diesel vehicle emissions and urban air quality. Second Report of the Quality of Urban Air Review Group, Institute of Public and Environmental Health. School of Biological Sciences, University of Birmingham, England.

Raizenne M, Neas LM, Damokosh AI, Dockery DW, Spengler JD, Koutrakis P *et al*. (1996). Health effects of acid aerosols on North American children: pulmonary function. Environ Health Perspect 104(5):506-14.

Ransom MR, Pope CA III (1992). Elementary school absences and PM10 pollution in Utah Valley. Environ Res 58:204-19.

Renwick LC, Donaldson K, Clouter A (2001). Impairment of alveolar macrophage phagocytosis by ultrafine particles. Toxicol Appl Pharmacol 172(2):119-27.

Rifai N, Ridker PM (2001). High-sensitivity C-reactive protein: a novel and promising marker of coronary heart disease. Clin Chem 47(3):403-11.

Ritz B, Yu F, Chapa G, Fruin S (2000). Effect of air pollution on preterm birth among children born in Southern California between 1989 and 1993. Epidemiology 11(5):502-11.

Romieu I, Meneses F, Ruiz S, Sienra JJ, Huerta J, White MC *et al* . (1996). Effects of air pollution on the respiratory health of asthmatic children living in Mexico City. Am J Respir Crit Care Med 154(2 Pt 1):300-7 .

Rosito GB, Tofler GH (1996). Hemostatic factors as triggers of cardiovascular events. Cardiol Clin 14(2):239-50.

Rothman KJ (1982). Causation and causal inference. In: Schottenfeld D, Fraumeni JF. Cancer Epidemiology and Prevention. Philadelphia, PA: W.B Saunders Co., 15-22.

Saldiva PHN, Bohm GM (1995). Air pollution and mortality in Sao Paulo, Brazil. Paper Faculty of Medicine, University of Sao Paulo :1-19.

Salvi S, Blomberg A, Rudell B, Kelly F, Sandstrom T, Holgate ST *et al*. (1999). Acute inflammatory responses in the airways and peripheral blood after short-term exposure to diesel exhaust in healthy human volunteers. Am J Respir Crit Care Med 159(3):702-9.

Salvi SS, Nordenhall C, Blomberg A, Rudell B, Pourazar J, Kelly FJ *et al*. (2000). Acute exposure to diesel exhaust increases IL-8 and GRO-alpha production in healthy human airways. Am J Respir Crit Care Med 161(2 Pt 1):550-7.

Samet J, Zeger S, Kelsall J, Xu J, Kalkstein L (1998). Does weather confound or modify the association of particulate air pollution with mortality? Environ Res 77:9-19.

Samet JM, Zeger SL, Dominici F, Curriero F, Coursac I, Dockery DW *et al*. (2000a). The National Morbidity, Mortality, and Air Pollution Study. Part II: Morbidity and mortality from air pollution in the United States. Res Rep Health Eff Inst (94 Pt 2):5-70; discussion 71-9.

Samet JM, Dominici F, Curriero FC, Coursac I, Zeger SL (2000b). Fine particulate air pollution and mortality in 20 U.S. cities, 1987- 1994. N Engl J Med 343(24):1742-9.

Sarnat JA, Schwartz J, Catalano PJ, Suh HH (2001). Gaseous pollutants in particulate matter epidemiology: confounders or surrogates? Environ Health Perspect 109(10):1053-61.

Sato S, Iso H, Naito Y, Kiyama M, Kitamura A, Iida M *et al*. (1996). Plasma fibrinogen and its correlates in urban Japanese men. Int J Epidemiol 25(3):521-7.

Schiller-Scotland CF, Hlawa R, Gebhart J (1994). Experimental data for total deposition in the respiratory tract of children. Toxicol Lett 72(1-3):137-44.

Schlesinger RB, Gorczynski JE, Dennison J, Richards L, Kinney PL, Bosland MC (1992). Long-term intermittent exposure to sulfuric acid aerosol, ozone, and their combination: alterations in tracheobronchial mucociliary clearance and epithelial secretory cells. Exp Lung Res 18(4):505-34.

Schwartz J (1993). Air pollution and daily mortality in Birmingham, Alabama. Am J Epidemiol 137:1136-47.

Schwartz J (1994a). Air pollution and daily mortality: a review and meta analysis. Environ Res 64:36-52.

Schwartz J (1994b). What are people dying of on high air pollution days? Environ Res 64:26-35.

Schwartz J (1994c). PM10, ozone, and hospital admissions for the elderly in Minneapolis-St. Paul, Minnesota. Arch Environ Health 49(5):366-74.

Schwartz J (1995). Short-term fluctuations in air pollution and hospital admissions of the elderly for respiratory disease. Thorax 50:531-8.

Schwartz J (1996). Air pollution and hospital admissions for respiratory disease. Epidemiology 7:20-8.

Schwartz J (1997). Air pollution and hospital admissions for cardiovascular disease in Tucson. Epidemiology 8:371-7.

Schwartz J (2000a). Assessing confounding, effect modification, and thresholds in the association between ambient particles and daily deaths. Environ Health Perspect 108(6):563-8.

Schwartz J (2000b). The distributed lag between air pollution and daily deaths. Epidemiology 11:320-6.

Schwartz J (2000c). Harvesting and long term exposure effects in the relation between air pollution and mortality. Am J Epidemiology 151:440-8.

Schwartz J (2001a). Is there harvesting in the association of airborne particles with daily deaths and hospital admissions? Epidemiology 12(1):55-61.

Schwartz J (2001b). Air pollution and blood markers of cardiovascular risk. Environ Health Perspect 109(Suppl. 3):405-9.

Schwartz J, Neas LM (2000). Fine particles are more strongly associated than coarse particles with acute respiratory health effects in school children. Epidemiology 11:6-10.

Schwartz J, Zanobetti A (2000). Using meta-smoothing to estimate dose-response trends across multiple studies, with application to air pollution and daily death. Epidemiology 11(6):666-72.

Schwartz J, Slater D, Larson TV, Pierson WE, Koenig JQ (1993). Particulate air pollution and hospital emergency room visits for asthma in Seattle. Am Rev Respir Dis 147:826-31.

Schwartz J, Dockery DW, Neas LM, *et al.* (1994). Acute effects of summer air pollution on respiratory symptom reporting in children. Am J Respir Crit Care Med 150:1234-42.

Schwartz J, Dockery DW, Neas LM (1996). Is daily mortality associated specifically with fine particles? J Air Waste Manag Assoc 46:927-39.

Schwartz J, Norris G, Larson T, Sheppard L, Claiborne C, Koenig J (1999). Episodes of high coarse particle concentrations are not associated with increased mortality. Environ Health Perspect 107(5):339-42.

Seaton A, MacNee W, Donaldson K, Godden D (1995). Particulate air pollution and acute health effects. Lancet 345:126-78.

Seaton A, Soutar A, Crawford V, Elton R, McNerlan S, Cherrie J *et al*. (1999). Particulate air pollution and the blood. Thorax 54(11):1027-32.

Sheppard L, Levy D, Norris G, Larson TV, Koenig JQ (1999). Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington, 1987-1994. Epidemiology 10:23-30.

Sherman M, Goldstein E, Lippert W, Wennberg R (1977). Neonatal lung defense mechanisms: a study of the alveolar macrophage system in neonatal rabbits. Am Rev Respir Dis 116(3):433-40.

Shukla A, Timblin C, BeruBe K, Gordon T, McKinney W, Driscoll K *et al*. (2000). Inhaled particulate matter causes expression of nuclear factor (NF)-kB-related genes and oxidant-dependent NF-kB activation in vitro. Am J Respir Cell Mol Biol 23:182-7.

Simpson RW, Williams G, Petroeschevsky A, Morgan G, Rutherford S (1997). Associations between outdoor air pollution and daily mortality in Brisbane, Australia. Arch Environ Health 52(6):442-54.

Smith RL, Spitzner D, Kim Y, Fuentes M (2000). Threshold dependence of mortality effects for fine and coarse particles in Phoenix, Arizona. J Air Waste Manag Assoc 50(8):1367-79.

Snipes MB, James AC, Jarabek AM (1997). The 1994 ICRP66 human respiratory tract dosimetry model as a tool for predicting lung burdens from exposures to environmental aerosols. Appl Occup Environ Hyg 12:547-54.

Snodgrass WR (1992). Physiological and biochemical differences between children and adults as determinants of toxic response to environmental pollutants. In: Guzelian PS, Henty CJ, Olin SS, eds. Similarities and differences between children and adults. ILSI Press, 35-42.

Soukup JM, Becker S (2001). Human alveolar macrophage responses to air pollution particulates are associated with insoluble components of coarse material, including particulate endotoxin. Toxicol Appl Pharmacol 171(1):20-6.

Spektor DM, Yen BM, Lippmann M (1989). Effect of concentration and cumulative exposure of inhaled sulfuric acid on tracheobronchial particle clearance in healthy humans. Environ Health Perspect 79:167-72.

Stieb DM, Beveridge RC, Brook JR, Smith-Doiron M, Burnett RT, Dales RE *et al*. (2000). Air pollution, aeroallergens and cardiorespiratory emergency department visits in Saint John, Canada. J Expo Anal Environ Epidemiol 10(5):461-77.

Swift DL, Strong JS (1996). Nasal deposition of ultrafine 218 Po aerosols in human subjects. J Aerosol Sci 27:1125-32.

Szabó BM, van Veldhuisen DJ, van der Veer N, Brouwer J, De Graeff PA, Crijns HJ (1997). Prognostic value of heart rate variability in chronic congestive heart failure secondary to idiopathic or ischemic dilated cardiomyopathy. Am J Cardiol 79(7):978-80.

Tan WC, Qiu D, Liam BL, Ng TP, Lee SH, van Eeden SF *et al*. (2000). The human bone marrow response to acute air pollution caused by forest fires. Am J Respir Crit Care Med 161(4 Pt 1):1213-7 .

Tashkin DP, Detels R, Simmons M, Liu H, Coulson AH, Sayre J *et al*. (1994). The UCLA population studies of chronic obstructive respiratory disease: XI. Impact of air pollution and smoking on annual change in forced expiratory volume in one second. Am J Respir Crit Care Med 149:1209-17.

Thurston GD, Ito K (2001). Epidemiological studies of acute ozone exposures and mortality. J Expo Anal Environ Epidemiol 11:1-9.

Thurston GD, Ito K, Lippmann M, Hayes C (1989). Reexamination of London, England, mortality in relation to exposure to acidic aerosols during 1963-1972 winters. Environ Health Perspect 79:73-82.

Thurston GD, Lippmann M, Scott MB, Fine JM (1997). Summertime haze air pollution and children with asthma. Am J Respir Crit Care Med 155(2):654-60.

Tiittanen P, Timonen KL, Ruuskanen J, Mirme A, Pekkanen J (1999). Fine particulate air pollution, resuspended road dust and respiratory health among symptomatic children. Eur Respir J 13(2):266-73.

Tolbert PE, Mulholland JA, MacIntosh DL, *et al.* (2000). Air quality and pediatric emergency room visits for asthma in Atlanta, Georgia. Am J Epidemiol 151(8):798-810.

Tsai FC, Apte MG, Daisey JM (2000). An exploratory analysis of the relationship between mortality and the chemical composition of airborne particulate matter. Inhal Toxicol 12(Suppl. 2):121-35.

Tsuji H, Venditti FJ Jr, Manders ES, Evans JC, Larson MG, Feldman CL *et al*. (1994). Reduced heart rate variability and mortality risk in an elderly cohort. The Framingham Heart Study. Circulation 90(2):878-83.

Tsuji H, Venditti FJ Jr, Manders ES, Evans JC, Larson MG, Feldman CL *et al*. (1996). Determinants of heart rate variability. J Am Coll Cardiol 28(6):1539-46.

U.S. Environmental Protection Agency (1989). An acid aerosols issue paper: health effects and aerometrics. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC, EPA-600/8-88-005F.

U.S. Environmental Protection Agency (1996). Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information: OAQPS staff paper. Office of Air Quality Planning and Standards, Research Triangle Park, NC, EPA-452\R-96-013.

U.S. Environmental Protection Agency (1999). The benefits and costs of the Clean Air Act 1990 to 2010. Office of Air and Radiation and Office of Policy, Washington DC, EPA-410-R-99-001.

U.S. Environmental Protection Agency (2000). Regulatory Impact Analysis: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements. Office of Air and Radiation, Research Triangle Park, NC, EPA-420-R-00-026.

van der Zee SC, Hoek G, Boezen HM, *et al.* (1999). Acute effects of urban air pollution on respiratory health of children with and without chronic respiratory symptoms. Occup Environ Med 56(12):802-12.

Vedal S, Petkau J, White R, Blair J (1998). Acute effects of ambient inhalable particles in asthmatic and nonasthmatic children. Am J Respir Crit Care Med 157:1034-43.

Verhoeff AP, Hoek G, Schwartz J, van Wijnen JH (1996). Air pollution and daily mortality in Amsterdam. Epidemiology 7:225-30.

Veronesi B, Oortgiesen M, Roy J, Carter JD, Simon SA, Gavett SH (2000). Vanilloid (capsaicin) receptors influence inflammatory sensitivity in response to particulate matter. Toxicol Appl Pharmacol 169(1):66-76 .

Vogelzang PF, van der Gulden JW, Folgering H, Kolk JJ, Heederik D, Preller L *et al*. (1998). Endotoxin exposure as a major determinant of lung function decline in pig farmers. Am J Respir Crit Care Med 157(1):15-8.

Ware JH, Ferris BG Jr, Dockery DW, Spengler JD, Stram DO, Speizer FE (1986). Effects of ambient sulfur oxides and suspended particles on respiratory health of preadolescent children. Am Rev Respir Dis 133:834-42.

Watkinson WP, Campen MJ, Costa DL (1998). Cardiac arrhythmia induction after exposure to residual oil fly ash particles in a rodent model of pulmonary hypertension. Toxicol Sci 41(2):209-16.

Watkinson WP, Campen MJ, Dreher KL, Su W-Y, Kodavanti UP, Highfill JW *et al*. (2000). Thermoregulatory effects following exposure to particulate matter in healthy and cardiopulmonary-compromised rats. J Therm Biol 25:131-7.

Wichmann H-E, Spix C, Tuch T, Wolke G, Peters A, Heinrich J *et al*. (2000). Daily mortality and fine and ultrafine particles in Erfurt, Germany: part I: Role of particle number and particle mass. Res Rep Health Eff Inst (98):5-86, discussion 87-96.

Wong TW, Lau TS, Yu TS, Neller A, Wong SL, Tam W *et al*. (1999). Air pollution and hospital admissions for respiratory and cardiovascular diseases in Hong Kong. Occup Environ Med 56 (10):679-83.

Woodruff TJ, Grillo J, Schoendorf KC (1997). The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States. Environ Health Perspect 105:608-12.

Wordley J, Walters S, Ayres JG (1997). Short term variations in hospital admissions and mortality and particulate air pollution. Occup Environ Med 54(2):108-16.

Yarnell JW, Baker IA, Sweetnam PM, Bainton D, O'Brien JR, Whitehead PJ *et al*. (1991). Fibrinogen, viscosity, and white blood cell count are major risk factors for ischemic heart disease. The Caerphilly and Speedwell collaborative heart disease studies. Circulation 83(3):836-44.

Yu O, Sheppard L, Lumley T, Koenig JQ, Shapiro GG (2000). Effects of ambient air pollution on symptoms of asthma in Seattle-area children enrolled in the CAMP study. Environ Health Perspect 108(12):1209-14.

Zanobetti A, Schwartz J (2000). Race, gender, and social status as modifiers of the effects of PM10 on mortality. J Occup Environ Med 42(5):469-74.

Zanobetti A, Schwartz J (2001). Are diabetics more susceptible to the health effects of airborne particles? Am J Respir Crit Care Med 164(5):831-3.

Zanobetti A, Schwartz J, Dockery DW (2000a). Airborne particles are a risk factor for hospital admissions for heart and lung disease. Environ Health Perspect 108(9):1071-7.

Zanobetti A, Schwartz J, Gold D (2000b). Are there sensitive subgroups for the effects of airborne particles? Environ Health Perspect 108(9):841-5.

Zeger SL, Dominici F, Samet J (1999). Harvesting-resistant estimates of air pollution effects on mortality. Epidemiology 10(2):171-5.

Zeger SL, Thomas D, Dominici F, Samet JM, Schwartz J, Dockery D *et al*. (2000). Exposure measurement error in time-series studies of air pollution: concepts and consequences. Environ Health Perspect 108(5):419-26.

Zemp E, Elsasser S, Schindler C, Kunzli N, Perruchoud AP, Domenighetti G *et al*. (1999). Long-term ambient air pollution and respiratory symptoms in adults (SAPALDIA study). The SAPALDIA Team. Am J Respir Crit Care Med 159(4 Pt 1):1257-66.

Zhang H, Triche E, Leaderer B (2000). Model for the analysis of binary time series of respiratory symptoms. Am J Epidemiol 151(12):1206-15.

Zock JP, Hollander A, Heederik D, Douwes J (1998). Acute lung function changes and low endotoxin exposures in the potato processing industry. Am J Ind Med 33(4):384-91.

8. Welfare Effects of Particulate Matter

8.1 Standards and "Welfare Effects"

"Welfare effects" includes all air pollutant impacts unrelated to human health. The manner in which these effects are evaluated depends on the legal authority for standard setting and how these effects bear on the standard in question. The California State standard setting environment is distinct from that under Federal law.

Under the Federal Clean Air Act (FCAA) (42 USC Ss 108 & 109) the National Ambient Air Quality Standard (NAAQS) for a particular pollutant consists of a "primary" standard aimed at protecting public health, and a "secondary" standard addressing welfare effects (if such effects exist). For gaseous air pollutants, such as ozone, the "primary-secondary" model allows the regulatory process to distinguish between an exposure (a specific concentration and duration) that causes human health impacts and other exposures that cause environmental and/or economic impacts.

Unlike a chemically homogeneous gaseous pollutant, particulate matter is a complex mixture of chemicals distributed over a wide range of particle sizes, with wide variation of chemical composition across particle size ranges. Moreover particle size and composition vary over time and between geographic areas. Consequently, the effects of particulate matter reflect its heterogeneous nature – different materials in different size ranges may have very different effects.

California law allows broad flexibility for air quality standards to address "public health, safety, and welfare, including, but not limited to, health, illness, irritation to the senses, aesthetic value, interference with visibility, and effects on the economy" [H&SC 39606(a)(2)]. In establishing the State PM10 Standard, the Air Resources Board declared that PM10 is "the fraction of inhalable particles which cause adverse health effects" and it should be "specifically addressed in a health-based standard" (ARB, 1982).

California has legal authority to define additional standards to specifically address other particulate matter effects. The PM10 standard is, therefore, not burdened with the requirement to cover all aspects of particulate matter pollution, and a separate State standard for "Visibility Reducing Particles" was created to address the dominant welfare effect of particulate matter - haze.

This section presents a brief overview of welfare effects and their regulation under State and Federal law to place the present PM10 review in the larger context of the role of particulate matter in the global environment.

8.2 Optical Effects: Visibility and Climate

The effects of particulate matter (aerosols) on visibility and climate are caused by the same optical processes. Visibility is reduced when aerosols interfere with light passing between an observer and a distant target; climate effects occur when aerosols interfere with incoming solar radiation or outgoing terrestrial radiation, changing the net energy balance between Sun and Earth. Where, how, and how intensely these interactions occur determines whether or not they are matters of regulatory concern. (The following discussion is highly simplified, the reader is referred to Friedlander (1977) for a full review of aerosol optics.)

8.2.1 How Particles Interact with Light

When a beam of electromagnetic radiation ("light") encounters the gases, particles and droplets that comprise the atmosphere, some light is scattered, some is absorbed, and some continues along its original path. The obscuring quality of a particular volume of air is termed "turbidity"; the experience of turbidity is the perception of "haze." The reduction of intensity of a beam of light as it moves through the atmosphere is termed "extinction," expressed as the "extinction coefficient" – the natural logarithm of the fractional change in intensity per unit distance (Middleton, 1952). Extinction is conventionally reported in units of "inverse megameters" (1/1,000,000m, or "Mm^{-1}"). Extinction is defined by the fundamental radiation transfer equation:

$$I_1 = I_0 \, e^{-Bext * d} \qquad (8.1)$$

where I_0 is the intensity of a beam at the beginning of the beam path, I_1 is the intensity at the end of the path, e is the root of natural logarithms (2.718…), B_{ext} is the extinction coefficient per unit distance, and d is the path length.

Under typical ambient conditions, extinction by various materials and processes is additive. Total extinction is the sum of scattering and absorption:

$$B_{ext} = B_{scat} + B_{abs}$$

Total extinction can be directly measured by observing the reduced intensity of a beam of light over a fixed distance, or scattering and absorption can be measured independently (monitoring methods are addressed below).

The strength of extinction is a function of the wavelength of the light, the density of the air, and the concentration, size and chemical composition of particles and droplets (aerosols). The extinction coefficient is additive, consisting of the sum of independent extinction due to n components of the atmosphere:

$$B_{ext} = \Sigma_{(1-n)} B_i * C \qquad (8.2)$$

where B_i is the extinction coefficient per unit mass for the i-th component, and C_i is the mass concentration of the i-th component.

8.2.2 Components of Extinction

Extinction can be represented as the linear sum of four generic components: scattering and absorption by both gases and particles. This is represented by the equation:

$$B_{ext} = B_{sg} + B_{ag} + B_{sp} + B_{ap}$$

Assessing the causes of strong extinction usually involves addressing each of these components separately.

Under typical ambient air conditions, B_{sg}, also known as "Rayleigh scattering," is a function of air density (thus a function of altitude), and proportional to the fourth power of wave number (inverse of wavelength):

$$B_{sg} (Mm^{-1}) = 12 * \rho \quad (500/\lambda)^4 / (.00123)$$

where ρ = air density (g/cm^3) and λ = wavelength (nm).

For green light (the middle of the visible range) at standard conditions:

$$B_{sg} = 12 \; Mm^{-1}$$

Light absorption by gases, B_{ag}, for clean air and visible light, is practically zero. In urban atmospheres, nitrogen dioxide absorbs blue light, causing a yellowing of the sky and distant

targets. Outside the visible range, gaseous components of the atmosphere exhibit strong absorption at various wavelengths in both the ultraviolet (especially ozone) and infrared (especially water vapor and carbon dioxide) portions of the electromagnetic spectrum.

B_{sp} is generally the largest component of extinction. For a given mass of aerosol, the largest determinant of scattering is particle size. If the aerosol size distribution and composition are held constant, then, for typical atmospheric particle loads, scattering will be proportional to particle concentration. If the particles contain hydrophilic chemicals (e.g., nitrates, sulfates), the size distribution may change with humidity. Raising humidity will promote particle growth through absorption of water, increasing scattering with no change in pollutant concentration.

The scattering power for a particular amount of aerosol can be expressed as effective surface area - the scattering cross-section (cm^2). If the volume or mass or aerosol is known, "scattering efficiency" can be expressed, respectively, as the volumetric scattering efficiency (cm^2/cm^3) or the mass scattering efficiency (cm^2/g). Using appropriate units:

B_{sp} (Mm^{-1}) = Efficiency (m^2/g) * Concentration ($\mu g/m^3$)

(see eqn. 8.2 above)

Particle size is very important for scattering (Friedlander, 1977).

The relationship between size and particle scattering efficiency for monochromatic light (a single wavelength) is plotted in Figure 8.1. The scattering cross section of particles much smaller than the wavelength of the light being scattered ($d/\lambda<0.1$) is negligible. For particles much larger than the wavelength ($d/\lambda>10$), the effective cross section tends toward twice the actual cross section. For particles near the wavelength, complex electrical interaction between light waves and particles accentuates scattering, increasing it to about 4 times the particle cross section for particles near $d/\lambda = 2$ (a process known as Mie scattering).

Expanding to all wavelengths of visible light, scattering efficiency is near zero for particles less than .05 μm diameter, less than 1 m^2/cm^3 for particles near 0.1 μm diameter, rises to a peak at about 10 m^2/cm3 in the range .4 to .7 μm diameter, then falls to less than 1 m^2/cm^3 for particles greater than 2 μm diameter and continues to decrease as the inverse of diameter for larger particles (Friedlander, 1977). Applying these physical characteristics to observed ambient aerosol size distributions, Friedlander (1977) calculated that light scattering is dominated by the population of particles between 0.2 and 2 μm diameter (Figure 8.1).

Absorption by aerosols, B_{ap}, is essentially a function of particle composition and total aerosol surface area. The strongest and most common absorbing aerosols are composed of nearly pure carbon ("elemental carbon", "EC", or "soot"); some soil materials also absorb some visible light (especially iron oxides), but with only a fraction of the efficiency of carbonaceous aerosols. Aerosols composed of a mixture of EC and other materials exhibit intermediate absorption efficiencies, roughly proportional to their EC content. Absorption is moderately sensitive to particle size. Very small particles ($d/\lambda<0.1$) don't interact efficiently with light waves. For particles with $d/\lambda>0.3$, absorption is roughly linear to total particle surface area, with the influence of particle size driven by the geometric decrease in surface area/volume ratio as size increases ($m^2/cm^3 \propto d^{2/3}$).

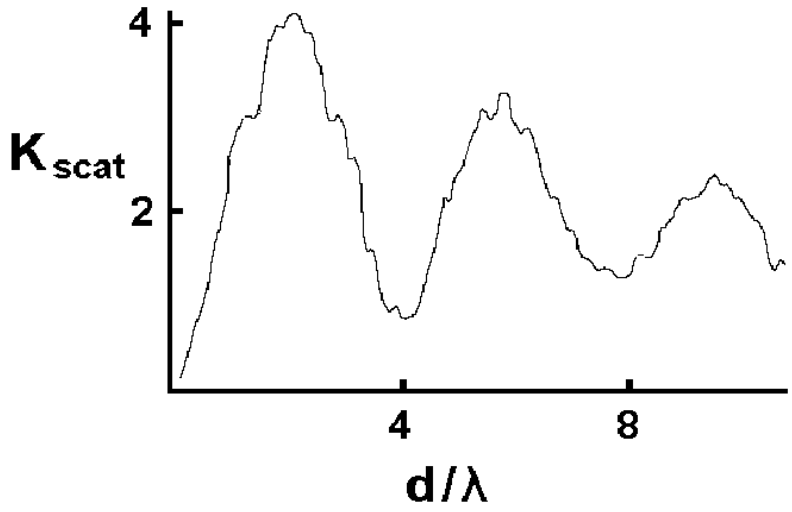


Figure 8.1a Monochromatic single particle scattering (Mie scattering; Friedlander, 1977).

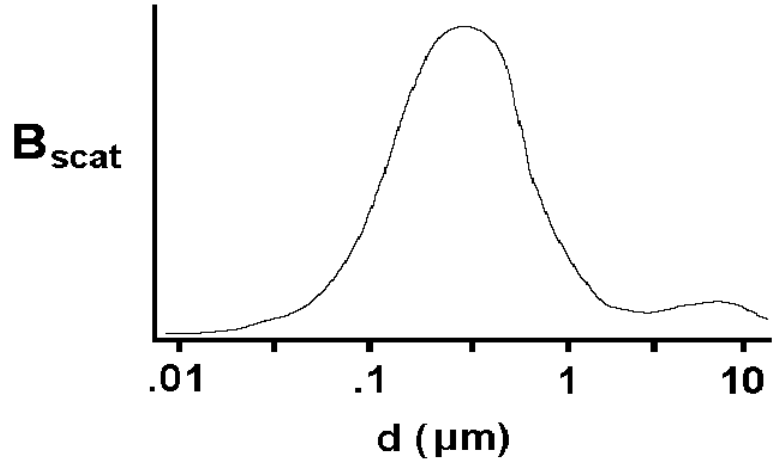


Figure 8.1b Particle light scattering vs. size in a typical urban aerosol (Friedlander, 1977).

8.2.3 PM10 and Extinction

Uniting the foregoing theoretical discussion and the discussion of PM composition in an earlier chapter (Chapter 3), it is evident that the smaller particles within PM10 (i.e., those below 2.5 μm diameter) play the dominant role in light extinction. Analysis of detailed aerosol data from over 5000 samples taken at 36 undeveloped rural sites across the United States (Sisler, et al., 1993) indicates that fine aerosols (PM2.5) exhibit 2 to 20 times more extinction efficiency per unit mass than do coarse particles (i.e., between 2.5 and 10 μm diameter), depending on chemical composition and relative humidity. For remote rural sites in California, their data (Sisler, et al., 1993) show that average coarse material light extinction is consistently less than 10 percent of the total. In urban areas, where fine material is more abundant, the coarse particle contribution to extinction is frequently even smaller.

8.2.4 Visibility

"Vision" is a psychophysical process involving light focusing and perception by the eye and image formation and interpretation by the brain. The process is subject to basic physiological limitations such as light sensitivity, spatial resolution, and color differentiation. Psychological processes control the brain's conversion of optic nerve signals into the perceptual components of vision, such as image formation, object recognition, and esthetic appreciation. Visual acuity varies among individuals due to interacting factors of physical and perceptual capabilities and acquired skill due to training and experience [this discussion is necessarily simplified, the reader is referred to Middleton (1952), ARB (1989), and Malm (1999)].

"Visibility" refers to the perceptibility of a distant target or scene. Variation of illumination, contrast, color, spatial frequency (target size and detail), background, foreground, etc., and the psychophysical variations among potential viewers combine to make "visibility" a very subjective concept. Managing visibility requires developing policy tools (such as air quality standards) that link physical qualities of the atmosphere to the subjective human experience of haze (ARB, 1989). This requires accepting a fundamental abstraction: regulating and managing the optical density of the air is a reasonable substitute for regulating the quality of human visual experience (ARB, 1989).

8.2.5 Measuring Visibility

8.2.5.1 Visual Range

In order to characterize atmospheric turbidity consistently and repeatably, measurements need to be standardized. "Visual Range" (V_r), in the parlance of meteorology or air pollution, is an operationally defined observation: the greatest distance at which a large black object can be distinguished from the background sky around a majority of the horizon circle. This method reduces the variation among definitions of "visibility," but imposes other strict limitations by requiring sites with clear views of the horizon in all directions and dark objects to view at varying distances in all directions. Moreover, it does not address differences among viewers. Nonetheless, visual range data are the best source of historical visibility information (Trijonis, 1980).

Visual Range data from many stations are significantly biased by lack of appropriate viewing targets. Historically, most Visual Range data have been recorded as part of routine weather observations at airports. Since low visibility impairs airport operations, "Airport Visibility" records are often biased toward reporting low visibility events, while moderate and good visibility are frequently grouped together as "greater than 10 miles" or "greater than 30 miles" (Trijonis, 1980). As weather observations have been increasingly automated, "Meteorological Visibility" (Visual Range) observations at many locations have been replaced with instruments calibrated

to replicate human observations; unfortunately, these instrumental records also replicate the bias toward measuring low visibility.

Visual Range from airport observations can be related to extinction if appropriate assumptions are applied. Human perception is much more sensitive to contrast than absolute light intensity (Middleton, 1952), so Visual Range can be restated as the distance at which a dark target (inherent contrast with the background sky ≅ 100%) is barely discernable to a human observer ["liminal contrast" threshold for detection ≅ 5% (Trijonis, 1980)]. The fundamental radiation transfer equation (eq. 1) applies for contrast as well as for intensity, so, substituting contrast for intensity in eq. 1 gives:

$$C_1 = C_0 \, e^{-Bext * d} \qquad (8.3)$$

where C_0 is the scene contrast at the target and C_1 is the apparent scene contrast at the viewer's location.

Algebraically transforming eq. 3 to relate distance (d, or in this case, V_r) to extinction (B_{ext}) and using the contrast assumptions above and units of Mm^{-1} gives:

$$V_r = 2996 / B_{ext} \qquad (8.4)$$

where V_r is in km,

or

$$V_r = 1857 / B_{ext}$$

where V_r is in miles.

The relationship in eq. 4 is generally known as the Koschmieder equation (Middleton, 1952).

Correcting for the limitations of airport data, Trijonis (1980) compiled a statewide assessment of visibility in California. Although there have been some reductions in aerosol loading in parts of the state, the general patterns he found still exist. No more recent statewide review exists. Figure 8.2 shows Trijonis' map of average visual range in California.

8.2.5.2 Instrumental Measurements

Since meteorological records are imperfect sources of visibility data, both Federal and California visibility monitoring programs use specialized monitoring methods designed to characterize "visual air quality" in a manner compatible with routine air quality management programs.

By measuring the physical property of "extinction" or its components (scattering and absorption) instrumentally, the "human factor" is eliminated altogether. Extinction can be related to measured aerosol characteristics (mass, size, chemical composition, etc.) both empirically (e.g., through regression analyses) and by calculating extinction "from first principles" using detailed knowledge of aerosol characteristics. These approaches allow management of visual air quality through the same types of measurement, modeling, and control programs that are used for other air quality purposes.

California's instrumental measurement of extinction, California Method "V", consists of side-by-side measurements of light scattering using a nephelometer and light absorption on a filter (modified from the "Coefficient of Haze" protocol), and supported by measurements of relative humidity (RH) (ARB, 1989). This provides direct observation of aerosol optical properties at the location of the monitoring site.

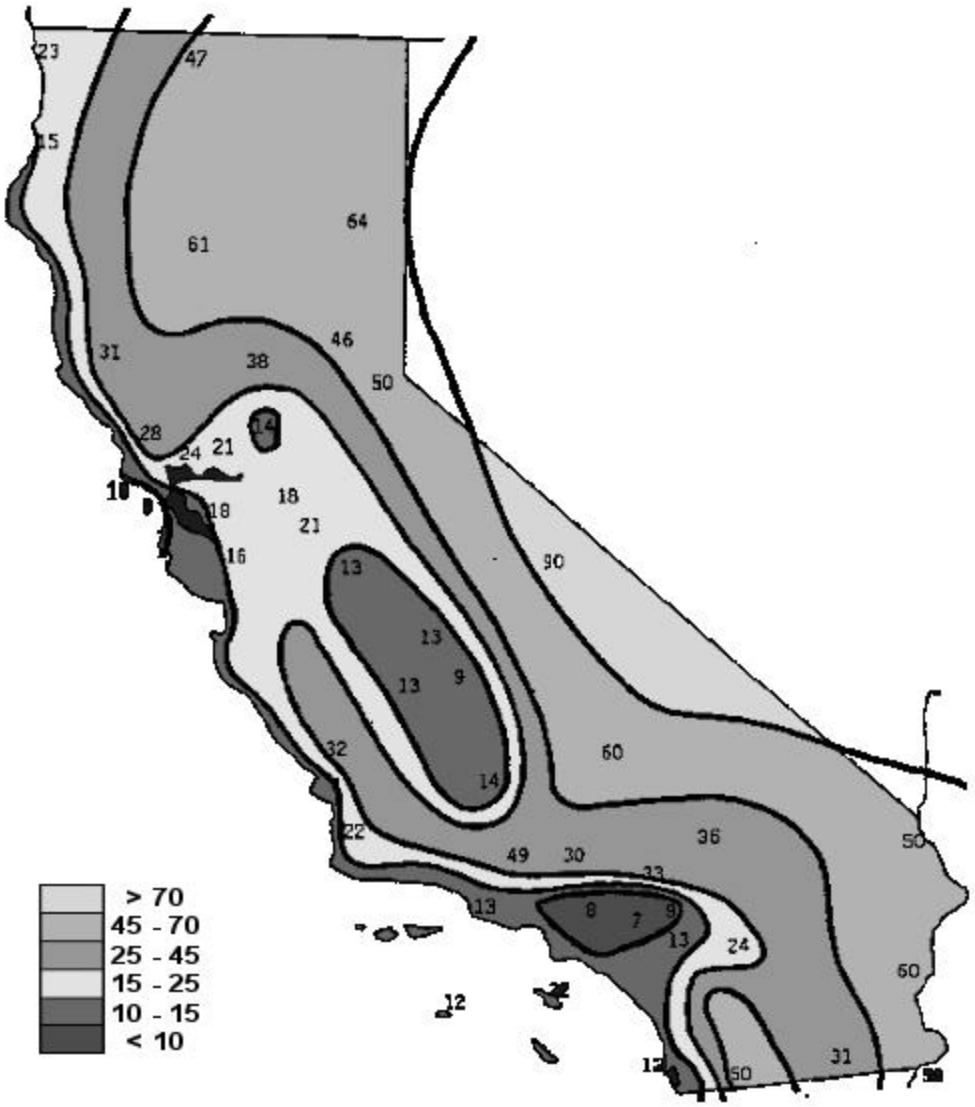


Figure 8.2 Average Annual Visual Range in California (Trijonis, 1980)

Determining the causes of the observed extinction depends on additional aerosol monitoring to identify the particular aerosol components present when visibility is poor, and then linking them to emission sources. Optical data (COH & nephelometer) consistent with Method V are available from 15 sites in the state. To date, implementation of full Method V visibility monitoring (i.e., including RH) has been restricted to a few sites in the South Coast Air Basin and the Lake County Air Basin.

The United States national visibility monitoring is done by the Interagency Monitoring of Protected Visual Environments (IMPROVE) program (Sisler, et al., 1993). The primary IMPROVE protocol consists of size-selective aerosol collection (total PM10 mass and PM2.5 mass and elemental analysis) supported by a long-path transmissometer to measure total

extinction over a fixed sight path near the monitoring site. Measurements of light absorption are taken from the PM2.5 particle filters; subtracting absorption from total extinction gives a measure of scattering. Some IMPROVE sites also employ nephelometers. Because the IMPROVE program combines optical and aerosol monitoring, the particular pollutants causing low visibility at an IMPROVE site can be assessed directly by analyzing the monitoring data. The IMPROVE network in California consists of six sites with records beginning in 1989, and 8 additional sites added in 2000.

8.3 Effects: Aesthetic, Economic, and Operational

Low visibility can result from natural causes (e.g., fog, volcanic eruption, forest fire smoke), from purely anthropogenic causes (e.g., industrial smoke, diesel exhaust), from mixtures of natural and anthropogenic aerosols (e.g., “agricultural haze” consisting of dust and combustion products), or from interactions of natural processes and anthropogenic activities (e.g., nitrate haze, smoke from prescribed fires). The fact of low visibility is not, of itself, cause for regulatory concern; rather, it is the combination of human cause and adverse effect on human activity that drives visibility regulation. While instrumental measurement and source identification can quantify the anthropogenic factors in the timing and intensity of poor visual air quality, identifying undesirable effects and determining appropriate levels of controls is wholly in the realm of policymaking.

There are three broad categories of effects due to reduced visibility: aesthetic, economic, and operational. Aesthetic effects, such as impairment of vistas in national parks, animate the present National visibility program. Economic effects, such as reduced tourism or depressed real estate values, are largely a secondary impact of aesthetic effects. Finally, operational impacts arise when low visibility interferes with “business as usual” such as airport operations, or causes short-term calamity (e.g., chain reaction accidents on freeways). Establishing visual air quality goals for each type of impairment involves balancing the effort and cost of control against the “value” (social and financial) of expected reductions in the frequency and intensity of visual impairment.

8.3.1 Aesthetic Effects

Aesthetic effects dominate the visibility regulatory landscape. The Federal visibility protection program derives from a tradition of National Park and Wilderness conservation based on eliminating all traces of human activity and preserving “pristine nature” in undisturbed enclaves – defined in the CAA as “Class I” areas. The FCAA defines the “national goal” as ‘‘prevention of any future, and the remedying of any existing, impairment of visibility in Class I areas which impairment results from manmade air pollution’’ (42 USC Sect. 169A).

Figure 8.3 Class I Areas.

By contrast, California's State standard for Visibility Reducing Particles follows a pattern derived from health-based air quality regulation. California applies a single minimum visibility value (maximum extinction level) uniformly across an entire air basin (presently, the statewide standard applies in all air basins except Lake Tahoe, where the standard is much more stringent). The logic of using a single-value standard is that regulating emissions that cause low visibility events will necessarily limit the frequency and severity of all levels of impairment, thus regulating the human experience of intense haze will also reduce the experience of intermediate levels of haze (ARB, 1989). The level of the standard represents a policy judgement that identifies regionally appropriate visibility goals (hence a more stringent standard for the Lake Tahoe air basin than for other areas).

8.3.2 Economic Effects

The economic effects of reduced visibility appear in the form of reduced prices for real estate (especially "view" properties), reduced demand for visibility-related recreation, and diffuse effects of perceived degraded environmental quality. (Delucchi, et al., 1996; Trijonis et al., 1985, Rowe, and Chestnut, 1982). Measuring the economic value of visibility (or the cost of impairment) involves translating human preference into monetary value – known as "willingness to pay" (WTP).

There are two general approaches to measure WTP. Surveys asking respondents to set a value on a change in environmental quality are termed "stated preference" methods. Studies using

statistical analysis of differential prices in real markets to infer the actual value of environmental amenities are termed “revealed preference” methods.

Loehman et al. (1994) measured the visibility and health risk WTP in the San Francisco Bay Area using stated preference data from a 1980 survey. Their methodology established three air quality classes (good, fair, poor; equivalent to V_r>10 mi., 10>V_r>6 mi., and V_r<6 mi.) and assigned respondents by residence to 5 sequentially ranked areas based on frequency and severity of pollution based on analysis of daily airport visibility data from around the region (we estimate equivalent PM10 cutpoint ranges as: V_r = 10 mi., 45-90 $\mu g/m^3$; V_r = 6 mi., 75-150 $\mu g/m^3$ depending on particle chemistry and size distributions). Respondents were asked to state how much they would pay per month to move up or to avoid moving down in air quality along the zonal scale. They found that overall individual WTP for visibility was about $0.10 per month (1980$) for each additional day per year of good air quality. It is interesting to note that this study also detected a “risk aversion” response. While visibility valuations were nearly symmetrical for improvement or avoidance, avoiding deterioration generally scored higher than improving air quality for health. Health based WTP to move up was relatively flat across all potential one-step changes, but WTP to avoid moving down increased with deteriorating air quality.

Trijonis et al. (1985) used the revealed preference method applied through multiple regression to analyze the value of visibility for residential real estate in California. Although somewhat dated, this study provides considerable insight into the effect of model formulation and variable specification on detecting WTP. Using a hybrid regression/principle component approach they eliminated the effects of spatial covariance between community characteristics and visibility, then tested various model forms for their explanatory power. Reporting the range of benefits calculated by the three best models for each area, they found, for a ten percent improvement in visibility, average home selling price in southern California would increase by 0.7 to 2.1 percent, while in the San Francisco Bay area, sales price would rise by 1.4 to 2.5 percent. Integrating over regional sales reported for 1978-79 produced economic benefits in the real estate sector of $250M to $617M (1979$) per year in southern California; and $190M to $220M (1979$) per year for the San Francisco Bay area. The breadth of analyses and use of multiple functional forms gives these results strong credibility and it is likely that they span the range of potential “true” values for visibility. There are no studies that address the current (2001) real estate market in California, but California’s spatial patterns of both real estate values and visibility reduction are still much like they were in 1980, so it is reasonable to assume that similar percentage value increments apply to today’s vastly more valuable real estate stock.

In the socioeconomic assessment of the Southern California Air Quality Management District’s (SCAQMD) 1997 Air Quality Plan (Lieu, 1996), SCAQMD staff constructed estimates of the economic value of improved visibility derived from both the revealed and stated preference methods. They reported aggregate annual benefits of $109 million in 2000 and nearly $1.1 billion in 2010; resulting in average annual benefits over the period 1997-2010 of $473M.

8.3.2.1 Controlling Both PM10 and Visibility Reducing Particles

The economic studies and the SCAQMD valuation discussed here were based on either modest incremental changes in air quality or assessing the ancillary benefits accompanying attainment of the annual 24-hour maximum health-based Federal PM10 standard (150 $\mu g/m^3$). The SCAQMD study assumes that all gains are achieved when the PM10 standard is attained. Although unreported in the literature, it is reasonable to expect that there would be additional benefits gained in attaining the State PM10 standard (at the time of the SCAQMD study the California standard was roughly 1/3 the level of the Federal standard) or the State Visibility Reducing Particles standard. While the reported data demonstrate that improving visibility has

substantial economic benefits, it is difficult to interpret these findings in relation to other target extinction levels or to extrapolate these findings to other areas of California. A full evaluation of the statewide benefits of attaining alternative PM or visibility standards has yet to be done.

8.3.3 Operational Effects

Operational impacts of low visibility vary depending on the sensitivity of individual activities to visibility impairment.

8.3.3.1 Roadways

Motor vehicle traffic has a low-sensitivity to PM-caused visibility impairment. Highway traffic requires "good visibility" for safe vehicle flow, yet traffic is not very sensitive to particulate air pollution. Highway visibility is "good" when drivers can clearly see vehicles, objects, or intersections far enough ahead to react to traffic conditions and maintain safe distance from other vehicles. This generally requires sight distances in the range of tens to hundreds of meters (AASHTO, 2001). In dry weather, very high particle concentrations are required to create light extinction levels sufficient to impair vehicle traffic (e.g., a V_r of 500 m implies fine particle concentrations in the range from 1300 to 2500 $\mu g/m^3$). Such high particle concentrations are generally due to short term local sources such as excavation dust, fires, or "dust devils" – events typically not detected by routine monitoring and thus must be regulated by nuisance rules, rather than through air quality standards.

8.3.3.2 Airports

Airport operations, like road traffic, require "good" visibility, but the higher speeds and greater distances involved translate into greater sensitivity to particulate extinction. Ground operation minima are very short – comparable to those for highways [FAA requires airports to begin "low visibility operations" when visual range is less than 1200 ft. (0.74 km) (FAA, 1996)]. Safe flight operations require that pilots have the ability to see an airfield well enough to land, to avoid land-based obstacles or other aircraft, and to generally operate safely under Visual Flight Rules (VFR); for this the FAA has established minimum visibility (V_r) for unrestricted operations at 3 miles (5.1 km) (FAA, 1996). This translates to PM10 concentrations ranging from 130 to 250 $\mu g/m^3$, depending on aerosol conditions.

8.3.3.3 Aircraft Flight Testing

California is home to the two most heavily used flight test facilities in the United States. Air space over the eastern Sierra and the western Mojave desert is reserved for the joint use of Air Force, NASA, and Army testing operations based at Edwards Air Force Base in Antelope Valley and Navy test operations based at China Lake in Indian Wells Valley. These facilities were sited in this region because of their year-round flying weather, excellent visibility, and proximity to California's aerospace industry. Activities at these facilities directly employ over 10,000 people and are the mainstay of the western Mojave regional economy. Unlike typical aviation, these facilities are extremely sensitive to reduced visibility because they employ optical tracking and recording of flight tests using powerful ground-based telescopic movie and video systems. Tracking each test from multiple sites, engineers are able to reconstruct flight dynamics of test or target aircraft, guided missiles, parachutes, or other test objects independent of onboard instrumentation (in some tests, onboard instrumentation is impossible, and the optical tracking is the sole flight record). To accomplish these tests, cameras must be able to track small objects in the sky from distances up to 20 miles (32 km) (VanCuren, 1982). In order to evaluate the threat to these operations due to air pollution, the Department of Defense (DoD) conducted the Research on Operations Limiting Visual Extinction (RESOLVE) project, an intensive visibility assessment in the region in the late 1980s (Trijonis, et al., 1988). While the DoD has not established absolute minimum visibility requirements for its operations, the RESOLVE study

identified anthropogenic pollutants as episodically contributing to reduced operational capability, and DoD adopted a policy of working with local, State and Federal air quality regulators to prevent further degradation in the study area. Conditions deemed adverse in the RESOLVE context are associated with V_r below about 80 km (48 mi), or PM2.5 on the order of 10 $\mu g/m^3$ or greater.

8.3.4 Visibility Regulation

8.3.4.1 Federal Regional Haze Program

The FCAA defines a "national goal" of the ''prevention of any future, and the remedying of any existing, impairment of visibility in Class I areas which impairment results from manmade air pollution'' (42 USC Sect. 169A). The program has two parts, one addressing the impacts of individual large air pollution sources ("Reasonably Attributable Impairment"- RAI) and the other addressing the cumulative effects of all sources ("Regional Haze").

The RAI program [40 CFR section 51.301(s)] is based on studying the direct aerosol impacts (termed "plume blight") of large pollution sources or small groups of sources such as smelters or power plants, and requiring controls on new sources or retrofits on existing sources to reduce their impacts below the threshold of perceptibility. The best-known example of RAI is the case of the Navajo Generating Station at Page, AZ, which was ordered to install additional emission controls after it was found to impact Grand Canyon National Park. No such RAI pairing of a large source and a Class I area has been identified in California.

The Regional Haze program (EPA, 1999) is intended to address the cumulative, diffuse effects of all air pollution sources in a region. Regional Haze involves virtually all sources distributed over a large area (a state or multiple states) and effects on one or many Class I areas. The Regional Haze program does not establish a single visual air quality goal; rather it requires that each State must determine, on a case-by-case basis, "natural conditions" at each Class I area within its boundaries. "Natural" conditions must be represented as a range of visual air quality, and the national goal is interpreted as requiring that emissions be controlled to bring ambient conditions for the best 20% of days to approximate the best 20% of "natural" conditions, and that the worst 20% of days be indistinguishable from the worst 20% of "natural" conditions. The 156 Class I areas in the United States are mapped in Figure 8.3.

California's responsibilities under the Regional Haze rules cover 29 in-State Class I areas, and an as yet undefined number of Class I sites in neighboring states.

Current visibility conditions at Class I areas in California range from near-pristine conditions at Redwood and Lassen Volcanic National Parks to substantially degraded at Sequoia National Park and San Gorgonio Wilderness. Although specific goals have not yet been set for California Class I areas, the likely range of such goals can be inferred from data for the cleaner IMPROVE sites. PM10 at Redwood National Park (a "clean" low altitude coastal site) has a long-term mean around 12 $\mu g/m^3$ and rarely exceeds 30 $\mu g/m^3$. At Lassen Volcanic National Park (a "clean" montane site) long-term mean PM10 is below 10 $\mu g/m^3$ and rarely exceeds 20 $\mu g/m^3$.

8.3.4.2 California AAQS for Visibility Reducing Particles

The California State Ambient Air Quality Standard for Visibility Reducing Particles (VRP) represents a policy judgement that a certain minimum degree of visibility is conducive to public welfare, regardless of location. This policy is manifested as a Statewide minimum dry air particle extinction limit of 0.23/km (230 Mm^{-1}) averaged from 9 AM to 5 PM (PST) when Relative Humidity (RH) is less than 70 percent. This is roughly equivalent to V_r = 10 miles. The standard is 0.07/km (70 Mm^{-1}) for the Lake Tahoe Air Basin (roughly equivalent to V_r = 30 miles). Equivalent PM10 concentrations when this standard is just met range from about 50 $\mu g/m^3$ for a

fine particle dominated urban setting (e.g., Sacramento in winter) to 90 or more $\mu g/m^3$ for a mixture of coarse and fine particles (e.g., Central Valley summer). The Lake Tahoe VRP limit equates to PM10 concentrations ranging from about 16 to 25 $\mu g/m^3$ over a similar range of aerosol characteristics.

State law permits the Board to adopt other standards for any Air Basin, although to date only Lake Tahoe has been singled out for additional protection.

8.4 Climate

Anthropogenic effects on climate have become very important international scientific and political issues. Understanding the scale of these effects, their causes, and anticipated harm, and identifying potential corrective actions are the subjects of major research programs. Beginning in the late 1980's, the World Meteorological Organization (WMO) and United Nations Environment Program (UNEP) have jointly sponsored the Intergovernmental Panel on Climate Change (IPCC), which has become the major international clearinghouse for assessing climate change (IPCC, 2001b) (the brief discussion presented here is largely based on the IPCC 2001 reports.) The initial focus of concern, both scientifically and for managing climate, was on so-called Green House Gases (GHGs) – CO_2, CH_4, etc. - but research over the last two decades has demonstrated that particles, too, have the potential to significantly alter climate processes.

Particles impact climate directly by modifying Earth's radiation balance through their interaction with both long wave (infrared) and short wave (visible) light, and indirectly by their role as condensation nuclei in cloud formation. This effect is termed "radiative forcing." Depending on chemistry, timing, and location, particles may either heat or cool the atmosphere.

Positive radiative forcing warms Earth's surface and lower atmosphere. Negative radiative forcing cools them. Natural factors, such as changes in solar output, explosive volcanic activity, snow, or cloud cover can also have radiative forcing effects. The planetary radiation balance is the net sum of all positive and negative forcing occurring together. Thus an effect such as climate warming by positive infrared forcing due to increasing CO_2 concentrations may be offset by negative forcing due to visible light scattering by "white" aerosols (e.g., sulfates) or enhanced by warming due to infrared and visible light absorption by "black" aerosols ("soot").

Determining the impact of anthropogenic PM emissions on climate requires properly accounting for all radiative forcing, natural and manmade, then determining the shift in net radiation that would occur if the anthropogenic component were removed, and finally calculating the change in climate that would result from that shift in radiation. While this is simple in concept, it is very difficult to implement because:

- We do not have a good inventory of all the aerosols in Earth's atmosphere.
- We do not know with certainty how much aerosol in Earth's atmosphere is due to anthropogenic activity.
- We do not know global PM emission and ambient aerosol distribution patterns with sufficient temporal and spatial resolution.
- We do not know how to partition secondary aerosol effects, such as cloud formation, between natural and anthropogenic condensation nuclei.
- We do not know how what co-effects would accrue to global-scale PM emission controls (CO_2 reduction, altered surface albedo, etc.).
- We do not have climate models with sufficient precision to reliably perform the climate effect calculation.

Figure 8.4 shows the relative positive or negative radiative forcing from various components of the climate system, with an assessment of the degree of certainty of climate knowledge in each area noted along the bottom of the figure. The major aerosol classes are briefly reviewed below.

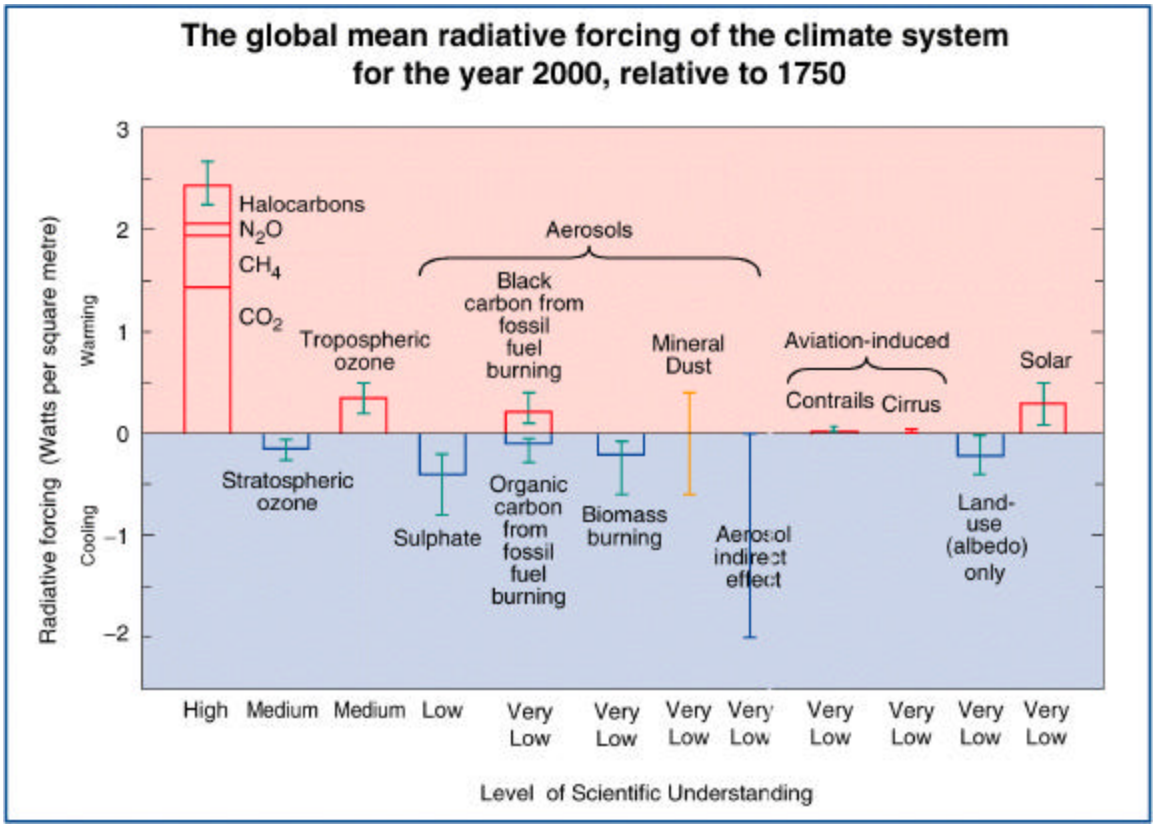


Figure 8.4 Summary of intensity and degree of scientific certainty of climate forcing by anthropogenic pollutants; note that aerosols' effects are both significant and highly uncertain (IPCC, 2001a).

8.4.1 Sulfate

The vast majority of sulfate aerosols are formed by the oxidation of gaseous sulfur compounds into sulfuric acid, which then combines with a metallic or alkaline ion to form a stable salt (Na_2SO_4, Mg_2SO_4, $(NH_4)_2SO_4$, etc.). Sulfate aerosols mostly form in heterogeneous (gas, droplet, and particle) atmospheric conversion, which tends to concentrate sulfate in fine aerosols (<2 μm diameter). When both humidity and sulfuric acid concentrations are high and sufficient neutralizing ions are not present, a liquid phase sulfuric acid aerosol can form.

Due to the hygroscopic nature of both sulfuric acid and sulfate salts, sulfate aerosols are prone to grow by accumulation of water, so that their effective optical cross section is enhanced far beyond the actual sulfate mass. Since sulfate aerosols are very efficient at scattering light, their impact on Earth's radiation balance is predominantly negative forcing due to backscatter of incoming solar radiation; this effect may be enhanced if their hygroscopicity contributes to increased daytime clouds or fog, or may be somewhat offset if they increase the presence of nighttime clouds or fog. The importance of pollutant sulfate in climate was only fully appreciated in the last decade; inclusion of sulfate cooling helped to significantly reduce the gap between

climate change predicted based on GHG calculations and observed secular temperature records (Charleson, et al., 1992). Future reductions in global pollutant sulfur emissions (necessary to manage impacts on public health and prevent "acid rain") may accelerate climate warming as the artificial cooling effect of sulfate is removed (IPCC, 2001).

The precursor sulfur compounds come from both natural and anthropogenic sources.

8.4.1.1 Natural Sulfate

Globally, most natural sulfate comes from biogenic production (primarily in the oceans), with volcanic emissions contributing modestly (e.g., hot springs and fumaroles) on a continuing basis, and occasionally very intensely (large eruptions). As a result, natural sulfate concentrations are somewhat higher over the oceans and lower over the continents. This tends to focus sulfate effects, suppressing solar input to the oceans (lowering heating and evaporation) while minimally altering radiation balance over continents. Large volcanic eruptions have been observed to cool the globe for months or years, an effect believed to be largely due to sulfate. Natural sulfate levels in the atmosphere have been estimated from observations and calculation of emissions, and their climatic effect estimated as well (Twomey, 1974; Twomey, 1977; Charleson, 1987).

8.4.1.2 Anthropogenic Sulfate

Anthropogenic sulfate is generated through the same pathways, but the precursor gases generally come from sulfur bound in fuels used in combustion processes (predominantly coal and petroleum). The potential effects of anthropogenic sulfate are strongest near industrialized regions where large amounts of fossil fuels are burned, thus the cooling effect is strongest over eastern North America, Europe, eastern Asia, and the oceanic and continental areas downwind of these regions (Charleson, 1992; IPCC, 2001a).

8.4.2 Nitrate

Nitrate aerosols form analogously to sulfate, and have similar optical properties. They are distinct from sulfate, however, in that nitrate salts are unstable and can return to the vapor phase when humidity drops or the surrounding air's concentration of precursor gases drops. The dynamics of nitrate aerosol formation and disappearance limit the scope of nitrate impacts on global climate processes.

Nitrates may play an important role on a local or regional basis, especially if their effect is amplified by contributing to changing fog frequency or persistence. Nitrates may be important in some regions as a damper on total aerosol reductions from sulfur control: sulfuric acid has a greater affinity for ammonia than does nitric acid, thus, in a region rich in both SOx and NOx, reducing sulfur emissions may not reduce total aerosol concentrations as nitrate replaces sulfate under humid conditions.

8.4.3 Carbon

Carbonaceous aerosols primarily come from incomplete combustion of fuels, consisting of pure unburned "elemental" carbon ("soot"), partially oxidized organic compounds, and some associated inorganic material ("ash"). In addition, some organic aerosols are produced by gas-phase oxidation of organic vapors – referred to as "secondary" organic aerosol. Carbonaceous aerosols can exhibit highly varied optical effects depending on particle size and chemistry. Major global sources of carbonaceous aerosols are biomass burning (wild fires, vegetation clearing, agriculture, and wood and charcoal used as domestic fuels), industrial and utility boilers, and motor vehicles. Global data on total carbonaceous aerosol emissions are highly uncertain, due primarily to the difficulty of accounting for biomass burning. Moreover, even when current biomass emissions are known, the task will remain to isolate the role of humans in both

the amount of burning we initiate and the changes in global biomass fuel patterns wrought by human alteration of the landscape.

Since carbonaceous aerosol emissions are closely linked with CO_2 emissions, properly calculating the aerosol effects alone may be misleading, since any effort to modify these emissions will undoubtedly be linked with significant changes in CO_2 emissions as well. Overall, the effect of carbonaceous aerosol is thought to be positive forcing, but the size of the effect and its regional distribution are highly uncertain.

8.4.3.1 Elemental Carbon

Elemental carbon (EC) aerosols strongly absorb light at all wavelengths, as well as scattering light in wavelengths near the size of the particles. EC's broad-spectrum light absorption gives it a strong potential for positive radiative forcing since it directly absorbs incoming sunlight, turning it into heat in the air containing the aerosol.

EC is produced in almost all combustion processes. The EC fraction of carbonaceous emissions is small in well-controlled fossil fuel combustion, with the notable exceptions of uncontrolled diesel engines, older jet engines, and open burning of oil-based fuels (e.g., burning contaminated waste fuel).

Biomass EC is highly uncertain, in part due to the lack of data on burning activity, and to the fact that the EC fraction is variable depending on fuel moisture and plant species. However, measurements have shown EC to be only about ten percent of biomass aerosol, suggesting that its effects would be overwhelmed by those of the OC and ash content.

8.4.3.2 Organic Carbon

Organic carbon (OC) aerosols generally exhibit a strong wavelength bias in absorption, weak in visible wavelengths and peaking in the ultraviolet. Since the peak of solar energy input is in the visible wavelengths, scattering of visible light has a greater effect on energy balance than UV absorption, thus OC aerosols' climate effects are believed to be weak negative forcing. OC aerosols are often part of a complex mixture ("smoke"), including OC, ash, and water. Because the inorganic fraction of smoke aerosols are generally weak absorbers at all wavelengths, and the entire mass is capable of scattering light, "smoke" aerosols are considered to show weak negative radiative forcing.

8.4.4 Mineral Dust

"Mineral dust" is generally derived from soil surfaces, either as a result of natural or anthropogenic causes. Since only particles with relatively long atmospheric lifetimes contribute significantly to global aerosol loading, mineral dust at the global scale is quite different from the dust air pollution regulators commonly encounter close to a source. Near-source mineral dust is composed of a variety of crystalline materials, including sand, fine rock fragments ("silt"), and clay particles. Sand and silt materials such as silica have high specific densities and generally fracture into compact shapes, thus coarse mineral particles (>5μm diameter) settle rapidly and have very short atmospheric lifetimes. Conversely, clays, having sheet crystal structures and much smaller particle dimensions, have very large surface to mass ratios and very small settling velocities. Global "background" mineral aerosol is thus finer (mass median diameter near 2μm) and often chemically distinct from most local-source mineral PM.

The optical properties of global mineral aerosols are not well known, nor are their global distributions. Mineral dust may cause either positive or negative radiative forcing, depending on chemistry (fraction of light absorbing minerals) and size (fines scatter more efficiently) (Tegen & Lacis, 1996; Alpert, et al., 1998). Seasonality of dust emission may also play a role in

determining net climate effect by altering the albedo of snow and ice or by positive or negative feedbacks with seasonal temperature cycles.

Mineral dust emissions are moderated by soil condition, plant cover, wind speed, soil wetness, and other factors. Human disturbance of soil can greatly increase dust emissions, both directly (tillage) and indirectly (overgrazing, ground water withdrawal, etc.) (Tegen et al., 1996). The fraction of global dust that is due to current human activity is highly uncertain. As with the biomass problem, determining a "natural" (no human effects) baseline will require unraveling the history of human land use and vegetation change as well as compiling emission inventories.

8.5 Vegetation and Materials Damage

The chemical diversity of particulate matter in the air gives is the potential to have a wide range of interactions with surfaces or water bodies on which it deposits. The most significant of these depositional effects involve the acid ions (primarily sulfuric and nitric) within the aerosol. Acid deposition occurs when aerosols or precursor gases deposit on leaves, soil, water, buildings, or other surfaces. Other components of PM also have deleterious effects, primarily in the form of soiling, and, in the cases of certain localities or particularly sensitive "receptors," damaging effects ranging from crop damage to deterioration of water quality.

8.5.1 Acid Deposition Programs

Nitrogen-containing gases and particles are the greatest source of airborne acidity in California. This is in sharp contrast to the eastern United States (U.S.), where precipitation chemistry is dominated by sulfur-containing acids. Nitrogen-containing acids are responsible for a major portion of acidity in precipitation, fogs and clouds, dry deposited gases, and particles within the state. Although annual precipitation acidity is ten-fold lower in California than in the eastern U.S., summertime concentrations and deposition of nitric acid vapor and particle nitrate are among the highest in the nation. While acute, short-term effects on human health and welfare (*i.e*., agricultural crops and man-made materials) were determined to be minor, long-term effects on human health, as well as aquatic and forest ecosystems, remain poorly known.

In 1980, the National Acid Precipitation Assessment Program (NAPAP) was established to investigate the causes and effects of acidic deposition in the U.S. While the cause of acidic precipitation is largely due to the dissolution of sulfur and nitrogen oxides in rain, the impacts of sulfur-derived acids were of principal concern in the eastern U.S., and the effects of nitrogen-derived acids were of primary interest in the western U.S. In consideration of the nitrogen-dominated rain chemistry of California, and the potential for distinct health and welfare effects from the eastern U.S., two five-year programs of monitoring and research were enacted by the California Legislature: the Kapiloff Acid Deposition Program (KADP) and the Atmospheric Acidity Protection Program (AAPP). Concentrations of acidic air pollutants in precipitation, fog, and dry-deposited particles and gases were measured in support of the KADP and AAPP by the Air Resources Board's (ARB) California Acid Deposition Monitoring Program (CADMP). Analyte levels in rain/snow and dry deposition have been reported in data summaries (Takemoto et al., 1996), final reports (Watson et al., 1991; Blanchard and Michaels, 1994), and the open literature (Blanchard and Tonnessen, 1993; Melack and Sickman, 1997). The major findings from the KADP and AAPP have also been documented in final reports, Annual Reports to the Governor and the Legislature (ARB, 1983-1986; 1988; 1991-1994a), a technical assessment (ARB, 1989), and the open literature (*e.g*., Takemoto et al., 1995).

8.5.2 Deposition

8.5.2.1 Acidity

Across the state the deposition of N-derived acidic gases and particles provides most of the atmospheric acidity and N to urban landscapes, and to mid-elevation forests in southern California. Blanchard et al. (1996) used precipitation chemistry data from the CADMP, the National Atmospheric Deposition Program/National Trends Network (NADP/NTN), and an alpine precipitation sampling network in the Sierra Nevada Mountains to estimate regional-scale rates of wet-deposited nitrate, sulfate, ammonium, calcium, and H^+ from 1985 through 1994 (Figure 8.5). Rates of wet sulfate, nitrate, and ammonium deposition were found to be <4, <3, and <4 kg S or N/ha/yr in at all sites, respectively (Blanchard et al., 1996). In comparison, rates of wet sulfate and nitrate deposition in eastern North America exceed 8.3 and 3.4 kg S or N/ha/yr, respectively, and deposition rates of ammonium are <3.1 kg N/ha/yr (Sisterson, 1991). In most years, wet nitrate deposition was estimated to be greater in urban areas of the South Coast Air Basin (SoCAB) and the southern Sierra Nevada, than in other parts of California. Along the northwest coast where wet sulfate deposition is highest, much of the sulfate is derived from sea salt. Uncertainties in the wet deposition estimates are ≤20 percent in the SoCAB, which has a large number of monitors, but are two to three fold higher in other parts of the state.

Comparisons of estimated NO_x emission and total N deposition rates (wet and dry) show that the deposition of oxidized N in the SoCAB accounts for 16-37 percent of the NO_x emitted in the Basin (Figure 8.6; Blanchard et al., 1996). The total N deposition at Fremont was about 11 percent of the NO_x emission rate in San Francisco Bay Area. Total N deposition rates at Bakersfield and Sacramento are about 76 and 32 percent of the NO_x emission rates in Kern and Sacramento County, respectively. Transport of NO_x from upwind areas could account in part for the relatively large deposition-to-emissions ratio at Bakersfield (Tracer Technologies, 1992).

8.5.2.2 Particulate Matter Concentrations

The CADMP dry deposition monitoring program was established in 1988 to determine spatial and temporal patterns of acidic pollutant concentrations in the state. Daytime and nighttime dry particle and gas concentrations were measured once every six days (Watson et al., 1991). Initially, the network consisted of ten sites located in Azusa, Bakersfield, Fremont, Gasquet, Long Beach, Los Angeles, Sacramento, Santa Barbara, Sequoia National Park, and Yosemite National Park. Over the years, data analyses indicated that acidic pollutants were a moderate-to-minor problem in California, and the number of monitoring sites was reduced, as well as the frequency and range of pollutants sampled. In September 1995, the CADMP dry deposition network was reduced to five sites (Azusa, Bakersfield, Long Beach, Los Angeles, and Sacramento) in urban areas. Also, instead of collecting daytime and nighttime samples of PM10 and PM2.5, only one 24-hour-average sample of PM2.5 was collected.

From 1989-1994, annual-average PM10 and PM2.5 concentrations declined at all ten sites. Representative data from five sites are shown in Figures 8.5 and 8.6. Most areas with high PM10 levels also have high PM2.5 concentrations. At rural sites (Gasquet, Yosemite, and Sequoia National Parks), annual average concentrations of PM2.5 were 4-6 $\mu g/m^3$. Near to Redwood National Park, Gasquet is far removed from most anthropogenic emissions sources, and provides an estimate of background ambient PM concentrations in California. On the western slope of the Sierra Nevada, Sequoia and Yosemite National Parks receive pollutants transported from the San Joaquin Valley by upslope flows. Compared to these rural sites, annual-average concentrations of PM2.5 are two to five times greater at urban locations.

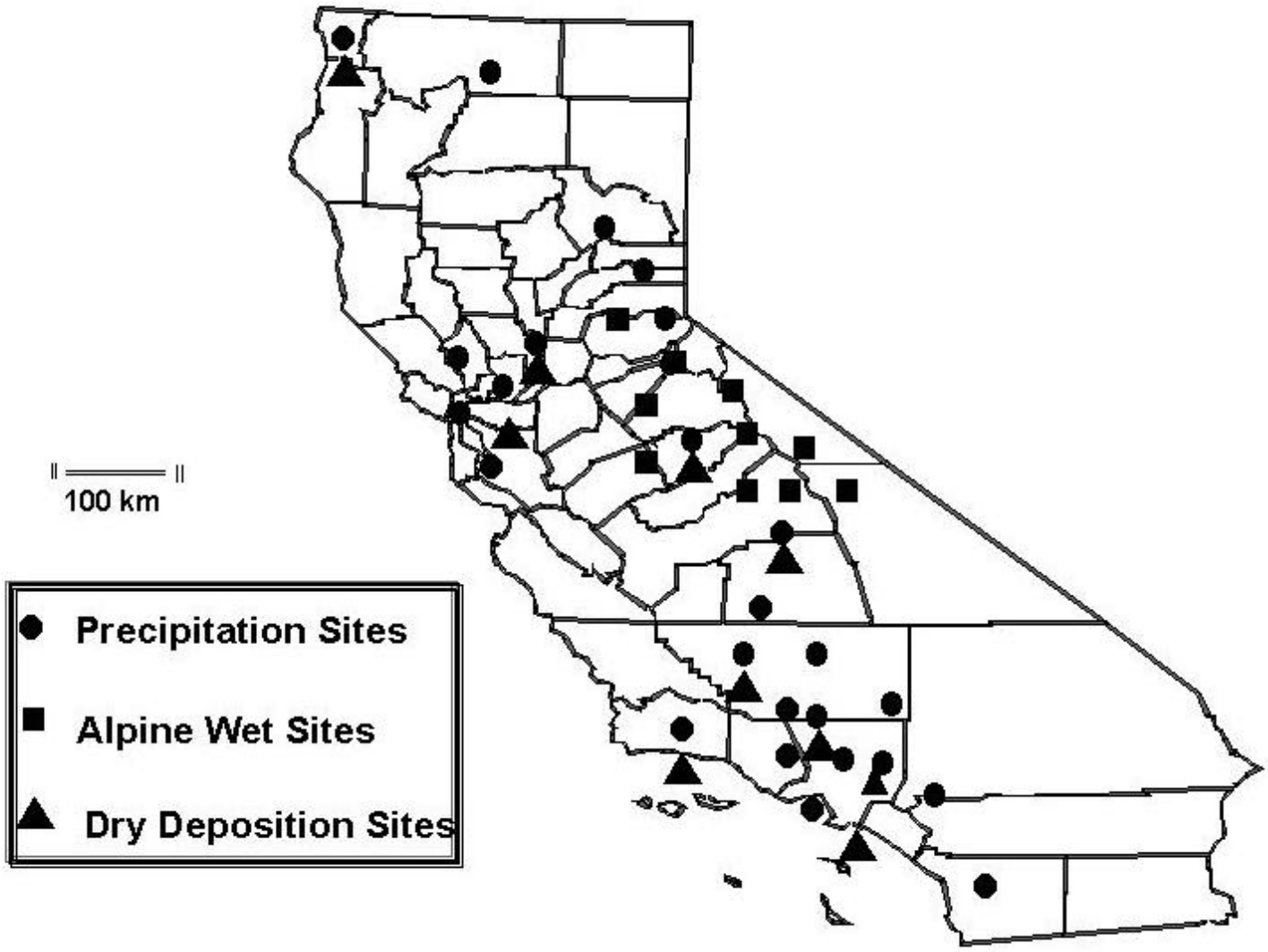


Figure 8.5 Location of CADMP, NADP/NTN, and Sierra Nevada Alpine Wet Deposition Monitoring Sites (Air Resources Board, 1983).

8.5.2.3 Acid Fog

Acidic fog has been associated with harmful air pollution episodes (e.g., London, the Meuse Valley in Belgium, and Donora, Pennsylvania), and reported to adversely affect materials, crops, and forests. From 1982 through 1989, ARB sponsored fog water sampling programs at seven sites in California. Fog water collected in the western portion of the SoCAB was found to be highly acidic, with pH values ranging from 1.7 to 4 (e.g., Jacob et al., 1985). Fog water collected at non-urban, coastal sites was less acidic (i.e., pH ranged from 3 to 7) due, in part, to the low alkalinity of marine atmospheres. In the eastern part of the SoCAB and the southern San Joaquin Valley, fogs were generally not as acidic due to high levels of acid-neutralizing ammonia

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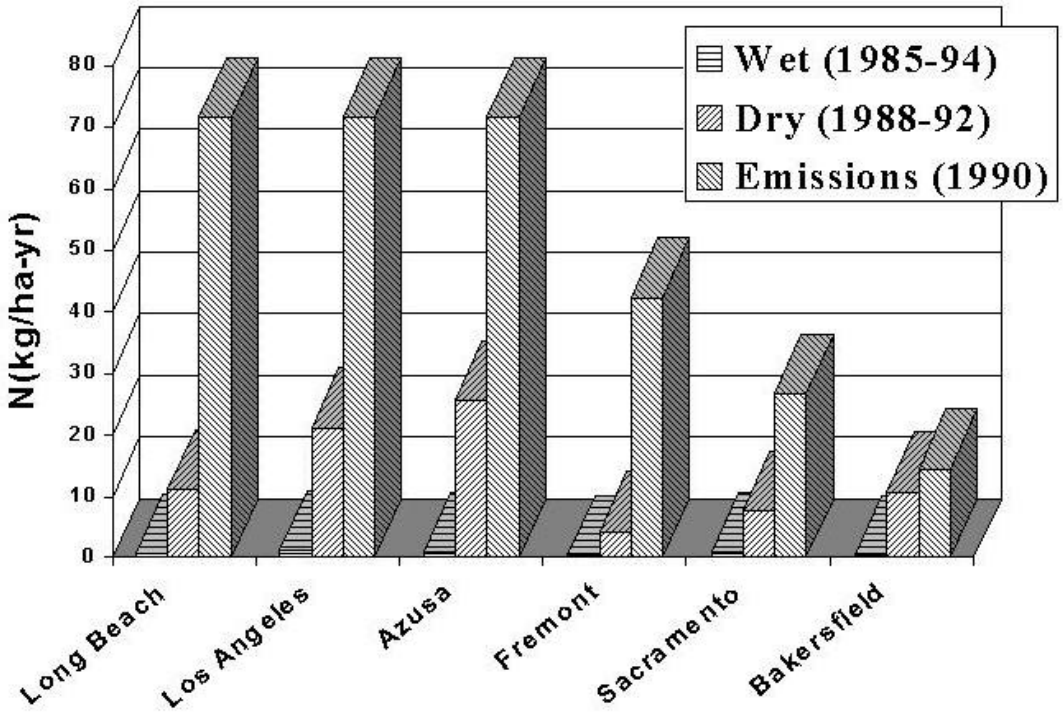

Figure 8.6 Rates of Oxidized N Emissions, and Wet and Dry N Deposition at Urban CADMP Sites (cf. Blanchard et al., 1996).

As in rain, the main contributors to fog acidity are nitric and sulfuric acid. Across the state, the nitrate-to-sulfate ratios in fog are typically about 3:1, but local emissions influence measured concentration ratios. For example, the 3:1 ratio typifies areas where motor vehicle emissions of NO_x dominate (e.g., Los Angeles), but may be close to 1:1 at sites in the southern San Joaquin Valley where sulfur emissions from oil production are significant. Concentrations of ammonium, nitrate, and sulfate ions are commonly 100-times higher in fog than in rain. High concentrations of chemical components in fog correlated well with the occurrence of photochemical smog events, as well as the physical processes of condensation and evaporation.

8.5.3 Effects

In this section the major findings from six research programs sponsored under the KADP and AAPP are summarized. These studies examined the atmospheric processes associated with acid deposition and its effects on human health, aquatic ecosystems, forest ecosystems, agricultural crops, and man-made materials. Statewide networks to monitor pollutant concentrations in wet and dry deposition were established to measure conditions in both urban and rural areas.

8.5.3.1 Aquatic Environments

Changes in surface water chemistry and precipitation chemistry may cause ecosystem-level alterations in the high elevation watersheds of the Sierra Nevada. Chronic acidification of high elevation surface waters in the Sierra Nevada has not been found, but episodic depressions in acid neutralizing capacity do occur. While no large-scale or widespread adverse ecological

impacts have been detected, many high elevation aquatic ecosystems are nitrogen-limited and potentially at risk from current levels of atmospheric nitrogen deposition (Melack and Sickman, 1997).

Currently, surface waters in the Sierra Nevada are not acidic enough to threaten the juvenile or adult stages of Sierra Nevada amphibians or fish. Of the five species of trout found at high elevation in the Sierra Nevada, three species spawn in the spring (rainbow, golden, and cutthroat), and two spawn in the fall (brown and brook). As a result they are differentially at risk from episodic acidification (Jenkins et al., 1994). In spring, the fertilized eggs of spring-spawning trout are at risk from snowmelt water, which is considerably more acidic than pre-melt surface water.

Episodic acidification of streams due to snowmelt or summer rains may decrease populations of some species of stream invertebrates. Vulnerable species identified in work done at Emerald Lake include the nymphs of mayflies and chironomid fly larvae (Hopkins et al., 1989; Kratz et al., 1994). When pH is lowered to 5.0 or below, for as little as eight hours, drift rates of vulnerable species increase, and much of the increased drift is due to mortality (i.e., drifting insects are killed by low pH).

Using the 1985 USEPA Western Lakes Survey, it was estimated that none of the 114 lakes sampled in the Sierra Nevada had been episodically acidified (ANC < 0) (Leydecker et al., 1999). These workers predicted that approximately six and ten percent of Sierra lakes would become episodically acidified if nitrate and sulfate deposition increases by 50 and 150 percent, respectively. No lakes would be chronically acidified in response to the above increases in nitrate and sulfate deposition.

In Lake Tahoe, studies (Jassby, et al, 1994) indicate that phytoplankton growth is not co-limited by the availability of nitrogen and phosphorus; rather, growth is limited by phosphorus alone, due to the deposition of atmospheric nitrogen. Nutrient input to Lake Tahoe, including airborne nitrogen and phosphorus, is not only a concern for ecosystem effects, but is believed to be a major factor in loss of clarity in the lake.

8.5.3.2 Forests

Nitrogen saturation has occurred in forested watersheds in the San Bernardino Mountains, and nitrate contamination of groundwater is of near-term concern. In future years, atmospheric nitrogen deposition could lead to forest soil nitrogen saturation in other areas such as the San Gabriel Mountains and southern Sierra Nevada. Ozone is the primary air pollution stressor of forests, and there is the potential for interactive effects with atmospheric nitrogen.

8.5.3.3 Crops

The acute effects of acidic fog on crops were of concern in the 1980s following reports of adverse S-derived fog and aerosol effects on human health (Graham, 1991). Two studies were funded to evaluate effects on winter and summer crops (Olszyk et al., 1987), and two species of conifer seedlings (Bytnerowicz et al., 1989). As the most extreme fog exposure, a pH 1.7 fog treatment was applied to simulate the pH 1.69 fog measured in Corona Del Mar by Hoffman and co-workers at the California Institute of Technology (Jacob et al., 1985). The responses of five crops were examined, and four crops exhibited yield reductions following 11 weeks of exposure to pH 1.7 fog (Olszyk et al., 1987). The damage to leaves caused by pH 1.7 fog decreased the amount of crop leaf area capable of performing photosynthesis. The observed reductions in crop yield were largely explained by decreases in whole plant photosynthesis. Similar findings were reported for white fir and ponderosa pine seedlings exposed to pH 2.0 fog for six weeks (Bytnerowicz et al., 1989).

8.5.3.4 Soil Chemistry

Concern over the effects of acidic deposition on agricultural soils emerged as a result of findings that suggested that excess inputs of N and S could lead to trace element nutrient deficiencies (*e.g.*, calcium). In a report by Mutters (1995), the nutrient requirements of selected crops were compared against annual inputs from fertilizer and the atmosphere to determine if imbalances could develop. Of the three elements examined (N, S, and calcium), there was a limited possibility that atmospheric N deposition could contribute to a build-up of nutrients that could adversely affect crop productivity. Given the lack of direct acidic deposition impacts on crop growth or yield, no additional research is needed. In terms of ARB's air quality goals, current farm practices appear to provide adequate protection from the harmful effects of acidic deposition.

8.5.3.5 Man-made Materials

Studies conducted in both the KADP and AAPP did not identify any significant damage to materials due to atmospheric acidity. While laboratory analyses indicate that NO_2 and nitric acid may damage painted surfaces, aluminum, and nylon fabric (Mansfeld et al., 1988), field studies in southern California found corrosion rates to be similar to rates in sites with clean air (Mansfeld and Henry, 1993).

8.6 References

Air Resources Board (ARB). 1994a. The Atmospheric Acidity Protection Program: Annual Report to the Governor and the Legislature, 1993. Research Division, Sacramento, CA.

Air Resources Board (ARB). 1993. The Atmospheric Acidity Protection Program: Annual Report to the Governor and the Legislature, 1992. Research Division, Sacramento, CA.

Air Resources Board (ARB). 1992. The Atmospheric Acidity Protection Program: Annual Report to the Governor and the Legislature, 1991. Research Division, Sacramento, CA.

Air Resources Board (ARB). 1991. The Atmospheric Acidity Protection Program: Annual Report to the Governor and the Legislature, 1990. Research Division, Sacramento, CA.

Air Resources Board (ARB). 1989. The Health and Welfare Effects of Acid Deposition in California: Technical Assessment. Research Division, Sacramento, CA.

Air Resources Board (ARB). (1989) "Instrumental Measurement of Visibility Reducing Particles," Technical Support Document, January, 1989.

Air Resources Board (ARB). 1988. Fifth Annual Report to the Governor and the Legislature on the Air Resources Board Acid Deposition Research and Monitoring Program. Research Division, Sacramento, CA.

Air Resources Board (ARB). 1986. The Fourth Annual Report to the Governor and the Legislature on the Air Resources Board Acid Deposition Research and Monitoring Program. Research Division, Sacramento, CA.

Air Resources Board (ARB). 1985. Third Annual Report to the Governor and the Legislature on the Air Resources Board Acid Deposition Research and Monitoring Program. Research Division, Sacramento, CA.

Air Resources Board (ARB). 1984. Second Annual Report to the Governor and the Legislature on the Air Resources Board Acid Deposition Research and Monitoring Program. Research Division, Sacramento, CA.

Air Resources Board (ARB). 1983. Acid Deposition Research and Monitoring Program Report to the Governor and the Legislature. Research Division, Sacramento, CA.

Air Resources Board (ARB). (1982). “Summary and Statement of Reasons for Proposed Rulemaking,” State Ambient Air Quality Standard for PM10, California Air Resources Board, Dec. 1982.

AASHTO (2001), A Policy on Geometric Design of Highways and Streets, 2001, American Association of Highway and Transportation Officials, Washington, D.C.

Alpert, P., et al. (1998), “Quantification of dust-forced heating of the lower troposphere,” Nature 395:367-370.

Blanchard, C.L. and H. Michaels. 1994. Regional Estimates of Acid Deposition Fluxes in California. Final Report, No. A132-149, Air Resources Board, Sacramento, CA. NTIS No. PB94207677.

Blanchard C.L. and K.A. Tonnessen. 1993. Precipitation-chemistry measurements from the California Acid Deposition Monitoring Program, 1985-1990. Atmos Environ 27A: 1755-1763.

Bytnerowicz, A., D.M. Olszyk, B.K. Takemoto, P.M. McCool, and R.C. Musselman. 1989. Effects of Acid Fog and Ozone on Conifers. Final Report, No. A6-114-32, Air Resources Board, Sacramento, California. NTIS No. PB89222715.

Charleson, R., J. Lovelock, M. Andreae, S. Warren (1987), “Oceanic phytoplankton, atmospheric sulfur, cloud albedo and climate,” Nature 326:655-661.

Charleson, R., et al. (1992), “Climate forcing by anthropogenic aerosols,” Science 255:423-430.

Crocker, T.D., and J. Shogren (1991) “Ex Ante Valuation of Atmospheric Visibility,” Applied Economics 23: 143-151.

Delucchi, M. A.. J. Murphy, D. R. McCubbin, J. Kim (1996) The Cost of Reduced Visibility Due to Particulate Air Pollution from Motor Vehicles , Report #13 in series: The Annualized Social Cost of Motor-Vehicle Use in the United States, based on 1990-1991 Data, University of California, Davis, UCD-ITS-RR-96-3 (13)

Engle, D.L. and J.M. Melack. 1997. Assessing the Potential Impact of Acid Deposition on High Altitude Aquatic Ecosystems in California – Integrating Ten Years of Investigation. Draft Final Report, No. 93-312, Air Resources Board, Sacramento, CA.

FAA (1996), AC No: 120-57A, Federal Aviation Administration, 12/19/1996.

Friedlander, S. K. (1977) Smoke Dust and Haze, J. Wiley & Sons, New York.

Graham, J.A. 1991. Direct health effects of air pollutants associated with acidic precursor emissions. NAPAP Report 22. IN: P.M. Irving (Ed).

Graham, J.A. 1991. Direct health effects of air pollutants associated with acidic precursor emissions. NAPAP Report 22. IN: P.M. Irving (Ed). Acidic Deposition: State of Science and Technology, Volume III, Terrestrial, Materials, Health, and Visibility Effects. Office of the Director, Washington, DC.

Harrison Jr., D. and D. L. Rubinfeld, "Hedonic Housing Prices and the Demand for Clean Air," Journal of Environmental Economics and Management 5: 81-102 (1978).

Hopkins, P.S., K.W. Kratz, and S.D. Cooper. 1989. Effects of an experimental acid pulse on invertebrates in a high altitude Sierra Nevada stream. Hydrobiol 171: 45-58.

Intergovernmental Panel on Climate Change (IPCC) (2001a), Climate Change 2001: The Scientific Basis, Contribution of Working Group I to the Third IPCC Assessment Report, J. T. Houghton, Y. Ding, D. J. Griggs, M. Noguer, P. J. van der Linden and D. Xiaosu (Eds.), Cambridge University Press, UK, 944 pp.

Intergovernmental Panel on Climate Change (IPCC) (2001b), Climate Change 2001: Summary for Policymakers (SPM), IPCC, Geneva, Switzerland.

Intergovernmental Panel on Climate Change (IPCC) (2001c), Climate Change 2001: Technical Summary (TS), IPCC, Geneva, Switzerland.

Jacob D.J., J.M. Waldman, J. W. Munger, and M.R. Hoffman. 1985. Chemical composition of fogwater collected along the California Coast. Environ Sci Technol 19: 730-736.

Jassby, A.D., J.E. Reuter, R.P. Axler, C.R. Goldman, and S.H. Hackley. 1994. Atmospheric deposition of nitrogen and phosphorus in the annual nutrient load of Lake Tahoe (California-Nevada). Water Resour Res 30(7): 2207-2216.

Jenkins, T. M. Jr., et al., 1994. Aquatic biota in the Sierra Nevada: current status and potential effects of acid deposition of populations, Final report, Contract A932-138. California Air Resources Board, Sacramento, CA.

Kratz, K.W., S.D. Cooper, and J.M. Melack. 1994. Effects of single and repeated experimental acid pulses on invertebrates in a high altitude Sierra Nevada stream. Freshw Biol 32: 161-183.

Leydecker, A., J.O. Sickman, and J.M. Melack. 1999. Episodic lake acidification in the Sierra Nevada, California. Water Resour Res (In press).

Lieu, S. et al. (1996) Final Socioeconomic Report For 1997 Air Quality Management Plan, South Coast Air Quality Management District, Diamond Bar, Ca.

Loehman, E.T., S. Park, and D. Boldt, "Willingness to Pay for Gains and Losses in Visibility and Health," Land Economics 70(4): 478-498, November (1994).

Malm, William C. (1999) Introduction to Visibility, Cooperative Institute for Research in the Atmosphere (CIRA), Colorado State University, Fort Collins, CO, May, 1999.

Mansfeld F. and R.C. Henry. 1993. Investigation of the Effects of Atmospheric Acidity upon Economically Significant Materials. Final Report, No. A932-113, Air Resources Board, Sacramento, CA. NTIS No. PB94126067.

Mansfeld, F., R. Henry, and R. Vijayakumar. 1988. Investigation of the Effects of Acidic Fog and Dew upon Materials. Final Report, No. A5-138-32, Air Resources Board, Sacramento, CA. NTIS No. PB90157496.

Middleton, W. E. K. (1952) Vision Through the Atmosphere, U. Toronto Press, Toronto, Canada.

Melack, J.M. and J.O. Sickman. 1997. Monitoring of Wet Deposition in Alpine Areas in the Sierra Nevada. Final Report, No. A932-081, Air Resources Board, Sacramento, CA. NTIS No. PB97151484.

Mutters, R.G. 1995. Atmospheric Deposition to Agricultural Soils. Final Report, No. 93-334, Air Resources Board, Sacramento, CA. NTIS No. PB95231379.

Olszyk, D.M., R.C. Musselman, A. Bytnerowicz, and B.K. Takemoto. 1987. Investigation of the Effects of Acid Deposition upon California Crops. Final Report, No. A5-087-32, Air Resources Board, Sacramento, CA. NTIS No. PB88225966.

Poole, E. 1999. Ambient air pollution in the Greater Vancouver area Evaluating air quality improvements with hedonic property value models Department of economics Simon Fraser University, Burnaby, B.C.

Rowe, R.D., and L. G. Chestnut, The Value of Visibility: Theory and Application, Abt Books, Cambridge, Massachusetts (1982).

Rowe, R.D., R. C. D'arge, and D. S. Brookshire, "An Experiment on The Economic Value of Visibility," Journal of Environmental Economics and Management 7(1): 1-19, March (1980).

Schulze, W.D., et al. (1983) "The Economic Benefits of Preserving Visibility in the National Parklands of the Southwest," Natural Resources Journal, 23(1):149-173.

Sisler, J. F., D. Huffman, D. Latimer, W. Malm, M. Pitchford (1993) Spatial and Temporal Patterns and the Chemical Composition of the Haze in the United States: An Analysis of Data from the IMPROVE Network, 1988-1991, Cooperative Institute for Research in the Atmosphere (CIRA), Colorado State University, Fort Collins, CO, February, 1993.

Smith, V.K., and J. Huang (1995) "Can Markets Value Air Quality? A Meta-Analysis of Hedonic Property Value Models", J. Political Economy 103:1, 209-227.

Takemoto, B.K., B. Cahill, C. Buenviaje, R. Abangan, and T.E. Houston. 1996. California Acid Deposition Monitoring Program: Wet Deposition Data Summary – July 1993 to June 1994. Air Resources Board, Sacramento, CA.

Takemoto, B.K., B.E. Croes, S.M. Brown, N. Motallebi, F.D. Westerdahl, H.G. Margolis, B.T. Cahill, M.D. Mueller, and J.R. Holmes. 1995. Acidic deposition in California: Findings from a program of monitoring and effects research. Water Air Soil Pollut 85: 261-272.

Tegen, I, and A. Lacis (1996), "Modelling of particle size distribution and its influence on the radiative properties of mineral dust aerosol," J. Geophys. Res. 101:19237-10244.

Tegen, I, and A. Lacis, I. Fung (1996), "The influence on climate forcing of mineral aerosols from disturbed soils," Nature 380:419-422.

Trijonis, J., M. Thayer, J. Murdoch, and R. Hageman (1985) Air Quality Benefit Analysis for Los Angeles and San Francisco Based on Housing Values and Visibility, Santa Fe Research Corporation, California Air Resources Board, Contract A2-088-32

Trijonis, J., et al. (1988), RESOLVE Project Final Report, Visibility Conditions and Causes of Visibility Degradation in the Mojave Desert of California, Naval Weapons Center, China Lake, CA. NWC TP 6869.

Trijonis, J., (1980) Visibility in California, Technology Service Corp., Santa Monica, CA, ARB Contract A7-181-30.

Twomey, S. (1974), "Pollution and the planetary albedo", Atmos. Environ. 8:1251-1256.

Twomey, S. (1977), Atmospheric Aerosols, Elsevier, Amsterdam, 237 p.

U.S. Environmental Protection Agency (USEPA). 1999. 40 CFR Part 51, Regional Haze Regulations, Federal Register Vol. 64, No. 126, Thursday, July 1, 1999, pp. 35714-35774.

U.S. Environmental Protection Agency (USEPA). 1995. Acid Deposition Standard Feasibility Study: Report to Congress. EPA 430-R-95-001a.

U.S. Environmental Protection Agency (USEPA). 1990. Acidic Deposition: State of Science and Technology, Volume III, Terrestrial, Materials, Health, and Visibility Effects. Office of the Director, Washington, DC.

van de Hulst, H.C. (1957) Light Scattering by Small Particles, Wiley, New York.

Watson, J.G., J.C. Chow, R.T. Egami, J.L. Bowen, C.A. Frazier, A.W. Gertler, D.H. Lowenthal, and K.K. Fung. 1991. Measurements of Dry Deposition Parameters for the California Acid Deposition Monitoring Program. Final Report, No. A6-076-32, Air Resources Board, Sacramento, CA. NTIS No. PB92167030.

9. Quantifying the Adverse Health Effects of Particulate Matter

There have been several recent published efforts to estimate the health benefits associated with reducing population exposures to PM. Ostro and Chestnut (1998) generated estimates of the health benefits associated with U.S. EPA's proposed standards for PM2.5. Kunzli et al. (2000) estimated the health effects attributed to traffic-related PM in three European countries. The U.S. EPA has embarked on several significant efforts to quantitatively evaluate the health risks associated with exposure to ambient PM10 and PM2.5. For example, the Staff Paper for particulate matter (U.S. EPA, 1996) summarized an analysis of health risks associated with attainment of alternative standards for PM2.5 and PM10. Section 812 of the federal Clean Air Act required the U.S. EPA to conduct an analysis of the health benefits of current federal air pollution legislation, which resulted in a report to the U.S. Congress (U.S. EPA, 1999). These efforts have undergone years of public review and comment as well as full peer review by the U.S. EPA's independent Science Advisory Board. We have, therefore, drawn considerably from prior efforts at the federal level, particularly in the development of concentration-response functions. We have also added California-specific concentration-response functions, whenever possible.

The objectives of this chapter are to quantify the health effects of PM in California and to employ the results of this effort to estimate the health benefits that would result from achieving the proposed air quality standards.

9.1 Health Effects Estimation Approach

Estimating the incidence of adverse health effects of PM involves four elements:

- Estimates of the changes in PM exposure levels.
- Estimates of the number of people exposed to PM at a given location.
- C-R functions that link changes in PM concentration with changes in the incidence of adverse health effects.
- Applicability of the C-R functions that are drawn from studies conducted in other parts of the country to California.

Each of these elements is discussed below.

9.1.1 Exposure Estimation and Assumptions

The basic procedure for determining exposures was first adopted by the ARB in 1993 to fulfill the requirements of section 39607(f) of the Health and Safety Code. Full details are provided in Guidance for Using Air Quality-Related Indicators in Reporting Progress in Attaining the State Ambient Air Quality Standards (September 1993). For this application, the concentrations and populations were associated by census tract and merged to assemble a distribution of exposures to different concentrations of PM.

Concentrations of many air pollutants, including PM, change significantly from one location to another. PM concentrations may be well under the standard in one location but above the standard less than 10 kilometers away. Accordingly, population exposures tend to be more accurate when the population data are highly resolved.

Population counts by census tract are used to determine population exposures to air pollutants. In addition, demographic data, such as age distributions, are available for each census tract. A

typical census tract contains several thousand people. Densely populated areas have many census tracts, while sparsely populated regions have few.

We estimated PM10 and PM2.5 concentrations per census tract using air quality data from monitors located at specified distances from the census tract centroid. Air pollutant concentrations from a network of air quality monitors are used to determine appropriate values at census tracts that lie between the monitors.

The concentration for a census tract is the weighted average of the concentrations at all monitors within a maximum allowed distance. For the present analyses of PM10 and PM2.5, the maximum distance was 50 kilometers except for 75 km in the Great Basin Valleys Air Basin. A small number of census tract populations were not included in the analyses because they are more than 50 km from any PM monitor. The population numbers are affected only slightly by different choices for the maximum distance.

The weight assigned to each monitor is the inverse square of its distance from the census tract. In this way, close monitors are more influential than distant ones. Although "boundaries," such as mountain ranges, were not used in the model, local monitors on each side of such boundaries dominate the calculated concentrations for census tracts in their respective regions.

In each air basin, we assumed that the population in a specific concentration bin is exposed to the mid-point concentration in that bin. We then estimated the population-weighted PM2.5 and PM10 annual arithmetic mean concentration in each air basin.

9.1.2 Data Used

Monitoring data for 1998 through 2000 were used from all monitors in the State meeting quality assurance criteria for valid data. Projected census tract data based on 1990 census data were used as the 2000 data were not yet available in the census tract format. The census data contains the shape, size and centroid of each census tract.

9.1.3 Exposure Model Results

Table 9.1 summarizes the results of the statewide assessment.

9.1.4 Exposed Population by Location

Health effects are related to the level of PM that individuals are exposed to. Because the levels of PM exposure vary from air basin to air basin, individuals in different air basins do not experience the same health effects. Estimating health effects by county is complicated somewhat because concentrations were estimated by air basin rather than by county in this analysis. The boundaries for air basins and counties are not always the same due to geographic characteristics. Therefore, county populations were divided to fit air basin boundaries.

We estimated the basin county population, i.e., the county population within an air basin, based on the county population percentage relative to the air basin population derived from California Department of Finance air basin population data and the 2000 census county population.

Table 9.1. Population-Weighted Average Particular Matter Annual Arithmetic Mean Concentration

Air Basin	PM2.5 (μg/m3)	PM10 (μg/m3)
Great Basin Valleys	8.50	16.71
Lake County	2.50	10.83
Lake Tahoe	7.50	20.83
Mountain Counties	16.60	22.96
Mojave Desert	10.00	21.60
North Coast	7.50	17.54
North Central Coast	7.50	24.25
Northeast Plateau	NA	12.97
South Coast	22.20	40.67
South Central Coast	11.80	23.04
San Diego	15.60	28.80
San Francisco Bay Area	15.80	21.67
San Joaquin Valley	22.30	39.48
Salton Sea	13.10	70.17
Sacramento Valley	12.30	24.49
Statewide Averages	18.5	33.11

9.1.5 Concentration-Response Functions

Concentration-response (C-R) functions are equations that relate the change in the number of adverse health effect incidences in a population to a change in pollutant concentration experienced by that population. This section discusses issues that affect health effect estimates and outlines epidemiological studies used for the basis of the C-R functions. Many C-R functions were used in the U.S. EPA Final Heavy-Duty Engine/Diesel Fuel Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit Analysis Results (U.S. EPA, 2000).

9.1.5.1 Basic C-R Function

Different epidemiological studies have been used to estimate the relationship between PM and a particular health endpoint at different locations. They may have different functional forms, PM concentrations, health endpoints, and relate to different populations. Some studies have assumed that the relationship between a health endpoint and PM is best described by a linear form, i.e., the relationship between a health endpoint (Y) and PM is estimated by a linear regression in which Y is the dependent variable and PM is one of several independent variables. Other studies have assumed that the relationship is best described by a log-linear form, i.e., the relationship between the natural logarithm of Y and PM is estimated by a linear regression. Most common functions used in this analysis are in log-linear form with a few exceptions using logistic regressions.

A log linear C-R function is:

$$\Delta y = y_0 \ (e^{\beta \Delta PM} - 1) \cdot pop$$

where:

Δy = changes in the incidence of a health endpoint corresponding to a particular change in PM

y_0 = baseline incidence rate per person

β= coefficient

ΔPM = change in PM concentration

pop = population of a particular group that a study considered.

The parameters in the functions differ depending on the study. Some studies of the relationship between ambient PM concentrations and mortality excluded accidental deaths from their mortality counts; others included all deaths. Some studies considered only members of a particular subgroup of the population, e.g., individuals 65 and older, while other studies considered the entire population in the study location. When using a C-R function from an epidemiological study to estimate changes in the incidence of a health endpoint corresponding to a particular change in PM in a location, it is important to use the appropriate value of parameters for the C-R function. That is, the measure of PM, the type of population, and the characterization of the health endpoint should be the same as or as close as possible to those used in the study that estimated the C-R function.

9.1.5.2 Baseline Incidences

The health effect baseline incidences are the baseline incidence rate in a specific location multiplied by the relevant population. In this analysis, California county mortality rates were used in the estimation of air pollution-related mortality. Hospital admissions were calculated at the state level for a given population age group based on “Patient Discharge Data 1998-1999”, California Office of Statewide Health Planning and Development, December 2000. All counties were assumed to have the same incidence rate for a given population age group. For some endpoints, such as respiratory symptoms, respiratory illnesses, and restricted activity days, we used estimates of baseline incidence rates from the studies reporting the C-R functions for those health endpoints because California specific baseline rates are not available.

9.1.5.3 Thresholds

Different assumptions about whether to apply thresholds, and at what levels, can have a major effect on health effects estimates. A very important issue in estimating PM health effects is whether it is valid to apply the C-R functions throughout the range of predicted changes in ambient concentrations, even changes occurring at levels approaching the natural background concentration (without any human activity).

There is some evidence that, at least for particulate matter, not only is there no threshold, but the PM effect coefficient may actually be larger at lower levels of PM and smaller at higher levels (Rossi et al., 1999). However, we used the background concentration of PM as a threshold for estimating the health effects presented in this analysis. As a result, adverse health effects may be underestimated.

The Point Reyes National Seashore in Northern California is located away from populated areas and other significant sources of particulate and particulate precursor emissions. Thus the PM concentration at this site may represent an estimate of PM concentrations in the absence of anthropogenic emissions. Data obtained from the IMPROVE program for Point Reyes from

March 1996 through February 1999 indicate that annual average concentrations were 4.55 $\mu g/m^3$ for PM2.5 and 10.97 $\mu g/m^3$ for PM10. In this analysis, we applied thresholds of 5 $\mu g/m^3$ for PM2.5 and 10 $\mu g/m^3$ for PM10 in all the epidemiological functions except for the long-term mortality functions where we used 9 $\mu g/m^3$ for PM2.5 and 18 $\mu g/m^3$ for PM10—the lowest concentration levels observed in the two long-term mortality studies. We assumed that all of these functions were continuous and differentiable down to threshold levels.

9.1.5.4 Mortality

Premature mortality may result from either short-term or long-term exposure to pollution concentrations. Short-term exposure may result in excess mortality on the same day or within a few days of increased exposure. Long-term exposure (over a year or more) may result in mortality in excess of what it would be if PM levels were generally lower. Long-term exposure may capture a facet of the association between PM and mortality that is not captured by short-term exposure.

Long-term epidemiological studies estimate the association between long-term (chronic) exposure to air pollution and the survival of members of a large study population over an extended period of time. Such studies examine the health endpoint(s) in relation to the general long-term level of the pollutant, for example, relating annual mortality to some measure of annual pollutant level. In contrast, short-term studies relate daily levels of the pollutant to daily mortality. By their basic design, daily studies can detect acute effects but not the effects of long-term exposures. A chronic exposure study design is best able to identify the long-term exposure effects, and may detect some of the short-term exposure effects as well. Therefore, a sum of estimated effects from both study types would likely result in some degree of double counting of the effects.

The following four studies were used to estimate PM related mortality.

9.1.5.4.1 Long-term Mortality (Krewski et al., 2000) Based on ACS Cohort

This study is a re-analysis of the Pope et al. (1995) study of PM2.5 associated mortality, using American Cancer Society (ACS) data. It essentially confirms the original findings. An advantage of Krewski et al. over Pope et al. is that the reanalysis uses the annual mean PM2.5 concentration rather than the annual median. Because the mean is affected more by high PM values than by the median, if high PM days are important in causing premature mortality, the annual mean may be preferable to the median as a measure of long-term exposure. We used this study to derive primary estimates of premature mortality.

The C-R function to estimate the change in long-term mortality is:

$$\Delta \text{Mortality} = - y_0 \ (e^{-\beta \Delta PM} - 1) \cdot pop$$

where:

y_0 = county-level all-cause annual death rate per person ages 30 and older

β = PM2.5 coefficient = 0.0046257, PM10 coefficient = 0.00231285

ΔPM = change in annual mean PM concentration

pop = population of ages 30 and older

σ_β = standard error of β PM2.5 = 0.0012046, PM10 = 0.0006023

Incidence Rate. To estimate county-specific baseline mortality incidence among individuals ages 30 and over, we used data from 1999 annual all cause deaths by age by county (Center for Health Statistics, California Department of Health, 1999).

Coefficient Estimate (β). The coefficient (β) for PM2.5 is estimated from the relative risk (1.12) associated with a mean change of 24.5 $\mu g/m^3$ (Krewski et al., 2000, Part II - Table 31).

Recent findings reported by Pope et al. of a new analysis of the American Cancer Society data show no association with long-term mortality for coarse particles (PM10 – PM2.5) (Pope et al., 2002). Based on the assumptions that: (1) only PM2.5 (fine PM) is associated with long-term mortality; (2) the reduction in PM10 will maintain the current proportion of PM2.5 in California; (3) and state average fine and coarse PM fraction is about 50-50, the coefficient for PM10 was derived by multiplying the PM2.5 coefficient by 0.5. Using this adjusted PM10 coefficient, we only calculated long-term mortality effects for the PM2.5 fraction of PM10. The standard error for PM10 was also adjusted accordingly.

Standard Error (σ_β). The standard error for PM2.5 was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Krewski et al., 2000, Part II – Table 31).

9.1.5.4.2 Long-term Mortality (Krewski et al., 2000) Based on Six-City Cohort by Dockery

Krewski et al., (2000) also reanalyzed the data from another prospective cohort study (the Harvard "Six Cities Study") authored by Dockery et al., (1993). The Dockery et al., study used a smaller sample of individuals from fewer cities than the study by Pope et al., (1995); however, it features improved exposure estimates, a slightly broader study population (adults aged 25 and older), and a follow-up period nearly twice as long as that of Pope et al., We used this study for alternative estimates of long-term mortality effects.

The C-R function is:

$$\Delta \text{ Mortality} = - y_0 \ (e^{-\beta \Delta PM} - 1) \cdot pop$$

where:

y_0 = county-level all-cause annual death rate per person ages 25 and older

β= PM2.5 coefficient = 0.013272, PM10 coefficient = 0.006636

ΔPM = change in annual mean PM concentration

pop = population of ages 25 and older

σ_β= standard error of β PM2.5= 0.00407, standard error of β PM10= 0.00204

Incidence Rate. To estimate county-specific baseline mortality incidence among individuals ages 25 and over, we used the data from 1999 annual all cause deaths by age by county (Center for Health Statistics, California Department of Health, 1999).

Coefficient Estimate (β). The coefficient (β) for PM2.5 is estimated from the relative risk (1.28) associated with a mean change of 18.6 (Krewski et al., 2000, Part I - Table 19c). The coefficient for PM10 was adjusted by multiplying the PM2.5 coefficient by 0.5 so that we only calculate a long-term mortality benefit for the PM2.5 fraction of PM10. The standard error for PM10 was also adjusted accordingly.

Standard Error (σ_β). The standard error for PM2.5 was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Dockery et al., 1993, Table 5)

9.1.5.4.3 Short-Term Mortality (Schwartz et al., 1996)

Schwartz et al., (1996) pooled the results from six cities in the U.S. and found a significant relationship between daily PM2.5 concentration and non-accidental mortality. Abt Associates, Inc. (1996b, p. 52) used the six PM2.5 relative risks reported by Schwartz et al., in a three-step procedure to estimate a pooled PM2.5 coefficient and its standard error. The first step estimates a random-effects pooled estimate of β; the second step uses an "empirical Bayes" procedure to re-estimate the β for each study as a weighted average of the β reported for that location and the random effects pooled estimate; and the third step estimates the underlying distribution of β, and uses a Monte Carlo procedure to estimate the standard error (Abt Associates, Inc., 1996a, p. 65).

The C-R function to estimate the change in mortality associated with daily changes in PM2.5 is:

$$\Delta \text{ Mortality} = - y_0 \ (e^{-\beta \Delta PM} - 1) \cdot \text{pop}$$

where:

y_0 = county-level daily incidence for non-accidental deaths per person of any age

β = PM2.5 coefficient (Abt Associates Inc., 1996a, Exhibit 7.2) = 0.001433

ΔPM = change in daily average PM2.5 concentration

pop = population of all ages

σ_β = standard error of β (Abt Associates Inc., 1996a, Exhibit 7.2) = 0.000129

9.1.5.4.4 Short-Term Mortality (Pooled California PM10 studies, Chestnut, et al., 2001)

A number of daily time-series studies have examined the PM-premature mortality relationship in California populations. Some of the study details and the PM relative risk results from these studies are presented in Table 9.2. Chestnut and Mills pooled PM10 results from each of the counties represented in this table in a random effects model. For counties with more than one set of PM10 results, those estimates were pooled first and the results from a fixed effects assumption were incorporated with the results for the remaining locations. Only PM10 results were used so no results from Kinney and Ozkaynak (1991) or from Ostro (1995) are included in the pooled estimate. The result of the pooled PM10 studies is shown in the last row of the table and it applies to all ages and non-accidental deaths.

Table 9.2. Daily time series study results of impact of PM on daily mortality in California.

Study	Study location (years)	PM measure used in study	Pollutant covariates included	Estimated Beta (std. Err)	Relative risk for a 10 µg/m^3 (95% CI)
Fairley, 1999	Santa Clara (1989-1996)	PM2.5	Ozone, CO, NO_2	0.004365 (0.001694)	1.045 (1.010, 1.080)
		PM10	None	0.001539 (0.000598)	1.016 (1.004, 1.027)
Kinney and Ozkaynak, 1991	Los Angeles (1970-1979)	KM[b]	Oxides	N/A (linear regression used)	1.008 (1.005, 1.012)
Kinney et al., 1995	Los Angeles (1985-1990)	PM10	Ozone	0.000488 (0.000284)	1.005 (0.999, 1.010)
Ostro, 1995	San Bernardino and Riverside Counties (1980-1986)	PM2.5 (est)	None	0.000000 (0.000311)	1.000 (0.994, 1.006) (full year)
Ostro et al., 1999	Coachella Valley (1989-1992)	PM10	None	0.001128 (0.000747)	1.011 (0.997, 1.026)
Samet et al., 2000b	Los Angeles County (1987-1994)	PM10	Ozone	0.000419 (0.000188)	1.004 (1.001, 1.008)
	San Diego County (1987-1994)	PM10	Ozone	0.001124 (0.000467)	1.011 (1.002, 1.021)
	Orange County (1987-1994)	PM10	Ozone	0.001025 (0.000523)	1.010 (1.000, 1.021)
	Santa Clara County (1987-1994)	PM10	Ozone	0.000369 (0.000350)	1.004 (0.997, 1.011)
	San Bernardino County (1987-1994)	PM10	Ozone	0.000310 (0.000687)	1.003 (0.990, 1.017)
	Alameda County (1987-1994)	PM10	Ozone	0.002000 (0.000572)	1.020 (1.009, 1.032)
Random Effects Pooling, Chestnut et al., 2001[c]	**All counties represented in table**	**PM10**	**N/A**	**0.000838 (0.000203)**	**1.008 (1.004, 1.012)**

a. Mortality in these studies is non-accidental mortality, which excludes deaths attributed to homicide, suicide, legal intervention, or other accidental causes.

b. KM is a measure of visual opacity in the air, which is related to particulate matter. The mean value for KM in this study was 25.

c. Only studies that measured PM10 were pooled in the random effects model.

9.1.5.5 Chronic Bronchitis (Abbey et al., 1995 and 1993, California)

Abbey et al. (1995) examined the relationship between estimated PM2.5 (annual mean from 1966 to 1977), PM10 (annual mean from 1973 to 1977), and total suspended particulate (TSP, annual mean from 1973 to 1977) and the same chronic respiratory symptoms in a sample population of 1,868 Californians. The initial survey was conducted in 1977 and the final survey in 1987. To ensure a better estimate of exposure, the study participants had to have been living in the same area for an extended period of time. In single-pollutant models, there was a statistically significant PM2.5 relationship with development of chronic bronchitis, but not for airway obstructive disease (AOD) or asthma; PM10 was significantly associated with chronic bronchitis and AOD; and TSP was significantly associated with all cases of all three chronic symptoms.

The C-R function to estimate the change in chronic bronchitis is:

$$\Delta \text{Chronic Bronchitis} = - y_0 \ (e^{-\beta \Delta PM} - 1) \cdot pop$$

where:

y_0 = annual bronchitis incidence rate per person = 0.00378 (Abbey et al., 1993, Table 3)

β = estimated PM2.5 coefficient = 0.0132, PM10 coefficient = 0.00932

ΔPM = change in annual average PM concentration

Pop = population of ages 27 and older without chronic bronchitis = 0.9465*population 27+

σ_β = standard error of β = 0.00680 for PM2.5, 0.00475 for PM10

Incidence Rate. The estimation of the incidence rate is detailed in "Final Heavy Duty Engine/Diesel Fuel Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit Analysis Results, Appendix C", U.S. EPA, December 2000.

Coefficient Estimate (β). The estimated coefficient (β) for PM2.5 is based on the relative risk (= 1.81) associated with 45 $\mu g/m^3$ change in PM2.5 (Abbey et al., 1995, Table 2). The estimated coefficient (β) for PM10 is based on the relative risk (= 1.36) associated with 60 $\mu g/m^3$ change in TSP (Abbey et al., 1993, Table 5). Assuming that PM10 is 55% of TSP and that particulate greater than 10 micrometers are harmless.

Standard Error (σ_β). The standard error for the PM2.5 coefficient (β) is calculated from the reported lower and upper bounds of the relative risk (0.98 to 3.25) (Abbey et al., 1995, Table 2).

9.1.5.6 Hospital Admissions

Studies of a possible PM-hospitalization relationship have been conducted for a number of locations in the United States, including California. These studies use a daily time-series design and focus on hospitalizations with a first-listed discharge diagnosis attributed to diseases of the circulatory system (ICD9-CM codes 390-459) or diseases associated with the respiratory system (ICD9-CM codes 460-519). Subcategories within these groups are also often examined, with variation between studies in how the categories are defined. Common subcategories within circulatory are cardiovascular, which includes heart attack, and cerebrovascular, which includes stroke. Common subcategories within respiratory are chronic obstructive pulmonary disease (COPD), asthma, and pneumonia. Various age grouping are also considered, which vary across studies.

Some studies have examined the relationship between air pollution and emergency room (ER) visits. Because most emergency room visits do not result in an admission to the hospital we treated hospital admissions and ER visits separately, taking account of the fraction of ER patients that were admitted to the hospital.

9.1.5.6.1 Hospital Admissions for COPD (Samet et al., 2000a, 14 Cities)

Samet et al. (2000a) examined the relationship between air pollution and hospital admissions for individuals age 65 and over in 14 cities across the country. Cities were selected on the basis of available air pollution data for at least four years between 1985 and 1994 during which at least 50% of days had observations between the city-specific start and end of measurements.

The C-R function to estimate the change in hospital admissions for COPD associated with daily changes in PM10 is:

$$\Delta \text{ COPD Admissions} = - y_0 \ (e^{-\beta \Delta PM} - 1) \cdot pop$$

where:

y_0.= daily hospital admission rate for COPD per person 65 and older = 2.59 E-5

β = PM10 coefficient = 0.00288

ΔPM = change in daily average PM concentration

pop = population age 65 and older

σ_β = standard error of β = 0.00139

Incidence Rate. COPD hospital admissions (ICD-9 codes: 490-492, 494-496) are based on "Patient Discharge Data 1998-1999," California Office of Statewide Health Planning and Development, 2000. Population data are from "Race/Ethnic Population with Age and Sex Detail, 1970-2040", California Department of Finance.

Coefficient Estimate (β). The coefficient is estimated from relative risk of 1.029 which is based on a 2.88 percent increase in admissions due to a PM10 change of 10.0 $\mu g/m^3$ (Samet et al., 2000a, Part II - Table 14).

Standard Error (σ_β) The standard error was calculated as the average of the standard errors implied by the reported lower and upper bounds of the percent increase (Samet et al., 2000a, Part II - Table 14)

9.1.5.6.2 Hospital Admissions for Pneumonia (Samet et al., 2000a, 14 Cities)

The C-R function to estimate the change in hospital admissions for pneumonia associated with daily changes in PM is:

$$\Delta \text{ Pneumonia Admissions} = - y_0 \ (e^{-\beta \Delta PM} - 1) \cdot pop$$

where:

y_0 = daily hospital admission rate for pneumonia per person 65 and older = 5.16 E-5

β = PM10 coefficient = 0.00207

ΔPM = change in daily average PM concentration

pop = population age 65 and older

σ_β = standard error of β = 0.00058

Incidence Rate. Pneumonia hospital admissions (ICD-9 codes: 480-487) are based on "Patient Discharge Data 1998-1999," California Office of Statewide Health Planning and Development, 2000. Population data are from "Race/Ethnic Population with Age and Sex Detail, 1970-2040", California Department of Finance.

Coefficient Estimate (β). The coefficient is estimated from relative risk of 1.021 which is based on a 2.07 percent increase in admissions due to a PM10 change of 10.0 $\mu g/m^3$ (Samet et al., 2000a, Part II - Table 14).

Standard Error (σ_β). The standard error was calculated as the average of the standard errors implied by the reported lower and upper bounds of the percent increase (Samet et al., 2000a, Part II - Table 14)

9.1.5.6.3 Hospital Admissions for Cardiovascular Disease (Samet et al., 2000a, 14 Cities)

The C-R function to estimate the change in hospital admissions for cardiovascular disease associated with daily changes in PM10 is:

$$\Delta \text{CVD Admissions} = - y_0 \ (e^{-\beta \Delta PM} - 1) \cdot pop$$

where:

y_0 = daily hospital admission rate for cardiovascular disease per person 65 and older = 1.58E-4

β = PM10 coefficient = 0.00119

ΔPM = change in daily average PM concentration

pop = population age 65 and older

σ_β = standard error of β = 0.00011

Incidence Rate. Congestive heart failure hospital admissions (ICD-9 codes: 390-429) are based on "Patient Discharge Data 1998-1999," California Office of Statewide Health Planning and Development, 2000. Population data are from "Race/Ethnic Population with Age and Sex Detail, 1970-2040", California Department of Finance.

Coefficient Estimate (β). The coefficient is estimated from a relative risk of 1.012 which is based on a 1.19 percent increase in admissions due to a PM10 change of 10.0 $\mu g/m^3$ (Samet et al., 2000a, Part II - Table 14).

Standard Error (σ_β). The standard error was calculated as the average of the standard errors implied by the reported lower and upper bounds of the percent increase (Samet et al., 2000a, Part II - Table 14)

9.1.5.6.4 Hospital Admissions for Asthma (Sheppard et al., 1999, Seattle)

Sheppard et al. (1999) studied the relation between air pollution in Seattle and non-elderly hospital admissions for asthma from 1987 to 1994. They used air quality data for PM10, PM2.5, coarse PM2.5-10, SO2, ozone, and CO in a Poisson regression model with controls for time trends, seasonal variations, and temperature-related weather effects. They found asthma hospital admissions associated with PM10, PM2.5, coarse PM2.5-10, CO, and ozone. The C-R function is based on a two-pollutant model with CO and PM2.5 and PM10 single-pollutant model:

$$\Delta \text{Asthma Admissions} = - y_0 \ (e^{-\beta \Delta PM} - 1) \cdot pop$$

where:

y_0 = daily hospital admission rate for asthma per person = 2.63 E-6

β = PM2.5 coefficient = 0.002505, PM10 coefficient = 0.002568

ΔPM = change in daily average PM concentration

pop = population of ages less than 65

σ_β = standard error of PM2.5 β = 0.001045, standard error of PM10 β = 0.0007674

Incidence Rate. Hospital admissions for asthma (ICD-9 code: 493) are based on "Patient Discharge Data 1998-1999," California Office of Statewide Health Planning and Development, 2000. Population data are from "Race/Ethnic Population with Age and Sex Detail, 1970-2040", California Department of Finance.

Coefficient Estimate (β). Based on a model with CO, the daily average coefficient is estimated from the relative risk (1.03) associated with a change in PM2.5 exposure of 11.8µg/m^3 (Sheppard et al., 1999, Table 3 and p. 28).

Standard Error (σ_β). The standard error was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Sheppard et al., 1999, p. 28).

9.1.5.6.5 Emergency Room Visits for Asthma (Schwartz et al., 1993, Seattle)

Schwartz et al. (1993) examined the relationship between air quality and emergency room visits for asthma in persons under 65, and 65 and over who lived in Seattle from September 1989 to September 1990. Using single-pollutant models they found daily levels of PM10 linked to ER visits in individuals younger than 65.

The C-R function to estimate the change in daily emergency room visits for asthma associated with daily changes in PM10 is:

$$\Delta \text{Asthma ER Visits} = - y_0 \ (e^{-\beta \Delta PM} - 1) \cdot pop$$

where:

y_0 = daily ER visits for asthma per person under 65 years old = 4.48 E-6

β = PM10 coefficient (Schwartz et al., 1993, p. 829) = 0.00367

ΔPM = change in daily average PM concentration

pop = population of ages 0-64

σ_β = standard error of β (Schwartz et al., 1993, p. 829) = 0.00126

Incidence Rate. Smith et al. (1997, p. 789) reported that in 1987 there were 445,000 asthma admissions and 1.2 million asthma ER visits. Assuming that all asthma hospital admissions pass through the ER room, then 37% of ER visits end up as hospital admissions. By subtracting out those visits that end up as admissions, ER visits = 1.7*asthma admission rate = 1.7*2.63 E-6 =4.48 E-6. Asthma hospital admissions (ICD-9 code: 493) rate are based on "Patient Discharge Data 1998-1999," California Office of Statewide Health Planning and Development, 2000, and population data are from "Race/Ethnic Population with Age and Sex Detail, 1970-2040", California Department of Finance.

9.1.5.7 Minor Illness

In addition to chronic illnesses and hospital admissions, there is considerable scientific research that has reported significant relationships between elevated air pollution levels and other morbidity effects. Controlled human studies have established relationships between air pollution

and symptoms such as cough, pain on deep inspiration, wheeze, eye irritation and headache. In addition, epidemiological research has found relationships between air pollution exposure and acute infectious diseases (e.g., bronchitis, sinusitis) and a variety of "symptom-day" categories. Some "symptom-day" studies examine excess incidences of days with identified symptoms such as wheeze, cough, or other specific upper or lower respiratory symptoms. Other studies estimate relationships for days with a more general description of days with adverse health impacts, such as "respiratory restricted activity days" or work loss days.

We selected a few endpoints that reflect some minor morbidity effects and carefully adjusted estimates to avoid double counting (e.g., adjusted minor restricted activity days by number of asthma attacks).

9.1.5.7.1 Acute Bronchitis C-R Function (Dockery et al., 1996)

Dockery et al. (1996) examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a study of 13,369 children ages 8-12 living in 24 communities in the U.S. and Canada. Health data were collected in 1988-1991, and single-pollutant models were used in the analysis to test a number of measures of particulate air pollution. The study found that there was a marginally significant relationship between PM and bronchitis.

The C-R function to estimate the change in acute bronchitis is:

$$\Delta Acute\ Bronchitis = -[\frac{y_0}{(1-y_0)\cdot e^{\ \Delta PM\beta} + y_0} - y_0\ ].pop$$

where:

Y_0= annual bronchitis incidence rate per person = 0.044

β = estimated PM2.5 logistic regression coefficient = 0.0272

ΔPM = change in annual average PM concentration

pop = population of ages 8-12

σ_β = standard error of β = 0.0171

Incidence Rate. The estimation of incidence rate is detailed in "Final Heavy Duty Engine/Diesel Fuel Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit Analysis Results, Appendix C", U.S. EPA, December 2000.

Coefficient Estimate (β). The estimated logistic coefficient is based on the odds ratio (= 1.50) associated with being in the most polluted city ($PM_{2.1}$= 20.7 µg/m^3) versus the least polluted city ($PM_{2.1}$=5.8 µg/ m^3) (Dockery et al., 1996, Tables 1 and 4). We applied the PM2.1 coefficient to PM2.5 and PM10.

Standard Error (σ_β) The standard error of the coefficient is calculated from the reported lower and upper bounds of the odds ratio (Dockery et al., 1996, Table 4)

9.1.5.7.2 Upper Respiratory Symptoms (Pope et al., 1991)

Using logistic regression, Pope et al. (1991) estimated the impact of PM10 on the incidence of a variety of minor symptoms in 55 subjects (34 "school-based" and 21 "patient-based") living in the Utah Valley from December 1989 through March 1990. The children in the Pope et al. study were asked to record respiratory symptoms in a daily diary. Pope et al. defined upper respiratory symptoms as consisting of one or more of the following symptoms: runny or stuffy

nose; wet cough; and burning, aching, or red eyes. The sample in this study was relatively small and is most representative of the asthmatic population, rather than the general population. The school- based subjects (ages 9 to 11) were chosen based on "a positive response to one or more of three questions: ever wheezed without a cold, wheezed for 3 days or more out of the week for a month or longer, and/or had a doctor say the 'child has asthma' (Pope et al., 1991, p. 669)." The patient-based subjects (ages 8 to 72) were receiving treatment for asthma and were referred by local physicians. Regression results for the school-based sample (Pope et al., 1991, Table 5) showed PM10 significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM10 effect. The results from the school-based sample are used here.

The C-R function used to estimate the change in upper respiratory symptoms is:

$$\Delta Upper\ Respiratory\ Symptoms = -[\frac{y_0}{(1-y_0)\cdot e^{\Delta PMb} + y_0} - y_0]\cdot pop$$

where:

y_0 = daily upper respiratory symptom incidence rate per person = 0.3419

β = estimated PM10 logistic regression coefficient = 0.0036 (Pope et al., 1991, Table 5)

ΔPM = change in daily average PM concentration

pop = asthmatic population ages 9 to 11 = 6.91% of population ages 9 to 11

σ_β = standard error of β (Pope et al., 1991, Table 5) = 0.0015

Incidence Rate. The incidence rate is published in Pope et al. (Pope et al., 1991, Table 2). Taking a sample-size-weighted average, one gets an incidence rate of 0.3419.

9.1.5.7.3 Lower Respiratory Symptoms (Schwartz et al., 1994)

Schwartz et al. (1994) used logistic regression to link lower respiratory symptoms in children with SO_2, NO_2, ozone, PM10, PM2.5, sulfate and H^+ (hydrogen ion). Children were selected for the study if they were exposed to indoor sources of air pollution: gas stoves and parental smoking. The study enrolled 1,844 children in 1984 into a year-long study. The study was conducted in different years (1984 to 1988) in six cities. The students were in grades two through five at the time of enrollment in 1984. By the completion of the final study, the cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

In single pollutant models SO_2, NO_2, PM2.5, and PM10 were significantly linked to coughing. In two-pollutant models, PM10 had the most consistent relationship with coughing. In models for upper respiratory symptoms, they reported a marginally significant association for PM10. In models for lower respiratory symptoms, they reported significant single-pollutant models, using SO_2, O_3, PM2.5, PM10, SO4, and H^+.

The C-R function used to estimate the change in lower respiratory symptoms is:

$$\Delta Lower\ Respiratory\ Symptoms = -[\frac{y_0}{(1-y_0)\cdot e^{\Delta PMb} + y_0} - y_0]\cdot pop$$

where:

y_0 = daily lower respiratory symptom incidence rate per person = 0.0012

β = estimated PM2.5 logistic regression coefficient = 0.01823

ΔPM = change in daily average PM concentration

pop = population of ages 7-14

σ_β = standard error of β = 0.00586

Incidence Rate. The proposed incidence rate, 0.12 percent, is based on the percentiles in Schwartz et al. (Schwartz et al., 1994, Table 2). The calculation is detailed in "Final Heavy Duty Engine/Diesel Fuel Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit Analysis Results, Appendix C", U.S. EPA, December 2000.

Coefficient Estimate (β). The coefficient is calculated from the reported odds ratio (= 1.44) in a single-pollutant model associated with a 20 $\mu g/m^3$ change in PM2.5 (Schwartz et al., 1994, Table 5).

Standard Error (σ_β). The standard error for the coefficient is calculated from the reported lower and upper bounds of the odds ratio (Schwartz et al., 1994, Table 5).

9.1.5.7.4 Asthma Attacks, (Whittemore and Korn, 1980)

Whittemore and Korn (1980) examined the relationship between air pollution and asthma attacks in a survey of 443 children and adults, living in six communities in southern California during three 34-week periods in 1972-1975. The analysis focused on TSP and ozone. In a two-pollutants model, daily levels of both TSP and O_3 were significantly related to reported asthma attacks.

The C-R function to estimate the change in the number of asthma attacks is:

$$\Delta\ Asthma\ Attacks = -[\frac{y_0}{(1-y_0)\cdot e^{\Delta PMb} + y_0} - y_0]\cdot pop$$

where:

y_0 = daily incidence of asthma attacks = 0.027 (Krupnick, 1988, p. 4-6)

β = PM10 coefficient = 0.00144

ΔPM = change in daily PM concentration

pop = population of asthmatics of all ages = 5.61% of the population of all ages.
σ_β = standard error of β = 0.000556

Incidence Rate. The annual rate of 9.9 asthma attacks per asthmatic is divided by 365 to get a daily rate. A figure of 9.9 is roughly consistent with the recent statement that "People with asthma have more than [a combined] 100 million days of restricted activity" each year (National Heart, Lung, and Blood Institute 1997, p. 1). This 100 million incidence figure coupled with the 1996 population of 265,557,000 (U.S. Bureau of the Census, 1997, Table 2) and the latest asthmatic prevalence rate of 5.61% (Current Estimates From the National Health Interview Survey, 1994, US Department of Health and Human Services, 1995, Table 57), suggest an annual asthma attack rate per asthmatic of 6.7.

Coefficient Estimate (β). Based on a model with ozone, the coefficient is based on a TSP coefficient (0.00079) (Whittemore and Korn, 1980, Table 5). Assuming that PM10 is 55 percent of TSP and that particulates greater than ten micrometers are harmless.

Standard Error (σ_β). The standard error is calculated from the two-tailed p-value (<0.01) reported by Whittemore and Korn (1980, Table 5), which implies a t-value of at least 2.576 (assuming a large number of degrees of freedom).

9.1.5.7.5 Work Loss Days (Ostro, 1987)

Ostro (1987) estimated the impact of PM2.5 on the incidence of work-loss days (WLDs), restricted activity days (RADs), and respiratory-related RADs (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average PM2.5 levels were significantly linked to work-loss days, RADs, and RRADs, however there was some year-to-year variability in the results. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function used here is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight.

The C-R function to estimate the change in the number of work-loss days is:

$$\Delta \text{ WLD} = - y_0 \ (e^{-\beta \Delta PM} - 1) \cdot \text{pop}$$

where:

y_0 = daily work-loss-day incidence rate per person = 0.00648

β = inverse-variance weighted PM2.5 coefficient = 0.0046

ΔPM = change in daily average PM concentration

pop = population of ages 18 to 65

σ_β = standard error of β = 0.00036

Incidence Rate. The estimated 1994 annual incidence rate is the annual number (376,844,000) of WLD per person in the age 18-64 population divided by the number of people in 18-64 population (159,361,000). The 1994 daily incidence rate is calculated as the annual rate divided by 365. Data are from U.S. Bureau of the Census (1997, Table 14) and current estimates from the national health interview survey (CDC/NCHS 1998, Table 41).

Coefficient Estimate (β). The coefficient used in the C-R function is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight.

Standard Error (σ_β). The standard error of the coefficient calculation is detailed in “Final Heavy Duty Engine/Diesel Fuel Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit Analysis Results, Appendix C”, U.S. EPA, December 2000.

9.1.5.7.6 Minor Restricted Activity Days (Ostro and Rothschild, 1989)

Ostro and Rothschild (1989) estimated the impact of PM2.5 on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for PM2.5, two-week average O_3 has highly variable association with RRADs and MRADs. Controlling for O_3, two-week average PM2.5 was significantly linked to both health endpoints in most years.

The study is based on a sample of individuals ages 18-65. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that the elderly are at least as susceptible to PM as individuals 65 and younger. The elderly appear more likely to die due to

PM exposure than other age groups (e.g., Schwartz, 1994c, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994a; Schwartz, 1994b).

The coefficient used in this analysis is a weighted average of the coefficients in Ostro and Rothschild (1989), Table 4, using the inverse of the variance as the weight. The C-R function to estimate the change in the number of minor restricted activity days (MRAD) is:

$$\Delta MRAD = - y_0 \ (e^{-\beta \Delta PM} - 1) \cdot pop$$

where:

y_0 = daily MRAD daily incidence rate per person = 0.02137

β = inverse-variance weighted PM2.5 coeffcient = 0.00741

ΔPM = change in daily average PM concentration

pop = adult population ages 18 to 65

σ_β = standard error of β = 0.0007

Incidence Rate. The annual incidence rate (7.8) provided by Ostro and Rothschild (1989, p. 243) was divided by 365 to get a daily rate of 0.02137.

Coefficient Estimate (β). The coefficient is a weighted average of the coefficients in Ostro and Rothschild (1989, Table 4) using the inverse of the variance as the weight.

9.1.6 Applicability of the C-R functions in California

Since many epidemiological studies do not incorporate results from California, one may expect that the health effects of particulate matter in California are different than those in the rest of the United States. One of reasons there may be differences is that the composition of particulate matter varies significantly by region, and it is possible that not all types of particulate matter have the same health effects. One obvious difference between particulate matter in California (and elsewhere in western states) and the rest of the country is that the sulfate aerosol content is much lower in California.

Samet et al. (2000a) provide data that allow a simple illustration of this difference. They report mean levels of several criteria air pollutants for 1987 to 1994 in 20 of the largest cities and metropolitan areas in the United States, including 6 in California: Los Angeles, San Diego, Santa Ana-Anaheim, San Jose, San Bernardino, and Oakland. Sulfur dioxide (SO_2) and nitrogen dioxide (NO_2) are gaseous pollutants, but they are precursors to the sulfate and nitrate aerosols that make up a significant share of PM10. Table 9.3 shows that PM10, ozone, and NO_2 are all somewhat higher, on average, in California cities than in other U.S. cities, with the largest difference in NO_2. SO_2, on the other hand, is dramatically lower in California cities. The slightly higher concentrations of PM10 and ozone in California cities reflects to some extent the warm temperatures and sunny skies that contribute to the photochemical formation of ozone and fine particulates. Dramatically lower SO_2 concentrations in California reflect that, to the extent that coal is burned by electric utilities and other industrial sources, it is low sulfur (western) coal that is used. Coal mined in the eastern United States, and widely used as a fuel for power plants and other industrial sources, tends to have substantially higher sulfur content, which has a direct relationship with ambient SO_2 concentrations.

Table 9.3. Comparison of mean concentrations of selected air pollutants, 1987-1994

	PM10 (μg/m^3)	Ozone (ppb)	SO_2 (ppb)	NO_2 (ppb)
Six California cities	35.1	24.7	1.4	28.6
Fourteen other U.S. cities	31.9	22.3	6.7	22.3

Source: Samet et al., 2000a.

Although there has been substantial discussion in the literature of potential differences in health effects of various PM10 constituents, and some studies have reported that sulfate aerosols are more likely than other constituents to be a primary culprit, the findings regarding sulfate have not been consistent. There is sufficient evidence of PM10 health effects in locations (e.g., Los Angeles) where the sulfate content of PM10 is relatively low.

Numerous time-series studies provide opportunities to compare results obtained in California to those obtained in other locations in the United States. Comparing the results for PM10 in Table 9.2, the relative risks range from 1.003 to 1.020, with a mean value of 1.009. The weighted mean relative risk for all counties in California for PM10 is 1.008, with a 95% confidence interval of 1.004 to 1.012. This is within the range of mean results for studies throughout the United States, and suggests that the mortality effects of PM in California are comparable to those found in other locations in the United States.

Samet et al. (2000b) present the relative risk results for 20 cities in the United States, all estimated using the same estimation approach and years of data. They also estimate a pooled relative risk across all locations. The pooled relative risk for 10 μg/m^3 PM10 results across all 20 locations was 1.005. Removing the California locations from these results and averaging the relative risk results from the remaining 14 city/counties results in an average relative risk value of 1.004. By comparison, the average relative risk for the six California locations was 1.009, and ranged from 1.003 to 1.020 across these six locations. This comparison suggests that the daily time-series results for PM10 from California are similar to, if not slightly higher than those from other locations across the country. These results contradict the hypothesis that PM health effects in California may be lower because of the significantly lower sulfate content of PM in the West.

Based on our observations, in cases where the EPA adopted C-R functions that do not incorporate results from California, or where the contribution from the California-based segment of the study population is unclear, the weight of available evidence from the other health outcome categories is not sufficient enough to argue that differences in the composition of the ambient PM in California or aspects of the California population make using results from locations outside of California inappropriate. We therefore selected functions which were drawn from the results of non-California locations when the California-specific C-R functions are not available.

9.2 Health Effects Results

Applying results from the available epidemiologic studies to California data on PM suggests significant effects for both mortality and morbidity. For example, applying the prospective cohort, long-term exposure studies, the change in ambient PM2.5 from current levels in California (as described in Chapter 6) to an annual average of 12 μg/m^3 for all California counties is associated, in the long term with approximately 6,500 (95% CI = 3,200 to 9,800) fewer cases of

premature mortality per year, or about 2.9% of all mortality in the population above age 30 (see table 9.4). Use of the short-term exposure studies, which only capture part of the total effects on mortality, generates a mean estimate of approximately 2,600 fewer premature deaths per year (95% CI of 2,200 to 3,100) using a standard of 12 $\mu g/m^3$ PM2.5.

Mean annual estimates of reduced hospitalization associated with moving from current concentrations of PM2.5 to 12 $\mu g/m^3$, are approximately 600 for COPD, 900 for pneumonia, approximately 1,500 for cardiovascular disease and 500 for asthma. These effects are all associated with relatively short-term exposures to PM; no effects associated with long-term exposures are included in the hospital estimates. These estimates are fairly close to those derived using the California Kaiser data on hospitalization, which suggest a reduction of approximately 2,100 cases of hospitalization for circulatory diseases, 1,500 for chronic respiratory disease and 700 for acute respiratory disease. Finally, among children ages 7 to 14, current concentrations of PM2.5 are estimated to result in about 209,000 (95%CI 81,000 – 323,000) excess days of lower respiratory symptoms per year.

The estimated health benefits associated with meeting a lower annual average PM10 standard are also significant. These estimates are an alternative and not in addition to the PM2.5 estimates. Based on the analysis of Krewski et al. (2000) of the ACS cohort, long-term effects are only attributed to the fine particle share of PM10, not to all of PM10. As noted above, the other major prospective cohort long-term exposure study (Dockery et al., 1993) did find an apparent association between PM10 and mortality, therefore this assumption leads to lower effects from PM10. In addition, several morbidity endpoints appear to be associated with long-term exposure to PM10. Applying the prospective cohort, long-term exposure studies, the change in ambient PM10 from current levels in California (as estimated in Chapter 6) to an annual average of 20 $\mu g/m^3$ for all California counties is associated, in the long term, with 6,500 premature deaths (95% CI = 3,200 to 9,800) (see table 9.6), about 3% of all mortality for the cohort above age 30. Use of short-term exposure studies generates a mean estimate of 2,300 (95%CI = 1,200 to 3,400) premature deaths per year.

Table 9.4 and Table 9.5 summarize the estimated health effects of reducing PM2.5 concentration from current levels to 12 $\mu g/m^3$ and to the non-anthropogenic background of 5 $\mu g/m^3$ in California.

Table 9.6 and Table 9.7 summarize the estimated health effects of reducing PM10 concentration from current levels to 20 $\mu g/m^3$ and to the non-anthropogenic background of 11 $\mu g/m^3$ in California.

Table 9.8 to Table 9.10 present the estimated mortality, chronic illness, and hospital admission effects of reducing PM2.5 concentration from current levels to the non-anthropogenic background in all California counties.

9.3 Uncertainties of Risk Estimates

Among the uncertainties in the risk estimates is the degree of transferability of the concentration-response functions to California. However, eight California cities were included in the long-term exposure-mortality study of PM2.5 (Krewski et al., 2000), which involved a total of 63 cities, while the short-term exposure-mortality estimates were derived from studies of nine California cities (see Chapter 9). Similar risk estimates for mortality associated with acute PM10 exposure have been observed in over 60 cities throughout the world. In addition, similar quantitative estimates of the morbidity outcomes have been reported in multiple cities and/or have been conducted in California. Therefore, generalizing these results appears reasonable. There is still some uncertainty, however, concerning the choice of the specific studies and concentration-response functions used in this risk assessment. In this case, we used

concentration-response functions that had been reviewed and judged as acceptable by U.S. EPA's Science Advisory Board. For example, although we used the results of single-day exposures in the short-term exposure-mortality studies, application of studies using multi-day averages would have generated higher effect estimates. As another example, the prospective cohort studies using the results from the ACS (Pope et al., 1995) and Harvard Six-Cities (Dockery et al., 1993) cohorts could have been pooled, producing a higher estimate than relying only on the Pope et al. study.

A second uncertainty involves the issue of co-pollutants. Specifically, it is possible that some of the estimated health effects include the effects of both PM and other correlated pollutants. Many of the daily exposure studies isolated an independent effect of PM and/or tested for possible interactions or joint effects with other pollutants. However, given inherent errors in measurement of exposure to ambient pollutant, it is possible that PM is serving as an index for a mix of combustion-related pollutants or other sources of pollutants. As indicated by Chen et al. (1999) either underfitting or misfitting a model has implications for statistical inference. Specifically, it is well recognized that omitted variable bias may result in biased estimates of both the coefficient and standard error. If the other pollutants are causally associated with the health endpoint, then clearly an effect attributed solely to PM would be biased. It should be noted, however, that SB25 requires OEHHA to consider possible effects of exposure to multiple pollutants in evaluating ambient air quality standards. Thus, insofar as the PM concentration-response association may include effects of other pollutants, this is in accordance with the statutory requirements. In addition, there is uncertainty related to the use of the existing network of monitors to represent community exposures. There will be some error in these measurements, depending on the location of these monitors and the spatial pattern of the pollutants.

Finally, estimates for only a subset of adverse outcomes are provided. For example, estimates of the effects of PM on cancer incidence and infant mortality are not provided. In addition, no estimates on averting behavior are provided. This would include measures that are taken to prevent symptoms from occurring in the first place, such as avoiding strenuous exertion on days with high PM, staying indoors, use of prophylactic medication, purchasing of air filters, and so forth.

Table 9.4. California Annual PM2.5 Health Effects Benefits from Achieving 12 μg/m^3 Standard*

Health Endpoint	Reference	Estimated Beta (Standard Error)	Avoided Incidence (cases/year)		
			5th Percentile	Mean	95th Percentile
Mortality					
Long-Term Exposures Mortality					
Ages 30+	Krewski et al., 2000	0.0046257 (0.0012046)	3,229	6,526	9,754
Short-Term Exposures Mortality					
All Ages	Schwartz, 1996	0.001433 (0.000129)	1,604	1,945	2,286
Chronic Illness					
Chronic Bronchitis (Age 27+)	Abbey, 1995	0.0132 (0.00680)	-59	5,749	10,907
Hospitalization					
COPD (ICD codes 490-492, 494-496), Age 65+	Samet et al., 2000a	0.002880 (0.001390)	33	600	1,154
Pneumonia (ICD codes 480-487), Age 65+	Samet et al., 2000a	0.002070 (0.000580)	391	864	1,331
Cardiovascular (ICD codes 390-429), Age 65+	Samet et al., 2000a	0.001190 (0.000110)	1,254	1,530	1,806
Asthma (ICD codes 493), Age 64-	Sheppard et al., 1999	0.002505 (0.001045)	86	470	846
Asthma-related ER Visits, Age 64-	Schwartz et al., 1993	0.003670 (0.001260)	386	1,167	1,930
Minor Illness					
Acute Bronchitis, Age 8-12	Dockery et al., 1996	0.02720 (0.01710)	-4,663	17,473	34,149
URS, Age 9-11	Pope et al., 1991	0.00360 (0.0015)	38,371	208,384	376,874
LRS, Age 7-14	Schwartz et al., 1994	0.01823 (0.00586)	81,284	208,638	323,322
Asthma Attacks, All ages	Whittemore and Korn, 1980	0.00144 (0.000556)	41,390	169,381	296,178
Work Loss Days	Ostro, 1987	0.0046 (0.00036)	1,227,554	1,445,391	1,661,848
Minor Restricted Activity Days –adjusted**	Ostro & Rothschild, 1989	0.00741 (0.0007)	6,175,290	7,413,386	8,635,934

* Base period 1998-2000
** To avoid double counting, the number of asthma attacks estimated were subtracted from the number of MRADs.

Table 9.5. California Annual PM2.5 Health Effects Benefits of Reduced PM2.5*

Health Endpoint	Reference	Estimated Beta (Standard Error)	Avoided Incidence (cases/year)		
			5th Percentile	Mean	95th Percentile
Mortality					
Long-Term Exposures Mortality					
Ages 30+	Krewski et al., 2000	0.0046257 (0.0012046)	4,659	9,391	13,999
Short-Term Exposures Mortality					
All Ages	Schwartz, 1996	0.001433 (0.000129)	3,312	4,014	4,714
Chronic Illness					
Chronic Bronchitis (Age 27+)	Abbey, 1995	0.0132 (0.00680)	-122	11,414	20,918
Hospitalization					
COPD (ICD codes 490-492, 494-496), Age 65+	Samet et al., 2000a	0.002880 (0.001390)	68	1,242	2,369
Pneumonia (ICD codes 480-487), Age 65+	Samet et al., 2000a	0.002070 (0.000580)	814	1,791	2,751
Cardiovascular (ICD codes 390-429), Age 65+	Samet et al., 2000a	0.001190 (0.000110)	2,608	3,180	3,750
Asthma (ICD codes 493), Age 64-	Sheppard et al., 1999	0.002505 (0.001045)	176	950	1,702
Asthma-related ER Visits, Age 64-	Schwartz et al., 1993	0.003670 (0.001260)	783	2,352	3,864
Minor Illness					
Acute Bronchitis, Age 8-12	Dockery et al., 1996	0.02720 (0.01710)	-9,567	32,923	59,724
URS, Age 9-11	Pope et al., 1991	0.00360 (0.0015)	77,367	418,985	755,504
LRS, Age 7-14	Schwartz et al., 1994	0.01823 (0.00586)	160,279	398,777	600,088
Asthma Attacks, All ages	Whittemore and Korn, 1980	0.00144 (0.000556)	84,439	344,532	600,679
Work Loss Days	Ostro, 1987	0.0046 (0.00036)	2,487,857	2,923,535	3,354,714
Minor Restricted Activity Days –adjusted**	Ostro & Rothschild, 1989	0.00741 (0.0007)	12,439,319	14,873,148	17,257,232

*Mortality estimates for achieving 9 $\mu g/m^3$, other effects to 5 $\mu g/m^3$ (background). Base period 1998-2000.
**To avoid double counting, the number of asthma attacks estimated were subtracted from the number of MRADs.

Table 9.6. California Annual PM10 Health Effects Benefits from Achieving 20 μg/m^3 Standard*

Health Endpoint	Reference	Estimated Beta (Standard Error)	Avoided Incidence (cases/year)		
			5th Percentile	Mean	95th Percentile
Mortality					
Long-Term Exposures Mortality					
Ages 30+	Krewski et al., 2000	0.00231285 (0.0006023)**	3,236	6,533	9,753
Short-Term Exposures Mortality					
All Ages	Pooled California Studies (Chestnut & Mills, 2001)	0.000838 (0.000203)	1,210	2,295	3,373
Chronic Illness					
Chronic Bronchitis (Age 27+)	Abbey, 1993	0.00932 (0.00475)	10	7,850	14,500
Hospitalization					
COPD (ICD codes 490-492, 494-496), Age 65+	Samet et al., 2000a	0.002880 (0.001390)	66	1,191	2,256
Pneumonia (ICD codes 480-487), Age 65+	Samet et al., 2000a	0.002070 (0.000580)	785	1,721	2,636
Cardiovascular (ICD codes 390-429), Age 65+	Samet et al., 2000a	0.001190 (0.000110)	2,514	3,063	3,611
Asthma (ICD codes 493), Age 64-	Sheppard et al., 1999	0.002568 (0.000767)	402	955	1,493
Asthma-related ER Visits, Age 64-	Schwartz et al., 1993	0.003670 (0.001260)	771	2,301	3,757
Minor Illness					
Acute Bronchitis, Age 8-12	Dockery et al., 1996	0.02720 (0.01710)	-9,883	31,557	54,379
URS, Age 9-11	Pope et al., 1991	0.00360 (0.0015)	78,599	424,492	763,139
LRS, Age 7-14	Schwartz et al., 1994	0.01823 (0.00586)	160,586	389,225	572,660
Asthma Attacks, All ages	Whittemore and Korn, 1980	0.00144 (0.000556)	83,128	338,270	588,195
Work Loss Days	Ostro, 1987	0.0046 (0.00036)	2,399,490	2,814,815	3,224,423
Minor Restricted Activity Days -adjusted***	Ostro & Rothschild, 1989	0.00741 (0.0007)	11,933,013	14,215,093	16,435,564

* Base period 1998-2000
** PM2.5 coeffecient and standard error were muliplied by 0.5 assuming only the PM 2.5 fraction of PM10 was associated with long-term mortality.
** *To avoid double counting, the number of asthma attacks estimated were subtracted from the number of MRADs.

Table 9.7. California Annual PM10 Health Effects Benefits of Reduced PM10*

Health Endpoint	Reference	Estimated Beta (Standard Error)	Avoided Incidence (cases/year)		
			5th Percentile	Mean	95th Percentile
Mortality					
Long-Term Exposures Mortality					
Ages 30+	Krewski et al., 2000	0.00231285 (0.0006023)**	3,734	7,534	11,241
Short-Term Exposures Mortality					
All Ages	Pooled California Studies (Chestnut & Mills, 2001)	0.000838 (0.000203)	2,148	4,069	5,969
Chronic Illness					
Chronic Bronchitis (Age 27+)	Abbey, 1993	0.0032 (0.00475)	16	13,530	24,141
Hospitalization					
COPD (ICD codes 490-492, 494-496), Age 65+	Samet et al., 2000a	0.002880 (0.001390)	118	2,112	3,967
Pneumonia (ICD codes 480-487), Age 65+	Samet et al., 2000a	0.002070 (0.000580)	1,401	3,061	4,671
Cardiovascular (ICD codes 390-429), Age 65+	Samet et al., 2000a	0.001190 (0.000110)	4,487	5,464	6,436
Asthma (ICD codes 493), Age 64-	Sheppard et al., 1999	0.002568 (0.000767)	703	1,664	2,586
Asthma-related ER Visits, Age 64-	Schwartz et al., 1993	0.003670 (0.001260)	1,349	3,992	6,465
Minor Illness					
Acute Bronchitis, Age 8-12	Dockery et al., 1996	0.02720 (0.01710)	-17,452	50,335	80,421
URS, Age 9-11	Pope et al., 1991	0.00360 (0.0015)	135,810	730,815	1,308,545
LRS, Age 7-14	Schwartz et al., 1994	0.01823 (0.00586)	270,413	631,880	899,973
Asthma Attacks, All ages	Whittemore and Korn, 1980	0.00144 (0.000556)	146,184	592,736	1,027,020
Work Loss Days	Ostro, 1987	0.0046 (0.00036)	4,195,917	4,910,652	5,612,157
Minor Restricted Activity Days –adjusted***	Ostro & Rothschild, 1989	0.00741 (0.0007)	20,717,957	24,564,726	28,272,025

* Base period 1998-2000
**Mortality estimates for achieving 18 $\mu g/m^3$, other effects to 11 $\mu g/m^3$ (background).
*** To avoid double counting, the number of asthma attacks estimated were subtracted from the number of MRADs.

Table 9.8. County Annual PM2.5 Mortality Effects Reducing Ambient PM to Background Levels*

County	Concentration Change (Current minus 9 ug/m^3)	Long-term Exposure Mortality Krewski, 2000, 63 cities, Age 30+				Short-term Exposure Mortality Schwartz, 1996, All ages			
		Population (age 30+)	5th Percentile	Mean	95th Percentile	Population (all ages)	5th Percentile	Mean	95th Percentile
ALAMEDA	6.8	830,217	156	317	474	1,443,741	120	145	170
ALPINE	0	745	--	--	--	1,208	0	0	0
AMADOR	7.6	23,696	6	12	18	35,100	5	6	7
BUTTE	3.3	115,129	16	32	47	203,171	17	21	25
CALAVERAS	7.6	27,521	7	14	21	40,554	6	7	8
COLUSA	3.3	9,750	1	2	3	18,804	1	1	2
CONTRA COSTA	6.8	560,627	97	197	296	948,816	81	99	116
DEL NORTE	0	16,430	--	--	--	27,507	1	1	1
EL DORADO, Lake Tahoe Basin	0	20,358	--	--	--	32,795	1	1	1
EL DORADO, Mountain Counties Basin	7.6	76,670	42	85	128	123,603	12	15	17
FRESNO	13.3	398,493	155	311	463	799,407	104	126	147
GLENN	3.3	14,402	2	4	5	26,453	2	2	3
HUMBOLDT	0	73,435	--	--	--	126,518	3	4	5
IMPERIAL	4.1	73,048	7	15	23	142,361	7	9	10
INYO	0	11,785	--	--	--	17,945	1	1	1
KERN, Mojave Basin	1	57,133	2	3	5	112,480	4	5	6
KERN, San Joaquinn Valley Basin	13.3	278,942	110	220	328	549,165	75	91	106
KINGS	13.3	65,080	20	40	59	129,461	13	16	19
LAKE	0	38,073	--	--	--	58,309	0	0	0
LASSEN	0	19,716	--	--	--	33,828	0	0	0
LOS ANGELES, Mojave	1	152,395	4	9	13	285,580	10	12	14
LOS ANGELES, South Coast Basin	13.2	4,927,449	1,763	3,546	5,274	9,233,758	1,086	1,316	1,545
MADERA	13.3	66,083	25	50	74	123,109	17	21	25
MARIN	6.8	167,482	27	55	82	247,289	22	26	31
MARIPOSA	7.6	11,432	3	6	9	17,130	2	3	3
MENDOCINO	0	52,390	--	--	--	86,265	2	3	3
MERCED	13.3	102,065	39	79	117	210,554	27	32	38
MODOC	0	6,043	--	--	--	9,449	0	0	0
MONO	0	7,604	--	--	--	12,853	0	0	0
MONTEREY	0	211,980	--	--	--	401,762	7	8	10
NAPA	6.8	75,990	18	37	56	124,279	15	18	21
NEVADA	7.6	61,115	15	30	44	92,033	11	14	16
ORANGE	13.2	1,576,527	475	956	1,422	2,846,289	324	393	461
PLACER, Lake Tahoe Basin	0	6,033	--	--	--	9,936	0	0	0
PLACER, Sac Valley Basin	3.3	144,794	12	25	38	238,463	15	18	22
PLUMAS	7.6	14,018	4	7	11	20,824	3	3	4
RIVERSIDE, Mojave Basin	1	16,644	1	1	2	30,908	1	2	2
RIVERSIDE, Salton Sea Basin	4.1	166,438	22	44	66	309,077	23	27	32
RIVERSIDE, South Coast Basin	13.2	649,109	267	538	800	1,205,400	186	226	265

County	Concentration Change (Current minus 9 ug/m^3)	Long-term Exposure Mortality Krewski, 2000, 63 cities, Age 30+				Short-term Exposure Mortality Schwartz, 1996, All ages			
		Population (age 30+)	5th Percentile	Mean	95th Percentile	Population (all ages)	5th Percentile	Mean	95th Percentile
SACRAMENTO	3.3	680,201	65	132	199	1,223,499	75	91	107
SAN BENITO	0	27,492	--	--	--	53,234	1	1	1
SAN BERNARDINO, Mojave Basin	1	197,817	5	11	17	393,170	14	17	21
SAN BERNARDINO, South Coast Basin	13.2	662,256	236	475	706	1,316,264	165	200	234
SAN DIEGO	6.6	1,550,162	276	560	839	2,813,833	222	269	316
SAN FRANCISCO	6.8	503,126	100	203	304	776,733	78	94	111
SAN JOAQUIN	13.3	294,878	125	251	374	563,598	83	101	119
SAN LUIS OBISPO	2.8	145,609	13	26	38	246,681	16	19	23
SAN MATEO	6.8	432,917	74	150	225	707,161	58	71	83
SANTA BARBARA	2.8	218,917	18	37	56	399,347	22	26	31
SANTA CLARA	6.8	960,713	134	271	406	1,682,585	107	130	153
SANTA CRUZ	0	146,100	--	--	--	255,602	5	6	7
SHASTA	3.3	98,835	12	25	38	163,256	13	16	19
SIERRA	7.6	2,400	1	1	2	3,555	1	1	1
SISKIYOU	0	28,852	--	--	--	44,301	0	0	0
SOLANO, Sac Valley Basin	3.3	67,412	5	11	16	121,998	6	7	9
SOLANO, San Francisco Basin	6.8	150,047	24	49	73	271,544	20	24	28
SONOMA, North Coast Basin	0	33,209	--	--	--	55,034	1	2	2
SONOMA, San Francisco Basin	6.8	243,531	49	99	149	403,580	41	49	58
STANISLAUS	13.3	233,429	96	193	287	446,997	65	79	93
SUTTER	3.3	43,620	5	9	14	78,930	5	6	8
TEHAMA	3.3	33,278	4	9	14	56,039	5	6	7
TRINITY	0	8,872	--	--	--	13,022	0	0	1
TULARE	13.3	179,625	72	145	216	368,021	48	58	68
TUOLUMNE	7.6	36,235	9	18	27	54,501	7	8	10
VENTURA	2.8	419,350	28	57	86	753,197	35	42	50
YOLO	3.3	83,401	7	15	23	168,660	9	11	13
YUBA	3.3	31,142	4	8	12	60,219	4	5	6
Statewide Total		***18,640,255***	***4,659***	***9,391***	***13,999***	***33,870,743***	***3,312***	***4,014***	***4,714***

* Base period 1998-2000

Table 9.9. County Annual PM2.5 Chronic Illness Effects Reducing Ambient PM2.5 to Background Levels*

County	Concentration Change (Current minus 5 ug/m^3)	Chronic Bronchitis Abbey, 1995, Age 27+ Est. β (std. Error) 0.0132 (0.00680)			
		Population (age 27+)	5th Percentile	Mean	95th Percentile
ALAMEDA	10.80	902,538	-4	429	804
ALPINE	3.50	781	0	0	0
AMADOR	11.60	24,742	0	13	23
BUTTE	7.30	122,055	0	40	77
CALAVERAS	11.60	28,456	0	14	27
COLUSA	7.30	10,459	0	3	7
CONTRA COSTA	10.80	598,543	-3	285	533
DEL NORTE	2.50	17,621	0	2	4
EL DORADO, Lake Tahoe Basin	2.50	21,343	0	2	5
EL DORADO, Mountain Counties Basin	11.60	80,377	0	41	76
FRESNO	17.30	432,034	-3	316	569
GLENN	7.30	15,362	0	5	10
HUMBOLDT	2.50	78,240	0	9	18
IMPERIAL	8.10	79,320	0	29	55
INYO	3.50	12,232	0	2	4
KERN, Mojave Basin	5.00	61,888	0	14	28
KERN, San Joaquinn Valley Basin	17.30	302,161	-2	221	398
KINGS	17.30	72,019	-1	53	95
LAKE	-	39,676	0	0	0
LASSEN	-	21,551	0	0	0
LOS ANGELES, Mojave	5.00	166,631	0	38	74
LOS ANGELES, South Coast Basin	17.20	5,387,730	-42	3,915	7,062
MADERA	17.30	71,142	-1	52	94

County	Concentration Change (Current minus 5 ug/m^3)	Chronic Bronchitis Abbey, 1995, Age 27+ Est. β (std. Error) 0.0132 (0.00680)			
MARIN	10.80	177,086	-1	84	158
MARIPOSA	11.60	11,917	0	6	11
MENDOCINO	2.50	55,283	0	6	13
MERCED	17.30	110,558	-1	81	146
		Population (age 27+)	**5th Percentile**	**Mean**	**95th Percentile**
MODOC	-	6,307	0	0	0
MONO	3.50	8,184	0	1	3
MONTEREY	2.50	231,186	0	27	53
NAPA	10.80	80,659	0	38	72
NEVADA	11.60	63,523	0	32	60
ORANGE	17.20	1,716,424	-14	1,247	2,250
PLACER, Lake Tahoe Basin	2.50	6,384	0	1	1
PLACER, Sac Valley Basin	7.30	153,220	-1	50	97
PLUMAS	11.60	14,517	0	7	14
RIVERSIDE, Mojave Basin	5.00	17,869	0	4	8
RIVERSIDE, Salton Sea Basin	8.10	178,691	-1	65	124
RIVERSIDE, South Coast Basin	17.20	696,897	-5	506	913
SACRAMENTO	7.30	734,152	-2	241	462
SAN BENITO	2.50	29,827	0	3	7
SAN BERNARDINO, Mojave Basin	5.00	214,586	0	49	95
SAN BERNARDINO, South Coast Basin	17.20	718,396	-6	522	942
SAN DIEGO	10.60	1,683,170	-8	786	1,476
SAN FRANCISCO	10.80	557,251	-3	265	497
SAN JOAQUIN	17.30	317,540	-3	232	418
SAN LUIS OBISPO	6.80	154,062	0	47	91
SAN MATEO	10.80	466,554	-2	222	416

County	**Concentration Change (Current minus 5 ug/m^3)**	**Chronic Bronchitis** **Abbey, 1995, Age 27+** **Est. β (std. Error) 0.0132 (0.00680)**			
SANTA BARBARA	6.80	235,598	-1	72	139
SANTA CLARA	10.80	1,050,455	-5	499	936
SANTA CRUZ	2.50	157,118	0	18	36
SHASTA	7.30	103,888	0	34	65
SIERRA	11.60	2,494	0	1	2
SISKIYOU	-	29,957	0	0	0
SOLANO, Sac Valley Basin	7.30	72,607	0	24	46
SOLANO, San Francisco Basin	10.80	161,609	-1	77	144
		Population (age 27+)	**5th Percentile**	**Mean**	**95th Percentile**
SONOMA, North Coast Basin	2.50	35,309	0	4	8
SONOMA, San Francisco Basin	10.80	258,935	-1	123	231
STANISLAUS	17.30	251,693	-2	184	331
SUTTER	7.30	46,746	0	15	29
TEHAMA	7.30	35,101	0	12	22
TRINITY	2.50	9,180	0	1	2
TULARE	17.30	194,596	-2	142	256
TUOLUMNE	11.60	37,927	0	19	36
VENTURA	6.80	450,600	-1	138	266
YOLO	7.30	90,504	0	30	57
YUBA	7.30	33,514	0	11	21
Statewide Total		***20,208,974***	***-122***	***11,414***	***20,918***

* Base period 1998-2000

Table 9.10. County Annual PM2.5 Hospitalization Reducing Ambient PM2.5 to Background Levels*

County	Concentration Change (ug/m^3)	Population (age 65+)	COPD (ICD codes 490-492, 494-496) Samet et al., 2000, Age 65+ 0.002880 (0.001390)			Pneumonia (ICD codes 480-487) Samet et al., 2000, Age 65+ 0.002070 (0.000580)			Cardiovascular (ICD codes 390-429) Samet et al., 2000, Age 65+ 0.001190 (0.000110)			Asthma (ICD codes 493) Sheppard et al., 1999, Age 64- 0.002270 (0.000948)			
			5th %tile	Mean	95th %tile	5th %tile	Mean	95th %tile	5th %tile	Mean	95th %tile	Population (age 64-)	5th %tile	Mean	95th %tile
ALAMEDA	10.80	147,591	2	43	82	28	61	95	89	109	129	1,296,150	6	33	60
ALPINE	3.50	120	0	0	0	0	0	0	0	0	0	1,088	0	0	0
AMADOR	11.60	6,329	0	2	4	1	3	4	4	5	6	28,771	0	1	1
BUTTE	7.30	32,056	0	6	12	4	9	14	13	16	19	171,115	1	3	5
CALAVERAS	11.60	7,373	0	2	4	1	3	5	5	6	7	33,181	0	1	2
COLUSA	7.30	2,135	0	0	1	0	1	1	1	1	1	16,669	0	0	1
CONTRA COSTA	10.80	107,272	2	31	59	20	45	69	65	79	93	841,544	4	22	39
DEL NORTE	2.50	3,448	0	0	0	0	0	1	0	1	1	24,059	0	0	0
EL DORADO, Lake Tahoe Basin	2.50	4,057	0	0	1	0	0	1	1	1	1	28,738	0	0	0
EL DORADO, Mountain Counties Basin	11.60	15,277	0	5	9	3	7	11	10	12	14	108,227	1	3	5
FRESNO	17.30	79,209	2	36	69	24	52	80	77	93	110	720,198	5	29	52
GLENN	7.30	3,431	0	1	1	0	1	1	1	2	2	23,022	0	0	1
HUMBOLDT	2.50	15,776	0	1	2	1	2	2	2	3	3	110,742	0	1	1
IMPERIAL	8.10	14,305	0	3	6	2	4	7	7	8	9	128,056	0	2	4
INYO	3.50	3,429	0	0	1	0	0	1	1	1	1	14,516	0	0	0
KERN, Mojave Basin	5.00	10,549	0	1	3	1	2	3	3	4	4	101,930	0	1	2
KERN, San Joaquinn Valley Basin	17.30	51,505	1	24	45	16	34	52	50	61	72	497,661	4	20	36
KINGS	17.30	9,557	0	4	8	3	6	10	9	11	13	119,904	1	5	9
LAKE	--	11,359	--	--	--	0	0	0	0	0	0	46,950	0	0	0
LASSEN	--	3,054	--	--	--	0	0	0	0	0	0	30,774	0	0	0
LOS ANGELES, Mojave	5.00	27,800	0	4	7	2	5	8	8	10	11	257,780	1	3	6
LOS ANGELES, South Coast Basin	17.20	898,873	23	410	780	270	592	908	863	1,053	1,241	8,334,885	63	337	603

County	Concentration Change (ug/m^3)	Population (age 65+)	COPD (ICD codes 490-492, 494-496) Samet et al., 2000, Age 65+ 0.002880 (0.001390)			Pneumonia (ICD codes 480-487) Samet et al., 2000, Age 65+ 0.002070 (0.000580)			Cardiovascular (ICD codes 390-429) Samet et al., 2000, Age 65+ 0.001190 (0.000110)			Asthma (ICD codes 493) Sheppard et al., 1999, Age 64- 0.002270 (0.000948)			
			5th %tile	Mean	95th %tile	5th %tile	Mean	95th %tile	5th %tile	Mean	95th %tile	Population (age 64-)	5th %tile	Mean	95th %tile
MADERA	17.30	13,596	0	6	12	4	9	14	13	16	19	109,513	1	4	8
MARIN	10.80	33,432	1	10	19	6	14	21	20	25	29	213,857	1	5	10
MARIPOSA	11.60	2,940	0	1	2	1	1	2	2	2	3	14,190	0	0	1
MENDOCINO	2.50	11,709	0	1	2	1	1	2	2	2	2	74,556	0	0	1
MERCED	17.30	20,004	1	9	17	6	13	20	19	24	28	190,550	1	8	14
MODOC	--	1,663	--	--	--	0	0	0	0	0	0	7,786	0	0	0
MONO	3.50	976	0	0	0	0	0	0	0	0	0	11,877	0	0	0
MONTEREY	2.50	40,299	0	3	5	2	4	6	6	7	8	361,463	0	2	4
NAPA	10.80	19,086	0	6	11	4	8	12	12	14	17	105,193	0	3	5
NEVADA	11.60	16,049	0	5	10	3	7	11	10	13	15	75,984	0	2	4
ORANGE	17.20	280,763	7	128	244	84	185	284	270	329	388	2,565,526	19	104	186
PLACER, Lake Tahoe Basin	2.50	1,302	0	0	0	0	0	0	0	0	0	8,634	0	0	0
PLACER, Sac Valley Basin	7.30	31,258	0	6	12	4	9	14	13	16	18	207,205	1	4	7
PLUMAS	11.60	3,725	0	1	2	1	2	3	2	3	3	17,099	0	0	1
RIVERSIDE, Mojave Basin	5.00	3,919	0	1	1	0	1	1	1	1	2	26,988	0	0	1
RIVERSIDE, Salton Sea Basin	8.10	39,192	0	9	16	6	12	19	18	22	26	269,885	1	5	9
RIVERSIDE, South Coast Basin	17.20	152,850	4	70	133	46	101	154	147	179	211	1,052,550	8	43	76
SACRAMENTO	7.30	135,875	1	27	51	17	38	59	56	68	80	1,087,624	3	19	34
SAN BENITO	2.50	4,315	0	0	1	0	0	1	1	1	1	48,919	0	0	1
SAN BERNARDINO, Mojave Basin	5.00	33,686	0	5	9	3	7	10	9	12	14	359,484	1	4	8
SAN BERNARDINO, South Coast Basin	17.20	112,773	3	51	98	34	74	114	108	132	156	1,203,491	9	49	87
SAN DIEGO	10.60	313,750	5	89	171	58	128	197	186	227	268	2,500,083	12	63	113
SAN FRANCISCO	10.80	106,111	2	31	59	20	44	68	64	78	92	670,622	3	17	31
SAN JOAQUIN	17.30	59,799	2	27	52	18	40	61	58	70	83	503,799	4	21	37
SAN LUIS OBISPO	6.80	35,685	0	7	13	4	9	14	14	17	20	210,996	1	3	6

County	Concentration Change (ug/m^3)	Population (age 65+)	COPD (ICD codes 490-492, 494-496) Samet et al., 2000, Age 65+ 0.002880 (0.001390)			Pneumonia (ICD codes 480-487) Samet et al., 2000, Age 65+ 0.002070 (0.000580)			Cardiovascular (ICD codes 390-429) Samet et al., 2000, Age 65+ 0.001190 (0.000110)			Asthma (ICD codes 493) Sheppard et al., 1999, Age 64- 0.002270 (0.000948)			
			5th %tile	Mean	95th %tile	5th %tile	Mean	95th %tile	5th %tile	Mean	95th %tile	Population (age 64-)	5th %tile	Mean	95th %tile
SAN MATEO	10.80	88,085	1	25	49	17	37	56	53	65	77	619,076	3	16	29
SANTA BARBARA	6.80	50,765	1	9	18	6	13	21	19	24	28	348,582	1	6	10
SANTA CLARA	10.80	160,527	3	46	89	30	67	103	97	118	140	1,522,058	7	39	70
SANTA CRUZ	2.50	25,487	0	2	3	1	2	4	4	4	5	230,115	0	1	3
SHASTA	7.30	24,861	0	5	9	3	7	11	10	12	15	138,395	0	2	4
SIERRA	11.60	629	0	0	0	0	0	0	0	0	1	2,926	0	0	0
SISKIYOU	--	8,040	--	--	--	0	0	0	0	0	0	36,261	0	0	0
SOLANO, Sac Valley Basin	7.30	11,292	0	2	4	1	3	5	5	6	7	110,706	0	2	3
SOLANO, San Francisco Basin	10.80	25,134	0	7	14	5	10	16	15	19	22	246,410	1	6	11
SONOMA, North Coast Basin	2.50	6,957	0	0	1	0	1	1	1	1	1	48,076	0	0	1
SONOMA, San Francisco Basin	10.80	51,020	1	15	28	10	21	33	31	38	44	352,561	2	9	16
STANISLAUS	17.30	46,697	1	21	41	14	31	47	45	55	65	400,300	3	16	29
SUTTER	7.30	9,755	0	2	4	1	3	4	4	5	6	69,175	0	1	2
TEHAMA	7.30	8,923	0	2	3	1	3	4	4	4	5	47,116	0	1	1
TRINITY	2.50	2,241	0	0	0	0	0	0	0	0	0	10,781	0	0	0
TULARE	17.30	35,917	1	16	31	11	24	36	35	42	50	332,104	3	14	24
TUOLUMNE	11.60	10,067	0	3	6	2	4	7	7	8	9	44,434	0	1	2
VENTURA	6.80	76,804	1	14	27	9	20	31	29	36	42	676,393	2	11	20
YOLO	7.30	15,782	0	3	6	2	4	7	6	8	9	152,878	0	3	5
YUBA	7.30	6,410	0	1	2	1	2	3	3	3	4	53,809	0	1	2
Statewide Total		***3,594,655***	***68***	***1,242***	***2,369***	***814***	***1,791***	***2,751***	***2,608***	***3,180***	***3,750***	***30,275,990***	***176***	***950***	***1,702***

* Base period 1998-2000

9.4 References

Abbey, D.E., B.E. Ostro, F. Petersen, and R.J. Burchette. 1995. “Chronic Respiratory Symptoms Associated with Estimated Long-Term Ambient Concentrations of Fine Particulates Less Than 2.5 Microns in Aerodynamic Diameter (PM2.5) and Other Air Pollutants.” *Journal of Exposure Analysis and Environmental Epidemiology* 5(2):137-159.

Abbey, D.E., F. Petersen, P.K. Mills, and W.L. Beeson. 1993. “Long-Term Ambient Concentrations of Total Suspended Particulates, Ozone, and Sulfur Dioxide and Respiratory Symptoms in a Nonsmoking Population.” *Archives of Environmental Health* 48(1):33-46.

Abt Associates Inc. 1996a. An Analysis of the Monetized Benefits Associated with National Attainment of Alternative Particulate Matter Standards in the Year 2007. Prepared for U.S. EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC. July 5.

Abt Associates Inc. 1996b. A Particulate Matter Risk Assessment for Philadelphia and Los Angeles. Prepared for U.S. EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC. July 3.

Air Resources Board. Guidance for using air quality-related indicators in reporting progress in attaining the State ambient air quality standards, Sacramento; 1993.

California Department of Health Services Office of Health Information and Research "Death Statistical Master Files", 1990

California Office of Statewide Health Planning and Development "Hospital Discharge Dataset", 1998 and 1999.

California State Department of Finance Demographic Research Unit "County Population Projections with Age, Sex and Race/Ethnic Detail" 1990-2040 in 10-year Increments, December, 1998

Centers for Disease Control and Prevention/National Center for Health Statistics. 1998. “Current Estimates from the National Health Interview Survey, 1995.” Series 10, No. 199.

Chen C, Chock DP, Winkler SL (1999). A simulation study of confounding in generalized linear models for air pollution epidemiology . Environ Health Perspect 107(3):217-22.

Chestnut et al. 2001. Evidence of PM Health Effects in California. Prepared for California Air Resources Board. July 31, 2001.

Dockery, D.W., J. Cunningham, A.I. Damokosh, L.M. Neas, J.D. Spengler, P. Koutrakis, J.H. Ware, M. Raizenne, and F. E. Speizer. 1996. “Health Effects of Acid Aerosols on North American Children: Respiratory Symptoms.” *Environmental Health Perspectives* 104(5):500-505.

Dockery DW, Pope CA III, Xu X, Spengler JD, Ware JH, Fay ME *et al*. (1993). An association between air pollution and mortality in six U.S. cities. N Engl J Med 329:1753-9.

Fairley, D. 1999. “Daily Mortality and Air Pollution in Santa Clara County, California: 1989-1996.” *Environmental Health Perspectives* 107(8):637-641.

Kinney, P.L. and H. Ozkaynak. 1991. “Associations of Daily Mortality and Air Pollution in Los Angeles County.” *Environmental Research* 54:99-120.

Kinney, P.L., K. Ito, and G.D. Thurston. 1995. “A Sensitivity Analysis of Mortality/PM10 Associations in Los Angeles.” *Inhalation Toxicology* 7:59-69.

Krewski, D., R.T. Burnett, M.S. Goldberg, K. Hoover, J. Siemiatycki, M. Jerrett, M. Abrahamonwicz, and W.H. White. 2000. *Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality.* Special Report. Health Effects Institute, Cambridge MA.

Kunzli N, Kaiser R, Medina S, Studnicka M, *et al.* (2000). Public health impact of outdoor and traffic-related air pollution: a European assessment. Lancet 356(9232):795-801.

National Heart, Lung, and Blood Institute. 1997. Guidelines for the Diagnosis and Management of Asthma: Expert Panel Report 2. National Institutes of Health. Bethesda, MD. NIH Publication No. 97-4051. July.

Ostro, B.D. 1987. "Air Pollution and Morbidity Revisited: A Specification Test." *Journal of Environmental Economics and Management* 14:87-98.

Ostro, B.D. 1995. "Fine Particulate Air Pollution and Mortality in Two Southern California Counties." *Environmental Research* 70:98-104.

Ostro BD, Chestnut L.. (1998). Assessing the health benefits of reducing particulate matter air pollution in the United States. Environ Res 76(2):94-106.

Ostro, B.D. and S. Rothschild. 1989. "Air Pollution and Acute Respiratory Morbidity: An Observational Study of Multiple Pollutants." *Environmental Research* 50:238-247.

Ostro, B.D., S. Hurley, and M.J. Lipsett. 1999. "Air Pollution and Daily Mortality in the Coachella Valley, California: A Study of PM10 Dominated by Coarse Particles." *Environmental Research* A81:231-238.

Pope III, C.A., R. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito, G.D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-Term Exposure to Fine Particulate Air Pollution." *Journal of the American Medical Association* 287:1123-1141.

Pope III, C.A., D.W. Dockery, J.D. Spengler, and M.E. Raizenne. 1991. "Respiratory Health and PM10 Pollution: A Daily Time Series Analysis." *American Review of Respiratory Diseases* 144:668-874.

Pope III, C.A., M.J. Thun, M.M. Namboodiri, D.W. Dockery, J.S. Evans, F.E. Speizer, and C.W. Heath Jr. 1995. "Particulate Air Pollution As a Predictor of Mortality in a Prospective Study of U.S. Adults." *American Journal of Respiratory and Critical Care Medicine* 151:669-674.

Rossi, G., M.A. Vigotti, A. Zanobetti, F. Repetto, V. Gianelle and J. Schwartz. 1999. Air pollution and cause-specific mortality in Milan, Italy, 1980-1989. *Arch Environ Health*. Vol. 54(3): 158-64.

Samet, J. M., S. L. Zeger, F. Dominici, F. Curriero, I. Coursac, D. W. Dockery, J. Schwartz, and A. Zanobetti. 2000a. *The National Morbidity, Mortality, and Air Pollution Study Part II: Morbidity, Mortality, and Air Pollution in the United States*. Health Effects Institute Research Report 94, Part II. June. 83 pp.

Samet, J.M., F. Dominici, F.C. Curriero, I. Coursac, and S.L. Zeger. 2000b. "Fine Particulate Air Pollution and Mortality in 20 U.S. Cities, 1987-1994." *The New England Journal of Medicine* 343(24):1742-1749.

Schwartz J (1994a). Air pollution and daily mortality: a review and meta analysis. Environ Res 64:36-52.

Schwartz J (1994b). What are people dying of on high air pollution days? Environ Res 64:26-35.

Schwartz J (1994c). PM10, ozone, and hospital admissions for the elderly in Minneapolis-St. Paul, Minnesota. Arch Environ Health 49(5):366-74.

Schwartz, J., D.W. Dockery, L.M. Neas, D. Wypij, J.H. Ware, J.D. Spengler, P. Koutrakis, F.E. Speizer, and B.G. Ferris Jr. 1994. "Acute Effects of Summer Air Pollution on Respiratory Symptom Reporting in Children." *American Journal of Respiratory and Critical Care Medicine* 150:1234-1242.

Schwartz, J., D. Slater, T.V. Larson, W.E. Pierson, and J.Z. Koenig. 1993. "Particulate Air Pollution and Hospital Emergency Room Visits for Asthma in Seattle." *American Review of Respiratory Diseases* 147:826-831.

Schwartz, J., D.W. Dockery and L.M. Neas. 1996. Is Daily Mortality Associated Specifically With Fine Particles. *Journal of the Air & Waste Management Association.* Vol. 46(10): 927-939.

Sheppard, L., D. Levy, G. Norris, T.V. Larson, and J.Q. Koenig. 1999. "Effects of Ambient Air Pollution on Nonelderly Asthma Hospitalizations in Seattle, Washington, 1987-1994." *Epidemiology* 10(1):23-30.

Smith D.H., D.C. Malone, K.A. Lawson, L.G. Okamoto, C. Battista, and W.B. Saunders. A national estimate of the economic costs of asthma. *American Journal of Respiratory and Critical Care Medicine* 1997;156:787-793.

U.S. Environmental Protection Agency. *Regulatory Impact Analysis: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements;* December, 2000. EPA-420-R-00-026. Office of Air and Radiation, Research Triangle Park, NC.

U.S. Environmental Protection Agency (1996). Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information: OAQPS staff paper. Office of Air Quality Planning and Standards, Research Triangle Park, NC, EPA-452\R-96-013.

U.S. Environmental Protection Agency (1999). The benefits and costs of the Clean Air Act 1990 to 2010. Office of Air and Radiation and Office of Policy, Washington DC, EPA-410-R-99-001.

Whittemore, A. and E. Korn. 1980. "Asthma and Air Pollution in the Los Angeles Area." *American Journal of Public Health* 70(7):687-696.

Appendix 1

Proposed Amendments to California Code of Regulations

PROPOSED AMENDMENTS TO TITLE 17, CALIFORNIA CODE OF REGULATIONS

Section 70100. Definitions.

Note: No changes to (a), (b), (c), (d), (e), (f), (g), (h), (i).

(j) Suspended Particulate Matter (PM_{10}10). Suspended particulate matter (PM_{10}10) refers to atmospheric particles, solid and liquid, except uncombined water as measured by a (PM_{10}10) sampler which collects 50 percent of all particles of 10 µm aerodynamic diameter and which collects a declining fraction of particles as their diameter increases and an increasing fraction of particles as their diameter decreases, reflecting the characteristics of lung deposition. Suspended particulate matter (PM_{10}10) is to be measured by ~~the size selective inlet high volume (SSI) PM_{10} sampler method in accordance with ARB Method P, as adopted in August 22, 1985, or by an equivalent (PM_{10}) sampler method~~ a California Approved Sampler (CAS) for PM10, for purposes of monitoring for compliance with the Suspended Particulate Matter (PM_{10}10) standards. Approved samplers, methods, and instruments are listed in Section 70100.1(a) below. A CAS for PM10 includes samplers, methods, or instruments determined by the Air Resources Board or the Executive Officer to produce equivalent results for PM10 with the Federal Reference Method (40 CFR, part 50, Appendix M, as published in 62 Fed. Reg., 38763, July 18, 1997).

(k) Fine ~~Total~~ Suspended Particulate Matter (PM2.5). Fine ~~Total~~ suspended particulate matter (PM2.5) refers to suspended atmospheric particles ~~of any size,~~ solid and liquid, except uncombined water as measured by a PM2.5 sampler which collects 50 percent of all particles of 2.5 µm aerodynamic diameter and which collects a declining fraction of particles as their diameter increases and an increasing fraction of particles as their diameter decreases, reflecting the characteristics of lung deposition. Fine ~~Total~~ suspended particulate matter (PM2.5) is to be measured by ~~the high volume sampler method or by an equivalent method~~ a California Approved Sampler (CAS) for PM2.5 for purposes of monitoring for compliance with the Fine Particulate Matter (PM2.5) standards. Approved samplers, methods, and instruments are listed in Section 70100.1(b) below. A CAS for PM2.5 includes samplers, method, and instruments determined by the Air Resources Board or the Executive Officer to produce equivalent results for PM2.5 with the Federal Reference Method (40 CFR, part 50, Appendix L, as published in 62 Fed. Reg., 38763, July 18, 1997).

Note: No changes to (l), (m), (n), (o).

(p) Sulfates. Sulfates are the water soluble fraction of suspended particulate matter (PM10) containing the sulfate ~~radical (SO_4)~~ ion (SO_4^{2-}) including but not limited to strong acids and sulfate salts, as measured by ~~AIHL Method No. 61 (Turbidimetric Barium Sulfate) (December 1974, as revised April 1975 and February 1976) or equivalent method~~ MLD Method 007 (based on high-volume size-selective inlet (SSI) sampling and ion chromatography), dated April 22, 2002.

Note: No changes to (q), (r), (t).

NOTE: Authority cited: Sections 39600, ~~and~~ 39601 and 39606, Health and Safety Code. Reference: Sections 39602 and 39606~~(b)~~, Health and Safety Code.

Section 70100.1. Methods, Samplers, and Instruments for Measuring Pollutants

(a) PM10 Methods. The following samplers, methods, and instruments are California Approved Samplers for PM10 for the purposes of monitoring for compliance with the Suspended Particulate Matter (PM10) standards:

(1) Federal Reference Method for the Determination of Particulate Matter as PM10 in the Atmosphere (40 CFR, Chapter 1, part 50, Appendix M, as published in 62 Fed. Reg., 38753, July 18, 1997). The specific samplers approved are:

(A) Andersen Model RAAS10-100 PM10 Single Channel PM10 Sampler, U.S. EPA Manual Reference Method RFPS-0699-130, as published in 64 Fed. Reg., 33481, June 23, 1999.

(B) Andersen Model RAAS10-200 PM10 Single Channel PM10 Audit Sampler, U.S. EPA Manual Reference Method RFPS-0699-131, as published in 64 Fed. Reg., 33481, June 23, 1999.

(C) Andersen Model RAAS10-300 PM10 Multi Channel PM10 Sampler, U.S. EPA Manual Reference Method RFPS-0669-132, as published in 64 Fed. Reg., 33481, June 23, 1999.

(D) Graesby Andersen/GMW Model 1200 High-Volume Air Sampler, U.S. EPA Manual Reference Method RFPS-1287-063, as published in 52 Fed. Reg., 45684, December 1, 1987 and in 53 Fed. Reg., 1062, January 15, 1988.

(E) Graesby Andersen/GMW Model 321B High-Volume Air Sampler, U.S. EPA Manual Reference Method RFPS-1287-064, as published in 52 Fed. Reg., 45684, December 1, 1987 and in 53 Fed. Reg., 1062, January 15, 1988.

(F) Graesby Andersen/GMW Model 321-C High-Volume Air Sampler, U.S. EPA Manual Reference Method RFPS-1287-065, as published in 52 Fed. Reg., 45684, December 1, 1987 and in 53 Fed. Reg., 1062, January 15, 1988.

(G) BGI Incorporated Model PQ100 Air Sampler, U.S. EPA Manual Reference Method RFPS-1298-124, as published in 63 Fed. Reg., 69624, December 17, 1998.

(H) BGI Incorporated Model PQ200 Air Sampler, U.S. EPA Manual Reference Method RFPS-1298-125, as published in 63 Fed. Reg., 69624, December 17, 1998.

(2) Continuous samplers:

(A) Andersen Beta Attenuation Monitor Model FH 62 C14 equipped with the following components: louvered PM10 inlet, volumetric flow controller, automatic filter change mechanism, automatic zero check, and calibration control foils kit*.

(B) Met One Beta Attenuation Monitor Model 1020 equipped the following components: louvered PM10 size selective inlet, volumetric flow controller, automatic filter change mechanism, automatic heating system, automatic zero and span check capability*.

(C) Rupprecht & Patashnick Series 8500 Filter Dynamics Measurement System equipped with the following components: louvered PM10 size selective inlet, volumetric flow control, flow splitter (3 liter/min sample flow), sample equilibration system (SES) dryer, TEOM sensor unit, TEOM control unit,

switching valve, purge filter conditioning unit, and palliflex TX40, 13 mm effective diameter cartridge*.

(b) PM2.5 Methods. The following samplers, methods, and instruments are California Approved Samplers for PM2.5 for the purposes of monitoring for compliance with the Fine Particulate Matter (PM2.5) standards:

(1) Federal Reference Method for the Determination of Particulate Matter as PM2.5 in the Atmosphere, 40 CFR, part 50, Appendix L, as published in 62 Fed. Reg., 38763, July 18, 1997 and as amended in 64 Fed. Reg., 19717, April 22, 1999. These must use either the WINS impactor or the U.S. EPA-approved very sharp cut cyclone (67 Fed. Reg., 15566, April 2, 2002) to separate PM2.5 from PM10. The specific samplers approved are:

(A) Andersen Model RAAS 2.5-200 PM2.5 Ambient Audit Air Sampler, U.S. EPA Manual Reference Method RFPS-0299-128, as published in 64 Fed. Reg., 12167, March 11, 1999.

(B) Graesby Andersen Model RAAS 2.5-100 PM2.5 Ambient Air Sampler, U.S. EPA Manual Reference Method RFPS-0598-119, as published in 63 Fed. Reg., 31991, June 11, 1998.

(C) Graesby Andersen Model RAAS 2.5-300 PM2.5 Sequential Ambient Air Sampler, U.S. EPA Manual Reference Method RFPS-0598-120, as published in 63 Fed. Reg., 31991, June 11, 1998.

(D) BGI Inc. Models PQ200 and PQ200A PM2.5 Ambient Fine Particle Sampler, U.S. EPA Manual Reference Method RFPS-0498-116, as published in 63 Fed. Reg., 18911, April 16, 1998.

(E) Rupprecht & Patashnick Partisol-FRM Model 2000 Air Sampler, U.S. EPA Manual Reference Method RFPS-0498-117, as published in 63 Fed. Reg., 18911, April 16, 1998.

(F) Rupprecht & Patashnick Partisol Model 2000 PM-2.5 Audit Sampler, as described in U.S. EPA Manual Reference Method RFPS-0499-129, as published in 64 Fed. Reg., 19153, April 19, 1999.

(G) Rupprecht & Patashnick Partisol-Plus Model 2025 Sequential Air Sampler, U.S. EPA Manual Reference Method RFPS-0498-118, as published in 63 Fed. Reg., 18911, April 16, 1998.

(H) Thermo Environmental Instruments, Incorporated Model 605 “CAPS” Sampler, U.S. EPA Manual Reference Method RFPS-1098-123, as published in 63 Fed. Reg., 58036, October 29, 1998.

(I) URG-MASS100 Single PM2.5 FRM Sampler, U.S. EPA Manual Reference Method RFPS-0400-135, as published in 65 Fed. Reg., 26603, May 8, 2000.

(J) URG-MASS300 Sequential PM2.5 FRM Sampler, U.S. EPA Manual Reference Method RFPS-0400-136, as published in 65 Fed. Reg., 26603, May 8, 2000.

(2) Continuous samplers:

(A) Andersen Beta Attenuation Monitor Model FH 62 C14 equipped with the following components: louvered PM10 size selective inlet, very sharp cut or sharp cut cyclone, volumetric flow controller, automatic filter change mechanism, automatic zero check, and calibration control foils kit*.

(B) Met One Beta Attenuation Monitor Model 1020 equipped the following components: louvered PM10 size selective inlet, very sharp cut or sharp cut cyclone, volumetric flow controller, automatic filter change mechanism, automatic heating system, and automatic zero and span check capability*.

(C) Rupprecht & Patashnick Series 8500 Filter Dynamics Measurement System equipped with the following components: louvered PM10 size selective inlet, very sharp cut or sharp cut cyclone, volumetric flow control, flow splitter (3 liter/min sample flow), sample equilibration system (SES) dryer, TEOM sensor unit, TEOM control unit, switching valve, purge filter conditioning unit, and palliflex TX40, 13 mm effective diameter cartridge*.

*Instrument shall be operated in accordance with the vendor's instrument operation manual that adheres to the principles and practices of quality control and quality assurance as specified in Volume I of the "Air Monitoring Quality Assurance Manual", as printed on April 17, 2002, and available from the California Air Resources Board, Monitoring and Laboratory Division, P.O. Box 2815, Sacramento CA 95814, incorporated by reference herein.

Note: Authority cited: Sections 39600, 39601 and 39606, Health and Safety Code.
Reference: Sections 39014, 39606, 39701, 39703(f) and 57004, Health and Safety Code; Western Oil and Gas Ass'n v. Air Resources Bd. (1984) 37 Cal.3d 502.

Section 70200. Table of Standards ***

[Note: no changes are proposed to standards for any substances not listed]

Substance	Concentration and Methods*	Duration of Averaging Periods	Most Relevant Effects	Comments
Suspended Particulate Matter (PM_{10}10)	50µg/m^3 PM_{10}10** ~~30µg/m^3 PM_{10}**~~ 20µg/m^3 PM10** ~~SSI Method in accordance with Method P~~ California Approved Sampler as listed in section 70100.1(a)	24 hour sample 24 hour samples, annual ~~geometric~~ arithmetic mean	Prevention of excess deaths, illness and restrictions in activity from short- and long-term exposures. Illness outcomes include, but are not limited to, respiratory symptoms, bronchitis, asthma exacerbation, emergency room visits and hospital admissions for cardiac and respiratory diseases. Sensitive subpopulations include children, the elderly, and individuals with pre-existing cardiopulmonary ~~from short-term exposures and of exacerbation of symptoms in sensitive patients with respiratory~~ disease. ~~Prevention of excess seasonal declines in pulmonary function, especially in children.~~	This standard applies to suspended matter as measured by PM_{10}10 sampler, which collects 50% of all particles of 10µm aerodynamic diameter and collects a declining fraction of particles as their diameter increases, reflecting the characteristics of lung deposition.
Fine Suspended Particulate Matter (PM2.5)	25µg/m^3 PM2.5** 12µg/m^3 PM2.5** California Approved Sampler as listed in section 70100.1(b)	24 hour sample 24 hour samples, annual arithmetic mean	Prevention of excess deaths and illness from short- and long-term exposures. Illness outcomes include, but are not limited to, respiratory symptoms, asthma exacerbation, and hospital admissions for cardiac and respiratory diseases. Sensitive subpopulations include children, the elderly, and individuals with pre-existing cardiopulmonary disease.	This standard applies to fine suspended matter as measured by PM2.5 sampler, which collects 50% of all particles of 2.5µm aerodynamic diameter and collects a declining fraction of particles as their diameter increases, reflecting the characteristics of lung deposition.
Sulfates	25µg/m^3 total sulfates, ~~AIHL #61 (Turbimetric Barium Sulfate)~~ MLD Method 007	24 hours	a. Decrease in ventilatory function b. Aggravation of asthmatic symptoms c. Aggravation of cardio-pulmonary disease d. Vegetation damage e. Degradation of visibility f. Property damage	This standard is based on a Critical Harm Level, not a threshold value.

* Any equivalent procedure which can be shown to the satisfaction of the Air Resources Board to give equivalent results at or near the level of the air quality standard may be used.

** These standards are violated when concentrations exceed those set forth in the body of the regulation. All other standards are violated when concentrations equal or exceed those set forth in the body of the regulation.

*** Applicable statewide unless otherwise noted.

**** These standards are violated when particle concentrations cause measured light extinction values to exceed those set forth in the regulations.

Note: Authority cited: Sections 39600, 39601(a) and 39606~~(b)~~, Health and Safety Code.
Reference: Sections 39014, 39606~~(b)~~, 39701 and 39703(f), Health and Safety Code.

Appendix 2

Findings of the Air Quality Advisory Committee

UCI
University of California, Irvine

4/29/2002

Dr. Alan C. Lloyd, Chair
California Air Resources Board
1001 I Street
Sacramento, CA 95812

Sacramento, CA

Dear Dr. Lloyd:

The Air Quality Advisory Committee met on January 23 and 24, 2002 to evaluate the draft document "Review of the California Ambient Air Quality Standards for Particulate Matter and Sulfates: Report to the Air Quality Advisory Committee." The examination of the current air quality standards and the recommendations for modification of those standards derived from the Children's Environmental Health Protection Act (Senate Bill 25) and a resulting document "Adequacy of California Ambient Air Quality Standards: Children's Environmental Health Protection Act" which was published as a staff report in 2000. SB 25 prompted an analysis of the scientific basis of the California air quality standards for particulate matter, sulfates, ozone, carbon monoxide, nitrogen dioxide, lead, and sulfur dioxide.

In response to SB 25, an up to date examination of the scientific information relevant to each of these standards that was published in peer reviewed documents was commissioned to determine if the current California standards were adequately protective of children's health. The staff of the Office of Environmental Health Hazard Assessment (OEHHA) made an analysis of the findings and recommended a list of standards that required re-review. The OEHHA analysis was deliberated by AQAC in a public meeting and the list of standards to be reviewed was prioritized. The standards for particulate matter and sulfate were deemed to be those with the highest priority for modification to protect the health of California's children.

In most respects, the committee was pleased with the document "Review of the California Ambient Air Quality Standards for Particulate Matter and Sulfates: Report to the Air Quality Advisory Committee." The committee went on record to complement the staffs of the ARB and OEHHA for performing a very comprehensive and careful compilation and analysis of the peer reviewed literature on sources, monitoring and health effects of ambient particulate matter. There were, however, some areas in which the AQAC required additional clarification and one key issue with which the AQAC disagreed with the OEHHA/ARB recommendations.

The draft document made the following recommendations that were endorsed by the AQAC.

Department of Community and Environmental Medicine
College of Medicine • 100 Faculty Research Facility • Irvine, CA 92697-1825
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- PM10 Annual-average standard – Lower the standard from the current 30 $\mu g/m^3$ to 20 $\mu g/m^3$ and revise the averaging method to an annual arithmetic mean.
- PM10 24-hour-average standard – Retain the current standard at 50 $\mu g/m^3$.
- PM2.5 Annual-average standard – Establish a new annual arithmetic mean standard at 12 $\mu g/m^3$.
- Sulfate 24-hour-average standard – Retain the current 25 $\mu g/m^3$ standard.
- For all of the PM standards, the concentrations noted above are to be established as "not to be exceeded."

The AQAC, however, did not agree with the assessment in the draft document that there was not sufficient scientific basis for establishing a 24-hour average PM2.5 standard. The AQAC requested the OEHHA and ARB staff to develop acceptable methodology for establishing a 24-hour PM2.5 standard and determined that the level and form of that standard.

The resulting recommendation was made.

- PM2.5 24-hour-average standard – Establish a new standard of 25 $\mu g/m^3$, not to be exceeded.

The AQAC met on April 3, 2002 and unanimously endorsed this recommendation and the statistical form of the standard that was proposed.

The specific comments of the AQAC on the draft document are appended to this letter.

The AQAC is extremely appreciative of the responsiveness and expertise of the the staffs of OEHHA and the ARB. We commend them on the excellent job they did in reviewing and summarizing the scientific literature in the complex area of particulate matter and in establishing a set of ambient air quality standards that will protect the health of California's citizens and especially their children.

Sincerely,

Michael T. Kleinman, Chair
Air Quality Advisory Committee

Cc: Bart Croes, Research Division
Richard Bode, Research Division

Summary Comments of the Air Quality Advisory Committee

The staffs of OEHHA and the ARB provided an excellent review of the current literature relevant to the sources, transport and health effects of ambient PM. The review provided a firm basis for establishing the needs for PM air quality standards and the committee was unanimous in its appreciation of the effort and diligence involved in producing the report.

The Air Quality Advisory Committee (AQAC) provided comments on a chapter by chapter basis and also addressed specific overarching questions that were submitted to them during their review of the report.

Children's protection, with an adequate margin of safety, is of paramount importance to public health. While the measurable injury and morbidity may be small, the degree to which PM exposures early in life contribute to lung compromise later in life (i.e. effects may be cumulative) has not been adequately researched. In addition, children with chronic lung diseases such as bronchopulmonry displasia, asthma and cystic fibrosis may be at special risk but, with the possible exception of asthma, there has been little research effort in these areas. Since asthma affects nearly 10% of the child population, the effects of PM on this group is of special importance. Although commented on in the draft document, it is important to recognize that children have higher minute ventilation rates per unit lung volume than do adults, hence their lungs receive greater doses of inhaled particles than do adults for comparable exposures.

The potential effects on children and the substantial evidence for short-term mortality and morbidity effects of PM in adults led this committee strongly identify that the major lacking of the report was the failure to set a 24-hour PM2.5 standard. The arguments for not having such a standard were judged to be weak. The specific justifications for considering that the justification was weak was addressed more fully, as per the specific comments below, and the comments were made available to the staffs of OEHHA and the ARB. The draft report had a very strong focus on mortality and certain chronic endpoints. Sufficient weight was not given to the large numbers of studies that provide data on short-term effects, including morbidity, that could have been considered as part of the basis for the 24 hour PM2.5 standard. The committee recommended that *a priori* criteria be established to guide decisions about the appropriate level and that a 24 hour PM2.5 standard be set.

Specific Comments on the Draft Report:

1. Executive Summary

Page 2, line 13-4, *"there are fewer studies.."* This statement is false and needs to be corrected.

2. Introduction

Regulations require that standards be reviewed when 'substantial new information becomes available' or at least once every 5 years. The committee suggests that some specific triggers for re-review might be new information on effects in susceptible

populations that might indicate erosion of margins of safety, or information bearing on the need for additional standards, e.g. a coarse particle standard (PM2.5-10).[1]

There are also data that suggest that ultrafine particles may be a size fraction that plays an important role in health effects. There are also metrics, other than mass of particles in a given size fraction, that might be better predictors of effects on health, including:

- Aerosol Acidity
- Aerosol Oxidant (peroxides, radicals)
- Ames Test Activity
- Polar and non-polar PAH
- Ultrafine Component ($1nm \leq d_p \leq 0.1\ \mu m$) [2]

An integrative approach to standard setting should be developed. Such an approach would improve ability to identify possible interactions between pollutants that might impact on the level set for a particulate standard. Such an approach might make it easier to recognize whether there are un-needed redundancies in standards. For example, it might be determined that a separate sulfate standard is not needed in the future. The chapter should be expanded to delineate future possibilities and triggers.

3. Physics and Chemistry of Particles

Pg 9 L 38 ultrafine are usually defined as $dp \leq 0.1\ \mu m$ (100 nm).

p. 12, l. 46, add reference Friedlander 2000[3]

4. Sources and Emission of Particles

It would be useful to contrast the emission inventory in Figure 4.1 with a pie chart derived from source-receptor modeling to show the impact of atmospheric chemistry, particle deposition and secondary formation.

5. Measurement of Particulate Matter

The committee agrees with the recommendations for changes to Title 17, California Administrative Code, Sections 70100(j) and 70200 to delete the current Method P and

[1] Professor Philip Hopke (Clarkson University), who is the Chair of the U.S.E.P.A. Clean Air Scientific Advisory Committee (CASAC) provided the following statement "In the decision by the U.S. DC Circuit Court of Appeals in American Trucking Associations, Inc., et al. vs. United States Environmental Protection Agency (97-1440), the court ruled that PM10 is an inappropriate indicator for coarse particles since it is confounded by the presence of PM2.5. EPA has not appealed this portion of the decision and thus, a new NAAQS for coarse particles, PM(10-2.5), will be promulgated in conjunction with the reconfirmation of the PM2.5 NAAQS. The proposal for measurement will be to use two side-by-side PM2.5 FRM samplers where the WINS impactor will be replace in one sampler with a straight tube. The difference between the two filter-based mass concentrations will be the measure of the coarse particle indicator. No decision has yet been made public as to the form or possible concentration ranges for this new PM coarse standard."

[2] Xiong and Friedlander, "Morphological Properties of Atmospheric Aerosol Aggregates", PNAS, Vol. 98, no. 21, pp. 11851-11856, 2001

[3] Friedlander, S. K., Smoke, Dust and Haze: Fundamentals of Aerosol Dynamics, 2nd edition, New York, NY: Oxford University Press, 2000.

replace it with a new Method P "Measurement Method for Particulate Matter in Ambient Air" Part I – Measurement of PM10 and Part II – Measurement of PM2.5. The committee also agrees with the recommended methods for adopting samplers that meet the Federal Reference Method requirements for PM10 and PM2.5 and to include continuous monitors whose data can be integrated and can be shown to correlate with co-located FRM samplers. The phrase 'high degree of statistical significance' (pg 43, L39; pg 44 L 4) is ambiguous and a more quantitative expression should be used.

The committee was especially supportive of the efforts being undertaken by ARB to validate continuous monitors. Continuation of these efforts is important because the possible health impacts of short-term, high level, excursions are not well understood and lack of adequate accurate short-term PM monitoring data is a primary reason for this.

The issue of sampling artifacts was raised in discussions. These included losses of volatile components under some sampling conditions and adsorption and conversion of gaseous species to particulate species on the surface of filters during sampling.[4] The use of quartz filters to avoid sulfate artifacts may lead to an overestimation of PM because of adsorption of organic vapors.[5] The possible impact of artifacts on air monitoring data from filter samplers, and methods to reduce the impacts of artifacts, should be discussed more fully in Chapter 5.

The committee makes the following recommendations:

a. Continue to evaluate continuous PM monitors for coarse and fine PM fractions.

b. Sample for coarse and fine PM separately, as opposed to using the difference between PM2.5 and PM10 filter weights.

c. Evaluate commercial continuous sulfate monitors to determine if they eliminate potential artifacts.

d. Chemical speciation should be performed to a much greater extent in California air samples. This data can be important for a number of reasons including source identifications using tracer, chemical mass balance and/or factor analytic methods. While the committee was split on whether chemical speciation would improve the

[4] Professor Freidlander has given the following example. The accumulation mode contains most of the aerosol water and serves as a site for sulfate formation by the SO2/H2O2 reaction. There is a possibility for additional sulfate formation in the aerosol filter used for sampling by reaction of SO2 and H2O2 which can dissolve in water containing aerosol already deposited in the filter. For example, consider the sequential passage through the filter of the parcels of gas, one high in SO2 concentration (from a power plant) and the other high in H2O2 (from vehicular emissions and photochemical processes). The gases may dissolve and react in the previously deposited water-containing aerosol. This would lead to artifact sulfate formation in the filter that might not have occurred in the air. In addition, the rate of diffusion from gas passing through the filter to collected aerosol is higher than the rate from a gas to a suspended particle because the diffusion rate increases with relative velocity between the gas and the deposited particles. Water vapor will continue to condense from the air on the deposited aerosol as the sulfate mass in the aqueous phase increases because of the hygroscopicity of the dissolved salts and polar organic compounds.

[5] Sioutas, personal communication, 2002

standard setting process, per se, it was clearly in favor of having more extensive analyses of the composition of ambient particles.

6. Exposure to Particles

The figure captions and legends are not informative. Most of the figures were not numbered. Even careful reading of the text left considerable confusion. Size distributions commonly are graphed with particle size increasing along the X-axis. Average total mass should be shown with each of the pie diagrams so that both the mass as well as fraction can be estimated for separate aerosol components.

Table 6.1 should also show annual arithmetic mean values, since this is the metric selected for the proposed standard.

Tables 6.1 and 6.2 need an explanation of the meaning of 'Max. Annual Avg.'

The differences in seasonal variation of PM10 and PM2.5 shown in the figures in this chapter need to be considered with respect to ability of PM10 regulations to also control PM2.5 exposures. The differences in sources and chemical composition underscore the importance of considering these separately with respect to setting regulations.

PM compounds with considerable spatial variability, such as ultrafine PM, transition metals, polar or non-polar polycyclic aromatic hydrocarbons (PAH) or elemental carbon may be potentially far more important toxicologically than PM2.5 mass, which is relatively uniform, spatially. There is considerable spatial variability of these species within a metropolitan area, consequently individual exposures to any of these compounds or size ranges may vary substantially. For example, in Los Angeles, while $PM_{2.5}$ and PM_{10} concentrations measured at various distances from highways (10-1000 meters) showed little spatial variability, particle number black carbon and organic carbon concentrations decreased rapidly with distance from highways (Zhu, et al., 2001). If these compounds are toxicologically more important than PM mass, individual exposure (and ultimately dose) may differ by more than one order of magnitude (depending on where individuals reside or spend the majority of their time) in areas where stationary PM10 or PM2.5 monitors would indicate relatively uniform population exposures.

Furthermore, ambient PM10 or PM2.5 aerosol consists of particles in size ranges spanning over 3 orders of magnitude, with equally variable deposition rates (and sites) in the respiratory tract. Exposures to aerosols at different locations/seasons with different size characteristics would result in vastly different PM doses of the exposed population. The stationary PM2.5 or PM10 data provide an overly simplified estimate of exposure, which will inevitably lead to substantial errors and uncertainly in linking health outcomes to PM mass concentrations.

The chapter summary (6.5.7) identifies various difficulties in using air quality monitoring central site data to develop and implement air quality standards. A more explicit discussion should be added explaining how such uncertainties are dealt with in the standard setting process.

7. Health Effects of Particulate Matter

The chapter was written in a somewhat fragmentary way and so rather than try to comment in a narrative fashion as was done for most of the other chapters, the committee's comments are provided on a page or section basis.

Page 116, lines 10-11, *"To the extent that PM may be causally related to*…". This statement ignores the fact that there may be real weather effects which confound PM effects away from the null, particularly in the colder-PM season in California. A more circumspect statement is required here.

Page 117, lines 35-43, *"In a separate study restricted to out-of-hospital*…". The thesis of this paragraph is not supported by some studies (see Levy, *et a*l., Epidemiology, 2001).

Therefore, this speculation needs to be tempered. This same comment applies to page 129, lines 36-43.

Page 131, 3 rd bullet. This statement is too strong. We really do not have a good qualitative estimate of the relative contribution of harvesting versus real shortening of life based on short-term studies

Page 142, lines 2-13. It also should be noted that cross-sectional studies are potentially

compromised by survivor bias, which would tend to lead to an underestimate of effect.

Page 143, lines 8 lines from bottom, *"…these effects were somewhat greater than*…". This reason does not seem very cogent in terms of the point being made. It would not at all be surprising if many years of exposure to PM carried a risk similar to that of 7 pack-years of smoking.

Page 155, lines 28-33. This statement needs to be more circumspect. The exposure evidence, to date, is weak at best, in relation to exposures likely to be experienced under ambient conditions by humans.

Page 163, lines 38-48. The argument here is not compelling. Moreover, the statement about the purpose of significance testing is simply wrong. The p-value expresses the long-range (i.e., over many repetitions of a study) of the probability of observing a result that actually observed, given some specified or unspecified null value. The p-value does not express the likelihood of results in a given study realization. A recent series of papers in Epidemiology on p-values should be consulted for a more useful discussion.

Page 167, lines 18-19. The quoted relationship between level of exposure and precision is not a causal argument at all. This statement should be removed. There could be a number of non-causal reasons—e.g., differential accuracy of measurement of exposure.

Page 170, L 46 Better justification for the assumption that 'only the fine particle share of PM10 is toxic' is required. The statement, per se, is not justifiable, scientifically and several papers are cited earlier that indicate that under some circumstances coarse PM is more toxic than fine PM. It would be useful to provide an analysis of the impact of that assumption on the level at which a standard should be set.

Page 170, L43-48, Given the almost 70 papers cited in Table 7.1 the emphasis placed on a single (Krewski) study needs explanation and justification. It is also important to differentiate how the OEHHA analysis that arrived at an annual average PM2.5

standard of 12 μg/m^3 from the USEPA analysis that used the same data but arrived at a 25 μg/m^3 annual average standard.

Page 172-173—Risk Estimates. There were a number of concerns with this section.

a. There needs to be a better explained rationale as to why 12 μg/m 3 was chosen as the level for the 24 hours standard. Why not 11 or 13 μg/m^3 ?

b. Improved methods for estimating the range of risk need to be incorporated into the standard setting process. Confidence intervals, although used by others, may not be appropriate. The use of a range of parameter estimates based on a variety of studies, preferably several that span the range of statistical approaches and study locations to quantitate the range of health effects that might be expected based on current data might be a better indicator. Expand the discussion on the potential effects of measurement error, and other sources of bias, on the estimates. The current discussion is sparse and excludes important papers such as Chen's EHP, 1999 paper on the consequence of poor model fitting for the occurrence of bias in effect estimates.

c. More emphasis should be placed on the respiratory morbidity effects in the risk assessment since they affect a large part of the population, especially children.

d. Some discussion is needed to explain why the relative incidences of acute morbidity effects are less than one might expect from the mortality estimates.

Page 174 L40 Can a % of population protected be suggested rather than 'nearly all?'

Page 178, 2nd paragraph It should be stated that studies of PM effects on the upper respiratory tract are few and far between, hence the question of whether particles 10 μm in diameter (that mainly deposit in the URT) will cause effects is unresolved. The statement 'not likely to cause serious health impacts' is an overstatement.

Page 179, Lines 30-34. The argument offered here as to why a 24 hour standard cannot be set does not make sense and is not consistent with the linear exposure-response relationship that has been observed across all short-term exposure time series studies. If the level of chronic exposure were confounding these effect estimates, it is hard to see how all of the studies would be consistent with a linear exposure-response function since each day's deaths would be the result of some people who die from chronic exposure and some who die from acute exposure. One would expect that areas with high chronic exposure would have more deaths/day due to the chronic effect in addition to those due to acute effects. On this basis, it is hard to see how a linear exposure-response relationship (on the log scale) would be observed across all short-term studies with varying levels of chronic exposure. Therefore this is not a valid argument for not setting a 24-hour PM2.5 standard. This same critique applies to the arguments on page 183, lines 26-30.

Page 180, paragraph 2. The argument that mortality rates are greater per unit change in PM concentration for long term studies versus short term studies is questionable. Although the rate may be higher for long term effects, the day to day PM variation is an order of magnitude greater than the year to year variation.

Page 181, Line 42-43 There are disconnects between PM10 and PM2.5 concentrations at some seasons of the year (as clearly shown in the figures in Chapter 6). It is not

clear that the short term PM10 standard will adequately control PM2.5 daily concentrations.

Page 187, paragraph 1 The committee disagrees with the OEHHA conclusion to not recommend a short term (24-hr) PM2.5 standard. As discussed in detail above, there are several arguments put forth but the committee felt that an adequate scientific rationale does exist for including a 24-hr PM2.5 standard in the recommendations.

Data on 4 major potential mechanisms (lung injury, inflammation, increased blood coagulation, and cardiac arrhythmias) suggest important short term effects.

8. Welfare Effects of Particulate Matter

The committee did not comment on welfare effects since our charge was the health effects basis for PM standards. The Chapter, however was a useful review of the topic.

9. Controls and Regulation of Particulate Matter

The summary of existing controls was not commented on. Again this provided a useful review of existing standards and controls.

10. Quantifying the Adverse Health Effects of Particulate Matter

Given the extensive list of morbidity outcomes that have been established and the large numbers of people affected, the emphasis on mortality as the sole rationale for PM standards seems unbalanced. The committee recommends that some method for integrating all of the health effect data into the process of arriving at protective air quality standards is needed.

Following submission of the initial AQAC comments to the staffs of OEHHA and the ARB, a reanalysis of the 24-hour PM2.5 standard was conducted. In developing a recommendation, the OEHHA and ARB staff:

- used statistical methods to examine the shape of the exposure-response relationships using two California data sets, and compared the results with those reported for other non-California data sets;

- tabulated the results of all time-series studies published in English, for which direct PM2.5 monitoring data were available, that have explored associations between low levels of ambient PM2.5 and daily mortality; and

- examined, with technical assistance from ARB staff, the upper tail of the PM2.5 distribution in California consistent with an annual average of 12 $\mu g/m^3$, based on data collected throughout California in 1999 and 2000.

Based on the results of these analyses, OEHHA recommended that the 24-hour PM2.5 standard be established at a level of 25 $\mu g/m^3$, not to be exceeded. The adoption of the recommendation for an annual PM2.5 standard of 12 $\mu g/m^3$ was considered to be an integral component of the proposal.

The AQAC had been concerned that the proposed standard based on attaining a 12 $\mu g/m^3$ annual average did not adequately protect against brief (i.e., one to several days) increases in PM2.5 levels. It was recognized that attainment of the recommended annual standard would help shift the entire PM2.5 distribution to the left, and would

influence peak concentrations. The committee indicated that a 24-hour standard would better protect Californians against significant short-term elevations of PM2.5.

The committee met in a public forum on April 3, 2002 to discuss the proposed 25 $\mu g/m^3$ PM2.5 24-hour standard. The AQAC endorsed the both the proposed standard and the process used to arrive at the standard. The committee agreed that the "not to exceed" form of the standard was appropriate.

This standard, in the AQAC's opinion, represents a balance between some competing issues. For example, in some areas, the 24-hour standard may dominate over the annual standard. However this competes with the need for the standards to provide an adequate margin of safety (as demanded by the legislature) and to take into account the potentially greater susceptibility of children to the effects of PM.

Specific Questions Addressed by the Committee

1. Have the key studies relevant to the recommendations been identified and appropriately interpreted? Are there any critical studies (published prior to 8/1/01) that have been omitted from review in this draft recommendation? Reviewers should bear in mind that the scientific foundation for the recommendations represents a focused evaluation of the critical literature, not an exhaustive compendium of all potentially relevant research.

   The OEHHA Staff has attempted a critical review of a very large, complex, and dynamic field involving different disciplines. The draft document is provides excellent reviews of current literature on PM exposure, epidemiology and toxicology. This does not mean that there are not major uncertainties and issues that need to be resolved about the toxic effects of PM, but the available (and quite exhaustive) literature has been properly reviewed and cited.

2. Have susceptible subpopulations been appropriately identified? Are there other sub-populations that may be at least as sensitive to PM exposure as those identified in the document? Is the scientific evidence related to infants and children correctly interpreted?

   Diabetics should be considered. In several single-city studies, the risk of PM-associated hospital admissions for heart disease for diabetics was double that for the general population (Zanobetti and Schwartz, 2001b; Zanobetti and Schwartz, 2001c). In addition, diabetics were found to have an increased risk of PM-associated mortality (Bateson and Schwartz, 2001). The scientific evidence regarding children and infants should also be considered beyond the immediate health effects. The impact on their caregivers (lost time from work and financial issues) and lost time from education could have significant societal effects.

3. Is there additional critical information that should be considered in estimating PM-related impacts on public health?

   Yes. The PM impacts on public health are estimated assuming population-based exposure models and PM mass concentrations measured at single outdoor monitoring sites as surrogates of population exposures to ambient air PM. The extent to which outdoor measurements accurately reflect PM exposures has been

the subject of considerable scientific debate. Results from early exposure studies such as those conducted as part of the Harvard Six Cities Study and the EPA Particle Total Exposure Assessment Methodology (PTEAM) Study, for example, suggested that personal PM exposures might differ substantially from outdoor concentrations due to contributions from indoor sources.

The link between central site and personal exposures need to be better defined and should be considered in future standard evaluations.

Also, as mentioned in the specific comments, above, the temporal and spatial variations in components of PM may significantly modify dose and biological responses. This is not given sufficient weight in the current standard setting process.

4. Have the uncertainties concerning the health effects of exposure to PM been adequately described?

   Major uncertainties that could be better discussed include the influence of indoor exposures, the link between central site and personal exposures, and the spatial and temporal variation in concentrations of toxic PM components.

5. Have potential differential exposure patterns among infants and children been examined sufficiently in the document?

   There are very scant data on this topic. This should be an area for additional research.

6. Is the overall approach to developing the recommendations for ambient PM standards transparent and appropriate? Specifically, are the recommendations for PM ambient air quality standards for California adequately supported by the underlying scientific rationale, specifically the:

   annual average for PM10?

   annual average for PM2.5?

   24-hr average for PM10?

   24-hr average for sulfates?

   The committee endorsed the recommendations for above four standards for the current period. There was discussion of the need for a future evaluation of the possibility that there is overlap between PM standards and the sulfate standard, to the extent that the sulfate standard might be considered redundant.

7. Given the state of the science, do you concur with OEHHA staff that there is insufficient evidence at present to develop a 24-hr average (or other short-term) standard for PM2.5?

   The committee recommended that a 24-hr PM2.5 standard be developed. This was accomplished and reviewed by AQAC on April 3, 1002. AQAC endorsed the new recommendation.

8. What do you see as the most important research issues to be addressed prior to the next cycle of review for PM?

- Evaluate regional differences in relationships between PM and gaseous co-pollutants;
- Characterize short-term PM exposures using validated continuous monitors;
- Speciate PM (metals, EC/OC, PAH's, NO_3);
- Characterize ultrafine exposures (Indoor, Outdoor, personal);
- Validate new or improved monitoring techniques, especially continuous monitors of PM2.5, PM10, coarse PM, sulfates that will allow specific questions to be addressed as to the most relevant averaging times for health-based particle standards;
- Increase our understanding of respiratory dosimetry and particle fate and transport in infants and children;
- Expand the base of studies on susceptibility of diabetics;
- Evaluate the relationship and mechanism of PM exposure and prenatal/neonatal health effects;
- Determine relationship(s) between ultrafine and coarse particulates versus different health outcomes;
- Define health effects/mechanisms of coarse, fine, and ultrafine PM and co-pollutants;
- Examine effects and mechanisms in cardiovascular subjects exposed to different size cuts of particles;
- Explore the roles of different chemical or metal constituents of PM in causing health effects.
- Using already established PM source emissions profiles and new state-of-the-art personal monitoring techniques, assess degree to which specific outdoor sources contribute to personal PM concentrations.
- As control strategies are implemented to achieve the proposed standards, it will be important to determine whether or not children and adults living in less affluent, more highly polluted, communities are receiving adequate benefit and protection.
- Our knowledge of the intractive effects of pollutants is inadequate for the development of comprehensive air quality improvement measures. The research base must be expanded and supported.

Appendix 3

Responses to Comments from the Air Quality Advisory Committee

Staff Responses to Comments from Members of the Air Quality Advisory Committee

The Air Quality Advisory Committee (AQAC) held public meetings in January and April, 2002 in order to review and consider public input on the recommendations in the draft PM report entitled "Review of the California Ambient Air Quality Standards for Particulate Matter and Sulfates." The Committee submitted their comments to the Air Resources Board (Board) and Office of Environmental Health Hazard Assessment (OEHHA) for response. The following is an overview of the written and oral comments provided by the members of the AQAC, and the corresponding responses from staff from the Board or OEHHA. Comments that address specific sections of the draft PM report, are referenced by page and line number, where appropriate.

We thank the AQAC for the time and effort each of its members dedicated to reviewing this document in order to ensure that its contents are based on a foundation of sound science and that the findings and recommendations contained within the report are protective of public health.

CHAPTER 1: EXECUTIVE SUMMARY

1. **COMMENT:** Page 2, line 13-4, *"there are fewer studies.."* This statement is false and needs to be corrected. **RESPONSE:** The statement in these lines of text suggests that there are fewer studies available on the mortality and morbidity effects associated with short-term exposures to PM than long-term exposure. The statement has been removed, in light of recent decisions, and the text has been revised to support the PM2.5 24-hour standard recommendation, based on short-term health effects.

CHAPTER 2: INTRODUCTION

2. **COMMENT:** Regulations require that standards be reviewed when 'substantial new information becomes available' or at least once every 5 years. The Committee suggests that some specific triggers for review might be new information on effects in susceptible populations that might indicate erosion of margins of safety, or information bearing on the need for additional standards, e.g. a coarse particle standard (PM2.5-10). There are also data that suggest that ultrafine particles may be a size fraction that plays an important role in health effects. There are also metrics, other than mass of particles in a given size fraction, that might be better predictors of effects on health, including: aerosol acidity; aerosol oxidant (peroxides, radicals); Ames Test activity; and ultrafine component ($1nm \leq d_p \leq 0.1\ \mu m$) . An integrative approach to standard setting should be developed. Such an approach would improve ability to identify possible interactions between pollutants that might impact on the level set for a particulate standard. Such an approach might make it easier to recognize whether there are un-needed redundancies in standards. For example, it might be determined that a separate sulfate standard is not needed in the future. The chapter should be expanded to delineate future possibilities and triggers. **RESPONSE:** These informative suggestions will be taken under advisement when we plan the next PM and sulfates standards review process.

CHAPTER 3: PHYSICS AND CHEMISTRY OF PARTICLES

3. **COMMENT:** Page 9, line 38, "ultrafine" particles are usually defined as $dp \leq 0.1\ \mu m$ (100 nm). Page 12, line 46, add reference to Friedlander 2000. **RESPONSE:** These corrections have been made.

CHAPTER 4: SOURCES AND EMISSION OF PARTICLES

4. **COMMENT:** It would be useful to contrast the emission inventory in Figure 4.1 with a pie chart derived from source-receptor modeling to show the impact of atmospheric chemistry, particle deposition and secondary formation. **RESPONSE:** One of the problems that may arise in attempting to perform this task is that the inventory pie chart, which is statewide and annual, would be difficult to pair with a chemical mass balance (CMB) pie chart that would be local and seasonal based on the data available.

CHAPTER 5: MEASUREMENT OF PARTICULATE MATTER

5. **COMMENT:** The Committee agrees with the recommendations for changes to title 17, California Code of Regulations, sections 70100(j) and 70200 to delete the current Method P and replace it with a new Method P "Measurement Method for Particulate Matter in Ambient Air" Part I – Measurement of PM10 and Part II – Measurement of PM2.5. The Committee also agrees with the recommended methods for adopting samplers that meet the Federal Reference Method requirements for PM10 and PM2.5 and to include continuous monitors whose data can be integrated and can be shown to correlate with co-located FRM samplers. The phrase 'high degree of statistical significance' (page 43, line39; page 44, line 4) is ambiguous and a more quantitative expression should be used. **RESPONSE:** A more quantitative description has been included in the revised text.

6. **COMMENT:** The Committee is especially supportive of the efforts being undertaken by ARB to validate continuous monitors. Continuation of these efforts is important because the possible health impacts of short-term, high level, excursions are not well understood and lack of adequate accurate short-term PM monitoring data is a primary reason for this.

   The issue of sampling artifacts was raised in discussions. These included losses of volatile components under some sampling conditions and adsorption and conversion of gaseous species to particulate species on the surface of filters during sampling. The use of quartz filters to avoid sulfate artifacts may lead to an overestimation of PM because of adsorption of organic vapors. The possible impact of artifacts on air monitoring data from filter samplers, and methods to reduce the impacts of artifacts, should be discussed more fully in Chapter 5. **RESPONSE:** A more detailed discussion has been included in the revised Chapter 5.

7. **COMMENT:** The Committee makes the following recommendations:

   a) Continue to evaluate continuous PM monitors for coarse and fine PM fractions.

   b) Sample for coarse and fine PM separately, as opposed to using the difference between PM2.5 and PM10 filter weights.

   c) Evaluate commercial continuous sulfate monitors to determine if they eliminate potential artifacts.

   d) Chemical speciation should be performed to a much greater extent in California air samples. These data can be important for a number of reasons including source identifications using tracer, chemical mass balance and/or factor analytic methods. While the Committee was split on whether chemical speciation would improve the standard setting process, per se, it was clearly in favor of having more extensive analyses of the composition of ambient particles. **RESPONSE:** While we are unable to incorporate them into this round of review, we will consider them the next time the PM and sulfates standards are reviewed.

CHAPTER 6: EXPOSURE TO PARTICLES

8. **COMMENT:** The figure captions and legends are not informative. Most of the figures were not numbered. Even careful reading of the text left considerable confusion. Size distributions commonly are graphed with particle size increasing along the X-axis. Average total mass should

be shown with each of the pie diagrams so that both the mass as well as fraction can be estimated for separate aerosol components**. RESPONSE:** The figures and graphs have been reviewed and revised, where appropriate, for clarity.

9. **COMMENT:** Table 6.1 should also show annual arithmetic mean values, since this is the metric selected for the proposed standard. Tables 6.1 and 6.2 need an explanation of the meaning of 'Max. Annual Avg.' **RESPONSE:** The text has been revised to clarify this issue.

10. **COMMENT:** The differences in seasonal variation of PM10 and PM2.5 shown in the figures in chapter 6 need to be considered with respect to ability of PM10 regulations to also control PM2.5 exposures. The differences in sources and chemical composition underscore the importance of considering these separately with respect to setting regulations.

    PM compounds with considerable spatial variability, such as ultrafine PM, transition metals, polar or non-polar polycyclic aromatic hydrocarbons (PAH) or elemental carbon may be potentially far more important toxicologically than PM2.5 mass, which is relatively uniform, spatially. There is considerable spatial variability of these species within a metropolitan area, consequently individual exposures to any of these compounds or size ranges may vary substantially. For example, in Los Angeles, while $PM_{2.5}$ and PM_{10} concentrations measured at various distances from highways (10-1000 meters) showed little spatial variability, particle number black carbon and organic carbon concentrations decreased rapidly with distance from highways (Zhu, et al., 2001). If these compounds are toxicologically more important than PM mass, individual exposure (and ultimately dose) may differ by more than one order of magnitude (depending on where individuals reside or spend the majority of their time) in areas where stationary PM10 or PM2.5 monitors would indicate relatively uniform population exposures.

    Furthermore, ambient PM10 or PM2.5 aerosol consists of particles in size ranges spanning over 3 orders of magnitude, with equally variable deposition rates (and sites) in the respiratory tract. Exposures to aerosols at different locations/seasons with different size characteristics would result in vastly different PM doses of the exposed population. The stationary PM2.5 or PM10 data provide overly simplified estimates of exposure, which will inevitably lead to substantial errors and uncertainly in linking health outcomes to PM mass concentrations. **RESPONSE:** It is important to point out that control measures are not part of the standard setting process; however, they do play a role and are taken into consideration in subsequent activities related to planning and attainment. However, we agree that characterizing uncertainty associated with measurements, seasonal variation, exposure characterization, and spatial and temporal variation is a very important part of the overall process. It is the goal of ARB and OEHHA to identify, characterize, and attempt to reduce and address these uncertainties in the most accurate manner possible as well as continue to focus on these uncertainties in future research, standard reviews, control and attainment processes, in order to ensure the protection of public health.

11. **COMMENT:** The chapter summary (6.5.7) identifies various difficulties in using air quality monitoring central site data to develop and implement air quality standards. A more explicit discussion should be added explaining how such uncertainties are dealt with in the standard setting process. **RESPONSE:** Sections 6.5.1 and 6.5.3 have been revised to provide a fuller description of the relationships between ambient and personal exposure. However, the section cited, 6.5.7, does not discuss the issue raised. Given the complex and variable relationship between ambient and personal exposure, along with the paucity of data available, ambient air quality standards are based on exposure estimates obtained from central site monitors.

CHAPTER 7: HEALTH EFFECTS OF PARTICULATE MATTER

12. **COMMENT:** Page 116, Lines 10-11, *"To the extent that PM may be causally related to*…" This statement ignores the fact that there may be real weather effects which confound PM effects away from the null, particularly in the colder-PM season in California. A more circumspect statement is required here. **RESPONSE:** The text has been modified taking this comment into account and now reads as follows: "To the extent that PM may be causally related to mortality and correlated as well with these meteorological variables, these multiple statistical controls could result in an underestimate of the effects of PM, though residual confounding by weather factors might also bias the PM effects away from the null hypothesis of no effect."

13. **COMMENT:** Page 117, Lines 35-43, *"In a separate study restricted to out-of-hospital*…" The thesis of this paragraph is not supported by some studies (see Levy, *et a*l., Epidemiology, 2001). Therefore, this speculation needs to be tempered. This same comment applies to page 129, lines 36-43. **RESPONSE:** The following sentences were added to the paragraph quoted above (Draft, p. 117, lines 35-43) to respond to the concern expressed: **"**However, deaths occurring among those outside of a hospital may represent individuals who are frail or without health insurance, or both. In contrast to the results reported by Schwartz et al. (1994b), Levy et al. (2001) did not find any association between PM10 and the incidence of primary cardiac arrest using a case-crossover analysis. This study, though, involved a small number of cases in Seattle, where relatively low levels of PM occurred during the study period [1988-1994, mean PM10 = 31.9 $\mu g/m^3$, mean PM2.5=18.4 $\mu g/m^3$]."

    The text on Draft, p. 129, has likewise been tempered in that we now refer to the "possibility" instead of "likelihood" of significant loss in life expectancy being "suggested" as opposed to "reinforced" by studies of out-of-hospital deaths.

14. **COMMENT:** Page 131, 3rd bullet. This statement is too strong. We really do not have a good qualitative estimate of the relative contribution of harvesting versus real shortening of life based on short-term studies. **RESPONSE:** The text has been modified, and now reads as follows: **"**Study results suggest that some, and perhaps a large fraction of, mortality associated with acute exposure is not the result of just a few days of life shortening." In the prior Draft the text had read: "The results indicate that much mortality associated …".

15. **COMMENT:** Page 142, Lines 2-13. It also should be noted that cross-sectional studies are potentially compromised by survivor bias, which would tend to lead to an underestimate of effect. **RESPONSE:** The following sentence has been added to the text, following the text indicated in the comment: **"**Moreover, in cross-sectional studies people who may have died from exposure-related illness are not included in the analysis. This "survivor bias" tends to underestimate effects of exposures (assuming that such effects exist).

16. **COMMENT:** Page 143, 8 lines from bottom, *"…these effects were somewhat greater than*…" This reason does not seem very cogent in terms of the point being made. It would not at all be surprising if many years of exposure to PM carried a risk similar to that of 7 pack-years of smoking. **RESPONSE:** The phrase referred to in the comment has been deleted.

17. **COMMENT:** Page 155, Lines 28-33. This statement needs to be more circumspect. The exposure evidence, to date, is weak at best, in relation to exposures likely to be experienced under ambient conditions by humans. **RESPONSE:** In response to this comment, as well as to several received from the public, the text has been modified to reflect a more tentative position regarding the strength of the evidence of systemic effects from exposure to ambient particles. The modified text reads as follows: "Taken together, these data suggest that inhalation of different sources of particles may initiate inflammatory events in human lungs, with some (albeit sparse) evidence of systemic impacts, including stimulation of bone marrow to accelerate production of inflammatory cells to respond to the pulmonary insult. However, these observations are subject to

the caveat that the results observed in the high-dose animal and *in vitro* experiments, as well as in the controlled human exposures, may or may not be directly applicable to humans exposed to ambient PM."

18. **COMMENT:** Page 163, Lines 38-48. The argument here is not compelling. Moreover, the statement about the purpose of significance testing is simply wrong. The p-value expresses the long-range (i.e., over many repetitions of a study) of the probability of observing a result that actually observed, given some specified or unspecified null value. The p-value does not express the likelihood of results in a given study realization. A recent series of papers in Epidemiology on p-values should be consulted for a more useful discussion. **RESPONSE:** Several changes were made in the text referred to in the comment, and an additional paragraph was added to reflect the concerns expressed.

19. **COMMENT:** Page 167, Lines 18-19. The quoted relationship between level of exposure and precision is not a causal argument at all. This statement should be removed. There could be a number of non-causal reasons—e.g., differential accuracy of measurement of exposure. **RESPONSE:** We have modified the text in response to this comment. Nondifferential, independent misclassification of either disease or exposure results in a bias towards the null hypothesis of no effect. Reduction of such misclassification, assuming that a causal relationship exists, should have the opposite effect. The comment raises the issue that a change in the strength of association accompanying a more precise measure of disease or exposure, either within or between studies, may be due to a change in something else such as measurement error, which would represent a noncausal explanation. Thus, to clarify the text in the Draft, we have added the caveat that, *with everything else held equal*, increasing the precision of measurement (and thereby decreasing the measurement error), would increase the strength of association, assuming that one is dealing with a causal relationship.

20. **COMMENT:** Page 170, Line 46. Better justification for the assumption that 'only the fine particle share of PM10 is toxic' is required. The statement, per se, is not justifiable, scientifically and several papers are cited earlier that indicate that under some circumstances coarse PM is more toxic than fine PM. It would be useful to provide an analysis of the impact of that assumption on the level at which a standard should be set. **RESPONSE:** Our justification for this assumption in the benefits analysis is the evidence provided by Krewski et al. (2000) in their reanalysis of the ACS cohort. The adjustment was based on the re-analysis of the ACS data set by Pope and others cited in Krewski et al. (2000), which shows that for long-term exposure, coarse particles were not associated with mortality. As explained in the text, this is a conservative approach, which may lead to an underestimate of the effects.

21. **COMMENT:** Page 170, Lines 43-48. Given the almost 70 papers cited in Table 7.1 the emphasis placed on a single (Krewski) study needs explanation and justification. It is also important to differentiate how the OEHHA analysis that arrived at an annual average PM2.5 standard of 12 $\mu g/m^3$ from the USEPA analysis that used the same data but arrived at a 25 $\mu g/m^3$ annual average standard. **RESPONSE:** The many papers referenced in Table 7.1 refer to acute effects of PM, whereas the Krewski et al. (2000) report is an exhaustive re-analysis of the two major studies of the chronic impacts of exposure to PM: the Harvard Six Cities study reported by Dockery et al. (1993) and the American Cancer Society Cohort reported by Pope et al. (1995). These are all described in the paragraphs in this section and in Section 7.4 "Chronic Exposure- Mortality." As noted in the Draft a couple of sentences prior to those referred to in the comment, "As reviewed in Sections 7.3 and 7.4, both short-term (daily or multi-day) and long-term (a year to several years) exposures to PM have been associated with mortality. Long-term exposure estimates are preferable since they include the effects of both long and short-term exposure and clearly represent a significant reduction in life expectancy." We believe that this explanation is clear and therefore have not modified the text in response to this comment. Moreover, the USEPA actually

proposed a 15 μg/m^3 annual average standard for PM2.5, not 25 μg/m^3. Ultimately, the decision regarding the level for any standard depends on the relative weights one wishes to accord to different studies, and how one deals with uncertainty. We cannot claim to know all of the thinking that went into the formulation of the USEPA's annual PM2.5 standard. However, as discussed in the document, there are a few studies linking PM2.5 with mortality and morbidity, in which the long-term mean concentrations were below 15 μg/m^3 PM2.5.

22. **COMMENT:** Page 172-173—Risk Estimates. There were a number of concerns with this section. **a.** There needs to be a better explained rationale as to why 12 μg/m^3 was chosen as the level for the 24 hours standard. Why not 11 or 13 μg/m^3? **b.** Improved methods for estimating the range of risk need to be incorporated into the standard setting process. Confidence intervals, although used by others, may not be appropriate. The use of a range of parameter estimates based on a variety of studies, preferably several that span the range of statistical approaches and study locations to quantitate the range of health effects that might be expected based on current data might be a better indicator. Expand the discussion on the potential effects of measurement error, and other sources of bias, on the estimates. The current discussion is sparse and excludes important papers such as Chen's EHP, 1999 paper on the consequence of poor model fitting for the occurrence of bias in effect estimates. **c.** More emphasis should be placed on the respiratory morbidity effects in the risk assessment since they affect a large part of the population, especially children. **d.** Some discussion is needed to explain why the relative incidences of acute morbidity effects are less than one might expect from the mortality estimates. **RESPONSE:** (a) We have provided a detailed rationale for the selection of 12 μg/m^3 in the recommendations section and have added two figures to make the argument more transparent. As we have indicated in the text, however, there is no clear zero-risk bright line. This concentration is below the means of the studies that have found important associations between PM2.5 and both mortality and morbidity. Specifically, consideration of a standard at 12 μg/m^3 places significant weight on the long-term exposure studies using the ACS and Harvard Six-Cities data (Dockery et al., 1993; Pope et al., 1995; Krewski et al., 2000). In these studies, robust associations were reported between long-term exposure to PM2.5 and mortality. The mean PM2.5 concentration was 18 μg/m^3 (range of 11.0 to 29.6 μg/m^3) in the Six-Cities study and 20 μg/m^3 (range of 9.0 to 33.5 μg/m^3) in the ACS study (see Figure 7.6). Thresholds were not apparent in either of these studies. In the Dockery et al. study, the relative risks are similar to the cities at the lowest long-term PM2.5 concentrations of 11 and 12.5 μg/m^3. Larger increases in risk don't occur until the long-term PM2.5 mean equals 14.9 μg/m^3. Therefore, an annual standard of 12 μg/m^3 would be below the mean of the most likely effects level and would provide a margin of safety. Targeting a long-term mean PM2.5 concentration of 12 μg/m^3 would also place some weight on the results of multiple daily exposure studies examining relationships between PM2.5 and adverse health outcomes (Table 7.2). These studies have long-term (three- to four-year) means in the range of 13 to 18 μg/m^3. A standard set at 12 μg/m^3 provides additional protection against mortality in adults associated with long-term exposure, as well as against a variety of morbidity effects in children (described in Section 7.6, above). In the opinion of OEHHA staff, an annual PM2.5 standard of 12 μg/m^3 would be likely to provide adequate protection of public health, including that of infants and children, against adverse effects of long-term exposure. (b) Depending on the health endpoint that is estimated, the confidence intervals reflect both the statistical uncertainty in a given study and the range of effects over several studies. In general, we have tried to use and adapt the analysis of benefits conducted by the U.S. EPA in its report to Congress, since that report has already undergone scientific peer review. (c) Many of the respiratory morbidity effects are included in the full analysis of the benefits of reducing PM provided in Chapter 10. We have simply discussed a subset of the endpoints in this section. (d) The results are a straightforward application of the results of the existing epidemiological studies and existing health outcome as reviewed in detail by U.S. EPA in its report to Congress. The effects estimated are a product of the exposed population, the risk per

unit and the change in air pollution. Measurement errors, difficulty in ascertainment, and sample selection bias could all affect the final risk estimates.

23. **COMMENT:** Page 174 Lines 40. Can a % of population protected be suggested rather than 'nearly all?' **RESPONSE:** At this point, we do not have adequate information to precisely determine the number of people in each subgroup that would be protected. Unfortunately, there is uncertainty about both the specific subgroups that may be sensitive as well as the number of people currently in each of the subgroups (i.e., the number of asthmatic children in California, or the number of frail elderly people with heart disease). Therefore, we are implying that by setting standards below the concentrations where health effects have been shown to occur, we are providing protection for a large segment of the population.

24. **COMMENT:** Page 178, 2nd paragraph. It should be stated that studies of PM effects on the upper respiratory tract are few and far between; hence the question of whether particles 10 µm in diameter (that mainly deposit in the URT) will cause effects is unresolved. The statement 'not likely to cause serious health impacts' is an overstatement. **RESPONSE:** We have modified the text to delete the phrase of concern, and to take into account the relative paucity of studies of the impact of particle deposition in the extrathoracic region.

25. **COMMENT:** Page 179, Lines 30-34. The argument offered here as to why a 24 hour standard cannot be set does not make sense and is not consistent with the linear exposure-response relationship that has been observed across all short-term exposure time series studies. If the level of chronic exposure were confounding these effect estimates, it is hard to see how all of the studies would be consistent with a linear exposure-response function since each day's deaths would be the result of some people who die from chronic exposure and some who die from acute exposure. One would expect that areas with high chronic exposure would have more deaths/day due to the chronic effect in addition to those due to acute effects. On this basis, it is hard to see how a linear exposure-response relationship (on the log scale) would be observed across all short-term studies with varying levels of chronic exposure. Therefore this is not a valid argument for not setting a 24-hour PM2.5 standard. This same critique applies to the arguments on page 183, lines 26-30. **RESPONSE:** The revised document now includes a recommendation for a 24-hr PM2.5 standard of 25 $\mu g/m^3$, not to be exceeded.

26. **COMMENT:** Page 180, paragraph 2. The argument that mortality rates are greater per unit change in PM concentration for long term studies versus short term studies is questionable. Although the rate may be higher for long term effects, the day to day PM variation is an order of magnitude greater than the year to year variation. **RESPONSE:** We have calculated the effects of moving from current concentrations to the standards. To do so, we assume that the annual change is made up of 365 similar daily changes. Given the linearity of the functions, however, this assumption is not biasing the results. Therefore, we are applying the evidence that a 10 $\mu g/m^3$ change would generate a larger effect from the studies of long-term exposure than from the short-term exposure. While short-term exposures certainly have greater variation over the year, they will be made up of some very small or zero changes and some large changes.

27. **COMMENT:** Page 181, Line 42-43. There are disconnects between PM10 and PM2.5 concentrations at some seasons of the year (as clearly shown in the figures in Chapter 6). It is not clear that the short term PM10 standard will adequately control PM2.5 daily concentrations. **RESPONSE:** The revised document now includes a recommendation for a 24-hr PM2.5 standard of 25 $\mu g/m^3$, not to be exceeded.

28. **COMMENT:** Page 187, paragraph 1. The committee disagrees with the OEHHA conclusion to not recommend a short term (24-hr) PM2.5 standard. As discussed in detail above, there are several arguments put forth but the committee felt that an adequate scientific rationale does exist for including a 24-hr PM2.5 standard in the recommendations. Data on 4 major potential

mechanisms (lung injury, inflammation, increased blood coagulation, and cardiac arrhythmias) suggest important short-term effects. **RESPONSE:** The revised document now includes a recommendation for a 24-hr PM2.5 standard of 25 $\mu g/m^3$, not to be exceeded.

29. **COMMENT:** Given the extensive list of morbidity outcomes that have been established and the large numbers of people affected, the emphasis on mortality as the sole rationale for PM standards seems unbalanced. The committee recommends that some method for integrating all of the health effect data into the process of arriving at protective air quality standards is needed. **RESPONSE:** We have revised the recommendations section so it is clear that the proposed standards will generate reductions in morbidity, as well as mortality. This is also reflected in the full analysis of benefits provided in Chapter 10.

CHAPTER 8: WELFARE EFFECTS OF PARTICULATE MATTER

30. **COMMENT:** The Committee did not comment on welfare effects since our charge concerns the health effects basis for PM standards. The Chapter, however was a useful review of the topic.

CHAPTER 10: QUANTIFYING THE ADVERSE HEALTH EFFECTS OF PARTICULATE MATTER

31. **COMMENT**: Given the extensive list of morbidity outcomes that have been established and the large numbers of people affected, the emphasis on mortality as the sole rationale for PM standards seems unbalanced. The Committee recommends that some method for integrating all of the health effect data into the process of arriving at protective air quality standards is needed. **RESPONSE:** We are in agreement with the need for balancing the rationale for standards between morbidity and mortality outcomes. However, this chapter does provide discussion related to morbidity effects, specifically pages 267 through 277 (Section 10.1.5.5 through Section 10.1.5.7), as well as Tables 10.4 through 10.10. Also note that the text in Chapter 10 is now contained within Chapter 9.

APPENDIX 3: REFERENCES FOR CHAPTER 7 RESPONSES

Chen C, Chock DP, Winkler SL (1999). A simulation study of confounding in generalized linear models for air pollution epidemiology. Environ Health Perspect 107(3):217-22.

Dockery DW, Pope CA III, Xu X, Spengler JD, Ware JH, Fay ME *et al*. (1993). An association between air pollution and mortality in six U.S. cities. N Engl J Med 329:1753-9.

Krewski D, Burnett R, Goldberg MS, Koover K, Siemiatycki J, Jerrett M *et al*. (2000). Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Res Rep Health Eff Inst (A special report of the Institute's Particle Epidemiology Reanalysis Project).

Levy D, Sheppard L, Checkoway H, Kaufman J, Lumley T, Koenig J *et al*. (2001). A case-crossover analysis of particulate matter air pollution and out- of-hospital primary cardiac arrest. Epidemiology 12(2):193-9.

Pope CA III (1996). Particulate pollution and health: a review of the Utah valley experience. J Expo Anal Environ Epidemiol 6(1):23-34.

Pope CA III, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE *et al*. (1995). Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Respir Crit Care Med 151:669-74.

Schwartz J (1994b). What are people dying of on high air pollution days? Environ Res 64:26-35.

Appendix 4

Summaries of Public Comments and Responses

LIST OF COMMENTERS

Written comments were received from the following individuals and groups:

1. American Lung Association of California, American Lung Association, Natural Resources Defense Council, Environmental Working Group, Committee for Law, Air, Water and Species, Transportation Solutions Defense and Education Fund (January 11, 2002)
2. Golden Gate University Environmental Law and Justice Clinic, Bayview Hunters Point Community Advocates, Bluewater Network, Communities for a Better Environment, Our Children's Earth Foundation (January 11, 2002)
3. Western States Petroleum Association (January 11, 2002)
4. Engine Manufacturers Association (January 11, 2002)
5. Center for Energy Efficiency and Renewable Technologies (January 8, 2002)
6. The Sierra Club (January 8, 2002)
7. Environmental Defense (January 11, 2002)
8. Marc Chytilo, Esq. representing unspecified groups (December 11, 2001)
9. Renee Sharp representing the Environmental Working Group (December 11, 2002)
10. Imperial County Air Pollution Control District (December 20, 2001)
11. Ford Motor Company (January 9, 2002)
12. Alliance of Automobile Manufacturers (January 10, 2002)
13. Frederick W. Lipfert, Ph.D. representing himself (January 9, 2002)
14. Engine Manufacturers Association (March 22, 2002)
15. Ford Motor Company (March 22, 2002)
16. Golden Gate University Environmental Law and Justice Clinic, Bayview Hunters Point Community Advocates, Bluewater Network, Communities for a Better Environment, Our Children's Earth Foundation (March 20, 2002)
17. American Lung Association of California, American Lung Association, Natural Resources Defense Council, Environmental Working Group, Medical Alliance for Healthy Air, Transportation Solutions Defense and Education Fund (March 25, 2002)
18. Alliance of Automobile Manufacturers (March 25, 2002)
19. Environmental Defense (April 2, 2002)
20. Golden Gate University Environmental Law and Justice Clinic, Our Children's Earth Foundation (February 21, 2002)
21. American Lung Association of California, American Lung Association, Natural Resources Defense Council, Environmental Working Group, Medical Alliance for Healthy Air, Transportation Solutions Defense and Education Fund (March 5, 2002)
22. Engine Manufacturers Association (December 5, 2001)

Responses to the Public Comments to AQAC

The individuals and entities listed above submitted written comments on the November 30, 2001 draft of the report "Review of the California Ambient Air Quality Standards for Particulate Matter and Sulfates" or the April 3, 2002 draft of the "Proposal to Establish a 24-hour Standard for PM2.5." The comments and responses are organized first by chapter, and secondarily by subject within the chapter of the draft report. The source of each comment is in parentheses following each comment, with the numbers referring to the list above.

CHAPTER 2: PROCEDURAL ISSUES

1. **COMMENT:** The time allotted for public review before the January 23 and 24, 2002 AQAC meeting was too short. (Commenters 4, 7, 19, 12, 22) **RESPONSE:** The comment period was extended until January 11, 2002. Also, public comments will be accepted up to and including the Board meeting scheduled for June 20 and 21, 2002.

2. **COMMENT:** Review procedures followed by U. S. EPA were not followed. (Commenter 12) **RESPONSE**: California law differs considerably in the procedural requirements for proposed regulatory actions. The procedures used by CARB/OEHHA are in accordance with the Health and Safety Code and the Administrative Procedures Act.

3. **COMMENT:** The report does not consider the environmental justice issue of people living near power plants and refineries who are likely to be exposed to localized PM plumes that have PM2.5/PM10 ratios higher than regional values. (Commenter 2) **RESPONSE**: The nature and degree of control for specific source categories of PM is related to the implementation of the standards, not to the choice of concentrations for the standards. The PM standards are based on health considerations, as specified in sections 39014 and 39606 of the Heath and Safety Code, so that the standards are designed to be health protective for all Californians, regardless of where they live. The standards apply equally to all areas of the State. After standards are promulgated, various emission standards and other control measures will be adopted by ARB and the Districts, in order to attain and maintain the standards. Environmental justice issues are considered during the control phase of the process. ARB's statewide programs reduce overall emissions to improve air quality all over the State, including in local neighborhoods. In addition, ARB is also pursuing special programs to reduce neighborhood-level pollution, for example, inspecting trucks for excess smoke, and evaluating technology to further reduce chrome emissions from plating shops.

4. **COMMENT:** The commenter pointed out that communities where a large portion of the population is low-income or of color are more exposed to ambient air pollution, and that consequently they are at greater risk of adverse health effects from PM exposure. In light of this, the commenter expressed concern that environmental justice may not have been adequately considered in the standards process. (Commenter 15) **RESPONSE**: Ambient air quality standards are the legal definition of clean air, and they apply equally throughout the state. Air pollution control plans and actions taken to bring about attainment with the standards are the responsibility of ARB for mobile sources, and the local air quality control and management districts for stationary sources. Issues of environmental justice are important considerations in evaluating and developing control strategies, both at the statewide and local levels, and must include consideration of local and/or neighborhood sources and impacts so as to bring the entire state into compliance with the standards.

CHAPTER 3: CHEMISTRY AND PHYSICS

5. **COMMENT:** A reference should be provided for the statement on pg. 11, lines 11-13. (Commenter 11) **RESPONSE**: Reference to Murphy et al, 1998 will be added to the next draft.

   Murphy, S.A., K.A. BeruBe, F.D. Pooley, R.J. Richards (1998), The response of lung epithelium to well characterized fine particles, Life Sciences. **62**: 1789-1799.

6. **COMMENT:** The sentence on pg. 12, lines 12-13 should be reworded. (Commenter 11) **RESPONSE**: This has been revised.

7. **COMMENT:** Measurements of optical properties for the fine particle fraction indicate that there is aerosol variation in both seasonal and monthly time scales (draft report pg. 17). This raises the issue of the spatial and temporal variation of real- (high)-time PM measurements and how that variation can be characterized, and what 24-hour PM measurements mean in the context of such variation. (Commenter 12) **RESPONSE**: In contrast to the traditional 24-hour average PM concentrations available from routine PM sampling networks, monitoring methods such as TEOMs and BAMs can provide hourly averaged concentrations. The higher temporal resolution with these monitors greatly increases our understanding of the processes leading to high 24-hour PM concentrations. Hourly data enable better assessments of the impact of dust storms, fires, transport, etc. on ambient PM concentrations. These hourly data can provide additional insights not only into the diurnal variations but also into seasonal and spatial differences. Results of PM continuous data analysis indicate that many urban monitoring sites in California exhibit a diurnal pattern with concentrations peaking during commute periods and being lowest during the afternoon, a pattern similar to diurnal profiles for carbon monoxide (CO) and oxides of nitrogen (NO_X). Continuous particulate monitoring methods have been deployed in recent years. The hourly data from these methods provide additional insight into the nature of the particulate problem and reduce the uncertainties associated with less than daily sampling frequencies. A total of about 36 continuous PM2.5-mass monitors are expected to be deployed throughout California by some time in 2002 as part of the California continuous PM2.5-mass monitoring network. Of these, 21 are already committed. The primary objective of continuous PM2.5 mass monitoring is to obtain diurnally resolved data. These data will be useful for public reporting, understanding diurnal and episodic behavior of fine particles, background monitoring, and transport assessment. California has two ambient air quality standards for inhalable PM, one with a 24-hour averaging time and an annual average standard. The primary objective of the 24-hour PM mass monitoring program is to identify areas where PM concentrations exceed one or both of the national or State PM standards. The Board designates areas based on ambient air quality data. An area is designated nonattainment if ambient PM concentrations in that area violate either of the State standards at least once during the previous three calendar years. In addition to collecting data for determining attainment status with respect to the national standards, PM monitoring sites must also satisfy other monitoring objectives, including transport assessment and assistance in health studies. To meet these objectives, air basins with high PM concentrations may need to have additional high time/spatially resolved monitoring sites to provide better geographical and temporal representation.

CHAPTER 4: EMISSIONS INVENTORY

8. **COMMENT:** The emissions inventory material presented is based on estimated inventory data, not on actual measurements. The report should at minimum present some validation comparisons between estimated and measured data. The inventory presentation should also include natural sources and background levels of PM. (Commenters 11, 12) **RESPONSE**: A discussion of the

validity of emission inventory compared to monitored data is beyond the scope of the standards report. ARB staff update the emission inventory triennially as required by Health and Safety Code section 39607.3. Available data show that emission inventory data are generally in reasonable agreement with ambient measured data, but refinements are continuously being applied to improve how well inventory data reflects ambient levels of air pollution

9. **COMMENT:** Natural PM sources are not included in the statewide emissions inventory. They should be because they can be a significant part of the daily PM level. (Commenter 11) **RESPONSE**: The PM inventory does not include natural sources of particulate, other than wildfires, nor are there plans to include it. Except for fire, the main sources of natural particulates are due to windblown dust from natural undisturbed lands. To date, there is no indication that this source contributes significantly to PM standard exceedances. (Note: the majority of windblown dust in Owens Valley, Imperial County, Mono Lake and other windblown dust regions is considered anthropogenic in origin). Except in cases of wildfires, which are included in the ARB emissions inventory, natural sources do not typically contribute meaningfully to elevated particulate matter levels.

10. **COMMENT:** What is included in the "Fuel Combustion" category? (Commenter 11) **RESPONSE**: The Fuel combustion category includes stationary air pollution sources such as electricity generation, oil refining, agricultural processing, etc. This will be clarified in the text.

CHAPTER 5: MONITORING ISSUES

11. **COMMENT:** Studies of the loss of semi-volatile compounds raise a serious concern with the staff recommendation in Ch. 5 to adopt the FRM for PM2.5. (Commenter 12) **RESPONSE:** From a monitoring standpoint, the potential for loss of semi-volatile compounds in sampling is a recognized shortcoming of any filter-based sampling method. There is no solution for this available at this time. Instruments are in development that may allow in-situ measurement of nitrogen species, including particulate matter nitrates. It is not likely that such instruments, when they become available, will be widely deployed in the monitoring network. The staff report mentions that loss of volatile species may lead to control strategies that are biased towards sources of fugitive dust and other primary particle sources. It is important to note that the development of control strategies is not based solely on ambient measurements made with the PM2.5 FRM. Emission inventories, chemical speciation analysis results, and other information are used to develop control strategies.

CHAPTER 6: EXPOSURE

12. **COMMENT:** Table 6.1 should be changed because it includes data from the Salton Sea Air Basin that ARB invalidated because the monitor was not sited so as to meet the requirements for a valid monitoring site. (Commenter 8) **RESPONSE**: It is correct that ARB has invalidated the data, and the Table will be corrected in the next draft.

13. **COMMENT:** An analysis of 24-hour PM2.5 monitoring data, with emphasis on areas projected to be in attainment of the annual average standard, is not presented in the report. (Commenter 1) **RESPONSE**: There are only about 2 years of PM2.5 data available using the U.S. EPA's federal reference method. Therefore, it is not possible to perform an extensive analysis, or to have an understanding of historical trends or year-to-year variability. However, we will include the requested analysis in the next draft of the report.

14. **COMMENT:** Captions to tables and figures in Section 6.4 need to be revised to reflect that the data are the percent of the population residing in areas that exceed given concentration levels. These data do not reflect actual or personal exposure as represented by the captions and text. (Commenters 11, 12) **RESPONSE**: The data reflect population exposure to outdoor or ambient PM levels. We will change the term "percent of population exposed to given PM levels" to "percent of population exposed to given ambient PM levels".

15. **COMMENT:** What is the effect of geographic barriers, such as mountain ranges, on estimating exposure? If the monitor is on the other side of a mountain than the population of a census tract, how does this affect estimation of exposure? (Commenter 11) **RESPONSE**: While it is true that we did not consider natural boundaries, such as mountains, in our exposure model, the results are useful for the following reasons: The distribution of monitoring sites in the South Coast Air Basin is dense compared to the width of the significance boundaries; and the use of the inverse of the square of the distance from monitors to census tracts limits any undue influence of more distant monitors (e.g., on the other side of the mountains).

16. **COMMENT:** Air quality trend data for estimating chronic effects are inadequate. Historical trends are not adequately discussed, and should encompass the time period from the 1940's to the present. (Commenter 12) **REPONSE:** Ambient PM10 trends for California from 1988 are presented in the ARB's 2000 almanac of air quality and emissions data. PM2.5 data are only available since 1998.

17. **COMMENT:** Emission trends should be presented from the 1940's to the present. (Commenter 12) **RESPONSE**: Emission trends are presented in the ARB's 2000 almanac of air quality and emissions data.

18. **COMMENT:** There is no information discussing the differences between current and background levels for PM10, although this information is presented for PM2.5. (Commenters 9, 12) **RESPONSE**: The PM2.5 information was provided as general background information. The report contains the information needed to calculate difference between current and background PM10.

CHAPTER 6: BACKGROUND CONCENTRATION OF PM

19. **COMMENT:** The concentration of background PM is a substantial portion of the standard. This means that very little anthropogenic PM can be contributed and attainment still be achieved. This means that the recommended standards are more stringent than at first apparent. The commenters request that ARB do more background PM analysis. They also challenge the use of Point Reyes National Seashore as a representative background site. (Commenters 11, 12) **RESPONSE**: We will expand our discussion of background sites. Background sites are intended to quantify regionally representative PM concentrations for sites located away from populated areas and other significant emission sources. Background concentrations for the PM2.5 program are defined as concentrations that would be observed in the absence of anthropogenic emissions of PM and the aerosol particles formed from anthropogenic precursor emissions of VOC, NO_X and SO_X. Sources of background PM include particles of soil and crustal material, organic particles from natural combustion processes such as wild fires, and organic aerosols formed from VOC emissions from vegetation. In addition, natural emissions of gaseous sulfur compounds contribute to the background sulfate component. However, it is very difficult to find true background sites. Depending on the season and meteorological conditions, even the monitoring sites located in pristine areas can be influenced by anthropogenic emissions and transport. This in turn may lead to higher annual average PM concentrations. Annual average PM concentrations from the IMPROVE network are presented in the table below (aggregated over a three year period, March

1996 to February 1999). We agree that different sites (e.g., a site for a desert locality, one in a forested areas, etc.) should be considered for determining regional background PM concentrations.

Site	Annual Average PM10 ($\mu g/m^3$)	Annual Average PM2.5 ($\mu g/m^3$)
Lassen Volcanic NP	5.06	2.68
Pinnacles NM	10.97	4.55
Point Reyes NS	12.42	4.01
Redwood NP	7.45	2.44
San Gorgonio WA	13.72	7.20
Sequoia NP	18.64	8.86
Yosemite	9.52	4.33

The comments mention that U.S. EPA is proposing to use a range of PM10 background of 4-8 $\mu g/m^3$ in the western U.S.

CHAPTER 6: FORM OF THE STANDARD/ ATTAINMENT DESIGNATIONS

20. **COMMENT:** It is important to consider the form of the standard and whether or not it leads to reasonable standards for attainment. (Commenter 11) **RESPONSE**: Under California law, criteria for attainment designation are not part of the ambient air quality standards. Attainment criteria are specified in a separate section of the California Health and Safety Code. The form of a standard defines a calculation using air quality data. The result of the calculation is often called the "design value". The California design value for standards with an averaging time of 24 hours or less is called the Expected Peak Day Concentration (EPDC). The degree of fluctuation for the EPDC is similar to the degree of fluctuation that affects design values based on the percentile-averaging procedure. Because California's 24-hour design value has fluctuations similar to the form recommended by the commenter, the proposed CA standard for 24-hour PM10 would have a similar relationship between the "perceived" and the "actual" stringency as does the commenter's recommended procedure.

    The comments do not accurately portray the in-use behavior of California's procedures. Experience with real-world data shows that the worst year is not inappropriately represented. In fact, the worst year, meteorologically speaking, typically receives all or most of the exclusions in any three-year period. The commenter's comments show that they are focusing on the false dichotomy between "expected exceedance" and "concentration based" forms for standards. The California form for the 24-hour standard integrates both of these concepts simultaneously; it is a concentration-based calculation that achieves an expected exceedance criterion. No stringency is added. No distortion is introduced between the specified level of the standard and the long-term concentration levels required to attain the standard. The performance of the California form (EPDC) already has a proven track record. It is stable, not volatile. It addresses a "one expected exceedance per year" objective. The commenter may argue for more allowed exceedances, but this should be done from a health basis, not a statistical basis. If more were allowed, a lower level standard might then be appropriate to achieve equivalent protection.

21. **COMMENT:** Criteria for attainment of the standard are unnecessarily stringent. (Commenter 11) **RESPONSE**: With reference to the 24-hour standards, see #16 above. If it becomes clear that using the highest annual average in the last three years is unnecessarily stringent; California statutes permit the form of the standard to be altered without requiring a full reevaluation of the

standard. Because annual averages do not fluctuate greatly from year-to-year (as the commenter notes elsewhere) it is not advisable at this time to alter the form of the proposed annual standard.

22. **COMMENT:** The method of determining compliance should be changed to that used by the U.S. EPA. (Commenters 11, 12) **RESPONSE**: The response to this comment is similar to an earlier comment (#20 and 21). Bounce is small for annual standards, even based on the maximum annual average in 3 years. If we learn that the average in 3 years (rather than the maximum) is protective, California can alter the form of the standard without requiring a complete reevaluation of the standard. When Health and Safety Code Section 39607 (e) was enacted, it separated the standard-setting and risk management functions. Federal rules make USEPA consider these all at once.

CHAPTER 6: EPDC

23. **COMMENT:** The exponential distribution of data used in calculating the EPDC tends to have a long tail, making the predicted "99.7th" percentile an unrealistically high extreme value. (Commenter 11) **RESPONSE**: The tail is not too long, as the commenter asserts. If the tail of the exponential distribution were not appropriate, the number of measured values above the calculated cutpoint (the EPDC) would be too low, that is, less than one per year on average. Annual reports concerning attainment designations show that the EPDC procedure works very well for PM10 when the 1-in-6 day sampling schedule is considered. Therefore, the tail is not too long.

24. **COMMENT:** The EPDC is an estimate of the maximum value in three years, it does not achieve the stated goal of "determining the peak 24-hour PM10 (or PM2.5) concentration expected to occur no more than once per year", and leads to hidden stringency. (Commenter 11) **RESPONSE**: This is not a correct characterization of EPDC. More than a decade of data shows that the commenter's contention is incorrect. The EPDC procedure automatically corrects for less than daily sampling frequency. No penalty results when samples are gathered less frequently than every day.

CHAPTER 6: THE CONTROLLING STANDARD

25. **COMMENT:** Currently the 24-hour standard is the controlling standard. If the staff recommendations are adopted, the new annual average PM10 standard will be approximately as stringent as the current 24-hour standard. Therefore, the driving force for regulation will be essentially unchanged. (Commenter 12) **RESPONSE**: This comment concerns the probable relationships between multiple standards, annual and 24-hour, for PM10. The reviewer correctly understands these relationships, and offers an alternative approach that relies on a 24-hour standard alone. The ARB staff discussed whether the multiple standards are useful and concluded that both the annual and 24-hour standards were useful, even if they were approximately equal in stringency. Policy and scientific issues that led to this conclusion include the following: (1) Some health scientists consider the annual PM data to be most reliably related to mortality, motivating an annual standard. (2) Air quality data clearly show that an annual standard alone would still admit some troublingly high PM concentrations for 24-hour periods during the year. (3) Though the annual and 24-hour standards would be approximately equivalent from a statewide viewpoint, areas with different PM composition are likely to show that each standard is controlling in some areas of California.

CHAPTER 6: LACK OF A PM2.5 STANDARD AND EXPOSURE

26. **COMMENT:** Having only an annual PM2.5 standard is not sufficient to protect against short-term PM2.5 peaks. (Commenters 1, 5, 6, 17) **RESPONSE**: The commenter is correct that PM2.5 levels could reach as high as the level set for PM10 (50 $\mu g/m^3$ at this point) if all the PM10 were in the form of PM2.5. The form of the CA standard for air quality measurements with averaging times of 24 hours or less is effectively the 364/365th, or the 99.73rd percentile, as only one day per year, on average, can be above the level set by the PM10 standard. The present form of the EPA 24-hour PM2.5 standard is based on the 98th percentile with a level of 65 $\mu g/m^3$. Clearly, the standards proposed for CA are much more protective. As proposed, the PM2.5 peaks would be controlled (limited) by the PM10 standard. The implied limit for PM2.5 is somewhat different for different areas of the state, depending on the fine versus coarse fractions of PM10. Nevertheless, the implied limit is less than 50 $\mu g/m^3$ throughout CA.

27. **COMMENT:** The short-term PM10 standard will not prevent short-term fine particle peaks in some areas where PM10 and fine particles are not highly correlated. (Commenters1, 6) **RESPONSE**: The 24-hour PM10 standard will restrain 24-hour PM2.5 concentrations to the same level as the level set for PM10. At this time the proposal sets the 24-hour PM10 level at 50 $\mu g/m^3$. Therefore, it is at least as protective as a PM2.5 standard set to 50 $\mu g/m^3$. However, because PM10 will include some coarse component, the proposed standard is more protective than a PM2.5 standard of 50 $\mu g/m^3$ would be by itself. The USEPA 24-hour PM2.5 standard is currently 65 $\mu g/m^3$.

28. **COMMENT:** Comments on the relative merits of the two methods suggested at the January 23 and 24, 2002 AQAC meeting for selection of a 24-hour PM2.5 standard. (Commenter 21) **RESPONSE**: These comments have been considered in development of the recommended 24-hour PM2.5 standard.

CHAPTER 6: SOURCE APPORTIONMENT

29. **COMMENT:** The source categories on the source apportionment charts in section 6.3.2 are inconsistent. (Commenter 11) **RESPONSE**: In the report, we explain that the source attribution data presented in the report were derived from a variety of studies with differing degrees of chemical speciation. Therefore, the source categories presented may be different among sites. For example, the fossil fuel combustion category is only presented for San Jose. As mentioned, this category included motor vehicles, refineries, and power plants. Throughout section 6.3.2 of the report we state that secondary ammonium nitrate is formed in the atmosphere from nitrogen oxides from motor vehicle exhaust and other combustion sources. We will further clarify that nitrogen oxides are from motor vehicle exhaust and other stationary combustion sources. We will add that sources of ammonia include animal feed lots, fertilizer application, and motor vehicles.

30. **COMMENT:** Section 6.3.1.4: wording changes and addition of 2 sentences are recommended. (Commenter 11) **RESPONSE**: We will add the following sentence at Pg. 57, line 5 to the next draft of the report: "The quality of source apportionment results depends on the adequacy of the chemical markers used for each potential source and of the ambient chemical composition data used in the analysis, as well as the inclusion of appropriate sources".

31. **COMMENT:** Section 6.3.2: The pie charts in this section need more explanation. (Commenter 11) **RESPONSE**: The temporal differences among the data presented on the source apportionment and on the ambient chemical composition pie charts is already indicated on the charts themselves.

In addition, we will specify if the data represent annual or seasonal averages, or averages of a few days in the text describing the data presented on each pie chart in the next draft of the report.

CHAPTER 6: INDOOR AND PERSONAL EXPOSURE

32. **COMMENT:** Add discussion of data variability from continuous monitoring sites, and use of 24-hour central monitoring site results as a surrogate for human exposure. (Commenter 12) **RESPONSE**: We have limited information on the diurnal variations of PM; two examples are presented in the report. We are in the process of deploying the State's network of continuous PM monitors, which will provide further data on diurnal variations in PM levels.

33. **COMMENT:** Section 6.5 should include discussion of the link between weather and indoor exposure, the effect of increasing air turnover in buildings, and building ventilation. The association between outdoor PM and health are confounded by exposure to indoor air pollutants. (Commenter 12) **RESPONSE**: It is true that indoor-outdoor differences in temperature and pressure (due to wind and mechanical ventilation) create pressure differences that affect AERs in buildings. For example, during mild, stagnant weather conditions the AERs can be very low, even in a home with open windows and a leaky building shell. This is because significant driving forces for infiltration are lacking. However, stagnant weather is not the norm.

    Additionally, the human factor plays a significant role. People use their home's windows, doors, and mechanical systems for heating, cooling, and ventilation, which can greatly modify the building's pressure characteristics. This can increase AERs, and hence, result in increased correlations between indoor and outdoor PM levels.

    Opening of windows and doors typically increases AERs. It can also increase the deposition of outdoor PM indoors and potential indoor resuspension of PM over long periods of time. Questionnaire data from ARB's adult activity pattern study showed that, on average, about one-third of Californians leave a door or window open all day, and 70% open a door or window for at least a few minutes per day (other than to enter or exit the home).

    Using mechanical ventilation systems can increase AERs. Whole-house fans, which are fairly common in much of California, can quickly equilibrate indoor and outdoor air in a home. Central heating and cooling systems can increase AERs when the pressure is imbalanced because of substantial duct leakage, which is fairly common. ARB's activity pattern study data indicated that about one-quarter of Californians use some type of fan, on average, to circulate the air. Operation of indoor ceiling or floor fans can resuspend surface PM, which may largely derive from outdoors.

    One caveat in reviewing AER data is that the 24-hour or multi-day averages may underestimate the AERs when people are actually home. These data may include large stretches of time when the house is vacant and closed up while the household members are working, attending school, and so on. These periods would have lower AERs that would reduce the average AER.

    Thus, a building's AERs, PM penetration rates, and indoor PM levels are in part dependent on weather, but in a complex manner that involves several other factors, such as window and door opening, that may not have linear relationships. For example, some of the highest outdoor PM levels in California occur during the fall season when the weather is relatively mild. In this season, cooling can usually be achieved by window opening and whole-house fans rather than air conditioning, which produces higher AERs than if one assumed that air-conditioning was used. This may help explain why outdoor PM levels had a substantial contribution to indoor PM in PTEAM homes during the Fall season.

It is unclear what is meant by the statement that regarding indoor pollutants as potential confounders of the outdoor – PM associations. Does this refer to pollutants of indoor origin, or indoor levels of pollutants? As stated in chapter 6, about ½ to the indoor PM mass comes has been estimated as coming from outdoor sources, and once indoors, some of that PM is available for resuspension, regardless of the day to day increases or decreases of AERs. As explained in Chapter 6, the correlation of personal exposures to ambient PM is variable but has been found to be substantial in more recent exposure studies with a longitudinal study design and in those focused on PM2.5. Thus, the ambient PM – health effects relationships seen in epidemiology studies that form the basis for the PM standard recommendations are robust despite the added exposure that may accrue from pollutants of indoor origin.

34. **COMMENT:** Ambient PM concentrations are not representative of actual personal exposure. People spend most of their time indoors. Use of outdoor PM concentrations to estimate exposure leads to confounded results and conclusions because of failure to consider indoor exposures. It should be assumed that indoor pollutants are potential confounders of the outdoor-PM associations until proven otherwise. (Commenters 11, 12) **RESPONSE**: We do not concur that indoor air pollutants are necessarily confounders. The major source of indoor PM, tobacco smoking, is usually adjusted for in epidemiological studies of outdoor PM. Other indoor air pollutant exposures that might affect the outdoor PM-health relationship, such as cooking emissions, do not introduce a known bias because they are not necessarily correlated with outdoor conditions. The relationship seen between outdoor PM and health effects in epdiemiology studies has been consistent across studies in different seasons and different meteorological conditions. We agree in part with the comment that buildings provide a level of protection against outdoor PM. This level of protection is highly variable, especially in the wide range of California's climate and building stock. The report will be revised to include an expanded discussion of the physical processes and human activities that affect the relationships among person, indoor, and outdoor PM concentrations.

35. **COMMENT:** Definitions of outdoor and ambient air are not consistent in the literature cited. Report should clarify these potential confusions. (Commenter 11) **RESPONSE**:**:** We agree. There is no regular distinction used for these terms. These terms are used differently, and often interchangeably in the general air pollution field, although in the personal exposure field, ambient usually refers to measurements at central monitoring station. Therefore, the distinction between "outdoor" and "ambient" is usually based on the scale over which the measurements are considered to be representative; however, this varies in relation to meteorological and other factors. Definitions of how these terms are used in various studies will be clarified in the report, where feasible, to make these distinctions more clear.

36. **COMMENT:** Section 6.5: The section contains internal contradictions. (Commenter 11) **RESPONSE**: The first portion of the comment addresses findings from one study of 30 individuals with COPD in Los Angeles (Linn et al., 1999). The conclusions noted in the comment are those of Linn et al., not ARB. In this study, the investigators examined blood saturation, blood pressure, and lung function, not mortality, as health endpoints. As indicated in the text, the findings regarding blood pressure were stronger for PM at the ambient monitoring station than for indoor or personal PM, and this is likely the basis for Linn et al.'s conclusion that ambient PM was linked to the health effects seen. The findings of this study apply to one small, sensitive segment of the population, and are not necessarily relevant to the health endpoint (daily mortality) upon which the level of the proposed standard is primarily based. The Linn et al. study was included in the report for completeness; it does not attenuate the credibility of findings of studies that identified

relationships between ambient levels of PM2.5 or PM10 and other observed health effects (e.g., daily mortality).

37. **COMMENT:** Section 6.5 does not include some of the available data on indoor/personal exposure. Several references are recommended. (Commenter 11) **RESPONSE**: We have reviewed the suggestions, and incorporated appropriate references.

38. **COMMENT:** Air conditioning use effects on past exposures should be considered in estimating past PM exposure. (Commenter 12) **REPONSE:** Most of the epidemiological studies in the U.S. have used data from the 1970's and later, and air-conditioning was already widely used in California and much of the U.S. by the 1960's. Therefore, past air-conditioning usage should not affect the results of these epidemiological studies.

39. **COMMENT:** There is no discussion of the personal cloud. (Commenter 12) **RESPONSE**: Section 6 will be modified to include such a discussion.

40. **COMMENT:** There is no discussion of the level of protection provided by buildings. (Commenter 12) **RESPONSE**: We agree that mechanical ventilation can affect indoor PM; this topic was included in the report. The report will be revised to expand the discussion of the effects of mechanical ventilation systems on indoor-outdoor air exchange. However, these effects do not alter the association observed between PM measured at ambient stations and the adverse health effects seen in the population. This is likely due to the relatively short time (6-7 hours) during a 24-hour period that people actually spend in office buildings, schools, and other large buildings with mechanical ventilation. Additionally, older individuals and those with serious illness do not generally spend time in such buildings.

41. **COMMENT:** Recent findings raise the issue of whether short-term peak exposures are more important than 24-hour or long-term exposures. In addition, the significant PM2.5 and PM10 exposures from indoor sources and personal activities represent a significant potential confounder. Because exposures to indoor particles are usually as large or larger than exposures to outdoor particles, indoor particles may represent a separate risk of equal or greater magnitude than ambient PM. (Commenter 12) **RESPONSE:** We agree that exposure to particles of indoor origin likely presents a separate risk of great magnitude. However, it is not the purpose of this document to address this specific issue. The available data on short-term or real-time exposures to indoor PM are currently very limited, but major studies on this topic are in progress. The potential risk from indoor PM is not really a confounder of the outdoor PM-health effects association seen in past epidemiological studies. As seen in recent longitudinal exposure studies, outdoor PM levels and personal PM exposure levels do correlate from day to day in a substantial portion of the population. This is not surprising, since about 2/3 of indoor particles are of outdoor origin, on average, as discussed in the report.

42. **COMMENT:** The Draft should discuss indoor and outdoor bioaerosols, especially the Cal Tech study (ARB, 1998). (Commenter 12) **RESPONSE:** The Cal Tech study examined the composition of allergens in roadside dust, and the contribution of those allergens to outdoor PM. Roadside dust can infiltrate or be tracked into buildings. It is acknowledged that both indoor and outdoor allergens are present in the air and in the indoor surface dust that can be resuspended. These allergens contribute to the allergy symptoms and asthmatic attacks in individuals. However, the relationship of roadside dust to indoor and personal exposure has not been well studied. The report discusses the various sources of biological contaminants in indoor PM, and it will be revised to include the findings of the Cal Tech study regarding outdoor PM.

43. **COMMENT:** Resuspension of large particles (>1 µm) "complicate and confound the analysis of exposures and health." (Commenter 12) **RESPONSE:** We agree that resuspension of particles can influence their contribution to indoor concentrations. However, this contribution does not bias the exposure-health effect studies because house dust largely consists of outdoor PM that has been transported indoors by air or track-in. Emissions from indoor resuspension are mainly dependent on human activities such as cleaning and moving about, and therefore would be expected to be independent of daily outdoor PM levels, and thus would not confound the correlation seen between ambient PM and adverse health effects.

44. **COMMENT:** "Because of the public policy implications of nitrate reduction, the Draft should discuss the subject (of indoor nitrate volatilization) in detail…". (Commenter 12) **RESPONSE**: Compared to ambient monitoring methods, the indoor, outdoor, and personal monitoring methods use lower flow rates, and the samples are usually collected immediately after 12 or 24 hours of sampling. Therefore, indoor sample losses of nitrate are expected to be minimal. A few laboratory and test house studies on this topic have been conducted, but field studies that examine nitrate composition of indoor, outdoor, and personal PM2.5 are currently underway. Concerning nitric acid deposition on indoor surfaces, it is not clear how important a nitrate removal mechanism this is in California buildings. Nitric acid can oxidize to form other volatile nitrogen oxides indoors, or perhaps react with indoor surface dust and indoor air pollutants to produce toxic or irritant pollutants. More research is needed in this area.

45. **COMMENT:** The commenter disputes the PTEAM results/conclusions presented. (Commenter 12) **RESPONSE**: The report does not state that indoor and outdoor PM are uncorrelated, but rather that higher correlations between outdoor and personal PM were obtained in longitudinal studies, as compared to correlations in cross-sectional studies such as PTEAM. The PTEAM investigators did find low indoor-outdoor correlations, however, despite the high air exchange rates. The report will be revised to clarify the indoor-outdoor correlations in PTEAM. The air exchange rates may have been higher than reported in some studies of homes, but are within the range observed in California's South Coast Air Basin. In this region and much of California, the milder climate encourages the use of open windows, whole house fans, and swamp coolers, except for the occasional heat wave when air conditioning may be used.

46. **COMMENT:** There is no information presented on the most frail sub-population, those in hospitals and nursing homes. (Commenter 12) **RESPONSE:** The report discusses the available studies regarding indoor and personal PM exposures of the elderly and ill. The report will be expanded to include the Lillquist et al. study, which measured indoor PM10 in 3 Utah hospitals, mostly in intensive care units that had extensive air filtration. However, this study showed that indoor-outdoor PM relationships were highly variable among the 3 hospitals and within each hospital.

CHAPTER 6: 24-HOUR PM2.5 STANDARD RECOMMENDATION

47. **COMMENT:** The relationship between the annual mean and the annual maximum implies that the annual average must be at or below the "background" level for PM2.5. (Commenters 13, 14) **RESPONSE**: The relationship between the annual average and the annual maximum reflects the influence of changing weather conditions and, to a lesser extent, changes in human activities. As emission control measures reduce the pollution generated by human activities, the ratio of the maximum to the average tends to decrease somewhat. Nevertheless, the Cal/EPA staff believes that the ratio is unlikely to be less than 2.5 when regions near attainment of the proposed 24-hour standard. The proposed 24-hour standard of 25 ug/m3 probably does imply an annual average between 8 and 10 ug/m3, which may be at or near "background" levels for PM2.5. Under such circumstances, the 24-hour standard would be the so-called "controlling" standard. That is, the

annual standard (12 ug/m3) would be met while the 24-hour standard still required additional emission reductions. Accordingly, the staff agrees with the commenter's statement that "the proposed 24-hour standard of 25 ug/m3 is considerably more stringent than the proposed annual standard of 12 ug/m3." The larger issue, however, is what an air quality standard represents. An air quality standard is meant to identify a concentration and averaging time that is "safe" for people to breathe. Whether such a standard can be "attained," is a different issue, an issue of risk management. Under California law, the risk management function is separated from the determination of an air quality standard in two ways -- through criteria for attainment and through planning requirements. Under CA law, ambient air quality standards are based solely on health and welfare considerations. There is no consideration as to whether the standard is attainable at any foreseeable time. In this sense, standards serve as goals for the air quality planning process.

Criteria for attainment

Small adjustments to the stringency of an air quality standard can be accommodated through modifications to the criteria for attainment. These criteria are not an intrinsic part of the standard under California statutes. However, criteria for attainment have been determined with an eye toward maintaining the health-protective nature of AAQ standards.

Planning requirements

The commenters assertion is that the proposed standard is not feasible, not attainable. Planning requirements in CA statutes recognize that one cannot do more than what is feasible. A plan containing all feasible measures is a satisfactory attainment plan. Therefore, draconian plans containing infeasible control measures would not be required by the proposed 24-hour standard for PM2.5.

48. **COMMENT:** A standard that is "not to be exceeded" imposes an unattainable goal, especially when concentrations must be very near background levels. (Commentors 14, 15) **RESPONSE**: The term "not to be exceeded" does not set implicit criteria for attainment. Criteria for attainment are set under the requirements of Section 39607(e) in the Health and Safety Code. Various AAQ standards that include the "not to be exceeded" language are attained under these criteria when the expected annual maximum equals the standard. The method used to compute the expected annual maximum (Expected Peak Day Concentration) is not subject to the large fluctuations anticipated by the commenters.

    The issue of "background" concentrations and attainability is primarily related to the level of the proposed standard (25 ug/m3) rather than the form of the standard. If the form of the standard were to be based on the measured annual maximum, the concerns raised by the commenters would be very appropriate. However, the default form of the proposed standard does not lead to these concerns.

CHAPTER 7: STUDIES USED FOR ANALYSIS

49. **COMMENT:** The Report did not review all studies, and the review was not objective for those studies that were reviewed. (Commenters 3, 4, 12) **RESPONSE:** The review covers hundreds of studies to address two key questions: (1) is there evidence of gravimetric PM10 and/or PM2.5 effects at or below current standard? (2) how strong is this evidence? The commenters suggest some specific studies that they feel should have been added. Some studies were not included because they did not include size-selected gravimetric particle exposure data. The other studies that were citied are discussed in the following paragraphs.

In the case of acute mortality outcomes, several Canadian studies (e.g., Burnett et al., 1998a,b) did not include PM10 measurements. In Burnett et al. (1998a) mortality was studied across 11 Canadian cities. However, PM10 was not measured. Burnett et al. (1998b) did not include PM10 measurements but rather estimated PM10 using TSP, SO4, and COH data. This makes the results difficult to interpret in terms of PM10. Furthermore, several of the Canadian studies reported high correlations between PM and gaseous pollutants, making it difficult to separate out the effects of different pollutants. The degree to which the various pollutants were acting as surrogates for one another cannot be discerned from these results. Zmirou et al. (1998) reported results of a large multi-center study of acute mortality in 10 European cities. PM10 data were not available. Black Smoke, a measure of optical absorbance of the aerosol, was used instead. In addition, given the locations and period of study – the data records ended in 1992 – it is unclear how to relate these exposure data to gravimetric PM measurements in the U.S. Particle sources and composition were likely to have varied substantially across cities; likewise, those cities as a group are likely to differ from the situation in the U.S. In any event, Zmirou et al. reported associations of both PM and SO_2 with mortality.

The Lipfert et al. (2000b) study results are now included in the PM document draft. As discussed in detail in the document, this study reports results and conclusions very different from previous studies, but there appear to be methodological differences that can account for these results. Results more similar to those obtained in the major cohort mortality studies were found when more conventional methods of analysis were used. There are two major issues with this analysis conducted by Lipfert et al. (2000b): (1) these researchers used highly specified, and likely over-specified, models that may have underestimated pollution effects, and; (2) these researchers used very localized (county level) and short-term segmented exposure data that may have introduced exposure estimation errors.

With regard to the first issue, the potential for model over-specification (described below) and resultant effect estimate bias is indicated by the authors' own results for smoking effects on mortality, which are apparently lower in this analysis compared to other studies. As noted by the authors: "The risk of current cigarette smoking (1.43) was somewhat lower than has been reported elsewhere, but other studies have not accounted for as many additional factors" (Lipfert et al., 2000b, p. 52). This suggests that over-specification is likely to be occurring in these models, potentially biasing the pollutant effect estimates downward, as well.

With regard to the second issue, Lipfert et al. (2000b) note that they obtained results closer to those reported by other researchers when using methods similar to those used by the others, rather than using the time-segmented approach. They state: "Responses to PM2.5 and PM15 differ greatly between the single period and the segmented periods…. The single-mortality-period responses without ecological variables are qualitatively similar to what has been reported before…" (Lipfert et al., 2000b, p. 68).

Thus, while this new cohort study gives results at variance from previous studies, there appear to be methodological issues that may account for these differences. When methods similar to studies published in the New England Journal of Medicine (Dockery et al., 1993) and the Journal of the American Medical Association (Pope et al., 1995) were used, Lipfert et al. (2000b) indicate that the results are similar to those published previously.

50. **COMMENT:** Consistency of results across studies and coherence of results across outcomes is limited. (Commenters 3, 12) **RESPONSE:** The most consistent aspect of the acute epidemiology results is the identification of statistically significant PM effects on mortality in a large number of studies conducted in over 20 cities in the U.S. as well as many in other countries. Not

surprisingly, risk coefficients reported from different locations vary somewhat. This may relate to variations in pollutant mixes, population characteristics, and analytic methodologies across the wide range of studies reported to date. As a group however, the acute mortality studies and, to a lesser extent, the morbidity studies present a consistent picture regarding the effects of PM on health.

The consistency of results among scores of epidemiological studies provides substantial evidentiary support for causality. Several hundred studies, conducted among different populations on five continents over multiple time periods, have reported small, but consistently elevated risks of daily mortality and diverse measures of morbidity (such as hospital admissions and emergency department visits for cardiac and respiratory causes, exacerbation of asthma, increased respiratory symptoms, restricted activity days, school absenteeism, and decreased lung function). Though the principal study design has been time-series analysis, modeling approaches have differed substantially among investigators; moreover, similar estimates of effect have been obtained with other study designs, including case-crossover and panel studies. The ranges of risk estimated in all these studies have been remarkably similar, despite the different PM source mixtures and size distributions, co-pollutant distributions, weather patterns, population characteristics (distributions of age, baseline health status, and access to health care; see Section 7.3, for example). Daily mortality and morbidity have also been linked with different measures of PM, as well, including TSP, PM10, PM2.5, the coarse fraction (PM10-PM2.5), black smoke, and ultrafine particles. It can be seen in Table 7.1 and Sections 7.3 through 7.6 that, with few exceptions, there is a consistent tendency for point estimates of relative risk to be greater than unity. If these findings were due to chance, one would expect a more nearly equal distribution of point estimates of risk above and below unity. In general, consistency of results across scores of investigations offers one of the strongest arguments favoring a causal relationship.

Coherence is considered to be present where there is evidence showing similar patterns of results for different health outcomes associated with a given pollutant. Strong evidence of coherence exists across the epidemiologic literature for PM. For example, PM has been associated with both mortality and hospital admissions in nearly 30 cities worldwide, more than 20 of which are in the US. As noted in several EPA scientific reviews, the effect sizes for total mortality generally fall in the range of 2.5 to 5.0% excess deaths per 50 $\mu g/m^3$ 24-h PM10. Similar effects are seen for cause-specific cardiovascular and respiratory mortality. Hospital admissions would be expected to exhibit larger effect sizes than those from mortality, and this is seen in the literature, where cardiovascular admissions increase from 3 to 6% per 50 $\mu g/m^3$ 24-h PM10 and respiratory admissions increase from 5 to 25% per 50 $\mu g/m^3$ 24-h PM10). Effects have also been observed in several panel studies by independent investigators, where elderly subjects are followed over time to assess changes in heart rhythm in association with ambient PM. The observed decreases in heart rate variability are consistent with increased risk of adverse cardiac events. A recent study (Peters et al., 2001a) went further and was able to demonstrate an association between both PM10 and PM2.5 and the onset of myocardial infarction. Thus, a coherent picture has emerged from a variety of different epidemiological approaches showing adverse effects of PM exposures among human populations.

Referring in particular to the time-series studies of mortality, Bates (1992) has argued that, if the PM-mortality relationship is causal, there should also be evidence of relationships between PM and health outcomes of lesser severity, such as hospitalizations, changes in lung function, and so forth, suggesting an ensemble of coherence among possible outcomes. This phenomenon has been observed in a number of areas throughout the world; perhaps the best illustration of such coherence in a given area is the studies undertaken in the Utah Valley. In addition to increases in PM-associated mortality, studies in this area have demonstrated statistically significant

relationships between ambient PM and respiratory hospitalizations, decrements in children's lung function, school absenteeism, respiratory symptoms, medication use among asthmatics, increased heart rate and decreased heart rate variability among elderly individuals (Pope, 1996; Pope et al., 1999a,b). Finally, there are over twenty cities in which associations between PM10 and both mortality and hospital admissions have been reported.

51. **COMMENT:** A fundamental limitation of the time series studies is their ecological nature. (Commenter 3) **RESPONSE:** The potential for "ecologic bias" is greatest in cross-sectional studies where it may be difficult or impossible to measure and control for potential geographic confounders such as cigarette smoking or income. In this case, all residents are often assigned countywide variables and assumed to have this common feature. This is the classical case of potential ecological bias. However, we note that we did not rely on any purely cross-sectional studies in our determination of likely concentrations associated with health effects. Rather, we used either prospective cohort studies or time-series studies. The prospective cohort studies control for potentially important individual-level risk factors, such as smoking, alcohol consumption, body mass index, educational status, occupational exposure, etc. Specifically, for most of the important risk factors associated with mortality, individual, nonecological data are used. In the time-series design, these concerns are largely eliminated since a single community is studied over time. Most potential confounders, such as smoking rates, are unlikely to vary from day to day in concert with air pollution levels. Potential confounders in the time-series design include weather factors, seasonality, and co-pollutants, all of which are carefully handled in much of the recent literature. Therefore, these studies are unlikely to suffer from ecological bias.

52. **COMMENT:** An important long-term exposure study by Lipfert was not adequately discussed. (Commenter 3) **RESPONSE:** We have now added a discussion to the document (section 7.4) about this study. Specifically, we have indicated that Lipfert et al. (2000b) recently reported preliminary results from a prospective cohort study of some 70,000 men enrolled by the U.S. Veterans Administration (VA) during the 1970s. This cohort is much smaller than the ACS cohort, and is made up of members who are not necessarily representative of the general population: the cohort was male, middle-aged (51 $\pm$ 12 years) and included a larger proportion of African-Americans (35%) than the U.S. population as a whole, as well as an extremely high percentage of current or former smokers (81%). Also, the cohort was selected at the time of recruitment as being mildly to moderately hypertensive, with screening diastolic blood pressure (DBP) in the range 90 to 114 mm Hg (mean 96, about 7 mm greater than the U.S. adult population average) and average systolic blood pressure (SBP) of 148 mm Hg. In addition, there were no extensive data collection forms to provide systematic information on such things as the presence of other risk factors (for hypertension) (Perry et al., 1982).

    In the air pollution analysis by Lipfert et al. (2000), pollutant levels of the county of residence at the time of entry into the study were used for analyses versus levels at the VA hospital area. While the use of monitors close to the subjects' residences at the start of the study theoretically might provide better exposure estimates than metro-area averages used in other studies, it may also have introduced exposure estimation error due to limited numbers of sites for each county, and possible residence changes within a metropolitan area over the years. Contextual socioeconomic variables were also assembled at the ZIP-code and county levels. The ZIP-code level variables were average education, income, and racial distribution. County-level variables included altitude, average annual heating-degree days, percentage Hispanic, and socioeconomic indices. Census tract variables included poverty rate and racial distribution. Countywide air pollution variables included TSP, PM_{10}, $PM_{2.5}$, PM_{15}, $PM_{15\text{-}2.5}$, SO_4, O_3, CO, and NO_2 levels at each of the 32 VA clinics where subjects were enrolled.

In addition to considering average exposures over the entire period, three sequential mortality follow-up periods (1976-81, 1982-88, 1989-96) were also considered separately in statistical analyses, which evaluated relationships of mortality in each of those periods to air pollution in the preceding, concurrent, or subsequent periods. The preliminary screening models used proportional hazards regression models to identify age, SBP, DBP, body mass index (BMI), age and race interaction terms, and present or former smoking as baseline predictors, with one or two pollution variables added. In the final model using 233 terms (of which 162 were interactions of categorized SBP, DBP, and BMI variables with age), the most significant nonpollution variables were SBP, DBP, BMI, and their interactions with age, smoking status, average ZIP education, race, poverty, height, and a clinic-specific effect.

The large number of "control" variables may well have led to over-specification of the study models, which could, in turn, cause underestimation of the effects of other risk factors (e.g., for pollution). Indeed, even the smoking effect on mortality in the Lipfert et al. study (2000b) is smaller than in other studies: "The risk of current cigarette smoking (1.43) was somewhat lower than has been reported elsewhere, but other studies have not accounted for as many additional factors" (Lipfert et al., pg. 52). This is a red flag that over-specification of the regression models (i.e., by including too many predictor variables) may have occurred, potentially biasing the pollutant effect estimates downward, as well.

The study's choice of pollutant exposure averaging times may also be the source of differences in relation to other studies. While the PM analyses considering segmented (shorter) exposure time periods gave unstable and differing results (including significantly negative mortality coefficients for some PM metrics), when methods consistent with those utilized in other studies were used (i.e., multi-year average PM concentrations), the authors reported that "(t)he single-mortality-period responses without ecological variables are qualitatively similar to what has been reported before (SO_4= > $PM_{2.5}$ > PM_{15})." Thus, methodological differences between Lipfert et al. (2000b) and the other major cohort studies may well be responsible for the different findings and conclusions reported by these authors.

CHAPTER 7: STATISTICAL MODELS

53. **COMMENT:** The assessment of co-pollutant effects is flawed. To be valid, studies must use multi-pollutant models. However, in many cases, where multi-pollutant models are used, PM coefficients decrease, suggesting no real effect from PM. The Report states there was no association between the effect estimates for each of the cities and the mean level of PM or other pollutants in the NMMAPS analysis of co-pollutant interactions. This is false. (Commenters 3, 12, 15, 18) **RESPONSE:** Understanding the role of co-pollutants as independent risk factors for acute mortality and morbidity outcomes is very important. Whereas in the past much of the epidemiological work focused largely or exclusively on PM, more recently many investigators have specifically addressed this issue by including other pollutants in the analyses. While a precise understanding of the relative impacts of PM and co-pollutants remains elusive, enough evidence currently exists to reach the following conclusion. Although gaseous pollutants such as ozone, CO, NO_2, and SO_2 are often associated with adverse health outcomes, the most consistent associations observed in the epidemiological literature are those involving PM. In studies including multiple pollutants in the analysis, PM has usually emerged as the most robust predictor of daily health outcomes.

PM associations have been reported in a wide variety of cities with different levels of, and correlations with, co-pollutants, including high and low SO_2 and ozone. In many cases, once PM effects have been accounted for in a study, the remaining co-pollutants have either not been

associated with the health endpoint(s) or else their inclusion in the model did not impact the estimated PM effect substantially. These observations of PM's 'robustness' lend increased confidence to the conclusion that PM exposures are the dominant, though perhaps not the sole, pollutant-related risk factor in the ambient environment.

Statistical issues must also be considered in this regard. It is important to recognize that co-pollutants are often correlated (or collinear) with PM over time due to the primary importance of weather patterns in determining ambient concentrations on any given day. In addition, most of the criteria air pollutants are generated through fossil fuel combustion and thus share common sources. This temporal correlation, depending on its magnitude, can make it difficult in a statistical sense to separate out the independent effects of different pollutants. Where correlations are relatively low (e.g., less than 0.5), it is often possible to derive reliable effect estimates for multiple pollutants included simultaneously in a regression, though the standard errors of those estimates will be inflated. Indeed, as noted above, many studies have been able to demonstrate independent PM effects in the presence of co-pollutants. However, where correlations are high (e.g., greater than 0.8), including additional pollutants in a model often cannot help determine which pollutant is most important, because risk coefficient estimates and their standard errors become very unstable. Any change in the significance of PM may thus be due to predictable statistical aspects of multi-collinearity and/or differential measurement error. Thus, caution must be exercised in interpreting results of multi-pollutant analyses when high degrees of correlation are present.

These points have been noted by many investigators, including Lipfert and Wyzga (1999) who state, "Single-pollutant regression results will likely overstate mean effects because of collinear relationships with other pollutants (*if the other pollutants have effects*, emphasis added), but multiple regressions may also yield misleading results under certain conditions, including high collinearity and differential measurement error…" This reference was cited by commenter 12.

An additional factor that must be kept in mind when interpreting results from multi-pollutant analyses that the temporal relationships between ambient concentrations and population exposures vary for different pollutants. The acute health effects captured by time-series epidemiological studies reflect associations between ambient concentrations and population health impacts. For these effects to represent a causal relationship, there must be a correlation over time between ambient concentrations and actual population exposures. This has been confirmed recently for PM2.5 in several studies, including an innovative study by Sarnat and colleagues (2001), who found no correlation between ambient concentrations and personal exposures for O_3, NO_2, and SO_2. Furthermore, it was shown that ambient O_3, NO_2, and SO_2 concentrations **did** correlate with personal PM2.5. While wider confirmation is needed, these findings imply that ambient concentrations of gaseous co-pollutants can serve as surrogates for personal PM2.5 exposures, which could lead in some cases to a false attribution of health effects to gaseous pollutants when, in fact, fine particles were the causative agent. Therefore, multi-pollutant models may not be suitable and the health effects attributable to ambient gases may be a result of PM2.5 exposure.

The NMMAPS study included gaseous co-pollutants along with PM in alternative regression models fit to all 90 cities. While the PM effect estimates diminished somewhat, they remained strongly significant. In the NMMAPS analysis of PM effect estimates as a function of inter-pollutant correlations, there was no evidence of significant changes in the PM effects across a range of cities that differed substantially in the degree to which PM correlated with other pollutants. Samet et al. (2000a p 27) stated: "As for the 20 cities, the effect of PM10 changed little with control for the other pollutants." Further, the HEI Review Panel (cited in Krewski et al., 2000

p. 75) concluded: "…the Panel agrees that in the 20 cities no convincing evidence suggests that PM10 effects on mortality are changed by the addition of either O_3, SO_2, NO_2 or CO concentrations to the models, suggesting that none of the other pollutants is responsible for the observed PM10 effects."

Regarding the prospective cohort studies by Pope et al. (1995) and Krewski et al. (2000), there are related issues when multi-pollutant models are used. While the PM2.5 estimate was decreased in the Krewski et al. (2000) sensitivity models that also included SO_2, this should not be interpreted as necessarily signifying that the PM2.5 effects are actually smaller than the single-pollutant models indicate. When one includes correlated variables in a regression at the same time, such as SO_2 and PM2.5 in the case of the Krewski et al. sensitivity analysis, this violates the basic assumption of the regression model of the independence of the predictor (x) variables, so the effect estimates are biased in these cases by the resulting model inter-correlations among the independent variables. The likely reason that SO_2 and PM2.5 are so correlated spatially is that they both are predominantly derived from a common source: fossil fuel combustion. This largely shared-source aspect of PM2.5 and SO_2 in the U.S. makes it very difficult for simultaneous regressions (e.g., those conducted by Krewski et al., 2000) to "partition" their respective effects. Thus, a finding that the PM2.5 effect estimates would be biased, and changed by the inclusion of a correlated variable such as SO_2 would not be unexpected. The new estimate based on the multi-pollutant model is not better, however, due to the fact that two correlated variables were in the model at the same time, which violates the underlying regression model assumption of independent (i.e., uncorrelated) predictor variables, and which almost certainly statistically biases this two-pollutant model's effect estimates in relation to the true effect estimates.

Indeed, in the HEI Report (Krewski et al., 2000), the original research authors note (on page 275) that: "We understand the inappropriateness of estimating many alternative statistical models that use many combinations of often correlated variables while searching for a preferred result or a statistical explanation for a disavowed result. We know that the Reanalysis Team, Expert Panel, Advisory Board, and Review Panel also understand the inappropriateness of such an approach. But, of course, it is hard to know when to stop. A systematic and skillful estimation of dozens (maybe even hundreds) of alternative statistical models with different variables and combinations of variables, even when it is done in the name of sensitivity analyses, will ultimately produce spurious associations."

It should be noted that the two-pollutant sensitivity model estimates by Krewski et al. (2000) of the PM2.5 effect still fell within the 95% confidence range of the single-pollutant model estimates, and with a relative risk estimate above 1.0, indicating that the PM2.5 effect estimates, though diminished for statistical reasons as discussed above, were actually not significantly changed by the addition of SO_2.

Overall, the statistical importance of SO_2 in the Krewski et al. (2000) sensitivity results seems unlikely to result from a true mortality health effect of SO_2 *per se*, but because it is another marker for fossil fuel combustion-related particles that form from the SO_2 emitted by these sources. In fact, the HEI Report (Krewski et al., 2000) notes (on page 233) that "The absence of a plausible toxicological mechanism by which sulfur dioxide could lead to increased mortality further suggests that it might be acting as a marker for other mortality-associated pollutants". Thus, the apparent SO_2-mortality association is most likely to result from the fact that it is a marker for the fossil fuel component of PM2.5 particles, and, in turn, of an enhanced toxicity of these fossil fuel combustion-related particles versus other PM2.5 particles, rather than from a distinct SO_2 health effect.

54. **COMMENT:** Weather is an uncontrolled confounder in many of the studies relied upon in the Report. (Commenters 11, 12) **RESPONSE:** Weather factors (e.g., temperature, humidity, dewpoint) have long been recognized as important potential confounders of the relationship between air pollution and acute mortality. It is well accepted that extreme heat events, as well as cold snaps, can lead to excess mortality. In addition, daily air pollution concentrations are closely linked to changes in weather. In view of these relationships, it is imperative that weather factors be controlled in time-series epidemiology studies. This has indeed been the case ever since the time-series design was first applied to the study of air pollution and mortality by Schimmel and Murawski in the 1970s.

    A variety of techniques have been used to control for weather factors in time-series studies of mortality and morbidity outcomes, including the use of linear terms, modeling extremes, and through nonparametric (nonlinear, data-driven) smoothing techniques. In addition, synoptic weather patterns have been used and data have been deseasonalized through smoothing functions. These methods are now developed to the point that there remains little concern among most analysts that weather factors could significantly confound the associations between air pollution and acute mortality or morbidity. The 1996 PM Air Quality Criteria Document of USEPA concluded that, "The observed PM effects are unlikely to be significantly confounded by weather." This conclusion was affirmed in the current draft PMAQCD which states, "the issue of potential confounding by weather was extensively examined in two studies as reviewed in the 1996 PM AQCD, and was considered essentially resolved." Later, in chapter 9, Integrated Summary, the CD states "The likelihood of PM effects being accounted for mainly by weather factors was addressed by various methods that controlled for weather variables in most studies (including some involving sophisticated synoptic weather pattern evaluations), and that possibility was found to be very unlikely."

    Additional support for the view that weather factors do not confound the observed PM effects is derived from the fact that PM associations have been observed in cities with climates that are cold (Detroit, Montreal, Minneapolis, other Canadian cities, Helsinki) and warm (Bangkok, Mexico City, Southern CA), and well as cities with high and low humidity. Therefore, a common weather confounder is unlikely. Further, effects have been reported in cities where PM peaks in summer (Philadelphia, Steubenville, many East Coast cities) and winter (Utah Valley, Santa Clara) or spring (Helsinki) and in cities with muted seasonal changes (Palm Springs, London, Netherlands, Bangkok).

55. **COMMENT:** The conclusion regarding the lack of threshold is unwarranted. (Commenters 3, 11, 12, 15) **RESPONSE:** There is no evidence yet available that identifies a population threshold for the acute mortality or morbidity effects of PM. There are many possible reasons for this. If, as expected, individual thresholds vary across the population, an analysis of aggregate population health data would tend to observe a continuous rise in health risk with increasing PM exposures. In addition, statistical power is usually very limited at the low end of the exposure range, leading to large standard errors on the risk estimates and an inability to statistically distinguish between linear and various nonlinear models, including threshold models. Finally, uncertainties in the relationship between ambient concentrations and population exposures introduce misclassification errors. It is acknowledged that the inability to identify a threshold using currently available data and methods does not mean that no thresholds exist at the individual level. While further work is needed in this area, at present there is insufficient evidence to identify a population threshold for the effects of PM. One exception to this conclusion is the work by Smith et al. (2000) for Phoenix which reported no association for PM2.5, as well as a potential threshold at around 20 $\mu g/m^3$ and based on the graphical analysis, effects at concentrations greater than 20 $\mu g/m^3$. Such a finding is not inconsistent with our findings and recommendations

For short-term exposure to PM, two general methods are available to address the issue of the existence of a threshold, or an ambient PM level below which there would be no risk of a significant adverse health outcome. First, it can be examined indirectly by considering data sets with very low mean ambient concentrations. Second, it can be examined directly by developing statistical tests that carefully model the shape of the concentration-response function. Both of these approaches appear to indicate the lack of an observable population threshold. Regarding the first method, several studies have been conducted in cities with low ambient concentrations of PM10, including Morgan et al. (1998) for Sydney, Australia (mean = 18 $\mu g/m^3$, based on conversion from co-located nephelometry data), Wordley et al. (1997) for Birmingham, UK (mean = 26 $\mu g/m^3$), Schwartz et al. (1996) for the Harvard Six-Cities (mean = 25 $\mu g/m^3$), Burnett et al. (2000) for the eight largest Canadian cities (mean =26 $\mu g/m^3$), and Gwynn et al. (2000) for Buffalo and Rochester (mean = 24 $\mu g/m^3$). In addition, several cities in the data set used by Samet et al. (2000a) have mean concentrations in the low 20s. Examination of these data indicates that the concentration-response functions are not driven by peak concentrations and that the slopes of these functions do not appear to increase significantly at higher concentrations.

Among the statistical approaches, Schwartz (2000a) simply examined the concentration-response relationship in 10 U.S. cities, restricting the data to only days where PM10 < 50 $\mu g/m^3$. The resulting risk estimates were statistically significant and greater than for that of the entire data set. Two other papers first addressed the issue of whether existing statistical techniques could identify a threshold, assuming one existed. Cakmak et al. (1999) simulated data with varying degrees of exposure measurement error, based on actual data from Toronto. They examined whether statistical models used in most air pollution epidemiology (including locally weighted smoothing techniques in Poisson regression models) would be able to detect thresholds in the PM-mortality association. They concluded that, if a threshold existed, it is highly likely that the existing statistical modeling would detect it. Many mortality papers have, in fact, examined the shape of the concentration-response function and indicated that a linear (nonthreshold) model fit the data well (Pope, 2000).

A different statistical approach was used by Schwartz and Zanobetti (2000) in their analysis of 10 U.S. cities. The authors combined concentration-response curves across the cities, after demonstrating that this approach produced unbiased estimates. Predicted values of the response function were estimated at 2 $\mu g/m^3$ intervals. Results from this approach did not provide any evidence for a threshold effect. Finally, Daniels et al. (2000) used an alternative statistical approach to test for the existence of a threshold using the 20 largest cities in the U.S. The authors considered three alternative log-linear regression models. One used a simple linear term for PM10, which could then be used as a basis for comparison with the other models. A second model used a cubic spline that would allow for nonlinearity in PM10 that could represent a threshold function. The third model presumed a threshold, in which a grid search was used to test for a concentration that would support a threshold. The results indicated that for the second model, which can allow for a threshold if the underlying data suggest one, a linear specification provided the best fit to the data. Analysis using the grid search model suggested that no threshold was apparent for either total mortality or cardiopulmonary mortality. Finally, using a goodness-of-fit test (Akaike's information criterion) to compare the simple linear nonthreshold model with models that would allow for a threshold concentration, the authors reported that there was no evidence to prefer the threshold models to the linear model.

Schwartz et al. (1996) examined the relationship of PM2.5 concentrations and daily mortality in the Harvard Six Cities dataset. When they restricted the analysis to days on which the PM2.5 24-hour

average concentrations equalled or exceeded 30 or 25 μg/m^3, Schwartz et al. (1996) found that the strong association persisted, suggesting that, if there is a threshold of effect, it cannot be found at concentrations in excess of 25 μg/m^3. On the other hand, Smith et al. (2000) statistically examined the threshold issue in data on mortality and ambient PM2.5 from Phoenix, AZ. They reported evidence of a significant change in the regression slope at a concentration of around 20 to 25 μg/m^3 PM2.5, suggesting the possibility of a threshold in this range. However, to our knowledge, this is the only study to report such a finding. Staff from OEHHA and the Bay Area Air Quality Management District (BAAQMD) analyzed data from the two published California studies involving 24-hour measurements of PM2.5 and daily mortality counts (in Coachella Valley [Ostro et al., 2000] and Santa Clara County [Fairley, 1999]). The modeling techniques used for the exposure-response functions included piecewise linear regression (e.g., utilizing several "hockey-stick" models), locally weighted smoothing in generalized additive models, trimming analysis (selectively deleting days with high PM2.5 values), and Bayesian models (comparing the likelihoods of various thresholds) to explore the evidence for a nonlinear exposure-response at low PM2.5 concentrations. In general, staff found that a linear, nonthreshold model within the concentration range of interest for PM2.5 provided an adequate fit to the data, while threshold (or other nonlinear) models provided no better fit. Except for the report of Smith et al. (2000), it appears that relationship between daily mortality and PM2.5 is likely well characterized by a nonthreshold model, consistent with the findings reported by others for PM10 (see above).

As indicated by Cakmak et al. (1999), measurement error in exposure could make it more difficult to find a threshold, assuming one exists. However, using a detailed simulation analysis, they report that for PM10 concentrations near the median and above (around 20 to 30 μg/m^3 and above), which is an area of concern for standard-setting, even if the correlation between personal exposure and ambient measurement is as low as 0.6 to 0.8, the models are 80% likely to detect a threshold, assuming one existed. Studies in the U.S. and Holland have shown time-series correlations of about 0.8 between personal and ambient exposure for both PM2.5 and sulfates. Therefore, given that dozens of studies have failed to detect anything besides a linear, nonthreshold concentration–response function, it is unlikely that measurement error by itself would explain the lack of a demonstrated threshold.

56. **COMMENT:** De-trending does not eliminate the need for season-specific time series analyses. (Commenters 11, 12) **RESPONSE:** De-trending is used in time-series analyses to remove the potentially confounding influence of strong seasonal cycles in both health and air pollution. We agree that season-specific analyses are valuable. However, year-round analyses after de-trending, the most prevalent approach available in the literature, still provide meaningful results on overall PM effects. Many of these approaches use a loess smoothing technique to control for seasonality. The loess smoothing technique can accommodate nonlinear and nonmonotonic patterns between time and other factors and the health outcome, offering a flexible nonparametric modeling tool. Including a smoothed variable in the model does not explain the underlying reason for the pattern over time, but controls for it statistically, allowing one to observe the relationship between daily mortality and environmental factors after the underlying trend in daily mortality is controlled for. Detailed analysis has demonstrated that these techniques are very effective in removing seasonal trends in the data. In addition, adding a locally weighted smooth of time diminishes short-term fluctuations in the data, thereby helping to reduce the degree of serial correlation. Serial correlation exists when the errors of the regression model are related over time, producing biased estimates of the variance of the explanatory variable coefficients. Finally, disaggregating the data by month or season introduces other problems into the analysis such as reduction in power, making it more difficult to find an effect given that one truly exists.

CHAPTER 7: 24-HOUR PROPOSAL FOR PM2.5

57. **COMMENT:** Strongly support establishment of 24-hour PM2.5 standard, but believe current proposal is insufficiently protective of public health. Should apply additional margin of safety to address issues of environmental justice. The annual and 24-hour standards are not protective of public health. (Commenters 16, 19) **RESPONSE:** Based on current evidence, the proposal provides sufficient protection of public health, although there is no risk-free level. Multiple analyses of the exposure-response relationships between PM2.5 and mortality indicate that the data can be fitted most parsimoniously with linear, nonthreshold models. Given the apparent linearity of the exposure-response relationships in the epidemiological data, it is difficult to determine at what concentrations within the PM2.5 distributions in each study adverse health effects begin. Intuitively, one would expect greater biological responses and larger numbers of adverse events occurring at higher concentrations, everything else being equal.

    The importance of the linear, nonthreshold exposure-response relationship cannot be overemphasized in light of legislation requiring that ambient air quality standards be "established at levels that adequately protect the health of the public, including infants and children, with an adequate margin of safety." (California Health & Safety Code Section 39606(d)(2)) If a threshold in the exposure-response curve cannot be identified, then specification of an "adequate margin of safety" becomes challenging. The approach OEHHA staff members have adopted in pursuit of this objective has therefore been to: (1) identify indicators of the distribution of PM2.5 (specifically the means and 98^{th} percentiles) in epidemiological studies that demonstrate the relationship of ambient fine particles with adverse health impacts, (2) recommend that the distribution of PM2.5 in California be reduced below the levels of these distributions, and (3) incorporate a margin of safety in the form of a standard "not to be exceeded", which will assure that the extreme values of the PM2.5 distribution in California will be lower (and in general substantially lower) than the 98^{th} percentiles of PM2.5 distributions in published studies.

    Without placing a short-term limitation on PM2.5 concentrations, recent experience in California indicates that even attainment of the recommended annual standard of 12 $\mu g/m^3$ will allow for excursions well into the range in which adverse effects, including mortality, have been identified in epidemiological studies. Notably, the modified EPDC analysis undertaken by the ARB staff indicates that for several large air basins, the estimated 98^{th} percentile of the PM2.5 distribution consistent with attainment of an annual standard of 12 $\mu g/m^3$ would be in excess of 40 $\mu g/m^3$. Thus, adoption of a 24-hour standard of 25 $\mu g/m^3$ would be intended to limit such excursions.

    Regarding the issue of environmental justice, we agree with the commenter that this is an important issue that needs to be reviewed and analyzed. However, we believe that environmental justice issues such as exposures of sub-populations to higher than average PM levels, are best addressed in the implementation phase of these standards, not in the setting of the standards themselves.

58. **COMMENT:** The proposed 24-hr PM2.5 standard does not acknowledge the lack of controlled experiments demonstrating effects at or around the level of the standard. Only controlled studies can credibly establish a causal relationship between PM exposure and health endpoints. The estimated risk is sensitive to model specification, city, data and control for weather. (Commenters 15, 18) **RESPONSE:** We disagree that only controlled studies are sufficient for causal inference, especially for study of PM and mortality. Most etiologic inference in medicine is based on epidemiological studies, not controlled exposures. Using generally accepted guidelines for causal inference, relationships between PM and adverse health impacts are addressed in Section 7.9 of the proposal, reviewed by AQAC in January 2002. Specifically, we carefully examined generally

accepted guidelines for causal inference, including: (1) the consistency of the findings; (2) the coherence of the study results; (3) the likelihood that findings are due to chance; (4) the possibility that findings are due to bias or confounding; (5) temporal sequence of the associations; (6) the specificity of the findings; (7) evidence for exposure-response relationships; (8) strength of the associations; and (9) the biological plausibility of a causal associations. These are based on informal guidelines for causal inference described by Sir Austin Bradford Hill, as modified by other epidemiologists (Hill, 1965; Rothman, 1982). The scientific evidence linking PM exposure to premature mortality and a range of morbidity outcomes appears to meet the generally accepted guidelines for causal inference in epidemiology. Much current research is now focusing on biological mechanisms in order to provide a more complete understanding of the effects of PM.

We agree that risk estimates are sometimes sensitive to city or region examined, model specification, control for weather, degree of measurement error, and inclusion of correlated co-pollutants. However, this does not invalidate assessment of causal relationship between ambient PM and adverse health outcomes.

59. **COMMENT:** The 24-hour proposal for PM2.5 ignores the nature of PM as a mixture, with constituents of varying toxicity. This may lead to control of the wrong components, with few health benefits. (Commenters 14, 15, 18) **RESPONSE:** There is an ongoing debate over whether toxicity is more related to particle size, mass, number and specific constituents. More research is clearly necessary. Any new information on this issue will be incorporated into ARB policy and standards development over time. However, it is generally accepted among researchers that combustion-related particles (e.g., diesel) are toxic and several articles are cited in the document that support this contention. There is sufficient scientific evidence on fine particles that warrant concern including: (i) they deposit throughout the lung and are retained in large quantities; (ii) they are linked in controlled exposure studies with lung inflammation; (iii) they easily penetrate residences; (iv) there are many epidemiological studies indicating associations with daily morbidity and mortality.

CHAPTER 7 : PARTICLE DOSIMETRY

60. **COMMENT:** The commenter raises questions related to fine particle dosimetry in the lung. The commenter notes a lack of discussion of particle dosimetry modeling in Section 7.1; specifically that there is no mention of the 1994 ICRP Human Respiratory Tract Dosimetry Model. The commenter cites work of Snipes et al. (1997) and others to argue that model estimates of doses of fine particles delivered and retained in the alveolar-interstitial (AI) region of the lung are too low to cause any toxic or adverse action, which therefore undermines any causal relationship between particle exposures and adverse health effects. The commenter succinctly summarizes several pages of comments as follows: "[T]he lung modeling data not only fail to support the proposed toxicity of fine particles as the cause of the statistical associations observed in epidemiological studies, but the dosimetry unequivocally shows that the daily alveolar deposits of fine particles and their potentially toxic components under present U.S. urban conditions are too low to be responsible for complex health effects like increased daily morbidity and mortality." (Commenter 12) **RESPONSE**: We have retitled Section 7.1 as "Particle Deposition, Clearance, and Dosimetry" to indicate that the section covers deposition and clearance as well as dosimetry. In addition, we have added a couple of paragraphs to the end of Section 7.1.1 incorporating data from the article by Snipes et al. (1997), cited by the commenter. While the commenter correctly indicated that the document should have additional information on particle dosimetry, we cannot agree with the assertion that the estimated doses are too low to have any toxic effect, for the following reasons: (1) mechanisms of particle-associated toxicity are incompletely understood, much less quantified; therefore, it is not possible to designate what constitutes a negligible dose; (2) to support the case

that daily doses are trivial, the commenter has selectively cited the metrics used by Snipes et al. (1997) – other metrics (e.g., particle number/surface area) suggest potentially greater exposures, especially to the conducting airways (i.e., bronchi and bronchioles); (3) the work by Snipes et al. (1997) is based on population average airway dimensions in the ICRP model and does not incorporate the large inter-individual differences in deposition related to variations in age, disease state, and pulmonary anatomy as well as ventilation patterns, short-term peak exposures, and so forth; (4) by focusing only on the alveolar-interstitial portion of the lung, the commenter assumes that exposures occurring in the bronchi and bronchioles are clinically unimportant. We cannot agree with the latter approach, both because some of the important adverse health effects associated with particle exposure are airway-related (e.g., exacerbation of asthma), and because the airway particle doses estimated by Snipes et al. (1997) are much greater than those predicted for the alveolar interstitial area, for both fine and coarse particles. Thus, as noted above, we have modified Section 7.1.1 to incorporate some of the particle dose estimates provided by Snipes et al. (1997) using the 1994 ICRP Human Respiratory Tract Dosimetry Model, but unlike the commenter, we cannot, for the reasons indicated, portray these doses as negligible. It follows, therefore, that we do not accept the assertion that the scale of the estimated doses precludes a causal relationship between particle doses and adverse health impacts. Additional detail is provided below.

Snipes et al. (1997) modeled particle size distributions as observed in environmental aerosols from Phoenix and Philadelphia. Table 1 summarizes the percent of total mass, number and surface area of three modes of the aerosols modeled: Fine, Intermodal, and Coarse.

Table 1. Percent of Total Mass, Particle Number, or Surface Area of Each of Three Modes for Philadelphia and Phoenix Aerosols (Snipes et al., 1997)

		Philadelphia			Phoenix	
Mode	**Fine**	**Intermodal**	**Coarse**	**Fine**	**Intermodal**	**Coarse**
Mass	48.2	7.4	44.2	22.4	13.8	63.9
Number	95.2	0.05	0.004	99.6	0.3	0.1
Surface Area	95.4	2.5	2.1	85.5	7.4	7.1
MMAD, µm	0.436	2.2	28.8	0.185	1.7	16.4

Table 2 summarizes model dose estimates for the alveolar-interstitial (AI) region from Snipes et al. (1997). Table 3 provides model AI dose estimates for the general population exposed to three different particle sizes determined by the U.S. EPA.

Since the mechanisms of particle-associated toxicity are unknown, it is not possible to predict with any degree of certainty what doses can be considered negligible. Similarly, the dose metric most closely linked with adverse effects is unknown. An examination of Table 2 shows that the values for a selection of reasonable dose metrics predicted for the fine particle mode in simulations based both on the Philadelphia and Phoenix aerosol particle size distributions cannot be considered "negligible" when compared to those of the larger sized fractions. The values from U.S. EPA for a general U.S. population use smaller particle sizes for all modes and would be expected to give even higher values for the AI dose metrics in Table 2.

The equilibrium burden in the AI region predicted by Snipes et al. (1997) is based on assumptions of dissolution-absorption properties that may not hold for lifetime simulations. To quote the

authors: "With respect to constructing accurate retained dose metrics for particles in the respiratory tract, *in vivo* dissolution-absorption rate characteristics are key determinants of particle clearance. These characteristics are more difficult to determine and were not done for the different modes of the Philadelphia and Phoenix aerosols. The approximations for dissolution-absorption rates *used in this article* could therefore yield only illustrative modeling results that would be improved with *accurate* values for these parameters."

Table 2. Model Estimates of Selected Dose Metrics for the Alveolar Interstitial Region*

Dose Metric	**Philadelphia**			**Phoenix**		
	Fine	Intermodal	Coarse	Fine	Intermodal	Coarse
μg/d	37.1	11.3	1.2	26.5	17.2	11.9
ng/cm^2-d	0.025	0.0077	0.00078	0.18	0.012	0.0081
ng/g tissue-d	34	10	1	24	16	11
Equilibrium burden μg/d	0.3	1	0.3	0.2	1	3
no. particles/cm^2-d	100	0.1	1E-6	100	0.1	1E-7

*Values are for inhaled aerosols 50 μg/m3, 24hr/d, 7d/wk. Snipes et al. (1997)

Table 3. Model Estimates of Particle Deposition in the AI Region for General Population*

Breather/Metric	**Fine**	**Medium**	**Coarse**
Normal Augmenter			
Percent	7.0	4.2	2.5
μg	69	42	25
Mouth Breather			
Percent	7.2	4.2	6.2
μg	71	42	62
MMAD, μm	0.0169	0.18	5.95

*U.S.EPA (2000) - online source

Notwithstanding the difficulties noted above, the commenter cites modeling results from Vostal (2000) indicating that "the estimates show that when the deposits are expressed in effects-related metrics, e.g., amounts of fine particles or their components deposited daily per square centimeter of lung surface in the alveolar/interstitial region, the deposits are of a very low magnitude and represent only fractions of nanograms mass (10^{-9})"… Vostal (2000) extended the findings of Snipes et al. (1997) using chemical speciation data on $PM_{2.5}$ from Houston, Texas (Tropp et al., 2000), assuming that Houston particles would be representative of Phoenix and Philadelphia, and by extension, cities in California. Vostal's results are summarized in Table 4. Vostal does not include organic carbon as a significant speciated component even though its mass was more than twice that of elemental carbon (3.3 vs. 1.5 μg/m^3); moreover, he does not include arsenic among toxic metals. Vostal (2000) and the commenter conclude that one of several potential mass-related dose metrics is the most relevant and that because estimates for this metric are very low for individual aerosol components, then the latter or their aggregate cannot be causally associated with adverse health effects. In the author's words "the 24 hr. levels of the deposited PM2.5 particles and their components are too low to produce a measurable health effect or be responsible for a complex biological endpoint like sudden changes in morbidity or mortality."

As noted above, the mechanism of toxicity for the observed adverse effects is unknown. While conventional mass dose metrics indicate low estimated doses for total particles and various components, we do not know if they are too low "in aggregate" to cause adverse effects by as yet unknown mechanisms. Also while the Alveolar-Interstitial region is considered by the commenter and the cited authors to be the most sensitive region of the lung in terms of particle-induced adverse effects, the conducting airways are also clearly a likely target tissue.

Table 4. Predicted Dosimetry of Fine Particles and Their Components in the Alveolar-Interstitial Region of the Lung (Vostal, 2000)*

Dose Metric Component	Philadelphia Fine	Philadelphia Fine & Intermodal	Phoenix Fine	Phoenix Fine & Intermodal
ng/cm^2-d				
Total mass	0.0182	0.0210	0.028	0.029
Sulfate (SO4)	0.0048	0.0056	0.0075	0.0077
Elemental carbon	0.0016	0.0018	0.0024	0.0025
Iron (Fe)	0.00018	0.00020	0.00027	0.00028
Trace elements except Fe	0.00010	0.00012	0.00015	0.00016
Toxic metals	0.000021	0.000024	0.000032	0.000033
ng/g Al tissue-d				
Total mass	24.8	27.8	37.6	38.8
Sulfate (SO4)	6.6	7.4	10.0	10.3
Elemental carbon	2.2	2.4	3.3	3.4
Iron (Fe)	0.24	0.27	0.37	0.38
Trace elements except Fe	0.136	0.152	0.207	0.213
Toxic Metals	0.027	0.030	0.041	0.042

*For residents inhaling an average annual $PM_{2.5}$ concentration of 17.5 μg/m^3 in the Philadelphia and Phoenix dosimetry models.

CHAPTER 7: BIOLOGICAL MECHANISMS

61. **COMMENT:** The commenter indicates that the Draft relies on high-dose toxicology studies, involving nonphysiological modes of exposure (especially intra-tracheal administration) to support the notion that there are biologically plausible explanations for the particle-associated adverse health effects reported consistently in the epidemiological literature. The commenter also criticizes the methodology of a paper cited by OEHHA (Nemmar et al., 2001b), in which the investigators had concluded that radiolabeled ultrafine particles could be detected in the blood shortly after inhalation. (Commenter 12) **RESPONSE:** We agree that high-dose intra-tracheal administration of particles or *in vitro* exposures of lung tissue are not necessarily representative of what might occur toxicologically when humans are exposed to ambient particles. Although we believe that there were sufficient caveats to this effect in the initial Draft, we have added several more qualifications throughout the text of Section 7.8, indicating the tentativeness of the state of the science regarding mechanisms of particle-associated toxicity and that one cannot directly extrapolate such findings to human exposures to ambient particles. Nevertheless, there are also several studies discussed in Section 7.8 involving potential mechanisms of particle-related cardiovascular and

pulmonary effects, in which the human subjects were exposed in daily life to ambient particles or in a controlled setting to particle levels consistent with occupational exposures, with ambient exposures in the developing world, or with peak exposure levels at busy intersections in rush-hour traffic. (See below) Thus, though we concur to some extent with the commenter, we would suggest that the concluding sentence to section 8 (unaltered) still expresses our view regarding potential biological mechanisms: "While the evidence is still fragmentary, it represents a dramatic advance from a few years ago, and begins to sketch a framework of biological plausibility for the time-series studies."

With respect to commenter's critique of the Nemmar et al. (2001b) report cited in the initial Draft, we think that subsequent publication of the work by these investigators addresses the methodological concerns expressed by the commenter (Nemmar et al., 2002). The comment raises an obvious concern that the investigators were clearly aware of, and which they have addressed sufficiently for the work to be published in a high-caliber medical journal (Circulation). Furthermore, even if the results of Nemmar et al. (2001a, b; 2002) were later found to be spurious, the potential for systemic pathophysiological effects related to pulmonary deposition of particles has been demonstrated by several other laboratories, and does not rest alone on the rapid absorption of particles into the blood. Thus, we have not modified the document in response to this comment..

CHAPTER 7: PULMONARY AND SYSTEMIC INFLAMMATION

62. **COMMENT:** The commenters discuss a variety of perceived shortcomings of several papers cited in the Draft in support of the notion that particle inhalation can result in inflammation in the lung, and suggest that the Draft should provide a much more critical discussion of these reports, which include several with very high doses relative to ambient concentrations. Exposures to near-ambient levels are needed to confirm the high-does experiments. (Commenters 11, 12) : Pulmonary inflammation is, in itself, a (normal) physiological, self-limiting response to respiratory stress. The papers cited in the Draft do not support the conclusion in the summary of section 7.8 that localized airway inflammation "provides mechanistic support for a causal relationship between ambient PM and the cardiopulmonary morbidity and mortality." In addition, the commenter states, "In the present form, the summary [of the section] is too strongly influenced by studies that use particle challenges much higher than those occurring under ambient levels and erroneously interprets small transient and beneficial changes in physiological defense mechanisms as indices of some as yet undocumented permanent pathological inflammation." (Commenter 12, pp. 68-73) The summary paragraph should exclude references about systemic effects because these are based on studies that may not be relevant to humans exposed to ambient PM. (Commenter 11) **RESPONSE:** In the original Draft, we recognized that studies such as those noted by the commenters have inherent limitations with respect to extrapolation to humans; however, we agree that the initial Draft did not sufficiently convey our understanding of some of these limitations. As noted in the response to the previous comment, we have added several qualifying remarks about the applicability of some of the experimental studies to ambient particulate matter exposures in humans. For instance, the summary paragraph in the revised Section 7.8.2 now reads: *"Taken together, these data suggest that inhalation of different sources of particles may initiate inflammatory events in human lungs, with some (albeit sparse) evidence of systemic impacts, including stimulation of bone marrow to accelerate production of inflammatory cells to respond to the pulmonary insult. However, these observations are subject to the caveat that the results observed in the high-dose animal and in vitro experiments, as well as in the controlled human exposures, may or may not be directly applicable to humans exposed to ambient PM."*

We have also added a sentence about the utility of low-level controlled human exposures to the paragraph that describes the limitations of the human diesel exposure studies.

The principal objective of Section 7.8 and its subsections was to illustrate that potential mechanisms to explain the epidemiological time-series observations are beginning to emerge, in contrast to the abyss of ignorance in this area just a few years ago. In addition, while strong evidence of biological mechanisms is certainly useful in assessing causal relationships between environmental exposures and disease, such evidence is not a *sine qua non* for causal inference.

We would take issue (as did members of the Air Quality Advisory Committee) with the assertion that localized inflammation should be interpreted as a (normal) physiological response rather than a pathological process. While inflammation in response to acute injury is a normal process, the inflammatory process can amplify oxidative stress, and result in the circulation of systemic chemical messengers that may have pathophysiological consequences. The assertion that pulmonary inflammation induced by exposure to ambient PM concentrations would be of little consequence is speculative at best, and is not based on sound science. Finally, the Draft does not interpret "small transient and *beneficial* changes in physiological defense mechanisms as indices of some as yet undocumented permanent pathological inflammation." (emphasis added) Section 7.8 and subsection 7.8.2 provide a description of pathophysiological events that may underlie acute responses to particulate matter air pollution, and do not refer to "permanent pathological inflammation."

63. **COMMENT:** The studies on bone marrow stimulation by PM exposure have significant methodological flaws – in the Tan et al. (2000) study of military recruits fighting wildfires in Indonesia there were likely confounding exposures ("the CO [carbon monoxide] levels would have likely been quite high" as well as "stress, exhaustion, and injury"), while the artificial mode of administration (intrapharyngeal) route and high dose of PM administered to rabbits (Mukae et al. 2001) precludes comparing these results with the human study. (Commenter 11) **RESPONSE:** The methods section of the Tan et al. (2000) paper indicates nothing about the subjects' fighting wildfires in *Indonesia*, but rather that they were national service men in a neighboring, but entirely different, country (*Singapore*) who undertook regular outdoor activities ("walking, marching, jogging, swimming, and obstacle training, as well as some indoor classroom activities") during a period of atmospheric haze resulting from the Indonesian fires. While it is possible that there may have been confounding exposures, those related to fire fighting (CO, stress, exhaustion, and injury) would not have been among them. As for the high-dose rabbit study (Mukae et al. 2001), the Draft indicates in several parts of Section 7.8 that the results of high-dose animal studies using nonphysiological routes of administration may have limited generalizability (see above responses). Thus, we have not changed the document specifically in response to this comment.

CHAPTER 7: EFFECTS ON THE CIRCULATION AND CARDIAC EVENTS

64. **COMMENT:** The published studies cited in the Draft have methodological omissions that vitiate their ability to explain mechanistically the results of the time-series studies linking cardiovascular outcomes to ambient PM. The associations repeatedly observed in epidemiological studies may be due to something else, such as "random changes in the progress of a chronic disease rather than by the variability of ambient PM pollution." (Commenter 12) **RESPONSE:** As noted in the response to the comment on Section 7.8.2, we have modified the Draft to indicate that this section is intended to convey that researchers have begun to identify biologically plausible mechanisms that may help explain the findings of the time-series studies. Neither the prior Draft nor the revised report claim that these studies provide definitive, uncontroverted proof of the specific mechanisms. The commenter provides no scientific foundation for the assertion that "random

changes" in cardiovascular disease status are responsible for the consistent, statistically significant associations between changes in PM pollution and serious exacerbations of cardiovascular disease (as represented by hospitalizations for ischemic heart disease).

65. **COMMENT:** The sentence indicating that one should be careful interpreting the controlled diesel exhaust studies should also indicate that high concentrations of PM were used. (Commenter 11) **RESPONSE:** We agree and have changed the sentence to read as follows: "This observation is subject to the caveat that three of these four studies involved exposures to high concentrations of diesel exhaust particles, which may not necessarily be representative of ambient PM generally."

66. **COMMENT:** Baseline levels of C-reactive protein were obtained 3 years after men were initially studied in the German MONICA study (Peters et al. 2001b). This appears to be problematic for a variety of reasons. (Commenter 11) **RESPONSE:** This study (Peters et al., 2001b) did not just look at comparisons of blood samples taken three years apart; the latter was just one of several comparisons undertaken demonstrating an association between ambient PM (measured as total suspended particles) and one blood marker of a systemic physiological response. In addition, this study is cited in the Draft as one of several interesting recent reports that *may* illustrate potential mechanisms relating exposure to ambient PM and cardiovascular outcomes.

67. **COMMENT:** The standard-setting process should be based on controlled experiments with a concentration range including the standard. Using the results of epidemiological studies and high-dose controlled exposure studies represents "a most disturbing development in the standard-setting process because it encourages advocacy through questionable extrapolations rather than scientific rigor." (Commenter 11) **RESPONSE:** The results of controlled exposure studies have generally been used in the formulation of short-term standards related to specific gases, exposures to which can be carefully tailored because of the uniform composition of the gas. In contrast, the heterogeneous nature of PM (size, physical state, chemical and biological composition, source mixtures), has until very recently posed a daunting challenge to the implementation of controlled human exposure studies involving ambient or concentrated ambient particles (other than model particles such as sulfuric acid). Therefore, the existing state and federal standards for PM have been based on epidemiological studies, recognizing the potential difficulties in the interpretation of such studies, particularly exposure misclassification. The limitations of epidemiological studies are acknowledged in the report, and have been taken into account in the recommendations for standards. This is not a new development in the standard-setting process, as suggested by the commenter: the existing California PM10 standards were set in 1983. In addition, the 24-hour SO_2 standard in California is also based solely on epidemiological studies. Moreover, epidemiological studies have been factored into the standard-setting process, at both state and federal levels, for ozone and nitrogen dioxide as well. Finally, controlled exposure studies are also subject to inherent limitations that affect their utility in standard-setting: (1) only short-term responses to relatively brief exposures (usually no more than several hours) can be evaluated; (2) there is often limited statistical power to detect effects, due to the typically small numbers of subjects; (3) controlling the experimental conditions may result in failure to capture effects found in complex real-world exposures; and (4) multiple selection biases in recruiting study subjects reduce the generalizability of such studies.

CHAPTER 7: DISTURBANCES OF THE CARDIAC AUTONOMIC SYSTEM

68. **COMMENT:** Limitations of study design and small numbers of subjects limit the utility of studies on heart rate variability (HRV) and others examining heart rate and rhythm; thus, it is premature to rely on these for deriving mechanistic hypotheses. However, "the Draft correctly cautions that 'it is unknown whether this relationship is causal or whether decreased HRV represents only an

epiphenomenon of more fundamental pathophysiological changes.'" On the other hand, it is "difficult to understand how the Draft concludes that studies of cardiac function in which high PM doses were administered to compromised experimental animals 'bolster the biological plausibility of the human studies' reporting statistically significant associations between ambient PM exposures and mortality and morbidity." (Commenter 12) **RESPONSE:** The Draft indicates that the human studies may have limited applicability for causal inference, as noted by the commenter. The full text of the sentence on animal studies in the Draft reads as follows: "Such investigations bolster the biological plausibility of the human studies, but are nevertheless limited by uncertainties related to cross-species extrapolation and high-level exposures used." Thus, in context, it is clear that OEHHA has indicated that the interpretation of the animal data is subject to inherent constraints. We have not changed the Draft in response to this comment.

CHAPTER 7: SUMMARY

69. **COMMENT:** The Draft "provides a thorough and nearly exhaustive listing of scientific data published on toxicology and potential mechanisms," but fails because: (1) there is no documentation that children are not protected by existing standards; (2) there is no "critical evaluation of the scientific validity and environmental relevance of the new data," which would demonstrate that the high doses used in these studies cannot be realistically extrapolated to ambient levels of exposure; (3) an authentically critical review would reveal that there is no "plausible and scientifically sound mechanism that would explain or support the causal role of low level PM pollution in the statistical associations observed in epidemiological studies." (Commenter 12) **RESPONSE:** This section was not intended to address the health-protectiveness of existing ambient air quality standards for PM. This issue was covered in more detail in OEHHA's review of all the health-based ambient air quality standards in California under the mandate of the Children's Environmental Protection Act during 2000, which is described in a joint staff report by the Air Resources Board and the Office of Environmental Health Hazard Assessment, entitled "Adequacy of California Ambient Air Quality Standards: Children's Environmental Health Protection Act," November 2, 2000.

    As noted in prior responses to comments, the revised Section 7.8 has been modified to clarify the limitations on the generalizability of the high PM doses used in experimental animal and in vitro studies, as well as the controlled human exposure investigations. However, it should be noted that a number of the epidemiological studies cited in this section examined potential mechanisms between ambient PM concentrations and acute responses (e.g., heart rate variability - Liao et al., 1999; Gold et al., 2000; Pope et al., 1999c; cardiac arrhythmias – Peters et al., 2000a). In studies such as these, cross-species and high-to-low dose extrapolations are not at issue.

CHAPTER 7: CAUSAL INFERENCE

70. **COMMENT:** There are sufficient difficulties in meeting each of the causal inference guidelines such that the Draft "significantly overstates the strength of the case for establishing causality for PM." The specific criticisms are generally presented in greater detail in other comments in this submission (e.g., Consistency and coherence of results, bias, confounding.) (Commenter 12) **RESPONSE:** We disagree with the commenter's assessment regarding causal inference. More detailed responses to the various specific points raised by the commenter are provided elsewhere in this appendix.

CHAPTER 8: WELFARE EFFECTS

71. **COMMENT:** Eleven minor comments on Chapter 8. (Commenter 11)

a) Pg. 229 line 40: There is a typographic error. **RESPONSE**: The commenter correctly identifies a typographic error – Rayleigh scattering is due to gases; variable should be Bsg. This has been corrected.

b) P230 lines 42-45: Absorption is much less size-sensitive than scattering. **RESPONSE**: The commenter correctly notes that absorption is much less size-sensitive than scattering. The sentence referred to emphasizes size effects on scattering; absorption is treated in the preceding sentences.

c) P233 lines 26-29: The commenter requests that a more recent statewide review of visibility be included. **RESPONSE**: A more recent statewide review of visibility would be desirable, but no such analysis exists. Contrary to commenter's contention, the data available (e.g. IMPROVE data for rural sites) do not show a significant improvement since the 1980s.

d) P240 Section 8.4: The commenter asserts that no adverse climate effects have been shown, and that reductions in some PM emissions may reduce aerosol cooling effects, thus exacerbating global warming. **RESPONSE**: While both statements are technically correct, the intensity of such effects, and California's contribution to them, are not known, and therefore can not be quantified in this document. The purpose for including this material in this document is to provide decisionmakers with a complete review of the potential consequences of regulating PM. The equivocal nature of current global assessments of climate effects of PM does not obviate the need for discussion.

e) P244 lines 21-25: The commenter asserts that PM – CO_2 linkage only exists for "natural emissions." **RESPONSE**: This is incorrect. On a continental to global scale, fossil fuel CO_2 emissions are highly correlated with combustion PM emissions, albeit at different mass ratios than for "natural" sources. The comment incorrectly implies that all biomass emissions are "natural;" in fact, a large fraction of vegetation burning is due to human ignition (see preceding paragraph on same page). Finally, natural dust emissions are completely uncorrelated with CO_2 emissions.

f) P249 Section 5.2.3: The data on California acid fog are dated. **RESPONSE**: The commenter correctly notes the California acid fog data are somewhat dated, and speculates that recent emission reductions may have ameliorated the problem. We are not aware of any more recent data, but would agree that present conditions are most likely no worse than when the data were collected, and present conditions may be somewhat improved due to decreased NO_x emissions statewide.

g) P250 Section 8.5.3: The commenter asserts that acidity effects are minimal. **RESPONSE**: We concur, but note that the purpose of this report is to review all effects of PM, not only those that currently pose serious risk.

h) P250 lines 19-24: The commenter asks what is the basis for the conclusion that aquatic systems are nitrogen limited and potentially at risk. **RESPONSE**: This paragraph should have referenced Melack and Sickman, 1997 (listed in references). This reference has been added.

i) P250 lines 25-31: Trout are not adversely affected by the present level of acid deposition. **RESPONSE**: The commenter notes that trout are not known to be adversely affected by present acid deposition, but confuses lack of effect with lack of risk. The Commenter correctly notes that there is a missing reference. It is: Jenkins, T. M. Jr., et al., 1994. Aquatic biota in

the Sierra Nevada: current status and potential effects of acid deposition on populations, Final Report, Contract A932-138. California Air Resources Board, Sacramento, CA.

j) P250 lines 38-42: Paragraph reports that it would take a 50 to 150 percent increase in acidic deposition to acidify the most sensitive Sierran lakes. **RESPONSE**: The commenter correctly notes that this provides no justification for additional controls. The purpose of including this information is to provide decisionmakers with an understanding of the "margin of safety" that exists under present circumstances.

CHAPTER 9: CONTROL ISSUES

72. **COMMENT:** There is no assurance that PM10 controls will effectively control PM2.5 as well. (Commenter 1) **RESPONSE**: California's PM10 control programs address both fine and coarse particles. Fine particles are typically controlled through statewide programs (such as reducing tailpipe emissions from cars and trucks, and requiring cleaner fuels) and district programs. These fine particle programs target both particulate precursors (such as NO_X and SO_X) and direct particulate emissions (such as diesel exhaust and woodsmoke). Coarse PM is generally controlled at the district level because sources tend to be local, for example dust controls. Because the ratio of coarse and fine particulate matter varies both geographically and seasonally, the types of additional measures needed to augment statewide controls must be tailored to local conditions.

CHAPER 10: BENEFITS ASSESSMENT

73. **COMMENT:** It is inappropriate to apply concentration-response functions to cities other than the one for which the function was derived. (Commenter 11) **RESPONSE**: This is a valid concern. To address this concern, we have assessed whether there is evidence that health effects of PM are different in California than in the rest of the country. Our conclusion was that there was not sufficient evidence to conclude that the health effects of PM are any different in California than they are anywhere else. The Samet et al. study of 90 cities shows a regional pattern of results with higher PM health effects in the Northeast than elsewhere in the country. Their results for California suggest that average effects in California are similar to the national average. It is true there is variability in results for a given health effect from different studies in different locations. Thus, there may be potential error in extrapolating coefficients from one location to another. Reasons for these differences are not identified sufficiently at this time to allow for adjustment for different locational characteristics. To the extent possible we have used studies from California, in order to lessen this potential bias. In some cases, such as restricted activity days, we have used a national study. The most important adverse health effect is premature mortality associated with ambient particulate pollution. Numerous studies have examined the impact of ambient particulate matter in areas throughout the United States, and the world. There is fairly good agreement that ambient particulate matter contributes to premature mortality. For our analysis, we have used the work by Krewski et al. (2000), which is widely considered the best epidemiological study to date examining the linkage between particulate matter and premature mortality. Krewski et al. included more than half a million participants from over 50 cities throughout the United States, including California.

The commenter also questions the validity of applying the coefficient estimated for one location to other locations because the coefficient is estimated based on the mean PM concentration for that location. Therefore, the coefficient cannot be applied to other cities unless the city of interest has the same mean PM concentration as the original study city. Krewski et. al. examined the issue of linearity or nonlinearity in the relationship between particulate matter and premature mortality, and

concluded that they could not rule out linearity. This suggests that it is not especially important whether the change in ambient particulate matter that we are examining is occurring at the mean or not.

To the extent possible, we have used the best epidemiological studies and baseline incidences appropriate for California. It is our judgment that using the best available methods is superior to a qualitative assessment or not do an assessment of the impact of air pollution.

The alternative implied by the comments is that we cannot say anything about the health benefits of air quality improvements in a location unless we have original health effects studies in that specific location. This is unreasonable.

74. **COMMENT:** There are no baseline data for some of the endpoints. (Commenter 11) **RESPONSE**: As a matter of fact, baseline incidence rates are available for each of the C-R functions that we have used. In the case of restricted activity days, we obtained national incidence rates from the National Center for Health Statistics. In the case of lower respiratory symptoms, we used a rate based on an epidemiological study of children in six US cities. The meaning of lines 17-20 on page 263 was that for some endpoints there were no baseline data other than the baseline data reported in the original studies.

    The comments also stated that it was not a scientifically valid methodology to use baseline incidence rates other than those reported in the original studies. In fact, the incidence rates reported in the original study from which we developed a C-R function are irrelevant for our application. The relevant issue is whether the C-R function and incidence rate are appropriate to estimate adverse health effects associated with air pollution in California. Clearly, some C-R functions and incidence rates are better than others. The question is whether the available data do a reasonable job of estimating the impact of air pollution on people's health in California. Once the best C-R function is selected, the original baseline incidence rates become irrelevant, because the C-R function we used is essentially a relationship of ratios or a percentage. The baseline rates or any scaling factor is not going to change the ratio. Therefore, instead of using the baseline incidence rate from the original study, we used California-specific baseline incidence rates when they were available.

75. **COMMENT:** All information used for the calculations in Tables 7.7 and 7.8 should be provided for public review. (Commenter 11) **RESPONSE**: Although we did not include PM concentration and population data in Tables 7.7 and 7.8, we did include PM2.5 concentration change and population data at county level in Table 10.9 and 10.10. With that, one can easily derive annual PM2.5 concentration. PM2.5 and PM10 concentration data at the air basin level were presented in Table 10.1. Baseline information for each study we used was described in detail in Sections 10.1.4 through 10.1.5.7. It is difficult to include all this information in a single table as suggested in the comments. However, this information will be added to the next version of the report.

76. **COMMENT:** The discussion describes only the thresholds for annual and long-term mortality. There is no description of how the short-term health-effect threshold was chosen, or of how background concentrations fluctuate. (Commenter 11) **RESPONSE**: To date, there is no clear evidence on whether there is a threshold of PM below which there are no detectable health effects. It is correct that long-term and short-term health effect thresholds could be different, and technically should be modeled differently. However, the intent of using annual average background as a threshold is to derive a more conservative PM health effects estimate as compared with not setting any threshold. It is likely that the short-term annual health effects

associated with background PM concentrations would be slightly higher if we use short-term-background PM concentrations because of the log linear functional form of the C-R function.

77. **COMMENT:** There are sign problems with the C-R functions. (Commenter 11) **RESPONSE**: We add negative signs to each C-R function, so that the result would be a positive number of health incidences avoided as a result of reducing PM concentration level. The definition of change in health incidence and PM concentration used in our calculation is baseline minus control. It is equivalent to the form of C-R function with a positive sign. However, we noticed a typo on page 262, line 34 – a negative sign before β should not be there – we will correct it in the next draft.

78. **COMMENT:** Table 10.2 and elsewhere: Many California cities are not included, leading to a consistency problem. (Commenter 11) **RESPONSE**: There are actually 12 California cities in the 90 NMMAPS cities. The report shows results for individual cities only on a chart, so it is difficult to determine the city-specific coefficients. This detail was provided for the 6 California cities in the 20-city analysis. As a result, we can only use the results for the 6 CA cities to evaluate whether the CA response is different than the national average.

APPENDIX 4: REFERENCES FOR CHAPTER 7 RESPONSES

Air Resources Board (1998). Allergens in paved road dust and airborne particles. August 1998. Sacramento, CA: California Environmental Protection Agency

Air Resources Board (2000). Adequacy of California's Ambient Air Quality Standards: Children's Environmental Health Protection Act. November 2, 2000. Sacramento, CA: California Environmental Protection Agency.

Bates DV (1992). Health indices of the adverse effects of air pollution: the question of coherence. Environ Res 59(2):336-49.

Burnett RT, Cakmak S, Brook JR (1998a). The effect of the urban ambient air pollution mix on daily mortality rates in 11 Canadian cities. Can J Public Health 89(3):152-6.

Burnett RT, Cakmak S, Raizenne ME, Stieb D, Vincent R, Krewski D *et al*. (1998b). The association between ambient carbon monoxide levels and daily mortality in Toronto, Canada. J Air Waste Manag Assoc 48(8):689-700.

Burnett RT, Brook JR, Dann T, Delocla C, Philips O, Calmak S *et al* . (2000). Association between particulate- and gas-phase components of urban air pollution and daily mortality in eight Canadian cities. In: Grant LD, ed. PM2000: Particulate Matter and Health. Inhal Toxicol 12(Suppl. 4):15-39.

Cakmak S, Burnett RT, Krewski D (1999). Methods for detecting and estimating population threshold concentrations for air pollution-related mortality with exposure measurement error. Risk Anal 19(3):487-96.

Daniels MJ, Dominici F, Samet JM, Zeger SL (2000). Estimating particulate matter-mortality dose-response curves and threshold levels: an analysis of daily time-series for the 20 largest US cities. Am J Epidemiol 152(5):397-406.

Dockery DW, Pope CA III, Xu X, Spengler JD, Ware JH, Fay ME *et al*. (1993). An association between air pollution and mortality in six U.S. cities. N Engl J Med 329:1753-9.

Fairley D (1999). Daily mortality and air pollution in Santa Clara County, California: 1989-1996. Environ Health Perspect 107(8):637-41.

Gold DR, Litonjua A, Schwartz J, Lovett E, Larson A, Nearing B *et al*. (2000). Ambient pollution and heart rate variability. Circulation 101(11):1267-73.

Gwynn RC, Burnett RT, Thurston GD (2000). A time-series analysis of acidic particulate matter and daily mortality and morbidity in the Buffalo, New York, region. Environ Health Perspect 108(2):125-33.

Hill AB (1965). The environment and disease: association or causation? Proc R Soc Med 58:295-300.

Krewski D, Burnett R, Goldberg MS, Koover K, Siemiatycki J, Jerrett M *et al*. (2000). Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Res Rep Health Eff Inst (A special report of the Institute's Particle Epidemiology Reanalysis Project).

Liao D, Creason J, Shy C, Williams R, Watts R, Zweidinger R (1999). Daily variation of particulate air pollution and poor cardiac autonomic control in the elderly. Environ Health Perspect 107(7):521-5.

Lillquist Dr, Lee Js, Ramsay Jr, Boucher KM, Walton ZL, and Jl Lyon. A comparison of indoor/outdoor PM10 concentrations measured at three hospitals and a centrally located monitor in Utah. Applied Occup. Environ. Hyg. 13(6), June 1998, p.409-415.

Linn WS, Gong, Jr H, Clark KW, Anderson KR. Day-to-day particulate exposure and health changes in Los Angeles area residents with severe lung disease. J Air Waste Manage Assoc 1999; Special Issue on PM; 49: PM108-115.

Lipfert FW, Wyzga RE (1999). Statistical considerations in determining the health significance of constituents of airborne particulate matter. J Air Waste Manag Assoc 49(9 Spec No):182-91.

Lipfert FW, Perry HM Jr, Miller JP, Baty JD, Wyzga RE, Carmody SE (2000b). The Washington University-EPRI Veterans' Cohort Mortality Study: Preliminary Results. Inhal Toxicol 12(Suppl. 4):41-73.

Morgan G, Corbett S, Wlodarczyk J, Lewis P (1998). Air pollution and daily mortality in Sydney, Australia, 1989 through 1993. Am J Public Health 88:759-64.

Mukae H, Vincent R, Quinlan K, English D, Hards J, Hogg JC *et al*. (2001). The effect of repeated exposure to particulate air pollution (PM10) on the bone marrow. Am J Respir Crit Care Med 163(1):201-9.

Nemmar A, Vanbilloen H, Hoylaerts MF, Hoet PH, Verbruggen A, Nemery B (2001a). Passage of Intratracheally Instilled Ultrafine Particles from the Lung into the Systemic Circulation in Hamster. Am J Respir Crit Care Med 164(9):1665-8.

Nemmar A, Vanbilloen H, Verbruggen A *et al.* (2001b). Evaluation of the passage of inhaled 99Tc-labelled ultrafine carbon particles into the systemic circulation in humans. Am J Respir Crit Care Med .

Nemmar A, Hoet PH, Vanquickenborne B, Dinsdale D, Thomeer M, Hoylaerts MF *et al*. (2002). Passage of inhaled particles into the blood circulation in humans. Circulation 105(4):411-4.

Ostro BD, Broadwin R, Lipsett MJ (2000). Coarse and fine particles and daily mortality in the Coachella Valley, California: a follow-up study. J Expo Anal Environ Epidemiol 10(5):412-9.

Perry HM Jr, Schnaper HW, Meyer G, Swatzell R (1982). Clinical program for screening and treatment of hypertension in veterans. J Natl Med Assoc 74(5):433-44.

Peters A, Liu E, Verrier RL, Schwartz J, Gold DR, Mittleman M *et al*. (2000a). Air pollution and incidence of cardiac arrhythmia. Epidemiology 11(1):11-7.

Peters A, Dockery DW, Muller JE, Mittleman MA (2001a). Increased particulate air pollution and the triggering of myocardial infarction. Circulation 103(23):2810-5.

Peters A, Frohlich M, Doring A, Immervoll T, Wichmann HE, Hutchinson WL *et al*. (2001b). Particulate air pollution is associated with an acute phase response in men; results from the MONICA-Augsburg Study. Eur Heart J 22(14):1198-204.

Pope CA III (1996). Particulate pollution and health: a review of the Utah valley experience. J Expo Anal Environ Epidemiol 6(1):23-34.

Pope CA III, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE *et al*. (1995). Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Respir Crit Care Med 151:669-74.

Pope CA III, Hill RW, Villegas GM (1999a). Particulate air pollution and daily mortality on Utah's Wasatch Front. Environ Health Perspect 107(7):567-73 .

Pope CA III, Dockery DW, Kanner RE, Villegas GM, Schwartz J (1999b). Oxygen saturation, pulse rate, and particulate air pollution: a daily time-series panel study. Am J Respir Crit Care Med 159:365-72.

Pope CA III, Verrier RL, Lovett EG, Larson AC, Raizenne ME , Kanner RE *et al*. (1999c). Heart rate variability associated with particulate air pollution. Am Heart J 138(5 Pt 1):890-9.

Rothman KJ (1982). Causation and causal inference. In: Schottenfeld D, Fraumeni JF. Cancer Epidemiology and Prevention. Philadelphia, PA: W.B Saunders Co., 15-22.

Samet JM, Zeger SL, Dominici F, Curriero F, Coursac I, Dockery DW *et al*. (2000a). The National Morbidity, Mortality, and Air Pollution Study. Part II: Morbidity and mortality from air pollution in the United States. Res Rep Health Eff Inst (94 Pt 2):5-70; discussion 71-9.

Sarnat JA, Schwartz J, Catalano PJ, Suh HH (2001). Gaseous pollutants in particulate matter epidemiology: confounders or surrogates? Environ Health Perspect 109(10):1053-61.

Schimmel H, Murawski TJ (1976). Proceedings: The relation of air pollution to mortality. J Occup Med 18(5):316-33.

Schwartz J (2000a). Assessing confounding, effect modification, and thresholds in the association between ambient particles and daily deaths. Environ Health Perspect 108(6):563-8.

Schwartz J, Zanobetti A (2000). Using meta-smoothing to estimate dose-response trends across multiple studies, with application to air pollution and daily death. Epidemiology 11(6):666-72.

Schwartz J, Dockery DW, Neas LM (1996). Is daily mortality associated specifically with fine particles? J Air Waste Manag Assoc 46:927-39.

Smith RL, Spitzner D, Kim Y, Fuentes M (2000). Threshold dependence of mortality effects for fine and coarse particles in Phoenix, Arizona. J Air Waste Manag Assoc 50(8):1367-79.

Snipes MB, James AC, Jarabek AM (1997). The 1994 ICRP66 human respiratory tract dosimetry model as a tool for predicting lung burdens from exposures to environmental aerosols. Appl Occup Environ Hyg 12:547-54.

Tan WC, Qiu D, Liam BL, Ng TP, Lee SH, van Eeden SF *et al*. (2000). The human bone marrow response to acute air pollution caused by forest fires. Am J Respir Crit Care Med 161(4 Pt 1):1213-7 .

Tropp RL, Chow JC, Kohl SD (2000). PM2.5 chemical speciation results in Texas. In. PM2000: Particulate Matter and Health, The Scientific Basis for Regulatory Decision-Making, Charleston, SC, 1/24-28/2000. Pittsburgh, PA: Air & Waste Management Assn.

U.S. Environmental Protection Agency (1996). Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information: OAQPS staff

paper. Office of Air Quality Planning and Standards, Research Triangle Park, NC, EPA-452\R-96-013.

Vostal JJ (2000). Statistical associations between ambient particulate matter and daily morbidity and mortality: Can we identify mechanisms responsible for these health effects? In: Proc. Air & Waste Management Assn., 93rd Ann Conf., Salt Lake City, 6/18-22/2000.

Wordley J, Walters S, Ayres JG (1997). Short term variations in hospital admissions and mortality and particulate air pollution. Occup Environ Med 54(2):108-16.

Zmirou D, Schwartz J, Saez M, Zanobetti A, Wojtyniak B, Touloumi G *et al*. (1998). Time-series analysis of air pollution and cause-specific mortality. Epidemiology 9:495-503.