Comments Regarding CASAC Advice on Reconsideration of the 2008 Primary Ozone National Ambient Air Quality Standard

Prepared for the Alliance of Automobile Manufacturers

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Introduction

As indicated in Lydia Wegman's January 25, 2011 Memo to Holly Stallworth of the EPA Science Advisory Board Staff, EPA is requesting additional advice from the Clean Air Scientific Advisory Committee (CASAC) on the strengths and limitations of the scientific evidence at issue in the Administrator's proposal for reconsideration. Although EPA is asking CASAC to focus on nine specific charge questions, the memo also indicates "...we would appreciate any further advice that CASAC wishes to provide." In addition, the memo cautions that Panel members should be mindful that they should consider only information that was in the record of the 2008 NAAQS review. Finally, the memo indicates that the Administrator looks forward to CASAC's consensus advice on the charge questions as she considers a standard in the range of 0.060 to 0.070 ppm.

The Alliance of Automobile Manufactures participated in the 2008 NAAQS review and the re-consideration, providing detailed comments to the Agency.[1] Based on a review of the EPA request, we offer comments below on the specific charge questions. Our comments also refer to the locations in earlier submissions of the Alliance and other interested parties where further detail can be found. However, we respectfully begin with comments to CASAC on the framework and process being followed in the teleconference.

First, because of the importance of the choice of an ozone NAAQS that is sufficient to protect the public health but not more stringent than necessary, we encourage CASAC to go beyond the charge questions and provide additional advice. For example, we raise additional important questions in these comments that CASAC should address. Second, we urge the CASAC Panelists to provide their advice without artificially enforcing a consensus view. If there is consensus, or a range of opinion, that information should be provided to the Administrator.

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[1] Comments of The Alliance of Automobile Manufacturers on EPA's Proposal for Reconsideration of the Primary and Secondary National Ambient Air Quality Standards for Ozone, March 22, 2010, Docket ID No. EPA–HQ–OAR–2005–0172- 12133.1. "Alliance Comments,"

Third, we ask CASAC to consider whether the limited time for discussion of each question, less than 18 minutes per question in the draft Agenda for the February 18, 2011 call, is sufficient to allow the Panel time to fully explore the issue and respond to the charge. In addition, there is no time on the Agenda to address other issues that Panelists may want to bring up. We would encourage the Panel to take the necessary time in a public format to discuss and develop its answers.

Fourth, we ask CASAC to address the difficulties the Panel faces because it is being asked to develop its views based only on the information available in the record of the 2008 review. The 2008 review was based primarily on the February 2006 Criteria Document that assessed literature through 2004 along with selected studies published in 2005 and 2006. It thus represents the state of science as of six years ago. Since the Clean Air Act requires EPA to update the "criteria" every five years, it follows that the "re-consideration" is not based on the accurate and latest science envisioned by the Clean Air Act requirement. In addition, since the Panelists are aware of the recent literature (and older studies that may be important but not included in the 2006 CD), it puts them in an awkward position to ask them to ignore new thinking about ozone health-related science.

Fifth, with regard to the newest science, there is substantial new information that calls into question a number of key assumptions EPA made in the re-consideration proposal. EPA carried out a 38-page Provisional Assessment (PA) of the post-2006 CD literature at the time of the re-consideration and reported that the newest literature does not materially change the conclusions of the Agency's science assessment. However, a detailed analysis of the PA and the relevant literature by the Alliance indicates that the PA does not reflect the latest science because it completely overlooks several significant new scientific studies, is not a thorough assessment of the studies it does summarize, and omits consideration of new information concerning a key issue in the review, the background of ozone. We urge the CASAC panel to read the critiques of the PA submitted to the Agency by the Alliance[2] and by the National Association of Manufacturers[3] (NAM) prior to the February 18, 2011 teleconference.

Sixth, we believe that CASAC needs to provide the Administrator with additional advice concerning the limitations of the method used to estimate background ozone. In the 1997 Review, the Administrator found that a standard set at a level of 0.07 ppm was not requisite to protect public health in part because, based on the 1996 CD, an 8–hour standard set at 0.07 ppm would be closer to peak background levels that infrequently occur in some areas due to non-anthropogenic sources of ozone. In the 2008 Review,

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[2] Alliance Comments, supra note 1, Appendix A, Review of More Recent Relevant Studies to the Choice of Ozone NAAQS.

[3] NAM, Attachment 1, A Detailed Review of the New Data in EPA's Provisional Assessment and the New Data Excluded from the Assessment Does Not Support a Reduction in the Ozone Standard, Docket ID No. EPA–HQ–OAR–2005–0172-12439.3.

EPA changed the way background was evaluated, relying solely on the use of a global atmospheric model to calculate mean background. EPA also neglected to estimate the extreme values of background even though the statistical form of the ozone NAAQS is an extreme value. This is an important omission that led the Agency and CASAC to assume, incorrectly, that a standard in the range of 0.06 to 0.07 ppm was substantially higher than background and achievable throughout the country.

Comments on the charge questions

In the following, the eight charge questions are repeated and Alliance comments are provided, along with references to sections of earlier public submissions that provide additional detail.

1. What is your advice on the overall strengths and limitations of the evidence from controlled human exposure and epidemiological studies and the results of the exposure and risk assessments, in the context of EPA's selection of a standard level within the proposed range that would be requisite to protect public health with an adequate margin of safety, including the need to protect susceptible populations, such as children and people with asthma?

The controlled human exposure studies provide a strong and consistent body of information on the effects of 1-to-3 hour and 8-hour exposures to ozone. The first effects - transient, reversible FEV_1 decrements - are the body's reflexive reaction to the presence of an irritant gas unrelated to sensations of discomfort. Such effects occur after exposures to 0.08 ppm for 6 to 8 hours when the subjects are exercising at a rate that would be considered strenuous when carried out intermittently for an eight-hour period. Whether such effects occur at 0.06 or 0.07 ppm is highly controversial since the answer depends on how the baseline is evaluated, how the precision of the test is considered, how the day-to-day variability of a subject is evaluated, and how the data is statistically analyzed. In either case, single incidences of the effects at 0.08 ppm (for either healthy or asthmatic subjects) were not considered to be adverse by CASAC and EPA staff in the 1997 review. Nothing in the body of controlled studies has changed to alter that view.

Since the first effects on the performance of lung function tests occur at 0.50 ppm in sedentary individuals, the vast bulk of personal exposures of either the general population or the susceptible population are far below the thresholds for the first effects identified in controlled studies. This provides a large margin of safety for the population as they go about their daily activities.

Other effects on the respiratory system, as discussed in the response to question #2, occur at higher concentrations as ozone triggers responses in the body's defense mechanisms. Since the threshold for even the first indications of an inflammatory response is higher than that for the reflexive FEV_1 response, no adverse health effects are to be expected at either the current 0.075 ppm standard or a more stringent standard.

In contrast to the controlled studies, the epidemiological studies have severe limitations

beyond those already acknowledged by the Agency. These limitations are discussed in the answer to questions #6 and #8 and in detail in the Alliance Comments Appendix B at pages 17 to 35.[4] Basically, the full pattern of results in comprehensive studies demonstrates a biologically implausible wide range of results from positive to negative and spatial and temporal variability that is not consistent with the spatial and temporal distribution of ozone. Therefore, it is inappropriate to assume that ozone is causing premature mortality or hospital admissions. The full pattern of results demonstrates a heretofore underestimated stochastic variability in the data. Although there may be somewhat more positive associations than negative associations in comprehensive studies, there is so much noise or variability in the data that identifying which positive associations may be real health effects and which are not is beyond the capability of current methods. Based on the body of controlled studies, a standard at the upper end of the range or the current 0.075 standard would adequately protect the public health.

2. Recognizing that controlled human exposure studies at 0.080 ppm O3 and above have provided evidence of other health effects, including inflammation and increased airway responsiveness which may occur through different physiological mechanisms than the reduction in FEV1, how should the results of these studies inform our understanding the health effects to healthy adults at exposures levels from 0.060 to 0.070 ppm?

As noted in the answer to question #1, other effects on the respiratory system occur at higher concentrations than those that cause FEV_1 decrements as ozone triggers responses in the body's defense mechanisms. For example, the immune system responses noted by EPA as the first indications of "inflammation" are physiological processes that occur in all living organisms under the stimuli of daily life. The first reported changes (that occur in humans with heavy exercise after 1- to 3-hours above a threshold of 0.18 to 0.20 ppm) are small and reversible and well within the range of physiological variability. They fall into the category of biochemical markers that the American Thoracic Society indicates do not necessarily imply adversity. The review by Mudway and Kelly[5] notes that for neutrophils transiting into the lung -- one of the earliest of these responses -- it is not clear if the response should be considered beneficial (functioning to clear necrotic cells) or detrimental (leading to an active inflammation with tissue injury). The 2006 CD notes that generally, "the initiation of inflammation is an important component of the defense process; however, its persistence and/or its repeated occurrence can result in adverse health effects." Since the threshold for even the first indications of an inflammatory response are higher than that for the reflexive FEV_1 response, no adverse health effects are to be expected at either the current 0.075 ppm standard or a more stringent standard.

Another effect that occurs at somewhat higher exposures than the FEV_1 decrements are mild symptoms and sensations of discomfort. These are also transient and reversible.

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[4] Alliance Comments, supra note 1, Appendix B, Comments of the Alliance of Automobile Manufacturers on EPA's Proposal to Revise Ozone NAAQS, filed October 9, 2007.
[5] I. Mudway and F. Kelly, *Ozone and the Lung: A Sensitive Issue*, Mol. Aspect. Med., 21, 1-48 (2000).

Thus, the inflammatory and other responses at 0.08 and higher ozone exposures when exercising do not inform the understanding of effects in the 0.06 to 0.07 ppm range. Additional relevant discussion can be found at pages 9-11 and 35-36 of Appendix B of Alliance Comments.

3. How should the results of the controlled human exposure studies at 0.060 ppm O3, showing effects on FEV1 and respiratory symptoms, in the context of the larger body of evidence from controlled human exposure studies, mentioned above, inform our understanding of the health effects to healthy adults at exposure levels from 0.060 to 0.070 ppm?

The controlled human exposure studies provide a strong and consistent body of information on the effects of 1-to-3 hour and 8-hour exposures to ozone. The first effects - transient, reversible FEV_1 decrements - are the body's reflexive reaction to the presence of an irritant gas unrelated to sensations of discomfort. Such effects occur after exposures to 0.08 to 0.12 ppm for 6 to 8 hours when the subjects are exercising at a rate that would be considered strenuous when carried out intermittently for an eight-hour period.

Whether such effects occur at 0.06 or 0.07 ppm is highly controversial since the answer depends on how the baseline is evaluated, how the precision of the test is considered, how the day-to-day variability of a subject is evaluated, and how the data is statistically analyzed. Public comments by the National Association of Manufacturers[6] and the Alliance[7] provide more detailed discussions of this issue.

The Alliance points out that the small group mean changes that are at issue are well within the range of normal measurement variability and well below the 10 to 20% changes which are considered moderate and potentially of health concern in the Staff Paper. Thus, the appropriate question in interpreting these studies is not whether there are small changes in the performance of lung function tests, but what these small changes mean for public health given the involuntary reflex mechanism causing the changes. NAM documents that a variety of environmental exposures and stresses to which many people, including children, are routinely exposed produce changes in pulmonary function tests of the same magnitude as proposed as "exposures of concern" for the ozone NAAQS. Many children experience minor exposures and stresses that result in transient FEV_1 decreases of 10-15%. Further, in some cases, these routine changes or effects occur consistently rather than infrequently, i.e. once per year.

In the 1997 review, single incidences of the FEV_1 effects at 0.08 ppm (for either healthy or asthmatic subjects) were not considered to be adverse by CASAC and EPA staff.

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[6] NAM, Attachment 2, Critical Review of the Health Data in EPA's 2008 Proposed NAAQS for Ozone Cited in the Current 2010 Proposed Rulemaking, Docket ID No. EPA–HQ–OAR–2005–0172-12439.4, at pages 1 to 7.

[7] Alliance Comments, Appendix B, at pages 9-11.

There was considerable discussion of this in the Staff Paper at that time. Nothing in the body of controlled studies has changed to alter that view. The acute decrements are transient, reversible decrements in the performance of the lung function test (that requires maximal inspiratory effort) due to a protective reflex and not a change in long-term lung function of any clinical or pathological significance.

The threshold for these first effects varies depending on ozone concentration, time of exposure and breathing rate. Since the first effects on the performance of lung function tests occur at 0.50 ppm in sedentary individuals, the vast bulk of personal exposures of either the general population or the susceptible population are far below the thresholds for the first effects identified in controlled studies. This provides a large margin of safety for the population as it goes about its daily routine.

The new data extending lung function effects to lower concentrations establishes that the response is non-linear in contrast to the linear assumption made in the 1997 review. This means that, for a given ozone exposure below the current standard, the risk of lung function decrements is now lower than thought in the 1997 review, when the standard was lowered to 0.08 ppm. The Second Draft Staff Paper in the 2008 review included a statement that the risk estimates "are now appreciably lower than in the last review."[8] The final Staff Paper removed the statement but the risk estimates remained.

Given these facts, making the ambient ozone standard more stringent with the aim of reducing the number of transient pulmonary function events and the number of "exposures of concern" will not likely have any effect on children's or adult's health.

4. With respect to the information from controlled human exposure studies at 0.060 ppm O3, what is the scientific importance of the small, group mean FEV1 decrements relative to the findings that 7 to 20% of the subjects experienced FEV1 decrements > 10%? Please consider this question from both a public health and a clinical perspective.

As discussed in the answer to question #2, small group mean FEV_1 decrements, whether or not statistically significant, are not medically significant. The EPA focus on a subset of responses in the 7 to 20 % range is also problematic since the Adams 2006 study was not designed to test for the presence or absence of high responders. EPA focuses on so-called responders but to have a very small mean difference in the presence of some larger decrements means that there are also subjects whose FEV_1 increased during the exposure. Thus, there is substantial variability in the subject's responses. For example, the range of individual FEV_1 changes in the filtered air group in these studies was -6.0 to 12.4 %.

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[8] U.S. Environmental Protection Agency, *Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper - Second Draft*, EPA-452/D-05-002, July 2006, at pages 5-71 and 6-27.

Dr. Roger McClellan, a former member and chair of CASAC, who has followed the human health effects literature for many years and Dr. Regis McFadden, a Professor of Medicine and a pulmonary physician with much research and clinical experience with asthmatics, have questioned EPA's interpretation of the data.[9] Dr. McClellan has indicated that the validity of EPA's reinterpretation of the data and the significance of the functional changes are debatable. Dr. McFadden describes the reported FEV_1 decrements in Adams' subjects when exposed to an ozone concentration of 0.08 ppm or less as "quite minimal and likely nondetectable by the subjects." Dr. McFadden does not regard such events as medically significant. He notes that the symptoms reported were barely discerned and the maximum effect (at 0.08 ppm) represented only a 6% increase over control. For example, the total mean symptom scores on the scale reported by Adams (2006) were only 2-4 units at 0.04 and 0.06 ppm out of a possible total score of 160. Adams had indicated that the differences in the symptoms between the 0.04 and 0.06 ppm exposures and the filtered air control were not significant. As the NAM Attachment 2 comments point out, EPA's conclusions concerning the variability in individual response and a higher level of response in a small subset of individuals are speculative and should not be used as a basis for a revised NAAQS.

5. The evidence, including that summarized above, indicates that susceptible populations may have greater responses than healthy people. In light of this evidence, how can we appropriately use the results of controlled human exposure studies conducted on healthy adults, as well as the epidemiological studies of susceptible groups, to inform a judgment on the effects of ozone exposure on susceptible populations?

With regard to the controlled studies, Dr. McFadden also reviewed the clinical data on whether asthmatics or other susceptible populations may have greater responses than healthy people as well as EPA's arguments as to why this might be the case. He concluded that the data at 0.12 ppm and higher do not show any major effects on asthmatics. He described EPA's arguments as to why there might be greater concern as a series of unproven assumptions. He concludes the assumptions remain an unproven theory regarding whether the responses seen in controlled studies would lead to clinical manifestations in terms of exacerbations of asthma or other adverse health effects.

Dr. McFadden provides a number of reasons for his conclusion that transient decreases in FEV, of 10-20% are not by themselves significant or meaningful to asthmatics. He points out that based on his experience from examining and studying thousands of patients for both clinical and research purposes that asthmatics typically will not begin to sense bronchoconstriction until their FEV_1 falls about 50% from normal. By way of comparison, he notes that asthmatic patients presenting to a physician or emergency room for treatment of acute exacerbations of their illness often have decreases in FEV_1 of 65-

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[9] Comments of E. R. McFadden, Jr., M.D. on Health Significance of Asthmatics' Responses Reported in Clinical Studies of Short-Term Ozone Exposure, September 14, 2007, Docket ID No. EPA–HQ–OAR–2005–0172-4054.

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70% from normal. In addition to Dr. McFadden's statement in the record, there are statements in the record by eight other Medical Doctors concerning the lack of clinical significance of transient, reversible FEV_1 decrements of 10 to 20 % for asthmatics.[10]

With regard to the epidemiological data, the Alliance Appendix B comments at pages 22 and 23 point out while there are some positive associations that implicate ozone as a responsible pollutant, taken as a whole the studies are mixed and inconsistent with regard to ozone and often implicate other pollutants. Overall, the data on susceptible populations is not any stronger than understood or assumed in the 1997 review. The mixed and inconsistent pattern is not surprising given the substantial stochastic variability in the data that is not acknowledged by the Agency. A large component of stochastic variability that goes well beyond just heterogeneity of response is apparent in the data from multi-city studies or comprehensive studies. The kinds of respiratory responses that EPA hypothesizes may occur in human populations from current ozone levels are not consistently seen in observational studies and, to the extent they are, the associations often implicate pollutants other than ozone. Therefore, there is little support for assuming that susceptible individuals are experiencing greater ozone effects than healthy individuals.

6. To what extent does your confidence that the effects observed in epidemiological studies are attributable specifically to O3 lessen or otherwise change, if at all, at the lower levels in the proposed range as compared to the higher levels?

This question is quite specific. Instead, EPA should be asking CASAC more generally about the interpretation of epidemiological studies as it informs the Administrator's decision. For example, the American Petroleum Institute offered additional questions regarding epidemiology that address key issues in the interpretation of the epidemiology - geographic heterogeneity, confounding by other pollutants and possible "double-counting" of effects, and the implications of the no-threshold assumption.[11] We urge CASAC to address all these issues.

The "effects" observed in epidemiological studies are associations not necessarily effects caused by or exacerbated by ozone. The overall pattern of associations in multi-city studies and in comprehensive studies in single cities is not biologically plausible. Analysis of time-series single-city associations in large multi-city studies such as the National Morbidity and Mortality Air Pollution Study (NMMAPS), shows significant variability of positive and "negative" associations unaccounted for in the published literature or EPA's analysis. Such a wide range that includes both strong positive and strong negative associations goes well beyond heterogeneity of response and is not biologically plausible.

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[10] A list of the statements is provided in Docket ID No. EPA–HQ–OAR–2005–0172-0052.3.
[11] January 25, 2011 letter from Howard Feldman to Assistant Administrator Regina McCarthy, Docket ID No. EPA–HQ–OAR–2005–0172-13001.1.

Air pollution time-series epidemiology studies suffer from problems associated with publication bias, model uncertainty, model selection issues, lack of adequate control for confounding variables such as other pollutants and weather, and exposure misclassification arising out of the poor correlation between ambient monitors and personal exposure. In its June 2006 letter to the Administrator, CASAC confirmed this view in evaluating mortality time-series studies, noting that "[b]ecause results of time-series studies implicate all of the criteria pollutants, findings of mortality time-series studies do not seem to allow us to confidently attribute observed effects specifically to individual pollutants."[12]

With regard to assuming that ozone associations are causal, there are a number of additional problems. Because of the wide range in individual city associations, shown for example in Figure 7-17 of the 2006 CD, combined results are usually presented. However, the combined association in winter is actually negative, so the emphasis in the literature has been on evaluating summer-only or all-year associations. It is incomprehensible that ozone would be causing mortality in the summer but protecting against mortality in the winter. The Agency has not fully evaluated all the possible reasons for this discrepancy and their policy implications. In multi-pollutant models for the summer case, the combined positive ozone association was reduced and became non-significant at all three lags evaluated as shown in Figure 7-31 of the CD.

In addition to the literature considered in the 2008 review, there is now a larger data base of multi-city studies which can inform the Administrator's decision. A full consideration of the ramifications of these studies would change the Administrator and CASAC judgments regarding an independent ozone mortality effect. Alliance and NAM comments summarize these studies.[13] In October, 2009, the Health Effects Institute (HEI) published Air Pollution and Health: A European and North American Approach (APHENA).[14] The APHENA project was designed to take advantage of the largest databases available, in the U. S. Europe, and Canada. As documented in the Alliance and NAM submissions, the pattern of combined associations reported in the APHENA study does not support EPA's claims of causal relationships between ozone and mortality or between ozone and hospital admissions.

There are at least six new multi-city studies that have evaluated ozone associations with mortality or hospital admissions since the 2006 CD. All the studies that report or plot the individual-city associations show a biologically impossible wide range of associations from positive to negative for mortality and hospital admissions. We note two relevant findings.

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[12] R. Henderson, CASAC Letter, EPA-CASAC-06-07, June 5, 2006 at page 3.
[13] Alliance Comments Appendix A and NAM Comments Attachment 1.
[14] Katsouyanni K. and Samet, J. (2009). Air Pollution and Health: A European and North American Approach (APHENA), *Health Effects Institute Report* 142, October, 2009.

When Bell et al., 2007[15] restricted the analysis to days with low ozone, in order to see if the small combined association persisted, the range in individual-community associations widened. For example, when the data was restricted to days with ozone less than 20 ppb, the range in individual city mortality associations for a 10 ppb increase in ozone was from – 20 % to + 30 %. It is inconceivable that such low ozone exposures would be causing a dramatic increase in mortality in one city and protecting against mortality in another. With such wide variation, the interpretation of a small combined association as a health effect is highly questionable.

There also is strong evidence of regional differences in ozone associations that are not consistent with ozone causing mortality. Bell and Dominici, 2008[16] and Smith, Xu, and Switzer, 2009[17] present results of combined associations broken down into the seven regions of the U.S. that were used in the NMMAPS PM studies. The combined ozone associations are significant and positive in some regions and negative or null in other regions. The spatial pattern is not consistent with ozone causing mortality. The spatial pattern, however, is similar to that for PM suggesting that ozone may be acting as an indicator of toxic PM constituents that are prevalent in some parts of the U. S. Other possible explanations also need to be considered.

Since the spatial and temporal pattern of associations in multi-city studies is not consistent with ozone causing premature mortality, the Agency should not assume that ozone has an effect on mortality at the levels under consideration.

7. EPA's exposure assessment quantified the number of all children and asthmatic children likely to be exposed to specific benchmark levels of ozone, including in particular 0.060 and 0.070 ppm. Considering the patterns of change in the estimates of exposures of concern at and above the 0.060 and 0.070 ppm benchmark levels, and the uncertainties and limitations in the estimates, what is the relative importance from a public health perspective of the estimated reductions in exposures of concern, as well as the exposures remaining, for alternative standards across the proposed range?

The exposure/exertion protocols used in the human clinical studies do not mimic typical human behavior so the results must be mapped onto realistic exposure/activity scenarios to determine the risk. The chance of experiencing an exposure of concern requires that a person be outside, at the location of the high ozone, at the time of peak ozone, and exercising heavily. During the 1996/1997 review, EPA staff undertook an extensive

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[15] Bell ML, Kim JY, Dominici F. Potential confounding of particulate matter on the short-term association between ozone and mortality in multisite time-series studies. Environ Health Perspect 2007;115:1591-5.

[16] Bell ML, Dominici F. Effect modification by community characteristics on the short-term effects of ozone exposure and mortality in 98 U.S. communities. Am J Epidemiol 2008;167:986-97.

[17] R. L. Smith, B. Xu, and P. Switzer, Reassessing the Relationship Between Ozone and Short-term Mortality in U.S. Urban Communities, Inhalation Toxicology, 29(S2), 37-61, 2009.

exposure and risk assessment with input from CASAC. To estimate the risk, staff made a series of conservative assumptions. For example, the analysis used an algorithm to assign ventilation rates based on individuals who exercised regularly and were motivated to reach a high ventilation rate. As a result, the 1996 Staff Paper acknowledged that the analysis allowed more high ventilation rates (hence greater risk) than would actually occur in the populations of interest -- outdoor workers, outdoor children, etc.

The 2008 review included an update to the exposure and risk assessment. The APEX model developed estimates of the number of all school age children and asthmatic school age children exposed one or more times a year to various 8-hour ozone concentrations (0.06 ppm, 0.07 ppm, and 0.08 ppm) while undergoing moderate exertion in a number of urban areas. These exposure metrics are discussed as benchmark "exposures of concern." The Agency noted that only a subset of the individuals in these groups would actually be expected to experience effects. To the extent that there are significant biases in the exposure and risk assessment, the interpretation of the public health protection afforded by either the current standard or various alternatives would change.

The Alliance Comments document at least five ways in which the clinical exposure and risk assessment is biased, overstating the exposures and risks that would accompany any of the alternative standards under consideration. Specifically, there are three ways that the APEX model used in the current risk assessment overestimates the number of occurrences of elevated 8-hour ozone exposures during strenuous work or play[18] and two ways in which the Risk Assessment (RA) overestimates the benefits of alternative standards.[19] Details can be found at pages 13 to 17 of Appendix B to the Alliance Comments. The American Petroleum Institute has also provided a detailed analysis that indicates that the exposure analysis overestimates the exposures of concern by at least a factor of three.[20]

Since there is substantial evidence that the exposures assessment overestimates the number of exposures of concern at any given ozone level, the estimated reductions from a more stringent standard shown in Table 3 of the January 19, 2010 proposal are also overestimated. Thus, the results of the exposure assessment overstate the impacts of a more stringent standard and should not be used until the biases are removed.

8. EPA's quantitative risk assessment estimated the numbers of occurrences of various ozone-related health effects associated with just meeting alternative standard levels down to a standard level of 0.064 ppm. Considering the patterns of change in the estimates of health effects in the risk assessment at the alternative

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[18] The model does not correct for ozone at person height compared to ozone at measurement height; the model treats the upper tail of the distribution of breathing rates incorrectly; the model predicts substantially more elevated ventilation rate occurrences than observed in real world data.
[19] The way the model rolls back ambient concentrations does not mimic the changes that will occur as emissions are reduced and man-made ozone is reduced; the background ozone assumed in the risk assessment is too low.
[20] Docket ID No. EPA–HQ–OAR–2005–0172-4141.2 at pages 27 to 29.

standard levels, and the uncertainties and limitations in the estimates, what is the relative importance from a public health perspective of the estimated reductions in risk, as well as the risk remaining, for alternative standards across the proposed range? Please consider this question in light of the scientific evidence as a whole.

The risk assessment included estimates of the lung function responses for various alternative standards as well as effects estimated from selected epidemiological associations. Each will be discussed in turn.

For the lung function responses, the exposures from the APEX model were used with dose-response functions for FEV_1 changes to estimate the number of moderate or greater lung function responses for all and asthmatic school age children. It is important to recognize, as noted above, that the lung function risk estimates are now appreciably lower than in the 1997 review when the 0.08 ppm standard was established. It is also important to recognize that the risk estimates are biased high because of the five factors referred to above that are discussed in the Alliance comments. Thus, the estimated number of lung function decrements reported in Table 2 of the January 19, 2010 proposal are substantial overestimates. In addition, incidences of exposure that might be expected to lead to decrements in the performance of lung function tests would not be discernable to the adult or child. In previous reviews of the ozone NAAQS, single incidences have not been deemed as adverse. The extreme value form of the current 8-hour standard, in itself, drastically limits the number of potential repeat exposures, providing a substantial margin of safety. Thus, for respiratory effects that we know are caused by ozone, the current standard is highly protective.

With regard to the risk estimates from epidemiology, there is substantial controversy over whether ozone is causing effects below the current standard. As noted above, the overall patterns in the ozone associations in comprehensive or multi-city studies are not biologically plausible. The Agency asserts plausibility by selecting positive associations from the literature. However, the mean personal exposures of the population are at or below the mean background, when considering the fact that people spend 90 percent of their time indoors where ozone concentrations are significantly lower than ambient. Such personal exposures do not trigger FEV_1 responses through the vagal mechanism and have not been shown to trigger inflammation. It is not clear how such low personal exposures could cause or contribute to the premature mortality or hospital admissions estimated in the risk assessment. Alliance Comments Appendix B at pages 17 to 35 and NAM comments Attachment 2 at pages 30 to 43 provide additional detail concerning the interpretation of the epidemiological results. As noted above, studies published since the 2006 CD confirm spatial and temporal patterns in the ozone mortality associations that are not consistent with causality.

Because of all the issues with the epidemiology, the Administrator should heavily discount the quantitative risk assessment using epidemiological associations. Instead, as EPA has reviewed the five criteria pollutants for which time-series epidemiology is available, by focusing on selected single-pollutant model results the Agency has claimed independent effects from four of the five pollutants. This is not scientifically defensible.

Before the

ENVIRONMENTAL PROTECTION AGENCY

Docket No. EPA–HQ–OAR–2005–0172

National Ambient Air Quality Standard for Ozone; Proposed Rule

75 Fed. Reg. 2938 (January 19, 2010)

Comments of

THE ALLIANCE OF AUTOMOBILE MANUFACTURERS

March 22, 2010

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THE ALLIANCE OF AUTOMOBILE MANUFACTURERS COMMENTS ON EPA'S PROPOSAL FOR RECONSIDERATION OF THE PRIMARY AND SECONDARY NATIONAL AMBIENT AIR QUALITY STANDARDS (NAAQS) FOR OZONE. 75 Fed. Reg. 2938 (January 19, 2010) Docket ID No. EPA–HQ–OAR–2005–0172

These comments are being submitted by the Alliance of Automobile Manufacturers (Alliance) on behalf of its member companies in response to the U.S. Environmental Protection Agency's (EPA or the Agency) Proposed Rulemaking for National Ambient Air Quality Standards for Ozone ("Reconsideration"). The Alliance is a trade association of eleven car and light truck manufacturers including BMW Group, Chrysler Group LLC, Ford Motor Company, General Motors Company, Jaguar Land Rover, Mazda, Mercedes-Benz, Mitsubishi Motors, Porsche, Toyota and Volkswagen. The Alliance's members account for over 74 percent of the new cars and trucks sold in the United States each year.

EXECUTIVE SUMMARY

The Environmental Protection Agency's (EPA's) proposal to lower the 8-hour primary ozone standard to a level within the range of 0.060-0.070 ppm and to establish a separate cumulative, seasonal secondary standard at a level within the range of 7-15 ppm-hours results from an inadequate technical review process that failed to include a full assessment of the latest science. The EPA should initiate a full and proper scientific review and issue a new proposal, in accordance with the NAAQS review process under the Clean Air Act (CAA). Because the proposed lower Ozone NAAQS is not based on a full and inclusive assessment of the latest science, the Alliance must oppose modifying the current standard of 0.075 ppm. The "provisional" review process that EPA employed during this reconsideration raises several important concerns which must be addressed before the Agency can move forward.

EPA's Provisional Assessment is a cursory examination of some of the latest science. The EPA's assessment lacks the depth of analysis that the Criteria Document (CD) or Integrated Science Assessment (ISA) would bring to a review of the science. The Provisional Assessment does not accurately reflect the latest science insofar as it overlooks several significant new scientific studies, does not thoroughly assess the studies it does summarize, and omits consideration of new information concerning a key issue in the prior review -- the background of ozone.

Studies published subsequent to the 2006 Ozone CD, were either not included in the Provisional Assessment or were overlooked by EPA. The omitted studies, highlighted in these comments and in Appendix A, question fundamental assumptions that led the Administrator to reconsider the current standard:

- The Health Effects Institute (HEI) recently published Air Pollution and Health: A European and North American Approach (APHENA)[1] study that analyzes the largest databases available on mortality and hospital admissions that may be related to ozone. This study compared multiple cities in the U.S., Canada and Europe and does not support EPA's claims of causal relationships between ozone and mortality or between ozone and hospital admissions.

- While noted in EPA's Provisional Assessment, based on new studies subsequent to the 2006 Criteria Document (CD), the pattern of results in these new multi-city studies of mortality and hospital admissions is not consistent with ozone having a causal role in mortality or morbidity below the current standard. In these multi-city studies of mortality and hospital admissions, the individual-city associations include a biologically impossible wide range of associations. In addition, the combined associations demonstrate seasonal and regional differences that are not consistent with a causal role for ozone.

- New controlled exposure studies that show no medically significant effects below 0.08 ppm.

- At least ten new studies that raise issues relevant to EPA's assumed background of ozone have been published subsequent to the 2006 CD and were not part of the EPA's Provisional Assessment. There is now new evidence that the computer model EPA relied on to evaluate policy-relevant background in the previous review substantially underestimated the contribution from soil NOx, lightning NOx, and stratospheric ozone.

INTRODUCTION

The Alliance and its members recognize the importance of ensuring clean air and setting air quality standards at a level that protects public health. While Gross Domestic Product, vehicle miles traveled, and energy consumptions have all increased, our air is cleaner today than it was a generation ago and air quality is continuing to improve.

On January 6, 2010, the U.S. Environmental Protection Agency (EPA) proposed lowering the NAAQS for ozone as a result of its reconsideration of the 2008 8-hour ozone standards. During 2008, EPA set both the "primary" and "secondary" standards at 0.075 parts per million (ppm). EPA recently proposed to lower the 8-hour primary ozone standard to a level within the range of 0.060-0.070 ppm and to establish a separate cumulative, seasonal secondary standard at a level within the range of 7-15 ppm-hours.

[1] Katsouyanni K. and Samet, J. (2009). Air Pollution and Health: A European and North American Approach (APHENA), *Health Effects Institute Report* 142, October, 2009.

EPA based this reconsideration on studies about the health and ecological effects of ozone that were assessed during the previous 2008 ozone review record. EPA conducted a "provisional" assessment of the more recent studies and found that the newer studies do not materially change the conclusions of its earlier science assessment. However, EPA should have undertaken a full assessment of these new studies.

In addition, EPA is proposing an accelerated schedule for designating areas as attainment, nonattainment, or unclassifiable for the primary ozone standard: (1) state Governors submit their recommendations by January 2011; (2) U.S. EPA promulgates final designations by July 2011; (3) designations take effect August 2011; and (3) states with nonattainment areas submit their state implementation plans describing how they will reduce pollution to meet the standards by December 2013. Depending upon the severity of the air quality problem, states would be required to meet the primary standard by 2014–2031. EPA is planning to propose an implementation rule in the spring of 2010 and issue a final rule as quickly as possible after the final ozone NAAQS which EPA plans to sign by August 31, 2010 in response to the current proposal of reconsideration. EPA is taking comment on whether to designate areas for a seasonal secondary standard on an accelerated schedule or a two-year schedule.

DETAILED COMMENTS

A. EPA Reconsiders 2008 NAAQS for Ozone

In September 2009, a year and a half after the 2008 ozone standards were finalized, EPA decided, on its own accord, to reconsider the 2008 ozone standards. On reconsideration, EPA chose not to rely on the latest scientific knowledge but instead utilized air quality criteria document (dated February 2006) that reviewed the science through 2004, with a few additional studies from 2005 that EPA found to be important.

The CCAA requires EPA to consider the most current science and consult with the Clean Air Scientific Advisory Committee's (CASAC's) scientific expertise in every NAAQS proceeding.

- Section 108(a)(2) - NAAQS must be based on Air Quality Criteria that accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health and welfare which may be expected from the presence of such pollutant in the ambient air, in varying quantities.

- Section 109(d)(2) - EPA must solicit CASAC's input in every instance in which the Administrator chooses to revise a NAAQS, which may be more frequently than at minimum five-year intervals that CAA section 109(d)(1) requires.

- Section 109(b)(1) and (2) – The statute mandates that "primary [and secondary] standards may be revised only in the same manner as promulgated."

EPA's Office of Research and Development "conducted a Provisional Assessment of the newer studies and found they do not materially change the conclusions of the Agency's earlier

science assessment."[2] The CAA, however, does not allow EPA to forego a full and complete analysis at the detailed and accurate level of an Integrated Science Assessment (ISA) or Criteria Document (CD).

B. EPA's Provisional Assessment of Newer Studies is Not an Accurate Review

EPA's Provisional Assessment cannot fulfill the CAA requirements regarding accuracy because it does not fully and completely analyze the required wide variety of health effects, inputs, welfare effects and policy-relevant background. In the Agency's own words, the review proceeded as follows:

> EPA is not relying on studies about the health and ecological effects of ozone that have been published since the science assessment was completed in 2006 to support the 2008 review.
>
> The reconsideration of parts of the 2008 final rule discussed in this notice is based on the scientific and technical record from the 2008 rulemaking, including public comments and CASAC advice and recommendations.
>
> Scientific and technical information developed since the 2006 Criteria Document will be considered in the next periodic review instead of this reconsideration rulemaking, allowing the new information to receive careful and comprehensive review by CASAC and the public before it is used as a basis in rulemaking that determines whether to revise the NAAQS.[3]

On the same Federal Register page, contrary to its statements above, the Agency asserts that its rationale for the proposed range of standard levels is based on a thorough review of the latest scientific information on human health effects associated with the presence of O_3 in the ambient air presented in the 2006 CD.

The Provisional Assessment is a cursory examination of some of the latest science. This is evidenced by the lack of analysis in the Provisional Assessment document which is only 38 pages long. This is in stark contrast to the 821 page first volume -- of three volumes in total -- of the CD analyzed during the 2006 ozone NAAQS review.

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[2] See Fact Sheet, Proposal to Revise the National Ambient Air Quality Standards for Ozone (Jan. 6, 2010) at 2; 75 Fed. Reg. at 2,944.

[3] See Fact Sheet, Proposal to Revise the National Ambient Air Quality Standards for Ozone (Jan. 6, 2010) at 2; 75 Fed. Reg. at 2,944.

Further, the Provisional Assessment lacks the depth that a CD- or ISA-level analysis would bring to a review of the science because it has not taken a fresh look at prior scientific studies. Instead, EPA is relying on the previous 2006 CD for the analysis of old science.[4]

The Provisional Assessment has not had the benefit of full review and input from CASAC, which held only one meeting -- a conference call on January 25, 2010 -- to provide any additional comments on EPA's proposed reconsideration of the ozone NAAQS. Also, the Provisional Assessment has not been made available for public comment.

Another concern is that the studies the Reconsideration Proposal relies on are now at least five years old. The 2006 CD assesses literature through 2004 along with selected "important" studies published in 2005.[5] It represents the state of science from five years ago. Since the CAA requires EPA to update the "criteria" every five years, it follows that the "reconsideration" is not based on the most accurate and latest science envisioned by that requirement.

In conclusion, the Provisional Assessment completely overlooks several significant new scientific studies, is not a thorough assessment of the studies it does summarize, and omits consideration of new information concerning a key issue in the prior review, the background levels of ozone.

Moving forward with a reconsideration based on a provisional review which omits much of the latest science directly conflicts with the goals and objectives articulated by EPA Administrator Lisa Jackson at her January 14, 2009 confirmation hearing:

> Science must be the backbone of what EPA does. The environmental and public health laws Congress has enacted direct the EPA Administrator to base decisions on the best available science. EPA's addressing of scientific decisions should reflect the expert judgment of the Agency's career scientists and independent advisors. If I am confirmed, I will administer with science as my guide. I understand the laws leave room for policymakers to make policy judgments. But if I am confirmed, political appointees will not compromise the integrity of the EPA's technical experts to advance particular regulatory outcomes.[6]

Since the lower Ozone NAAQS is not based on a full and inclusive assessment of the latest science, the Alliance must oppose the standard as currently proposed.

C. <u>Provisional Assessment Does Not Reflect the Latest Science</u>

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[4] The Alliance submitted detailed comments on the old science in 2007. See comments of the Alliance attached hereto as Appendix B and referred to as "Alliance Comments (Oct. 9, 2007)."

[5] CD at page I-iii

[6] "Statement of Lisa P. Jackson, Hearing on Nominations, Committee on Environment and Public Works, United States Senate, January 14, 2009."

Documented in Appendix A, attached and incorporated in these comments are studies published subsequent to the Ozone CD which question the fundamental assumptions that led the Administrator to reconsider the current standard. In summary:

- The Health Effects Institute (HEI) recently published Air Pollution and Health: A European and North American Approach (APHENA)[7] study. The APHENA project was designed to take advantage of the largest databases available on mortality and hospital admissions that may be related to ozone and showed inconsistent and non-robust relationships between these health effects and ozone. The APHENA study was not included in the Provisional Assessment.

- The pattern of results in other new observational studies that are noted in the Provisional Assessment but published subsequent to the 2006 CD, is not consistent with ozone having a causal role in mortality or morbidity below the current standard. In these new multi-city studies of mortality and hospital admissions, the individual-city associations include a biologically impossible wide range of associations. In addition, the combined associations demonstrate seasonal and regional differences that are not consistent with a causal role for ozone.

- There is now substantial new evidence that the computer model EPA relied on to evaluate policy-relevant background in the previous review substantially underestimated the contribution from soil NOx, lightning NOx, and stratospheric ozone. New science concerning background ozone was not included in the Provisional Assessment.

EPA is inaccurate when it asserts that "…the requirement that primary standards include an adequate margin of safety was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of the standard setting."[8] Uncertainties about background levels demand a different legal treatment than uncertainties about the health or welfare effects of man-made emissions.

CASAC itself recognized the necessity for following the regular five year process. In its April 7 2008 letter to Administrator Johnson, CASAC assumed EPA would follow the regular NAAQS review process for ozone, not a shortened reconsideration process.

> In doing so, the participating members of the CASAC Ozone Review Panel are unanimous in strongly urging you or your successor as EPA Administrator to ensure that these

[7] Katsouyanni K. and Samet, J. (2009). Air Pollution and Health: A European and North American Approach (APHENA), Health Effects Institute Report 142, October, 2009.

[8] 75 Fed. Reg. at 2,940.

> recommendations be considered during the next review cycle for the Ozone NAAQS that will begin next year[9] (*Emphasis added*)

In exercising her judgment to revise the existing 2008 Ozone NAAQS, the Administrator must provide a reasonable analysis for the change. Specifically, EPA must explain why its earlier judgment regarding what is requisite to protect public health or welfare no longer governs. Given that EPA's estimate of health risks has declined, and that the general understanding of the uncertainty surrounding the results of the health studies has increased, EPA cannot persuasively say why it must depart from its 2008 decision that the 2008 standard is requisite to protect public health.

CONCLUSION

The NAAQS review process, with its attendant Integrated Science Assessment that integrates all the latest and most accurate science and examines its results and shortcomings in detail, is the most sound scientific way to proceed. It is also the process that the CAA requires. Anything less than a full and complete analysis that includes the most current science risks is inadequate and subjects the overall process to unnecessary delays and unsound scientific results. EPA's reliance on a provisional review that is neither thorough nor scientific in its approach is contrary to the statutory requirements for NAAQS review and conflicts with the goals and objectives articulated by Administrator Jackson. The Agency should initiate a full and proper scientific review and issue a new proposal, in accordance with the CAA NAAQS review process.

[9] Henderson, R. (2008) Letter from CASAC Chairman Rogene Henderson to EPA Administrator Stephen Johnson. April 7, 2008, EPA–CASAC–08–009. Also see Ozone NAAQS Reconsideration proposal at page 2992 with regard to the primary standard and at page 3018 with regard to the secondary standard. 75 Fed. Reg. 2992, January 19, 2010.

APPENDIX A

REVIEW OF MORE RECENT STUDIES RELEVANT TO THE CHOICE OF OZONE NAAQS

This Appendix reviews recent human clinical and observational health effects studies, evaluating the extent to which they confirm or rebut the assumptions and conclusions in the EPA proposal. In addition, new studies relevant to EPA's assumptions about background ozone are reviewed.

Observational or epidemiologic studies published subsequent to the CD demonstrate a pattern of results that is not consistent or coherent with ozone below the current standard having a causal role in mortality or morbidity. Alliance comments on the July 11, 2007 Proposed Rule[10] pointed out evidence of model selection uncertainty, publication bias, confounding, and stochastic variation in the epidemiological data base that EPA relied upon to support the assumption that ozone was causing health effects below the then current standard. As discussed in the following, the new studies published since the 2006 ozone CD reinforce and support these concerns.

The APHENA study was not included in the Provisional Assessment and does not support EPA's claims of causal relationships between ozone and mortality or between ozone and hospital admissions.

In October, 2009, the Health Effects Institute (HEI) published Air Pollution and Health: A European and North American Approach (APHENA). The APHENA project was designed to take advantage of the largest databases available. These had been developed by the three groups of investigators for earlier studies: 1) the Air Pollution and Health: A European Approach Phase 2 (APHENA2) involving 32 cities; 2) the National Morbidity, Mortality, and Air Pollution Study (NMMAPS), conducted in the 90 largest U.S. cities; and 3) multicity research on the health effects of air pollution in 12 Canadian cities. Each database included air pollution monitoring data for particulate matter and ozone; health outcome data in the form of daily mortality for all ages, for persons younger than 75 years, and for persons 75 years or older (from all nonaccidental causes [all cause]); cardiovascular disease, or respiratory disease); and daily hospital admissions for persons 65 years or older (for cardiovascular and respiratory disease). Other database variables used for APHENA included weather data and a number of

[10] Comments of the Alliance of Automobile Manufacturers on EPA's Proposal to Revise National Ambient Air Quality Standards for Ozone, 72 Fed. Reg. 37,818 (July 11, 2007), dated October 9, 2007.

socioeconomic and other variables known or suspected to influence mortality or hospital admissions.

In the original studies, each of the three groups used different modeling methodologies and entered different variables into their models. Although each group found positive and significant relationships between PM_{10}/O_3 and mortality and some morbidity endpoints, the magnitude of the relationships differed by geographic region. One goal of APHENA was to use common methodologies and variables to reanalyze the data sets. The APHENA investigators intended to create a central repository for all three of the time-series databases and use a common quality assurance approach. In addition, they would conduct analyses on a combined, pooled dataset to study a variety of sensitivity issues, including effect modification. They would then investigate the sensitivity of the estimates to a variety of smoothing methods and to the number of degrees of freedom. They also intended to explore reasons for the geographical heterogeneity of the effect estimates seen in their original studies. Another important goal of the program was to understand the extent of coherence between mortality and hospitalizations using data from cities in North America and Europe.

In the original analyses, all three groups used a two-stage approach. In the first stage, risks were estimated for the individual cities; in the second stage, evidence across the cities were combined. Each group used different methods to perform both stages in the original analyses. In APHENA, the investigators wanted to identify a preferred way to do both stages and apply common methodologies to the three data sets. For the first stage, they identified two smoothing techniques, natural splines (NS) and penalized splines (PS), and decided to use a number of degrees of freedom choices. They chose to use 3, 8 and 12 degrees of freedom and also the number of degrees of freedom chosen by minimizing the partial autocorrelation function (PACF).

For the second stage analyses, the two approaches used in original NMMAPs and the European studies represented the two major approaches used at the time to pool estimates. NMMAPS used Bayesian hierarchical regression models, while the Europeans used metaregression models. However, the APHENA investigators could not determine which was the best method, so they decided to use the models interchangeably.

Using the two smoothing techniques together with the four choices for the degrees of freedom and three choices of lags (0-1 day, 1 day and distributive lags which provided the cumulative effects of days 0 through 2) for each health outcome, the investigators ran a total of 24 different models for ozone. In addition, subsets of these choices were also used to examine the effects of controlling for PM_{10} and seasonal variations.

The results showed that the differences between the PS and the NS were very small in most cases and that the number of degrees of freedom tended to give similar results when greater than 6-8 degrees of freedom where used.

The overall modeling results for the mortality and morbidity models are summarized in Table 1 and 2, respectively. The denominator in the tables is the total number of different models that were run for each health effect outcome examined and the numerator is the number of models that resulted in a positive and statistically significant relationship between ozone and

the health effect outcome. The way to interpret these tables is as follows: high ratios are suggestive of a robust and consistent relationship, while low ratios are suggestive of no significant relationship. Intermediate values of the ratio suggest inconsistent and non-robust relationships that are dependent upon the model selected. Since there is no *priori* way to determine the "correct" model, it is not possible to determine whether a small number (low ratio) significant and positive relationship represents a real causal relationship or if they are false positives that can occur by chance or by confounding.

The "all cause," "all ages" mortality results indicate a consistent relationship with ozone in Canada but somewhat less consistent relationships in Europe and the U.S. When the results for the two different age groups are examined, the interpretation of the results becomes even less clear. For ≥ 75 years of age, a consistent relationship still holds in Canada, but the European and U.S. relationships become less consistent. When compared to the results for the < 75 years of age group, the results are implausible as they suggest that ozone is affecting the younger group more than the older group, which goes against conventional wisdom. Controlling for PM makes the positive relationship for the older group disappear in all three locations, but the positive effect remains for the younger group (except in the U.S., where no relationship is evident). At all three locations a consistent summertime relationship is seen at all three locations, but vanishes in Europe and the U.S. when PM is controlled. PM controlled model results were not presented for the Canadian data. In any event, the results are not consistent with the existence of a causal relationship between ozone and "all cause" mortality.

The cardiovascular mortality/ozone modeling results are somewhat confusing. A clear positive relationship was found only in Canada and only for the ≥ 75 years of age group. Few significantly positive relationships were found for either age group for the other locations and no relationship was found in Canada for the younger age group. When PM is controlled for, few significant relationships remain. The summer-only results suggest significant relationships in Europe and the U.S., but the relationships vanish when PM is controlled. Taken altogether, these results do not support a causal relationship between ozone and cardiovascular mortality when the models are controlled for PM.

The cardiovascular hospital admissions/ozone results are also confusing. The annual results show a few significant model-dependent relationships in Canada and the U.S. but none in Europe. When PM is controlled for, a few significant, model-dependent relationships remain in Canada, disappear in the US, but become consistently significant in Europe. The European results defy logic and were dismissed by the APHENA authors as a strong positive relationship was evident for respiratory hospital admissions and PM_{10}. The summer-only results at all three locations show no significant relationships. In contrast to the cardiovascular mortality results, the respiratory mortality modeling results consistently show no relationship, with one exception. None of the annual results at any location show any significant relationship between ozone and respiratory mortality. However for the summer, consistent significant results are found, but only in Canada. Significant model-dependent results are seen in Europe and the US, but they disappear when controlled for PM. PM-controlled results for Canada were not presented. The respiratory hospital admissions show consistent significant relationships with ozone in Canada that disappears when PM is controlled. In the U.S. and Europe, a few significant, model-dependent relationships are seen that persist when PM is controlled. However, during the summer, when ozone is the highest and the strongest relationships would be expected, no

significant relationships are found in either the U.S. or in Europe. Consequently, the weight of evidence does not support a causal relationship between ozone and respiratory hospital admissions.

The APHENA results do not support EPA's claims of causal relationships between ozone and mortality or between ozone and hospital admissions. This is shown in Table 1 for mortality and in Table 2 for hospital admissions.

Table 1: APHENA modeling results for mortality

Cause of Death	Canada	Europe	United States
All Cause – all ages	24/24	15/24	12/24
≥ 75 yrs	23/24	2/24	6/24
< 75 yrs	18/24	22/24	10/24
All Cause PM controlled – all ages	4/8	8/16	0/16
≥ 75 yrs	0/8	3/16	0/16
< 75 yrs	5/8	14/16	0/16
All Cause – summer only	9/9	18/18 (4/12)*	18/18(0/12)*
Cardiovascular – ≥ 75 yrs	24/24	3/24	2/24
< 75 yrs	0/24	8/24	2/24
Cardiovascular –PM controlled ≥ 75yrs	0/8	0/16	0/16
< 75 yrs	0/8	5/16	2/16
Cardiovascular – summer only	0/6	8/12(0/8)*	11/12(0/8)*
Respiratory – all ages	0/24	0/24	0/24
≥ 75 yrs	0/24	0/24	0/24
Respiratory – PM controlled – all ages	0/8	0/16	0/16
≥ 75 yrs	0/8	0/16	0/16
Respiratory – summer only	6/6	4/12(0/8)*	2/12(0/8)*

*Denotes the PM controlled ratio

The numerators represent the number of models that showed a positive and statistically significant relationship between O_3 and mortality, while the denominator is the total number of models run.

Table 2: APHENA Modeling Results for Hospital Admissions for Patients 65 Years and Older

Type of Admission	Canada	Europe	United States
Respiratory	18/24	8/24	7/23
Respiratory – PM controlled	0/8	7/16	5/16
Respiratory – summer only	3/3	0/4	0/4
Cardiovascular	5/24	0/24	3/24
Cardiovascular – PM controlled	3/8	16/16	0/16
Cardiovascular – summer only	0/4	0/4	0/4

The numerators represent the number of models that showed a positive and statistically significant relationship between O_3 and admissions while the denominator is the total number of models run.

Based on new studies subsequent to the 2006 CD, the pattern of results in new multi-city studies of mortality and hospital admissions is not consistent with ozone having a causal role in mortality or morbidity below the current standard.

There are now a number of new multi-city studies that have evaluated ozone associations with mortality or hospital admissions since the 2006 CD. The largest of these is the APHENA study discussed above. All the studies that report or plot the individual-city associations show a biologically impossible wide range of associations from positive to negative for mortality (Bell et al., 2007[11]; Franklin and Schwartz 2008[12]; Cakmak *et al*., 2006[13]; Bell and Dominici, 2008[14]) and hospital admissions (Medina-Ramon *et al*., 2006[15]). For example, the range in individual city associations in Bell and Dominici, 2008 is from - 3 % to + 4 % per 10 ppb increase in the daily average ozone over the previous week. In the Bell *et al*., 2007 study the range in individual city results is from – 5 % to + 10 % per 10 ppb increase in the average of the current and previous day's ozone. In the Franklin and Schwartz, 2008 study the range is from – 1 % to + 3 % for a 10 ppb increase in summertime current day ozone. When Bell *et al*., 2007 restricted the

[11] Bell ML, Kim JY, Dominici F. Potential confounding of particulate matter on the short-term association between ozone and mortality in multisite time-series studies. Environ Health Perspect 2007;115:1591-5.
[12] Franklin M, Schwartz J. The Impact of Secondary Particles on the Association between Ambient Ozone and Mortality. Environ Health Perspect 2008;116:453-8.
[13] Cakmak S, Dales RE, Judek S. Do gender, education, and income modify the effect of air pollution gases on cardiac disease? J Occup Environ Med 2006b;48:89-94.
[14] Bell ML, Dominici F. Effect modification by community characteristics on the short-term effects of ozone exposure and mortality in 98 U.S. communities. Am J Epidemiol 2008;167:986-97.
[15] Medina-Ramon M, Zanobetti A, Schwartz J. The effect of ozone and PM10 on hospital admissions for pneumonia and chronic obstructive pulmonary disease: a national multicity study. Am J Epidemiol 2006;163:579-88.

analysis to days with low ozone, in order to see if the association persisted, the range in individual-community associations widened. When the data was restricted to days with ozone less than 20 ppb, the range in individual city associations was from – 20 % to + 30 %. It is inconceivable that such low ozone exposures would be causing a dramatic increase in mortality in one city and protecting against mortality in another. With such wide variation, the interpretation of a small combined association as a health effect is highly questionable.

The spatial and temporal variation in combined ozone associations with mortality is also not consistent with causality. All of the newest studies evaluated either all-year or warm period ozone associations. However, Dominici *et al*., 2005[16] showed that the combined association in winter was actually negative. When the negative association in winter was discussed in the ozone CD, it was deemed most likely to occur due to the inverse correlation of ozone and particulate matter in the winter. Thus, the likely explanation was that ozone was acting as an inverse indicator of PM. If ozone is acting as an indicator of PM in the winter, it should also be acting as an indicator of PM in the summer.

There also is strong evidence of regional differences in ozone associations that are not consistent with ozone causing mortality. Bell and Dominici, 2008 present results of combined associations broken down into the seven regions of the U.S. that were used in the NMMAPS PM studies. The combined ozone association is statistically significant in only two regions, the Northeast and Industrial Midwest. In the other five regions the combined associations are not statistically significant. In two of the regions, the Southwest and the Urban Midwest, the combined associations are actually negative. The spatial pattern is not consistent with ozone causing mortality. The spatial pattern, however, is similar to that for PM suggesting again that ozone may be acting as an indicator of the toxic PM constituents that are prevalent in the Northeast U.S. Another strong possibility, in addition to confounding by other pollutants, is that the associations represent residual confounding. In either case, the spatial and temporal variation in ozone/mortality associations is not consistent with ozone causing mortality. It is not logical that ozone would be causing harm in one season but protective in another. Also, it is not logical that ozone would be harmful in some regions of the country but protective in others. In either case, the spatial and temporal variation in ozone/mortality associations is not consistent with ozone causing mortality.

In multi-pollutant models, the combined mortality associations are not statistically significant, as shown by Dominici *et al*, 2005. A number of the recent studies evaluated potential confounding by PM, but confounding by sulfate, PM, other gases, as well as by meteorological variables all need to be fully assessed. Smith, Xu, and Switzer,[17] in a study that is not referenced in the Provisional Assessment, show that the ozone association is affected by various model selection choices. They also report substantial regional variability similar to that reported by Bell and Dominici, 2008 with some regions having combined negative associations. The ozone CD indicates that the pollutant most correlated with ozone in the summer is sulfate, especially in

[16] Dominici F, McDermott A, Daniels M, et al. Revised Analyses of the National Morbidity, Mortality, and Air Pollution Study: mortality among residents of 90 cities. J Toxicol Environ Health A 2005; 68:1071-92.
[17] R. L. Smith, B. Xu, and P. Switzer, Reassessing the Relationship Between Ozone and Short-term Mortality in U.S. Urban Communities, Inhalation Toxicology, 29(S2), 37-61, 2009.

the Eastern U. S., thus noting that the main potential confounders of interest for ozone in the summer are PM2.5 and sulfate. Indeed, Franklin and Schwartz, 2008 found that the combined ozone association was reduced substantially when sulfate was included in the model. As in the case of single-pollutant models, the range in the individual city associations in multi-pollutant models reported by Franklin and Schwartz was large (from negative to positive) in the eighteen cities they evaluated.

Additional examples of the influence of uncertainty due to model selection are now available. These include new studies of temperature and health (Keatinge and Donaldson, 2006[18]) that indicate that weather-related factors are confounding ozone associations. The ozone CD acknowledged[19] that variations in treatment of weather can change the results by a factor of two and that publication bias can inflate the perceived association by a factor of three.

With the substantial stochastic variation evident in the observational studies of mortality and hospital admissions, it is not surprising that observational studies of lung function report mixed and inconsistent results. The new studies noted in the Provisional Assessment provide many examples where the effects on respiratory morbidity that EPA assumes have not been confirmed. For example, an observational study of healthy hikers, Girardot *et al*., 2006 found no association between ozone concentrations and a decrease in lung function. The Provisional Assessment also notes three studies evaluating the association between short-term exposure to ozone and airway inflammation that failed to find an effect (Adamkiewicz *et al*., 2004; Ferdinands *et al*., 2008; and Liu *et al*., 2009). Studies of asthma exacerbations continue to report inconsistent ozone effects as well as effects with other pollutants (Schildcrout *et al*. 2006 study, in particular).

Controlled exposure studies published subsequent to the CD continue to show no medically significant effects below 0.08 ppm.

The October 2007 Alliance comments pointed out that the small changes in group mean FEV_1 (spell out) below 0.08 ppm in exercising individuals are not medically significant. They are the same order of magnitude as the normal measurement variability for an individual. The new studies noted in the Provisional Assessment do not change that view. In fact, we now know that the small, transient changes in the performance of lung function tests are caused by a reflex action as part of the body's response to an irritant gas.

Studies published subsequent to the 2006 CD raise issues relevant to the Agency's assumed background of ozone.

The estimation of background ozone in the 2007 review departed from EPA's approach in prior reviews by focusing on only mean background and by relying solely on one modeling study that used a global-scale model. This led to a major disagreement between the Agency and public reviewers. EPA's Response to Significant Comments Document noted that:

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[18] W. Keatinge and G. Donaldson, Heat acclimatization and sunshine cause false indications of mortality due to ozone, Environmental Research, 100, 387-393 (2006).
[19] CD at page 7-96.

> Several commenters questioned EPA's focus on mean background rather than the extremes of the background, believing that it resulted in EPA's failure to propose an attainable standard. Further, others mentioned that when EPA established the current primary standard for ozone, the EPA Administrator specifically rejected a standard of 0.070 ppm as being too close to background levels. The Alliance stated that reliance on one particular global model is not a scientifically sound way to estimate the contribution of uncontrollable ozone to an extreme value standard.[20]

EPA's response to these comments was that questions of implementation and attainability consequences of a NAAQS are legally irrelevant in determining which standards are requisite to protect public health and that, therefore, EPA did not consider these comments in its decision-making process.[21]

A recent review by Reid *et al*., 2008 lays out the important policy implications of the disagreement over the level of background ozone, noting:

> In their recent publications, Lefohn *et al*. (2001) and Cooper *et al*. (2005) maintain that significant inputs occur of air of stratospheric origin, bringing with it ozone concentrations of 60 ppb or higher. On the other hand, research done using a modeling approach with the GEOS- CHEM model supports background ozone concentrations that are much lower, often in the range 25 to 35 ppb. This large discrepancy has significant implications for policy development, because if the background concentration is indeed as high as 60 ppb, even if only occasionally, extremely stringent controls could be required to achieve a design value which would satisfy the standard of 80 ppb, the current U.S. value, let alone 75 ppb, the new standard.[22]

If a standard is set in the range proposed by the Administrator or recommended by CASAC, the discrepancy is even more important, with the possibility that EPA may set a standard that is unattainable even with complete elimination of all U.S. anthropogenic precursor emissions. This is clearly not a result envisioned by Congress. Therefore, it is incumbent on EPA to review the latest scientific information related to background ozone in this proposed reconsideration before the standard is made any more stringent. The Provisional Assessment

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[20] U.S. Environmental Protection Agency, Responses to Significant Comments on the 2007 Proposed Rule on the National Ambient Air Quality Standards for Ozone (RTC), March 2008, at page 166.

[21] RTC at page 166. EPA's response also noted "In addition, section 109(d) requires EPA to reevaluate the NAAQS no later than five years after completion of the last review, and determine whether new or revised NAAQS are appropriate in light of current science, as reflected in the updated air quality criteria. Issues of implementation or attainability are not a basis to delay conducting and finishing the review and making the decisions required under section 109(d)."

[22] N. Reid, D. Yap, and R Bloxam, The potential role of background ozone on current and emerging air issues: An overview, Air Qual Atmos Health (2008) 1:19–29.

omits any discussion of new science on the various sources of ozone. This is a serious omission. In the following, additional recent studies relevant to the assessment of background are noted along with a short discussion of their importance.

Jaegle, *et al*., 2005 used satellite observations of NO_2 columns, found that soil NOx emissions were twice as high as assumed in the GEOSS-CHEM model. The Alliance referenced the 2005 study in its ozone comments but EPA dismissed the Jaegle *et al*., 2005 study since it was a "new" study not evaluated in the CD. The Alliance raised this issue since it would result in the model misallocating ozone between man-made and natural sources. That study is now five years old and should be considered by EPA in its reconsideration of the O_3 standard.

Cooper *et al*., 2007[23] documents that lightning NOx is a significant source of tropospheric ozone, particularly over the Southeastern U.S. This is important because, even though the ozone is formed in the upper troposphere, it will influence surface ozone at a later time.

Hudman *et al*., 2007[24] found that the GEOS-CHEM estimates of lightning NOx emissions had to be increased by a factor of four to match recent upper tropospheric NOx measurements. This is another case in which the GEOS-CHEM version EPA relies upon will misallocate the split between natural and man-made ozone.

Koumoutsaris, *et al*., 2008[25] used the GEOS-CHEM model to evaluate the interannual variability of tropospheric ozone and CO. They report that the model reproduced the observed variability in CO well but that the model was less successful in the case of ozone. They attribute the poorer performance for ozone to the model's poor representation of stratospheric chemistry and dynamics. This is another example of the limitations of the GEOS-CHEM model that EPA relies upon.

Singh *et al*., 2007[26] evaluated four global models including GEOS-CHEM against tropospheric measurements of reactive nitrogen species. The authors show that the four models deviate from each other and from the observations at all altitudes, demonstrating that there is still substantial uncertainty in the modeling of ozone precursors that was ascribed to uncertainty in sources, meteorology, and mechanisms in the models. If the modeling of ozone precursors is

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[23] O. R. Cooper, M. Trainer, A. M. Thompson, S. J. Oltmans, D. W. Tarasick, J. C. Witte, A. Stohl, S. Eckhardt, J. Lelieveld, M. J. Newchurch, B. J. Johnson, R. W. Portmann, L. Kalnajs, M. K. Dubey, T. Leblanc, I. S. McDermid, G. Forbes, D. Wolfe, T. Carey-Smith, G. A. Morris, B. Lefer, B. Rappengluck, E. Joseph, F. Schmidlin, J. Meagher, F. C. Fehsenfeld, T. J. Keating, R. A. Van Curen, and K. Minschwaner, Evidence for a recurring eastern North America upper tropospheric ozone maximum during summer, J. Geophys. Res.,112, D23304, doi:10.1029/2007JD008710, 2007.

[24] Hudman, R. C., et al. (2007), Surface and lightning sources of nitrogen oxides over the United States: Magnitudes, chemical evolution, and outflow, J. Geophys. Res., 112, D12S05, doi:10.1029/2006JD007912.

[25] Koumoutsaris, S., I. Bey, S. Generoso, and V. Thouret (2008), Influence of El Nin˜ o – Southern Oscillation on the interannual variability of tropospheric ozone in the northern midlatitudes, J. Geophys. Res., 113, D19301, doi:10.1029/2007JD009753.

[26] Singh, H. B., et al. (2007), Reactive nitrogen distribution and partitioning in the North American troposphere and lowermost stratosphere, J. Geophys. Res., 112, D12S04, oi:10.1029/2006JD007664.

substantially uncertain, the modeling of the ozone produced from those precursors is also substantially uncertain.

Langford *et al*., 2009 shows that the stratospheric contribution to surface ozone is significant, and can lead to exceedances of the 2008 NAAQS ozone standard in a major U.S. metropolitan area. The GEOS-CHEM model application relied upon by EPA had concluded that stratospheric impacts at ground-level are never substantial. Langford *et al*. point out that the conclusions of Fiore *et al*., 2003 that EPA relies on are at odds with many other studies which have presented evidence for significant stratospheric contributions to surface ozone at both high altitude sites and near sea level. The assumption EPA makes that stratospheric intrusions, if present, can be easily identified and handled as exceptional events has not been verified.

Tarasick *et al*., 2007[27] and Lightener *et al*., 2009[28] and Terao *et al*., 2008[29] report additional evidence of stratospheric input. Tarasick *et al*. point out that while stratospheric intrusion events sometimes directly impact the surface, much more frequently, intrusion events are observed to reach the middle troposphere, where they mix in and contribute to background ozone.

The Trickl *et al*. 2009[30] study of stratospheric-tropospheric exchange using lidar is important because it documents that the available models are, still, far from being quantitative in describing the intrusion phenomena. They conclude that high spatial resolution models are needed to capture the events and that the data they collected contradicts the frequently heard statement that stratospheric-tropospheric transport has a minor influence on the tropospheric ozone budget.

Cooper *et al*., 2010[31] reports an increase in springtime background ozone mixing ratio from Asian transport that the authors conclude may hinder compliance with the U.S. ozone air quality standard. The authors note that an increase in free tropospheric ozone will also affect the surface. They also note that while summertime extreme ozone events in many U.S. urban areas have decreased, some rural and marine sites in the western U.S. show increasing ozone possibly due to increasing background ozone.

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[27] Tarasick, D. W., et al. (2007), Comparison of Canadian air quality forecast models with tropospheric ozone profile measurements above midlatitude North America during the IONS/ICARTT campaign: Evidence for stratospheric input, J. Geophys. Res., 112, D12S22, doi:10.1029/2006JD007782.

[28] Lightner, K. J., W. W. McMillan, K. J. McCann, R. M. Hoff, M. J. Newchurch, E. J. Hintsa, and C. D. Barnet (2009), Detection of a tropospheric ozone anomaly using a newly developed ozone retrieval algorithm for an up-looking infrared interferometer, J. Geophys. Res., 114, D06304, doi:10.1029/2008JD010270.

[29] Terao, Y., J. A. Logan, A. R. Douglass, and R. S. Stolarski (2008), Contribution of stratospheric ozone to the interannual variability of tropospheric ozone in the northern extratropics, J. Geophys. Res., 113, D18309, doi:10.1029/2008JD009854.

[30] T. Trickl, H. Feldmann, H.-J. Kanter, H.-E. Scheel, M. Sprenger, A. Stohl, and H. Wernli. Forecasted deep stratospheric intrusions over Central Europe: case studies and climatologies, Atmos. Chem. Phys. Discuss., 9, 2223–2288, 2009.

[31] O. R. Cooper, D. D. Parrish, A. Stohl, M. Trainer, P. Ne´de´lec, V. Thouret, J. P. Cammas, S. J. Oltmans, B. J. Johnson, D. Tarasick, T. Leblanc, I. S. McDermid, D. Jaffe, R. Gao, J. Stith, T. Ryerson, K. Aikin, T. Campos, A. Weinheimer, and M. A. Avery, Increasing springtime ozone mixing ratios in the free troposphere over western North America, Nature, 463, 344-348 (2010).

A more extensive analysis of ozone trends from 1980 to 2005 at surface sites in the U.S. by Lefohn, *et al*., 2008[32] demonstrates a pattern that is not consistent with the Agency's assumed background. Lefohn *et al*. document reductions in various ozone metrics that correspond to the former 1-hour standard, the current 8-hour standard and the proposed W126 standard.

Reductions have occurred in and near the major populated areas of the country where one would expect reductions from the nation's ozone precursor control program. However, there are also many sites with no statistically significant trends. Many of the sites with no significant trends in 8-hour ozone and W126 are located in the rural and remote West. Given the many federal programs that have reduced VOC, NOx, and CO throughout the region[33] along with the state and Federal programs that have reduced ozone in the major populated areas of the coastal states (and hence the ozone transported from those coastal states into the remote West), the lack of trend is puzzling if the background ozone is as low in the remote West as EPA assumes. The likely explanation is that the uncontrollable background of ozone (from various uncontrollable sources, such as the stratosphere, biogenic VOC and NOx, lightning NOx, and transport of anthropogenic ozone from outside the U. S.) is substantially larger than EPA assumes.

In summary, there is now substantial new evidence that the global model EPA relied on to evaluate policy-relevant background substantially underestimated the contribution from soil NOx, lightning NOx, and stratospheric ozone. In addition, the coarse horizontal resolution in the model renders it incapable of resolving the phenomena that contribute to attainment or nonattainment of an extreme value standard of the form proposed by the Administrator.

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[32] A. S. Lefohn, D. Shadwick, and S. J. Oltmans, Characterizing long-term changes in surface ozone levels in the United States (1980–2005), Atmos. Environ., 42, 8252-8262 (2008).

[33] EPA's trend data indicates that CO concentrations have been reduced 79 % nationwide between 1980 and 2008, VOC concentration, while not measured as extensively, have been reduced a comparable amount, and NO_2 concentrations have been reduced by 46 % over the same time period.

APPENDIX B

COMMENTS OF THE ALLIANCE OF AUTOMOBILE MANUFACTURERS ON EPA'S PROPOSAL TO REVISE OZONE NAAQS 72 FED. REG. 37,818 (JULY 11, 2007)

OCTOBER 9, 2007

BEFORE THE
ENVIRONMENTAL PROTECTION AGENCY

Docket NO. EPA-HQ-OAR-2005-0172

National Ambient Air Quality Standards for
Ozone; Proposed Rule

Comments of

THE ALLIANCE OF AUTOMOBILE MANUFACTURERS

October 9, 2007

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THE ALLIANCE OF AUTOMOBILE MANUFACTURERS COMMENTS ON EPA'S PROPOSAL TO REVISE NATIONAL AMBIENT AIR QUALITY STANDARDS FOR OZONE

72 Fed. Reg. 37, 818 (July 11, 2007)

These comments are being submitted by the Alliance of Automobile Manufacturers (Alliance) on behalf of its member companies in response to the U.S. Environmental Protection Agency's (EPA or the Agency) Proposed Rulemaking for National Ambient Air Quality Standards for Ozone (Proposed Rule). The Alliance is a trade association of nine car and light truck manufacturers including BMW Group, Chrysler LLC, Ford Motor Company, General Motors, Mazda, Mitsubishi Motors, Porsche, Toyota and Volkswagen. One out of every ten jobs in the United States is dependent on the automotive industry.

The Alliance members share the concerns of our customers and the American public about this nation's air quality and recognize the importance of assuring clean air that protects public health. While GDP, vehicle miles traveled, and energy consumptions have all increased, our air is cleaner today than it was a generation ago and air quality is continuing to improve. According to EPA's most recent air trends analysis, between 1970 and 2006, when gross domestic product increased 203 percent, vehicle miles traveled increased 177 percent and energy consumption increased 49 percent, total emissions of the six principal air pollutants dropped by 54 percent.[1] Investments and improvements in vehicle technology and manufacturing processes are a major part of this success story. Today's vehicles are 99 percent cleaner than vehicles of the 1970s, which has resulted in dramatic reductions in smog-forming emissions.[2]

Further, significant and additional emission reductions will occur over the next 20 years as a result of already promulgated regulations for mobile sources. According to EPA's own statistics, nitrogen oxide emissions from light duty vehicles will be reduced by over 70 percent by 2030 as the result of the Tier II Vehicle and Gasoline Sulfur Program.[3] These reductions will help to reduce ozone concentrations across the country, and the improvements to air quality as a result of these reductions will occur *regardless* of whether the existing ozone standard is revised.

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[1] U.S. Environmental Protection Agency, Comparison of Growth and Emissions, 1970-2006, *available at* http://www.epa.gov/airtrends/images/comparison70.jpg (last visited Oct. 2, 2007).

[2] Alliance of Automobile Manufacturers, Environment, *available at* http://www.autoalliance.org/environment/ (last visited Oct. 5, 2007).

[3] http://www.epa.gov/tier2/faqs.htm.

EXECUTIVE SUMMARY

Under Section 109(a) of the Clean Air Act (CAA), 42 U.S.C. § 7409(a), EPA is required to set national ambient air quality standards (NAAQS) for criteria pollutants. NAAQS include (a) a primary standard, which in the judgment of the Administrator, based on air quality criteria and allowing for an "adequate margin of safety," is "requisite" to protect the public health and (b) a secondary standard, which in the judgment of the Administrator, based on air quality criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the air pollutant.[4] EPA last reviewed and revised the ozone standard in 1997, when it promulgated the current 8-hour standard (0.08 ppm) to replace the previous, less stringent 1-hour standard (0.12 ppm) (referred to as the "1997 Review").[5]

The Alliance has carefully examined the evidence in the Ozone Criteria Document (CD), EPA's Staff Paper (SP) and in this proposed rule. The current ozone standard is protective of public health with an adequate margin of safety and protective of public welfare in accordance with Section 109. There is no basis for EPA to revise the standard at this time.

These comments focus on the reasons why EPA's scientific justification and legal grounds for lowering the existing ozone standard are unfounded and not supported by the relevant scientific body of literature and evidence produced since the Agency's 1997 Review.

1. The clinical and epidemiology evidence indicate less risk from ozone exposure than in 1997.

Although new clinical studies since 1996 have added to the available data, the fundamental understanding of effects at 0.08 ppm and above has not changed since the last review. In fact, the latest controlled human exposure studies shows there is less risk of lung function decrements and symptoms now than assumed in the 1997 Review. The data, that EPA has used to extend lung function effects to lower concentrations, establishes that the response is non-linear in contrast to the linear assumption made previously. The public health significance of these potential risks has also declined. The percent of the population undergoing exposures of concern is significantly smaller in the current modeling than originally thought in 1996/1997.[6] The Second Draft SP relied upon this evidence in concluding that the estimates of lung function effects "are now appreciably lower than in the last review"[7] to support the view that the current standard protects the public health with an adequate margin of safety. Nothing in the final SP or proposed rule contradicts this evidence. Therefore, the new clinical studies do not provide a basis for lowering the NAAQs.

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[4] 42 U.S.C. § 7409(b).

[5] 62 Fed. Reg. 38,855 (July 18, 1997).

[6] U.S. Environmental Protection Agency, *Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper - Second Draft*, EPA-452/D-05-002, July 2006, at 5-71 ("Second Draft SP").

[7] Second Draft SP at 6-27.

2. Time-series studies do not provide support for causality of effects but instead confirm significant uncertainties present in the 1997 Review.

Time-series epidemiology studies suffer from problems associated with publication bias, model uncertainty, model selection issues, lack of adequate control for confounding variables, such as other pollutants and weather, and exposure misclassification arising out of the poor correlation between ambient monitors and personal exposure. In its June 2006 letter to the Administrator, EPA's Clean Air Scientific Advisory Committee (CASAC) confirmed this view in evaluating mortality time-series studies, noting that "[b]ecause results of time-series studies implicate all of the criteria pollutants, findings of mortality time-series studies do not seem to allow us to confidently attribute observed effects specifically to individual pollutants."[8]

Analysis of time-series single-estimate results and more complete data sets, such as the National Morbidity and Mortality Air Pollution Study (NMMAPS), shows significant variability of positive and "negative" responses unaccounted for in the published literature or EPA's analysis.

3. The evidence on premature mortality is too limited in nature and should not be relied upon in basing conclusions or estimating health effects from exposure to ozone.

In its June 2006 letter to the Administrator, CASAC questioned whether the effect of individual pollutants, such as ozone, can be determined from time-series mortality studies because these studies implicate all of the criteria pollutants.[9] Furthermore, due to the significant potential for exposure misclassification, the overall utility of the time-series mortality estimates is severely limited. The results from time-series studies of 95 individual cities, shown in Figure 7-17 of the CD, are inconsistent at best, ranging from strongly negative to strongly positive.[10] Such a wide range is not biologically plausible.

4. EPA's risk estimate for mortality and certain respiratory endpoints fails to confirm an increase risk from 1997.

EPA's risk assessment fails to account for several major uncertainties, the correction of which would result in a significant reduction in the estimated risk. In estimating risk, EPA assumes causality. As discussed above, time-series studies have produced an implausibly wide range of associations from strongly positive in some cities, to no effect in some cities to strongly negative (implying protective effects) in some cities. In accordance with the recommendations of the National Academy of Sciences (NAS), the risk assessment should address this pivotal uncertainty by including a probabilistic

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[8] R. Henderson, CASAC Letter, EPA-CASAC-06-07, June 5, 2006 at 3 ("June 2006 CASAC Letter").

[9] June 2006 CASAC Letter at 3.

[10] *See* U.S. Environmental Protection Agency, *Air Quality Criteria for Ozone and Related Photochemical Oxidants*, Vol. I of III, EPA 600/R-05/004aF, Feb. 2006, Figure 7-17 at 7-90 ("CD").

analysis of the major uncertainties, including the possibility the associations may not be causal.[11] Given the magnitude of the uncertainties and EPA's failure to conduct an integrated uncertainty analysis, the current state of the scientific evidence supports retention of the current 8-hour standard.

5. The CAA does not require EPA to set standards that eliminate all risk.

EPA's obligation under the CAA is to establish standards that are "neither more nor less stringent than necessary" to protect public health.[12] This means establishing standards at a level that reduces risk sufficiently to protect public health with an adequate margin of safety. A careful review of the scientific evidence shows that lowering the current ozone standard would result in controls unnecessary to protect public health.

6. Since EPA has not provided a reasonable analysis supporting its new judgment on what is requisite to protect public health or welfare, its actions could be found to be "arbitrary and capricious."

In exercising his judgment to revise the existing NAAQS, the Administrator must provide a reasonable, scientific analysis for the change. Specifically, EPA must explain why its earlier judgment regarding what is requisite to protect public health or welfare no longer governs. To reverse its position in the face of precedent that is not persuasively distinguished is arbitrary and capricious.[13] Given that EPA's estimate of health risks has declined and new information on the public health significance of these effects suggests less cause for concern, EPA cannot persuasively say why it must depart from its 1997 decision that the current standard is requisite to protect public health.

7. EPA has not correctly evaluated the potential for further risk reduction as a result of its proposed change to the current primary standard for ozone.

EPA has taken the largely unsupportable step of substituting modeled ozone background concentrations for ambient monitoring based background. The new modeled concentrations, which suggest significantly lower concentrations of ozone background levels, suffer from several flaws, including a significant under-estimation (by a factor of 2) of NOx emissions from soils. An independent analysis shows that employing the more realistic ozone background levels used in the 1997 Review would eliminate over 95 percent of EPA's estimated risk from exposure to ozone without correcting for additional biases in EPA's risk assessment.

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[11] National Research Council, Estimating the Public Health Benefits of Proposed Air Pollution Regulations, The National Academies Press, Washington, DC, September 2002.

[12] *Whitman v. American Trucking Ass'ns, Inc.*, 531 U.S. 457, 473 (2001) (citation omitted).

[13] *Motor Vehicle Mfr.'s Ass'n v. State Farm Mut. Auto. Ins. Co.*, 463 U.S. 29, 42 (1983) (presumption "against changes in current policy that are not justified by the rulemaking record").

8. EPA has not adequately considered that in many areas of the U.S., additional reductions beyond those needed to meet the current primary standard for ozone, may not be achievable with known control technology and could impose ruinous economic consequences.

The situation in several nonattainment areas is direr than implied by EPA in its proposed rule. Several nonattainment areas have recently submitted State Implementation Plans (SIPs) that fail to demonstrate attainment with the current standard, even when considering all currently known control technologies. Although the Administrator is not allowed to consider the costs of attaining the standard, it is clear that the CAA was not intended to impose ruinous conditions on industry or the American public. In some areas, the near elimination of all stationary and mobile source emissions would be required to meet the current standard. Moreover, in making a policy judgment on what is requisite to protect public health, the Administrator should consider the adverse impacts on health and quality of life from such stringent measures, particularly on the low income populations.

9. CASAC's conclusion that the current primary standard for ozone is unjustified and is based on unsupportable assumptions.

Based on the information presented to CASAC from the GEOS-CHEM model, CASAC was led to believe that ozone background levels never exceed 0.040 ppm. As a result, CASAC incorrectly assumed that a standard as low as 0.060 ppm is attainable and that, therefore, exposures of concern could be essentially eliminated. CASAC also erred in evaluating the risk of clinical effects. Although the Second Draft SP showed that the risk of lung function effects is lower than thought in 1997, the current CASAC panel appeared to believe that the APEX model used in the risk assessment for the clinical effects systematically under-predicted measured values.[14] In actuality, the model significantly overestimated the number of exposures of concern as detailed elsewhere in these comments.

10. EPA's estimate of non-mortality health benefits, while small, still overestimates the potential benefit of meeting a lower primary standard

The benefits of a 0.070 ppm standard (or any of the alternatives proposed by EPA) depend on highly questionable assumptions concerning mortality causality. EPA's Regulatory Impact Analysis (RIA) acknowledges that the direct benefits from ozone reduction itself are $190 million per year for the 0.070 ppm alternative if it is assumed that ozone does not cause premature mortality.[15] Given EPA's estimate of the morbidity effects of ozone in the RIA are highly exaggerated due to model selection issues and other factors discussed in these comments, all the alternative standards below the current

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[14] *See, e.g.,* R. Henderson, CASAC Letter, EPA-CASAC-07-001, October 26, 2006, at 11.

[15] U.S. Environmental Protection Agency, *Regulatory Impact Analysis of the Proposed Revisions to the National Ambient Air Quality Standards for Ground-Level Ozone*, EPA-452/R-07-008, July 2007, at 6-2 ("RIA").

standard are likely to achieve very little if any additional health benefits, even though achievement of such standards will require a massive investment and dramatic change in the quality of life for Americans.

11. A careful review of the existing scientific evidence demonstrates that the existing secondary standard for ozone is requisite to protect public welfare.

EPA's recommendation to use a new seasonal secondary ozone standard does not address the uncertainties and limitations associated with the use of such a standard. Limitations include: (1) the lack of ozone monitoring data at vegetation height in rural areas; (2) the likelihood that extreme ozone background levels overlap with the ranges recommended for the secondary standard; and (3) the inability of the seasonal metric to account for the variable uptake of ozone by plants. The new data developed since 1997 does little to address these important uncertainties.

A new secondary standard also raises significant implementation issues. For instance, emission inventories (particularly for agricultural emissions) and modeling tools needed to implement a seasonal standard, particularly in the Western United States, are not currently available. Establishing a secondary standard without these tools would impose an unreasonable implementation burden on states and local communities.

DETAILED COMMENTS

Introduction

Section 109 of the Clean Air Act requires the Administrator to set a primary national ambient air quality standard (NAAQS), the attainment and maintenance of which in the judgment of the Administrator are requisite to protect the public health with an adequate margin of safety. EPA's obligation under the CAA is to establish standards that are "neither more nor less stringent than necessary" to protect public health. This means establishing standards at a level that reduces risk sufficiently to protect public health with an adequate margin of safety. Lowering the current ozone standard would result in controls unnecessary to protect public health.

A careful review of the scientific evidence shows that EPA's understanding of the potential risks from exposure to ozone at the current standard remains largely the same as in 1997, and that the actual risk from exposure to levels below the standard, based on clinical evidence, may now be lower than what was assessed in 1997. In addition, it is clear from the Second Draft of EPA's Staff Paper that the percent of the population undergoing exposures of concern is significantly smaller in the current modeling than originally thought in 1997. As EPA states in the preamble, its task under Section 109 is to "establish standards that are neither more nor less stringent than necessary for these purposes."[16] Lowering the standard in light of the 1997 determination would result in controls that would be unnecessary to protect public health.

Since EPA has not provided a reasonable scientific analysis supporting its new judgment on what is requisite to protect public health or welfare, its actions could be found to be "arbitrary and capricious." In exercising his judgment to revise the existing NAAQS, the Administrator must provide a reasonable, scientific analysis for the change. Specifically, EPA must explain why its earlier judgment regarding what is requisite to protect public health or welfare no longer governs. To reverse its position in the face of precedent that is not persuasively distinguished is arbitrary and capricious.

The weight of the science supports retention of the current ozone standard as requisite to protect public health and welfare. There is nothing in the science that deviates from EPA's 1997 analysis, and EPA failed to adequately explain its change of position. A careful review of the scientific studies EPA relies on to justify a lower standard illustrates that EPA's current estimates of risk are inflated and unreliable. EPA's own Regulatory Impact Analysis (RIA) shows that many areas of the country today are struggling to meet the current standard and that many more areas will be unable to attain a tighter standard with known technology. Requiring areas to meet a more stringent standard would impose "ruinous" conditions on the American public contrary to the intentions of the CAA.

[16] 72 Fed. Reg. 37,818, 37,820 (July 11, 2007).

As these comments will show, the state of the science indicates that the current standard is requisite to protect public health and welfare, and that EPA has not provided a reasonable explanation to deviate from its finding in 1997.

I. Comments on EPA's Proposal to Revise the Primary Standard for Ozone.

EPA proposes to revise the primary NAAQS for ozone by reducing the current 8-hour standard of 0.08 ppm to a level within the range of 0.070 to 0.075 ppm. In addition, EPA seeks comments on alternative levels, down to 0.060 ppm, and up to retaining the current standard.[17]

An exhaustive review of the administrative record and the underlying studies on which the proposed revisions are based shows that a revision of the current 8-hour ozone NAAQS cannot be justified. Any tightening of the current standard would not meet the statutory test -- that the "attainment and maintenance of [such standard], … allowing an adequate margin of safety, are requisite to protect the public health." CAA at §109(b)(1). To be "requisite to protect the public health," the standard must be "not lower or higher than is necessary." *Whitman v. American Trucking Associations*, 531 U.S. 457, 476 (2001). As the Administrator found in 1997, the current standard meets that test. While there have been many additional studies since that time, as demonstrated in the detailed comments below, those studies do not show that the risk from ozone is any greater than was recognized in the previous rulemaking, and the level of uncertainty over the existence of those risks has increased. EPA should retain the existing primary ambient NAAQS for ozone.

A. The Clinical and Epidemiology Evidence Indicate Less Risk from Ozone Exposure than in 1997.

1. The risk of respiratory effects is substantially reduced from what was estimated in the 1997 Review.

EPA is correct in concluding that controlled human exposure studies offer the best information on ozone effects. Although there are a wide range of epidemiological studies that report associations of ozone with health endpoints, the proposed rule notes that the epidemiological studies are not themselves direct evidence of a causal link between exposure to ozone and the occurrence of the effects.[18]

A careful examination of the clinical data (including the new data) shows that there is less risk of lung function decrements and symptoms than was assumed in the 1997 review. Key findings from the clinical data include the following:

The data extending lung function effects to lower concentrations establishes that the response is non-linear in contrast to the linear assumption made in the previous review. The preamble notes that the logistic form provides a very good fit to the combined data set used in

[17] 72 Fed.Reg. at 37,818 (July 11, 2007).
[18] 72 Fed. Reg. at 37,879.

the current Risk Assessment.[19] Although the final Staff Paper (SP) does not show a direct comparison between the dose-response assumed in 1996 and the current dose-response, the Second Draft SP did include such a comparison in Figure 5-2 a., b., and c. The Figure clearly shows that the response rates for FEV_1 decrements >10%, >15%, and >20% are lower at all concentrations below the current standard with the logistic form.[20] This means that, for a given ozone exposure below the current standard, the risk of lung function decrements is now lower than thought in the previous review.

Although there are many differences between the 1996 and 2007 clinical risk assessment methodologies, it is clear from the Second Draft SP that the percent of the population undergoing exposures of concern is significantly smaller in the current modeling than originally thought in 1996/97. The Second Draft SP, which was the first draft that included staff's recommendations for the level of the standard, used the fact that the estimates of lung function effects "are now appreciably lower than in the last review" to support the view that the current standard protects the public health with an adequate margin of safety.

Finally, although respiratory symptoms in healthy children were estimated in the 1996 review, that endpoint was not included in the current review since a number of field studies failed to find such effects.

2. The clinical data do not support the reversal of EPA's 1997 determination that the current standard is requisite to protect the public health with an adequate margin of safety.

EPA's conclusions from the 1997 Review are unaltered by more recent clinical studies and, thus, do not support a revised determination that the current 8-hour standard is no longer sufficient to protect public health with an adequate margin of safety.

The proposed rule notes that, in controlled human exposures, the first effects on the performance of lung function tests occur at 0.50 ppm in sedentary individuals. EPA focuses on FEV_1, the volume of air expired in the first second during maximal expiratory effort. With increased levels of physical activity and breathing rate, the threshold for the first effects decreases. However, the thresholds for the first effects in controlled studies are still considerably above the vast bulk of personal exposures of the general population. During the 1997 Review, EPA staff concluded that the lowest range within which 1- to 3-hour exposures to ozone at heavy exertion produce group mean statistically significant lung function decrements is 0.12 to 0.16 ppm.[21] They concluded that the lowest range for 6- to 8-hour exposures at moderate exertion is 0.08 to 0.12 ppm.[22] The exercise utilized in the 6- to 8-hour controlled exposure studies was intended to simulate work performed during a day of heavy to severe manual labor by outdoor

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[19] 72 Fed. Reg. at 37,858.
[20] Second Draft SP, Figure 5-2 a., b., c. at 5-17. col. 2.
[21] U.S. Environmental Protection Agency, *Review of National Ambient Air Quality Standards for Ozone Assessment of Scientific and Technical Information, OAQPS Staff Paper*, EPA-452/R-96-007, June 1996, at 49-150. ("1996 Staff Paper")
[22] 1996 Staff Paper at 150.

laborers.[23] Although the ventilation rate (40 L/min) is described by EPA as "moderate exercise," activity maintained at this level for 6 to 8 hours has to be considered "heavy" or "strenuous work or play." The staff conclusions regarding lowest effect levels for various respiratory endpoints in the current review are unchanged from the previous review with one exception, which is discussed below.

More importantly, in addition to establishing the presence or absence of physiological changes, the question of the public health significance of any measured changes needs to be addressed. In the previous review, there was considerable discussion of this question and a framework for evaluating the adversity of reported changes was developed with one set of criteria for healthy individuals and another for individuals with compromised respiratory systems. The two frameworks for evaluating adversity in the previous review are included, unchanged, in the current SP.

The Criteria Document (CD) acknowledges that there is a neurally-mediated mechanism that is responsible for the lung function decrements and symptoms seen in the clinical studies. A body of studies now shows that the first effects of ozone are caused by reflex responses of vagal nerve receptors. This response is the body's reflexive reaction to the presence of an irritant gas and is unrelated to sensations of discomfort. Such a risk would be considered both safe and tolerable. The CD acknowledges that these effects are transient, of a reflex nature, and reversible.[24]

In the previous review, CASAC and staff determined that single, acute moderate lung function responses in healthy individuals should not be considered adverse. For asthmatics, staff previously concluded, based on discussions with medical experts, that single ozone exposures that resulted in moderate responses were not likely to interfere with normal activity nor would it result in increased frequency of medication or the use of additional medications. There is no scientific basis for changing these conclusions in the current review. In fact, in the proposed rule, the Administrator concurs noting that "[w]hile some experts would judge single occurrences of moderate responses to be a 'nuisance,' especially for healthy individuals, a more general consensus view of the adversity of such moderate responses emerges as the frequency of occurrence increases."[25] The concern over multiple occurrences arises not because of established effects but because of the potential for it leading to adverse consequences. However, there has been little or no discussion in the current review of the number of occurrences that should be considered adverse or of the public health significance of multiple occurrences, given the reflex mechanism that is responsible for the transient FEV_1 changes.

Although new clinical studies since 1996 have added to the available data, the fundamental understanding of effects at 0.08 ppm and above has not changed since the last review. In 1996, EPA concluded that a wide array of health effects were attributed to short-term

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[23] W. McDonnell, H. Kehrl, S. Abdul-Salaam, P. Ives, L. Folinsbee, R. Devlin, J. O'Neil and D. Horstman, *Respiratory response of humans exposed to low levels of ozone for 6.6 hours*, Arch. Environ. Health, 46, 145-150 (1991).

[24] CD at 6-44 to 6-45.

[25] 72 Fed. Reg. at 37,850.

(1 to 3 hrs.), prolonged (6 to 8 hrs.) and long-term exposure to ozone at levels as low as 0.08 ppm. The 1997 Review found acute health effects induced by short-term exposures to ozone concentrations as low as 0.12 ppm, occur generally while subjects are engaged in heavy exercise. These effects include pulmonary function responses, transient respiratory symptoms, effects on exercise performance, increased airway responsiveness, increased susceptibility to respiratory infection and transient pulmonary inflammation. Similar health effects in the 1997 Review were observed following prolonged exposures to ozone at concentrations as low as 0.08 ppm and at lower levels of exercise.[26] EPA noted that some of the studies reporting effects at 0.08 ppm included Follinsbee *et al.* (1991), 1994, Gong *et al.* (1986), Hazucha *et al.* (1992), Horstman *et al.* (1990, 1995), Horvath *et al.* (1991), Koren *et al.* (1988,1989 and 1991), Lin *et al.* (1994) and McDonnell *et al.* (1991).

However, new data (Adams 2006) extended the database on lung function effects down to 0.06 ppm and 0.04 ppm for 6 to 8 hours with moderate exercise.[27] The interpretation of the latest data is controversial. In the Second Draft SP, EPA indicated that the lowest ozone effect level for pulmonary function decrements and increased respiratory symptoms was 0.08 ppm for 6.6 hours,[28] but in the final SP, this was lowered to 0.06 ppm for group comparisons[29] and 0.04 ppm for individuals.[30] The change occurred for several reasons. First, EPA adjusted the Adams 2006 study's results based on the assumption that the performance on lung function tests generally improves after exposure to the filtered air control. In addition, EPA also re-analyzed the data with a different statistical test. With these changes EPA drew completely different conclusions from those of the study's author, with regard to possible statistical significance of group mean results. The author of the original study has subsequently objected to the conclusions of this re-analysis. EPA also inappropriately relied on the results in individual subjects in drawing their conclusions.

It is important to note that the group mean decrement, even with the adjustment for the filtered air response was less than 3 percent, which is within the 3 to 5 percent range of normal measurement variability for an individual and substantially less than the 10 to 20 percent range discussed as adverse lung function decrements. For example, the range of individual FEV_1 changes in the filtered air group in these studies was -6.0 to 12.4 percent. In fact, decrements of 3 percent or less are noted in the SP as having no interference with normal activity for either healthy persons or persons with impaired respiratory systems. Given that the observed effects from the Adams 2006 study are not medically significant, the debate over whether the study demonstrates a statistically significant effect at specific ozone exposure levels is not meaningful. For this reason, the Administrator cannot rely on the Adams 2006 study as evidence to support a

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[26] 1996 Staff Paper at 24; 62 Fed. Reg. at 38,859.

[27] W. Adams, *Comparison of chamber 6.6 h exposures to 0.04-0.08 ppm ozone via square-wave and triangular profiles on pulmonary responses*, Inhalation Toxicology, 18, 127-136 (2006).

[28] Second Draft SP, Table 3-4, at 3-78.

[29] U.S. Environmental Protection Agency, *Review of National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper*, EPA-452/R-07-007, July 2007, Table 3-4, at 3-84 ("SP").

[30] SP at 3-1.

conclusion of significant public health effects below the current standard when the effects observed are not considered medically significant.

3. EPA's risk assessment based on the clinical studies does not support the conclusion that a tightening of the standard is requisite to protect the public health.

EPA's interpretation of the risks based on clinical studies is flawed and does not support the conclusion that a more stringent standard is needed.

The exposure/exertion protocols used in the human clinical studies do not mimic typical human behavior so the results must be mapped onto realistic exposure/activity scenarios to determine the risk. The chance of experiencing an exposure of concern requires that a person be outside at the location of the high ozone, at the time of peak ozone and exercising heavily. During the 1996/1997 review, EPA staff undertook an extensive exposure and risk assessment with input from CASAC. To estimate the risk, staff used the output of a probabilistic exposure model together with concentration-response functions derived from the clinical studies. The conservative analysis, documented in the 1996 Staff Paper, assumed lung function decrements started at 0.04 ppm, assumed no attenuation of effects, assumed that children experienced the same level of symptoms as adults (available data suggests otherwise), ignored smoking status that would attenuate responses in outdoor workers, and used human time/activity and exertion data from California as well as other locations. The EPA analysis also used an algorithm to assign ventilation rates based on individuals who exercised regularly and were motivated to reach a high ventilation rate. As a result, the 1996 Staff Paper acknowledged[31] that the analysis allowed more high ventilation rates (hence greater risk) than would actually occur in the populations of interest -- outdoor workers, outdoor children, *etc.*

The 1996 exposure and risk assessment demonstrated that there will be a minimal number of days of ozone-related respiratory effects with 8-hour concentrations up to 0.09 ppm and employing statistical forms allowing up to five exceedances per year. The risk analysis based on human clinical studies was one of the major factors in the Administrator's choice of the current ozone standard. While the primary standard is required to allow for an adequate margin of safety, the extreme value form of the current 8-hour standard, in itself, adds a substantial margin of safety in that it drastically limits the number of potential repeat exposures.

The current review includes an update to the exposure and risk assessment. The APEX model developed estimates of the number of all school age children and asthmatic school age children exposed one or more times a year to various 8-hour ozone concentrations (0.06 ppm, 0.07 ppm, and 0.08 ppm) while undergoing moderate exertion in a number of urban areas. These exposure metrics are discussed as benchmark "exposures of concern." The proposal notes that only a subset of the individuals in these groups would actually be expected to experience effects.[32] The benchmark metrics were evaluated for both "as-is" ozone concentrations for the

[31] 1996 Staff Paper at 102.
[32] 72 Fed. Reg. at 37,855.

years 2002 to 2004 and then for the ozone concentrations estimated to occur upon meeting alternative standards. The exposures from the APEX model were then used with the dose-response functions for FEV_1 changes to estimate the number of moderate or greater lung function responses in the same groups for both the "as-is" air quality and that estimated upon attainment of various alternative standards.

The proposed rule notes that the Administrator considered the number of persons exposed at or above the 0.07 ppm benchmark and the clinical risk estimates along with other factors in evaluating the adequacy of the current standard.[33] In fact, the proposed rule notes specifically that the Administrator focused on the risks remaining upon just meeting the current standard in considering the adequacy of that standard.[34] In addition, the Administrator relied significantly on the exposure and risk assessment results in choosing the range of 0.070 to 0.075 ppm for the proposed revised primary standard. To the extent that there are significant biases in the exposure and risk assessment, the interpretation of the public health protection afforded by either the current standard or various alternatives would change.

There are at least five ways in which the clinical exposure and risk assessment is biased, overstating the exposures and risks that would accompany any of the alternative standards under consideration. Specifically, there are three ways that the APEX model used in the current risk assessment overestimates the number of occurrences of elevated 8-hour ozone exposures during strenuous work or play and two ways in which the Risk Assessment (RA) overestimates the benefits of alternative standards. The five areas are:

1. The CD acknowledges that ozone exposure is lower at "person" height compared to "measurement" height (3-15 meters).[35] For example, Wisbeth *et al.*[36] measured the increment between ozone at 2 and 10 meters and reported an average 13 percent difference. In addition to the height differential, ozone monitors are also sited in open areas removed from sources so as to capture the highest ozone concentrations expected in an area. Since downwind sites are usually the design value sites, they will dominate the upper tail of the ozone distribution and yet may not reflect the overall outdoor exposures in the vicinity of the site. If people spend time outdoors in closer proximity to streets or in areas with more surface area (buildings, *etc.*) to quench ozone, their exposures will be below that measured at the monitor. The APEX model assumes that whatever ozone is interpolated from the monitor measurement is the actual ozone exposure in the outdoors-other microenvironment. The Langstaff Memorandum acknowledges the issue of vertical variation in ozone but indicates that the Agency does not plan to address it due to a lack of data.[37] This difference was corrected in the vegetation risk assessment but not in the

[33] 72 Fed. Reg. at 37,871.
[34] 72 Fed. Reg. at 37,871.
[35] *See* CD at 3-15, AX3-190-91.
[36] A. Wisbeth, G. Meiners, T. Johnson, and W. Ollison, *Effect of monitor probe height on measured ozone concentration*, Paper No. 96-RA111.02, presented at the 89th Annual Meeting of the Air & Waste Management Association, Nashville, TN, June 1996.
[37] J. Langstaff, Technical Memorandum, *Analysis of Uncertainty in Ozone Population Exposure Modeling*, Jan. 2007, at 21-22 ("Langstaff Memorandum") .

human risk assessment. In the vegetation risk, the metric summing concentrations of 0.06 ppm and higher was halved with a 10 percent vertical correction. By analogy, a vertical correction in the human risk assessment would likely halve the number of human exposures of concern at ground level. Because this effect would correct a bias in the exposure calculations, it is particularly important to evaluate and include it in the risk assessment before the final rule.

2. The upper tail of the distribution of breathing rates -- equivalent ventilation rates (EVR) -- is particularly important because it is a critical factor in determining the number of exposures of concern. EPA assumes that all exposures between 13 and 27 EVR respond as though they were at 20 EVR, substantially overestimating the risk since there are many more 8-hour occurrences of ozone at 13 EVR than at 20 and many more at 20 EVR than at 27. This results in an overestimation of the number of exposures of concern. The 1996 CD showed that 1- to 2-hour ozone responses vary with EVR and Ollison has shown that the 8-hour responses in the Folinsbee/Horstman data do too.[38] Thus, the dose-response curve varies with EVR. The distribution of exposures also varies with EVR. We expect many more 8-hour occurrences of ozone exposures at 13 EVR than at 20 and many more at 20 EVR than at 27. Thus, the approximation that all exposures between 13 and 27 EVR respond as though they were at 20 EVR substantially overestimates the risk.

Since the model calculates EVR to a much finer scale, the distribution of EVR should be presented in the risk assessment and dose-response curves should be developed as a function of EVR to avoid the overestimation of risk. At a minimum, this issue should be evaluated in a sensitivity analysis. The Langstaff Memorandum indicates that the EVR cutpoints input to APEX are selected for the risk calculations and are not considered as uncertain for the purposes of the uncertainty analysis.[39] However, it would be simple to break the wide EVR range used for the risk calculations into a number of smaller bins to test the hypothesis that the use of such a wide EVR range biases the overall result. In addition, the American Petroleum Institute (API) has provided EPA with a dynamic statistical model that can be implemented in APEX and provide more highly refined FEV_1 decrements.[40] The sensitivity of APEX predictions to the use of the API model should be evaluated prior to the issuance of the final rule.

3. The model predicts more elevated ventilation rate occurrences than observed in real world data. Langstaff acknowledged that the "values produced by the ventilation rate algorithm may exhibit an excessive degree of variability."[41] The final sensitivity analysis for APEX includes a comparison of predicted ventilation rates with mean values in the literature, but the upper tails of the distribution which impact the risk estimates were not

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[38] American Petroleum Institute Comments on EPA's Dec. 13, 1996 Proposed Ozone Rule, Appendix A.

[39] Draft Langstaff Memorandum at 43.

[40] Memorandum from Ted Johnson, TRJ Environmental, to John Langstaff and Harvey Richmond, U.S. EPA, *An algorithm for APEX that estimates one-minute ozone-induced FEV_1 decrements*, (July 31, 2006) (included as attachment to Memorandum from Will Ollison, API Energy, to Karen Martin, U.S. EPA (Aug. 29, 2006) (EPA-HQ-OAR-2005-0172-0037.1)).

[41] Draft Langstaff Memorandum at 42.

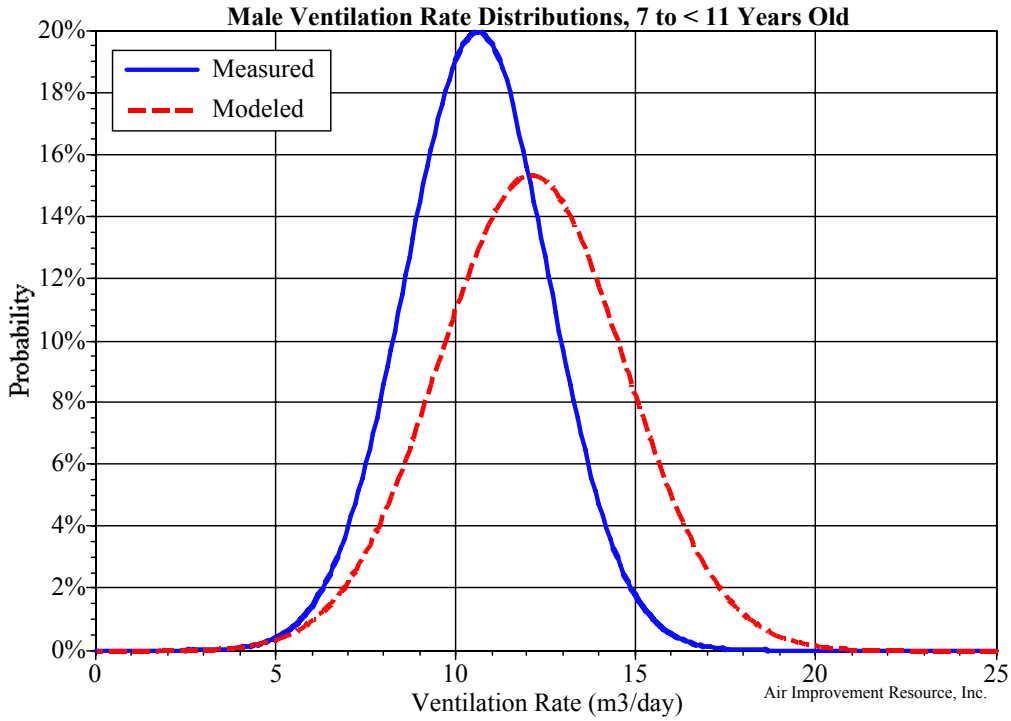
compared.[42] This is an important oversight because the upper percentiles of ventilation rate are responsible for the exposures that cause the perceived risk. In the comparison of the APEX modeled values with the measured ventilation rates from Brochu *et al.* 2006,[43] the model over-predicted mean daily ventilation rates for persons below age 11 and over age 40. More importantly, the model had a much higher standard deviation at all ages. This suggests that the upper percentiles of ventilation rates in the model are substantially above those measured in a database of over 30,000 person-days from a cohort of over 2200 free-living individuals between the ages of 3 and 96. This is particularly important since the comparison was not available to CASAC before the panel provided its recommendations. A detailed analysis of the upper tails of the distribution of predicted and measured ventilation rates must be carried out before the final rule is issued.

The following Figure 1 shows that the APEX model EPA uses in the risk assessment significantly overestimates the breathing rates of male children, particularly for the upper tails of the distribution that are responsible for the exposures of concern discussed in the proposed rule. The data underlying these distributions (means and standard deviations) come from Table 25 in the 2007 Langstaff Memorandum on uncertainty in the exposure model. In fact, of the 16 comparisons in Table 25, for eight age groupings each of males and females, 15 had substantially higher modeled ventilation rates compared to the data reported by Brochu, *et al.* 2006 at the upper end of the distribution.

[42] J. Langstaff, Technical Memorandum, *Analysis of Uncertainty in Ozone Population Exposure Modeling*, Jan. 31, 2007 at 52 ("2007 Langstaff Memorandum") (EPA-HQ-OAR-2005-0172-0174).

[43] *See* P. Brochu, J. Ducre-Robitaille, and J. Brodeur, *Physiological daily inhalation rates for free-living individuals aged 2.6 months to 96 years based on doubly labeled water measurements: comparison with time-activity-ventilation and metabolic energy conversion estimates*, Int. J. Hum. Ecol. Risk. Asses., 12, 736-761 (2006).

Figure 1: Comparison of Measured *vs.* Modeled Daily Ventilation Rates for 7- to 10-Year Old Boys



4. The EPA risk assessment uses the current distribution of ozone concentrations as a baseline input. To estimate risk associated with a given standard, the model "rolls back" ozone levels to the given standard. There are several problems with the rollback methodology EPA used. It is arbitrary and does not mimic the changes that will occur as emissions are reduced and man-made ozone is reduced. Importantly, EPA ignores background when rolling back the distribution of ozone concentrations at individual sites.

Currently, NOx emissions suppress ozone a significant portion of the time *via* the NO + O_3 reaction. As ozone precursor emissions are reduced, there is a reduction in the highest daily 8-hour ozone concentrations but lower ozone concentrations actually increase. The CD notes this behavior (*see* CD at 3-32 to 3-35) referring to Figures 3-17 and 3-18 which show the year-to-year trends in the distribution of mean daily 8-hour averages and the 95th percentile of daily 8-hour averages across the country. The text indicates that the highest ozone concentrations decreased over the past 15 years, but there has been little change in the middle of the distribution and a slight increase in ozone concentrations near the bottom of the distribution because of reduced titration of ozone with NO in response to reductions of NOx emissions. EPA's rollback misses the increase in ozone concentrations near the bottom of the distribution as the man-made influence on ozone is decreased. EPA's rollback overestimates the change in ozone at mid- and low

concentrations and, thereby, overestimates the benefits of alternative standards. The rollback should be applied to the man-made portion of ozone not the entire distribution.

5. The background ozone assumed in the risk assessment is too low. Since the risk assessment subtracts the risk from background, a higher background will result in a lower risk from attaining the current standard. As demonstrated elsewhere in these comments, the Agency's approach to estimating background is flawed. As in previous ozone reviews, both the mean and extremes of background need to be evaluated in order to properly account for the ozone that is not controllable through control of U.S. man-made emissions.

Corrections to the exposure and risk assessment for these five major biases should substantially increase the health benefits and reduce the potential risk associated with attaining the current standard. The corrected risk assessment would confirm that the current standard is protective of public health in accordance with the requirements of Section 109 of the CAA.

Even with significant overestimates of the number of exposures associated with exercise, the APEX results presented in the SP demonstrate that the current standard limits the number of persons and person-occurrences of ozone exposures to 0.08, 0.07, and 0.06 ppm for 8-hours with moderate exertion (strenuous work or play). Correcting major biases would significantly reduce the estimates further and confirm the protectiveness of the current standard and the unlikelihood of achieving additional public health benefits from lowering the standard.

B. Time-Series Studies Do Not Provide Support for Causality of Effects But Instead Confirm Significant Uncertainties Present in the 1997 Review.

1. In interpreting the epidemiological evidence, EPA downplays the major new findings concerning uncertainty due to model selection issues.

Model selection uncertainty relates to confounding of air pollutant associations by temporal trends, weather and co-pollutants. EPA acknowledges that the uncertainties in the estimates of ozone effects (or those of any other pollutant) are understated by consideration of the statistical uncertainty of the fitted model alone. Much more uncertainty arises from the lack of information regarding the choice of appropriate models for adjusting confounding by other co-variates, and the choice of appropriate lag structures. As Lumley and Sheppard (2003) point out:

> Estimation of very weak associations in the presence of measurement error and strong confounding is inherently challenging. In this situation, prudent epidemiologists should recognize that residual bias can dominate their results. Because the possible mechanisms of action and their latencies are uncertain, the biologically correct models are unknown. This model selection problem is

> exacerbated by the common practice of screening multiple analyses and then selectively reporting only a few important results.[44]

Others have also pointed out the critical importance of model choice, particularly when effect estimates are small. For example, Smith *et al.* caution:

> From a statistical point of view, the common epidemiological practice of choosing variables (including lagged variables, co-pollutants, etc.) that maximize the resulting effect estimates is a dangerous approach to model selection, particularly when the effect estimates are close to 0 (i.e., RR close to 1).[45]

Smith *et al.* note that Lumley and Sheppard (2000)[46] showed that the effect of choosing lags in this fashion has a bias which is of the same order of magnitude as the relative risk being estimated.

The revised analyses necessitated by the problems with the commonly used software for time-series analyses clearly show that methods used for controlling temporal trends and weather can profoundly affect the results. To make matters worse, there appears to be no objective statistical test to determine whether these factors have been adequately controlled. The Health Effects Institute Expert Panel[47] for the re-analysis states, "Ritov and Bickel (1990)[48] have shown, however, that for any continuous variable, no strictly data-based (*i.e.,* statistical) method can exist by which to choose a sufficient number of degrees of freedom to insure that the amount of residual confounding due to that variable is small. This means that no matter what statistical method one uses to select the degrees of freedom, it is always logically possible that even if the true effect of pollution is null, the estimated effect is far from null due to confounding bias." The expert panel concluded further, "Neither the appropriate degree of control for time, nor the appropriate specification of the effects of weather, has been determined for time-series analyses". In other words, it is impossible to adjust temporal trends without accurate information from external sources regarding the appropriate degrees of freedom to use. Such information simply does not exist.

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[44] T. Lumley and L. Sheppard, *Time-series analyses of air pollution and health: straining at gnats and swallowing camels?* Epidemiology, 14, 13-14, 2003.

[45] R. Smith, P. Guttorp, L. Sheppard, T. Lumley and N. Ishikawa, *Comments on the Criteria Document for Particulate Matter Air Pollution*, Northwest Research Center for Statistics and the Environment Technical Report Series No. 66, July 2001.

[46] T. Lumley and L. Sheppard, *Assessing seasonal confounding and model selection bias in air pollution epidemiology using positive and negative control analyses*, Environmetrics, 11, 705-717 (2000).

[47] Health Effects Institute, Special Report: *Revised Analyses of Time-Series Studies of Air Pollution and Health*, Health Effects Institute, Cambridge, Massachusetts, at 267, 269 (2003).

[48] Y. Ritov and P. Bickel, *Achieving information bounds in non- and semi-parametric models*, Ann. Stat., 18, 925-938 (1990).

With regard to uncertainty due to model selection, the Koop and Tole 2004[49] conclude:

> Point estimates of the effect of numerous air pollutants all tend to be positive, albeit small. However, when model uncertainty is accounted for in the analysis, measures of uncertainty associated with these point estimates became very large. Indeed they became so large that the hypothesis that air pollution has no effect on mortality is not implausible. On the basis of these results, we recommend against the use of point estimates from time-series data to set regulatory standards for air pollution exposure.

EPA has failed to adequately account for the significance of model uncertainty in either its qualitative or quantitative assessment of the health effects from exposure to ozone. Given that the small positive results from time-series studies may reflect residual bias of the models due to weather, temporal or other unaccounted confounding factors, EPA cannot and should not draw conclusions on causality from these studies.

2. In interpreting the epidemiological studies, EPA relies on single estimate and single-site analyses, and fails to address the effect of publication bias.

The SP and proposed rule discuss three new meta-analyses of ozone mortality associations and point out that they come to similar conclusions. When these studies were published, they were accompanied by two commentaries referenced in the CD but not the SP or proposed rule. The commentary by Goodman is particularly insightful.[50] It notes that the implications of the EPA-sponsored exercise of funding three separate meta-analyses "go far beyond the question of the ozone mortality effect." He cautions that "depending on published single-estimate, single-site analyses are an invitation to bias." He notes that "the most plausible explanation is the one suggested by the authors, that investigators tend to report, if not believe, the analysis that produces the strongest signal; and in each single-site analysis, there are innumerable model choices that affect the estimated strength of that signal." Although the proposed rule mentions the issue of publication bias, it is discounted by asserting that ozone-mortality associations remained after accounting for the bias. However, the current methods for estimating and correcting publication bias are only partial and incomplete. For example, the current method of plotting the effect size *versus* the standard error (referred to as a funnel plot) and using the trim and fill method to provide a symmetrical plot, resulted in the order of a 10 percent reduction in effect size in the Ito *et al*.[51] meta-analysis, but Bell *et al.*[52] and Goodman noted factors of three or more reduction in effect size comparing the published single-city studies with the comprehensive NMMAPS data set. The reasons for the discrepancy is likely the result

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[49] G. Koop and L. Tole, *Measuring the Health Effects of Air Pollution: to What Extent Can We Really Say that People are Dying from Bad Air*, J. of Environmental Economics and Management, 47, 30-54. (2004).

[50] S. Goodman, *The Methodologic Ozone Effect*, Epidemiology, 16: 430-435 (2005).

[51] K. Ito, S. de Leon, and M. Lippmann, *Associations between ozone and daily mortality*, Epidemiology, 16: 446-457 (2005).

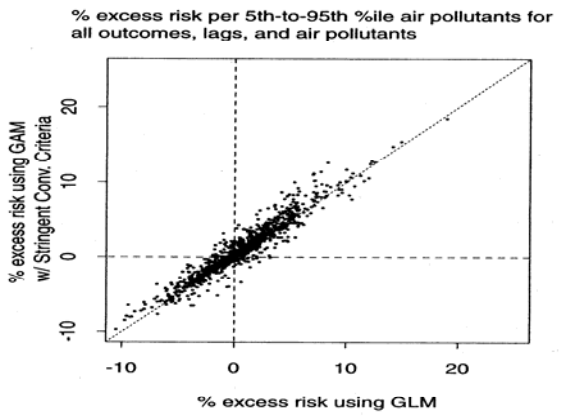
[52] M. Bell, F. Dominici, and J. Samet, *A meta-analysis of time series of ozone and mortality with comparison to the National Morbidity, Mortality, and Air Pollution Study*, Epidemiology, 16: 434-445 (2005).

of many non-random model selection choices in the conduct of the study in addition to the non-random choice of whether to publish.

3. Analysis of more complete data sets, such as NMMAPS, suggests significant variability of positive and "negative" responses unaccounted for in the published literature or in EPA's analysis.

While there are some inverse or negative air pollution associations reported in the literature (implying an unlikely protective effect from exposure to ozone), the NMMAPS study shows that there are many more "negative" associations in the data than in the literature. When the statistical issues with the General Additive Model (GAM) were raised, Ito[53] systematically re-analyzed the 1220 separate air pollution mortality and morbidity associations that were included in the original Lippmann *et al.* 2000 study of Detroit, comparing the results using the General Linear Model (GLM) to those with the suspect GAM. As shown in Figure 2, there was a wide range of negative and positive excess risks (associations) in Detroit when a large number of pollutants, lags and morbidity and mortality endpoints were considered. All the combinations of pollutant, lag and health outcome evaluated in the original Lippmann study were considered plausible candidates for air pollution health effects. Ito showed in separate figures that the wide range of associations occurred for each pollutant, including ozone. Although the focus in the original Lippmann study, as it is in almost all the published literature, was on the positive associations, Ito's plot shows that there are many negative associations in the data.

Figure 2: Comparison of Generalized Additive Model With Generalized Linear Model[54]



[53] K. Ito, *Associations of Particulate Matter Components with Daily Mortality and Morbidity in Detroit, Michigan*, in Health Effects Institute, Special Report: Revised Analyses of Time-Series Studies of Air Pollution and Health, May 5, 2003, at 143-156.
[54] *Id.*

Although there may be somewhat more positive associations than negative associations, there is so much noise or variability in the data that identifying which positive associations may be real health effects and which are not is beyond the capability of current methods. Thus, time-series epidemiology of air pollution associations is a very blunt tool. CASAC raised this issue in a June 2006 letter to the Administrator, noting that "because results of time-series studies implicate all of the criteria pollutants, findings of mortality time-series studies do not seem to allow us to confidently attribute observed effects specifically to individual pollutants."[55] Further, due mainly to exposure misclassification concerns, they questioned the utility of the time-series mortality estimates.

If all positive associations (or excess risks) are assumed or concluded to be health effects then all negative associations should be considered health benefits due to ozone or other air pollutants. Since no one believes the observed negative associations are proof that air pollutants actually provide health benefits on a day-to-day basis, the assumption that all positive associations are health effects is also not plausible. For example, the various ozone/mortality associations in 95 U.S. cities shown in Figure 7-17 of the CD at 7-90 (ranging from - 6 to + 10% per 0.02 ppm increase in 24-hr. average ozone in the previous week) would imply that ozone is dangerous in about a third of the cities, but is harmless or even beneficial in about half the cities. This is not biologically possible, if it is assumed that these associations represent causal relationships.

Instead of focusing only on and relying on the studies in the literature reporting positive associations, the Agency should address questions such as how there can be such a wide variation of ozone associations both positive and negative in the data. It is not biologically plausible that ozone would be causing mortality in some cities in the summer and protecting against mortality in others. Similarly, it is not plausible that ozone is, on balance, dangerous in the summer and protective in the winter.

4. EPA has a duty under the Data Quality Act to consider all the results, including both positive and non-positive results.

The 2000 Data Quality Act requires federal agencies to issue information quality guidelines ensuring the quality, utility, objectivity and integrity of information that they disseminate and provide mechanisms for affected persons to correct such information.[56] According to federal guidelines objectivity involves both the presentation and substance of information. In order for information to be considered objective, however, it must be presented in an accurate, clear, complete and unbiased manner, although data subjected to formal, independent, external peer review is presumed to be of acceptable objectivity, the presumption is rebuttable.[57] EPA's analysis of the epidemiology does not meet the standard of objectivity required under the 2000 Data Quality Act because it ignores the inherent variability of the data by focusing on only the positive results.

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[55] June 2006 CASAC Letter at 3.

[56] 44 U.S.C. § 3501 *et seq.*

[57] *See Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility, and Integrity of Information Disseminated by Federal Agencies*; Republication, 67 Fed. Reg. 8452, 8459 (Feb. 22, 2002).

5. The new data on asthmatics and symptoms/medication use does not provide clear and convincing evidence of an increase in risk due to ozone exposure that was not present in the 1997 Review.

EPA incorrectly concludes that the new evidence on asthmatics suggests an increased risk compared to that assumed in the previous review:

> The Administrator also notes that clinical studies, supported by epidemiological studies, provide important new evidence that people with asthma are likely to experience larger and more serious effects than healthier people from exposure to O_3.[58]

The discussion of clinical studies in Section 6.3.2 of the CD indicates that there are some studies that report slightly higher functional responses in asthmatics compared to normal subjects, some studies that report similar responses, and one new study that reports lesser responses in asthmatics. In the 1996 review, the Agency considered this issue and developed separate guidelines for evaluating the adversity of respiratory effects for healthy individuals and those with impaired respiratory systems. The same guidelines are included in the SP. Therefore, there is no important new information from clinical studies that changes the interpretation of risk for asthmatics relative to healthy individuals compared to that in the previous review. However, the overall risk for both groups is lower than thought in 1996 due to the non-linear nature of the pulmonary function responses noted above.

The SP refers to a number of panel studies that report associations of ozone with daily symptoms/asthma medication use. The SP and CD stress the multi-city study of Mortimer *et al.* 2002 as providing a sample of asthmatics that is most representative of the U.S. The SP cites the CD as indicating that the panel studies as a group indicate a positive association between ambient concentrations and respiratory symptoms, and increased medication use in asthmatics.[59] The evidence is characterized as much stronger than in the previous review.

The reliance on single-pollutant results from Mortimer *et al.* 2002, however, is highly misleading. The Mortimer study presents the results of single- and multi-pollutant models. Because the various possible confounders were not all measured in the eight cities, the results are shown for various subsets of the data. Only SO_2 was measured in all eight cities, and when SO_2 was included in two pollutant models, the ozone coefficient was reduced from 1.16 to 1.11 and became non-significant. For seven areas both SO_2 and NO_2 were available. In multi-pollutant models the ozone coefficient was reduced to 1.06 and again was non-significant. In three cities PM_{10} was also available, and in those areas the ozone coefficient was reduced to 1.04 in a 2-pollutant model with PM_{10} and to 1.00 in a multi-pollutant model with all four pollutants. As the authors indicate, only SO_2 remained significant in the seven urban areas with complete data for the three gases. Overall, each of the four pollutants was associated with symptoms in single-pollutant models and the strongest association was with NO_2 in single-pollutant models and with

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[58] 72 Fed. Reg. at 37,878.
[59] SP at 3-10.

SO_2 in multi-pollutant models. In fact, the authors of the study specifically mention that they focus on ozone as a good marker of summer air pollution and refer to the study's finding as effects of summertime air pollution rather than ozone, *per se*.

A new study by Schildcrout *et al.*[60] evaluated the relation between various pollutants and asthma exacerbations (daily symptoms and use of rescue inhalers) among 990 children in eight U.S. cities (including Boston) and reported associations with several pollutants, but ozone was unrelated to exacerbations. When the new Schildcrout *et al.* study is considered along with the nine other studies that are described in the CD as having limited or a lack of evidence for ozone effects, the overall findings relative to effects of ozone on asthmatics on symptoms and medication use should be described as mixed and inconsistent rather than clear and very strong evidence of an ozone effect.

In addition to man-made air pollutants, confounding by bioaerosols may also be a possible explanation for some of the variation in results for asthmatics. Some bioaerosols are known to exacerbate asthma but are generally not considered or included in air pollution studies. During the 1996 review, EPA staff included moderate or severe pain on deep inspiration in the exposure and risk assessment. However, the current CD correctly notes that there is no consistent evidence of an association between ozone and respiratory symptoms in healthy children. Therefore, the Agency does not include this health outcome in the current risk assessment. For asthmatics, however, the current risk assessment does include an estimate of respiratory symptoms in Boston using associations reported by Gent *et al.* 2003.

Given the Schildcrout *et al*. study, the lack of adequate control for other pollutants (man-made or biogenic), and the overall inconsistent results in the epidemiology data, EPA cannot conclude that the health risk to asthmatics from exposure to ozone is greater than the risk observed in 1997.

6. The claim that a more stringent ozone standard will address the growing asthma problem in the U.S. is without scientific merit.

a. There is no evidence that ozone levels at or below the current standard cause asthma.

Although there has been an increase in asthma in both developed and developing nations in the past 20 or 30 years, outdoor air pollution, in general (and ozone, in particular), has been decreasing. Outdoor air pollution cannot be the cause of the increase in asthma. Changes in living conditions in the indoor environment due to energy conservation may play a role by increasing exposure to common household allergens such as mold, dust mites and animal dander. A recent Annapolis Center Report[61] concludes that certain allergens and irritating chemicals, as

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[60] J. Schildcrout, L. Sheppard, T. Lumley, J. Slaughter, J. Koenig and G. Shapiro, *Ambient air pollution and asthma exacerbations in children: An eight-city analysis*, Am. J. Epidemiology, advance access published June 23, 2006.

[61] Asthma and Air Quality, A Report of the Annapolis Center for Science-Based Public Policy, Annapolis, MD, 2004.

well as passive smoking can cause asthma, but that there is insufficient evidence to determine whether many common indoor or outdoor air pollutants cause asthma.

b. The epidemiology data do not show that ozone levels at or below the current standard cause asthma.

Dockery *et al.*[62] evaluated asthma prevalence and respiratory symptoms in a group of 13,369 8 to 12 year old children in 24 communities and reported that the prevalence of asthma or asthmatic symptoms was not associated with chronic exposure to ozone. McConnell *et al.*[63] followed 3,535 children with no history of asthma from 12 communities in Southern California. The risk for developing asthma was not greater in high ozone communities compared to low ozone communities. However, in a subset of children, those playing three or more sports, there was an increased risk of developing asthma in the high ozone communities but not in the low ozone communities. Because the number of children in the subset was very low, it is not clear if this is a true effect of ozone combined with high exercise levels. If it is true, the effect is limited to a subset of the population and associated with ozone levels in Southern California in the mid-1990s – levels that were substantially *above* the current standard.

There are many different environmental stimuli that can trigger or aggravate an asthma attack, including weather, changes in weather, bioaerosols and indoor allergens. The Institute of Medicine has concluded that various indoor pollutants, primarily biologic agents, and some chemical agents cause exacerbation of asthma.[64] For traditional outdoor air pollutants, the Annapolis Center concludes the situation is not as clear because the data are not as consistent as for the major indoor triggers. Although ozone is a candidate for exacerbating asthma, the epidemiologic literature is not consistent and implicates all the major outdoor pollutants to some degree or another.

C. The Evidence on Premature Mortality is too Limited in Nature and Should Not Be Relied Upon in Basing Conclusions on the Health Effects from Exposure to Ozone.

In the 1996 review, there was limited evidence of an ozone association with daily mortality. The 1996 CD concluded that an association has been suggested, but that the strength of any such association remained unclear at that time. Since then, there has been considerable study of the association of daily mortality with ozone and several other major pollutants. The current SP and the proposed rule place significant emphasis on the question of ozone mortality effects, especially from the results of multi-city analyses.

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[62] D. Dockery, J. Cunningham, A. Damokosh, L. Neas, J. Spengler, P. Koutrakis, J. Ware, M. Raizenne and F. Speizer, *Health Effects of Acid Aerosols on North American Children: Respiratory Symptoms*, Environmental Health Perspectives, 104: 500-505, 1996.

[63] R. McConnell, K. Berhane, F. Gilliland, S. London, T. Islam, W. Gauderman, E. Avol, H. Margolis and J. Peters, *Asthma in exercising children exposed to ozone: a cohort study*, The Lancet, 359: 386-391, 2002.

[64] Institute of Medicine, *Clearing the Air: Asthma and Indoor Air Exposures*, 2000.

1. CASAC itself had reservations on the use of the data.

Because of all the issues with the publication bias, model uncertainty, confounding, *etc.* discussed above, time-series epidemiology of air pollution associations is a very blunt tool. CASAC raised this issue in a June 2006 letter to the Administrator noting that "[b]ecause results of time-series studies implicate all of the criteria pollutants, findings of mortality time-series studies do not seem to allow us to confidently attribute observed effects specifically to individual pollutants."[65] Further, due mainly to measurement error issues, they questioned the likelihood of ozone itself causing mortality and noted the limitation that measurement error obscures thresholds in time-series studies, adding additional concerns about the utility of the time-series mortality estimates.[66]

2. A new NMMAPS analysis that extends the previous analyses, and the Huang *et al.* (2005) analysis of a subset of 19 large U.S. cities is limited, and does not support a finding of causality.

a. There are major deficiencies in the Bell et al. 2004 paper, including the omission of the winter ozone results and limited analyses regarding other pollutants.

There are major deficiencies in the new NMMAPS analysis, Bell *et al.* 2004, that omitted winter ozone results and limited the analyses regarding other pollutants. If the positive combined association of ozone with mortality in the summer is causal, it implies that ozone levels well within background are linked to premature mortality. Since the ozone exposures of the frail population (since they spend their time indoors) will be roughly half or less than that measured at central monitors, the paper implies that levels well below background (average exposures of 0.01 to 0.015 ppm) are causing significant mortality. This is not biologically plausible. Bell *et al*. argue that the pattern of association they observe would be anticipated for ozone since ozone produces acute inflammatory responses in the lung. However, the doses of ozone that result in initial inflammatory changes are considerably elevated compared to the doses the frail population experiences on a day-to-day basis.

In addition, while Bell *et al.* focus on the combined associations, the results from the 95 individual cities, shown in Figure 7-17 at 7-90 of the CD, range from strongly negative to strongly positive (-6 to + 10% increase in mortality per 0.02 ppm increase in 24-hr. average ozone in the previous week). Such a wide range is not biologically plausible.

Finally, Bell *et al.* (2004) provided adjustment for the potential confounding by PM_{10}. However, Bell *et al.* did not have the necessary air quality data to provide similar and adequate adjustment for the potential confounding effects of fine PM, which EPA has concluded also causes mortality. Rather, Bell *et al.* only provided adjustment for the long-term average of $PM_{2.5}$. It is well known that ozone is not highly correlated with PM_{10}. Rather, ozone levels,

[65] June 2006 CASAC Letter at 3.
[66] CASAC Letter, EPA-CASAC-06-07, June 5, 2006, at 3 and 4.

particularly in summer, are highly correlated with $PM_{2.5}$. Thus, it was not possible for Bell *et al.* to conclude that the acute ozone mortality signal they observed was due to ozone exposure alone.

b. The pattern of individual-city associations in the Huang et al. study is not consistent with a causal association, and may reflect the confounding role of another pollutant.

The largest multi-city study, the National Morbidity and Mortality Air Pollution Study (NMMAPS), evaluated air pollutant-mortality relations in the 90 largest U.S. cities. In 2002, statistical issues with the GAM commonly used in such time-series studies were raised and many time series studies (particularly particulate matter studies) were re-analyzed. The SP thus refers to the original NMMAPS analysis and the NMMAPS re-analysis, but focuses on a new NMMAPS analysis[67] that extends the previous analyses and the Huang *et al.* 2005 analysis of a subset of 19 large U.S. cities.

In the original NMMAPS analysis and in the re-analysis in single-pollutant models, ozone had a small positive combined association in summer, but also a small negative combined association in winter.[68] In multi-pollutant models for the summer case, the combined positive ozone association was reduced and became non-significant at all three lags evaluated as shown in Figure 7-31 of the CD. Ozone is not the only pollutant for which NMMAPS seasonal results have been published to date; a seasonal PM_{10} analysis has also been carried out.[69] Peng *et al.* used updated mortality data from 1987-2000 in 100 cities and found that summer was the only season for which the combined PM_{10} effect was statistically significant.

In the Huang *et al.* study of 19 cities, the pattern of individual-city associations is not consistent with a causal association. For example, the range in individual-city associations is from –6 to + 16% per 0.02 ppm increase in 24-hour ozone and lower ozone levels in the cities were associated with higher mortality associations.[70] Also, Huang *et al.* actually reported that inclusion of PM_{10} markedly reduced the ozone mortality coefficients, in many cases to non-statistically significant levels. The SP also discusses the Gryparis *et al.* 2004 23-city study and the Schwartz 2005 14-city study. Both studies also reported a very wide range of individual city associations ranging from negative to positive. Schwartz notes the negative association in winter and suggests that it may reflect the negative association between wintertime ozone and primary air pollutants. Schwartz goes on then to ask "might not the positive association in the summer likewise reflect confounding with some other pollutant?" These and other possible explanations need to be rigorously considered by EPA.

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[67] M. Bell, *et al.*(2004), at 2372-2378.
[68] F. Dominici, *et al.* (2005), at 1071-1092.
[69] R. Peng *et al.* (2005), at 585-594.
[70] CD at 7-104.

3. While the SP downplays confounding by co-pollutants, a more comprehensive review of the data implicates other pollutants as potential causes of the reported mortality associations in time-series studies.

The SP continues to downplay confounding by co-pollutants resulting in the conclusion that single-pollutant model results should be the baseline for the quantitative risk assessment. However, several major findings from the Stieb *et al.* 2002, 2003 meta-analyses (cited in the CD) show that confounding by co-pollutants remains a major problem. Stieb *et al.* evaluated mortality associations for each of the major pollutants and found a very similar distribution of associations for each in their analysis of 109 separate mortality/time-series studies. For example, the patterns in Figures 1 to 5 of Stieb *et al.* 2002 are remarkably similar to one another. They also reported a similar pattern of associations with various categories of death and stronger associations in the warm season than in the cold season for <u>all</u> the pollutants. When multi-pollutant models were compared to single-pollutant models, the associations were reduced in the case of each pollutant. In the case of ozone, the combined association became non-significant in multi-pollutant models. Since various investigators included different pollutants as potential confounders and different modeling strategies, the multi-pollutant analyses available in the literature are not a systematic evaluation of the issue. Nevertheless, the relative risk reported in single-pollutant model results was reduced substantially for each pollutant when multiple-pollutant models were used. This suggests that the effect observed may not be due to ozone but rather from exposure to other pollutants, such as $PM_{2.5}$, that are highly associated with ozone exposures.

The analysis of the NMMAPS data and findings of the Stieb *et al.* studies, and the results of the HEI re-analysis, implicate all the pollutants as potential causes and/or confounders of excess mortality in time-series studies, and show that stronger associations in the summer are not restricted to ozone. Since the Stieb *et al.* meta-analyses like the other meta-analyses are subject to publication bias; the associations reported are likely to overstate the true associations. Nevertheless, the pattern of results should not be affected by publication bias the way the magnitude is.

The NMMAPS database is valuable because, by including all the largest U.S. cities with data, it avoids the issue of publication bias. On the other hand, it is questionable to use the same approach to adjust for meteorological factors in cities with widely differing weather conditions (*e.g.,* Los Angeles *versus* Chicago). The wide range of ozone associations in the data, using the same methodology, demonstrates the inherent noise or variability in the data. The same wide pattern of mortality associations ranging from strongly positive to strongly negative is observed in the individual NMMAPS data for all the pollutants studied (PM_{10}, ozone, CO, NO_2, and SO_2) at all the lags studied.[71]

[71] J. M. Heuss and J. J. Vostal, *Comments on the Fourth External Review Draft of Air Quality Criteria for Particulate Matter*, EPA 600/P-99/002aD, June 2003, prepared for General Motors Corporation, Aug. 28, 2003.

In the HEI re-analysis, in single-pollutant models, ozone had a small positive combined association in summer, but also a small negative combined association in winter. In multi-pollutant models for the summer case, the combined positive ozone association was reduced and was not significant at all three lags evaluated.

The SP and CD dismiss concern over confounding by gases since the peaks of the various pollutants occur in different seasons and because ozone is generally not highly correlated with other pollutants. This is unwarranted since the Stieb *et al.* meta-analysis shows that the mortality associations for these potential confounders are strongest in the warm period. The cause or causes of these seasonal patterns are not clear. It could be a seasonally varying bias that is yet unidentified. It could be related to toxic constituents (gases or particles) that have a summer maximum. Since it occurs for each pollutant, confounding by as yet not understood weather effects is a likely candidate. As discussed in the next section, many air pollution analysts dismiss weather effects but the substantial seasonal trends in the health data are caused by subtle effects related to changes in weather and human behavior. Therefore, confounding by weather is a candidate.

4. Weather factors remain a key confounder in the mortality analysis relied on by EPA.

A recent paper by Keatinge and Donaldson[72] provides important new insights into the issue of modeling weather effects in ozone studies. They evaluated whether mortality that is often attributed to ozone and other pollutants in hot weather results from confounding by neglected weather factors. Their analysis was restricted to days when the mean daily air temperatures exceeded 18 degrees°C in Greater London from 1991 to 2002, and evaluated mortality counts at an age greater or equal to 65. The adjustment for acclimatization was based on the characteristic pattern that has been reported by various investigators that the rise in mortality on hot days is followed by a prolonged reduction in mortality lasting at least 14 days. When only current temperature (average of days 0 to –2) was considered in the model, significant mortality was attributed to ozone. When they allowed for cumulative exposure to heat throughout the summer and for sunshine (which contributes to heat stress at any given temperature), the ozone association was reduced by a factor of 10 and was no longer statistically significant. This study indicates that previously neglected weather factors may be confounding the mortality analyses relied on in the SP. It is noted in the CD that variations in treatment of weather can change the results by a factor of 2 and that publication bias can inflate the perceived association by a factor of 3.[73] The Keatinge and Donaldson analysis suggests that previously overlooked weather factors can reduce the association by a factor of 10. Thus, the fact that the uncertainty due to model selection is much larger than the typical confidence limits on any given statistical association should be highlighted by EPA and considered in the interpretation of the risk assessment.

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[72] W. Keatinge and G. Donaldson, *Heat acclimatization and sunshine cause false indications of mortality due to ozone*, Environmental Research, 100, 387-393 (2006).

[73] CD at 7-96.

D. EPA's Risk Estimate for Mortality and Certain Respiratory Endpoints Fail to Confirm an Increase in Risk.

There are five significant sources of uncertainty in EPA's risk assessments for mortality and respiratory health effects:

- *EPA failed to show that the associations between ozone and health endpoints are causal. The risk assessment, therefore, should include a probabilistic consideration that the associations may not be causal.*

In contrast to the acute respiratory effects in clinical studies where we know the observed effects are due to the exposure of the pollutant being assessed, the first and main issue in interpreting the epidemiologic associations is determining the cause of the observed effects. Chapter 5 of the Second Draft SP acknowledged such noting "the most important uncertainty is the extent to which the associations between O_3 and the health endpoints included in the assessment actually reflect causal relationships."[74] (The final SP acknowledges the five sources of uncertainty as "major" at pages 5-42 and 5-43) Although there clearly are positive associations in the literature for these health endpoints, the overall pattern of associations is not biologically plausible. In multi-city studies, there is an implausibly wide range of associations for mortality from strongly positive in some cities, to no effect in some cities to strongly negative (implying protective effects) in some cities. The full range for several multi-city studies is shown below in Table 1.

Table 1

Range of Ozone Individual-City Single-Pollutant Mortality Associations in Multi-City Studies Per Standard Ozone Increment as Defined in the CD (subject to model selection uncertainty, but not publication bias)

NMMAPS II	(warm season)	-6% to + 6%
Gryparis *et al.*	(warm season)	-8% to + 8%
Gryparis *et al.*	(year-round)	-12% to + 6%
Bell *et al.* 2004	(warm season)	not reported, but large as shown in CD Figure 7-17
Huang *et al.* 2005	(warm season)	-3% to + 8%

With this range of heterogeneity, the practice of using Bayesian techniques to shrink the city-specific estimates towards the overall mean is highly questionable. The individual-city estimates are dramatically pulled towards the mean and this masks the heterogeneity, especially when the heterogeneity includes both no effect and protective effects.

As noted above, the SP fails to fully acknowledge that the body of studies since 1996 demonstrates major uncertainties and issues related to model selection and publication bias.

[74] Second Draft SP at 5-76.

When the full range of studies in the CD is viewed, the range is also very wide but shifted to more positive findings. This is shown below in Table 2. The heterogeneity implied by these ranges is biologically implausible. While the input from the panelists in the December 2005 CASAC consultation varied regarding the use of these data, there was much more skepticism regarding causality than was the case in the PM review. This is reflected in CASAC's June 5, 2006 letter to the Administrator that raises serious issues regarding the use of time-series studies.[75] The risk assessment, therefore, should include a probabilistic consideration that the associations may not be causal. In fact, the Regulatory Impact Analysis (RIA) does evaluate the case assuming that ozone mortality associations are not causal.

Table 2

Range of Ozone Individual-City Single-Pollutant Associations in Studies Included in CD Figures per Standard Ozone Increment as Defined in the CD (subject to model selection uncertainty and publication bias)

Figure 7-8	Emergency visits for asthma	- 20% to + 90%
Figure 7-9	Respiratory hospitalizations	- 17 % to + 17 %
Figure 7-13	Cardiovascular hospitalizations	- 12 % to + 35 %
Figure 7-14	All-cause mortality year-round	- 8 % to + 7 %
Figure 7-19	All-cause mortality by season	- 4 % to + 17 %

While staff indicate that there is clear and very strong evidence supporting the judgment that the relationships are causal for respiratory symptoms in asthmatic children and respiratory hospital admissions, staff acknowledges that there is greater uncertainty regarding mortality, describing the overall body of evidence only as highly suggestive.[76] However, as noted above, the latest multi-city study of asthmatic children in eight cities, including Boston, found associations with other air pollutants but not with ozone. In addition, the Mortimer study implicates air pollution in general, not ozone in particular. Therefore, the data for asthmatic effects in children is best described as mixed and inconsistent rather than clear and very strong evidence of an ozone effect. The results for respiratory hospital admissions, similarly, are more inconsistent than staff acknowledges.

- *Ambient monitors do not provide an adequate estimate of personal exposure.*

A second major uncertainty staff discusses is related to the adequacy of ambient monitors as surrogates for population exposure.[77] CASAC's June 5, 2006 letter points out that "personal exposure to ozone is not reflected adequately, and sometimes not at all, by ozone concentrations measured at central outdoor monitoring sites."[78] CASAC also pointed out that personal

[75] June 2006 CASAC Letter at 3.
[76] Second Draft SP at 5-76.
[77] Second Draft SP at 5-35; SP at 5-43.
[78] June 2006 CASAC Letter at 3.

exposures are much lower than ambient and that the susceptible population would be exposed to very low concentrations of ozone indeed.[79] Finally, CASAC pointed out that ozone measurement error obscures true thresholds in the concentration-response relationship and that this effect worsens with increasing degrees of measurement error.[80]

Since the true underlying personal exposure-response relationships implied by the disputed ozone/mortality associations are stronger by a factor of 2 to 4 than the concentration-response relationships suggest (due to the fact that indoor exposures are 2 to 4 times lower than outdoor exposures), the range of results in multi-city studies is actually 2 to 4 times larger (both positive and negative) than that indicated in Table 1. Such a wide range is biologically impossible.

In addition, the fact that the underlying personal exposure-response relationships implied by these associations are stronger by a factor of 2 to 4 than the ambient concentration-response relationships provides an even greater challenge to explain how low ozone concentrations, within the range of background, can be causing the premature mortality estimated in the risk assessment. If one takes the ozone associations at face value as evidence of effects, then one also has to explain how ozone can be both damaging in some cities and protective in others as well as damaging, on balance, in the summer and protective, on balance, in the winter. This pattern is not plausible and is not adequately acknowledged or discussed in the SP or proposed rule. The wide pattern is shown in Figure 7-17 at 7-90 of the CD, but downplayed as just heterogeneity due to differences in the cities.

- *The shape of the empirically-estimated concentration-response function is uncertain due to uncertainties in model selection, shape of the response and confounders.*

A third major area of uncertainty noted by staff is a set of issues related to the empirically-estimated concentration-response relationships.[81] These include uncertainty related to model selection, shape of the response and confounding. As we document above, the uncertainty in the "true" relation is much larger than the statistical uncertainty in any given association. While staff concludes that a linear response down to background should be applied, there are many reasons why this should not be the baseline estimate. CASAC rejected this approach for PM even though the PM database was viewed as more consistent. From the controlled human studies, we know that the body has mechanisms to deal with oxidative stress and the first responses are non-linear, transient and reversible. Without strenuous work or play for several hours or more, these responses are not found. Respiratory symptoms in healthy children were not included as an endpoint in this review because the CD concluded that there was no consistent evidence of an association between ozone and respiratory symptoms in children. Earlier drafts of the CD and SP concluded that the evidence for respiratory emergency room visits is inconsistent and inconclusive, as is the evidence for cardiovascular hospital

[79] June 2006 CASAC Letter at 3.
[80] June 2006 CASAC Letter at 4.
[81] Second Draft SP at 5-34, 5-76; SP at 5-42.

admissions. Assuming that there are mortality effects at background levels violates the coherence guideline from toxicology whereby lesser effects are seen at lesser doses and greater effects at greater doses. For all these reasons, mortality effects and any effects down to background levels of ozone should be severely discounted.

- *EPA's reliance on GEOS-CHEM for estimates of background concentrations significantly underestimates ozone background levels.*

The fourth major uncertainty relates to the use of the GEOS-CHEM model results for policy-relevant background.[82] As documented in comments submitted to EPA by Air Improvement Resources Inc. on behalf of the Alliance [EPA=HQ-OAR-2005-0172-0019] (dated January 17, 2006), the model will systematically underestimate the peak background levels. Therefore, several alternative higher background assumptions should be used. They could include a constant 0.040 ppm background, scaling the seasonal pattern from the Trinidad Head data, and a probabilistic formulation that provides yearly peak 8-hour values in the range of 0.060 to 0.075 ppm.

- *EPA fails to adequately consider the air quality benefits of meeting the current standard and overestimates the benefits of alternative standards.*

The fifth source of uncertainty relates to the adjustment to the air quality data to simulate just meeting alternative standards. The SP indicates that the shape of the distribution upon just meeting the current standard or an alternative standard is not known.[83] Importantly, EPA ignores background when rolling back the distribution of ozone concentrations at individual sites. EPA's rollback misses the increase in ozone concentrations near the bottom of the distribution as the man-made influence on ozone is decreased. The rollback overestimates the change in ozone at mid- and low concentrations and, thereby, overestimates the benefits of alternative standards. The rollback should be applied to the man-made portion of ozone not the entire distribution. The Agency is in a position to estimate the magnitude of this bias before the final rule using the ozone modeling carried out for the RIA, and correct the risk assessment before a final decision is reached.

As a result of all these uncertainties, the risk results presented in Chapter 5 of the SP and summarized in the proposed rule severely mischaracterize the magnitude of the risk and the certainty with which the risk is known. Since there are now more issues and inconsistencies related to the interpretation of the time-series studies than there were in 1996, when the decision was made not to rely on ozone/mortality associations, a similar decision is warranted for this review. EPA should acknowledge and discuss the full range of mortality associations in the literature, and the public should be informed that this range is not biologically plausible. Comparisons of multi-city results with meta-analyses have demonstrated a major issue with publication bias as noted by Goodman. Goodman's caution about publication bias and the

[82] Second Draft SP at 5-35, 5-77; SP at 5-43.
[83] SP at 5-43.

current weaknesses of single-site reports, along with Koop and Tole's caution[84] about the use of point estimates from time-series to set regulatory standards, need to be carefully considered by the Administrator before the final rule.

1. New studies on emergency room visits and hospital admissions do not provide supporting evidence of a causal relationship.

Although EPA's interpretation of the data on emergency room visits has changed, the evidence remains weak with inconsistent results. The literature on emergency room visits shows a very wide range of single-pollutant ozone associations. For asthma–related emergency room visits, the range is from –20 to + 80% per standard ozone increment (0.02 ppm change in 24-hr. ozone or 0.03 ppm change in 8-hr. ozone) as shown in Figure 7-8 at 7-68 of the CD. For total respiratory hospital admissions, the range is from –15 to +15% per standard ozone increment as shown in Figure 7-9 at 7-73 of the CD.

Although the data remained the same, EPA's interpretation changed during the ozone review process. In the Second Draft CD and the First Draft SP, the conclusion was drawn that the body of evidence remains inconclusive regarding effects of ozone on the risk of emergency department (ED) visits.[85] However, that conclusion was removed from the final CD and SP and replaced with a statement indicating that there are generally positive associations in summer analyses for asthma ED visits.

The proposal incorrectly notes that the studies concluded that co-pollutants are not significant confounders:

> Most studies in the United States and Canada indicated positive, statistically significant associations between ambient O_3 concentrations and respiratory hospital admissions in the warm season. However, not all studies found a statistically significant relationship with O_3, possibly because of very low ambient O_3 levels. Analyses for confounding using multipollutant regression models suggest that copollutants generally do not confound the association between O_3 and respiratory hospitalizations. Ozone effect estimates were robust to PM adjustment in all-year and warm-season only data.[86]

A number of rigorous evaluations of this literature come to different conclusions regarding consistency and possible confounding. Anderson, *et al.* 1998[87] reported that ozone, SO_2, NO_2, and particles all had positive associations with emergency asthma admissions in London, but that

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[84] G. Koop and L. Tole, *Measuring the Health Effects of Air Pollution: to What Extent Can We Really Say that People are Dying from Bad Air*, J. of Environmental Economics and Management, 47, 30-54. (2004).

[85] *See* U.S. Environmental Protection Agency, *Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information: OAQPS Staff Paper – First Draft*, EPA-452/D-05-002, Nov. 2005 at 3-8 (referring to the second draft CD at 7-62 as concluding that "the body of evidence remains inconclusive regarding the effects of O_3 on the risk of emergency department visits.")

[86] 72 Fed. Reg. at 37,832.

[87] H. Anderson, A. Ponce de Leon, J. Bland, J. Bower, J. Emberlin and D. Strachen, *Air pollution, pollens, and daily admissions for asthma in London 1987-92*, Thorax, 53, 842-848 (1998).

there was a lack of consistency across age groups and seasons. Anderson *et al.* also evaluated 15 other studies of air pollution and daily asthma emergency visits and admissions in the literature and concluded "Taken overall, it is apparent that the evidence is not coherent as to whether there is an effect of pollution or the responsible pollutant." They list a number of possible reasons for the lack of consistency, including false negatives due to lack of statistical power and false positives due to chance, multiple significance testing, post *hoc* hypothesis testing and publication bias. They also note differences in pollution levels and mix between cities, the presence of highly correlated pollutants, and that pollutants acting as surrogates for unmeasured pollutants or ambient aeroallergens may be involved. They conclude that, while there is evidence that all of the pollutants may have an effect on asthma, there is a lack of consistency in the specific pollutant responsible.

The review of ED studies during the 2004 ozone standard review in California indicated that if hospital admissions are affected by ozone, then it is likely that ED visits would also be affected, and to a greater degree. However, the ED results are less consistent and summarized as being inconclusive, as noted in the first draft SP. The California review[88] noted that of 20 studies they evaluated, 16 reported at least one significant association involving ozone. Given that studies without effects are difficult to publish and that these studies often include multiple comparisons, it is not surprising that there would be a number of positive associations in the literature. However, the California review also acknowledged that there is no clear pattern of association with specific respiratory disease outcomes.[89] For asthma ED visits, the results are described as variable. In the 27 studies of daily ED visits included in Table AX7-3 at AX7-40 of the CD, there is a very wide range of results with some multi-year studies reporting positive associations in only some years, with some ozone associations not robust to inclusion of other pollutants, with many single-pollutant associations being positive for only one of many lags or age groups. Therefore, it is hard to accept this data as strong evidence of consistent effects.

For hospital admissions, the 1996 risk assessment and the current risk assessment both use results from the Thurston *et al.* 1992 study of New York City. As such, this adds no new information. Also, the current risk assessment for hospital admissions uses the results from a study reporting no consistent increase in respiratory hospital admissions attributed to ozone in Los Angeles, an area with very high ozone levels. EPA uses one isolated result from the Linn *et al.* 2000[90] study of hospital admissions disregarding the totality of the results of the study, and the author's conclusions regarding a lack of ozone association with hospital admissions. Overall, the risk coefficients for the studies EPA used for their 2006 risk assessment are similar to those they used in 1996. The slight increase in projected rates of hospital admissions is attributed entirely to EPA's use of a lower background ozone level in the current risk assessment. See the general discussion of ozone background levels in Section H.

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[88] California Air Resources Board, *Review of the California Ambient Air Quality Standard for Ozone*, *Staff Report,* Vol III, Mar. 11, 2005, at 10-39.

[89] California Air Resources Board, *Review of the California Ambient Air Quality Standard for Ozone*, *Public Review Draft*, Vol. II, June 21, 2004, at 12-37.

[90] W. Linn, Y. Szlacheis, H. Gong, P. Kinney and K. Berhane, *Air pollution and daily hospital admissions in metropolitan Los Angeles*, Environmental Health Perspectives, 108, 427-434 (2000).

2. Overall, the school absence data are weaker than noted in the proposed rule and do not confirm causality or effects below the current standard.

A review of the literature shows that the results of school absence studies are highly dependent on the statistical approach employed. The SP notes that the association between school absences and ozone was assessed in two relatively large field studies.[91] The SP discusses the Chen *et al.* 2000 and Gilliland *et al.* 2001 studies of school absences, but quotes the CD that further replication is needed before firm conclusions can be drawn. The final CD discusses three analyses of the school absence data from the Southern California Children's Health Study (Gilliland *et al.* 2001, Berhane and Thomas 2002, and Rondeau *et al.* 2005) that used different statistical approaches and assumptions, and came to very different conclusions concerning the presence and magnitude of an ozone effect using the same data set.[92] This is an indication that the choice of the statistical approach used to analyze the data can change the findings dramatically. Overall, the evidence for school absence associations with ozone is weaker than noted in the SP or the proposed rule.

E. EPA Improperly Characterizes the Impact of New Information Since the Last Review, Particularly as it Relates to Mechanisms of Ozone Action and Support for Causality of Mortality and Other Important Epidemiological Health Endpoints.

Controlled studies with animals and cell systems fail to provide evidence for either systemic effects or cardiovascular effects except at very high dose levels. There is now a large body of controlled studies with animals and cell systems that provide information on ozone effects and the mechanisms that cause those effects. The determination of the medical significance of single or repeated exposures to ozone at various levels and the plausibility of various health effects needs to be evaluated with consideration of the body's defense mechanisms against pollutants in general and an irritating gas such as ozone, in particular. These defenses include the presence of antioxidants in the epithelial lining fluid, the mucociliary clearance system, alveolar macrophages and aspects of the immune system that protect the lung from physical injury, infectious disease and inhaled particles. The CD notes that ozone reacts preferentially with ascorbate, reduced glutathione, and urate (all antioxidants) in the epithelial lining fluid in a protective response.[93] Ozone also reacts with the carbon-carbon double-bond in polyunsaturated fatty acids. The products of these reactions can signal cellular responses such as inflammation. The CD notes that various models of these reactions do not agree as to whether ozone can penetrate the lining fluid and reach the epithelial cells.[94]

The review by Mudway and Kelly[95] indicates that ozone is a highly reactive gas that is consumed by reactive processes on reaching the first interface in the lung and the lung lining

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[91] SP at 3-12.
[92] CD at 7-57 to 7-60.
[93] CD at 5-5.
[94] CD at 5-2.
[95] I. Mudway and F. Kelly, *Ozone and the Lung: A Sensitive Issue*, Mol. Aspect. Med., 21, 1-48 (2000).

fluid compartment. They note that reactions between ozone and antioxidants tend to dominate in this compartment and these are generally thought of as beneficial or protective interactions. In their review, Mudway and Kelly discuss a range of antioxidant defenses. Mudway and Kelly further conclude that when ozone is relatively low-- probably less than 0.08 ppm -- the majority of ozone will be neutralized by reactions with uric acid in the nasal passages and upper airways.

As the ozone dose increases, the first effects of ozone are transient, reversible lung function decrements that are a reflex response due to vagal nerve receptors that involuntarily inhibit maximal inspiratory effort. This response is the body's reflexive reaction to the presence of an irritant gas and is unrelated to sensations of discomfort. Although it is well-established that ozone causes transient, reversible lung function decrements, the observational evidence argues against the presence of chronic lung function decrements related to ozone which, if present, would be a public health concern.[96] The acute decrements are decrements in the performance of the lung function test (that requires maximal inspiratory effort) and not a change in lung function of any clinical or pathological significance. The threshold for these first effects varies depending on ozone concentration, time of exposure and breathing rate. At somewhat higher exposures, there are mild symptoms and sensations of discomfort that also occur. These are also transient and reversible.

As the dose is increased, the reflex response noted above and then immune system responses begin to come into play. Even the immune system responses which are noted as the first indications of "inflammation" are physiological processes that occur in all living organisms under the stimuli of daily life. The first reported changes (that occur in humans with heavy exercise after 1- to 3-hours above a threshold of 0.18 to 0.20 ppm) are small and reversible and well within the range of physiological variability. They fall into the category of biochemical markers that the American Thoracic Society indicates do not necessarily imply adversity. For example, Mudway and Kelly note that for neutrophils transiting into the lung -- one of the earliest of these responses -- it is not clear if the response should be considered beneficial (functioning to clear necrotic cells) or detrimental (leading to an active inflammation with tissue injury). The CD notes that generally, "the initiation of inflammation is an important component of the defense process; however, its persistence and/or its repeated occurrence can result in adverse health effects."[97]

Based primarily on animal studies, after repeated doses that induce inflammation or acute high concentration doses, the acute inflammation can develop into chronic inflammation, continued inflammation can alter the structure and function of pulmonary tissue, and tissue injury and/or inflammation can interfere with the lungs response to other challenges such as other pollutants, viruses, bacteria, and allergens. In general, lesser effects are seen at lesser doses and greater effects at greater doses. There is little evidence for either systemic effects or cardiovascular effects from the literature on controlled studies unless the doses are such that there are strong respiratory effects.

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[96] 72 Fed. Reg. at 37,833.
[97] CD at 5-32.

In controlled studies, there are many instances of attenuation of some ozone responses with repeated exposures. In a general sense, these are manifestations of the body's defense mechanisms dealing with and adjusting to the presence of an irritant gas.

There is also now a large body of information related to ozone health effects from observational (statistical correlation) studies that can lead to a hypothesis of exposure cause and health effects but cannot confirm cause and effect. Since observational (statistical correlation) studies cannot by themselves establish cause and effect, a number of factors need to be evaluated before any given association of ozone with a health outcome can be viewed as causal or a real health effect. These factors include the strength, consistency, coherence and biologic plausibility. Biologic plausibility involves both the kinds of effects seen and the doses that cause the effects.

Based on the kinds of effects seen in well-designed controlled human exposure studies, one might predict finding acute lung function decrements, respiratory symptoms, and respiratory inflammation in susceptible subjects or in the healthy population in observational studies. One might also consider evidence of chronic respiratory problems associated with ozone plausible. One would not expect to see associations with cardiovascular hospital admissions or cardiovascular mortality, but one might not be surprised to see associations with respiratory hospital admissions or emergency room visits or possibly respiratory mortality. With regard to dose plausibility, one would expect to see thresholds in the data with effects at higher ozone concentrations or in subjects who exercise outdoors in the afternoon.

When the epidemiological literature of statistical correlation studies is compared to this expectation, there are areas of agreement and areas of disagreement. The small lung function decrements reported in field studies of children at summer camps and in other populations spending time outdoors may be real but, based on EPA's guidelines are not medically of concern.[98] Moreover, EPA indicates there is no consistent association with symptoms in healthy children or adults. In addition, there is less consistency in the association with symptoms or medication use in asthmatics than EPA claims. The data on respiratory hospital admissions or emergency room visits is also less consistent than EPA claims. Whether the positive ozone associations in the literature for respiratory morbidity are real health effects is not clear at present.

The fact that there is no consistent chronic ozone signal in the data is probably a result of the very low day-to-day exposures of either the general population or the susceptible population. For example, in cities where the day-to-day monitored ozone concentrations range from 0.01 to 0.08 ppm, the personal ozone exposures of the population are on the order of 0.01 to 0.04 ppm

[98] In the 1996 review, the results of six summer camp studies were analyzed to estimate a 2 to 3% decrease in FEV_1 at 0.12 ppm ozone, which is well within the range of normal measurement variability and well below the 10 to 20% changes which are considered moderate and potentially of health concern in the SP. Additional studies published since the 1996 review, for the most part, confirm the earlier results. The issue in interpreting these studies is not whether there are small changes in the performance of lung function tests, but what these small changes mean for public health given the involuntary reflex mechanism causing the changes.

during normal activity.[99] The mean personal exposures of the population are at or below the mean background, when considering the fact that people spend 90 percent of their time indoors where ozone concentrations are significantly lower. Such personal exposures do not trigger moderate FEV_1 responses through the vagal mechanism and have not been shown to trigger inflammation.

It is not clear how such low personal exposures could cause or contribute to the premature mortality estimated in the risk assessment. The lack of a cardiovascular hospital admissions signal in the data is not consistent with the mainly cardiovascular mortality associations with ozone. The likelihood that systemic effects or cardiovascular effects are causing mortality at the low day-to-day personal exposures of the population is also extremely low. As noted in the CD, by protecting against respiratory effects, systemic effects will likely be protected against also.[100] While the possibility of acute cardiovascular effects is raised in the CD based on limited epidemiologic findings, the controlled study of Gong *et al.*[101] which exposed subjects to 0.3 ppm ozone for three hours with exercise found little direct effect of ozone on the heart or vasculature for hypertensive or healthy subjects even though many pertinent variables were evaluated. Gong concluded that they did not find convincing evidence of significant direct cardiovascular effects from ozone exposure, analogous to effects reported in laboratory animals. The indirect effect they report, impaired gas exchange, can be explained as an effect secondary to the respiratory effects of the high ozone exposures.

Another possibility that is discussed in the CD and SP to explain a small positive warm season ozone association with mortality is that ozone is acting as a surrogate for other photochemical products. However, as noted above, greater warm season associations have been shown for all pollutants and the CD indicates that most widely present non-ozone oxidants have not been shown to have adverse effects in toxicological studies.[102]

Assuming that there are mortality effects down to background violates the coherence guideline from toxicology, whereby lesser effects are seen at lesser doses and greater effects at greater doses. For example, Figure 5-15 at 5-79 of the SP shows that the vast bulk of the estimated mortality is attributable to days on which the 24-hour ozone is 0.040 ppm or less. This ozone level was assumed as mean background in the prior review. Since people spend 90 percent of their time indoors where the ozone exposure is ¼ to ½ of the outdoor level, the personal exposures implicated as potentially causing mortality are well below background and there are no medically significant physiological ozone responses at such levels.

As discussed above, the single-pollutant time-series studies that are the basis for the respiratory admissions and mortality estimates are subject to a number of criticisms, regarding

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[99] CD, Table 3-3, at 3-64.
[100] CD at 5-57.
[101] H. Gong, R. Wong, R. Sarma, W. Linn, E. Sullivan, D. Shamoo, K. Anderson and S. Prasad, *Cardiovascular effects of ozone exposure in human volunteers*, Am. J. Respir. Crit. Care Med., 158, 538-546 (1998); A full report of the Gong *et al.* 1998 study is available as a report to the California Air Resources Board, Final Report, Contract No. 93-327, May 2000.
[102] CD at 7-2.

publication bias, model selection uncertainty, and an unrealistic range of positive and negative associations in systematic analyses. For all these reasons, EPA should assign significantly less weight to mortality effects and any other respiratory effects extrapolated down to background levels based on these studies.

Similarly, in both the prior review and the current review, the possibility that there is no threshold for ozone effects was considered, primarily based on the observational results. The proposed rule now admits uncertainty with regard to threshold effects:

> In summary, the Criteria Document finds that, taken together, the available evidence from clinical and epidemiological studies suggests that no clear conclusion can now be reached with regard to possible threshold levels for O_3-related effects (EPA, 2006a, p. 8-44). Thus, the available epidemiological evidence neither supports nor refutes the existence of thresholds at the population level for effects such as increased hospital admissions and premature mortality.[103]

The likelihood of a threshold should also be considered in drawing any conclusions regarding potential mortality risks from exposure to ozone levels at or below the current standard.

F. The Clean Air Act Does Not Require EPA to Set Standards That Eliminate All Risk.

Section 109 of the CAA requires the Administrator to set a primary standard, the attainment and maintenance of which in the judgment of the Administrator, are requisite to protect the public health with an adequate margin of safety. EPA's task is to establish standards that are "neither more nor less stringent than necessary" to protect public health. This does not mean zero risk, but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety. As Justice Breyer noted:

> Section 109(b) (1) directs the Administrator to set standards that are "requisite to protect the public health" with "an adequate margin of safety." But these words do not describe a world that is free of all risk -- an impossible and undesirable objective. . . . Nor are the words "requisite" and "public health" to be understood independent of context. We consider football equipment "safe" even if its use entails a level of risk that would make drinking water "unsafe" for consumption. And what counts as "requisite" to protecting the public health will similarly vary with background circumstances, such as the public's ordinary tolerance of the particular health risk in the particular context at issue. The Administrator can consider such background circumstances when "decid[ing] what risks are acceptable in the world in which we live."[104]

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[103] 72 Fed. Reg. at 37,840.

[104] *Whitman*, 531 U.S. at 494-95 (Breyer, J., concurring in part and concurring in judgment) (citation omitted).

EPA states in the preamble that its task under Section 109 is to establish standards that are neither more nor less stringent than necessary for these purposes. Lowering the standard in light of the 1997 determination would result in controls that would be more than necessary to protect public health.

The Supreme Court has agreed with EPA that "requisite" under Section 109 means "sufficient, but not more than necessary."[105] The material presented in the CD and SP do not support EPA's proposal to lower the standard. As discussed further in these comments above, EPA's own assessment of the risks fails to show an increase in risk from exposure to ozone. In fact, as stated above, much of the evidence supports a reduction in risk. Given EPA's data showing limited risks from exposure to the current standard and EPA's analysis of the studies which shows greater, not less uncertainty, in the likelihood that the observed effects are caused by ozone, EPA should retain the current standard. As EPA states in the preamble, its task under Section 109 is "to establish standards that are neither more nor less stringent than necessary."[106]

G. Since EPA Has Not Provided a Reasonable Analysis Supporting its New Judgment on What is Requisite to Protect Public Health or Welfare, its Actions Could Be Found to Be "Arbitrary and Capricious."

In exercising his judgment to revise the existing NAAQS, the Administrator must provide a reasonable analysis for the change. Specifically, EPA must explain why its earlier judgment regarding what is requisite to protect public health or welfare no longer governs. To reverse its position in the face of precedent that is not persuasively distinguished is arbitrary and capricious.[107] Given that EPA's estimate of health risks has declined and our understanding of the uncertainty surrounding the results of the health studies has increased, EPA cannot persuasively say why it must depart from its 1997 decision that the current standard is requisite to protect public health.

H. EPA Has Not Correctly Evaluated the Potential Risk Reduction as a Result of its Proposed Change to the Current Ozone Standard.

In the 1997 final ozone rule, EPA estimated, based on monitoring data, that background levels from natural sources could be as high as .07 ppm. This fact was important to EPA's decision regarding the level of ozone deemed requisite to protect public health with an ample margin of safety:

> As many commenters have noted, based on information in the Criteria Document with regard to ambient concentrations of O_3 from background sources, an 8–hour standard set at a 0.07 ppm level would be closer to peak background levels that infrequently occur in some areas due to non-anthropogenic sources of O_3

[105] *Whitman*, 531 U.S. at 473 (citation omitted).
[106] 72 Fed. Reg. at 37,820.
[107] *Motor Vehicle Mfr.'s Ass'n*, 463 U.S. at 42 (presumption "against changes in current policy that are not justified by the rulemaking record").

> precursors, and thus more likely to be inappropriately targeted in some areas on such sources. After taking into account the public comments, and for the reasons outlined above, the Administrator finds that a standard set at a level of 0.07 ppm is not requisite to protect public health with an adequate margin of safety.[108]

EPA's analysis of background levels in the current review is wholly deficient and does not support a lowering of the standard.

1. In contrast to the 1997 Review, EPA failed to evaluate the extreme values of ozone background levels.

Uncontrollable ozone background levels due to transport of stratospheric air (high in ozone) into the troposphere, transport of man-made polluted air from locations outside the U.S., and photochemical formation from natural NOx and VOC emissions can significantly constrain an area's ability to reduce ozone concentrations. As discussed in the next section, the uncontrollable background also plays a pivotal role in EPA's estimate of the potential health benefits of further reductions in the ozone standard. In previous ozone reviews, EPA evaluated this background primarily based on measured observations of ozone and other compounds in remote locations. In the current review, the CD discusses estimates of background from both observations and models, but the SP relies entirely on one global chemical transport modeling study to model an estimated mean background.

Because of the form of the standard, the background related to the extremes of the distribution of the daily maxima, as well as the means, must be evaluated as was done in the 1997 Review. Ozone background levels must be evaluated for each ozone metric being considered for the primary and secondary standards. Since extreme value forms of the ozone standard are being proposed, then measured ozone values or modeled estimates of the extremes of policy-relevant background (PRB) <u>must</u> be considered. In previous ozone reviews, both the means and extremes of background were evaluated. For example, the Agency's Staff Paper in the previous review concluded:

> …a reasonable estimate of the background O_3 concentrations near sea level in the U.S. for a 1-hour daily maximum during the summer is usually in the range of 0.03 to 0.05 ppm. At clean sites in the western U. S., the maximum annual hourly values are in the range of 0.06 to 0.075 ppm.[109]

In the current review, the SP relies solely on the Fiore *et al.* 2003[110] global modeling study to estimate mean background. However, the CD notes several important limitations of that study and the need for additional work to evaluate PRB with an ensemble of models comparing the

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[108] 62 Fed. Reg. at 38,868.

[109] U.S. Environmental Protection Agency, *Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information: OAQPS Staff Paper*, EPA-452/R-96-007, June 1996 at 20.

[110] *References denoted by author and year in the text can be found in the CD and SP .*

model results with observations.[111] The large grids in the global model mix air parcels of different composition and origin in an unrealistic way, severely limiting its ability to accurately simulate local photochemical activity, intercontinental transport, and the impact of stratospheric air inserted into the troposphere. The CD notes the need to include models with greater resolution to understand variability on shorter time scales and variability due to processes that are not captured in the global models. Until this is done, a range of background estimates from observations only should be used by the Agency. Because of the acknowledged limitations of global models, observations should be the primary source for estimates of extreme values of background at specific locations.

The failure of the current final SP to provide information on the extremes of background is a serious omission. As a result, the ranges recommended by CASAC and EPA staff overlap with the extremes of background and are unlikely to be achievable in some areas of the country.

2. EPA's definition of PRB incorrectly excludes man-made emissions from Canada and Mexico.

The Agency defines a PRB as those concentrations that would result in the U.S. in the absence of anthropogenic emissions in North America; *i.e.* leaving no room for ozone caused from human activities. However, EPA's definition of PRB is flawed for several reasons. The model exercise EPA relied on omits the contribution from the emissions from agricultural activities and it omits consideration of the contribution to ozone from anthropogenic emissions in Mexico and Canada. While there can be reductions in emissions from agricultural activities, as with other human activities, it would be impossible to drive them to zero. No one contemplates the cessation of human activities including the practice of growing crops and raising animals in the U.S. Omitting the impact of Canadian and Mexican emissions is also problematic because, even though the U.S. may have treaties with these countries, zero anthropogenic emissions and ceasing human activities in these countries is not a possible future outcome that can be controlled by U.S. regulations.

3. EPA does not provide sufficient rationale for relying on modeled results over monitoring data for assessing background ambient ozone levels.

The SP notes that because of long-range transport from anthropogenic source regions within North America, estimates of PRB cannot be derived solely from measurements of ozone.[112] However, it goes on to state that PRB estimates must be based on modeling. This is not true.[113] While the transport of plumes from urban areas is well documented in the literature, the CD offers no observation-based analyses to demonstrate that the many instances of ozone greater than 0.06 ppm that are measured at remote monitoring sites are all caused by transport from urban areas. As documented in Volume II (Annexes 2 and 3) of the CD, there are still major uncertainties and limitations involved in using atmospheric models. The question is not

[111] CD at 3-55.
[112] SP at 2-54.
[113] SP at 2-54.

whether to use observations or models, both observations and models should be used to estimate PRB.

For example, a recent paper by Jaegle, *et al.*[114] used satellite observations of NO_2 columns to derive estimates of NOx emissions from fuel combustion, biomass burning and soils. They conclude that there is a significant role for soil NOx emissions at northern mid-latitudes during summer, where they account for nearly half that of the fuel combustion source and a doubling of the inventory used in the GEOS-CHEM model. They further conclude that the contribution of soil emissions to background ozone is likely to be underestimated by the current generation of chemical transport models.

There are NOx emissions from soils due to microbial processes. Fertilized agriculture produces the highest soil NOx emissions. By turning off the agriculture source in their PRB analyses, Fiore *et al.* systematically underestimated uncontrollable background. Since the total soil source is twice as high as used in the GEOS-CHEM model, Fiore *et al.* also misallocates the NOx between fuel combustion and natural sources. This will result in a misallocation of ozone between man-made and natural sources. The presence of a large soil NOx source in the Great Plains may also help explain the discrepancy between the locations of elevated SUM06 and W126 and the major populated areas of the country.

4. The proposed rule ignores the conclusions of several researchers and previous conclusions of EPA regarding maximum background levels.

Recently, Vingarzan[115] reported the range of annual maxima at background stations in the U.S. and Canada. The annual maxima (1-hour) at Western U.S. and Canadian background sites approached or exceeded 0.08 ppm.

The level of background ozone first became a policy issue in 1971 when EPA set the NAAQS for Photochemical Oxidants at a concentration of 0.08 ppm for 1-hour, not to be exceeded more than once per year. In 1978, Singh *et al.*[116] reported an analysis of long-term ozone data from remote sites that indicated summertime average daily 1-hour maxima in the 0.04 to 0.05 ppm range but maximum 1-hour concentrations that can approach or exceed 0.08 ppm in the spring. Singh *et al.* concluded that achievement of a yearly 1-hour ozone standard of 0.08 ppm may be impossible.

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[114] L. Jaegle, L. Steinberger, R. Martin and K. Chance, *Global partitioning of NOx sources using satellite observations: Relative roles of fossil fuel combustion, biomass burning, and soil emissions*, Faraday Discussions, 130: 407-423 (2005).

[115] R. Vingarzan, *A review of surface ozone background levels and trends*, Atmospheric Environment, 38, 3431-3442 (2004).

[116] H. Singh, F. Ludwig and W. Johnson, *Tropospheric ozone concentrations and variabilities in clean remote atmospheres*, Atmos. Environ., 12, 2185-2196 (1978).

When EPA revised the federal 1-hour ozone standard in 1979 to 0.12 ppm, the Agency acknowledged that:

> Field measurements at some remote sites, where man-caused ozone is likely to be negligible, have shown low-but not insignificant- rates of exceedances of the 0.08 ppm level originally proposed for the secondary standard.[117]

In 1989, Logan[118] reported the results of an analysis of ozone data from rural locations in the U.S. Logan reported that ozone concentrations above 0.080 ppm were common in rural areas of the Eastern U.S. in spring and summer (occurring between about 2 and 8% of the time) but were unusual at remote western sites, occurring less than 0.5% of the time. Logan also pointed out that concentrations of NOx in rural areas of the east are frequently high enough to permit significant photochemical formation of ozone during favorable weather conditions, but that NOx concentrations are much lower in remote regions of the west. The low NOx concentrations in the west are expected due to the low population and emissions density, and effectively limit the local formation of ozone in these air masses. Logan also reported that the median ozone concentrations of 0.03 to 0.04 ppm were similar at rural sites across the country even though there is a much greater population and emission density in the Eastern U.S. than in the Western U.S.

Lefohn and Foley[119] reported in 1991 on an analysis of ozone data from 26 Class I national parks and wilderness areas. For the seven cleanest sites, the yearly maximum 1-hour average concentrations were in the range of 0.06 to 0.075 ppm.

In 1996, Altshuller and Lefohn[120] published an analysis of background ozone in the planetary boundary layer of the U.S. They selected 11 sites for analysis. The criteria they used included using sites receiving the cleanest air masses from the upwind flow off a continent or ocean, and sites isolated from the influence of urban plumes or regional ozone formation from anthropogenic emissions. They reported that the maximum 1-hour concentrations in the Western U.S. in the April through October period ranged from 0.050 to 0.098 ppm and the maximum 1-hour concentrations at coastal sites ranged from 0.044 to 0.080 ppm. For comparison, daily maximum 1-hour concentrations are typically 1.3 times higher than daily maximum 8-hour concentrations.

In 2001, Lefohn *et al.*[121] reported an analysis that concluded that hourly levels of 0.05 and 0.06 ppm are often exceeded at Northern U.S. and Canadian sites specifically selected to minimize anthropogenic influences. Most occurrences were in April and May but sometimes as

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[117] 44 Fed. Reg. 8202, 8212 (Feb. 8, 1979).

[118] J. Logan, "*Ozone in Rural Areas of the United States*," J. Geophys. Res., 94, D6, 8511-8532 (1989).

[119] A. Lefohn and J. Foley, *Estimated Surface-level Ozone Exposures in Selected Class I Areas of the United States*, paper 91-144.2, presented at the 84th Annual Meeting of the Air & Waste Management Association, Vancouver, British Columbia, June 1991.

[120] A. Altshuller and A. Lefohn, *Background Ozone in the Planetary Boundary Layer Over the United States*, J. Air & Waste Management Association, 46, 134-141 (1996).

[121] A. Lefohn, S. Oltmans, T. Dann, and H. Singh, *Present-day variability of background ozone in the lower troposphere*, J. Geophys. Res., 106, D9, 9945-9958 (2001).

late as June. They concluded that many of these occurrences were associated with natural ozone sources, particularly upper tropospheric and stratospheric air.

5. Because the PRB varies substantially, there will be times and places where the background approaches or exceeds the levels recommended by CASAC for both the primary and secondary standards.

The SP attempts to limit the discussion of policy relevant background to the meteorological conditions generally conducive to peak urban ozone formation. While this is often the limiting case for development of control plans, it would not be under the standards recommended by CASAC. The issue is not only what the background is on days of maximum photochemical potential, but also what the background is on days with the highest background.

To illustrate, if the background is 0.04 ppm and the standard is 0.07 ppm, the amount of ozone that can be allowed to be formed from man-made emissions is only 0.03 ppm. So even with a 0.04 ppm background, a 0.07 ppm standard would allow little room for human activities. On a day when the background is 0.06 ppm, there would be even less margin for human activities. While this illustration over-simplifies the complex chemical and meteorological processes involved in ozone formation and transport, it demonstrates that the variability of background due to transport of ozone from upwind natural and non-U.S. man-made sources can make some of the standards under consideration unattainable.

A practical example of this issue is found in Winner and Cass.[122] Winner and Cass modeled the Los Angeles Basin over an entire year and evaluated VOC and NOx controls from 0 to 95 percent. They concluded that the current 8-hour standard may be unattainable in the basin even with complete elimination of man-made emissions and, importantly, they found that the days with lower than peak ozone were less responsive to VOC and NOx reductions than the peak ozone days. This means that the days that are most difficult to control are not necessarily the days that produced the peak concentrations in the historical base year.

6. The lack of consideration of the extremes of ozone background levels is an omission that led both the Agency and CASAC to assume that the recommended standards are attainable.

Ozone background concentrations are an important factor in setting and achieving ozone air quality standards and in estimating risk. Background concentrations also play a role in the designation of attainment or nonattainment, in the development of State Implementation Plans (SIPs), and in the ultimate achievability of any specific standard. EPA's failure to accurately assess ozone background extremes resulted in recommendations for new standards that are unachievable.

It is clear from the structure of the CAA that Congress assumed that air quality standards would focus controls on man-made pollution and would be achievable in reasonable timeframes.

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[122] D. Winner and G. Cass, *Effect of Emissions Control on the Long-Term Frequency Distribution of Regional Ozone Concentrations*, Environ. Sci. Technol., 34, 2612-2617 (2000).

Once a standard is set and designations of attainment/nonattainment are made, the CAA requires states to prepare SIPs to achieve the standard. Under Subpart 1, Section 172(a)(2)(A) of the CAA, non-attainment areas must attain the primary standard "as expeditiously as practicable, but no later than 5 years from the date such area was designated nonattainment."[123] The Administrator may extend the attainment date for a period of no greater than ten years based on the severity of nonattainment and the feasibility of pollution control measures.[124] Under the ozone-specific provisions of Subpart 2, the Act assigns ozone nonattainment areas with a series of separate deadlines and control requirements based on the degree of nonattainment of the former 0.12 ppm 1-hour standard. In both frameworks, the underlying assumption is that the standard can be met with man-made controls.

The penalty for failure to meet by the designated deadline is a bump-up to a higher category with a longer deadline but also more prescriptive control requirements in the control region. The penalty for failure to plan properly is federal sanctions.

In recommending a range of 0.060 to 0.070 ppm, CASAC apparently assumed that an 8-hour standard in this range was ultimately achievable from control of U.S. man-made emissions. As documented above, it is not at all clear that this is the case. One of the specific charges for CASAC under the CAA (Section 109(d)(2)(c)(iii)) is to "advise the Administrator on the relative contribution to air pollution concentrations of natural as well as anthropogenic activity."[125] A complete reading of the CD indicates that reliance on one particular global model is not a scientifically sound way to estimate the contribution of uncontrollable ozone to an extreme value standard.

The CAA also requires CASAC (Section 109(d)(2)(c)(iv)) to "advise the Administrator of any adverse public health, welfare, social, economic, or energy effects which may result from various strategies for attainment and maintenance of such national ambient air quality standards."[126] Accurate assessment of ozone background levels is key to the photochemical modeling used in the SIP development. Photochemical grid modeling has moved from the modeling of multi-day episodes in specific urban areas to modeling ozone concentrations throughout a season over large regions. For the latter situation, an accurate depiction of stratospheric/tropospheric exchange and subsequent transport is necessary. However, the simulation of stratospheric intrusions is notoriously difficult in global models because stratospheric-tropospheric exchange is an intermittent process—a series of strong exchange events separated by more quiescent periods. These events insert stratospheric air high in ozone into layers or filaments in the troposphere. Only rarely do these intrusions penetrate to ground level intact, but they intermittently insert high concentrations of ozone into the troposphere much more often. The layers or filaments of stratospheric ozone eventually mix with surrounding air in ways which are not yet fully understood, influencing ozone levels, photochemical activity and lifetimes of various species. Because of the complexity of these phenomena, an exceptional

[123] 42 U.S.C. § 7502(a)(2)(A).
[124] 42 U.S.C. § 7502(a)(2)(A).
[125] 42 U.S.C. § 7409(d)(2)(c)(iii).
[126] 42 U.S.C. § 7409(d)(2)(c)(iv).

events policy would not be able to identify the contribution of stratospheric intrusions to ground-level ozone.

When the EPA Administrator set the current federal 8-hour standard, she considered how close an 0.07 ppm standard would be to background, noting that "As many commenters have noted, based on information in the [CD] with regard to ambient concentrations of O_3 from background sources, an 8-hour standard set at a 0.07 ppm level would be closer to peak background levels that infrequently occur in some areas due to non-anthropogenic sources of O_3 precursors, and thus more likely to be inappropriately targeted in some areas on such sources."[127]

The lack of consideration of the extremes of background is an omission that led both the Agency and CASAC in the current review to assume that the standards recommended are attainable. If an 8-hour ozone standard were established at the lower end of the range recommended by CASAC, virtually the entire country would be designated nonattainment, even areas that are far removed from significant man-made emissions. It would be impossible to develop rational implementation plans for some areas and, for other areas, complete elimination of man-made emissions might be required to demonstrate attainment.

I. EPA Has Not Adequately Considered That in Many Areas of the U.S. Additional Reductions, Beyond Those Needed to Meet the Current Standard, May Not Be Achievable With Known Control Technology and Could Impose Ruinous Economic Consequences.

EPA correctly notes that some cities will have difficulty in complying with the current standard within the next decade and invites comment on whether EPA can count the health benefits from these reductions:

> As a related concern, a number of areas – including some of the cities involved in the risk assessment – will have difficulty in complying with the current 8-hour standard within the next decade. As a result, the full public health gains in these areas from a more stringent 8-hour standard are unlikely to be realized for a number of years. In light of the fact that these public health gains may not fully materialize within the attainment date structure set forth in the Clean Air Act, some commenters question whether the Agency can or should consider these projected gains as a health based criterion for its decision making. EPA requests comment on this view.[128]

The situation in several areas is direr than implied in the preceding paragraph. The SIPs prepared for the South Coast Air Basin and San Joaquin Valley of California that were due to EPA in June 2007 lay out the emission reductions required to attain the current 8-hour ozone standard in these air basins. In both cases, the emission reductions required for attainment that were determined using photochemical modeling are massive. Both these areas have been reducing emissions of ozone precursors for many decades, yet they still require an additional 75

[127] 62 Fed. Reg. at 38,868.
[128] 72 Fed. Reg. at 37,881.

percent reduction in NOx emissions from current levels to demonstrate attainment.[129] The overall reduction from uncontrolled levels is well in excess of 90 percent and the required reduction in emissions per capita or per unit of economic activity compared to uncontrolled levels is even greater. Neither District can identify the control strategies that will provide such additional massive reductions so the Districts are each asking to be reclassified to the 'extreme' category so they can take advantage of Section 185(e)(5) of the CAA, which allows extreme areas to submit approvable plans that rely on future advancements in technology, the so-called "black-box" emission reductions.

The San Joaquin Valley District concludes that currently available technology and known technology scheduled for future availability cannot provide sufficient emission reductions to fill the attainment gap. It is noted that this would be the case even if money was no object. In fact, the District concludes that shutting down all businesses, or shutting down agriculture, or removing all truck traffic, or removing all passenger cars, or replacing all motor vehicles with the newest and cleanest vehicles at the cost of $60 billion would not fill the attainment gap. The District notes that these extreme and unrealistic measures were evaluated only to illustrate the magnitude of the problem.

The use of the black box allows EPA to approve a SIP without having specific identified controls, but the experience with the black box in the South Coast District has not been favorable. After decades of research and development, the technologies for pollution control are mature and the improvements available now are small and incremental. There are no more low-hanging fruit that are available to provide major reductions in emissions in California. Therefore, it is unlikely that either District will be able to identify measures (short of drastic measures such as those noted above that would cause economic havoc and be a direct contradiction of the Supreme Court decision) to attain the current ozone standard within the timeframes required by law, even given the extreme classifications.

In addition, the Winner and Cass modeling study, noted above, raises the possibility that the modeling of current ozone episodes may not be the limiting case for ozone attainment and even more emission reductions may be required than in the 2007 District air quality plans.

EPA's estimate of health benefits from lowering the ozone standard is based on a definition of policy relevant background that assumes man-made emissions can be eliminated. This, however, goes beyond the reach of the Act and is in direct contradiction with the Supreme Court decision. As the proposal notes:

> As discussed by Justice Breyer in *Whitman v. American Trucking Associations*, however, this interpretation of § 109 does not require the EPA to eliminate every health risk, however slight, at any economic cost, however great, to the point of

[129] *See* San Joaquin Unified Air Pollution Control District, *2007 Ozone Plan*, Apr. 30, 2007; South Coast Air Quality Management District, *Final 2007 Air Quality Management Plan*, June 2007.

"hurtling" industry over "the brink of ruin," or even forcing "deindustrialization."[130]

J. The Agency's Estimates of Health Benefits Are Highly Sensitive to Assumptions Regarding Background Levels.

The proposed rule notes that changes in the assumed background can either double or halve the estimated mortality effects.[131] This sensitivity to 0.005 ppm changes in background is a demonstration that the bulk of estimated mortality comes from days with ozone concentrations that are very low. This is not biologically plausible.

The sensitivity of clinical respiratory effects to background assumptions is not as large, but is still very important. For example, the Risk Assessment Technical Support Document (TSD)[132] shows that the number of exposures of lung function responses of healthy and asthmatic children (Tables 3-28 and 3-31, respectively) is reduced with a 0.005 ppm increase in PRB almost as much as a lowering of the standard from 0.084 to 0.074 ppm.[133] Thus, the choice of background is extremely important in evaluating the adequacy of the current standard and in estimating the public health impact of a more stringent standard.

Given all the issues with EPA's assumed background noted above, the Agency should re-evaluate its assumed background and then re-evaluate the clinical risks. Only then can the adequacy of the current standard be evaluated on a sound basis. The proposed rule recognizes the importance of this issue and indicates additional sensitivity analyses to background will be carried out.[134]

K. CASAC's Conclusion That the Current Standard is Unjustified is Based on Unsupportable Assumptions.

CASAC's recommendation to tighten the ozone standard was not based on fundamental changes in the science from the 1996 review, but rather on EPA's decision to lower its estimate of mean ozone background concentrations and ignore the extremes of background. In 1996, EPA staff and CASAC agreed on a range for the 8-hour standard between 0.07 and 0.09 ppm.[135] When queried individually, no one on the 1996 panel recommended a standard below 0.08 ppm. Currently, CASAC recommends a standard between 0.060 and 0.070 ppm.

The major reason for the difference between the two panel's recommendations is that the current panel assumed that mean background was substantially lower than previously thought in 1997. The current CASAC panel did not carefully consider the extremes of background and so

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[130] 72 Fed. Reg. at 37,820 (quoting *Whitman*, 531 U.S. at 494).
[131] 72 Fed. Reg. at 37,862, n.45.
[132] E. Post *et al., Ozone Health Risk Assessment for Selected Urban Areas*, EPA 452/R-07-009, July 2007.
[133] U.S. Environmental Protection Agency, *Ozone Health Risk Assessment for Selected Urban Areas*, EPA-452/R-07-001, Jan. 2007, Tables 3-28, 3-31, at 3-70, 3-73 ("RA TSD").
[134] 72 Fed. Reg. at 37,857, n.40.
[135] G. Wolff, CASAC Letter, EPA-SAB-CASAC-LTR-96-002, Nov. 30, 1995 ("1995 CASAC Letter").

incorrectly assumed that uncontrollable background was substantially lower than thought in the previous review. Based on the information presented to CASAC from the GEOS-CHEM model, CASAC was led to believe that ozone background levels never exceed 0.040 ppm. As a result, CASAC incorrectly assumed that a standard as low as 0.060 ppm is attainable and that, therefore, exposures of concern could be essentially eliminated.

In the previous review, CASAC concluded that the estimates of risk for various alternative standards were very similar after reviewing the risk assessments for both clinical and epidemiological endpoints. Specifically, the 1996 panel concluded that there was no "bright line" that distinguished any of the proposed standards as being significantly more protective of public health.[136] In making this judgment, the 1996 panel focused on the overlap and uncertainty between the estimates of the percent responding rather than on the absolute number of incidences.[137] In contrast, the current CASAC Panel focused on the absolute number of incidences, even though they are only a very small fraction of the potential number of incidences, and again, the differences in incidences at alternate standards are minimal.

Beyond the error in evaluating background, there are several other ways in which CASAC erred in evaluating the scientific information. Although the Second Draft SP showed that the risk of lung function effects is lower than thought in 1997, the current CASAC panel appeared to believe that the APEX model used in the risk assessment for the clinical effects systematically under-predicted measured values.[138] In actuality, the model significantly overestimated the number of exposures of concern as detailed elsewhere in these comments.

Although CASAC raised serious questions regarding the use of time-series studies to set regulatory standards in its June 2006 letter, those concerns were ignored in the October 2006 letter which uncritically accepted single-pollutant positive associations as evidence of health effects. CASAC failed to provide a reason why the issues raised in the June 2006 letter no longer applies. Since the science did not change, the more logical conclusion is that the concerns raised in CASAC's June 2006 letter still apply and should be considered by EPA in its review.

For example, the June 2006 letter highlights the issue of exposure measurement error in time-series studies. In addition, the October 2006 letter acknowledges that "Panel members have little insight as to what we would find if we had actual exposure measurements."[139] Nevertheless, in the October 2006 letter, CASAC claims that ED visits for respiratory disease, especially asthma, are relatively strong and relatively consistent, citing Figure 3-4 of the SP as evidence.[140] However, the apparent consistency in the figure reflects only that there are a number of reports of positive associations with various health endpoints in single-pollutant models in the literature.

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[136] *See* the October 2006 CASAC Letter at 11.
[137] 1995 CASAC Letter at 2-3.
[138] *See* R. Henderson, CASAC Letter, EPA-CASAC-07-001, Oct. 24, 2006 at 11 ("Oct. 2006 CASAC Letter").
[139] October 2006 CASAC Letter at 10.
[140] October 2006 CASAC Letter at 10.

In making a judgment with regard to consistency, CASAC did not consider the way EPA chose to present the data to establish consistency. If these were controlled studies, we would ask how many total experiments were conducted and how many were positive. We should know both the numerator (the number of positive associations) and the denominator (the total number of studies) before we make a judgment about consistency. For much of the epidemiology, we have only the numerator. For a given health endpoint, we do not know how many different models, cities, lags, *etc.* were evaluated. For purposes of drawing conclusions, there is a major difference between 6 out of 6 positive associations of ozone *versus* 6 out of 60 or 6 out of 600. CASAC did not evaluate the way EPA chose to present the data to establish consistency. In fact, the more rigorous analysis of the full body of data by Anderson *et al.* 1998 establishes that there is little consistency regarding ozone association with ED visits.

L. Tropospheric Ozone Protects Against UV-Induced Health Effects.

It is well-established that tropospheric ozone has a beneficial effect along with stratospheric ozone in protecting public health from solar UV radiation. A reduction in tropospheric ozone would act to increase UV exposures and UV-induced health effects. This topic is discussed in Section 10.2 of the CD and the conclusion is drawn that the beneficial effects of tropospheric ozone cannot be assessed quantitatively at this time. This conclusion is troubling and inconsistent with the science. The health effects from exposure to UV radiation are substantial and ozone's causal link to UV radiation is better established than is the case for ozone and daily mortality. For example, the CD concludes that "[t]he current evidence strongly suggests a causal link between exposure to UV radiation and the incidence of both nonmelanoma and melanoma skin cancer."[141] The CD stresses the uncertainties and limitations in estimating beneficial effects and generally characterizes the impacts as small and uncertain. While this may be true, it is also true of the adverse health effects from exposure to ozone implied by many of the epidemiological associations discussed above. When EPA responded to a court's remand to consider UV-related effects of ground level ozone, various estimates of these effects (made by EPA staff, EPA consultants and staff at other government agencies) were discussed at some length.[142] In all cases, there was no disagreement with the fact that ground-level ozone has beneficial effects. The only disagreements centered on the magnitude of the effect or the certainty with which it can be estimated.

EPA now argues that one needs to do a detailed analysis similar to the APEX exposure model to estimate the beneficial UV-related effects, and that all the necessary inputs for such a calculation are not available. EPA, however, does not adhere to this same view when estimating detrimental effects of ground-level ozone on mortality. Instead, a much cruder observational approach is tolerated that has enormous uncertainty associated with it. This created a clear double standard.

The proposed rule asks for available studies or data that would be relevant to conducting a critical assessment with reasonable certainty of UV-induced health outcomes and how evidence

[141] CD at 10-29.
[142] 68 Fed. Reg. 614 (Jan. 6, 2003).

of UV-induced health outcomes might inform the Agency's review of the primary ozone standard.[143]

Even though EPA indicates it cannot make reliable quantitative estimates of beneficial ozone impacts for this rulemaking, quantitative estimates of UV-related health effects have been made by EPA and others for various reasons. For example, as part of the estimate of the global burden of disease, the World Health Organization (WHO) has estimated that excessive UV exposure is responsible for about 1.5 million disability-adjusted life years and over 60,000 premature deaths per year world-wide primarily due to cataracts and skin cancer.[144] The situation is complex because people need some UV to maintain adequate Vitamin D levels. In the current review, the potential benefits of ground-level ozone should be weighed along with the potential harm, using similar approaches for each type of effect.

The Alliance recommends that EPA use the same methods the WHO and the Agency did when estimating UV effects and allocate a reasonable portion of those effects to ground-level U.S. man-made ozone. If EPA does not accept such an approach, at a minimum, the Agency should acknowledge that its risk assessment estimates of ozone-mortality effects do not provide sufficient certainty.

M. The Scientific Data and Precision of Ozone Measurement Monitors Does Not Support Specifying the Standard to the Third Decimal Place.

In recommending the ozone standard be specified to the third decimal place, CASAC overstates the precision and accuracy of current ozone measurements. CASAC recommended that the ozone standard be specified to the third decimal place in ppm because current measurement technology "allows accurate measurements of ozone concentrations with a precision of parts per billion."[145] Although California recently specified an 8-hour ozone standard to the third decimal place in ppm, the California Air Resources Board (CARB) staff acknowledges that the third decimal place is an uncertain digit.[146] At the 0.070 ppm level, CARB staff indicated that ozone measurements are uncertain in the third decimal place to the degree of plus or minus 0.003 ppm. Another way of expressing this uncertainty is that 95 percent of the true values will be within two standard deviations, or plus or minus 0.006 ppm. The EPA estimate of precision noted in the SP of plus or minus several percent is consistent as is the precision reported for the CASTNET sites where 98 percent of the audits were within plus or minus 10 percent. In discussing the uncertainty due to measurement error, Langstaff notes that federal reference and equivalent methods are required to have a precision of 0.01 ppm for 1-hour average concentrations (40 CFR Ch.1, Section 53.21) and that interference with other pollutants

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[143] 72 Fed. Reg. at 37,837.

[144] R. Lucas, T. McMichael, W. Smith, and B. Armstrong, *Global Burden of Disease from Solar Ultraviolet Radiation, Environmental Burden of Disease*, Series No. 13, World Health Organization, Geneva, 2006.

[145] Oct. 2006 CASAC Letter at 5.

[146] California Air Resources Board, Staff Report, *Review of the California Ambient Air Quality Standard for Ozone, Staff Report*, Vol II, Mar. 11, 2005, at 6-3.

and humidity can lead to errors in measurements.[147] Given this level of precision, the current round-off procedure is appropriate.

The proposed rule refers to an analysis by Cox and Camalier (2006) that purports to show the impact of measurement error on the design value is small -- the order of 1 ppb for precision and 1 ppb for randomly occurring instrument bias.[148] However, there is no reason to assume that the bias is random. EPA has considerable data on the site-specific precision and bias estimates. For example, Langstaff reports bias estimates for individual Boston monitors varying between -7 and + 11 % in 2003 taken from the larger EPA database.[149]

Given that the current requirement for the precision of monitors is specified to only two decimal places, the staff analysis is not sufficient to support a change to the round-off procedure. Making attainment determinations on the basis of the implied precision of the third decimal place would be inappropriate.

N. While EPA Should Retain the Current Standard, the Form of the Standard Should Be Changed to the Fifth Highest Daily Maximum 8-Hour Average Concentration.

While the evidence suggests that the current standard should be retained, EPA should revise the form of the standard to the fifth highest daily maximum 8-hour average concentration. If, as expected, ozone levels attributed to U.S. anthropogenic emissions continue to fall while background levels of ozone from biogenic sources and international sources continue to rise, more areas will be classified as nonattainment due to meteorological variations. A more stable statistic would aid states in their planning efforts and provide regulated entities greater certainty as to the control requirements they must meet. Changing the form of the standard to the fifth highest daily maximum 8-hour average concentration better comports with the health risks, which increase with significance with frequency of exposure. The likelihood of any adverse effect warranting protection under the Act with only two exceedances per year of the current standard is minimal.

[147] 2007 Langstaff Memorandum at 11 (citing CD at 2-25 to 2-26).
[148] 72 Fed. Reg. at 37,874.
[149] 2007 Langstaff Memorandum, Table 2 at 11-12.

II. Comments on EPA's Proposed Secondary Standard.

The EPA proposal[150] indicates that the Administrator would revise the current 8-hour secondary standard with one of two options. The first would be to promulgate a cumulative, seasonal standard expressed as an index of the annual sum of weighted hourly concentrations cumulated over the 12-hour period from 8:00 a.m. to 8:00 p.m, during the 3-month period in the ozone season with the highest index values. This index is referred to as W126, and it would be set within the range of 7 to 21 ppm-hours. The second option would be to make the secondary standard identical to the proposed primary 8-hour standard. EPA is also soliciting comments on an alternative approach that would establish a suite of secondary standards that would contain different ambient levels, with each standard set at a level requisite to protect public welfare based on the variation in plant effect, use and/or location. Under this approach, the specific standard applicable would depend on the classification of areas according to intended use and on the nature and sensitivity of the plant species in that area.

The Alliance believes that there is no evidence suggesting that the current secondary standard should be revised. The existing standard is requisite to protect the public welfare, and should not be altered.

A. A Careful Review of the Existing Scientific Evidence Demonstrates That the Existing Secondary Standard is Requisite to Protect Public Welfare.

1. All the issues and problems that led the Administrator in 1997 to reject the option of a seasonal ozone metric are still relevant.

In the 1996 Federal Register notice proposing revised ozone standards, the Administrator recognized many limitations and uncertainties in the available data and analyses regarding secondary standards. The proposal acknowledged that "the selection of a single averaging time for a national standard will of necessity be a compromise…" and "specifying the form of a seasonal exposure index to correspond to the relationship between vegetation response and O_3 exposure is complicated by the many biological variables that influence the uptake of O_3 by the plant and plant responses to such uptake."[151]

The 1996 proposal also acknowledged that "[b]ecause of a lack of monitoring data, national air quality typical of agricultural crop growing areas has not been characterized."[152] Some of the most important caveats and uncertainties concerning the exposure and risk assessments for crop yield that were listed in the proposal included "[e]xtrapolating from exposure-response functions generated in open-top chambers to ambient conditions,…the lack of a performance evaluation of the national air quality extrapolation,…the methodology to adjust modeled air quality to reflect attainment of various alternative standard options,… and inherent

[150] 72 Fed. Reg. at 37,818.
[151] 61 Fed. Reg. 65,716, 65,738 (Dec. 13, 1996).
[152] 61 Fed. Reg. at 65,740.

uncertainties in models to estimate economic values associated with attainment of alternative standards."[153]

Discussing the quantitative analyses carried out at that time, the proposal indicated that "the Administrator acknowledges the significant uncertainties in these analyses and recognizes that these benefits should be regarded as rough approximations."[154] Because of the lack of new data or substantive improvements in the risk assessment, these same issues remain today, contributing a similar degree of uncertainty, as was the case in the prior review.

The 1996 CASAC Panel did not reach consensus on key issues regarding protection of vegetation. Of the four Panel members with expertise in ozone vegetation effects, two indicated that the open-top chamber experiments produced results that overestimated the effects of ozone on plant yield; the other two did not agree with there being a positive bias.[155] With regard to the seasonal cumulative metric under consideration at that time, SUM06, two of the experts thought the cumulative form was appropriate but they disagreed as to the appropriate level. The other two experts were uncomfortable with a SUM06 form because they felt it lacked a biological basis.[156]

The main reasons given for the lack of agreement were lack of sufficient rural ozone data and the lack of relevant plant exposure studies. The Panel concluded that "[f]ive years from now, if we do not have the results of research coupling ozone air quality and plant biology under conditions more representative of ambient field conditions, to avoid the shortcomings of the open-top chamber experiments, then we will continue to be hampered by our inability to come to consensus on the levels of air quality that are protective of vegetation and ecosystems at the most reasonable cost."[157]

Ten years have now elapsed, and the same concentration-response functions from the open-top chamber studies of the 1980s are still the only viable data to use to estimate crop loss and the called-for increase in rural ozone monitoring has not taken place. The 1996 CASAC Panel agreed that the estimates of crop loss at that time were highly uncertain.[158]

Although the current CASAC panel recommends a stringent cumulative secondary standard, there is little in the new data that reduces the uncertainty recognized in 1996. In fact, the current CASAC panel's recommendations for the appropriate range of the standard come from a 1997 workshop on secondary ozone standards.[159] If the workshop recommendations are to be used for standard setting, then the studies underlying their basis, as well as the method for reaching the recommended values, should be stated in more detail than in the cited workshop

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[153] 61 Fed. Reg. at 65,740.
[154] 61 Fed. Reg. at 65,743.
[155] G. Wolff, CASAC Letter, EPA-SAB-CASAC-LTR-96-006, Apr. 4, 1996 at 2 ("1996 CASAC Letter").
[156] 1996 CASAC Letter at 3-4.
[157] CASAC Letter 96-006, Apr. 4, 1996, at 5.
[158] 1996 CASAC Letter at 3.
[159] *See* W. Heck and E. Cowling, *The need for a long-term cumulative secondary ozone standard – an ecological perspective*, Environmental Manager, pp. 23-33, January 1997.

summary. The basis for establishing a standard should be transparent and reproducible. One of the current CASAC Panel points out that the state of the science regarding vegetation effects has not changed very much since the last review and, in a minority view, indicates that the case has not been made for a biological basis for either the SUM06 or W126 metric.[160]

2. There are problems, flaws and concerns with the SP analysis and vegetation risk assessment that preclude the establishment of a separate secondary standard to protect vegetation.

a. Extremes of background ozone concentrations were not considered.

The same analyses, arguments, and data that apply to consideration of background for the primary standard also apply to the secondary standard. As discussed above, the global model EPA relied on is not capable of resolving the phenomena that contribute to attainment or nonattainment of secondary ozone standards throughout the country. In fact, even a more refined, higher-resolution model, the Community Multi-Scale Air Quality (CMAQ) modeling system , which EPA evaluated when developing an "as-is" W126 exposure surface performed extremely poorly in predicting the observed 2001 SUM06 and W126 in the west. When the model was used to predict the SUM06 values at 300 western locations, it had over 300 percent normalized mean error and almost a 300 percent normalized mean bias. This means that the sources and processes that contribute to substantial levels of both SUM06 and W126 in rural and remote areas in the Western U.S. are not well-captured in state-of-the-art chemical transport models. Since CMAQ was developed to focus on the impact of U.S. man-made pollution sources, the poor prediction performance suggests other sources and processes are responsible for a substantial portion of the elevated SUM06 and W126 levels observed in rural and remote western areas.

b. The pattern of current W126 levels suggests contributions from both man-made emissions and uncontrollable background.

The map of "as-is" W126 levels included as Figure 7-6 at 7-28 of the SP shows W126 levels exceeding 7 ppm-hr throughout many areas of the country. Although some areas of elevated W126 occur in and downwind of major man-made source areas, other elevated W126 areas occur in less populous areas including: Utah, New Mexico, Wyoming, Kansas and Oklahoma, states where population and density of man-made emissions are only a small fraction of that in the Eastern U.S.

The overall pattern of W126 suggests both a contribution from U.S. man-made emissions and a background component that could involve biogenic emissions, agricultural emissions, long-range transport from non-U.S. sources and a stratospheric component. In particular, W126 levels greater than 7 ppm-hr. exist in many locations throughout the country, including many rural and remote western sites well-removed from anthropogenic emission source areas. These

[160] 2007 CASAC Letter at C-17- C-18.

levels could be explained in part by NOx emissions from soils due to microbial processes. Jaegle, *et al.*[161], using satellite observations of NO_2 columns, found that soil NOx emissions were twice as high as assumed in the GEOS-CHEM model. This will result in a misallocation of ozone between man-made and natural sources. The presence of a large soil NOx source in the Great Plains also may help explain the discrepancy between the locations of elevated SUM06 and W126 and the major populated areas of the country.

c. EPA and CASAC relied on an inadequate model in recommending secondary standards that fall within the range of uncontrollable background.

EPA and CASAC assume, incorrectly, based on the Fiore *et al.* 2003 global modeling analysis, that SUM06 or W126 is not confounded by background ozone. However, substantial levels of both SUM06 and W126 in rural and remote areas are not obviously related to known man-made source areas, and are likely due to such factors as soil NOx emissions. As noted above, both models and observations must be used when determining the extent of uncontrollable ozone. Otherwise, a secondary ozone standard may be implemented that is unattainable in reality.

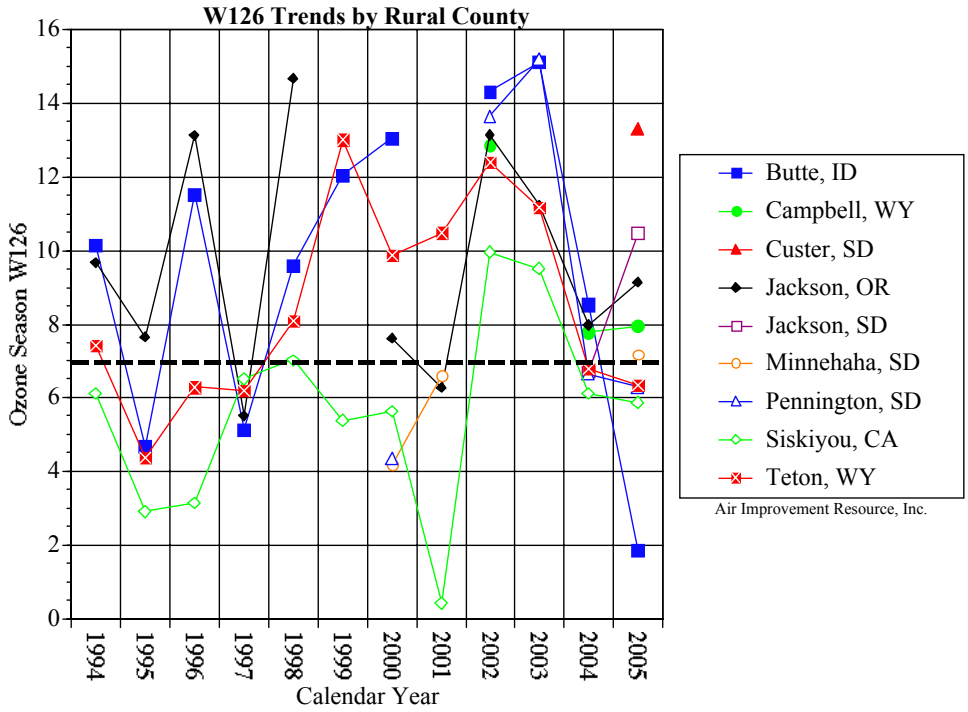
In Figure 3, longer-term W126 data are plotted for a series of sites across the Western U.S. from the California/Oregon border across Idaho, Wyoming and into South Dakota. All of these sites are substantially removed from major emission sources and have W126 values generally in the range of 0 to 20. Note also that there are wide variations from year-to-year. There are several important implications from this data.

i. Even sites removed from obvious source areas have a substantial number of hours annually at or above 0.06 ppm, often on the order of 100 to 200 hours per year. Importantly, all these sites have 8-hour design values of 0.06 or greater.

ii. The W126 metric varies substantially from year-to-year even in rural and remote areas lacking any obvious influence from anthropogenic sources. In addition, in 2001, the year used by EPA to model "as-is" W126, it appears to be an anomaly compared to all the other years. Thus, the ranges recommended for a W126 standard are even more stringent and more likely confounded by background than apparent at first glance. With such large variations, if a W126 or other cumulative standard is set using only one year's data, areas could bounce in and out of attainment, causing havoc with implementation planning efforts. Even three years of data does not appear to provide a stable statistic for implementation planning.

[161] L. Jaegle, *et al.* (2005) at 130, 407-423.

Figure 3



d. There is no new data on vegetation effects suitable for establishing secondary standards.

The vegetation risk assessment utilizes the crop yield loss and seedling growth data used in the previous review. Due to the lack of new data, the vegetation risk assessment does not materially reduce the major uncertainties that were acknowledged in the 1996/1997 review.

e. The choice of an appropriate ozone metric to protect vegetation is difficult.

Although cumulative ozone metrics offer advantages over the form of the primary standard to protect vegetation, the limitations and simplifications involved with cumulative ozone metrics are still of concern. For example, the simplifying assumptions that plant exposure to ozone equals plant response, and that there is a uniform threshold or response function for plant injury are both known to be incorrect. In spite of these issues, EPA staff and CASAC recommend a seasonal cumulative secondary ozone standard. Although more biologically-based flux-based models offer promise, there are insufficient data to apply them at present. The decision to ask for comments on establishing a suite of secondary standards is an implicit acknowledgement of the problems with a national seasonal cumulative standard. However, there is insufficient data to establish a suite of standards that would vary with vegetation type and/or location and it is not clear how such a suite of standards could be implemented under the current CAA.

f. The European experience with cumulative standards has been disappointing.

While both EPA staff and CASAC recommend a cumulative ozone metric, the European experience with cumulative ozone metrics has been disappointing. In a series of recent papers,[1] European researchers have acknowledged that the AOT40[2] critical level adopted in Europe has proven to be unrealistic and unattainable and have called for revisions to the approach. In addition, it has been reported that visible symptoms at 83 sites in five countries had no relation to the cumulative exposure metric and that crown defoliation of beech was only related to ozone exposure at very high exposure levels, seven times the European critical level for forests.

In fact, in the European approach, the cumulative seasonal metric was chosen as a first step that evaluated the potential risk to vegetation, while recognizing that it does not take into account environmental factors that modify ozone uptake, plant defense mechanisms, or differences in ozone tolerance between species. The Europeans are now working on their second level approach which will be flux-based.

g. The analysis used to discriminate between cumulative standards and the current 8-hour standard is flawed.

A major consideration in the choice of a seasonal standard was the assessment of the extent to which an 8-hour standard would provide protection for vegetation. The assessment involved a comparison of the "as-is" W126 exposure (Figure 7-6 of the SP) with the W126 exposure rolling back 8-hour ozone concentrations to meet the current standard (Figure 7-7 at 7-30 of the SP). This comparison is misleading, because the rollback approach assumes that there will be no reduction in ozone at sites that currently attain the 8-hour standard. In reality, there are many national and regional emission reduction programs that will reduce man-made ozone throughout the country, and measures adopted through the SIP process that will also reduce ozone in current attainment as well as nonattainment areas.

The Agency is fully aware that the rollback approach underestimates the benefits of attaining the alternative standards. The RIA used the monitor rollback approach for some of its calculations, and the Agency acknowledges that the approach understates the benefits that would occur due to implementation of actual controls because controls implemented to reduce ozone concentrations at the highest monitor will result in reductions in ozone at attaining monitors and other nonattaining monitors.[3] If the rollback approach understates the benefits of alternative standards it also overstates the risk remaining after just meeting the standard.

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[1] *See* M. Ferretti, F. Bussotti, V. Calatayud, M. Schaub, N. Krauchi, B. Petriccione, G. Sanchez-Pena, M. Sanz and E. Ulrich, *Ozone and forests in South-West Europe – What have we learned?* Environmental Pollution, 145, 652-655 (2007), that summarizes the findings from monitoring of forest conditions and ozone at 83 sites in France, Italy, Luxembourg, Spain and Switzerland over the years 2000-2002.

[2] The AOT40 metric is the seasonal accumulated dose of the hourly concentrations above a threshold of 0.040 ppm.

[3] RIA at 6-99, 8-15

The form of the current 8-hour standard, while not designed to protect vegetation, does focus control on the precursors of man-made ozone and consequently will reduce the overall level of man-made ozone throughout the country. EPA's analysis to date does not evaluate the impact of the nation's ozone control program on W126 in a scientifically sound manner. Before a decision regarding the protectiveness of an 8-hour standard is made, a realistic evaluation of the ozone reductions throughout the country must be made using state-of-the-art photochemical models. The draft RIA did not include such an analysis.

In addition, the EPA analysis has focused on determining if the proposed seasonal air quality targets could be exceeded in areas where the 8-hour standard is met. Starting with the basics, however, an analysis of the exposure-response data to determine if effects are observed for exposures which do not exceed the 8-hour standard has not been presented. The ecological effects data are provided generally as seasonal means or cumulative (SUM06, W126) values, which may include multiple exceedances of the 8-hour standard. This is particularly true for the crop studies that form the basis of the EPA and CASAC recommendations, which have multiple hourly peaks > 0.10 ppm.

h. The estimates of crop loss are substantially lower than those of the previous review.

The SP acknowledges that the estimates of crop loss for the species evaluated are substantially lower than in the previous review. As a result, the economic benefits associated with attaining various alternative standards have been substantially lowered from previous ozone yield loss studies.[4] It is now widely accepted that ozone concentrations at the "standard measurement height" are not the same as the ozone exposures at plant height. When this factor was corrected in the vegetation risk assessment, the SUM06 and W126 exposures at plant height were approximately half those at measurement height.

i. Studies of foliar injury do not support a cumulative metric.

The map of locations where foliar injury was present and absent in 2001 (Figure 7-19 at 7-62 of the SP) shows a patchwork of responses that, in many cases, are not obviously correlated to major areas of man-made precursor emission. Although foliar injury is observed in areas that meet the current 8-hour standard, there are also high and low SUM06 areas that experience foliar injury and areas that do not. Similarly, the European experience indicates that there was little or no relation between a cumulative metric, AOT40, and foliar injury at 83 sites in five countries.

At a site in the Northeast Corridor of the U.S. where ozone levels are elevated, Davis and Orendovici[5] report that there was no readily apparent relationship between SUM06 ozone levels and incidence of ozone stipple. SUM06, by itself, was not a reliable predictor of ozone foliar injury, but when included with N100 (the number of hours of 0.100 ppm or higher) and drought stress, a cumulative ozone metric such as W126 did have substantial predictive power.

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[4] SP at 7-52.

[5] D. Davis and T. Orendovici, *Incidence of ozone symptoms on vegetation within a National Wildlife Refuge in New Jersey*, Environmental Pollution, 143, 555-564, 2006.

Although the SP mentions the possible use of N100 along with a cumulative ozone metric, it took no position on the question. Clearly, this is an important issue that should be evaluated fully by EPA.

j. CASAC made its recommendations based on the draft SP that contained errors overstating the magnitude of vegetation effects.

There were major errors in the translation of the results of the vegetation risk assessment in the Second Draft SP, overstating the magnitude of crop loss from ozone. For example, the table summarizing the results of the crop loss risk assessment in the appendices to the second draft SP (Table 7E-4) reported the maximum yield loss for various crops (8 crops and 7 fruits and vegetables) but labeled the numbers as the median yield loss. This grossly overstated both the yield loss from current ozone levels, as well as the yield gain from attaining any of the alternative secondary standards under consideration. While this was corrected in the final SP, CASAC made its recommendations concerning the secondary standard based on the draft text that contained numerous errors overstating the magnitude of vegetation effects. Moreover, the SP still only discusses the maximum yield loss figures. The actual distribution of yield loss/gain can only be found in Section 5.4.1 and Appendix G of the TSD,[6] which indicates that the mean and median yield gains from attaining any of the alternatives under consideration are quite small – the order of 1 percent or less for even the most stringent standards. For example, the mean yield gain for cotton (compared to the 2001 "as-is" baseline) from attaining a 13 ppm-hr W126 standard is 0.19 percent and the mean yield gain for soybeans from attaining a 13 ppm-hr W126 standard is 0.14 percent. In contrast, the second draft SP had indicated that there would be the orders of a 7 % mean yield gain for cotton and 2.5 % for soybeans.

k. The recommended range for a cumulative standard needs to be adjusted upward.

The CASAC suggestions for the range of a SUM06 or W126 standard come from a consensus-building workshop held in 1997. The EPA staff's recommended range also considered the results of the 1997 Review and risk assessment. Since neither of these sources considered the difference between the ozone monitored in the network and the ozone exposure at plant height, these ranges should be re-evaluated accordingly.

If ozone monitoring is continued under the current guidelines, the ranges for a seasonal secondary standard would have to be adjusted upward. Based on the reduction in ozone exposure at plant height of a factor of 2 compared to that at measurement height, the range should be increased by roughly a factor of 2. Neither CASAC nor EPA took this into account in making their recommendations concerning the level of the secondary standard.

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[6] J. Lehrer, M. Bacou, B. Blankespoor, D. McCubbin, J. Sacks, C. Taylor and D. Weinstein, *Technical Report on Ozone Exposure, Risk, and Impact Assessment for Vegetation*, EPA 452/R-07-002, Jan. 2007, Appendix G.

l. The tools needed to implement a seasonal ozone standard are not in place.

The placement of current ozone monitors is not appropriate for evaluating vegetation exposure in terms of either spatial coverage or monitor height. Therefore, a new monitoring network must be designed and implemented before designations of attainment or nonattainment occur. Based on work-to-date, a model suitable for developing control strategies for a cumulative seasonal standard, particularly in the Western U.S., is not available. In addition, major improvements would be needed in grided emission inventories throughout the U.S. particularly for agricultural emissions. Therefore, the tools needed to implement a seasonal ozone standard are not in place.

When the timeframe required implementing a new monitoring network, to collect data for several years, and to develop and approve an implementation plan is considered, the next ozone review will have been completed before implementation plans will be in place. Ozone reviews are required every five years and EPA recently changed the review process dramatically to meet the statutory requirement. As an example of the timeframe needed to implement a standard when a new monitoring network is required, the $PM_{2.5}$ standards were set in 1997 and the SIPs are due to EPA in 2007.

All this assumes that tools are available when needed, sources responsible for the nonattainment can be identified, and the standard is attainable in the sense that reduction or elimination of the identified sources is at least theoretically possible. To the extent that any of these steps are not possible, the setting of a seasonal secondary standard will not be fruitful and will not aid materially in the protection of vegetation.

III. Comments on EPA's Regulatory Impact Analysis for the Agency's Proposal to Revise the National Ambient Air Quality Standards for Ozone.

The Regulatory Impact Analysis (RIA) EPA issued on August 2, 2007 estimates potential incremental benefits and costs of reaching a standard in the year 2020 in the range EPA proposed (0.070 to 0.075 ppm, and at two other levels on which the Agency is taking comment (0.065 and 0.079 ppm).

The RIA concludes that the annual net benefits for implementation of the proposed standards (*i.e.,* 0.070 to 0.075 ppm) in 2020 range between -$20 billion and +$23 billion. Because of the high degree of uncertainty in these calculations, EPA cannot estimate whether costs will outweigh benefits, or *vice versa*.[168] An analysis of the RIA suggests that many of the potential benefits may not be realized while the estimated cost is likely to be significantly greater than the estimates suggest. The RIA shows that attainment of any alternative ozone standard is going to require massive new control programs that will affect most parts of the country and many sectors of life. After the application of every known control measure, substantial areas of the country still fail to attain the current standard.

A. EPA's Alleged Benefits From Lowering the Ozone NAAQS Are Not Likely to Occur.

Most of the benefits estimated in the RIA are due to the avoidance of premature mortality. For the upper-end estimate of the benefits, EPA assumes that premature mortality is linked causally to both ozone and $PM_{2.5}$. The Alliance takes issue with both of these causal assumptions. For the lower-end estimate, EPA assumes premature mortality is only linked to $PM_{2.5}$ and not to ozone. Because emissions of NO_x will need to be reduced to meet any of the proposed alternative ozone standards, ambient concentrations of fine particulate nitrate, a component of $PM_{2.5}$, will also be reduced. In fact, estimated benefits from reduced $PM_{2.5}$ pollution make up between 13 and 99 percent of the estimated benefits of meeting the alternative standards.

Since only observational studies are used in the RIA, judgments are necessary in selecting the health endpoints and concentration-response functions to include from a wide range of available associations in the literature. As indicated above, there are major issues of model selection and potential biases involved in interpreting the air pollution epidemiologic literature. The current practice of using central station monitoring data, central station weather data, and available health statistics yields many weak positive associations for various pollutants in time-series analyses. However, the full range of associations in the literature is very wide, with both positive and negative associations, and it is known that the methodology is subject to problems of measurement error and exposure misclassification, as well as severe collinearity between weather and pollution variables. When the uncertainty due to model selection issues is added and the potential biases are considered, the interpretation of a subset of positive findings as

[168] RIA at 8-10.

causal becomes problematic. Therefore, the use of epidemiological studies for health effect benefit estimation is highly uncertain.

The most important uncertainty relates to the assumption of causality. The second most important uncertainty is due to the assumption of no threshold for most of the evaluated endpoints. The SP indicates that the bulk of estimated premature mortality accrue at 24-hour concentrations below 0.04 ppm which is close to the background assumed in the previous review. The CD and SP acknowledge that only a few ozone studies have examined the shape of the concentration-response function. The Special Panel of the Health Effects Institute Review Committee recently raised several cautions in interpreting epidemiological concentration-response results. They point out[169] that measurement error could obscure any threshold that might exist and note that city-specific concentration-response curves (for particulate matter, in this instance) exhibited a variety of shapes. They also caution that the standard approach of minimizing the Akaike Information Criterion may not be appropriate for choosing between models. The HEI Panel finally cautions that lack of evidence against a linear model should not be confused with evidence in favor of it. CASAC has raised similar concerns regarding the inability of time-series studies to identify the presence or absence of thresholds. The use of a linear ozone effect model as the default should not be accepted until it is demonstrated with controlled studies that low doses within the range of background are capable of eliciting significant biological effects.

There are specific concerns with each of the health endpoints included in the RIA, as discussed in detail in the following sections.

1. EPA's estimates of acute mortality rely on weak epidemiology evidence and incorrectly assume causality.

The RIA acknowledges considerable uncertainty in the ozone/mortality relationship including the possibility that the ozone/mortality association may not be causal. However, the RIA inappropriately goes on to include estimates of health benefits that presume acute mortality from exposure to ozone. These estimates are based on the Bell *et al.* 2004 NMMAPS analysis, and the three meta-analyses sponsored by EPA in 2005. Since the proposed rule acknowledges that the three meta-analyses are subject to publication bias, those results should not be included in the final RIA.

There are several reasons why even the NMMAPS results are not consistent with a causal relationship and should not be used as the basis of a health benefits analysis. The RIA notes CASAC's concern that time-series analysis is a blunt tool that implicates all pollutants and it notes EPA's request that the National Academy of Sciences (NAS) evaluate the evidence.

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[169] *See* Commentary by Health Review Committee in M. Daniels, F. Dominici, S. Zeger and J. Samet, *National Morbidity, Mortality, and Air Pollution Study, Part III: Concentration-Response Curves and Thresholds for the 20 Largest U.S. Cities*, Health Effects Institute Research Report Number 94, Part III, May 2004.

The CD and SP acknowledge that there is a lack of consistent effect on respiratory mortality. Although this is blamed on a lack of statistical power, CASAC questions this interpretation.[170] In addition, the Anderson *et al.* 2004 study reported no association of respiratory mortality with ozone in a meta-analysis of European studies. There were no statistically significant associations of ozone with respiratory mortality in the 12 studies evaluated. The overall risk was 1.000 per 10 $\mu g/m^3$ increase in ozone. While there was a weak signal of an association of ozone with cardiovascular mortality, the finding of a cardiovascular but not respiratory mortality signal from ozone in single-pollutant models is hard to explain as a causal relation. It is not coherent with the staff conclusion that the ozone signal in hospital admissions is with respiratory not cardiovascular admissions. In the Anderson study, the combined association of ozone with respiratory hospital admissions was not statistically significant for the five cities available for analysis.

Anderson discusses the issue of publication bias acknowledging that it is a common and possibly universal problem in our research culture. Furthermore, they note that there are particular reasons why it might occur in time-series studies. These include the large number of associations evaluated and the possibility for a selection bias involving the results included in a paper. They also mention a lag selection bias that can be overcome with multi-city studies such as NMMAPS. They further point out that publication bias can result in false conclusions being drawn in the hazard identification stage of a risk assessment and result in an inflation of the magnitude of the health impacts. They also recognize that the methods of detecting publication bias are not without problems.

In an earlier meta-analysis, Levy *et al.* 2001 describe their result as providing weak epidemiologic support for ozone mortality and as potentially biased because it excludes studies that found ozone to be insignificant. After listing a number of such studies and noting that nearly all the studies found ozone not to be a predictor of mortality including all studies in warmer climates, Levy *et al.* concludes this information must "…give one pause in using our pooled estimate as a representative measure of ozone mortality risks." Levy *et al.* also discuss the difficulties of separating temperature or weather and ozone effects, cautioning that "possible confounding by weather leaves lingering doubt about whether the ozone findings are causal, an issue that would be difficult to resolve epidemiologically."

Although the RIA relies on the Bayesian adjusted summer ozone/mortality associations from the Bell *et al.* 2004 NMMAPS analysis, the individual-city data shown in Figure 7-17 of the CD demonstrate a much wider range of associations. Such a wide range with many negative associations and few statistically significant positive associations is not consistent with a causal ozone effect on mortality. There is another NMMAPS analysis that also raises important concerns. The NMMAPS re-analysis[171] reported a small positive combined association for ozone with mortality in the summer, but a small negative combined association in the winter at lag 0. The lag 0 results were stronger than the lag 1 and lag 2 results. However, an association

[170] October 2006 CASAC Letter at 10.

[171] F. Dominici, A. McDermott, M. Daniels, S. Zeger and J. Samet, *Revised analyses of the National Morbidity, Mortality, and Air Pollution Study: Mortality among residents of 90 cities,* J. Toxicology and Environmental Health, Part A, 68, 1071-1092 (2005).

of ozone at lag 0 may be problematic since the ozone peaks in the afternoon and mortality is distributed throughout the day, so that a lag 0 association potentially runs afoul of the temporality requirement that the cause precede the effect.

The wide range of individual city results in NMMAPS, the overall negative winter association demonstrated in NMMAPS, and the lack of a significant summer combined association for the 90 largest U.S. cities in the NMMAPS multi-pollutant models all erode the RIA case for causality. The only reported NMMAPS ozone results in multi-pollutant models were for the summer. In each case, when other pollutants were included, the ozone association was reduced and became non-significant. If the ozone associations are not robust to inclusion of other pollutants in the summer when there was a combined small positive association in single pollutant models, it is very unlikely that they could be causal.

2. The RIA incorrectly bases benefit estimates from reduced hospital admissions for respiratory disease on selective studies.

The RIA inappropriately uses selected positive associations of ozone with respiratory hospital admissions from the literature. As discussed in the comments above concerning this endpoint, there is less consistency in the hospital admissions and emergency department ozone/association data than EPA acknowledges. As shown in the NMMAPS mortality analyses, the range of individual city results in multi-city analyses with a consistent methodology is very large (with both positive and negative associations) and not biologically plausible. The wide range of positive and negative associations in Ito 2003 also indicates that assuming that selected individual city positive associations are real health effects overstates the situation. The time-series studies in the literature can be used to implicate many different air pollutants as potentially causing respiratory hospital admissions and emergency department visits, but as Anderson *et al.* 1998 point out there is lack of consistency in the pollutant responsible or the specific respiratory endpoint affected.

This conclusion is consistent with the emerging realization in the scientific community that there is increasing evidence for caution in interpreting weak air pollution associations. In order to fully consider the range of ozone/hospital admissions associations in the literature, the presence of negative studies and studies with negative associations must be acknowledged and the lower limit of estimated zero should be zero. In fact, the RIA acknowledges that the confidence limits for the pooled estimate for asthma-related emergency room visits includes negative values, so the lower limit for this endpoint is indeed zero.[172]

[172] RIA at 6-16.

3. The data on school absences are weaker than noted in the proposed rule and are not reliable for purposes of estimating benefits.

The RIA uses associations between school absences and ozone from Chen *et al.* 2000 and Gilliland *et al.* 2001. However, both the SP and CD indicate that further replication is needed before firm conclusions can be drawn regarding the presence of ozone effects on school absences. The Gilliland *et al.* study of school absences reported a large effect but there is a question of residual seasonal confounding in the study. Furthermore, Gilliland *et al.* report a substantially larger effect in low ozone communities *versus* the effect in high ozone communities, a highly counterintuitive result that necessitates additional explanation. Potential confounding by bioaerosols was not evaluated in the study. Thus, it would be particularly important to replicate the study in another cohort and over all seasons.

The many issues with model selection that have arisen in recent years raise additional concerns that the result may not be robust. Specifically, the final CD discusses three analyses of the school absence data from the Southern California Children's Health Study (Gilliland *et al.* 2001, Berhane and Thomas 2002, and Rondeau *et al.* 2005) that used different statistical approaches and assumptions and came to very different conclusions concerning the presence and magnitude of an ozone effect <u>using the same data set</u>. This is an indication that the choice of the statistical approach used to analyze the data can change the findings dramatically. Overall, the evidence for school absence associations with ozone is weaker than noted in the proposed rule.

The large reported ozone effect on respiratory absences in Southern California is disproportional to the lack of substantial effects on respiratory symptoms seen in school children residing in the Basin. There are several studies of respiratory symptoms in California that failed to detect symptomatic effects. These include Delfino *et al.* 1996, Delfino *et al.* 1997b, Linn *et al.* 1996 and Ostro *et al.* 2001. There is a particularly important study of asthmatic, wheezy and healthy children in Southern California that is not considered by EPA. The HEI study by Avol *et al.*[173] evaluated potential acute respiratory effects of ambient ozone in the spring and summer. About 200, 10- to 12-year old children from Lancaster, San Dimas, Upland, Mira Loma, Riverside and Lake Arrowhead were studied in the spring and late summer of 1994 with monitored hourly ozone levels exceeding the federal 1-hour standard in each season, and with peak hourly levels up to 0.20 ppm in the summer period.

Furthermore, there was no consistent or clear role of ozone in producing changes in reported symptoms or medication use. There was also no discernible pattern of diurnal lung function changes by season, ozone level, or health group. The authors note that failure to detect clear evidence of acute pulmonary effects in the three groups may be due to insufficient elevation of ozone levels during the study, although ozone concentrations exceeded 0.08 ppm during the study. The authors note that children in their study on average did not spend a comparable amount of time outdoors or exercising at high levels to the time used in the chamber studies.

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[173] E. Avol, W. Navidi, E. Rappaport and J. Peters, *Acute effects of ambient ozone on asthmatics, wheezy, and healthy children*, Health Effects Institute Report Number 82, 1998.

Finally, the lack of a substantial respiratory symptom signal in several California studies is inconsistent with the large effect on school absences reported by Gilliland *et al.* This difference needs to be acknowledged and discussed in the RIA.

4. EPA should include a lower bound of zero for the benefits of minor restricted activity days.

The RIA uses an association of 2-week average ozone with minor restricted activity days from Ostro and Rothschild 1989 for this endpoint. The authors note that the association with ozone was highly variable but statistically significant. They also note that there was no association with respiratory-related restricted activity days (RRADs). Minor Restricted Activity Days (MRADs) are days when people reduce their activity but do not miss work or school.

Levy *et al.* 2001 point out some statistical issues with the study. Levy *et al.* raise concerns that the study may not properly measure an independent ozone effect. Levy *et al.* note that of the six years in the study, 1976-1981, the regression coefficient was significantly negative in 1977 and 1981. Although the combined result was significantly positive, Levy *et al.* caution that the magnitude of the ozone impact on MRAD should be considered somewhat uncertain given that "only one older study with statistical issues is available."

Since ozone was not significantly associated for any year with RRAD, it is difficult to discern what the actual health effect on a MRAD might be and how it could be caused by ozone. Given these issues and the presence of negative as well as positive study results in different years, the lower bound estimate should be zero.

5. Given questions of PM speciation and other uncertainties, the lower bound of the ancillary $PM_{2.5}$ benefits should also be zero.

The RIA acknowledges that ancillary benefits from reduced $PM_{2.5}$ pollution make up between 13 and 99 percent of the estimated benefits of meeting the alternative standards. (RIA at 8-10) Because emissions of NO_x will need to be reduced to meet any of the proposed alternative ozone standards, ambient concentrations of fine particulate nitrate, a component of $PM_{2.5}$, will also be reduced. Since the baseline for the RIA assumes sufficient controls to attain the new $PM_{2.5}$ standards, there are several reasons why reduced nitrate concentrations, especially at concentrations below the new PM standards, would not be expected to impact mortality.

- The toxicology of nitrate and other PM components indicates that nitrate is among the least toxic PM components. PM air pollution is a mixture of many different kinds of particles

that vary by three orders of magnitude in toxicity per unit mass (AICGH, 2006)[174] and nitrate is among the least toxic of these components. (Schlesinger and Cassee, 2003).[175]

- Nitrate exposures of the frail population are substantially below that measured in ambient air. Ammonium nitrate is known to decompose to gaseous nitric acid and ammonia as it penetrates indoors where people spend 90 percent of their time with the nitric acid formed being lost on surfaces.[176] Numerous studies report substantially reduced nitrate concentrations indoors compared to ambient concentrations with lower indoor/outdoor (I/O) ratios for nitrate than for other ambient PM components such as sulfate.

- Spatial and temporal distribution of nitrate is opposite to the acute health effects signal from PM. Ambient nitrate is highest in winter and higher in the west than in the east. A seasonal NMMAPS analysis is available (Peng *et al.* 2005)[177] with updated mortality data from 1987-2000 in 100 cities. Summer was the only season for which the combined effect of PM_{10} was statistically significant. An analysis by geographic regions showed a strong seasonal pattern in the northeast with a peak in the summer. The authors note several possible explanations. One obvious hypothesis is that the most toxic particles have a spring/summer maximum and are more prevalent in the northeast. The acute PM health effects signal from this large multi-city study is not consistent with nitrate-caused mortality.

- The spatial distribution of nitrate is also not consistent with causation of cardiovascular hospital admissions based on the results of the Dominici *et al.* (2006)[178] study of $PM_{2.5}$ hospital admissions associations for 204 U.S. urban counties. Dominici *et al.* report a clear difference in the combined associations between the eastern and western regions of the country. The combined hospital admissions association is positive for cardiovascular outcomes in the east but negative in the west except for heart failure, which is positive in both areas. However, there is an ample literature of small positive associations of hospital admissions in single pollutant models with a wide range of air pollutants, particularly for heart failure, for which the most consistent associations are reported.[179] The Dominici *et al.* results are not consistent with an effect of either generic $PM_{2.5}$ or nitrate on cardiovascular hospital admissions and, indeed, the authors point out the need to shift the focus of research

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[174] American Conference of Governmental Industrial Hygienists, 2006 Threshold Limit Values and Biological Exposure Indices, Cincinnati, Ohio.

[175] R. Schlesinger and F. Cassee, *Atmospheric secondary inorganic aerosols: The toxicological perspective as a basis for health effects risk assessment*, Inhalation Toxicology, 15, 197-235 (2003).

[176] M. Lunden, T. Thatcher, D. Littlejohn, M. Fischer, T. Kirchstetter, N. Brown, S. Hering and M. Stolzenburg, *Building a Predictive Model of Indoor Concentrations of Outdoor $PM_{2.5}$ in Homes*, Report under U.S. Dept. of Energy Contract No. DE-AC03-76SF00098, prepared by Lawrence Berkeley National Laboratory and Aerosol Dynamics Inc., Berkeley, CA, September 2001.

[177] R. Peng, F. Dominici, R. Pastor-Barriuso, S. Zeger and J. Samet, *Seasonal analyses of air pollution and mortality in 100 U.S. Cities*, Am. J. Epidemiol, 161, 585-594 (2005).

[178] Dominici F., Peng, R., Bell M., Pham.L., McDermott, A., Zeger, S. and Samet, J., Particles*, Air Pollution and Hospital Admissions for Cardiovascular and Respiratory Diseases*, J. American Medical Association, 2006, 295, 1127-1134.

[179] *See* M. Goldberg, *et al.,* Env. Res., 91, 8-20 (2003) which reports similar small associations with congestive heart failure for every pollutant included in the analysis.

to identifying those characteristics of particles that determine their toxicity. The authors also note that their combined result is several-fold lower than other hospital admission associations they cite from the literature, suggesting publication bias in that literature.

The implications of these two large multi-city studies are that the $PM_{2.5}$ associations in the RIA inflate the magnitude of potential benefits from control of generic $PM_{2.5}$ and that the associations should not be applied to estimate the benefits of nitrate reductions.

- Regional differences in the chronic mortality signal are inconsistent with nitrate causing chronic mortality. There is a major difference in the chronic $PM_{2.5}$ mortality signal between the east and west in the data the RIA relies on that is inconsistent with nitrate-causing chronic mortality. The RIA relies on the Pope *et al.* 2002 update of the Pope *et al.* 1995 American Cancer Society Study (ACS) to estimate the ancillary nitrate benefits. However, the lack of a $PM_{2.5}$ association with mortality in western cities in the ACS cohort was noted by EPA (Grant 2001)[180] in a presentation to the CASAC PM Panel. Staff indicated an excess risk from 10 $\mu g/m^3$ $PM_{2.5}$ of +29 percent in the industrial midwest, +25 percent in the southeast, +14 percent in the northeast, and –9 percent in the west (west is a combination of cities in the northwest, southwest, upper midwest, and Southern California NMMAPS geographic regions). The different regional signals and other findings from the HEI re-analysis[181] of the chronic PM health studies all raise questions concerning the interpretation of the chronic $PM_{2.5}$ associations as a universally applicable chronic PM health effect. For the RIA, the lack of a chronic mortality signal in the west, where nitrate is a major component of ambient PM, is not consistent with the assumption that nitrate causes chronic mortality.

- The expert solicitation that the Agency uses in a sensitivity analysis never addressed the experts' views on causality of ammonium nitrate or other specific PM components. Therefore, the sensitivity analysis regarding causality is incomplete, as it applies to ancillary nitrate reduction impacts.

- As noted above, all the estimated ancillary $PM_{2.5}$ benefits from more stringent ozone standards accrue from $PM_{2.5}$ concentrations below the new $PM_{2.5}$ NAAQS that the Administrator decided was protective of public health with an adequate margin of safety.

For all these reasons, the lower bound of ancillary $PM_{2.5}$ benefits due to NOx reductions should be zero.

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[180] Grant, L.; EPA Staff Presentation to CASAC, July 23, 2001; *Key Revisions and Scientific Issues for Second External Review Draft of Air Quality Criteria for Particulate Matter,* Slide 46.

[181] For example, the Krewski *et al.* 2001 re-analysis showed that the increased risk was cardiovascular not respiratory, that one gaseous pollutant, SO_2, had a strong association with mortality, that when SO_2 was included in the model the PM all-cause mortality association was materially reduced and became non-significant, and that the increased mortality was experienced in the portion of the cohort that had a high school education or less.

B. Even Though the Benefits Are Uncertain, the Costs of Meeting a More Stringent Ozone Standard Will Occur and the EPA Analysis Likely Underestimates Those Costs.

In order to conduct the RIA, EPA used a photochemical grid model to model two different scenarios and then used selected grid modeling results to extrapolate and estimate the number of additional tons of precursor control that would be needed for attainment of the alternatives under consideration.

1. The RIA shows many existing nonattainment areas will not attain the current standard with all known control technologies, even when applied to regions outside the nonattainment area.

The first modeling scenario, designated as the 2020 Baseline includes all national emission control programs that are on the books or being proposed and many additional local and regional NO_x and VOC control programs that are thought necessary to bring most of the U.S. in attainment with the existing 8-hour ozone NAAQS of 0.08 ppm. The RIA reports (Figure 3.4 of the RIA) that ten counties would not attain the current standard by 2020 with all these known or available controls. The remaining nonattainment areas include counties in the Houston metropolitan area, the Lake Michigan area, the Northeast Corridor, and the South Coast and San Joaquin Valley areas of California. Although only ten counties would actually experience ozone levels above the current standard in 2020 in this simulation, the areas affected by such nonattainment are much larger. For the baseline, the geographic areas where additional known controls were applied are extensive, including much of the midwest and the Northeast Corridor and all of Texas and California. This baseline analysis also shows that the application of all known controls would result in 82 counties that exceed 0.075 ppm standard, which is the high-end of the range in the proposed rule.

The second model simulation added a hypothetical national control strategy for attaining a 0.070 ppm standard by 2020 to the controls in the baseline case. Although described as applying all known or demonstrated controls, this emission scenario is a mix of adding known controls in additional geographic areas, adding controls assuming that current technologies can be applied to smaller and smaller sources of the same type, and adding hypothetical controls that are extensions of existing technology that EPA assumes will be available and effective in the needed time-frame. After applying the hypothetical "known" controls over most of the Eastern U.S. and throughout five additional western states -- Nevada, Utah, Colorado, Arizona and New Mexico -- the simulation was only able to show partial attainment of the 0.070 ppm goal. In fact, after these hypothetical controls were added to the baseline, nine counties still exceeded the current standard, 50 exceeded 0.075 ppm and 126 exceeded the 0.070 ppm goal as shown in Figure 3.13 of the RIA. No estimates for the number of counties exceeding the 0.060 ppm alternative recommended by many environmental activists were reported.

To summarize, the RIA acknowledges that known and reasonably anticipated controls would likely be insufficient to bring many areas into attainment with either the current or

alternative standards.[182] In other words, the Agency does not know how the current standard can be attained throughout the country.

2. The RIA relies on unknown control technology to demonstrate attainment.

Since the measures in the hypothetical national control strategy were not sufficient to bring much of the country into compliance with the alternative standards, a third emissions scenario for 2020 was developed using unknown technology to further reduce NOx emissions so that all places (except two areas in California) would be in compliance with the alternative standards by 2020. This exercise involved developing estimates of the emissions needed to provide full attainment but it did not involve testing in the grid model whether those emission reductions would, in fact, provide full attainment.

The emission reductions in this scenario were estimated from grid modeling runs of the impact of NOx reductions. A single national estimate of the reduction in NOx and VOC in terms of parts per billion ozone reduced per kiloton of precursor reductions was used together with the extent of remaining nonattainment to estimate that several million additional tons of NOx reductions would provide for attainment of the more stringent alternatives under consideration in the RIA. The cost of full attainment was then estimated based on historical experience with NOx controls in terms of dollars per ton of NOx removed. Even though EPA does not know how these reductions will be achieved, the costs are estimated by extrapolating the cost from known technologies.

3. The RIA correctly characterizes this extrapolation of costs from known to unknown technologies as a highly speculative exercise. Actual costs are likely to be significantly higher.

The RIA characterizes the extrapolation of costs from known to unknown technologies as a highly speculative exercise, [183] and describes the resulting estimates as providing guidance on the general magnitude of costs and benefits not precise predictions. As standards across multiple pollutants with overlapping precursors are tightened, states and affected industries will move further down the list of cost-effective known and available controls, until uncontrollable background and anthropogenic emissions are reached. The nation has had a major national effort to develop and apply controls to reduce ozone and particulate matter emissions for over 40 years. Although much progress has been made, achieving even the current ozone standard throughout the country will require either the invention and application of substantial additional technology or curtailment of economic activity. Thus, the current standard is a very stringent standard.

The estimated costs in the RIA, as large as they are, may significantly underestimate the true costs of the alternative standards. As the RIA notes, the estimated emission reductions are based on generalized precursor ozone relationships while the actual relationships are highly

[182] RIA at 8-11.
[183] RIA at 8-11.

variable across the country.[184] The full attainment emission reductions were restricted to NOx controls, but the remaining ozone problems tend to occur in areas with the greatest population and emissions density (modified by the extent to which the area's meteorology is conducive to ozone formation). These areas are the large metropolitan areas where portions of the domain are VOC-limited and NOx reductions can both increase and decrease ozone formation.[185] Thus, full attainment in these areas may be more difficult than EPA has assumed in the RIA. The generalized precursor relationships were derived from 36 km grid modeling runs which tend to exaggerate the benefits of NOx control and mask the benefits of VOC control.

All the remaining major nonattainment areas have sophisticated ongoing grid modeling efforts supported in many cases with extensive field studies. Some of these areas were not able to develop and submit attainment demonstrations for the current standard by the June 2007 deadline due to modeling issues and the lack of known controls. Others did develop attainment demonstrations that depended on the black box for as yet unidentified controls. The RIA acknowledges that in urban areas with a high population density, ozone is often VOC-limited (RIA at 2-1) and that in these areas NOx reductions can increase ozone. The RIA also notes that between the NOx-limited and VOC-limited extremes there is a transitional region where ozone is relatively insensitive to changes in NOx and VOC. To the extent that the mix of national and local controls results in areas being in the transitional region, where ozone formation tends to maximized, it may take massive reductions in one precursor or the other to come into attainment. Given the large biogenic emissions of both VOC and NOx that need to be included in the modeling, such massive reductions may not be feasible or achievable in some locations. In the final RIA EPA needs to test its full attainment strategies in a nested grid model to insure that attainment of the ozone standard is feasible.

In conclusion, the RIA notes that tightening the ozone standard will incur significant but uncertain costs.[186] The RIA concludes that the hypothetical national strategy to attempt to attain the 0.07 ppm alternative would cost $3.9 billion per year and that the estimated reductions for full attainment of the 0.07 ppm alternative would cost $13 to $26 billion per year. (For the 0.065 ppm standard the estimated national cost, excluding full attainment in California, is $17 to $46 billion per year. No estimate for the 0.060 ppm standard was made. While, as documented above, the benefits of an 0.07 ppm standard (or any of the alternatives) depend on highly questionable assumptions concerning causality, it is, without question, demonstrably clear that achieving such a standard will require massive additional emission reductions costing the order of several tens of billions of dollars per year on top of the already massive national costs of attaining the current standard. The RIA acknowledges that the direct benefits from ozone reduction itself are $190 million per year for the 0.07 ppm alternative if it is assumed that ozone does not cause premature mortality.[187] Since the morbidity effects of ozone in the RIA are

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[184] RIA,, at 8-16.
[185] *See* J. Heuss, D. Kahlbaum and G. Wolff, *Weekday/Weekend Ozone Differences: What Can We Learn from Them?* J. Air & Waste Management Association, 53, 772-778 (2003); G. Yarwood, K. Bonyoung, T. Stoeckneius, and J. Heiken, *Modeling Weekend and Weekday Ozone in Southeast Michigan*, ENVIRON International Corporation Draft Final Report prepared for National Renewable Energy Laboratory, Golden, CO, July 30, 2007.
[186] RIA at 8-16.
[187] RIA at 6-2.

highly exaggerated as demonstrated above, all the alternative standards below the current standard are expected to be highly cost-ineffective.