

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 (PM₄₀10). Suspended particulate matter (PM₄₀10) refers to atmospheric particles, solid and liquid, except uncombined water as measured by a (PM₄₀10) 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 (PM₄₀10) is to be measured by the size-selective inlet high volume (SSI) PM₄₀ sampler method in accordance with ARB Method P, as adopted in August 22, 1985, or by an equivalent (PM₄₀) sampler method a California Approved Sampler (CAS) for PM10, for purposes of monitoring for compliance with the Suspended Particulate Matter (PM₄₀10) 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 (SO₄) ion (SO₄²⁻) 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)	50µg/m ³ PM ₁₀ ** 30µg/m ³ PM ₁₀ ** 20µg/m ³ PM ₁₀ ** 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 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 (PM _{2.5})	25µg/m ³ PM _{2.5} ** 12µg/m ³ PM _{2.5} ** 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 PM _{2.5} 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 (Turbidimetric Barium Sulfate) MLD Method 007	24 hours	a. Decrease in ventilatory function b. Aggravation of asthmatic symptoms c. Aggravation of cardiopulmonary 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



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.

- PM10 Annual-average standard – Lower the standard from the current 30 $\mu\text{g}/\text{m}^3$ to 20 $\mu\text{g}/\text{m}^3$ and revise the averaging method to an annual arithmetic mean.
- PM10 24-hour-average standard – Retain the current standard at 50 $\mu\text{g}/\text{m}^3$.
- PM2.5 Annual-average standard – Establish a new annual arithmetic mean standard at 12 $\mu\text{g}/\text{m}^3$.
- Sulfate 24-hour-average standard – Retain the current 25 $\mu\text{g}/\text{m}^3$ 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 $\mu\text{g}/\text{m}^3$, 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 bronchopulmonary dysplasia, 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 PM_{2.5} 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 PM_{2.5} standard. The committee recommended that *a priori* criteria be established to guide decisions about the appropriate level and that a 24 hour PM_{2.5} 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 (PM_{2.5-10}).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 ($1\text{ nm} \leq d_p \leq 0.1\ \mu\text{m}$)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 $d_p \leq 0.1\ \mu\text{m}$ (100 nm).

p. 12, l. 46, add reference Friedlander 2000³

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 PM_{2.5}. 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 PM_{2.5} NAAQS. The proposal for measurement will be to use two side-by-side PM_{2.5} FRM samplers where the WINS impactor will be replaced 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 PM_{2.5}. The committee also agrees with the recommended methods for adopting samplers that meet the Federal Reference Method requirements for PM₁₀ and PM_{2.5} 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 PM_{2.5} 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 PM_{2.5} shown in the figures in this chapter need to be considered with respect to ability of PM₁₀ regulations to also control PM_{2.5} 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 PM_{2.5} 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 PM₁₀ 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 PM_{2.5} monitors would indicate relatively uniform population exposures.

Furthermore, ambient PM₁₀ or PM_{2.5} 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 PM_{2.5} or PM₁₀ data provide an overly simplified estimate of exposure, which will inevitably lead to substantial errors and uncertainty 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 al.*, *Epidemiology*, 2001).

Therefore, this speculation needs to be tempered. This same comment applies to page 129, lines 36-43.

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

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 $\mu\text{g}/\text{m}^3$ from the USEPA analysis that used the same data but arrived at a 25 $\mu\text{g}/\text{m}^3$ 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 $\mu\text{g}/\text{m}^3$ was chosen as the level for the 24 hours standard. Why not 11 or 13 $\mu\text{g}/\text{m}^3$?
- 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 PM_{2.5} 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 PM_{2.5} 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 $12 \mu\text{g}/\text{m}^3$, 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 $25 \mu\text{g}/\text{m}^3$, not to be exceeded. The adoption of the recommendation for an annual PM2.5 standard of $12 \mu\text{g}/\text{m}^3$ was considered to be an integral component of the proposal.

The AQAC had been concerned that the proposed standard based on attaining a $12 \mu\text{g}/\text{m}^3$ 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 PM_{2.5}.

The committee met in a public forum on April 3, 2002 to discuss the proposed 25 µg/m³ PM_{2.5} 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 subpopulations 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 PM_{2.5}?

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 PM_{2.5}?

The committee recommended that a 24-hr PM_{2.5} 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, NO₃);
- Characterize ultrafine exposures (Indoor, Outdoor, personal);
- Validate new or improved monitoring techniques, especially continuous monitors of PM_{2.5}, 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 PM_{2.5} 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 (PM_{2.5-10}). 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 ($1\text{nm} \leq d_p \leq 0.1 \mu\text{m}$). 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 $d_p \leq 0.1 \mu\text{m}$ (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 PM_{2.5}. The Committee also agrees with the recommended methods for adopting samplers that meet the Federal Reference Method requirements for PM₁₀ and PM_{2.5} 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, line 39; 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 PM_{2.5} 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 PM_{2.5} shown in the figures in chapter 6 need to be considered with respect to ability of PM₁₀ regulations to also control PM_{2.5} 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 PM_{2.5} 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 PM₁₀ 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 PM_{2.5} monitors would indicate relatively uniform population exposures.

Furthermore, ambient PM₁₀ or PM_{2.5} 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 PM_{2.5} or PM₁₀ data provide overly simplified estimates of exposure, which will inevitably lead to substantial errors and uncertainty 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 al.*, *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 $\mu\text{g}/\text{m}^3$, mean PM_{2.5}=18.4 $\mu\text{g}/\text{m}^3$].”*
- 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 $\mu\text{g}/\text{m}^3$ from the USEPA analysis that used the same data but arrived at a 25 $\mu\text{g}/\text{m}^3$ 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 $\mu\text{g}/\text{m}^3$ annual average standard for PM_{2.5}, not 25 $\mu\text{g}/\text{m}^3$. 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 PM_{2.5} standard. However, as discussed in the document, there are a few studies linking PM_{2.5} with mortality and morbidity, in which the long-term mean concentrations were below 15 $\mu\text{g}/\text{m}^3$ PM_{2.5}.

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 $\mu\text{g}/\text{m}^3$ was chosen as the level for the 24 hours standard. Why not 11 or 13 $\mu\text{g}/\text{m}^3$?
 - 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 $\mu\text{g}/\text{m}^3$ 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 PM_{2.5} and both mortality and morbidity. Specifically, consideration of a standard at 12 $\mu\text{g}/\text{m}^3$ 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 PM_{2.5} and mortality. The mean PM_{2.5} concentration was 18 $\mu\text{g}/\text{m}^3$ (range of 11.0 to 29.6 $\mu\text{g}/\text{m}^3$) in the Six-Cities study and 20 $\mu\text{g}/\text{m}^3$ (range of 9.0 to 33.5 $\mu\text{g}/\text{m}^3$) 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 PM_{2.5} concentrations of 11 and 12.5 $\mu\text{g}/\text{m}^3$. Larger increases in risk don't occur until the long-term PM_{2.5} mean equals 14.9 $\mu\text{g}/\text{m}^3$. Therefore, an annual standard of 12 $\mu\text{g}/\text{m}^3$ would be below the mean of the most likely effects level and would provide a margin of safety. Targeting a long-term mean PM_{2.5} concentration of 12 $\mu\text{g}/\text{m}^3$ would also place some weight on the results of multiple daily exposure studies examining relationships between PM_{2.5} and adverse health outcomes (Table 7.2). These studies have long-term (three- to four-year) means in the range of 13 to 18 $\mu\text{g}/\text{m}^3$. A standard set at 12 $\mu\text{g}/\text{m}^3$ 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 PM_{2.5} standard of 12 $\mu\text{g}/\text{m}^3$ 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 PM_{2.5} 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 PM_{2.5} standard of 25 µg/m³, 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 µg/m³ 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 PM_{2.5} 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 PM_{2.5} daily concentrations. **RESPONSE:** The revised document now includes a recommendation for a 24-hr PM_{2.5} standard of 25 µg/m³, 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) PM_{2.5} 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 PM_{2.5} 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 PM_{2.5} standard of 25 µg/m³, 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 Health 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 PM_{2.5}-mass monitors are expected to be deployed throughout California by some time in 2002 as part of the California continuous PM_{2.5}-mass monitoring network. Of these, 21 are already committed. The primary objective of continuous PM_{2.5} 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) **RESPONSE:** 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 ($\mu\text{g}/\text{m}^3$)	Annual Average PM2.5 ($\mu\text{g}/\text{m}^3$)
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 Geronio 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 $\mu\text{g}/\text{m}^3$ 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 PM_{2.5}) 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 ($50 \mu\text{g}/\text{m}^3$ 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 $65 \mu\text{g}/\text{m}^3$. 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 $50 \mu\text{g}/\text{m}^3$ 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. (Commenters 1, 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 $50 \mu\text{g}/\text{m}^3$. Therefore, it is at least as protective as a PM2.5 standard set to $50 \mu\text{g}/\text{m}^3$. However, because PM10 will include some coarse component, the proposed standard is more protective than a PM2.5 standard of $50 \mu\text{g}/\text{m}^3$ would be by itself. The USEPA 24-hour PM2.5 standard is currently $65 \mu\text{g}/\text{m}^3$.
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 ⅓ 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 PM_{2.5}. 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 epidemiology 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 PM_{2.5} 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) **RESPONSE:** 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 PM_{2.5} 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 ($>1 \mu\text{m}$) “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 PM_{2.5} 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 PM_{2.5} 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 PM_{2.5}. (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 $\mu\text{g}/\text{m}^3$ probably does imply an annual average between 8 and 10 $\mu\text{g}/\text{m}^3$, which may be at or near "background" levels for PM_{2.5}. 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 PM_{2.5}.

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 PM_{2.5} 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 cited 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 SO₂ 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 PM_{2.5} 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, PM_{2.5}, the coarse fraction (PM₁₀-PM_{2.5}), 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 $\mu\text{g}/\text{m}^3$ 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 $\mu\text{g}/\text{m}^3$ 24-h PM₁₀ and respiratory admissions increase from 5 to 25% per 50 $\mu\text{g}/\text{m}^3$ 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 PM_{2.5} 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 ± 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, PM₁₀, PM_{2.5}, PM₁₅, PM_{15-2.5}, SO₄, O₃, CO, and NO₂ 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 ($SO_4 = > PM_{2.5} > PM_{10}$).” 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, NO₂, and SO₂ 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 SO₂ 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 PM_{2.5} in several studies, including an innovative study by Sarnat and colleagues (2001), who found no correlation between ambient concentrations and personal exposures for O₃, NO₂, and SO₂. Furthermore, it was shown that ambient O₃, NO₂, and SO₂ concentrations **did** correlate with personal PM_{2.5}. While wider confirmation is needed, these findings imply that ambient concentrations of gaseous co-pollutants can serve as surrogates for personal PM_{2.5} 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 PM_{2.5} 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 O₃, SO₂, NO₂ 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 SO₂, 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 SO₂ 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 SO₂ 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 SO₂ 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 SO₂ 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 SO₂.

Overall, the statistical importance of SO₂ in the Krewski et al. (2000) sensitivity results seems unlikely to result from a true mortality health effect of SO₂ *per se*, but because it is another marker for fossil fuel combustion-related particles that form from the SO₂ 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 SO₂-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 SO₂ 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 PM_{2.5}, as well as a potential threshold at around 20 µg/m³ and based on the graphical analysis, effects at concentrations greater than 20 µg/m³. 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 $\mu\text{g}/\text{m}^3$, based on conversion from co-located nephelometry data), Wordley et al. (1997) for Birmingham, UK (mean = 26 $\mu\text{g}/\text{m}^3$), Schwartz et al. (1996) for the Harvard Six-Cities (mean = 25 $\mu\text{g}/\text{m}^3$), Burnett et al. (2000) for the eight largest Canadian cities (mean = 26 $\mu\text{g}/\text{m}^3$), and Gwynn et al. (2000) for Buffalo and Rochester (mean = 24 $\mu\text{g}/\text{m}^3$). 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 $\mu\text{g}/\text{m}^3$. 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 $\mu\text{g}/\text{m}^3$ 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 PM_{2.5} concentrations and daily mortality in the Harvard Six Cities dataset. When they restricted the analysis to days on which the PM_{2.5} 24-hour

average concentrations equalled or exceeded 30 or 25 $\mu\text{g}/\text{m}^3$, 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 $\mu\text{g}/\text{m}^3$. On the other hand, Smith et al. (2000) statistically examined the threshold issue in data on mortality and ambient PM_{2.5} from Phoenix, AZ. They reported evidence of a significant change in the regression slope at a concentration of around 20 to 25 $\mu\text{g}/\text{m}^3$ PM_{2.5}, 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 PM_{2.5} 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 PM_{2.5} values), and Bayesian models (comparing the likelihoods of various thresholds) to explore the evidence for a nonlinear exposure-response at low PM_{2.5} concentrations. In general, staff found that a linear, nonthreshold model within the concentration range of interest for PM_{2.5} 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 PM_{2.5} 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 $\mu\text{g}/\text{m}^3$ 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 PM_{2.5} 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 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.

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 $\mu\text{g}/\text{m}^3$ 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 $\mu\text{g}/\text{m}^3$ would be in excess of 40 $\mu\text{g}/\text{m}^3$. Thus, adoption of a 24-hour standard of 25 $\mu\text{g}/\text{m}^3$ 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 PM_{2.5} 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/m³); 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 PM_{2.5} 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/cm²-d				
Total mass	0.0182	0.0210	0.028	0.029
Sulfate (SO ₄)	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 (SO ₄)	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/m³ 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-dose 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 (intraparyngeal) 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 SO₂ 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 – CO₂ linkage only exists for “natural emissions.” **RESPONSE:** This is incorrect. On a continental to global scale, fossil fuel CO₂ 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 CO₂ 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.

CHAPTER 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 PM_{2.5} concentration change and population data at county level in Table 10.9 and 10.10. With that, one can easily derive annual PM_{2.5} concentration. PM_{2.5} 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

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