

Ozone Standard Review Staff Report Summary of Comments (by Commenter) And Staff Responses

Note: Comments are in regular type, and responses are *italicized*.

David Bates

Several suggestions/differences in emphasis related to specific health studies, but no disagreement with conclusions/findings.

1. Page 11-12: The point might be made that there is concordance between the dosimetric calculations of the target area for the highest concentration of ozone (the terminal bronchiole), and the observed morphological effects, which is the centriacinar region. The dosimetric calculations also indicate the higher delivered dose of ozone as the tidal volume increases, and this is consistent with the increased effects on exercise.

This point is addressed in the report in the section on the effective dose concept.

2. Page 11-15: The complex problem of the variation of effect with different time courses of ozone delivery is well described. The genetic basis for differences in sensitivity to ozone demonstrated in breeding experiments deserves more analysis.

We are unclear what breeding experiments the commenter is referring to. Although it is generally agreed that there is a genetic contribution to between-subject differences in sensitivity to ozone, research on this topic is just beginning. Investigations into possible contributions from several genes are underway, but data are not yet available.

3. Page 11-45: If the length of time between exposures is important, how can this be related to the time course of exposure that would usually occur to an exposed child? This is mentioned on Page 11-46: "The episodic nature of ambient exposure conditions in humans suggests that reliable assessments of risk must include a clear understanding of the impact of cyclic exposure ". There is no follow-up as to how this might be done.

The statement on p 11-46 was not meant to imply that we could quantitatively evaluate the effects of cyclic exposure in adults or in children, but to point out the responses observed in animals from cyclic exposure. Animal studies give some indications as to exposure intervals of concern, but unfortunately the data are not readily extrapolated to humans at this point.

4. Page 11-48: First paragraph: the FEV1 has the smallest coefficient of variation, but the FEF25-75 is much more sensitive than the FEV1 to changes in terminal bronchioles. More emphasis on the work of Weinman on the small

airway effects of ozone is needed. This is important to offset the early FVC change which is due to stimulation by ozone of the C-fiber system – the changes in small airways are slower to resolve and very likely more important in terms of long term effects.

The issue of small airway effects has been alluded to with reference to several papers reviewed in the Staff Report, although it has not been discussed as a separate topic. We will add a section discussing small airway effects in the revised report. It should be noted that there is little literature on the effects of ozone on the small airways, and more research on this topic would be useful.

5. Page 11-51: Is it fair to assume that human variability in response to ozone is genetic in origin? What is the role of anti-oxidants such as superoxide dismutase? What about the protective effect of Vitamin C?

It is likely that the largest part of the variability is genetically based. Differences between individuals in superoxide dismutase or other anti-oxidant enzymes are largely genetically determined. Anti-oxidant vitamins have been shown to influence responsiveness to ozone, but do not necessarily fully mitigate responses to ozone exposure. Antioxidant vitamin supplements are likely a modifying factor, rather than a determinant factor in responsiveness.

6. Page 11-52: The reader should be told that although a single subject may have a meaningful threshold value for the effects of ozone, no such threshold is derivable for a group if a statistically significant shift in the mean is taken as the criterion of some effect.

This is true. A group mean value does not represent a population threshold. In fact, due to the variability between individuals, determining a population threshold implies finding the threshold level for the most responsive people, which is likely to be a very low concentration. We will clarify this point in the revised report.

7. Page 11-87: Insufficient attention is given to the work of Frank, R. et al (Repetitive ozone exposure of young adults: evidence of persistent small airway dysfunction: Am J Respir Crit Care Med 164: 1253-1260; 2001). The reference is quoted on page 11-226. In evaluating acute exposure data, it is important to separate the early FVC effect due to stimulation of the C-fiber system, and the later and more persistent small airway effects as shown by these authors. Their work also suggests that the reduced effect of ozone on subsequent days after an initial effect is to be explained by the protective mucus layer induced by the inflammatory response to the exposure on the first day, which has the effect of diminishing the response on subsequent days. These observations are relevant to standard setting.

We will discuss this paper more fully in the section to be added on small airway function. However, it should be pointed out that the primary measure of small

airway function used in this paper is a unique measurement that was developed by the investigators. It has not been validated or used in any other study. In addition, although the investigators speculate that increased mucus production may explain their findings, there were no measurements made in the experiment that could support or refute the suggested mechanism.

8. Page 11-92: The complex data on asthmatics is well described here.

Thank you for the comment.

9. Page 11-110: The emphasis on the joint ozone/allergen exposures is important, even though, as noted on the top of page 11-111, “they do not directly contribute to the evaluation of the level of the standard”. It should be noted here that sequential exposures to ozone and allergens must be very common in real life situations.

We covered this information because it addresses a common exposure pattern, and one that explores a possible explanation for observations that asthmatics have higher risk of being admitted to the emergency room or hospital on high ozone days.

10. Page 11-112: Summary: the Southern California Children’s study found that lung development, as judged by lung function tests, was being adversely affected by exposures to vehicle exhausts, but higher exposures to ozone were without effect.

These studies are discussed on pg 12-52, in the review of epidemiology studies. Although ozone effects on lung function were weak, associations between ozone exposure and other effects were found.

11. Page 11-114; second paragraph: the point might be made that exacerbations of asthma are now thought to be primarily inflammatory in nature and hence aggravation by ozone, which causes inflammation at very low doses, is to be expected.

We agree that it is quite likely that for many asthmatics, inflammation related to ozone exposure may represent an additive effect and be of particular concern. We will alter our text to reflect this point.

12. Page 11-127: Penultimate paragraph: might be better expressed as follows: “Chronic obstructive pulmonary disease, as well as chronic asthma, lead to nonuniform distribution of inhaled air in the lungs. This will have the effect of increasing the delivered dose of an inhaled pollutant to the regions of the lung which are relatively over-ventilated”.

Thank you for the suggestion. We will consider the wording of the paragraph.

13. Page 11-149: The interaction between heat stress and the effects of ozone is important, and as noted below, there have been recent attempts to separate the higher mortality in heat waves into the deaths attributable to heat and the deaths attributable to the concomitant elevated ozone levels. Increased temperature leads to increased ventilation, which in turn will increase the delivered dose of ozone to the lungs.

This point is well taken, and in fact heat may contribute to increased delivered dose. However, activity levels, especially outdoors, tend to be lower on very hot days. Also, the chamber studies that have investigated this topic did not find that concurrent heat exposure altered responses compared to those observed with completion of the same protocol at room temperature.

14. Page 11-172: Second paragraph: note the work of Frank et al which suggests that the mucus secretion initiated by the first ozone exposure plays a part in lessening the effect (on FVC) of subsequent exposures. It should be noted that it is not clear whether successive exposures result in a reduced effect at the level of the small airways, although the work of Christian et al noted on Page 11-173 suggests that the effects on distal airways may also be attenuated. As noted on Page 11-174, whether this applies to lung tissue is unclear. These distinctions should be made clear in the Summary on page 11-174. My opinion is that the reduced FVC response on successive exposures cannot be assumed to indicate a reduction of effect in other parameters within the lung.

See comments above regarding the Frank et al. paper page 11-87. Christian et al. do not report small airway function data, although the bronchoalveolar lavage fluid analysis suggests that with four consecutive days of exposure to ozone some, but not all, inflammatory measures had shifted toward the normal range. However, the measured values suggest that after four days of exposure inflammation was still evident in the lower airways. We agree with the commenter's opinion that FVC is not necessarily representative of all responses. We will edit the text for clarity.

15. Page 11-177: In the Summary, a reference should be given to the reduction in exercise performance noted at ozone levels of 0.06 ppm.

Thank you for the suggestion. We will add this to the document.

16. Page 11-198: Tokyo-Yokohama asthma was almost certainly due to high particulate and SO₂ levels and had nothing to do with ozone. It is not really relevant to this review.

Your point is well taken. We had it in the document because it was an early recognition that air pollution might affect asthma. We will clarify this in the document.

17. Page 11-200: Peden's observation about an increased eosinophilic response should be put earlier when the interaction of ozone and allergens was being reviewed.

Thank you for this suggestion. We will consider this.

18. Page 11-207: First paragraph: more emphasis should be given to this work in the interaction between combined O₃ and allergen exposures.

Although interesting, this material is not part of the basis for the standards recommendations; it serves as important supporting material. In addition, it is difficult to extrapolate between monkeys and humans so that the material could be used quantitatively.

19. Page 11-211: Pollutant mixtures: More discussion is needed on the factors affecting simultaneous exposure to ozone on the one hand, and to vehicle exhaust on the other. Perhaps a few paragraphs specifically on patterns of exposure would be helpful. This is because PM_{2.5} in the urban environment is associated with a variety of adverse health effects.

Little is known about combined exposure to particulate matter and ozone in human or animal subjects. The small amount of available literature suggests that ozone is more significant than particulate matter in inducing acute respiratory effects.

CHAPTER 12:

20. An important point should be mentioned at the outset, which is that it is now known that a peak in asthma attendances and admissions occurs in the third week of September. This was first documented in Vancouver (see Environ Research 51: 51-70; 1990 quoted in another context in the reference list here) but has since been shown by the group at McMaster (see ATS Abstracts) to occur across Canada. It is independent of air pollution, but may interfere with ongoing panel studies by obscuring an association with air pollution during other periods of the year. See Gent et al 2003 quoted here for a September asthma peak not detected by the authors, which might have affected their ongoing panel study. See annotation of the Gent study also in the second paragraph on Page 12-5.

This is not addressed in the epidemiological literature we reviewed. We will investigate this point, and revise the section appropriately. The fall peak in asthma would on average add noise to epidemiological studies but could also

bias results of an individual study if by chance it correlated with either an episode (unlikely given the season) or a trough in ozone concentrations. We will note this in the document. For the studies that are of longer-term duration such as those examining hospital admissions, this should not have a major impact on the findings.

21. Page 12-3: In relation to data on PM2.5 and ozone in Mexico City, see comment on pg. 11-211.

See response to comment on pg. 11-211 above.

22. Page 12-4: A comment should be added to the note on Brauer's study that the ozone exposures were measured by personal badges as well as by an ozone monitor very close to the workers.

We will add this to the document.

23. Page 12-7: The recent study by Hall et al of the economic costs of school absences, based on the Gilliland study, might be noted here.

We can note the study. However, the Hall study examines the quantitative implication of the Gilliland study for the L.A. basin. We have conducted our own quantification using this study and others, using more recent and complete data.

24. Page 12-23: I was surprised that no mention was made of the Atlanta study: FRIEDMAN, M.S., POWELL, K.E., HUTWAGNER, L., GRAHAM, L.M., & TEAGUE, W.G. Impact of changes in transportation and Commuting behaviors during the 1996 Summer Olympic Games in Atlanta on Air Quality and Childhood Asthma. JAMA 2001: 285; 897-905.

For many people, the documentation of a reduced adverse health effect synchronous with a reduced ambient ozone level constitutes very convincing evidence that the data being derived from epidemiological associations is real. My own opinion is that this study deserves special emphasis, not least when the effect of a possible "standard" is being discussed.

Thank you for pointing this out. Omission of this study was an oversight. We intend to add it to the next draft of the report.

25. Page 12-25: This comment on the Petroeschovsky study in Brisbane fails to make two important points, first that it involved over 13,000 hospital admissions for asthma, and second that aerosol sulfates were not present so the effect was due to ambient ozone alone.

Thank you for this suggestion. We will add these two good points to the text.

26. Page 12-39: Last paragraph: “On this issue, the evidence is fairly supportive of independent effects for ozone”. This is too weak a statement in my opinion. It should read: “On this issue, the evidence is conclusive that ozone is responsible for exerting direct effects” – see data from Mexico City and from Brisbane and Atlanta already discussed.

We will remove the word “fairly” and just say supportive.

Joint submission endorsed by: American Chemistry Council, California Business Properties Association, California Cement Manufacturers Environmental Coalition, California Chamber of Commerce, California Citrus Mutual, California Cotton Ginners Association, California Cotton Growers Association, California Farm Bureau Federation, California Independent Oil Marketers Association, California Independent Petroleum Association, California League of Food Processors, California Manufacturers and Technology Association, California Natural Gas Producers Association, Construction Materials Association of California, Council of Shopping Centers, Industrial Environmental Association, National Association of Industrial and Office Properties-California Chapters, Nisei Farmers League, Retail Industry Leaders Association.

1. There is not sufficient scientific support for the proposed 8-hr standard.

The commenter may misunderstand the CA definition of ambient air quality standards. In California, ambient air quality standards represent the highest concentrations for selected averaging times that are unlikely to induce adverse effects (H&S Code 39014). The standards represent the greatest outdoor exposure that is acceptable. The number of people who experience these exposures is immaterial.

The averaging times have been selected to represent common exposure patterns. The 1-hr average standard relates to peak exposure concentrations, and also represents a frequent duration of outdoor activity for many people, for example, children playing after school, adults exercising, people doing yard work or home maintenance for a relatively short time period. In this case, the standard means that for a 1-hr exposure, the maximum average ozone concentration estimated to be without adverse consequences is 0.09 ppm. Likewise, the 8-hr average standard relates to both the ozone concentration profile frequently observed in down wind areas and the activity pattern of outdoor workers, and adults and children who spend multi-hour periods in outdoor activity, including work, play and recreation. In this case, the standard means that for an 8-hr exposure, the highest average concentration estimated to be without adverse effects is 0.070 ppm.

The concept of margin of safety includes the idea that a standard must be set at a level below the lowest concentration at which adverse effects have been

documented to provide protection for potentially sensitive subjects who were not included in the study groups. Since State law requires that ambient air quality standards protect the most sensitive people in the population, we have looked not only at group mean responses, the basis of U.S. EPA developed ambient air quality standards, but have also evaluated individual responses. The scientific literature clearly shows that there is a very wide range of responses among individuals. This is not adequately factored into U.S. EPA ambient air quality standards. In the case of our 1-hr recommendation, multi-hour exposure studies did not find statistically significant responses with exposure to 0.10 ppm during the first one to two hours of a 6.6 to 8 hr exposure, while there are group mean and individual changes of concern with 2-hr exposure to 0.12 ppm ozone. This suggests a threshold in exercising people somewhere below 0.12 ppm and above 0.10 ppm for one to two hour exposures. We have included a margin of safety, and recommended a 1-hr standard of 0.09 ppm.

The body of findings from studies of 6.6 hr exposures to 0.08 ppm ozone indicates that about 26% of people who undergo similar exposures will experience symptoms and pulmonary function decrements of 10% or larger, with some experiencing decrements in excess of 30%. Since responses are related to the inhaled dose, larger decrements, and a larger fraction of people experiencing effects would be expected if the exposure period had been extended from 6.6 to 8 hours. This led to the conclusion that an 8-hr average concentration of 0.08 ppm was not adequately protective of public health. The few data available suggested that multi-hour exposure to 0.04 or 0.06 ppm ozone was unlikely to result in adverse responses.

In the case of the 8-hr average recommendation, there is less guidance for determining an adequate margin of safety, since only one chamber study at 0.04 ppm, and one at 0.06 ppm have examined responses to ozone concentrations below 0.08 ppm. Both studies found no significant pulmonary function or symptoms effects at the group level, although there were a few individual responders at 0.06 ppm. The margin of safety is supported by several epidemiologic studies, which report associations between ozone and a wide range of severe health outcomes. While we agree that this margin of safety is a more uncertain estimate than available for the 1-hr average standard, it incorporates consideration of all available data.

The primary health endpoints from the chamber studies used to develop these recommendations are acute responses (decrements in pulmonary function, respiratory symptoms, airway hyperreactivity and airways inflammation). Reduced lung function is not a benign effect because it is due to a neural reflex, as asserted by some commentators. Activation of the neural reflex represents an attempt by the body to limit inhalation of a toxic substance, in this case ozone, to protect the airway lining tissues from oxidant damage, and resulting airway inflammation. Furthermore, reduced lung function and symptoms can reduce ability to work, as well as to participate in healthful exercise and recreation.

These seemingly minor effects, temporarily reduced lung function and symptoms, can impact on ability to earn a living, and to maintain a healthy lifestyle, and clearly qualify as adverse by ATS standards, both physiologically and as aspects of quality of life. Asthmatics already have underlying chronic airway inflammation and reduced lung function. The additional ozone insult to the airway can result in exacerbation of asthma. Children are disproportionately impacted by asthma as they have higher prevalence rates, and the highest hospitalization rates are for 0-4 year olds. This is likely due at least partially to physics – the airway resistance is inversely proportional to the 4th power of the radius. Thus in a small child a little airway constriction can result in serious breathing difficulty. This will be clarified in the revised report

In addition, repeated episodes of airway inflammation lead to morphological changes in the lungs, and may contribute to long-term respiratory health impacts. Animal studies clearly support this line of reasoning. There is also convincing evidence that children who grow up in high ozone communities have lower lung function values at maturity than children who grow up in low ozone communities (Kunzli et al., 1997; Galizia and Kinney, 1999). This is a significant finding, in that low lung function is a known risk factor for chronic lung disease and premature death. The epidemiologic studies include such endpoints as premature mortality, hospitalization for respiratory and cardiovascular disease, emergency room visits for asthma, and respiratory symptoms.

Although no directly stated, this comment may include concern that ARB/OEHHA and U.S. EPA have recommended different ozone standards. There are several differences in the California standard review process that may clarify for the commenter why California and the U.S. EPA review the same literature and arrive at different recommended standards. California law requires that the standards protect the most sensitive subgroup of the population. This requires that we consider the range of individual responses to different exposure protocols to understand the range of variability in the population as a whole, and then to base our recommendations on the sensitive sub-group. In contrast, U.S. EPA primarily looks at group mean responses, with little consideration of the variability among individuals.

Second, California standards are based solely on health considerations, not on risk analysis. As noted above, our model, set by State law, is for selection of a concentration and averaging time combination that is unlikely to induce adverse effects in anyone who happens to undergo that exposure pattern. The exposure patterns used are based on a combination of patterns identified by ambient air quality monitoring, and on likely outdoor activity patterns. California standard setting does not consider the likelihood of exposure. As noted above, in California, ambient air quality standards represent the highest concentrations for selected averaging times that are unlikely to induce adverse effects. Furthermore, the proposed standards are based on responses of subject groups most likely to have significant exposure – people who are active outdoors.

Third, when EPA last considered the ozone standard in 1996/1997, there were far fewer epidemiologic studies showing severe outcomes associated with ozone exposure.

2. The proposed 8-hr average is not the appropriate form for such a standard.

The California Code of Regulations (Title 17 section 70200) establishes the form of the ambient air quality standard for ozone as “not to be exceeded”. The Expected Peak Day Concentration methodology used for area attainment designations is defined in Title 17 of the California Code of Regulations section 70306 Appendix 2. This section is unrelated to the section of the Health & Safety Code that has been opened in the present regulatory action. The EPDC method for attainment designation can be changed, but a completely separate regulatory action would be required from that for standard review. We have not opened the attainment designation procedure for review, and have no plans to do so.

3. The proposed standard would be lower than relevant background concentrations and as such is not attainable.

Our analysis determined that 0.04 ppm is a reasonable average background ozone concentration. This value is in agreement with the conclusions of the 1996 U.S. EPA ozone criteria document, and also with the World Health Organization’s 2000 document outlining Air Quality Criteria for Europe. See page 50 for an in-depth discussion of the comments received on background ozone (Chapter 4).

American Petroleum Institute & Western States Petroleum Association & Paul Switzer, Stanford University

1. The epidemiological studies of ozone and mortality use inadequate models. The report does not adequately address the statistical concerns with these models.

See #3 below.

2. It is not appropriate to use PM epidemiological studies to assess acute ozone mortality effects.

We agree that there are methodological issues with the ozone epidemiology literature, and these are discussed and acknowledged in the Staff Report. However, the recommended standards are not based primarily on epidemiology. They are based on controlled human exposure studies. Epidemiologic studies do figure into the margin of safety considerations since they strongly suggest the possibility of severe health outcomes. Thus, even if there are uncertainties about the actual effect level, measurement error, and treatment of weather and time trend, these studies are too numerous and the effects too severe to be ignored.

3. Epidemiological studies about the effects of ozone exposure on mortality and other serious health endpoints need further analysis.

API provided a commentary that points out several issues relative to time-series studies, and seeks to discredit the findings, primarily of ozone mortality studies. While the issues the commenter raises are not new, it is interesting that they focus on epidemiology and mortality, in that neither epidemiology nor mortality formed the primary basis for the standard recommendations. In the past 8 to 10 years, the focus of air pollution epidemiology has been PM. The issues the commenter raised have been investigated at length with reference to PM. Unfortunately, few studies have been designed with ozone-related hypotheses, and consequently few of the issues raised have been adequately investigated with reference to ozone. This is acknowledged in the Staff Report, which includes considerable discussion of statistical modeling issues associated with the epidemiologic literature on ozone. The Staff Report also addresses other modeling issues not mentioned by the commenter as they relate to the different types of epidemiology studies discussed in the Staff Report.

The commenter uses the NMMAPS study to support the view that ozone effects are highly variable between cities, and consequently uncertain. However, greater variability may be expected among cities simply because the effect estimate is so low and small variation in co-factors may exert more influence. This is why meta-analytic results from a large set of cities are used in preference to results from single cities. Variability is not a good enough reason to discount all of the studies. While NMMAPS (which is primarily a PM study that reports a few results for ozone) is an important study, the observation that it found a number of negative associations for ozone suggests that some of the modeling methods used may not fully control for seasonality and time trend.

It is likely that statistical modeling designed to remove weather confounding in PM studies is not the same as what would be used to control for weather confounding relative to ozone, and that the modeling requirements for removing this confounding vary by weather pattern between geographical areas. Most ozone results, particularly for mortality, come from studies that have been modeled for PM effects, and were part of the analysis of possible confounding factors, not primary analyses. Evaluation of the controlled exposure literature clearly shows that there is no biological plausibility for the reported negative effect, implicating inadequate statistical modeling and perhaps measurement error of exposure.

The commenter also asserts that heat and humidity effects may totally confound the effects of ozone. However, it is unlikely that weather would totally explain away these effects. The existing time series studies suggest that the temperature effect is very immediate; mortality usually occurs on the day of or day after high temperature. Humidity doesn't appear to play an independent role (Schwartz et al., 2004). Most existing studies carefully control for these effects and still report

an independent effect of ozone. Also, summer-specific studies also report effects of ozone. Regardless, temperature, of course, peaks in the summer while mortality peaks in the winter so the correlation between the two is usually very low or negative; therefore, failure to control for temperature is unlikely to generate a positive association between ozone and mortality. In fact, as reported in our recommendation document, a study by Thurston and Ito showed that when weather was modeled most carefully using non-linear functions, the effect estimate for ozone increased. Thus, it does not appear that temperature is responsible for reported associations between ozone and mortality. In addition, ozone is often elevated in a given city for several days, and not all ozone excursions are accompanied by temperatures that are high enough to cause mortality. Regarding interactive effects, human studies on this subject indicate that concurrent heat exposure does not impact responses to ozone. NMMAPS used basically the same weather and time trend modeling methodology in all cities, regardless of the local weather patterns, which may not adequately address differences in pattern among cities. It is likely that if the weather and time parameters had been modeled correctly and differently in each city, there may have been fewer negative, biologically implausible results for some cities. There are many possible explanations for the heterogeneity in the effect estimates and the fact that Samet et al. could not identify any effect modifiers is not evidence that they don't exist. Only six general socioeconomic status (SES) variables were tested. Factors such as monitor placement, spatial variability, SES, background health status, use of air conditioners, and housing characteristics all could contribute to heterogeneity in response. Finally, while it is possible that no benefits would result from ozone reductions, the existing meta-analysis of studies suggest that, on average, health benefits would occur. A few null findings cannot lead us to ignore all of the positive findings and the meta-analysis results.

The commenter raises the issue of heterogeneity of ozone exposure within a study area. Since ambient ozone is a regional pollution, most studies that have examined this issue report fairly similar concentrations and a high intra-city correlation among monitors on a daily basis. Regardless, random exposure measurement error would tend to reduce the likelihood of finding an effect, and would be unlikely to result in a positive and significant association.

The commenter raises the issue of a possible non-linear effect of ozone and mortality. Previous studies have suggested that the functions look fairly linear in response. However, it is true that if there is significant measurement error in exposure, it will be more difficult to find a threshold if one exists. However, as discussed above, an absolute threshold at the population level is unlikely. In addition, we are not able to reestimate the functions that have been reported in many of these studies. Finally, if the models are, in fact, non-linear, the resulting positive slope estimate would have to be larger than that produced by the linear function. This increase may fully offset the application of a threshold.

The commenter raises the issue of lag selection and ozone averaging time as impacting interpretation of ozone health effects. Based on human and animal studies, ozone effects would be expected within a day or two of exposure. There are also some studies, which suggest greater effects from cumulative exposures over 3 to 5 days. This has been considered in our interpretation of the literature. Further, since all ozone averaging times (i.e., 1, 8, 24 hr) are highly correlated, it is difficult to use epidemiology results to determine the specific averaging time of interest. As noted above, epidemiology literature is not the primary basis for either the concentrations or averaging times recommended. Epidemiology was used in a qualitative manner, as support for the controlled exposure studies.

The commenter points out that staff has not addressed mortality displacement in the Staff Report, and that this is necessary if epidemiologic studies are to be used as the basis for ambient air quality standards. It is true that we have not discussed this topic in the report. However, issues of displacement are more appropriate when one is attempting to determine the amount of life years lost and for economic valuation issues. It is not necessarily relevant for standard setting purposes.

4. A more precise and quantitative definition of adverse effects is needed.

Adverse effects were evaluated in accordance with the American Thoracic Society guidelines outlined in the Staff Report. An effect was considered significant if it was large enough to reduce or limit work or exercise capacity, or was sufficient to impact quality of life. Obviously, some of the categories suggested in the guidelines do not pertain to effects observed with ozone exposure; however, we believe that we have appropriately applied the recommended criteria.

5. Further justification for the Staff Report's recommendations is needed.

In California, ambient air quality standards represent the highest concentrations for selected averaging times that are unlikely to induce adverse effects (H&S Code 39014). The standards represent the greatest outdoor exposure that is acceptable. The number of people who experience these exposures is immaterial.

The averaging times have been selected to represent common exposure patterns. The one hour average standard relates to peak exposure concentrations, and also represents a frequent duration of outdoor activity for many people, for example, children playing after school, adults exercising, people doing yard work or home maintenance. In this case, the standard means that for a 1-hr exposure, the maximum ozone concentration estimated to be without adverse consequences is 0.09 ppm. Likewise, the 8-hr average standard relates to both the long, lower concentration, broad ozone concentration profile frequently observed in down wind areas, and also reflects the activity pattern of

outdoor workers, and adults and children who spend multi-hour periods in outdoor activity, including work, play and recreation. In this case, the standard means that for an 8-hr exposure, the highest average concentration estimated to be without adverse effects is 0.070 ppm.

The concept of margin of safety includes the idea that a standard must be set at a level below the lowest concentration at which adverse effects have been documented, to provide protection for potentially sensitive subjects who were not included in the study group. Since state law requires that ambient air quality standards protect the most sensitive people in the population, we have looked not only at group mean responses, the basis of U.S. EPA developed ambient air quality standards, but have also evaluated individual responses. The scientific literature clearly shows that there is a very wide range of responses among individuals. This is not adequately factored into U.S. EPA ambient air quality standards. In the case of our 1-hr recommendation, multi-hour exposure studies did not find statistically significant responses with exposure to 0.10 ppm during the first one to two hours of exposure, while there were group mean and individual changes of concern with 2-hr exposure to 0.12 ppm ozone. This suggests a threshold in exercising people somewhere between 0.10 and 0.12 ppm for one to two hour exposures, the same conclusion reached in the 1987 review of the State ozone standard. We also concluded that the margin of safety applied in the existing State ozone standard was adequate, and recommended retention of the existing 1-hr standard of 0.09 ppm.

In the case of the 8-hr average recommendation, there is less guidance for determining an adequate margin of safety, since there is only one study at 0.04 ppm, and one at 0.06 ppm. The body of findings from studies of 6.6 to 8 hr exposures to 0.08 ppm ozone indicates that about 26% of people who undergo similar exposures will experience symptoms and pulmonary function decrements of 10% or larger, with some experiencing decrements in excess of 30%. The study at 0.04 ppm found no significant pulmonary function or symptoms effects. Unfortunately, the one study at 0.06 ppm has not appeared in the peer-reviewed literature, although it has been published as a research report. The data on 6.6 to 8 hr exposures led to the conclusion that an 8 hr average concentration of 0.08 ppm was not adequately protective of public health, and that multi-hour exposure to 0.04 or 0.06 ppm ozone was unlikely to result in adverse responses. The epidemiological study by Tolbert et al. (2000), one of the few available that used an 8-hr averaging time, examined the shape of the concentration response function and found evidence for a population threshold in the ozone concentration range of 0.070 to 0.10 ppm. We selected the bottom of this range as the margin of safety. While we agree that this margin of safety is a more uncertain estimate than available for the 1-hr average standard, it incorporates all of the available data, and is substantially based on controlled human exposure data.

The primary health endpoints used to develop these recommendations are acute responses (decrements in pulmonary function, respiratory symptoms, airway hyperreactivity and airways inflammation). Reduced lung function is not a benign effect because it is due to a neural reflex. Activation of the neural reflex represents an attempt by the body to limit inhalation of a toxic substance, in this case ozone, to protect the airway lining tissues from oxidant damage, and resulting airway inflammation. Furthermore, reduced lung function and symptoms can reduce ability to work, as well as participate in healthful exercise and recreation. These seemingly minor effects, temporarily reduced lung function and symptoms, impact on ability to earn a living, and to maintain a healthy lifestyle, and clearly qualify as adverse by ATS standards, both physiologically and as aspects of quality of life. Repeated episodes of airway inflammation lead to morphological changes in the lungs, and may contribute to long-term respiratory health impacts. Animal studies clearly support this line of reasoning. There is also evidence that children who grow up in high ozone communities have lower lung function values at maturity than children who grow up in low ozone communities (Kunzli et al., 1997; Galizia and Kinney, 1999). This is a significant finding, in that low lung function is a known risk factor for chronic lung disease and premature death. Furthermore, asthmatics already have underlying chronic airway inflammation and reduced lung function. The additional ozone insult to the airway can result in exacerbation of asthma. Children are disproportionately impacted by asthma as they have higher prevalence rates and the highest hospitalization rates are for 0-4 year olds. This is likely due at least partially to physics – the airway resistance is inversely proportional to the 4th power of the radius. Thus in a small child a little airway constriction can result in serious breathing difficulty.

There are several differences in the California standard review process that may clarify for the commenter why California and the U.S. EPA review the same literature and arrive at different recommended standards. California law requires that the standards protect the most sensitive subgroup of the population. This requires that we consider the range of individual responses to different exposure protocols to understand the range of variability in the population as a whole, and then to base our recommendations on the sensitive sub-group. In contrast, U.S. EPA primarily looks at group mean responses, with little consideration of the variability among individuals.

Second, California standards are based solely on health considerations, not on risk analysis. As noted above, our model is for selection of a concentration and averaging time combination that is unlikely to induce adverse effects in anyone who happens to undergo that exposure pattern. The exposure patterns used are based on a combination of patterns identified by ambient air quality monitoring, and on likely outdoor activity patterns. California standard setting does not consider the likelihood of exposure. As noted above, in California, ambient air quality standards represent the highest concentrations for selected averaging times that are unlikely to induce adverse effects. Furthermore, the proposed

standards are based on responses of subject groups most likely to have significant exposure – people who are active outdoors.

Third, when EPA last considered the ozone standard in 1996/1997, there were far fewer epidemiologic studies showing severe outcomes associated with ozone exposure.

6. Additional research on human subjects in the range of 0.04 to 0.08 ppm (multi-hour exposures) is needed.

We agree that additional research at lower exposures would be informative. However, this does not negate the evidence from studies done at 0.08 ppm that a substantial fraction of the population (26%) is likely to experience pulmonary function decrements greater than 10% as well as symptoms if they undergo 6.6 hr exposures to 0.08 ppm. It should also be noted that we are proposing an 8-hr averaging time, which is longer than that of the studies on which the standard recommendation is based. Consequently, due to the larger inhaled dose of ozone, a larger portion of the population would be expected to have decrements greater than 10% and have symptoms when exposure is extended from 6.6 hr to 8 hr.

7. Additional quantification of the uncertainties, individually and in combination, is warranted and needed.

The uncertainties in epidemiological findings are discussed in the Staff Report, and because of them, the epidemiological data were used in a qualitative fashion, supporting the quantitative findings of the controlled exposure studies. It is unclear what the commenter means by the uncertainties in controlled study results. The exposure and protocol conditions in these studies are very closely controlled, and consequently the inhaled ozone dose can be accurately estimated. On an individual level, responses to ozone are very consistent over time periods of at least one year (section 11.4.2.1.4). The range of responsiveness between individuals has also been investigated, and been shown to be very wide at all ozone concentrations investigated (section 11.4.2.1.4). For example, the range of FEV1 response with 6.6 hr exposure ranged from +10% to –40% with exposure to 0.08 ppm ozone; from +5 to –45% with exposure to 0.10 ppm ozone; and from +5% to –50% with exposure to 0.12 ppm ozone. The ranges are similar for shorter exposures with somewhat higher ozone concentrations. This information is presented in section 11.4.2.1.4 of the Staff Report. In addition the range of individual responses is presented in the review of studies and the tables of Chapter 11 of the Staff Report.

8. The background ozone concentration is not 0.04 ppm, but is frequently much higher.

See page 50 for an in-depth discussion of the comments received on background ozone (Chapter 4).

Our analysis determined that 0.04 ppm is a reasonable average background ozone concentration. This value is in agreement with the conclusions of the 1996 U.S. EPA ozone criteria document, and also with the World Health Organization's 2000 document outlining Air Quality Criteria for Europe.

9. The exposure scenarios used in controlled studies do not reflect ambient conditions, particularly the square wave multi-hour protocol. This protocol does not consider the non-linear dose-response relationship, or that responses are related to the dose rate, not just the concentration.

The commenter is correct that the dose rate is more important than the concentration alone. This is why we have recommended two standards, one with a 1-hr averaging time, and one with an 8-hr averaging time. This will insure that during any eight hour period that meets the 0.070 ppm 8-hr standard, there will be no hour with an average ozone concentration over 0.09 ppm.

10. Controlled studies should not compare response consequent to ozone exposure with that after filtered air exposure. The baseline should be background.

The commenter asserts that the baseline for comparison of effects should be background (i.e., 0.04 ppm) rather than filtered air because responses are related to the change in ozone concentration, not the concentration itself. This is erroneous. The biological responses caused by ozone are not linear functions, as the commenter apparently assumes, but rather are exponential. The human exposure data clearly indicate that responses to ozone exposure are proportional to the inhaled dose of ozone, which is the product of ozone concentration, breathing rate, and exposure duration. Consequently, a very large number of exposure scenarios can be invented that would result in an inhaled dose that is likely to induce adverse responses. Although there are no data suggesting effects at 0.04 ppm, it is theoretically possible that a sufficiently long exposure with a high exercise level could result in an inhaled dose that is large enough to induce adverse effects, but based on available data, this is unlikely. The data also point to the existence of a threshold, particularly on the individual level, which appears to be below 0.12 ppm for 1 to 3 hour exposures, in heavily exercising subjects, and 0.08 ppm for 6.6 hour exposures, in moderately exercising subjects. Since 0.04 ppm appears to be below the threshold, use of 0.04 for the baseline for calculating responses to ozone exposure would be unlikely to change the conclusions reached.

11. It appears that staff has turned to analysis of ambient air quality to show the relationship between exposure for the 1-hr standard and alternative concentrations for an 8-hr standard.

Perhaps the commenter has misunderstood the purpose of the analysis of the relationship between 1-hr and 8-hr average ozone concentrations. The recommendations were based on the health literature. The analyses comparing the 1-hr (0.09 ppm) and recommended 8-hr (0.070 ppm) standards were presented to indicate the relationship between the two standards. The analysis indicated that either standard by itself would not be protective of public health. Specifically, an area could attain one standard but still be out of attainment relative to the other standard. Therefore, we recommended adding an 8-hour standard while retaining the 1-hour standard. We will revise the Staff Report to prevent this misunderstanding.

12. The linear rollback method is not appropriate.

The rollback method was developed using actual monitored data from California, and represents the behavior of real data. The methodology and data tables and figures that support the approach are presented in the appendix to chapter 10 of the Staff Report.

13. The 8-hr proposed standard is not attainable.

The proposed 8-hour standard will be difficult to attain. However, California law does not require that ambient air quality standards be based on ease of attainability; it requires that they be based on health effects.

Alliance of Automobile Manufacturers

1. The appropriate measurement of background ozone must be considered part of the proposed AAQS. The proposed standards are at or overlap background.

Our analysis determined that 0.04 ppm is a reasonable average background ozone concentration. This value is in agreement with the conclusions of the 1996 U.S. EPA ozone criteria document, and also with the World Health Organization's 2000 document outlining Air Quality Criteria for Europe. See page 50 for an in-depth discussion of the comments received on background ozone (Chapter 4).

2. The correlation between measured clinical health effects and impact on public health has not been established.

This issue has several parts. First, the commenter states that a new mechanism for pulmonary function decrements and respiratory symptoms has been reported - a vagal nerve reflex. Two recent papers are cited along with the claim that this is a recent finding. Actually, the vagal nerve reflex contribution to responses to ozone has been known since the 1970's. The commenter appears to believe that since a nerve reflex mechanism is involved, there is no reason for concern.

However, the reflex is a protective response to an inhaled irritant, the purpose of which is to reduce exposure of the lung tissue to the irritant. In other words, the body recognizes that inhaled ozone is potentially injurious, and attempts to reduce inhalation, and thereby exposure, by reflexively reducing lung function and tidal volume. This, in turn, can reduce work capacity. Decrements in lung function in response to ozone can be large, and can also contribute to exacerbations of lung disease including asthma. The argument that a reflex response does not represent an adverse response that is significant is incorrect.

Second, the commenter asserts that the baseline for comparison of effects should be background (i.e., 0.04 ppm) rather than filtered air because responses are related to the change in ozone concentration, not the concentration itself. This is erroneous. The biological responses caused by ozone are not linear functions, as the commenter apparently assumes, but rather are exponential. The human exposure data clearly indicate that responses to ozone exposure are proportional to the inhaled dose of ozone, which is the product of ozone concentration, breathing rate, and exposure duration. Consequently, a very large number of exposure scenarios can be invented that would result in an inhaled dose that is likely to induce adverse responses. Although there are no data suggesting effects at 0.04 ppm, it is theoretically possible that a sufficiently long exposure with a high exercise level could result in an inhaled dose that is large enough to induce adverse effects, but based on available data, this is unlikely. The data also point to the existence of a threshold, particularly on the individual level, which appears to be below 0.12 ppm for 1 to 3 hour exposures, in heavily exercising subjects, and 0.08 ppm for 6.6 hour exposures, in moderately exercising subjects.

The commenter raises issues with the Staff conclusions as to sensitive subpopulations, and asserts that the Staff Report concludes that young adults are the most sensitive population for pulmonary function decrements and symptoms, that older adults and children are less sensitive, and that COPD patients and smokers are unlikely to experience marked respiratory effects. This argument involves several misconceptions. Responses to ozone are related to the inhaled dose, not solely to the concentration. The Staff Report concluded that children, people who are active outdoors, and outdoor workers were most likely to inhale sufficient doses of ozone to induce adverse effects. While data suggest that older adults have smaller pulmonary function and symptoms responses than similarly exposed young adults, there are individual exceptions, and there are no data on airway reactivity or inflammation on older adults. However, older adults who have reduced pulmonary function with ozone exposure typically also have symptoms, as well. Consequently, a complete picture of the risks to active older adults is not available in the current literature. The available data on children suggest that they have similar pulmonary function changes as young adults who inhaled comparable doses of ozone, but they tend to report few symptoms. There are no data available from chamber studies on airway responsiveness or inflammation for children, although there is no reason

to think that they would not have responses similar to those of adults. The commenter misinterprets the Staff Report statement regarding the lack of symptoms reports by children, asserting that their lack of reported symptoms indicates lower risk. The few controlled studies on children have involved children from about 8 to 12 years of age. In reality, there are several possible explanations for this largely uninvestigated topic. It is unknown whether children really don't have symptoms, are unwilling to articulate them due to social concerns that they might disappoint the investigators, or whether they are unable to understand or articulate them. In any case, this difference in responses between adults and children is of concern from a risk management perspective, because for whatever reason, children appear to have little appreciation that their bodies have activated reflex induced pulmonary function decrements as a means to reduce toxic exposure.

Of additional importance is that asthma is an important health endpoint for children. Prevalence rates of asthma are higher in children than adults, and children 0-4 years old have the highest hospitalization rates of all age groupings. Small children have small airways and thus are more prone to breathing difficulties due to the relationship between airway caliber and resistance. Thus, children are disproportionately impacted by air pollutants that exacerbate asthma. Ozone can exacerbate asthma and may induce asthma in children who are very active outdoors (McConnell et al., 2002).

The commenter raises several issues relative to the discussion of morphological effects. While it is not clear how to extrapolate findings of animal studies to likely human responses, the fact that similar changes in morphometry have been observed in multiple animal species, albeit with differences in apparent sensitivity, makes it likely that similar responses also occur in humans. Sections 11.3.3 and 11.3.4 discuss responses of animals to long term ozone exposure and also the influence of the interexposure interval with repeated acute exposures on morphological responses. The text makes clear that the time sequence of repeated exposures affects tissue responses, and that the timing of a repeat exposure relative to the status of the injury-repair cycle influences the outcome. It is not entirely true that responses diminish over time. Animal studies clearly show that repeated acute exposures can have residual effects that accumulate over time.

It is unclear why the commenter asserts that there is no likelihood that the population most at risk (people who are active outdoors) will experience a large number of repeated peak exposures. People who are regularly active outdoors will experience a significant number of repeated high exposures if they live in areas with more than a few annual exceedances of the ozone standard (for example, the South Coast Air Basin, Sacramento, and the San Joaquin Valley). True, many people spend most of their time indoors. But the population of California includes a large number of children, many of whom spend a significant amount of time outdoors, many recreational athletes, and outdoor workers.

These people will experience multiple peak exposures per year by virtue of their lifestyle patterns. The southern California Children's Study suggests that ozone may induce asthma in very active children (McConnell et al., 2002). The fact that ozone concentrations have declined considerably over the past 40+ years does not negate scientific data indicating that significant adverse effects are still possible in people who inhale a sufficient dose of ozone. The magnitude of these effects may be smaller due to the lower peak ozone concentrations currently observed, but this does not alter the conclusion that current ozone concentrations can induce adverse responses in people who inhale a sufficient dose due to their activity patterns.

The commenter requests that the issue of an effect threshold be discussed more fully. While the Staff Report does not explicitly use the term threshold in discussion of the controlled exposure studies, it does clearly present the lowest effect levels found in the available literature for all available endpoints, and details them both in the summary of the controlled studies chapter, and in the recommendation (Chapter 8).

The commenter requests modification of a sentence in the final paragraph of the chapter summary that refers to epidemiology studies and their limitations. The subject of the limitations of epidemiology studies is discussed at length in Chapter 12, which addresses at length the concern of the commenter.

3. The inherent weaknesses in epidemiology studies need to be formally recognized.

We have attempted to outline the various weaknesses of the epidemiology studies in each of the four major sections of the epidemiology chapter, including extensive discussion of uncertainties, issues related to statistical modeling, and potential weaknesses of epidemiology studies in general, as well as specific to individual studies that impact on the conclusions that can be drawn from the literature. It is true that this chapter is not as comprehensive as the controlled studies chapter. This is because we relied primarily on the chamber studies for the development of the standard with the epidemiology studies playing a supportive role and weighing in on the margin of safety. Regarding the issue of GAM-related problems in the mortality studies, many of these studies have now been reanalyzed, although mostly for PM. The general conclusion from this reanalysis is that, for the most part, using other smoothing functions such as penalized or natural splines does appear to drastically alter the general results. In some cases the estimated effect estimate falls and in some cases it rises or stays about the same. In multi-city analyses, the general findings are often the same as the original GAM results. In general, the ozone studies have not undertaken as much examination. However, new analyses of the NMMAPS focusing on mortality confirms an association between ozone and premature mortality, with an effect estimate generally similar to that previously reported.

We agree that the statistical modeling strategy is extremely important in evaluating and interpreting these studies. This is why the chapter spends quite a few pages discussing modeling and interpretation issues as they relate to each of the four topical categories of studies evaluated. But two general findings seem apparent: (1) that the results do not appear to change when other smoothing models are used, including parametric smoothing techniques; and (2) that, in general, more careful control of weather tends to increase the size and statistical significance of the ozone effect.

Publication bias is unlikely an issue with this literature, as almost all of it was designed to investigate PM effects, and any ozone results presented were part of the sensitivity analyses and investigation of potentially confounding factors relative to the main focus, PM. Consequently, there is little reason to suppose that negative findings have been suppressed. In fact, there is reason to suppose that any ozone related findings would be presented to show that the PM results were not influenced by ozone. In addition, the new analysis of the NMMAPS data on ozone has been recently published, and this is a study that inherently has no publication bias. Finally, the WHO has adjusted their estimates of their meta-analysis of European studies to address the possibility of publication bias. They still report an association between ozone and both all-cause and cardiovascular mortality.

4. Chapter 7 is an analysis of potential peak exposure, not actual exposure. Exposure analysis should include consideration of the probability that a person will receive an exposure of concern.

The comment suggests a misunderstanding of the purpose of Chapter 7. The chapter is an analysis of statewide air quality. It gives an indication of the number of people who live in areas where ozone concentrations reach the level of concern. It is not a risk analysis. The source of confusion may be that the Health & Safety Code calls this sort of characterization of statewide air quality "exposure", although it is not exposure in the sense of personal exposure assessment.

5. The staff recommendation is not adequately substantiated. The selected margin-of-safety interval has not been quantified or substantiated.

The commenter begins this topic by disagreeing with identification in the year 2000 of the standard for ozone as being possibly inadequate (SB25 standard prioritization process). The prioritization process involved a brief review of recent scientific literature, and a determination as to whether or not there was evidence that the various air quality standards might be inadequate, particularly in regards to infants and children. Standards deemed possibly inadequate were prioritized for full review, partially based on the frequency of exceedences of the existing standards, as well as the sorts of effects identified. Chapter 7 clearly shows that

most Californians live in areas where peak ozone concentrations frequently exceed the current state standard. Personal exposure is not the issue here.

The commenter seems to misunderstand the meaning of ambient air quality standards. In California, ambient air quality standards represent the highest concentrations for selected averaging times that are unlikely to induce adverse effects (H&S Code 39014). The standards represent the greatest outdoor exposure that is acceptable. The number of people who experience these exposures is immaterial. The commenter recommends inclusion of a risk analysis, such as performed by U.S. EPA. Such an analysis is not required for California ambient air quality standards. California ambient air quality standards are based solely on health effects, and as noted above, the risk of exposure has no bearing on what constitutes the maximal exposure that is unlikely to induce adverse responses. Such an analysis would not change the conclusions Staff has drawn.

The averaging times have been selected to represent common exposure patterns. The one hour average standard relates to peak exposure concentrations, and also represents a frequent duration of outdoor activity for many people, for example, children playing after school, adults exercising, people doing yard work or home maintenance. In this case, the standard means that for a 1-hr exposure, the maximum ozone concentration estimated to be without adverse consequences is 0.09 ppm. Likewise, the 8-hr average standard relates to both the long, lower concentration, broad ozone concentration profile frequently observed in down wind areas, and also reflects the activity pattern of outdoor workers, and adults and children who spend multi-hour periods in outdoor activity, including work, play and recreation. In this case, the standard means that for an 8-hr exposure, the highest average concentration estimated to be without adverse effects is 0.070 ppm.

The concept of margin of safety includes the idea that a standard must be set at a level below the lowest concentration at which adverse effects have been documented to provide protection for potentially sensitive subjects who were not included in the study group. Since state law requires that ambient air quality standards protect the most sensitive people in the population, we have looked not only at group mean responses, the basis of U.S. EPA developed ambient air quality standards, but have also evaluated individual responses. The scientific literature clearly shows that there is a very wide range of responses among individuals. This is not adequately factored into U.S. EPA ambient air quality standards. In the case of our 1-hr recommendation, multi-hour exposure studies did not find statistically significant responses with exposure to 0.10 ppm during the first one to two hours of exposure, while there were group mean and individual changes of concern with 2-hr exposure to 0.12 ppm ozone. This suggests a threshold in exercising people somewhere between 0.10 and 0.12 ppm for one to two hour exposures. We have included a margin of safety, and recommended a 1-hr standard of 0.09 ppm because the total population studied

at these concentration was small, and would not have included people who represent the full range of sensitivity.

In the case of the 8-hr average recommendation, there is less guidance for determining an adequate margin of safety, since there is only one study at 0.04 ppm, and one at 0.06 ppm. The body of findings from studies of 6.6 to 8 hr exposures to 0.08 ppm ozone indicates that about 26% of people who undergo similar exposures will experience symptoms and pulmonary function decrements of 10% or larger, with some experiencing decrements in excess of 30%. The study at 0.04 ppm found no significant pulmonary function or symptoms effects. Unfortunately, the one study at 0.06 ppm has not appeared in the peer-reviewed literature, although it has been published as a research report. The data available led to the conclusion that an 8-hr average concentration of 0.08 ppm was not adequately protective of public health, and that multi-hour exposure to 0.04 or 0.06 ppm ozone was unlikely to result in adverse responses. Also, an epidemiological study by Tolbert et al. (2000) that examined the shape of the concentration response function suggested that a population threshold might be evident in the ozone concentration range of 0.070 to 0.10. We selected the bottom of this range to incorporate a margin of safety. In addition, several other epidemiologic studies suggest the possibility of effects below the concentrations where effects are observed in the chamber studies.

Next, the commenter suggests that the effects reported in the scientific literature are isolated, transient and reversible, and therefore not of significance. We believe that we have appropriately applied the ATS guidelines for adverse health effects. Admittedly, the most common effects attributable to ozone based on the chamber studies (pulmonary function changes, respiratory symptoms, airway hyperreactivity and airways inflammation) are acute and are reversible once exposure decreases below a threshold level. We agree that these are in some sense "potential effects" in that not all people will have the exposures on which the recommendations are based. But, to reiterate, the number of people is not the issue. The standards represent the maximum single exposures unlikely to induce adverse effects in exposed people. As we discussed in the Staff Report, there is evidence that repeated responses can lead to morphological changes in the lungs.

Reduced lung function is not a benign effect because it is due to a neural reflex as the commenter asserts. Activation of the neural reflex represents an attempt by the body to limit inhalation of a toxic substance, in this case ozone, to protect the airway lining tissues from oxidant damage, and resulting airway inflammation. Furthermore, reduced lung function and symptoms can reduce ability to work, as well as participate in healthful exercise and recreation. These seemingly minor effects, temporarily reduced lung function and symptoms, impact on ability to earn a living, and to maintain a healthy lifestyle, and clearly qualify as adverse by ATS standards, both physiologically and as aspects of quality of life. Repeated episodes of airway inflammation lead to morphological changes in the lungs, and

may contribute to long-term respiratory health impacts. Animal studies clearly support this line of reasoning. There is also evidence that children who grow up in high ozone communities have lower lung function values at maturity than children who grow up in low ozone communities (Kunzlie al., 1997; Galizia and Kinney, 1999). This is a significant finding, in that low lung function is a known risk factor for chronic lung disease and premature death. Furthermore, asthmatics already have underlying chronic airway inflammation and reduced lung function. The additional ozone insult to the airway can result in exacerbation of asthma. Children are disproportionately impacted by asthma as they have higher prevalence rates, and the highest hospitalization rates are for 0-4 year olds. This is likely due at least partially to physics – the airway resistance is inversely proportional to the 4th power of the radius. Thus in a small child a little airway constriction can result in serious breathing difficulty.

The next section of the comments recommends that ARB/OEHHA adopt the federal process and procedures for development of ambient air quality standards. As discussed above, federal law related to processes and procedures governing establishment of federal ambient air quality standards does not apply to California. California law dictates the process and procedures that must be followed in development and promulgation of ambient air quality standards. We have followed the process required by the California Administrative Procedure Act, and do not have jurisdiction to change it.

There are several differences in the process that may clarify for the commenter why California and the U.S. EPA review the same literature and arrive at different recommended standards. California law requires that the standards protect the most sensitive subgroup of the population. This requires that we consider the range of individual responses to different exposure protocols to understand the range of variability in the population as a whole, and then to base our recommendations on the sensitive sub-group. In contrast, U.S. EPA primarily looks at group mean responses, with little consideration of the variability among individuals. Second, California standards are based solely on health considerations, not on risk analysis. As noted above, our model is for selection of a concentration and averaging time combination that is unlikely to induce adverse effects in anyone who happens to undergo that exposure pattern. The exposure patterns used are based on a combination of patterns identified by ambient air quality monitoring, and on likely outdoor activity patterns. California standard setting does not consider the likelihood of exposure. As noted above, in California, ambient air quality standards represent the highest concentrations for selected averaging times that are unlikely to induce adverse effects. Furthermore, the proposed standards are based on responses of subject groups most likely to have significant exposure – people who are active outdoors. Finally, since the U.S. EPA review in 1996/97, dozens of epidemiologic studies have been published documenting an effect of ozone on several severe health outcomes including mortality and hospitalization.

With reference to the controlled exposure studies, the commenter points out that airway hyperresponsiveness and pulmonary inflammation occur at 0.18 to 0.20 ppm, with one to three hour exposures with heavy exercise, and at 0.08 ppm with 6.6 hr exposure. Since these are the lowest concentrations at which these endpoints have been evaluated, as is noted in the Staff Report, it is unknown whether these effects occur at lower concentrations. While a single episode of airways inflammation induced by ambient concentrations of ozone is unlikely to have long-term consequences, the reality is that the most populated parts of California have multiple exceedances of the State ozone standard each year. In addition, large, heavily populated parts of the state often have concentrations at or near those reported in the literature to induce airways inflammation. As noted in the section on morphological effects of repeated ozone exposures, such a pattern of injury and repair cycles causes changes in the kind of cells lining the airways, increases collagen formation which can lead to reduced airway compliance, a feature of several chronic lung diseases, and in children exposed early in life, to changes in airway architecture and lung development. There is ample evidence that these constitute effects of concern.

The commenter has misunderstood the statement referring to the value of animal studies in elucidating human health effects. Animal studies have provided considerable information on biological mechanisms and tissue effects that can not be studied in humans. The fact that these effects have been documented in more than one mammalian species and in multiple strains of animals suggests that these effects are common to mammals. True, they do not inform as to the relative sensitivity of humans compared to the various species and strains, but that does not negate the value of the information they provide.

The commenter complains that the Staff Report does not include discussion of the statistical form of the standard (attainment test, or expected peak exposure concentration - EPDC). The EPDC methodology is not part of the standard setting process in California. The procedure is established in section 70306 Appendix 2 of Title 17 of the California Code of Regulations. This section is unrelated to those that have been opened in the present regulatory action. The EPDC method can be changed, but a completely separate regulatory action would be required from that for standard review.

6. The EPDC method for determining attainment is too complex, not robust, and is too stringent. The federal method should be adopted. Alternate method proposed by commenter.

We thank the commenter for the suggested alternate attainment designation method. However, this is not relevant to the standard review process. The area attainment designation process is dealt with under a separate regulatory framework (Title 17, California Code of Regulations sections 70300 through 70306)

7. The federal method/process of standard review should be followed by CA.
8. A more iterative process would allow an opportunity to reconcile differences in the interpretation of the science.

These two comments are related to the process used by ARB/OEHHA to propose revision of the CA ozone standard. The Alliance recommends that ARB/OEHHA adopt the federal model in which there are several drafts of a document similar to the EPA criteria document, several rounds of public peer review, and then recommendation of a standard. They also state that the public has been excluded from participation in development of the policy recommendation.

The requirements of the federal Clean Air Act, and those governing promulgation of federal regulations do not apply to state regulations. California law dictates the process and procedures to be followed for standards review and revision. We have followed the requirements of the California law governing review of ambient air quality standards in our review and in the development of our recommendations. The public has the opportunity to participate in the process. The public is free to comment on each draft of the Staff Report and its recommendations, to comment to the Air Quality Advisory Committee, and directly to the Board at its public hearing of the item.

Engine Manufacturers Association

1. The report needs to better address whether the results of human exposure studies actually meet the criteria as adverse health effects established by the American Thoracic Society.

Adverse effects were evaluated in accordance with the American Thoracic Society guidelines outlined in the Staff Report. An effect was considered significant if it was large enough to reduce or limit work or exercise capacity, or was sufficient to impact quality of life. Obviously, some of the categories suggested in the guidelines do not pertain to effects observed with ozone exposure, however, we believe that we have properly applied the recommended criteria. Admittedly, the most common effects attributable to ozone (pulmonary function changes, respiratory symptoms, airway hyperreactivity and airways inflammation) are acute and are reversible once exposure decreases below a threshold level ends. They are not, however isolated, given that the literature shows that about 25% of people who undergo an 8 hr exposure to 0.08 ppm ozone are likely to have reductions in lung function and respiratory symptoms, along with airway inflammation.

Reduced lung function is not a benign effect because it is due to a neural reflex as some commentators assert. Activation of the neural reflex represents an attempt by the body to limit inhalation of a toxic substance, in this case ozone, to protect the airway lining tissues from oxidant damage, and resulting airway

inflammation. Furthermore, reduced lung function and symptoms can reduce ability to work, as well as participate in healthful exercise and recreation. These seemingly minor effects, temporarily reduced lung function and symptoms, impact on ability to earn a living, and to maintain a healthy lifestyle, and clearly qualify as adverse by ATS standards, both physiologically and as aspects of quality of life. Repeated episodes of airway inflammation lead to morphological changes in the lungs, and may contribute to long-term respiratory health impacts. Animal studies clearly support this line of reasoning. There is also evidence that children who grow up in high ozone communities have lower lung function values at maturity than children who grow up in low ozone communities (Kunzli et al., 1997; Galizia and Kinney, 1999). This is a significant finding, in that low lung function is a known risk factor for chronic lung disease and premature death. Furthermore, asthmatics already have underlying chronic airway inflammation and reduced lung function. The additional ozone insult to the airway can result in exacerbation of asthma. Children are disproportionately impacted by asthma as they have higher prevalence rates and the highest hospitalization rates are for 0-4 year olds. This is likely due at least partially to physics – the airway resistance is inversely proportional to the 4th power of the radius. Thus in a small child a little airway constriction can result in serious breathing difficulty.

2. A better evaluation of the human exposure/chamber studies is needed.

The commenter raises questions about the design of the controlled studies with regard to undue physiological stress, measurement and form of the ozone exposure, possible subject response bias, statistical analysis methods, applicability of the results to the overall population, and differences among studies. This series of comments reflects a misunderstanding on the part of the commenter as to what an ambient air quality standard represents under California law, and the considerations that state law requires when reviewing ambient air quality standards.

The basic protocols and methodologies used for the human chamber exposure studies are standardized, and have been essentially unchanged for about 30 years. These protocols were designed to simulate several possible outdoor exposure scenarios. Typically, people who are outdoors are not continually at rest, but are at least intermittently involved in some sort of physical activity. The one-hour continuous exercise protocol simulates the sort of outdoor exposure a recreational athlete or person pursuing an exercise program, such as jogging, would experience. The two-hour intermittent exercise protocol simulates children playing, after school sports and less intense personal exercise programs, outdoor home maintenance, moderate recreational activity, and yard work. The 6.6- to 8-hour protocols simulate a full day of outdoor work. The ventilation rates used in these studies are based on research that has measured ventilation for a variety of activities. Because of these factors, we believe that the protocols adequately simulate real-world activity patterns, and disagree that the protocols

cause undue physiological stress. We will add some text more fully describing the basic protocols and methodologies.

It is unclear why the commenter focuses on only four human exposure studies in their commentary, when there are many studies that have similar findings. The usual statistical design for these studies is a repeated measures analysis of variance design in which each subject completes all exposures, and serves as his/her own control. A few studies from the U.S. EPA lab have assigned each subject to only one exposure group, but in these cases, the groups for each condition were considerably larger to provide sufficient statistical power to the analyses. It is typical to investigate whether the data set is normally distributed, and then to use parametric or nonparametric analysis of variance, as appropriate. Since this method focuses on the variance of the responses to the different conditions, it does not reveal information as to between subject variability. The commenter raised an issue regarding the parametric t-tests used to compare intra-exposure time points in Horstman et al. (1990). The investigators compared the intraexposure time points using both t-tests and MANOVA, and point out that while the latter is more appropriate for the data set, it is also negatively biased due to the small number of degrees of freedom. The commenter also points out that many of the subjects in this study did not demonstrate clear dose-response relationships on an individual level. As Horstman et al. discuss, this may be partly due to the similarity of the inhaled effective dose for the 0.08 and 0.10 conditions, in addition to within subject variability, and the nonlinearity of the dose response relationship, which is typically exponential.

It is true that not all studies present individual level data, but we evaluated the range of responses to ozone exposure to the extent that individual data were available. The difference in mean responses between different studies also gives some information on the range in responses between individuals. Subjects are more likely to conclude that they have been exposed to ozone if they begin to develop respiratory symptoms or perceive that it is more difficult to breathe than if they smell it because ozone quickly dulls the sense of smell. In fact, some investigators put a trace of ozone in the chamber at the time the subject enters so that initial entry conditions seem the same, no matter what the actual exposure is. The commenter suggests that subjects could be faking their responses because they conclude, based on smell, that they are exposed to ozone. It is impossible to fake consistent lung function tests. If the subjects had been faking, their test values would be highly inconsistent. In fact, the reason it is customary to have subjects perform two to three tests per sampling period (that must agree within 5%) is to preclude the possibility of the subject failing to make maximal effort.

The commenter recommends that since we have proposed standards for one and eight hour averaging times only studies that used these exposure durations should be considered. We disagree. Analysis of the database includes consideration of the total inhaled dose of ozone, in addition to the averaging time.

While the ozone concentration is the most important determinant of effects, total dose also matters. This allows comparison of studies with different durations of exposure (i.e., 1-3 hours). It is true that most of the multi-hour exposure studies used a 6.6 hour exposure protocol, while the recommended multi-hour standard is an eight hour average. Since responses are proportional to inhaled dose, if anything, we would expect that the effects with exposure to 0.08 ppm would be greater if the 6.6 hour exposures were extended to eight hours. This in itself justifies a lower ozone concentration on the grounds of the longer averaging time, and the correspondingly increased inhaled dose of ozone.

The commenter attempts to attribute at least part of the ozone effect to temperature and humidity, and asserts that we have not adequately considered this potential confounder. The literature does not support the reviewer's contention. We reviewed all available studies addressing this subject in the Staff Report. The data indicate that temperature and humidity do not affect responses to ozone. In addition, there is no literature suggesting that heat or humidity, in the absence of ozone, alter lung function or respiratory symptoms.

The commenter questions whether the general population is capable of the sorts of exposures that were used in the published literature, and expresses the opinion that the exposure patterns studied are irrelevant for the general population. This is not the point, and the commenter appears to misunderstand the definition of ambient air quality standards in California, which is different from that used by U.S. EPA. In California, ambient air quality standards represent the highest concentration for a given averaging time that is unlikely to induce adverse responses in people who experience that exposure. It is irrelevant how many people might actually experience that exposure. Active people and outdoor workers are not less deserving of adequate protection from adverse effects caused by air pollution than less active people.

3. The report does not adequately convey the caveats or conflicting results contained in the epidemiology literature on ozone.

The commenter expresses the view that longitudinal cohort studies are more powerful than time series studies for evaluating air pollution health effects. This is not likely true for ozone, although it appears to be the case for PM. In addition, each type of epidemiologic study design has advantages and disadvantages. The prospective cohort studies are powerful in terms of the importance of their health endpoint and the implications of the findings for both standard setting and impact assessment. On the other hand, both panel studies and time-series studies have some very powerful aspects as well such as the ability to minimize confounding, deal with seasonality, and reduce measurement error in exposure. Human and animal exposure studies indicate that ozone effects are more acute than chronic, although there is evidence for morphological effects with long-term, high concentration exposure, and some evidence for reduced lung function with

long-term exposure. On this basis, we would expect time-series studies to more likely show positive associations with adverse effects.

The commenter goes on to make comments on the various types of epidemiological studies. Under longitudinal studies, the commenter discusses Gent et al. (2003) at length. This is actually a field-type study, and is discussed in section 12.1 of the Staff Report. The commenter does not raise any issues that are not pointed out in the chapter. The commenter goes on to discuss the findings of several papers from the Children's Health Study, and offers nothing that has not been considered in the chapter. The study by Frischer et al. (1999) is discussed as a long term study of lung function growth. This is not the case. The Frischer study investigated the influence of seasonal ozone exposure on lung function by comparing measurements obtained at the beginning and end of the summer ozone seasons. The study and the Staff Report indicate that it is unknown whether the somewhat lower lung function measured at the end of the summer ozone season represents a permanent change, or whether it would reverse over the low ozone season. Consequently, the study adds nothing to the commenter's argument. The commenter reaches pretty much the same conclusion as the Staff Report with reference to long-term consequences of ozone exposure.

The commenter next discusses time-series studies, largely on the issue that most have not been reanalyzed since discovery of the default convergence criteria problem in the S-Plus software for the generalized additive model. The issues raised by the commenter regarding the S-Plus software and model specifications and sensitivity analyses are all acknowledged and discussed in the chapter. We made it clear that we did not consider time series studies using the S-Plus generalized additive model, unless they had been reanalyzed. The commenter raises significant statistical modeling issues, but the report acknowledges them. Furthermore, as stated previously, we did not use epidemiological literature as the primary basis for the ozone standards recommendations. Epidemiological literature served in a supporting, qualitative capacity.

The paper by Koop and Tole (2004) asserts that there are multiple statistically acceptable models to describe time series data sets, and that there is no consensus as to which is/are the "real" one(s). This is true – the subject has been raised before. Koop and Tole suggest a Bayesian averaging methodology to address this problem. They claim that the available time series literature includes too few potentially explanatory variables. They propose an approach that is purely statistical, and includes every possible variable they can think of, and all possible interactions of these variables. Unfortunately, they also include variables and lag times that have been shown by physiological research to have no biological plausibility. There is no reason to include variables or lag times in the models that can be excluded a priori on physiological grounds. Inclusion of such variables complicates the models, can lead to computational difficulties, and

confuses interpretation of the results. Contrary to the commenter's assertion, a great deal is known about ozone that is useful in selecting a particular lag time or potential confounder. In addition, the approach included weather variables in the regression model that relate to mortality only because they impact air pollution concentrations, and that would not have an independent effect. Therefore, these variables should not be considered confounders if one is trying to assess the causal effects of air pollution. Finally, it is a problem that the authors of the comment base all of their conclusions on findings from only one city where up to 90 cities have been used in some of the meta-analyses. Single city studies have limited ability for inference in this case.

The commenter's statement that people are generally eating better, exercising more and smoking less is belied by even a cursory look at recent public health reports that obesity is epidemic, and at the high sales volume of foods of questionable nutritional value.

We believe that the caveats, limitations and various statistical modeling issues raised by the commenter with reference to the epidemiological literature have been acknowledged in the report, and taken into consideration in the conclusions drawn. As noted above, epidemiology is not the primary basis of the recommended standards.

While we agree that the relative sensitivities to ozone of rodents, monkeys and humans is unknown, the results from animal exposures provide important information as to biological mechanisms by which ozone could induce adverse effects, and that could support a conclusion as to whether chronic ozone exposure could plausibly have adverse consequences. These results are presented, not as proof of effects at ambient concentrations, but as showing that such effects are plausible. These studies also provide important mechanistic support for epidemiological findings. We have not used this literature as a basis for our recommendations, but as supportive material.

4. A more thorough discussion of the effects of ozone on susceptible populations including children and asthmatics needs to be included in the final report.

We have evaluated what literature there is on responses of children and potentially sensitive groups to ozone, and believe that we have drawn fair and reasonable conclusions. We agree that the number of studies available on these subgroups is limited. We will add additional discussion on asthma as a health endpoint that disproportionately impacts children.

We will attempt to clarify the justification for the margin of safety, and discuss our reasoning in more detail.

5. The report needs to assess the impacts on human health from historical and documented reductions in ozone levels.

We agree that this would be interesting and helpful information. Unfortunately, there is no data available that would address the issues raised. In the case of PM, there are the historical London, Meuse Valley and Donora, PA episodes of extremely high PM concentrations. There are no similar ozone events, although Friedman et al. (2001) in Atlanta reported a reduction in asthma ER visits when ozone levels decreased when city traffic was rerouted during the Atlanta Olympics. In addition, there are no studies that have investigated the magnitude of public health benefits that have accrued from the reductions in ozone concentrations over the past 40 years.

Natural Resources Defense Council

1. The proposed standards are not adequately protective, and do not include an adequate margin of safety.

We believe that the commenter has misinterpreted the epidemiological literature used to support the conclusion that the proposed standards are not adequately protective. The concentrations cited by the commenter are the annual average of daily peak concentrations. This average includes values obtained on days there was little ozone because it rained, was winter, or the meteorological conditions were not conducive to ozone formation. The only conclusion that can be made from the data cited is that effects have been reported in cities with low annual averages of the peak daily measures. This does not mean that the effects in those cities actually occurred at the annual mean of the daily peak concentrations.

Hal Levine

1. There is not enough emphasis on indoor contributions to exposure.

The ambient air quality standards are for outdoor air, and reflect the highest concentration for a given averaging period that is unlikely to induce adverse responses in anyone who undergoes outdoor exposure.

Carl Selnick from San Diego APCD

1. Several typos were pointed out.

Thank you for pointing out these errors – we will correct them in the final report.

Joint submission endorsed by: American Lung Assoc. of CA, Environment California, Environmental Defense, Kirsch Foundation, National Parks Conservation Assoc., Merced/Mariposa County Asthma Coalition, Fresno

***Metro Ministry, Sierra Club CA, Medical Alliance for Healthy Air,
Community Medical Centers***

1. Support the recommendations.

Over 200 submissions from private citizens

1. All in favor of the recommendations.

Summary of Responses to Comments on Chapter 10: Quantifying the Adverse Health Effects of Ozone

A. Key Comments

1. Question of causality from epidemiologic studies; use chamber studies instead

Usually, epidemiologic studies by themselves cannot “prove” causality. However, it is important to mention in this context that ozone has the benefit of numerous human chamber and animal studies, and extensive knowledge about biological mechanisms, so there is more than sufficient information supporting a causal relationship between ozone and cardiopulmonary health. The key question is the magnitude of the relationship and the shape of the CR function (including thresholds) for the population at large which epidemiologic studies can provide including a wide range of potential health outcomes.

There are a number of reasons for using epidemiologic studies. While human chamber studies have the merit of being controlled experiments, they usually involve small sample sizes that do not include the most sensitive subpopulations, and cannot capture severe outcomes like hospitalization or premature death. Lagged or cumulative effects are similarly omitted, and only a limited range of exposures is examined. In short, human chamber studies are helpful to support causality and to determine effects of short-term exposure on measures like lung function in generally healthy individuals, but they cannot give us the general population response to exposure to ozone in the presence of other pollutants. For the latter purpose, epidemiologic studies which incorporate varying populations, exposure scenarios and behaviors, and health outcomes would best serve to isolate the human response to a particular pollutant and be the source of quantitative estimates for health impact assessment.

2. Ozone mortality estimates

There’s some misinterpretation of the long-term epidemiologic evidence. In the Harvard Six Cities, ozone levels were similar in the six cities, so the study did not have the power to detect ozone-related effects, (which is different from not finding associations). In the most recent American Cancer Society publication, summer ozone shows a positive and nearly statistically significant association with cardiopulmonary mortality, so there is consistency with the time-series literature.

The two meta-analyses of the worldwide literature by WHO and Levy et al. have yielded consistent estimates, so the real question remains as to whether NMMAPS is a better approach for estimating the effects than a literature meta-analysis. There are concerns regarding publication bias in the meta-analyses, but

there are concerns that the NMMAPS statistical approach overcontrols for weather. Given this, it is entirely appropriate to have bounding estimates that have NMMAPS as a lower bound and WHO/Levy/Steib as an upper bound – as was discussed in the Chapter. In addition, staff plans to revise the WHO estimate to consider correction for publication bias and to consider results of recently completed meta-analyses of ozone mortality studies when they are published. These meta-analyses indicate associations between ozone and mortality and do not include an effect estimate of zero within their range of estimates. Further, there is some possibility that the technique used to correct for potential publication bias is not appropriate and therefore may lead to an underestimate. Thus, results will be examined and presented as a probable range accordingly.

3. Threshold assumptions

In our next version, we will examine two different cases regarding thresholds: one in which no threshold is assumed and another with an assumed threshold. However, for the latter case to be empirically correct, the concentration-response functions need to be adjusted to correctly fit the assumption. We will utilize information on the ER visits studies to suggest the size of the slope coefficient with and without an implied threshold. These relative slope estimates will then be used to adjust all of the CR functions for sensitivity checks

4. Estimation of exposures

Staff recognizes the assumption of equal distribution of population across each county is an oversimplification of the true population distribution but is not likely to cause significant bias in either direction. Regardless, we plan to perform a sensitivity check on the exposure estimation methods by interpolating air quality measurements from nearby monitors to derive exposures for each census tract. Health benefits would then be calculated at the census tract level using census population.

5. Rollback scheme

During the period that begins today and ends at some future attainment date, many factors will affect how ozone levels will change in each California air basin. These factors, which include patterns of population growth, emergence of new technologies, and strategic decisions by air quality managers, are more or less uncertain. Into this uncertain future, we projected the benefits of attaining the proposed ozone standards based on rational but necessarily speculative ozone projections. All methods of projecting ozone are speculative, but we believe our approach is subject to fewer difficulties compared to the other approaches that we considered or that others have recommended.

The concern regarding the same proportional change (above 0.040 ppm background) in ozone applied at all locations within an air basin reflects the observation that ozone usually does not change at the same rate throughout a basin. For example, our analysis of ozone changes in the South Coast Air Basin since 1980 shows that ozone at different locations changed in somewhat different proportions. However, while past performance is not a guarantee of future performance, it is a good indicator. The factors that produced historical changes in ozone will not necessarily follow the same path in the future. In addition, these factors may not be the same in other air basins as they are in the South Coast Air Basin. Therefore, we consider it quite appropriate to focus on the required change in the ozone design value at each design site.

For a basin to attain the standard, the design value (characterizing high ozone) at the design site (the site with the highest design value) must be reduced to the level of the standard. For each basin, the proportional change required of the design value at the design site was applied to ozone at all sites in the basin. Data from the South Coast Air Basin indicate that this approach may understate the actual benefits that would accrue when the standard is attained. That is, the proportional change (historically) at the design site was less than the proportional change found for almost all other locations in the basin.

Another suggestion is that photochemical simulation models be used to project daily ozone changes within California air basins. After all, the chief use of these models is to project future ozone as a key part of the planning process. Unfortunately, that pathway is not feasible. In the planning process, it is common for the model to calculate in great detail the response of a single set of high-ozone days, called an "episode", to alternative emission reduction scenarios. To project the benefits of attaining the standard, however, the response of all days or all types of days must be addressed. To apply a reasonable set of alternative emission reduction scenarios to a set of episodes representing all types of days in all California air basins would require many hundreds of model runs. Although simplifications could be imposed to limit the number of model runs to a feasible number, the simplifications would then lead to criticisms similar to those raised concerning the method we chose to use.

6. Conversion factors for study results based on various averaging times

As we reported in the document, an empirical examination of the California monitoring data indicates that the assumed national ratios are similar to those found in the highly populated areas of the State.

B. Comments by Commenter

Donald H. Stedman

1. Commenter suggested adding 2001-2003 data in Figure 10.1, which currently stops at year 2000.

The purpose of Figure 10.1 is to demonstrate the rate of change in long-term ozone trends from 1980's. It now stops at year 2001. Since ozone did not change much from 2001 to 2003, adding 2 more years of data would not change the results.

2. Commenter suggested examining the health effect changes from the past to the present as a way to validate the current approach of predicting the benefits from attaining the standards in the future.

Many changes have occurred between 1980-82 and the present, including population growth, demographic shifts, health care system changes, etc. It would be nearly impossible to simply eyeball past data to validate the current estimates. However, several studies conducted in locations including, but are not limited to, the Utah Valley, Dublin, Hong Kong, and (the former East) Germany and Los Angeles have validated that health improvement occurs after discrete changes in air pollution levels.

3. Commenter suggested there might be errors in the rollback formula for OzAttain (ozone under attainment scenario).

The formulae are correct. In Stedman's example, a basin maximum B_{max} of 0.18, a standard of 0.09 and a background BG of 0.04 would lead to the rollback factor RF of 0.64. Thus, a current ozone value of 0.15 would be rolled back to $0.04 + (1-0.64)(0.15-0.04) = 0.08$, not 0.09. The rollback methodology was not designed to bring all current ozone values into attainment; rather, it was designed as a reasonable expectation of what would occur as the high values coming into attainment.*

Suresh Moolgavkar

1. (p. 1) Thurston and Ito's 2001 paper showed that the estimated effects of ozone on mortality were sensitive to how temperature was controlled

This is true; however, their conclusion was that studies that more appropriately captured weather trends (with non-linear relationships) found higher ozone CR functions. Therefore, it is likely that some of the earlier studies underestimated the effects of ozone.

2. (p. 1) At the end of the first paragraph, and elsewhere in this critique and others, it is stated that the associations in epidemiologic studies cannot lead to inferences of causality.

Usually, epidemiologic studies by themselves cannot “prove” causality. However, it is important to mention in this context that ozone has the benefit of numerous human chamber and animal studies, and extensive knowledge about biological mechanisms, so there is more than sufficient information supporting a causal relationship between ozone and cardiopulmonary health. The key question is the magnitude of the relationship and the shape of the CR function (including thresholds), which epi studies, and only epi studies, can provide.

3. (p. 1-2) Argued about the assumption of a threshold for emergency room visits but not for other health outcomes.

We are re-examining the studies to address this inconsistency. In our next version, we will examine two different cases regarding thresholds: one in which no threshold is assumed and another with an assumed threshold. However, for the latter case to be empirically correct, the concentration-response functions need to be adjusted to correctly fit the assumption. We will utilize information on the ER visits studies as to the slope coefficient with and without an implied threshold. These relative slope estimates will then be used to adjust all of the CR functions for sensitivity checks.

4. (p. 2) Commenter states that the long-term exposure studies do not report associations between ozone and mortality.

There’s some misinterpretation of the long-term epidemiologic evidence here. In the Harvard Six Cities, ozone levels were similar in the six cities, so the study did not have the power to detect ozone-related effects, (which is different from not finding associations). In the most recent American Cancer Society publication, summer ozone shows a positive and nearly statistically significant association with cardiopulmonary mortality, so there is consistency with the time-series literature.

5. (p. 2) Regarding the conversion factors applied to epidemiologic study results for various averaging times of ozone measurements, commenter suggests that the national ratios may not be precise.

Commenter may overstate the potential level of imprecision and the implications. As we reported in the document, an empirical examination of the California monitoring data indicates that the assumed national ratios are similar to those found in the highly populated areas of the State. In any case, it is likely that the conversions contribute only a small amount of uncertainty, under the assumption that there is not significant dose-rate dependence.

6. (p. 3-4) Commenter questioned ARB's use of mortality estimate by WHO and that if we do use these estimates, suggests that we should use estimate that corrects for publication bias.

The two meta-analyses of the worldwide literature by WHO and Levy et al have yielded consistent estimates, so the real question is whether NMMAPS is a better approach for estimating the effects than a literature meta-analysis. There are concerns regarding publication bias in the meta-analyses, but there are concerns that the NMMAPS statistical approach overcontrols for weather. Given this, it is entirely appropriate to have bounding estimates that have NMMAPS as a lower bound and WHO/Levy/Steib as an upper bound – as was discussed in the Chapter. In addition, staff plans to revise the WHO estimate to consider correction for publication bias and to consider results of recently completed meta-analyses of ozone mortality studies when they are published. These meta-analyses indicate associations between ozone and mortality and do not include an effect estimate of zero within their range of estimates. Further, there is some possibility that the technique used to correct for potential publication bias is not appropriate and therefore may lead to an underestimate. Thus, results will be examined and presented as a probable range accordingly.

7. (p. 4) Regarding ozone and hospital admissions, commenter suggests that results from the WHO report be used and questions whether results in Thurston & Ito are peer-reviewed.

Staff will take a closer look at the WHO report and investigate whether a bounding distribution parallel to that for mortality might make sense. However, it is reasonable to use the WHO estimates for mortality but not hospitalization. While death is death everywhere, the health care systems vary significantly between the US and Europe, so what one is hospitalized for may also vary. The argument that the book chapter is not peer-reviewed does not hold since the three studies underlying the estimate are peer-reviewed, and the pooling approach was simple inverse-variance weighting, a method commonly used for meta-analyses.

Stan Hayes

1. (p. 2) At a number of points, commenter (and others) raises the argument that human chamber studies should be used instead of epidemiological studies for benefits assessment.

There are a number of reasons for using epidemiologic studies. While human chamber studies have the merit of being controlled experiments, they usually involve small sample sizes that do not include the most sensitive subpopulations, and cannot capture severe outcomes like hospitalization or premature death. Lagged or cumulative effects are similarly omitted, and only a limited range of

exposures is examined. In short, human chamber studies are helpful to support causality and to determine effects of short-term exposure on measures like lung function in generally healthy individuals, but they cannot give us the general population response to exposure to ozone in the presence of other pollutants. For the latter purpose, epidemiologic studies which incorporate varying populations, exposure scenarios and behaviors, and health outcomes would best serve to isolate the human response to a particular pollutant and be the source of quantitative estimates for health impact assessment.

2. (p. 3) Commenter questions the assumption of log-linearity or linearity of the CR functions for values below the levels of the standards.

As mentioned previously, the concern about linearity can be partially addressed with an explicit sensitivity analysis that captures the “hockey stick” CR function in an appropriate way. As a result, Staff will perform some sensitivity analysis which assumes a threshold model and which adjusts the estimated slope.

3. (p. 4) Commenter suggests examining the log-linearity issue by using human clinical data from controlled chamber studies. He mentions explicitly in the first paragraph that healthy young adults were the target population in the controlled chamber studies by Avol, Kulle, and McDonnell.

Using the shape of the dose-response curve for healthy young adults to draw inferences about the shape of the population dose-response curve is highly suspect. One would certainly expect that there may be susceptible individuals that would have a greater response to ozone and that are not included in the chamber studies. If individuals have heterogeneity in the levels at which they respond, there may be a tendency toward linearity (or at the very least, toward lower thresholds than were observed in chamber studies).

4. (p. 5) Commenter suggests including chronic effects such as those studied in Gauderman et al.

Since the Chapter did not include any chronic exposure effects, the discussion about Gauderman is not relevant. However, there are multiple other epidemiologic studies that have documented effects of ozone on lung development, and these have been supported by animal studies. Regardless, Staff thought the evidence of an effect associated with long term exposure, while plausible, was not sufficient at this time.

5. (p. 6) Commenter states that quantitative estimation of mortality and morbidity benefits should be deferred until substantial additional research is conducted.

While caution is warranted in using the current literature to estimate benefits, deferring this work altogether effectively assumes zero benefit and implies that one can never proceed with risk assessment in the presence of uncertainty

(since uncertainty is always present). It is a better approach to acknowledge the uncertainties and come up with reasonable bounding estimates, rather than to ignore the effect altogether. With some additional sensitivity analyses and discussion of key uncertainties, the output will be superior to not having any quantitative analysis. There are a compelling number of studies linking both morbidity and mortality to exposure to ozone at current ambient concentrations. In addition, several meta-analyses on ozone mortality are being submitted for publication, representing a reasonable basis for quantification.

Allen Lefohn

- 1.(p. 3) Commenter questions the background level assumption of 0.04 ppm and asserts that the benefits assessment is sensitive to the selection of a level for background ozone.

Various citations included in the comment imply that our use of 0.04 ppm as the background level for ozone was based on analyses for which the commenter asserts a detailed array of flaws. We do not consider it necessary to defend the questioned analyses in this venue, because our choice of 0.04 ppm for background was not based on the analyses cited. Instead, it was based on the simple empirical observation that as ozone has improved in California, the distributions of 1-hour and 8-hour daily maximum concentrations have "piled up" around the 0.04 ppm level. That is, ozone concentrations higher than 0.04 ppm decrease, but they tend to stop improving in the neighborhood of 0.04 ppm. Please see our more detailed response to the comment regarding the background assumption in the set of comments on that chapter of our report.

Further, the commenter states that the benefits assessment would be sensitive to the choice of background level. A table supporting this contention was included in the commenter's submittal. Based on the example presented in the table, it seems more appropriate to say that the calculated benefits are affected by the choice of background rather than sensitive to the choice of background. We agree that the calculated benefits are affected by the choice of background. However, the magnitude of the effect is relatively small and would not alter the general picture.

The table includes alternative choices for background from 0.040 to 0.070 ppm. Since the vast bulk of the benefits attributed to ozone reductions represent the highly populated coastal and valley regions of the state, any background level significantly above 0.04 ppm is highly doubtful. In these areas, a very few days under extremely unusual circumstances might have a "policy-relevant" background greater than 0.04 ppm ozone, but the incidental frequency of such days means they would have a negligible effect on the overall assessment of benefits. When one compares the results in the table provided for 0.04 ppm to

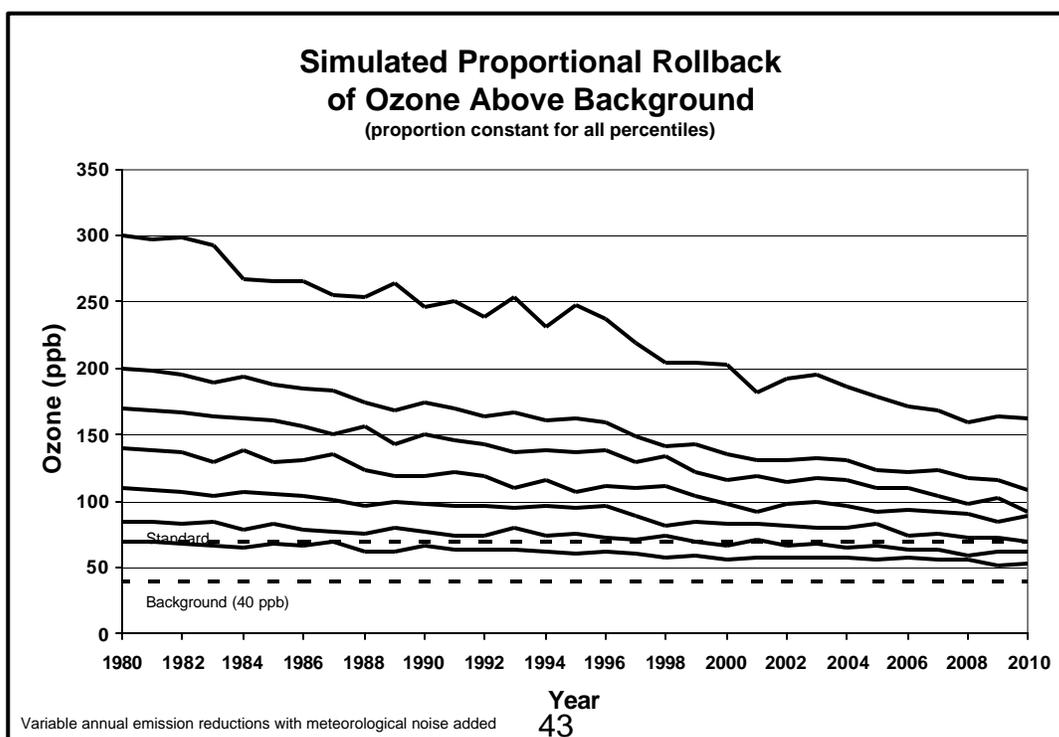
the results for 0.05 ppm, the differences are minor. Accordingly, we take the writer's analysis as more supportive than critical of our choice of 0.04 ppm for background ozone.

2. (p.3-4) Commenter questions the proportional linear rollback method, making the observation that the percentile trends in the South Coast analysis do not convince that a constant rate of reduction occurs across the range of ozone concentrations.

The Appendix shows a similar downward trend in percentiles of ozone maximum observations at each site in the South Coast Air Basin. This suggests that the rollback method used is a reasonable approximation. In fact, it is the most defensible approach among alternative methods, for the alternative methods would likely lead to results that are well within the uncertainty bounds presented in our report

Our interpretation of the South Coast analysis is that the rates of reduction in the portion of each concentration that is above background are more similar than they are different. The rates do not need to be identical to support our rollback methodology. They need to be similar enough to support the general application of one proportion in an uncertain future.

The figures we provided support our rollback approach because the lines are roughly parallel. The lines need not be "straight" to support our "linear" rollback method. They need only be "proportionally" parallel and converge as they near 0.04 ppm or some lower concentration. The following picture is a simulated example of the ideal pattern that would support our method. The figure shows striking similarity to those based on measured data from the South Coast Air Basin from 1981 - 2001.



3. (p. 4-5) Commenter is concerned with the use of epidemiologic data for this work and whether causality can be implied.

As indicated above, the epidemiological studies are not meant to establish causality by themselves, but in the presence of many other studies, are meant to quantify the relationship between ozone concentrations and population health effects. It is not inconsistent to document large uncertainties in the epidemiological literature and to quantify health benefits, as long as the uncertainties are acknowledged and quantified to some extent – as was done in our Chapter.

4. (p. 6) Commenter objects to accruing health benefits below the proposed standards.

As mentioned above, the fact that human chamber studies do not show statistically significant effects below 0.08 ppm does not imply that there are no health risks for susceptible individuals at those levels. All of the epidemiological studies used document effects below the 0.08 concentration.

5. (p. 13) Commenter states that the benefits would have been reduced from the estimated values to 14-24% if one only considers benefits between the current levels and the proposed standards only.

This estimate may not hold since an analysis incorporating a threshold at the proposed standard would have needed to have a significantly greater slope above that point to appropriately capture the information from the epidemiological studies. Exactly what the difference would be requires careful analysis, and staff plans to address this issue via a sensitivity analysis for at least one health endpoint.

6. (p. 44) Commenter notes that there are seasonal differences in ozone CR relationships.

The fact that there are seasonal differences in ozone CR relationships does make interpretation of annual average estimates somewhat problematic, but the argument is overstated. It is unclear what is driving the seasonal differences, but activity patterns related to ambient temperature likely play a role. Given the mild climate in CA, the seasonality is likely more muted, which would imply that using a US-wide estimate would tend to underestimate the effects in CA (by including too much of the wintertime relationship). Regardless, we are not able to re-estimate the original studies published by other researchers.

7. (p. 46) Commenter argues that the sharp disagreement between summer and winter does not argue that weather has not been adequately addressed and that higher ozone appears to be beneficial in the winter.

This is not true. Rather, the sharp disagreement may point out the importance of different activity patterns (time outdoors) during the seasons, the possibility of a population threshold, or the possibility of poor modeling of potential confounders such as weather. Also, the statement that higher ozone appears to be beneficial in the winter is overstated – most of the literature seems to show highly statistically insignificant relationships in the winter and there is no biological mechanism to support this assertion. Finally, the most recent reanalysis of NMMAPS shows very similar effect estimates using the full year of data versus the warm season.

8. (p. 47, 49) Commenter is concerned with the inter-city differences among ozone effect estimates.

Inter-city differences are accounted for in random effects modeling, so the uncertainty behind these differences has been addressed in the epidemiologic studies. There are many possible explanations for the heterogeneity in the effect estimates. For example, the fact that NMMAPS imposed very similar weather and time smoothers for all cities may have resulted in model mis-specifications. In addition, factors such as monitor placement, spatial variability, socioeconomic factors, background health status, use of air conditioners, and housing characteristics all could contribute to heterogeneity in response. So long as it is addressed properly, variation should not be the reason for ignoring the positive associations reported in many of the existing studies.

9. (p. 52) Commenter argues that a relation between exposure and response may be non-linear, hence opting for a linear model can result in regulatory decisions that will not produce the desired mitigation of health effects.

Just because a linear CR function might have significant regulatory implications does not mean that one should not use a linear CR function if the evidence shows that it is appropriate to do so. As stated above, staff plans to address the assumption of linearity via a sensitivity analysis.

10. (p. 52-53) Commenter questions the compatibility of linear CR functions in the context of individual variations.

*The argument regarding the compatibility of linear CR functions in the context of individual variations is not correct. If there are individual-specific response thresholds to ozone, and those thresholds are distributed normally across the population (which the central limit theorem would support), then the population CR curve would resemble a cumulative normal distribution, which is linear at low doses. See Schwartz et al., *The Concentration-Response Relation Between PM_{2.5} and Daily Deaths, Environ Health Perspect 110: 1025-1029 (2002)* for a detailed discussion of this point.*

11. (p. 53) Commenter argues that the re-analyzed results of the NMMAPS study show a negative effect of ozone mortality in the winter, hence acute mortality studies do not show sufficient evidence for calculating mortality effects from ozone exposure.

The most recent reanalysis of the NMMAPS shows no appreciable difference between ozone and mortality relationships for the whole year versus the warm season. Our analysis relies on WHO results and discusses NMMAPS as a lower bound. Additional meta-analyses of U.S. mortality may be published soon and if so, these estimates will be incorporated into our analysis. Although uncertainties in the estimates clearly remain, it would be inappropriate to ignore the vast scientific literature suggestive of a mortality effect.

12. (p. 55) Commenter states that Pope et al (2002) did not discern an ozone effect on total mortality even when restricted to summer months and to specific causes of death.

The estimate in the ACS cohort using summertime ozone is of borderline significance ($p \sim 0.07$). Most epidemiologists would agree that using a p -value of 0.05 as the only indicator of association is inappropriate. Rather, one has to consider the potential biases that may exist in the study (i.e., factors that, in this case, may lower the likelihood of finding an association), the biologic mechanism involved (i.e., in this case, the evidence for inflammation and other effects), and, the related evidence (i.e., epidemiologic evidence of effects on hospital admission and emergency room visits, and toxicological evidence of inflammation and lung restructuring)

Cover letter from WSPA

On page 4, the author argues that benefit estimates predicated on mortality be removed given artifacts of the analysis methodology.

The points about human chamber studies and the appropriateness of conducting analyses in the presence of uncertainty have already been made. One additional point can be made here: what is the logic in arguing for the omission of mortality but the inclusion of morbidity effects? Similar issues regarding seasonality and the use of observational epidemiology would hold for other health endpoints.

John Heuss

1. (p. 2) Commenter questions whether the health effects estimation approach has drawn well from methods used at the federal level.

The Section 812 analysis from U.S. EPA is not specifically focused on ozone, but it includes ozone with explicit determination of ozone exposures and CR

functions. Numerous other regulatory impact analyses by EPA have included ozone and followed an identical approach. Since the intent of the CA analysis is parallel with that of EPA's analysis, it makes sense to use similar methods. It would be worthwhile to mention the ozone health risk assessment conducted by EPA as part of their recommendation for the ozone standard in 1997. However, staff would like to point out that it does not have direct relevance for quantifying the full scope of benefits to the population from attaining the ozone standard since the 1997 analysis only used human chamber studies.

2. (p. 3) Commenter asserts that a linear rollback method is not appropriate because the proportional linear rollback was applied to concentrations above a 0.04 ppm background level and that ozone formation is highly non-linear

Our rollback calculation was calibrated and applied based on the "portion above 0.04 ppm" for each measured concentration. This means that 0.04 ppm rather than 0.00 ppm was the effective rollback target. We believe this target is well established from the empirical data, which indicate a range around 0.04 ppm is suitable for background ozone. The overall evaluation of benefits is not especially sensitive to alternative background levels in the neighborhood of 0.04 ppm.

The well-known non-linear nature of ozone chemistry relates chiefly to the quantitative response of ozone to quantitative reductions in the ozone precursors, VOC (a.k.a., ROG) and NOx. In this case, we are not postulating any particular reductions in VOC and/or NOx. Rather, we only assume that the standard has been attained by whatever emission reductions were needed. The linear aspect to our rollback calculations is the use of a basin-specific proportion (a linear factor) applied to the portion above background for each measured value.

3. (p. 3) Commenter suggests that ozone trends in other California locations (than the South Coast Air Basin) be evaluated to test the assumption of the linear rollback method.

We do not believe that additional study of other California air basins is needed. South Coast covers a vast population, and it is where we have seen a dramatic downward trend in ozone concentrations. Many people found the results of our analysis in the South Coast Air Basin quite "surprising". It seems that experience and general scientific understanding did not correctly align expectations in this case. We believe that the South Coast work sufficiently demonstrates that our roll-back procedures "make sense".

4. (p. 3) Commenter suggests using GIS methods and population by census tract to assign exposures to each monitor.

Staff recognizes the assumption of equal distribution of population across each county is an oversimplification of the true population distribution but is not likely to cause significant bias in either direction. Regardless, we plan to perform a sensitivity check on the exposure estimation methods by interpolating air quality measurements from nearby monitors to derive exposures for each census tract. Health benefits would then be calculated at the census tract level using census population.

- 5.(p. 3) Commenter argues that due to time spent indoors, the population surrounding a monitor is not actually continuously exposed to the concentrations at the monitor.

It is factually correct that actual exposure will tend to be less than the reported ambient concentration, given time spent indoors. However, the epidemiological studies are based on the central site monitors, making exposure estimates at these monitors the most appropriate values to use in the health impact assessment.

6. (p. 4) Commenter states that the finding of a cardiovascular but not respiratory mortality signal from ozone in single-pollutant models is hard to explain as a causal relation.

The fact that effects were seen with cardiovascular but not respiratory mortality could be explained by the relatively low baseline rate for the latter, resulting in low statistical power to detect an effect. Also, many deaths from respiratory disease are likely to be coded as related to cardiovascular death. Finally, there are biological mechanisms, which would render cardiovascular deaths to be a plausible outcome.

7. (p.4) Commenter mentions that Anderson et al. evaluated the potential for publication bias.

Staff recognizes the publication bias correction used in the Anderson et al. study for the WHO. However, it is also possible that the trim and fill method used to correct for potential bias is not correct since that method was initially proposed for estimates that all came from the same population. Nevertheless, our analysis will include a new estimate that corrects for publication bias. In addition, if the new meta-analyses funded by EPA are published prior to completion of our standard development, we will incorporate those estimates into our quantification as well.

8. (p. 5) Here and elsewhere, commenter raises the argument that the staff should include a lower effect estimate of zero.

There is some non-zero probability that the effects are not causal. However, staff proposes to use the existing meta-analytic studies currently available.

These studies do not include zero within the confidence interval. Over the last several years, many studies have reported associations between short-term exposure to ozone and resultant mortality with intervals that do not include zero. This is particularly the case when temperature and time trend are carefully modeled with non-linear smooth terms. However, unlike the examination of particulate matter, researchers have not conducted the full range of sensitivity analyses using ozone. Therefore, we will add some discussion in our text regarding the uncertainties in the estimates that are not incorporated into the confidence intervals..

9. (p. 5) Commenter mentions that Thurston & Ito's work on hospital admissions relied on the meta-analysis based exclusively on studies in cold climates.

Staff notes that the cold climate/warm climate argument made in Levy et al (2001) and Thurston & Ito (1999) in regard to hospital admissions had to do with air conditioning, which is far more prevalent in Alabama than in California. The lack of air conditioning in many parts of California may serve to increase the penetration of ozone into the homes and increase the estimated effect. However, we do not have enough California-specific studies at this time and we cannot ignore the existing literature on this issue.

Summary of Responses to Comments on Chapter 4: Background Ozone

Note: There is considerable overlap among the comments on Chapter 4. To avoid repetition, comments/responses are numbered so that redundant comments can be referenced to a single response.

General Comments:

1. Extensive comments on the discussion of background ozone were provided by two commenters, the Alliance of Automobile Manufacturers and the American Petroleum Institute. These commenters focused on two issues: observed levels of “background” ozone, and potential difficulty in attaining the 8-hour standard due to exceedances caused by “background” ozone.

The latter issue is addressed in responses to comments presented elsewhere in this document. In summary, ARB’s position on this issue is that California law requires the standard to be based on health effects alone. California standards represent the highest concentrations for selected averaging times that are unlikely to induce adverse health effects. Furthermore, the proposed standards are based on responses of subject groups most likely to have significant exposure – people who are active outdoors. Problems that may be encountered in attaining the standard through emission control programs are relegated to the air quality control planning process, and are not properly part of the standard setting process. Under State law, issues regarding actions needed to attain State standards are addressed under the air quality planning provisions of Health and Safety Code sections 40912 through 40930.

The discussion of “background” ozone provided in the Staff Report is provided as part of a general review of the characteristics of ozone as an atmospheric pollutant. The presence (or absence) of a “background” concentration of any particular pollutant is not specifically addressed in State law regarding setting air quality standards. The effect of non-anthropogenic ozone on determinations of attainment status is a separate issue dealt with in California’s Area Designation Criteria (Appendix 2 to California Code of Regulations, Title 17, Sections 70300 through 70306). The following discussion addresses comments bearing on “background” ozone concentrations.

Alliance of Automobile Manufacturers, General Comments by Casimer J. Andary, Director, Regulatory Programs; Technical Comments prepared by Jon M. Heuss and Dennis F. Kahlbaum, Air Improvement Resources, Inc.

1. The appropriate measurement of background ozone must be considered as part of the proposed AAQS (Ambient Air Quality Standard). This issue will impact whether the proposed standards overlap with natural (or transported

from outside of California) levels of pollutants in the air. The staff review uses one model (Fiore *et al.*, 2002) to evaluate background ozone concentrations. We identify specific concerns with that modeling approach and present analyses and data from a variety of sources that conflict with the assessment. The scientific literature on background ozone indicates that the proposed standards overlap with background concentrations. We also provide an analysis demonstrating that the elimination of essentially all human activity in California will still leave portions of California unable to attain the proposed standards. We recommend a broader discussion of background level ozone in the document, including natural fluctuations and measurements at clean sites to allow comparison of concentrations with the proposed standards. We also note that, from a policy perspective, the overlap of background concentrations with the proposed standards is in conflict with implementation requirements for California air districts to develop plans to meet the standards.

As stated above, the discussion of "background" ozone is provided as informative supporting information. Under California law, the level of the standard is to be based on health effects data, and in this context "background" levels of a pollutant are not relevant to standard setting.

The commenter is incorrect in suggesting that the Staff Report discussion relies solely on the work of Dr. Fiore; the text summarizes the work of several investigators and does not rely solely on models. We do not agree with the commenter's contention that the proposed standard "overlaps background." While there is uncertainty regarding background ozone in California, the preponderance of evidence cited in the Staff Report supports an estimate of 40 ± 10 ppbv as typical for the low altitude populated areas of the State. The lack of more detailed information does not preclude California from acting on this standard. Furthermore, data presented in the section on health benefits indicates that the observed frequency of concentrations at or near the proposed standard is decreasing at multiple locations in California, which would not be the case if background concentrations were commonly near 70 ppbv. Although exceptional events may occur that are beyond reasonable control, such as stratospheric ozone intrusion, current ARB regulations provide that these rare meteorological events, when documented, can be excluded from determining compliance with the air quality standard (CCR Title 17 sections 70300 through 70306, Appendix 2).

2. There are several concerns with the analysis. First, it relies on one modeling study and does not account for known criticisms and limitations of the model. Second, we have found a large body of ozone observations that show annual maximum ozone concentrations in remote monitoring sites in the western United States that equal or exceed the proposed 8-hour standard. Third, the conclusions of several other researchers and the United States Environmental Protection Agency (U.S. EPA) concerning maximum

background levels should be considered in the review. Fourth, there are studies of stratospheric ozone, which demonstrate that its impact is larger, more widespread, and more difficult to identify than assumed in the review. Fifth, the review uses the standard as the typical case when various background studies shows it is an extreme value. Sixth, the analysis of background is not consistent with the background assumed by ARB in its assessment of the impact of transported pollutants on ozone in California.

Each of these criticisms is addressed in turn below.

1. The review relies on the Fiore et al. (2002) modeling study to estimate the various components of background ozone. There are a number of problems with this approach. First, it is a model calculation with a global transport model that was not designed to address the components of background specifically in California. The model was run for the summer of 1995, so it was not aimed at evaluating the various sources of ozone over the entire year. As documented in the following, it is not a reliable tool to estimate the mean value or the range of background in California that might influence the attainability of the proposed standards.

Since there is no such thing as a measurement record for “background” ozone in California, ARB believes that the Fiore et al. (2002) study provides a reasonable first estimate. We also cited global and regional modeling and analyses from other authors (e.g. Lelieveld and Dentener, 2000; Galani et al., 2003). The reviewer correctly states that the model is uncertain, but does not suggest an alternative model or systematic estimation procedure.

2. The GEOS-CHEM model Fiore et al. used employs a coarse 2° latitude by 2.5° longitude horizontal grid that the authors acknowledge cannot resolve the steep gradients in surface heating near coastal sites that determine the depth of the mixed layer. The authors indicate that this compromises the simulation over coastal urban environments. In addition, the authors list the inability to resolve topography in California as another problem that manifests itself in the Central Valley of California. The limitations of the model in simulating coastal urban environments and the Central Valley are important in that these are the areas of California with the greatest population and hence man-made emissions.

To begin with, characterizing the Staff Report analysis as solely based on the Fiore et al. (2002) modeling is incorrect. The comment’s assertion that the model may understate down-mixing over California is speculative and contradicts the bulk of information available. For global-scale processes, the coarse resolution is reasonable – even localized stratospheric intrusions in mid-latitudes are the result of synoptic scale “Tropopause Folding Events” (TFEs) in which stratospheric air is incorporated into the upper troposphere due to vertical motion induced by cold fronts. These tend to have geographic scales on the order of

several to tens of degrees and occur at altitudes generally above 10 km (30,000 ft). As noted in section 4.1.2.1 of the Staff Report, most TFEs produce layers of enhanced ozone at elevations of 5 – 6 km (15,000 – 18,000 ft) with weaker ozone signals down to about 3 km (10,000 ft) – altitudes well removed from the populated coastal lowlands or the low-altitude San Joaquin Valley. Terrain interactions with these layers will be generally restricted to higher elevations of the Sierra Nevada, not the lower Coast Ranges or the floor of the Central Valley. Detailed study of TFEs over Europe (referenced in the Staff Report) showed only about 2% of TFEs deliver stratospheric ozone to elevations below 1 km (3000 ft).

Regarding the problem of not resolving surface ozone in populated coastal zones, Fiore et al. (2002) observed that the model tends to **overpredict** surface ozone concentrations in grid cells that include coastal ocean and highly populated land areas. This is due to extending the coastal shallow mixing layer too far inland (and contradicting the commenter's assertion that background is underestimated). This error would tend to overpredict grid-cell-wide natural ozone concentrations as well, so that applying the Fiore et al. (2002) results in the coastal areas of California incorrectly extends elevated coastal plain ozone concentrations into the coastal mountains. In determining the ozone contribution due to long range transport or stratospheric downmixing, this error is irrelevant outside the coastal zone.

Dynamical considerations support this interpretation. The cold Pacific Ocean causes strong, persistent inversions to overlie California's coastal plains at elevations from 300 m to 500 m (1000 – 1500 ft), and similar shallow nocturnal inversions are also common in the Central Valley – thus down-mixing of mid-troposphere ozone (whether from in-situ formation or TFEs) below about 1 km (3000 ft) is even more unlikely in California than elsewhere in North America. For free troposphere ozone to descend to near sea level would require an extraordinary degree of vertical mixing in the atmosphere – a situation inimical to accumulation of high concentrations of pollutants near the surface, thus these events are not expected to be additive with local accumulation of anthropogenic ozone. Moreover, TFEs generally occur in late winter or early spring, well outside the California ozone season (summer and fall). Observational data presented by Newchurch et al. (2003) further support the case that the impact of this error is overprediction. Ozonesonde data from coastal California show that the local inversion drives surface “background” ozone down in summer – the reverse of the pattern at other ozonesonde sites in the U.S. The Newchurch data will be added to the Staff Report to clarify the effect of shallow inversions on surface ozone in undeveloped areas.

3. A source of non-anthropogenic ozone that is important in California is photochemical production from reactions of NO_x that comes from microbial action in the soil and lightning with biogenic hydrocarbons from vegetation. Another complicating factor in California is increased NO_x emissions from soil

related to fertilizer use. The model was not designed to accurately simulate these sources and processes in California.

The estimates of lightning – produced ozone cited in the Staff Report are independent of the Fiore et al. (2002) model. The literature cited indicates that lightning is not a significant source of ozone at low altitudes. Furthermore, lightning is a relatively rare phenomenon in Mediterranean climates (compared to most midlatitude land masses).

Pedogenic (soil-produced) NO_x is highly uncertain, but it is thought to be dependent on temperature and the activity state of vegetation and soil organisms. The protracted dry season in most of California forces natural vegetation over much of the State into semi-dormancy during the dry months, and areas with substantial summer precipitation (mountains, northwest coast) do not experience high temperatures. While ARB cannot precisely estimate the ozone production due to natural soil NO_x emissions in California, it is unlikely to be atypically large. Surface ozone production in areas remote from anthropogenic precursor sources has been observed to be uniformly limited to concentrations well below the level of the proposed standard. Data on 19th century ozone concentrations measured in Europe and the U.S. (Bojkov, 1986) show that spring peak ozone partial pressures were about 4 ± 1 mPa (30-50 ppbv) in the Midwestern U.S. and ranged from 2 – 3 mPa (20-30 ppbv) in Europe. This point will be clarified by adding a discussion of the Bojkov (1986) data to the Staff Report.

Fertilizer emissions are considered in ARB's own modeling for ozone management in the Central Valley, and are not reasonably included in "natural" sources of ozone precursors. Biogenic hydrocarbons have been observed to react with anthropogenic NO_x to enhance ozone downwind of urban areas, as discussed in the Staff Report (Sect. 4.1.3.2) but this, because it is dependent on a local anthropogenic precursor whose sources are already within ARB's regulatory purview, is not properly considered "background" ozone.

4. Any global model contains many assumptions and simplifications that simply cannot be fully evaluated. The GEOS-CHEM model is but one of a number of such models. Fusco and Logan (2003) evaluated the GEOS-CHEM model and report that the model estimates somewhat higher production and loss rates of ozone than other chemical transport models, as much as 15 to 30 %. Since the net photochemical production of ozone is determined by the difference between these two large numbers (a large chemical source term and a large chemical sink term), the net production cannot be precisely determined. They note that differences in modeled photochemical production and loss rates affect the relative importance of the stratospheric source giving examples of other models that indicate a much larger role for the stratospheric source in summer and in winter. Adding to the complexity is that assumptions have to be made about the cross-tropopause flux of ozone

and ozone deposition at the surface, quantities that each have significant uncertainty, too. There are other aspects of the chemical transport models that are also highly uncertain. For example, there is disagreement over how many ozone molecules are produced, on average, from each NO molecule emitted. The recent NARSTO Synthesis Report indicates that more recent studies have reduced the estimated ozone production efficiency from 7 to 10 molecules ozone per molecule NO_x emitted down to 1 to 3. In addition, the NARSTO report acknowledges there is substantial disagreement over key factors such as the magnitude of U. S. United States biogenic VOC emissions (uncertain by a factor of 2 or 3) and natural NO_x emissions from soil and lightning.

The Staff Report discusses the work of Fiore et al. (2002) because it is the only modeling study to date that explicitly treats background ozone in California. Other modeling studies addressing hemispheric to global-scale ozone distributions are available; to the degree they can be compared with the Fiore et al. (2002) results, they do not contradict ARB's interpretation. This particular comment is based on a selective reading of the Fusco and Logan (2003) paper. Their critique of GEOS-CHEM is presented in the context of using that model to estimate global ozone trends. Fusco and Logan (2003) present comparisons with ozonesonde data that show GEOS-CHEM to perform well in this application, with errors compared to low altitude measurements on the order of 10 ppbv or less – generally within the standard deviation range of the measurements.

5. Fusco and Logan (2003) also express concern with the accuracy of the method imposed to simulate the annual flux of ozone across the tropopause, noting that an incorrectly modeled seasonal cycle, as appears likely in the case of the GEOS-CHEM model, could adversely affect the response of the modeled ozone to the stratospheric flux. In summary, there are a large number of questions concerning the conclusions derived from the model, in general, and more specifically in California. Thus, it is not a reliable tool to estimate mean background in California much less the range of background that might influence the attainability of the proposed standards.

Again, this is a selective reading of Fusco and Logan (2003). A full reading of their report shows that GEOS-CHEM tends to overpredict stratospheric downmixing, while being somewhat uncertain on in-situ tropospheric ozone dynamics, with the result that it has a small positive bias in springtime. This would tend to make GEOS-CHEM overestimate background ozone in California.

6. Since the staff recommends that the proposed standards be defined as concentrations not to be exceeded, the Chapter should evaluate the extreme values or yearly maxima of policy relevant background. There is now a substantial body of ozone observations that shows annual maximum 8-hour ozone concentrations in remote monitoring sites in the western United States that equal or exceed the staff's proposed 8-hour standard. This data is

relevant to the issue of a regional, policy relevant background that will hinder the attainability of the proposed standard. Therefore, it should be included and discussed in the Chapter. In California, the ARB has provided the peak 8-hour indicators for all the air basins in the 2004 Almanac as well as in Chapter 7. The yearly maximum 8-hour concentrations in Lake County have averaged 0.069 ppm for the past 20 years and equaled or exceeded 0.07 ppm in 11 of the past 20 years. In the North Coast, the yearly maxima have averaged 0.072 ppm over the past 20 years and equaled or exceeded 0.070 ppm in 13 of the past 20 years. Inspection of the figures in Chapter 7 shows that the proposed 0.070 ppm standard would put the entire state out of attainment.

This comment again refers to attainment issues; not the health effects that drive the standard setting process (see response to General Comment 1). In addition, the comment implies, incorrectly, that measured values in these areas are completely unaffected by anthropogenic emissions. Measured values in these areas reflect a combination of background regional concentrations and the impact of anthropogenic emissions.

7. Alliance commenters present data on ozone concentrations in rural areas across the U.S., then conclude: The idea that peak background is 0.04 ppm is inconsistent with the data from the cleanest of the California air basins where the population and emissions density is only a minute fraction of that in California. While transport from other more populated California air basins may play a role from time to time in the ozone values measured in the most remote air basins, the large fraction of daily 1-hour maximum and daily 8-hour maximum concentrations that are reported as 0.04 ppm and greater in the tables and figures in section 7.3.6 demonstrate a much higher policy relevant background than indicated in Chapter 4.

Data for a much less industrialized period (Bojkov, 1986) suggest that the 40 ± 10 ppbv average presented in the Staff Report is consistent with surface ozone concentrations observed in the absence of modern transportation, utility, and industrial emissions. Recitation of rural concentrations, absent dynamical analyses to support interpretation as “background” are not, of themselves, persuasive that mean background is much higher. The commenters themselves cite modeling they performed for the South Coast Air Basin based on the episode of August 3-7, 1997 that showed that, with “all anthropogenic emissions in the modeling domain turned off (both U. S. and Mexican)... the peak 8-hour ozone during the episode was 37 to 46 ppb.”

ARB agrees that there are may be occasional events of “background” ozone that show higher concentrations, but, as argued in the Staff Report, such events are unlikely to coincide with local ozone production sufficient to exceed the proposed air quality standard.

8. Alliance commenters present an extended discussion of stratospheric downmixing and putative observations of surface ozone impacts of stratospheric downmixing. They conclude: The known patterns of tropospheric folds together with the ground-level ozone- ^7Be analyses by Wolff et al. suggest that stratospheric ozone also contributes significantly to ground-level ozone during times when man-made ozone is present.

The processes controlling the concentrations and survival of stratospheric ozone after it moves into the lower troposphere are very complex. Although ^7Be is radiogenically produced in the stratosphere, its concentrations are not linearly related to stratospheric ozone, especially after movement into the troposphere. The referenced paper suggests recurring downward stratospheric ozone transport over the eastern U.S. More recent analyses of ^7Be and ^{10}Be data from Europe (Zanis et al., 2003) indicates that most stratospheric transport events (STEs) are short lived, and that ^7Be observed during periods of anticyclonic circulation (associated with regional ozone events in the eastern U.S.) is potentially a biased estimator for stratospheric ozone transport due to reduced Be removal rates in dry air and the accompanying high insolation that accelerates in-situ ozone formation by tropospheric photochemistry.

9. In addition to the examples in the references noted above, there are several cases in the references presently included in the chapter of elevated ozone transported long distances that contain a mixture of anthropogenic and stratospheric air. In these situations, routine monitoring data will not be able to distinguish the anthropogenic contribution from the stratospheric contribution. Although the ARB and the U.S. EPA have “exceptional event” policies, it is likely that only a small portion of the stratospheric intrusions that affect ground-level ozone concentrations will be uniquely identified and thereby qualify for the exceptional event policy.

An air mass carrying ozone from a “classic” STE can be distinguished from one carrying anthropogenic ozone by its chemistry. An anthropogenic ozone plume would contain elevated concentrations of long-lived combustion-related gases, such as CO and CO₂; the CO/CO₂ ratio would be elevated, and there would be accompanying combustion-related aerosols, including sulfates and carbonaceous species. In contrast, stratospheric ozone would not be accompanied by other gaseous or aerosol pollutants and would be marked by very low relative humidity (RH) since there is little water in the stratosphere to begin with, and compressional heating during descent to the surface would drive RH very low. Determination of an “exceptional event” would rely primarily on meteorological analyses and chemical evidence, where available, that support a showing that synoptic conditions were compatible with stratospheric ozone intrusion.

10. We would like to see this available literature included in the review of the role of stratospheric ozone on ground level background. It appears from these other studies cited that the Galani et al. (2003) study is not typical of Europe or of the U. S.-relevant studies.

The characterization of ARB's review as being based solely on Galani et al. (2003) is incorrect. ARB believes that the observational record presented by Galani et al. (2003) is representative. Moreover, we find broad consistency in the balancing of stratospheric intrusion and in-situ formation as explanation for tropospheric ozone across the many papers coming from the large, integrated STACATTO program, the general discussion of Lelieveld and Dentener (2000), ozonesonde data, GEOS-CHEM modeling, and other sources. ARB will update the Staff Report to reflect discussion of these responses.

11. Problems with Comparing Average Behavior with an Extreme Value Standard: Chapter 4 focuses on background as it may apply to the stable, stagnant conditions that produce the highest ozone concentrations from man-made emissions. For example, it is argued that some background sources generally peak in other seasons than man-made ozone and that they are generally not major contributors to observed peak ozone. However, the review proposed an extreme value standard that applies everywhere in California all the time. So the range of background during worst case urban episodes is not the only concern. The evidence from observations around the globe and modeling is that the factors and processes that affect ozone levels in the atmosphere are very complex. There are complex chemical and dynamic processes involved that interact in a variety of ways. Stratospheric intrusions create elevated ozone plumes that may persist or mix with neighboring air. Under certain conditions, long-range transport of man-made ozone or its precursors from continent to continent is observed. Large-scale plumes originating in the stratosphere and plumes from long-range transport and plumes from nearby urban areas can all cause elevated ozone levels exceeding the proposed state standards. Sometimes ozone from these sources is mixed together so that one cannot identify a specific source. It just takes one combination of the many different combinations of these sources to violate the state standard.

Determination of attainment or violation of State standards does not solely rely on identifying the highest measured concentration at a monitoring site. Statistical filtering is used to avoid arbitrary determination of attainment status due to very rare or unique situations (Guidance for Using Air Quality-Related Indicators in Reporting Progress in Attaining the State Ambient Air Quality Standards, ARB Research Report 93-49, 1993, pp. 21-26). In addition, measured values above the standard that can be shown to be very rare or associated with unusual weather or sources beyond regulatory control can be removed from consideration as exceedances of the standard through the State's Attainment Designation process. Designations and the procedures for designation are

subject to public review and comment since the California Health and Safety Code (H&SC) requires the Board to periodically review the criteria it uses for making State area designations and both the H&SC (section 39608) and the regulations covering designation criteria (CCR, title 17, section 70306) require the Board to review the area designations annually and to redesignate areas as new information becomes available.

12. The Background Used in ARB Transport Assessments: The March 2001 ARB Staff Report, in reference to background level ozone, states the following: “For instance, clean air, such as the air mass over the Pacific Ocean has a normal background of 4 pphm. Areas in the mountains may have background concentrations of 5 or 6 pphm...” (March 2001 Staff Report at page D-2) Since 4 pphm is the same as 0.04 ppm or 40 ppb, the ARB, in assessing transport, considers the normal or average clean air background coming off the Pacific to be 0.04 ppm. This contradicts the statement on page 4-11 that the maximum clean air background is 0.04 ppm. The “clean air” boundary conditions used in photochemical modeling also specify 0.04 ppm ozone because it is widely accepted as an average clean air background. The normal background at elevation noted in the March 2001 Staff Report of 0.05 or 0.06 ppm is very close to the proposed 8-hour standard of 0.070 ppm, so that fluctuations around the normal background will likely cause violations of the proposed standard.

The reviewer has identified an error in the text of the 2004 Staff Report – the referenced statement in the Summary should read “The models reviewed here indicate that average “natural background” ozone near sea level is in the range of 15 – 35 ppbv, with a maximum monthly mean of about 40; at altitudes above 2 km stratospheric intrusions can push peak “natural background” concentrations to 45 – 50 ppbv.” This will be corrected in future versions of the Staff Report.

13. Policy Relevant Background Levels: Given that the extreme values of background can approach or exceed the proposed standards, the proposal allows little or no room for ozone from mankind’s activities. With a policy relevant background that varies substantially, there will be times and places where the background approaches the 70 ppb level of the proposed 8-hour standard. The Chapter limits the discussion of policy relevant background to the meteorological conditions conducive to peak urban ozone formation. While this is currently the limiting case for development of control plans, it may not be under a 70 ppb standard. If the policy relevant background is 40 ppb and the standard is 70 ppb, the amount of ozone that can be formed from man-made emissions is only 30 ppb. So even with a 40 ppb background, the proposed standard allows little room for man’s activities. On a day when the background is 60 ppb, the margin for man’s activities will be only 10 ppb. On a day when the background is 70 ppb, there is no margin for man’s activities. While this illustration over-simplifies the complex chemical and meteorological

processes involved in ozone formation and transport, it demonstrates that transport of ozone from upwind natural and non-California man-made sources can make the proposed standard unattainable.

The case presented has an internally modeled “background” that is in the range of 37 – 46 ppb, well within the range discussed in the Staff Report. Peak ozone events in Southern California depend on strong local temperature inversions and overlying synoptic high pressure. These conditions preclude rapid down-mixing of stratospheric air to the surface. Slow downmixing from the stratosphere at this time of year is relatively weak, and any stratospheric air present near the surface will be many days old and strongly diluted, thus high natural “background” ozone is not expected during such an episode. No case is made for why much higher “background” ozone can be assumed in discussing these findings. Note that ARB cannot comment in detail on the commenter’s modeling exercise since the details of the model specifications have not been included in the comments.

14. Even with a 40 ppb background advected into the South Coast Air Basin, the degree of emission control required to attain a 70 ppb standard is unreasonable. The Alliance of Automobile Manufacturers asked ENVIRON International to carry out photochemical modeling of Southern California to investigate whether an 8-hour ozone standard of 0.070 ppm could be achieved... even 95 % additional control of the man-made VOC and NO_x from current 1995 baseline is not enough to attain the proposed 8-hour standard... The difficulty in finding additional emission reductions to enable the South Coast to attain the federal 1-hour standard is well known. It has led to the use of long-term or “black box” emission reductions within the Basin in order to demonstrate attainment of the federal 1- hour standard... The 90% control of man-made emissions beyond the 2003 AQMP did bring [other] air basins below the 70 ppb proposal, but when Mexican emissions were added back in, the proposed 8- hour standard was exceeded in the San Diego and Salton Sea air basins... For other situations in which there is an additional contribution from natural sources or transport of non-California man-made ozone or ozone precursors, the margin for manmade ozone associated with the 70 ppb standard will be reduced. In much of California, the reactions of biogenic NO_x emissions (that maybe increased due to fertilizer use) and biogenic VOC will contribute additional uncontrollable ozone that will add to the regional background coming off the ocean.

See response to General Comment 1.

15. Summary of Chapter 4: Background Ozone in California: In summary, the scientific literature on background ozone indicates that it is highly variable and can reach levels close to the current California 1-hour standard. There is ample evidence that the proposed 8-hour standard will be exceeded, as a result of the regional background from natural and non-California sources, in

all California air basins and throughout much of the Western U. S. including many national parks. The ARB discussion of background relies on an unverified model calculation and discounts the large body of observations and analyses around the world that indicate higher maximum background concentrations than ARB assumes.

ARB does not concur with the commenter's assertion that ozone in California, absent a contribution from in-State anthropogenic precursor sources, can approach the current 1-hour standard. ARB accepts that there may be some (as yet unquantified) potential for exceedances of the 8-hour standard due to a combination of natural and non-California ozone sources, and anthropogenic ozone production. However, we believe that these will be infrequent in time and space, generally restricted to high altitude, and that there are administrative mechanisms in place to address unusual events such as stratospheric ozone intrusion. Nonetheless, the putative existence of such events is not relevant to the standard setting process (see response to General Comment 1).

16. In addition, the scientific literature and the U.S. EPA ozone scientific review support a higher maximum background than ARB assumes. The review states that the influence of tropopause folding events that insert high concentrations of ozone from the stratosphere into the troposphere will be easily recognized and dealt with by the exceptional events policy. However, as documented in the references noted above, there is evidence that these events may not be easily identified. The policy relevant background varies spatially and temporally. It varies substantially on both seasonal and short-term time scales, and policy relevant background levels leave little room for man's activities.

ARB recognizes that there may be occasions when "background " ozone contributes to exceedances of the proposed standard. However, as discussed above, health effects levels, not attainability of the standard, are the primary determinants of the standard, and there are administrative mechanisms in place to address exceedances caused by exceptional events such as stratospheric ozone intrusion.

Allen S. Lefohn, Ph.D., A.S.L. & Associates, Helena, Montana: Comments prepared for American Petroleum Institute, Washington, D.C., and Western States Petroleum Association, Sacramento, CA, Dated August 27, 2004.

Note: Dr. Lefohn submitted nearly 50 pages of comments. Much of his submission is devoted to a literature review and presentation of extensive monitoring data. Point-by-point citation and reply would reiterate discussions in the Staff Report and revisit issues dealt with above. ARB staff have reviewed Dr. Lefohn's materials, and we present here responses to his major points, conclusions, and criticisms of the Staff Report. The responses here are organized according to Dr. Lefohn's summary.

The comments focus is provided on the following issues:

1. Estimates of *policy-relevant background* concentrations need to consider the important contribution from stratospheric O₃, as well as other natural sources;

Stratospheric ozone intrusion is explicitly treated in the Staff Report. Discussion of significant historical ozone data cited by Dr. Lefohn (Bojkov, 1986) will be added to the Staff Report. The measurements cited by Dr. Lefohn are within the range discussed in the Staff Report.

2. There is large variability among global models on the attribution of the contribution of natural O₃ to the background;

ARB agrees that there is wide variation among models, however much of the disagreement is due to the difficulty of comparing results across models with different vertical and horizontal resolutions. In preparing the Staff Report ARB selected recent modeling studies that are constrained by recent observations and theoretical understanding of ozone formation and transport.

3. The California Ambient Air Quality Standard for Ozone Document (CAAQSOD) states that ground level impacts from fires are typically in the range of 15-25 ppb. Such is not necessarily the case;

ARB agrees with Dr. Lefohn that there are documented cases of very large fires, such as the Yellowstone fires of 1988 that have produced elevated ground level ozone measurements. We disagree with Dr. Lefohn's contention that such an event may be missed by California's exceptional event policy. The Yellowstone fires were, by their nature, a very rare ("once a century") event, with smoke impacts across several states – such an event would be impossible to overlook as an "exceptional event." The lower ozone impact estimates presented in the Staff Report were based on California's fire experience and relate to recurring large fires in the State, not extreme events.

4. Given the limitations discussed in this report with the Lin et al. (2000) and Jaffe et al. (2003b) trending analyses, the scientific evidence for an Asian influence on surface O₃ concentrations on the United States is weak and further research efforts are required;

ARB agrees that Asian ozone is not, at present, a significant source of enhanced ozone in California. The Staff Report included the modeling and anecdotal evidence in order to provide the reader with a complete picture of the exogenous ozone sources that may enhance locally formed ozone in California. We agree that further research on this problem is desirable. We disagree with Dr. Lefohn's interpretation of the data presented by Jaffe et al. (2003b) as suggestive that elevated springtime ozone at Mt. Lassen is more likely to be due to stratospheric

intrusion rather than long range transport. The persistent vertical stratification characteristic of the meteorology in the region makes both sources more likely to be detected at the Lassen site. Regardless of the source, the Mt. Lassen springtime ozone peaks are the types of event that ARB expects would be subject to review as “exceptional events.”

The table of “rural” ozone concentrations Dr. Lefohn quotes from the paper by Lin et al. (2000) needs some comment as well. ARB rejects the central assumption of that paper that the data represent sites not strongly influenced by urban and industrial ozone. Many of the sites listed by the AIRS database as “rural” are in fact downwind of major urban areas. The narrowing of the frequency distributions of ozone concentrations observed at such sites is fully explainable by the effects of changing NO_x / VOC ratios in upwind areas and the reduced effect of local NO_x titration near the monitoring sites due to improved motor vehicle emission controls. We believe the data in the paper are not proof of the contention of growing background, Asian or local.

5. The CAAQSOD emphasizes that the violations associated with the proposed 0.070 ppm 8-hour average standard would occur during the summertime, when stratospheric O₃ contributions are thought to be minimal. However, when one characterizes the hourly average concentrations collected in 2003 for 184 monitoring sites in California, one finds that violations of the proposed 8-hour average standard occur during spring, summer, and fall;

ARB disagrees with Dr. Lefohn’s interpretation of the monitoring data. The Staff Report acknowledges the potential for elevated background ozone concentrations at high altitude sites, but we are unconvinced of the “natural” origin of many of the ozone peaks listed, and the commenter does not provide dynamical analyses to support such interpretation. ARB does admit to the potential for infrequent standard exceedances due in part to influx of exogenous ozone, however any exceedance caused by stratospheric ozone intrusion or wildfire would be subject to “exceptional events” review under State law.

6. Because violations of the proposed 8-hour average standard occur during spring summer, and fall, policy-relevant background concentrations that occur during seasons other than summer will have to be characterized so that emission control actions result in optimum reductions in hourly average O₃ concentrations;

ARB agrees that “off season” ozone peaks above the proposed standard will be encountered at California monitoring sites. The sources of these peaks will need to be investigated in the course of designating “design days” for control plans and to insure that exceedances due to natural or transport processes outside ARB’s regulatory control are not misinterpreted.

7. At some monitoring sites in California, when stratospheric O₃ predominates in comparison to anthropogenic sources during the spring, it may not be possible for regulators to control hourly average concentrations in the 0.05 – 0.06 ppm range using emission reduction strategies;

ARB agrees that current control strategies may not be effective for ozone concentrations in this range, however, as discussed in the response to comment 1 above, the standard is to be based on health effects, and, moreover, the proposed standard would not require any regulatory action for ozone concentrations in this range.

8. The empirical data provide a solid indication to CAAQSOD that policy-relevant background O₃ hourly average concentrations, as defined on page 4-1, are more than likely higher than the 15-35 ppb discussed in the document. Using models that provide highly uncertain concentration estimates provides an overly optimistic message to those who are responsible for implementing control strategies.

We agree that reading the long term mean values presented in the Staff Report as absolute maxima could be misleading. The text in the Staff Report will be revised to insure such a misreading is precluded.

9. In some of the modeling efforts to estimate natural background O₃ concentrations within North America, investigators removed all anthropogenic emissions of NO_x, CO, and nonmethane hydrocarbons (including NO_x emitted from aircraft and fertilizer, but not biomass burning). Because the State of California does not plan to eliminate all anthropogenic emissions of NO_x, CO, and nonmethane hydrocarbons (including NO_x emitted from aircraft and fertilizer), the estimates for the range of hourly average policy-relevant background concentrations will be greater than the 4-hour afternoon average background (i.e., natural background, in North America and anthropogenic and natural background outside of North America) values estimated by these models.

ARB does not propose or project that all anthropogenic sources of ozone precursors in California could be eliminated. The background discussion is intended to give readers a sense of the scale of the in-State anthropogenic contribution to observed ozone concentrations. Comparing model results with and without in-State anthropogenic sources provides a cross-check on estimates based on interpreting the literature on global ozone formation processes and reporting the scant data available from preindustrial sampling.

10. The proposed 8-hour standard of 0.07 ppm is violated in pristine places, such as Yellowstone National Park in Wyoming. The ambient concentrations experienced at Yellowstone National Park in the springtime represent policy-relevant background as defined in Chapter 4 of the CAAQSOD. This implies

that the proposed 8-hour standard will be difficult to attain in some areas that are affected by stratospheric O₃ during the spring and that perhaps the methodology used by Staff to propose the form and level of the 8-hour standard provides highly uncertain results.

As stated above, ARB accepts the possibility that some rural sites, especially those at high elevation, may experience occasional ozone concentrations in excess of the proposed standard that are not obviously linked to local or upwind in-State emissions of ozone precursors, and we plan to address these through our "exceptional events" policy.

Comments were received from:

1. David Bates
2. Carl Selnick
3. Hal Levin
4. Joint submission endorsed by: American Lung Assoc. of CA, Environment California, Environmental Defense, Kirsch Foundation, National Parks Conservation Assoc., Merced/Mariposa County Asthma Coalition, Fresno Metro Ministry, Sierra Club CA, Medical Alliance for Healthy Air, Community Medical Centers
5. Natural Resources Defense Council
6. American Petroleum Institute/Western States Petroleum Association
7. Engine Manufacturers Association
8. Alliance of Automobile Manufacturers
9. Joint submission endorsed by: American Chemistry Council, California Business Properties Association, California Cement Manufacturers Environmental Coalition, California Chamber of Commerce, California Citrus Mutual, California Cotton Ginners Association, California Cotton Growers Association, California Farm Bureau Federation, California Independent Oil Marketers Association, California Independent Petroleum Association, California League of Food Processors, California Manufacturers and Technology Association, California Natural Gas Producers Association, Construction Materials Association of California, Council of Shopping Centers, Industrial Environmental Association, National Association of Industrial and Office Properties-California Chapters, Nisei Farmers League, Retail Industry Leaders Association.
10. Over 200 submittals from private citizens, all in favor.