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Honorable Carol M. Browner
Administrator
U.S. Environmental Protection Agency
401 M Street, SW
Washington, DC 20460

Subject: Clean Air Act Compliance Analysis Council (CAACAC)
Physical Effects Review Subcommittee (CAACACPERS)
Initial Review Pertaining to the Physical Effects
Review Documents for Criteria Air Pollutants and
Methodology for Quantifying Health Effects for the
Clean Air Act (CAA) Section 812 Retrospective
Benefit-Cost Analysis

Dear Ms. Browner:

In response to the Congressional mandate of Section 812, of the CAA and at the request of the Agency's Office of Policy Analysis and Review (OPAR) and the Office of Policy Analysis (OPA), the Clean Air Act Compliance Analysis Council (CAACAC) Physical Effects Review Subcommittee (CAACACPERS) reviewed the draft physical effects documents and the methodology pertaining to quantifying health effects for the Clean Air Act (CAA) Section 812 retrospective benefit-cost analysis.

The CAACACPERS met on November 15 and 16, 1994 to receive briefings from Agency staff and discuss issues with the Agency staff and the public. In addition, the CAACACPERS held a public teleconference on April 12, 1995 and a public meeting on May 18, 1995 to review Agency drafts of additional sections of its overall analysis of the Section 812 Retrospective Benefits and Costs, and to finalize the Council's report. Most of the findings and recommendations contained in this report were conveyed verbally to the Agency staff during the second day of the November 15-16, 1994 meeting.

We note that the Agency is attempting to respond to a Congressional directive to conduct a national assessment. However, it has been recognized from the outset by all the parties involved that the Agency clearly has limited resources

provided to it for this daunting task, and that many of the actions required of the Agency go beyond the capabilities of current state-of-the art assessments. Further, while significant human health, human welfare, and ecological benefits result from controlling many air pollutants, it is very difficult to achieve a credible, much less a thorough and unambiguous evaluation, of the human health, human welfare, and ecological benefits and costs without a sustained program over the long-term. In our view, it is clear that the major commitment necessary to make any significant progress in this area has not yet been made.

Congress and the Agency have to ask themselves whether they want to commit the time and resources needed for more comprehensive evaluations. The Subcommittee believes that this exercise is worthwhile, and indeed necessary. Without evaluation, how can the Agency and the Congress tell which programs are working, which need attention, and which need to be cut or expanded? In doing such an evaluation, it is important to give a complete list of effects. Where there is any ability to do so, it is important to quantify and monetize the benefits - at least within an order of magnitude.

We offer the following specific comments in the belief that constructive advice at this stage can help the Agency make significant improvements in the assessment process needed for the Section 812 and other future mandates. We also note that neither Congress nor the EPA implemented the CAA in a way designed to expedite evaluation. It is not surprising, therefore, that quantifying benefits and costs is so difficult and uncertain at this point.

- a) **Coordination and Management:** The CAA Section 812 retrospective analysis and the subsequent prospective analysis are very substantial and important exercises. However, the retrospective study does not presently appear to be sufficiently coordinated - either across the various physical effects assessments, or across all major model components. Various EPA offices and researchers (and their contractors) have used different strategies to address problems, which limit the linkages in the study and the consistency of presentation. Although this study should be pushing the frontiers of benefits assessment, it is, in fact, well behind many similar studies performed for other governmental entities in terms of coordination, use of available knowledge, and assessment of uncertainty.

The project needs more emphasis on careful and forceful coordination, planning, and consistency. The Agency needs a modeling team leader to structure model frameworks for physical effects quantification and linkages to other model components. The leader can assure that the team identifies and addresses specific critical modeling strategies and issues.

- b) **A Comprehensive Assessment:** In Section 812, Congress specified that the assessment should be "comprehensive" and that it should "consider all of the economic, public health, and environmental benefits... [emphasis added]." We interpret this to mean that Congress was directing the Agency not to be conservative in the sense of minimizing the likelihood that benefits would be overstated. Rather, Congress appears to have been asking the Agency to produce an Assessment that was inclusive of not only well documented and measured effects and values, but also those effects and values for which there is limited information. In other words, Congress was asking the Agency to take some risks by listing and quantifying effects that are not well documented in the literature so as to reduce the likelihood that it would produce an underestimate of the true benefits.

With the notable exception of the analysis of the association between ozone and mortality, the draft document is not responsive to this congressional directive. Rather, it appears to have been prepared with an emphasis on statistical and scientific conservatism. Although this is an understandable, defensible position for the Agency to adopt for many circumstances (for example in setting regulations that have to be defended in a legal arena), it may not inform Congress about all of the possible benefits (or lack of benefits) associated with implementation of the CAA.

- c) **Uncertainty Analysis:** A major deficiency in the draft document is the lack of any adequate treatment of uncertainty. The manner in which uncertainty is treated has important implications for the way in which physical-effects dose-response functions are developed and expressed. And it is important that the Assessment present a clear picture of the degree of confidence, or

lack thereof, that readers can place on the components of the Assessment.

- d) **Ecological Effects:** The effects of air pollutants on human health have dominated this analysis thus far, and it does not appear that ecological effects will catch up within the short time and limited budget that remain for this project. However, one of EPA's missions is to protect ecosystems from adverse effects of pollutants. By definition, a benefit-cost analysis is stated in terms of dollars. However, some quantification involves effects that cannot be monetized by current methods. Protocols will be needed to express ecological and other nonmonetary values and to compare them to values expressed in monetary terms. For many of the human health effects, valuation in monetary terms may be all that is needed. However, when ecological effects eventually are addressed, many ecological values (including non-use values) may not easily be expressed in monetary terms. Although ecological effects are difficult to comprehensively quantify and value in economic terms, the absence of at least a qualitative analysis that is on equal footing with a human health analysis will be conspicuous and will leave EPA open to sharp criticism.

The enclosed report also provides comments on a number of more technical issues including: the selection of impacts for quantification; omissions, biases, and uncertainty analyses; sensitivity analysis; and the over-reliance on clinical, as opposed to epidemiological, data.

Lastly, the Council strongly recommend that the Administrator and the Congress allocate adequate resources to the Agency and other entities to build a core of expertise to continue the difficult, but necessary exercise pertaining to quantifying health, welfare and ecological effects. It is important that this effort be sustained over the long term. Such expertise could logically focus on the many challenges identified in this report to you and the Congress.

We look forward to receiving your reactions to our recommendations as we continue our reviews of the documents

pertaining to the health effects of air toxics, and to ecological and welfare effects.

Sincerely,

A handwritten signature in black ink, appearing to read 'Richard Schmalensee', written in a cursive style.

Dr. Richard Schmalensee
Chair, Clean Air Act Compliance
Analysis Council
Science Advisory Board

A handwritten signature in black ink, appearing to read 'Morton Lippmann', written in a cursive style.

Dr. Morton Lippmann
Chair, CAACAC Physical Effects
Review Subcommittee
Science Advisory Board

ENCLOSURE

NOTICE

This report has been written as a part of the activities of the Science Advisory Board, a public advisory group providing extramural scientific information and advice to the Administrator and other officials of the Environmental Protection Agency. The Board is structured to provide a balanced, expert assessment of scientific matters related to problems facing the Agency. This report has not been reviewed for approval by the Agency; hence, the comments of this report do not necessarily represent the views and policies of the Environmental Protection Agency or of other Federal agencies. Any mention of trade names or commercial products does not constitute endorsement or recommendation for use.

ABSTRACT

The Physical Effects Review Subcommittee (CAACACPERS) of the Clean Air Act Compliance Analysis Council (CAACAC) of the Science Advisory Board (SAB) has reviewed the Agency's draft physical effects documents and the methodology pertaining to quantifying health effects of criteria air pollutants for the Clean Air Act (CAA) Section 812 retrospective benefit-cost analysis. CAACAC responded to five specific questions raised in the charge to the Subcommittee and also provided more general comments and suggestions relating to this topic.

CAACACPERS believes that, despite considerable shortcomings in these documents, the Agency has laid out a useful framework. The Subcommittee identified a number of technical issues requiring resolution, and made a number of specific recommendations, including: more systematically identify and document the selection of impacts to be specifically included and excluded; conduct sensitivity and uncertainty analyses; more carefully balance and specify the uses of epidemiological and clinical data; identify a method complementary to the standard valuation endpoints, so endpoints that do not lend themselves to monetary valuation can also be considered equitably in a cost-benefit analysis; rectify inconsistencies in the selection of coefficients; and investigate mitigation behavior. The Subcommittee offers many other comments and emphasizes where fundamental improvements are needed.

The Subcommittee strongly recommends that the Administrator and the Congress allocate adequate resources to the Agency to enhance its core of expertise to continue the difficult, but necessary exercise pertaining to quantifying human health, human welfare, and ecological effects over the long term. Such expertise could logically focus on the many challenges inherent in cost-benefit analyses.

Key Words: Clean Air Act, Cost-Benefit Analysis, Physical Effects, Air Pollutants, Ozone, Particulate Matter, Lead, Carbon Monoxide, Sulfur Dioxide, Nitrogen Dioxide, Economic Valuation.

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1. EXECUTIVE SUMMARY

In response to the Congressional mandate of Section 812 (Appendix A, CAA, 1990), and at the request of the Agency's Office of Policy Analysis and Review (OPAR) and the Office of Policy Analysis (OPA), the Clean Air Act Compliance Analysis Council (CAACAC) Physical Effects Review Subcommittee (CAACACPERS) reviewed draft physical effects documents and the methodology pertaining to quantifying health effects for the Clean Air Act (CAA) Section 812 retrospective benefit-cost analysis for criteria pollutants.

In addition to answering the questions in the Charge (see section 2), the Subcommittee also focused on the broader mandate from Congress regarding the Section 812 study. The following highlights summarize the main points of this review and are offered in the belief that constructive advice at this stage can help the Agency make significant improvements in the assessment process needed for the Section 812 and other future mandates. We also note that neither Congress nor the EPA implemented the CAA in a way designed to expedite evaluation. It is not surprising, therefore, that quantifying benefits and costs is so difficult and uncertain at this point. Congress and the Agency have to ask themselves whether they want to commit the time and resources needed for more comprehensive evaluations. The Subcommittee believes that this exercise is worthwhile, and indeed necessary. In doing such an evaluation, it is important to give a complete list of effects, and when possible, to quantify and monetize the benefits - at least within an order of magnitude.

The Agency's draft documents reviewed are a first step toward confronting the challenge of the Congressional mandates of Section 812 of the CAA of 1990. The following points are offered as a summary of the findings and recommendations of the Subcommittee as it dealt with the charge and identified major issues:

- a) A Comprehensive Assessment: In Section 812, Congress specified that the Assessment should be "comprehensive" and that it should "consider all of the economic, public health, and environmental benefits... [emphasis added]." With the notable exception of the analysis of the association between ozone and mortality, the draft document is not responsive to this congressional

directive. Rather, it appears to have been prepared with an emphasis on statistical and scientific conservatism. Although this is a defensible position for the Agency to adopt for many circumstances (for example in setting regulations that have to be defended in a legal arena), it may not inform Congress about all of the possible benefits associated with implementation of the CAA.

- b) Coordination and Management: The CAA Section 812 retrospective analysis and the subsequent prospective analysis are very substantial and important exercises. However, the retrospective study does not presently appear to be sufficiently coordinated - either across the various physical effects assessments, or across all major model components. Various EPA participants have used different strategies to address problems, which limit the linkages in the study and the consistency of presentation. This study should be pushing the frontiers of benefits assessment, but it is, in fact, well behind many similar studies in terms of coordination, use of available knowledge, and assessment of uncertainty.

The project needs more emphasis on careful and forceful coordination, planning, and consistency, particularly in the area of modeling. The Agency needs to assure that specific modeling strategies and issues are identified and addressed

- c) Selection of Effects for Inclusion in the Assessment: Balancing the objective of performing a comprehensive analysis against the cost of completing the assessment requires a systematic approach to the selection of effects-- one that focuses resources on the most substantial effects for which literature exists for benefits assessment, while not ignoring other effects of potential interest or concern. We recommend a more comprehensive approach to the screening of potential effects and the selection of effects for quantification and valuation. Specifically, we recommend:
- 1) Providing a more complete listing and identification of known and suspected physical effects.

- 2) Screening known and suspected physical effects to formally identify and focus on the most substantial impacts for which there is literature to develop a damage assessment.
 - 3) Employing a consistent format for reporting omissions, biases, and uncertainties in all study components, and especially in all physical-effects components.
- d) Selection of Pollutant Species for Analysis: The analyses received initially were restricted not only to the criteria pollutants but, within them, to the specific entities monitored in the ambient air, i.e., SO₂, NO₂, and the mass concentrations of PM₁₀ and PM_{2.5}. This is inappropriate for sulfur oxides (SO_x), nitrogen oxides (NO_x), and particulate matter (PM). There were essentially no analyses made for the effects of other SO_x, such as sulfuric acid aerosol and its neutralization products, of other NO_x, such as nitric acid vapor and nitrate salts, or of other PM components, such as trace metals or toxic organic particles.
- e) Selection of Health End Points: Given that the purpose of this Assessment is to estimate the economic value of air quality changes resulting from the implementation of the CAA of 1970, the Subcommittee has reservations about the specific health endpoints selected for detailed quantitative analysis. An example of an effect given too much attention is reduced lung function following short-term peaks in exposure to ozone. In some instances, recent epidemiological evidence of more adverse effects that are also more readily monetized was overlooked.
- f) Omitted Physical Effects: The documents presented to the Subcommittee specifically address only the health effects of the index pollutants within the criteria pollutant category. In addition to omitting any discussion of other air pollutants within the criteria pollutant category, the documents reviewed did not

address health effects of air toxics, welfare effects, and ecological effects. Although recognizing the limited resources available to EPA to complete this project, we urge EPA to perform some sensitivity analyses to prioritize the levels of efforts to be invested in air toxics, and welfare and ecological effects assessments.

- g) Uncertainty analysis is not adequately treated: A major deficiency in the draft document is the lack of any adequate treatment of uncertainty. It is not too late to make a decision about how uncertainty will be handled in the assessment. The manner in which uncertainty is treated has important implications for the way in which physical effects dose-response functions are developed and expressed.

- h) Ozone: The Ozone Section of the document is seriously unbalanced. There is an inappropriate emphasis on the findings from clinical studies, as well as questionable interpretation of the clinical study implications. The Assessment needs to link more closely the results of the clinical, field, and epidemiologic studies to provide a firmer basis for the health costs of ozone exposures based on the exposure-response relationships from the studies of exposed populations.

- i) Sulfur Oxides: The stated goal of limiting the analysis to the gaseous sulfur oxides has not been explained or justified. Most importantly, this section does not come to grips with:
 - 1) The role of SO₂ as a precursor of acidic aerosol formation;
 - 2) The ways in which SO₂ emission controls have affected exposures to acidic sulfate aerosols; and
 - 3) The health benefits of the reductions in such exposures.

- j) Particulate Matter: The mortality effects of particulate matter are likely to be the dominant ones in terms of economic impact. It is unfortunate that the draft is incomplete as it stands in several respects. First, it needs to account for other acute-mortality studies that satisfy the selection criteria.

Second, it must address the nature and significance of acute mortality in terms of the extent of life-shortening for those who die in excess on polluted days. Third, it should consider evidence on differences in mortality risk across age groups, e g, under 65 years vs. 65 and older. Finally, the biggest deficiency is that it ignores the differences in annual average (cross-sectional) mortality rates among communities.

- k) Carbon Monoxide: The section on CO presents, in a clear manner, the current information pertaining to the health risks of exposure. As it notes, the most consistent data relating low-level concentrations to health measures come from studies based on latency to anginal pain induced by exercise. Linking these findings to mortality from heart disease or to accelerated myocardial damage is a difficult speculative exercise, but offers the most reasonable way to assess the benefits of reduced exposure. If the lower-bound estimate includes zero, this can be stated. At a minimum, the Agency should conduct a preliminary assessment using worst-case assumptions to establish an upper bound.

- l) Nitrogen Oxides: It is defensible to exclude aerosol forms of NO_x from this Section, if they are adequately discussed in Section 3 on Particulate Matter. However, such an exclusion needs to be stated more explicitly. In the case of NO_x, that still leaves all of the vapor-phase nitrogen oxides to be discussed in this Section. Thus, this Section is obligated to review the health effects associated with nitric acid, and possibly nitrous acid (as well as those associated with NO₂).

- m) Lead: This is one of the better-written products provided by the Agency. The Subcommittee notes that the Agency's draft document recognizes the broad spectrum of lead toxicity and attempts to provide quantitative risk assessments for a variety of endpoints. This aim, however, misses some of the subtleties of the lead literature. Specifically:
 - 1) No explicit role is accorded to other measures of neurotoxicity;

- 2) Cross-sectional and other studies fail to exploit the relevant dose-response behavioral data;
 - 3) Inadequate attention is paid to the interaction of social class and the expression of lead toxicity;
 - 4) Effects in adults are accorded a narrow point-of-view
 - 5) Observations of other effects of lead are important to understanding fully the risk factors
 - 6) Remobilization of stored lead has been ignored
 - 7) The document gives inadequate discussion to the doses and responses chosen, the approximate nature of the functional form, the perils of extrapolating very far from the median of the data, and the question of thresholds
 - 8) There is no discussion of how changes in mean blood lead levels in adults will be predicted as a function of changes in lead emissions
- n) Ecological Effects: The effects of air pollutants on human health have dominated this analysis thus far; and it does not appear that ecological effects will catch up within the short time and limited budget that remain for this project. However, one of EPA's missions is to protect ecosystems from adverse effects of pollutants. Protocols will be needed to express ecological and other nonmonetary values and to compare them to values expressed in monetary terms. Currently, the documents only discuss valuation in monetary terms. For many of the human health effects, this may be all that is needed. However, when ecological effects eventually are addressed, many ecological values (including non-use values) may not easily be expressed in monetary terms. Although ecological effects are difficult to comprehensively quantify and monetize, the absence of at least a qualitative analysis that is on equal footing with a human health analysis will be conspicuous and will leave EPA open to sharp criticism.

2. INTRODUCTION AND OVERVIEW

The CAACACPERS met on November 15 and 16, 1994 to receive briefings, and have discussions with the Agency staff and the public. The Subcommittee also conducted a public teleconference on April 12, 1995 and held a public meeting on May 18, 1995 to review additional draft documents related to this topic.

The basic charge presented to the Subcommittee is as follows.

- a) Are each of the elements of the methodology developed by EPA sufficiently valid and reliable from a scientific standpoint for purposes of the CAA Section 812 assessments?
- b) The Congress and the EPA intend that the CAA Section 812 retrospective analysis should provide the most comprehensive possible statement about potential benefits of historical reductions in air pollution. This includes reporting on the potential significance of effects for which there may be no scientific consensus regarding the magnitude or even the existence of a specific effect. Given this statutory and administrative goal, is the methodology developed by EPA sufficiently comprehensive in terms of plausible physical health, welfare, and ecological consequences of exposure to the relevant air pollutant?
- c) If the answer to question 2 (above) is negative, what are the physical outcomes of pollutant exposure which the Agency has omitted?
- d) For the physical outcomes already included in the methodology paper, as well as those which should be considered for inclusion pursuant to question 2 (above), are there potentially relevant data pertaining to quantitative or qualitative estimation of the effect which should be considered for inclusion in the methodology? What specifically are the sources of these data and how might they be best utilized?
- e) What is the strength of the scientific evidence underlying each of the physical effects functions or models already included in the methodology paper? What is the strength of each element of additional

scientific evidence suggested for inclusion pursuant to questions 3 and 4 above?

3. METHODOLOGY AND OVERVIEW OF APPROACH

One question posed to the SAB was whether EPA's proposed approach is suitable for quantifying health effects pertinent to Section 812 of the CAA, with the stated goal of providing the basis for a benefits analysis that is "sufficiently comprehensive in terms of plausible health, welfare, and ecological consequences of exposure to the relevant air pollutant." Unfortunately the current draft of the Overview document fell short of these aims. The Introduction to the document refers to welfare and ecological effects as well as health effects, and several passages in the document are applicable to these broader aims. However, most of the document discusses issues pertinent only to the quantification of a limited number of effects on human health. We recommend that the title of the Overview be changed to reflect the limited aims of the present Section 812 Assessment, or that the analyses be broadened to be consistent with the original aims.

The health overview beginning on page 3 did not accurately describe the approaches used or say anything about the comparative advantage of each. Toxicological and clinical studies are needed to establish causality. Quantification is generally a combination of toxicology (causality, functional form, and sensitive populations) and epidemiology (i.e., "real world" conditions and quantification of population response rates).

The issue of mitigation behavior was not addressed in the general methods section or in any of the chapters provided. Individuals in controlled exposure studies cannot mitigate exposures, whereas individuals in real life situations sometimes can affect their exposures. Thus, the results of clinical studies may overstate damages. On the other hand, ignoring mitigation in epidemiologic studies results in understated damage. For example, the number of observed health effects per capita for any pollutant level is reduced by mitigation, but mitigation results in some costs to the affected individuals, such as reduced activities and increased use of medication.

In redrafting the overview chapter, one option that could be considered is to reorganize by effect rather than by pollutant. This would naturally lead to a discussion of real world exposures of mixed pollutants as well as to 'secondary' pollutants formed in the atmosphere. It may allow for a better assessment of the dose/response relationships discussed at the end of each chapter

and avoid the issues associated with "double counting" of effects. This reorganization of the material would not affect the air quality analyses/modeling activities also associated with this exercise. Perhaps, more importantly, it could place the modeling exercise in context, because the actual model outputs of a pollutant mixture could be used rather than the pollutant-by-pollutant output.

4. OVERALL CONCLUSIONS AND RECOMMENDATIONS

4.1 Coordination and Management

The CAA Section 812 retrospective analysis and the subsequent prospective analysis are very substantial and important exercises. However, the retrospective study does not presently appear to be sufficiently coordinated - either across the various physical effects assessments, or across all major model components. Various EPA offices and researchers (and their contractors) have used different strategies to address problems, which limit the linkages in the study and the consistency of presentation. Although this study should be pushing the frontiers of benefits assessment, it is, in fact, well behind many similar studies performed for state and other federal agencies, in terms of coordination, use of available knowledge, and assessment of uncertainty.

The project needs more emphasis on careful and forceful coordination, planning, and consistency. With consistency, important linkages will be made between major study components, appropriate comparisons of costs and benefits can be made, uncertainty can be treated in a like manner in all study components, and the level of professionalism will be consistent in all work elements.

The Agency needs a modeling team leader to structure model frameworks for physical effects quantification and linkages to other model components. The leader can assure that the team identifies and addresses specific modeling strategies and issues.

4.2 Selection of Effects for Inclusion in the Assessment

Balancing the objective of comprehensiveness against the cost of completing the assessment requires a systematic approach to the selection of effects that focuses resources on the most substantial effects for which literature exists for benefits

assessment, while not ignoring other effects of potential interest or concern. The draft overview chapter provides a good, short summary of the approach that was taken, and provides reasonably good justification for the options selected within that narrowly defined approach. However, we recommend a more comprehensive approach to the screening of potential effects and the selection of effects for quantification and valuation. Specifically, we recommend:

- a) Providing a more complete listing and identification of known and suspected physical effects.
- b) Screening known and suspected physical effects to formally identify and focus on the most substantial impacts for which literature to develop a damage assessment exists. Ample research to assist with this screening exists. It is important that a clear statement be provided on what is included in the quantification and what is not. Substantiation for the selections of the literature should also be provided. The selection process should focus on key health, ecological, and welfare impacts that are defended by the screening, with the other effects left to be addressed later, if and when additional resources become available. The Subcommittee would also like to emphasize that both monetary and non-monetary values should be looked at; that is, dollar benefit impacts should not be the only basis for selecting and evaluating impacts.
- c) Employing a consistent format for reporting omissions, biases, and uncertainties in all study components, and especially in all physical-effects components. This reporting can be in tabular form, listing the omission, bias, or uncertainty, the direction of bias (if known), and any comments on the potential significance of the omissions.

4.3 Selection of Pollutant Species for Analysis

Another major problem with the overview chapter and companion documents is the lack of any apparent strategic planning leading to a comprehensive framework for analysis. This is evident from the analyses being not only restricted to the criteria pollutants but, within them, to the specific entities

that are monitored in the ambient air. This is a suitable limitation for carbon monoxide (CO), but not for sulfur oxides (SO_x), nitrogen oxides (NO_x), and particulate matter (PM). Within these pollutant classes, analyses were largely restricted to the effects of SO₂, NO₂, and the mass concentrations of PM₁₀ and PM_{2.5}. As a consequence, essentially no analyses were made for the effects of sulfuric acid aerosol and its neutralization products, of nitric acid vapor and nitrate salts, of trace metals, or of toxic organics as either vapors or particles.

The reductionist approach taken also complicates and obscures the opportunities to take some promising approaches to determining the benefits resulting from the 1970 CAA. The control costs incurred have largely been associated with source-strength reductions regarding criteria pollutants themselves and precursors of secondary pollutants. Imposed on motor vehicles, power plants, space heating, and fuel processing, these controls have led to known or calculable source reductions in emissions of CO, hydrocarbons, SO₂, NO_x, and coarse particles. The SO₂ and NO_x reductions have also led to less well defined, but calculable reductions in secondary pollutants such as SO_x and NO_x aerosols and ozone (O₃). Furthermore, the efforts to control ambient concentrations of O₃ and PM have led to reductions in ambient concentrations of CO, as well as much greater reductions in source emissions of trace metals and hydrocarbons (as both vapors and aerosols) than any efforts to control them through NESHAPS (National Emission Standards for Hazardous Air Pollutants) as hazardous or toxic air pollutants. Analyses should be done to show the effects of: a) NO_x source controls on ambient concentrations of O₃ and nitric acid; b) the effect of SO₂ and NO_x source controls on ambient concentrations of acidic aerosols and on acidic deposition in the environment; and c) the effects of reductions in O₃ on the formation of acidic aerosols. Such an analysis most likely would show, for example, that the benefits from NO_x source control lie more in their effect on O₃ formation, formation of air toxics through photochemical reactions, and acidic deposition, than on reduction of health effects directly attributable to NO₂. The overview chapter is incomplete without a discussion of such interrelationships between the various pollutants within and among the categories of the National Ambient Air Quality Standard (NAAQS) and NESHAPS pollutants.

4.4 Health-Effects End Points Selected

Given that the purpose of this Assessment is to estimate the economic value of air quality changes resulting from the

implementation of the CAA of 1970, we have reservations about the specific health endpoints selected for detailed quantitative analysis. An example of an effect given too much attention is the changes in lung function following short-term peaks in exposure to ozone. No economic data or methods were identified for estimating the economic values of such effects. Furthermore, to the extent that changes in lung function are associated with other symptoms and endpoints that are being modeled, there is a potential for double counting of effects.

On the other hand, several health end points of potential economic significance were not modeled. Both economic valuation studies and epidemiological studies support the development of dose-response functions for a) respiratory hospital admissions, minor restricted activity days, and acute respiratory symptoms due to exposures to ozone and b) asthma, restricted activity days, and childhood bronchitis due to exposures to particulate matter.

Also, the discussions of health effects for each pollutant were inconsistent and incomplete. The material is presented with limited indication of how it will be used and integrated into the overall assessment. For example:

- a) The introductory chapter noted the limited usefulness of clinical (controlled human exposure) studies for this assessment, yet the ozone chapter focused on clinical studies with little explanation as to why. The fundamental purposes for including controlled-exposure studies are to establish a basis for the effects observed in epidemiologic studies and to establish evidence for effect thresholds. The fundamental reasons for not requiring controlled exposure studies are the costs to conduct valid exposure analyses (which are not required with the use of epidemiologic studies) and the limited usefulness of the endpoints that are measured. These reasons, and others, were not sufficiently discussed.
- b) The selection of studies, or of dose-response parameters, was inconsistent. In some cases a best study was selected by judgement, while in other cases meta-analysis was used. In these cases, the criteria for selection of studies for inclusion into the meta-

analysis were not made clear. A consistent analysis framework should be adopted.

- c) Inconsistent and limited treatment was given to portraying the range of dose-response results for use in uncertainty analysis. In some health sections, the highest and lowest coefficient were reported. In others, a meta-function standard error was reported (or appeared likely to be the strategy that would be used). These two approaches have a considerably different interpretation. Without consistency in the treatment of uncertainty, in the physical-effects study component, and across all other major analysis components, any propagation of uncertainty may be of limited meaning.
- d) Evidence for and against thresholds for health effects was often not discussed. What are the assumptions EPA uses for the base case, and why? What sensitivity analysis will be done, and why? Base-case threshold assumptions can be made and defended, and the analyses can be conducted with alternative assumptions about what are practical thresholds (below which effects may still occur but where the dose-response curve may become very flat or where there is little basis to extrapolate existing data to low levels).
- e) Some health effects may overlap. The treatment of double counting should be addressed in each chapter.

4.5 Omitted Physical Effects

The documents presented to the Subcommittee specifically address only the health effects of the index pollutants within the criteria pollutant category. Discussion of other air pollutants within the criteria pollutant category, health effects of air toxics, welfare effects such as effects on forests crops, visibility and materials, and ecological effects was omitted. Although recognizing the limited resources available to EPA to complete this project, we urge EPA to perform some sensitivity analyses to prioritize the levels of efforts to be invested in air toxics, and welfare and ecological effects assessments. No plan to handle this challenge was apparent in the Methodology

Overview document. Expert opinion may provide the only way to approach this challenge.

4.6 Uncertainty Analysis

A major deficiency in the draft document is the lack of any adequate treatment of uncertainty. It is now past time to make a decision about how uncertainty will be handled in the assessment. The manner in which uncertainty is treated has important implications for the way in which physical-effects dose-response functions are developed and expressed.

The options for dealing with uncertainty include the formal specification and propagation of uncertainty using probability distributions on key variables and applying Monte Carlo simulations or other related techniques; the specification of high and low values for key parameters and the calculation of upper and lower bounds based on them; and sensitivity analysis. We prefer the formal analysis of uncertainty because it generates much more information about the overall uncertainty that results from the combinations of uncertainties about components of the assessment. For example, using high and low values will usually lead to unrealistically wide bounds around the true value, at least if the individual component uncertainties are independent of each other, as they often will be. We also recommend that important omissions, biases, and uncertainties be listed and their potential significance to the assessment be discussed.

We note that the CAACAC also discussed this set of issues in its letter of March 24, 1993 to the Administrator (SAB, 1993).

4.7 Peer-reviewed literature

The document clearly presents the case that the analysis will include data from some literature that is not peer-reviewed. While this is necessary in some instances, we would strongly argue that the window for inclusion of non peer-reviewed literature be restricted to about two years, sufficient for it to be published. For example, we suggest limiting the use of non-peer-reviewed literature to supportive analyses rather than core components. Clearly, if a critical component of the analysis methodology is based on non-peer reviewed literature, the analysis is suspect. Some explicit criteria for inclusion and exclusion of "gray" literature needs to be formulated.

4.8 Additional Issues and Recommendations

In addition to answering the specific questions in the Charge, the Subcommittee broadened its scope of coverage to focus on the broader mandate from Congress regarding the Section 812 study.

Attempts to quantify the costs of air pollution have been undertaken over the past three decades, beginning with Ridker and Henning (1967), Ridker (1967), and Lave and Seskin (1970). These efforts led to more active discussion on the reliability of assuming a causal relationship from statistical associations. In 1989, a Congressional Research Service Report to Congress (Congressional Research Service Report, 1989) noted, in the summary:

"..we conclude that though there is no doubt that significant health benefits result from controlling some air pollutants, it is not currently feasible to produce an unambiguous evaluation of the health benefits."

In Section 812, Congress specified that the Assessment should be "comprehensive" and that it should "consider all of the economic, public health, and environmental benefits... [emphasis added]." We interpret this to mean that Congress was directing the Agency not to be conservative in the sense of minimizing the likelihood that benefits would be overstated. Rather Congress appears to have been asking the Agency to produce an assessment that was inclusive of not only well documented and measured effects and values, but also those effects and values for which limited information exists. In other words, Congress was asking the Agency to take some risks of overstating benefits so as to reduce the likelihood that it would produce an underestimate of the true benefits.

With the notable exception of the analysis of the association between ozone and mortality, the draft document is not responsive to this Congressional directive. Rather, it appears to have been prepared with an emphasis on statistical and scientific conservatism. Although this is an understandable, defensible position for the Agency to adopt for many circumstances (for example in setting regulations that have to be defended in a legal arena), it may not inform Congress about all of the possible benefits (or lack of benefits) associated with implementation of the CAA. The Methodology Overview should discuss the issues of conservatism in the face of limited information about some effects and describe its approach to meeting the mandate of Congress to avoid undue conservatism.

The Council offers the following comments in the belief that constructive advice at this stage can help the Agency make

significant improvements in the assessment process needed for the Section 812 and other future mandates. We also note that neither Congress nor the EPA implemented the CAA in a way designed to generate a database that could be used to expedite evaluation. It is not surprising, therefore, that quantifying benefits and costs in the absence of much relevant data is so difficult and uncertain at this point.

Congress and the Agency have to ask themselves whether they want to commit the time and resources to future evaluations. The Subcommittee believes that this effort is worthwhile, and indeed necessary. Without evaluation, how can the Agency and the Congress tell which programs are working, which need attention, and which need to be cut or expanded? In doing such an evaluation, it is important to give a complete list of effects. Where there is any ability to do so, it is important to quantify and monetize the benefits - at least by an order of magnitude.

The Agency should focus its resources on estimating the important benefits within each program. For example, the Agency can neglect effects that are a factor of ten smaller than the largest effects in a category -- They are unlikely to make a significant contribution. This rule-of-thumb will allow the Agency to focus its resources on the categories of interest and not waste time or effort on categories that would not affect the policy implications of the evaluation.

Currently, the documents only discuss valuation in monetary terms. For many of the human-health effects, this may be all that is needed. However, when ecological effects eventually are addressed, EPA might be forced to conclude that many ecological values (including non-use values) cannot easily be monetized. EPA and the Congress will need protocols for expressing values in non-monetary terms, and for comparing them to those values that will be expressed in monetized terms. In effect, a methodology is needed to decide how to maintain two complementary benefits columns -- monetary and non-monetary values.

Another major problem is that the documents failed to confront the challenge of Section 812 in the CAA of 1990. The overall need is clearly stated in the first two pages of the overview chapter (October 11 draft). Thereafter, the focus suddenly narrows to "effect categories that have a direct effect on human health." Furthermore, the balance of the overview chapter, and the content of the following six Sections on criteria pollutants, makes it evident that analyses are focussed almost entirely on acute health responses, with considerably less attention to cumulative tissue damage resulting from long-term, low-level exposures and their benefits in terms of chronic health-care costs, lost time from work or school, and diminished quality-of-life in people with chronic health damage.

In order for EPA to be responsive to the mandate of Section 812 of the CAA, its final report will need to take a broader view of not only human-health effects of criteria pollutants, but also other major class of pollutants and their effects. These include the effects of hazardous air pollutants on human health, as well as the effects of air pollution on visibility, ecosystems, forest and agricultural productivity, and on welfare effects such as soiling and damage to materials and equipment. It should be noted that the SAB's CAACAPERS received draft documents in April, 1995 related to the above topics, discussed them in an open public meeting on May 18, 1995, and will provide a future advisory document to the Agency on these issues.

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APPENDIX A--SPECIFIC TECHNICAL COMMENTS

OZONE

General Comments

The Ozone Section of the document is seriously unbalanced. The specific reasons for this conclusion are provided in the detailed critique that follows, but the imbalance is primarily the consequence of inappropriate emphasis on the findings from clinical studies, as well as questionable interpretation of the clinical study implications. The clinical studies are very important, because they confirm that the functional and symptomatic effects reported in field studies of natural populations engaged in outdoor recreational activities and/or vigorous work schedules can be seen under rigorously controlled experimental conditions. They are also important, because they demonstrate that concurrent lung inflammation, not detectable in field studies, occurs and that it persists during repeated daily exposures despite the attenuation of functional and symptomatic responses. This provides mechanistic support for the epidemiological associations observed between repeated exposures and the exacerbation of asthma. By more closely linking the results of the clinical, field, and epidemiologic studies, a revised document could provide a firmer basis for the health costs of ozone exposures based on the exposure-response relationships from the studies of exposed populations.

In this revised discussion, the authors need to recognize that the paradigm for the quantitative assessment of the pulmonary function responses of humans to exposure to ozone has recently shifted, as agreed by CASAC (Clean Air Scientific Advisory Committee) consensus during their meeting in July, 1994 and by the WHO-EURO (World Health Organization, European Region) Working Group on Air Quality Guidelines at their meeting in Bilthoven, The Netherlands, October, 1994 (Lippmann, 1995, personal communication). There is now a body of credible data from field studies on human populations in natural settings to establish the nature and extent of human pulmonary function responses to ambient ozone exposures, and their uncertainty. In this new paradigm, the findings in controlled laboratory studies provide support for the observed effects in natural populations, rather than the other way around, as stated in the draft chapter.

The effect of ozone on mortality remains to be established, but warrants careful attention. Although the Kinney and Ozkaynak (1991) studies report positive ozone effects, other mortality studies have not found a statistically significant ozone relationship. EPA does not address how it would use the Kinney and Ozkaynak results. Will the coefficient from LA or NY be

used? Or would they be averaged? Will these values be the central coefficient (ignoring or giving little or no weight to other negative studies)? What confidence will be given to the selected coefficients and why?

The macro-epidemiology studies are noticeably absent, including those for restricted activity days (e.g., Ostro *et al.*, 1989; Portney *et al.*, 1986), acute respiratory symptom days (Krupnick *et al.*, 1990), and respiratory hospital admissions (Burnett *et al.*, 1994; Thurston *et al.*, 1992, 1994, Pope, 1991; Schwartz, 1994a,b). This omission is noticeable because these endpoints and studies are easier to use than the selected controlled exposure studies, these endpoints and studies are regularly used in other benefit analyses, and because many of these endpoints and studies are included in the particulate matter chapter. Related to these points is our concern with the emphasis in this Section of the draft on physical effects of uncertain significance for human health and for which there is little or no empirical data on people's willingness to pay to avoid these effects.

As noted in the prior CAACAC Subcommittee review on transport and transformation modelling, a potentially major issue arises when attempting to reconstruct historical O₃ exposure via simple urban plume (box) models. Historical emissions of NO_x were higher than today. Therefore the spatial distribution of NO_x emission densities in urban areas would be different today in the absence of controls, and thus the spatial distribution of maximum hourly O₃ values would also be different. Simple urban plume models may not adequately capture this shift in O₃ and therefore may improperly characterize the control versus no-control population exposure difference. One possible approach is to use the O₃ predictions from the RADM (Regional Acid Deposition Model) model exercise to predict the control/no-control ratios of O₃ exposure and then use these ratios to adjust current (present day) O₃ levels. This would provide a spatially averaged O₃ exposure difference for use in retrospective analyses.

This section also needs other significant revisions. It must address: standard issues of thresholds; double counting; omissions, biases, and uncertainties; and the selection of central dose-response function coefficient values.

Specific Comments

Page 1-3: Line 9 from top: "the average changes in lung function are generally small and are a matter of controversy in regard to their medical significance." As noted below, the issue is whether an FVC (Forced Vital Capacity) change is indicative of induced inflammation. Fuller discussion is needed.

Page 1-8: bottom of page: A recently presented study by Thurston *et al.* (1994). A full text of this manuscript would be important, as the abstract does not mention all the important findings. The authors studied asthmatic children at a summer camp for a week in three consecutive years. The children had to report to an office (where there was doctor and a nurse) if they felt they needed medication. Air pollutants were measured at the camp. The usual decrements in lung function with O₃ levels were noted, but in addition there was a monotonic relationship between the requests for medication and the ozone level at concentrations lower than 100 ppb. This study should also be included in Table 1-3.

Page 1-12: Section 1.2.2: This extended discussion of symptoms does not include any mention of whether the induction of symptoms is important. Does it indicate that inflammation has occurred? Why does the FVC fall early in ozone exposure? As it stands, it is clinically incomplete. See below. Also, the review should cite the recent paper on symptoms in children participating in the Harvard six-cities study (Schwartz *et al.*, 1994).

Page 1-12: A new section is needed to summarize and discuss the evidence of induced inflammation in the human lung after ozone exposure. On page 1-21 in section 1.2.7, it is noted. that : "Indications of ongoing tissue inflammation of subject exposed to O₃ have been reported in several studies." This is far too weak a statement for the contemporary evidence. The time course of the induced inflammation has been well summarized by Koren and Devlin and their associates (Koren, *et al.*, 1991). This reference is useful in this regard.

Page 1-22 (first paragraph): The following statement is quoted from the criteria document: "However, the time course of this inflammatory response and the O₃ exposures necessary to initiate it, have not yet been fully elucidated" . This is entirely unsatisfactory in the light of present evidence (Koren, *et. al.*, 1991). Furthermore, the statement at the end of the second paragraph on page 1-22 referring to Devlin's work states: "However these results have not yet been fully evaluated." What does this mean? We recommend that the authors revisit these issues with experts at HERL (Health Effects Research Laboratories) laboratories of EPA. One would have expected an up-to-date and sophisticated discussion of these issues in this document.

A more suitable summary would state:

"It has been well established that an early effect of ozone is to cause an inflammatory response in the human lung. The pattern of increases in the cells in bronchoalveolar lavage specimens after ozone exposure in normal subjects, followed

by the appearance of inflammatory mediators for as long as 18 hours after the exposure, has been convincingly shown."

The relationship between the onset of symptoms and the reduction in FVC, with the onset of inflammation is less clear. It appears that the magnitude of induced function defect and the severity of the inflammatory response are not closely associated (Frampton *et. al.*, 1994); if this is the case, then the question arises of whether the use of the function test response as a guide to safe exposure level is appropriate. What is the clinical significance of an induced inflammatory response? in normal subjects? in asthmatics? These question must be directly addressed.

It should also be noted that the effect of ozone (120 or 240 ppb) in inducing a inflammatory response in the nose of asthmatic and non-asthmatic subjects has been studied (McBride *et. al.*, 1994). This showed that an inflammatory response was found in asthmatic but not in normal subjects.

Page 1-14: Section 1.2.5: Aggravation of existing respiratory disease:

This section should contain a synthesis of information on asthma. The statement quoted here from the 1986 Ozone Criteria Document is inappropriate, as it is seriously out of date. The Draft Ozone Criteria Document (U.S. EPA, 1995) recently submitted by EPA to CASAC includes a full and up-to-date summary of the known effects of ozone; the clinical and epidemiological data bases are well covered. Additionally, a synthesis of present data on asthma would have told the reader:

- a) Exacerbations of asthma (including fatal asthma) are now believed to be due to acute inflammation in the airways (Laitinen *et. al.*, 1993; Kuwano *et. al.*, 1993).
- b) The early and prolonged induction of airway inflammation by ozone is well documented.
- c) Although in quantitative terms the function test response and the bronchial reactive response in asthmatics to ozone may be similar to that in normals, the effect in asthmatics is exerted on an already depressed level of function, and an already aggravated airway responsiveness. Thus the inference invited from the quotation from the 1986 Ozone Criteria Document, that ozone is not more of a risk to asthmatics than to normals, is not an accurate representation of the circumstances. Kreit's 1989 paper (Kreit *et al.*, 1989) is quoted, but misinterpreted.

- d) Based on the nasal lavage studies, asthmatics show an inflammatory response to ozone that is not shown by non-asthmatics (McBride *et. al.*, 1994).
- e) There is now strong epidemiological evidence (see below) that asthma is made worse by existing ozone levels.

In the light of these observations, the comment on Page 1-28 in the second line: "However, there is no consensus about the magnitude of the difference in sensitivity between asthmatics and other individuals" should be deleted.

Page 1-17: Section 1.2.6: This section is seriously out of date. References in Tables 1.5 and 1.6 include several as abstracts which have now been completely published (White, *et. al.*, 1994; Cody, *et. al.*, 1992; Thurston, *et. al.*, 1994; Burnett *et. al.*, 1994; and Lipfert and Hammerstrom, 1992), and one that is not mentioned (Schwartz, 1994a). To summarize this very large bank of epidemiological data:

- a) The Ontario data has now been analyzed by four different groups of investigators (Bates and Sizto, 1987); Lipfert and Hammerstrom, 1992; Thurston *et. al.*, 1994b; and Burnett *et. al.*, 1994). All find a strong association of respiratory hospital admissions with ozone.
- b) Although in the eastern Great Lakes region, ozone is associated with peaks of acid aerosol, several studies indicate that the primary effects relationship is with ozone. PM₁₀ effects appear to be additional and separate.
- c) In Burnett's analysis of admissions to 168 hospitals in Ontario between 1983 and 1988 (Burnett, *et. al.*, 1994), there is a monotonic relationship between respiratory admissions and ozone the day before. These data should be used in economic estimates and assumed to apply in the Northeast of the continent.
- d) White's data (White *et. al.*, 1994) show a direct association between hospital emergency visits and ozone levels in Atlanta. The levels of acidity here are not much different from those in New Jersey. Together with Cody's data from eight New Jersey hospitals (Cody *et. al.*, 1992), the conclusion should be drawn that ozone at levels below 120 ppb is aggravating asthma.

- e) Schwartz's recently published data on hospital admissions for pneumonia in the elderly in Detroit (Schwartz, 1994a) are supportive. He has been able to show separate effects for PM_{10} and for ozone.

This document should have analyzed the Lipfert, Burnett, Thurston, Cody, and White and Schwartz data, and presented a synthesis of ozone impact on emergency visits and on hospital admissions as a basis for cost estimates.

Page-1-22: Section 1.2-8: The effect of ozone on macrophage is noted here, but this should be under Section 1.1. The discussion is also incomplete because the reader is not told the possible significance of this experimental finding. Does it indicate that ozone levels might affect the incidence or severity of pneumonia? (See comment under Page 1-17 above on Schwartz's recent study of pneumonia in Detroit).

Page 1-22: Section 1.2.9: It is true that animal-exposure data are important for estimating the likelihood of chronic effects of ozone. But the point should be mentioned that it is difficult to extrapolate from rat data to humans, because the rat lung has been shown to be less sensitive to ozone than the human lung, probably because a lower concentration of ozone is delivered to the periphery of the rat lung than to the human. See Hatch *et al.*, (1994) for a recent discussion of this. This important work was done at the HERL in North Carolina.

Page 1-28: Section 1.4.1. It is surprising to find the development of complex formulae to calculate pulmonary function test responses to ozone, together with symptomatic responses in Section 1.4-2, when the strength of the epidemiological data has been denied. An individual does not go to a hospital emergency department, much less get admitted, complaining of a 2% loss of FEV_1 (Bates, 1992). This misplaced emphasis represents a serious lack of balance.

SULFUR OXIDES

The stated goal of restricting this section to the gaseous sulfur oxides has not been explained or justified. It could be justified by a disclaimer that the particulate sulfur oxides would be fully covered in Section 3 - Particulate Matter, if that were indeed the case. This issue will be addressed further in our comments on Section 3. In any case, the stated goal is not followed because an extensive, if inconclusive text on the historic $PM-SO_2$ epidemiology is included in this section.

Most importantly, this section does not come to grips with:
(1) the role of SO_2 as a precursor of acidic aerosol formation;

(2) the ways in which SO₂ emission controls have affected exposures to acidic sulfate aerosols; and (3) the health benefits of the reductions in such exposures. Without such considerations, this section is woefully inadequate.

PARTICULATE MATTER

General Comments

There is little doubt that particulate matter (PM) causes health problems. The questions are: Which particles? Which effects? And what is the dose-response relationship? Recent reviews which address particulate matter should be cited, and the following points should be noted:

- a) In the past, PM dose-response studies have focused on total PM₁₀ and health effects (or even TSP), although a few had started to look at constituents of PM₁₀. EPA takes this approach, as well, in its draft document.
- b) In the past year, considerable new evidence allowed investigation into the relative significance of some constituents (e.g., SO_x aerosols), and
- c) This issue should be investigated by the Agency. There will still remain issues of attribution, verification, and double counting; however, considering the newer literature will provide a much clearer picture of the likely health effects.

The mortality effects are likely to dominate in terms of economic impact. It is unfortunate that the draft is incomplete as it stands in several respects. First, it needs to account (as does Table 3-13) for other acute-mortality studies that satisfy the selection criteria. Second, it must address the nature and significance of acute mortality in terms of the extent of life-shortening for those who die in excess on polluted days. Third, it should consider evidence on differences in mortality risk across age groups, e.g., under 65 years vs. 65 and older. Finally, the biggest deficiency is that it ignores the differences in annual average (cross-sectional) mortality rates among communities (Dockery *et al.*, 1993, Pope *et al.*, 1995).

The secondary aerosols formed in the atmosphere from SO₂ and NO_x precursors can often account for 25 to 40% of the ambient PM. While the NO_x aerosol health effects literature is meager and inconclusive, the SO_x aerosol literature is not. Extensive evidence of health effects associated with exposure to strong acid aerosol (H⁺) exists, and even more literature relating exposures to health effects associated with exposures to SO₄⁼ exists. Whether SO₄⁼ itself produces such effects is an open

question, because ambient SO_4^- may simply indicate the presence of H^+ . In any case, SO_4^- often shows closer associations to the effects, than do simultaneous measurements of PM_{10} or $\text{PM}_{2.5}$, and is often a major mass fraction of $\text{PM}_{2.5}$.

The SO_x aerosol health-effects literature cannot be ignored in the benefits analysis. Either it must be included in a revised Section 3, or alternatively in a revised Section 2.

Although a consensus regarding the effects of PM on certain health endpoints, including mortality, is rapidly evolving, a major issue still remains regarding the application of the results to a retrospective benefits analysis. Specifically, how are we to estimate historical PM_{10} concentrations in the U.S. given the fact that we only have TSP (Total Suspended Particulates) values for most of the past 25 years? One approach is to bound the PM_{10} values over time. A reasonable lower bound on historical PM_{10} is to assume that it has remained constant at present-day values. An upper bound can be derived assuming that present-day PM_{10} to TSP ratios, on a city-by-city basis, have remained constant over time. This would allow a reconstruction of historical PM_{10} based on historical TSP. This latter approach represents an upper limit on historical PM_{10} because it is certain that emissions of larger particles ($>10 \mu\text{m}$) have been controlled more than emissions of smaller particles ($<10 \mu\text{m}$) over the past 25 years in the U.S.

An alternative approach would be to use historical measurements of sulfate to assess fine particle concentration trends over time. Particulate sulfate measurements do exist over time, as well as site-specific comparisons with various fine-particle mass measurements. This analysis presumably would fall within the bounds described above. The danger with this approach is that it could be misconstrued as supportive of a hypothesis that sulfate particles are the causative agent.

CARBON MONOXIDE

The CO document presents, in a clear manner, the current information pertaining to the health risks of exposure. As it notes, the most consistent data relating low-level concentrations to health measures come from studies based on latency to anginal pain induced by exercise. Linking these findings to mortality from heart disease or to accelerated myocardial damage is a difficult speculative exercise but offers the most reasonable way to assess the benefits of reduced exposure.

Behavioral effects, which played a large role in the earlier CO literature, have diminished in importance with the inability of investigators to reliably reproduce such effects. Exercise performance in healthy subjects suggests diminished capacity at

environmentally relevant CO levels, but the data are rather sparse and the question has been pursued only at

high levels of activity in standardized situations. The document should point out the potential importance of modifications in voluntary exercise.

Overall, this Section seemed on track and well done until it concluded (p. 4-14, #1 lines, 5, 6) that: "A concentration-response function based on the Allred *et al.* (1989) data will not be developed and used in the current assessment." Why not? The results of this multi-center controlled human exposure study are important, along with the results of other related and consistent clinical lab studies, because they address cardiac ischemia, a significant risk to health, and show that subjective responses are correlated with objective measures. Specifically, the time to onset of angina is linked to changes apparent on electrocardiograms. Much weaker and less conclusive results were used for this purpose in other Sections. If the lower-bound estimate includes zero, this can be stated. At a minimum, the Agency should conduct a preliminary assessment to either establish order of magnitude values, or a worst case bounding analysis .

NITROGEN OXIDES

General Comments

As noted in Section 2 on Sulfur Oxides, it is defensible to exclude aerosol forms of NO_x from this Section, if they are adequately discussed in Section 3 on Particulate Matter. However, such an exclusion needs to be stated more explicitly. In the case of NO_x, that still leaves all of the vapor-phase nitrogen oxides to be discussed in this Section. Thus, this Section is obligated to review the health effects associated with nitric acid, and possibly nitrous acid (as well as those associated with NO₂). It fails to do so and is, therefore, deficient. The draft is also deficient in its treatment of NO_x chemistry. It should be noted that the only relevant equilibrium is between NO and NO₂, not the other nitrogen oxides.

The conclusion that outdoor NO₂ concentrations are "poor predictors of personal exposures" ignores the recent work done in Los Angeles (Neas, *et al.*, 1991). They co-located NO₂ passive monitors both indoors and outdoors and regressed these weekly values against personal badge values. The outdoor values accounted for between 40 and 50 percent of the personal exposure; the remainder was attributed to indoor values. These recent results temper the conclusions of the chapter. Additionally, recent European work on NO₂ showing detrimental effects on

asthmatic children, should also be looked at by the Agency (Moseler *et al.*, 1994).

Detailed Comments

Page 5-2: Section 5.2-1: The recent work from HERL showing that preexposure to NO₂ in healthy women increases the effect of a subsequent exposure to O₃, should be cited (Hazucha *et al.*, 1994,). It is important to note that NO₂ pre-exposure exerts an effect not only on the subsequent O₃ effect on FEV₁, but also in increasing the airway responsiveness.

The recent Swedish work should be noted (Strand *et al.*, 1994), even though it is still only in Abstract Form. In studies of 18 asthmatics sensitive to birch or timothy grass, exposure to 0.5 ppm NO₂ at rest for 30 minutes was shown to increase the late asthmatic reaction when the subject was subsequently exposed to the allergen. The immediate reaction was unchanged.

Page 5-9: Section 5.3:

The reasons for thinking that asthmatics may be a sensitive group have been described above. In an 8-month panel study of asthmatics in Denmark, Moseholm and his colleagues (Moseholm *et al.*, 1993) found that both SO₂ and NO₂ exposures were associated with worsening of the asthmatic state. This paper might be quoted.

Page 5-11: Last paragraph:

Surely it would be useful to develop a risk estimate regardless of the sources and nature of the exposure to NO₂. It will never be possible to determine exactly, for each individual in society, what made up the cumulative exposure. The quotation from the NO₂ Criteria Document at the head of this page stresses the consistency of the observations, and recent work provides a basis for estimating the exposures. Outdoor NO₂ can only add to indoor levels.

The Subcommittee agrees that some recent data showing an increased risk of asthma exacerbation in Birmingham, England, associated with closer residence to a major highway (Edwards *et al.*, 1994) do not allow identification of the nature of the hazard (which might as well be PM₁₀ as NO₂), far less an estimate of exposure. Nor does the note that the asthma associated with exposures to the dust from soybeans in Barcelona only occurred after several days of NO₂ being elevated (Castellsague *et al.*, 1992) permit a risk estimate. The significant correlation that was reported between outdoor NO₂ levels and hospital emergency visits for acute respiratory disease in the elderly in Vancouver (Bates *et al.*, 1990) can be used in cost estimates, however, because the regression can be computed.

If none of these strategies is deemed solid enough to compute a damage associated with outdoor NO₂ levels, the document should end with some such statement as:

The contemporary data indicate that raised NO₂ levels (both indoor and outdoor) are associated with adverse health outcomes. Currently, the data are not solid enough to permit a damage estimate - but it is clear that any damage estimates which ignore completely the effects of NO₂ are necessarily underestimates.

With the above caveats, this section provides a useful and generally well-balanced review of a notoriously difficult pollutant.

LEAD

General Comments

This is one of the better-written products provided by the Agency. The Subcommittee notes that the Agency's draft document recognizes the broad spectrum of lead toxicity and attempts to provide quantitative risk assessments for a variety of endpoints. This aim, however, misses some of the subtleties of the lead literature. Specifically:

a) No explicit role is accorded to other measures of neurotoxicity: The document focuses on IQ (Intelligence Quotient) as "the predominant measure of neurotoxicity." Other indications of adverse central nervous system (CNS) actions are recognized indirectly, but no explicit role is accorded them. For example, Section 6.1 emphasizes effects on hematopoiesis, but fails to mention the even more critical effects on neurochemistry. We do not expect a comprehensive review of such data in a document of this kind, but they should be acknowledged given that the primary developmental effects are expressed in behavioral toxicity.

b) Cross-sectional and other studies fail to exploit the relevant dose-response behavioral data: Section 6.2.2-2.2 (Cross-Sectional and Other Studies) mentions conduct disturbances as one criterion of toxicity, but fails to cite some relevant data. For example, Needleman *et. al.*, (1979) demonstrated a clear dose-response relationship between tooth lead levels and items on a teacher rating scale describing such disturbances. Yule *et. al.* (1981) observed a similar relationship. Such data provide transparent connections between exposures and adverse effects that supplements the IQ data. The data of Needleman *et. al.* (1990a,b; 1979) which show a

correlation between tooth lead values in the early primary grades and subsequent success in high school, are also important guides to the evaluation of risks and benefits.

c) Inadequate attention is paid to the interaction of social class and the expression of lead toxicity: Although mentioned (page 7), inadequate attention is paid to the interaction of social class and the expression of lead toxicity. It is not solely the Cincinnati studies (Carson et al., 1989; Rae, 1983) that demonstrate such a phenomenon. Winneke et al., (1982), in Dusseldorf, reported similar results. Such interactions should be included in any attempts to describe benefits.

d) Effects in adults are accorded a narrow point-of-view: Section 6.2.2.1 (Effects in Adults) focused on evidence for a threshold. In other sections, a Lowest Observed Adverse Effect Level (LOAEL), which is actually the criterion under discussion, was modified by uncertainty factors. Why was lead treated differently?

e) Observations of other effects of lead are important to understanding fully the risk factors: Section 6.2.3 notes a relationship between higher prenatal blood lead levels and reductions in gestational age and birth weight. Such observations are important not only for estimating lead's contribution to infant mortality but for examining the contributions of lead to reduced IQ scores. Lowered gestational age and birth weight are risk factors for a variety of developmental disturbances.

f) Remobilization of stored lead has been ignored: The document notes the transfer of lead from mother to fetus but does not fully describe the hazards of excessive bone stores in the mother which may be recruited during pregnancy. Similarly, the release of bone stores of lead during aging may provide another kind of risk.

g) Dose-response relationships: The dose-response relationships are the key to benefits estimation, because this provides the basis for quantification. The documents give inadequate discussion to the doses and responses chosen and the appropriate caveats. The approximate nature of the functional form, the perils of extrapolating very far from the median of the data, and of the question of thresholds need to be discussed. Much more attention should be given to discussing the quantification of the dose-response function and what legitimate uses of the equations are.

h) Modeling adult lead uptake and changes in blood lead levels: There is no discussion of how changes in mean blood lead levels

in adults will be predicted as a function of changes in lead emissions.

ECOLOGICAL EFFECTS

The following comments are based on our review of several reports from an outside contractor (Industrial Economics, Inc.) that reviewed valuation methods and their applicability to this Assessment. These reports were provided to the Subcommittee as background. Their review was not part of the charge to Subcommittee.

General Comments

The objective of the documents delivered prior to the review meeting was to present the elements of the methodology by which the EPA will provide a comprehensive analysis of the potential benefits and liabilities from changes in air quality in the United States. The research is designed to evaluate in an aggregate and comprehensive manner impacts due to air quality on: (i) human health; (ii) natural and intensively-managed ecosystems (and at-risk cohorts); (iii) visibility; and (iv) materials. The documents provided by the Agency excluded items ii - iv. Comments are offered relative to items ii-iv in anticipation of how the Agency plans to develop the methodology.

As it often appears to happen in other cost-benefit analyses, human health has dominated this analysis thus far; and it does not appear that ecological effects will catch up within the short time and limited budget that remain for this project. However, one of EPA's missions is to protect ecosystems from adverse effects of pollutants. No other Federal agency has this as a primary mandate (U.S. EPA/SAB, 1990b), whereas other agencies share the mandate to protect human health. It seems likely that this Assessment will once again highlight our lack of information to quantify ecological effects with adequate certainty.

This Subcommittee is concerned that natural resource valuation is likely to be simply an addendum to the evaluation provided for human health. If so, it would inaccurately reflect the mission of the Agency, the overwhelming data demonstrating a linkage between human health and sustainability of natural resources, and the intrinsic value of natural ecosystems (U.S. EPA/SAB, 1990b).

Although consideration of issues of economic valuation is beyond the scope of the Subcommittee charge from the Agency, the Subcommittee offers this additional commentary for consideration. Ecological effects are difficult to comprehensively quantify and value in economic terms. However, the absence of at least a

qualitative analysis that is on equal footing with a human health analysis will be conspicuous and will leave EPA open to sharp criticism. Quantitative valuation clearly has difficulties associated with translating ecological effects into monetary terms in the valuation process (U.S. EPA/SAB, 1990a). Congress and the EPA Administrator should be reminded of this continuing deficiency. Although the value assigned to a single human life and other morbidity effects might at first appear to be so high as to overshadow any other possible cost-benefit categories, some people believe non-use ecological values might be significant relative to the monetary value of human lives -- if only enough effort was expended to fully value ecosystem functions in monetary and non-monetary terms. Until such efforts are supported, Congress will not be presented with a truly comprehensive view of the benefits associated with implementing the CAA. Quantitative ecological risk assessment (Suter, 1993) might provide an approach to deriving a valuation methodology. We encourage the Agency not to ignore this quantitative methodology.

The effort to address physical (including biological and ecological) effects can best proceed if the exposure modeling is done in a way that complements the effects research. In the past, most of the exposure modeling has been done from the perspective of the atmospheric sciences community, whereas most of the effects research is done from the perspective of the biologists. The isolation of the disciplines has resulted in analyses that often can not be linked in the respective camps.

Finally, it was clear that the Congress anticipated that the research would be comprehensive, exhaustive and integrative. However, the picture that emerged in the documents and discussions was one of isolated, fragmented activities without a cohesive framework. Given the scope of the project, it is imperative that the fragmentation be replaced with an approach that is far more integrated. Otherwise, inadequate results are predictable.

Specific Comments

Specific comments on quantification and assessment of ecosystem impacts follow:

a) **Wildlife:** Wildlife were not mentioned in the background documents provided to the Subcommittee, even though surface-water quality and fisheries were mentioned. If human health is a concern regarding rural air pollution, it seems reasonable to

believe that at least some mammalian and perhaps non-mammalian wildlife species might also be adversely affected by rural air pollution. Although little is known about wildlife toxicology, it is likely that there are adverse effects of air pollution on wildlife. This assessment should acknowledge the probability of some adverse effects; therefore, some positive expected benefits of reduced air pollution. benefits of reduced air pollution will not be counted).

b) **Pollutants:** For aquatic ecosystems, acidification (an indirect effect of SO_x and NO_x) and air toxics are much more important than CO, O₃ and PM. The background document appeared to implicitly recognize this. The roles of SO_x and NO_x should be made clear. And the importance of airborne toxics (e.g., metals in addition to lead) to ecosystems should be emphasized. Ecological risk assessment techniques that recently have been developed at Oak Ridge National Laboratory appear to be the most appropriate methods for evaluating the effects of air toxics that are transferred into aquatic ecosystems. Such techniques were not mentioned in the documents available to the committee.

c) **Ecological services:** It is difficult to decipher how this issue is being handled, and we encourage that the document be specific. Ecological systems provide "services" that need to be addressed including nutrient cycling, water processing, air cleansing, recreation, wildlife habitats, pollutant degradation, etc. These are benefits that society derives and values.

d) **Food-web transport:** Ecological systems process pollutants in the environment. In many cases, the processing degrades chemicals to innocuous forms. In other cases, transport and transformation of chemicals through terrestrial and aquatic food webs result in increasing toxicity and in biomagnification. Notable cases are mercury (via methylation) and polycyclic aromatic hydrocarbons (via food web contamination).

e) **Geographical scale:** The scale of the evaluation was not discussed, and the issue is not trivial. Many of the pollutants have residence times that result in their distribution being hemispherical and/or global (e.g., ozone, mercury, trace organics). If the scale of the analysis is simply regional or continental, the assessment process will not capture the significance of changes outside of the continental United States.

f) **At-risk cohorts:** It is widely recognized that there is a standard of error or margin of safety used as a basis for air quality standards, particularly with respect to human health (i.e., appropriate to protect sensitive members of the population). The same concept is also appropriate in some aspects of welfare effects but is handled in separate legislation (e.g., PSD legislation, Endangered Species Act). The emphasis

was supposed to be on at-risk cohorts in both human and non-human effects. Although at-risk cohorts were supposed to be considered in evaluating human and non-human effects, the ecological-effects reports ignored the concept of at-risk cohorts.

g) **Aggregate effects across pollutants:** Will the effort appropriately address interactions among pollutants? The literature strongly suggests that a pollutant-by-pollutant analysis is inaccurate and too simplistic. This also applies to human-health and welfare effects.

h) **Concept of sustainability:** Ecological sustainability involves "impacts on the environment that are irreversible or of long duration compared to human perspectives" (U.S. EPA/SAB, 1990b, p. 35); furthermore, the sustainability of human activities is determined by the resilience of the ecological systems on which economies depend (Arrow *et al.*, 1995). In the ecological literature, this concept has emerged as one means of developing a better appreciation for the value of ecosystems. We encourage EPA to be cognizant of this approach.

APPENDIX B GLOSSARY OF TERMS AND ACRONYMS

ARDS	<u>A</u> cute <u>R</u> espiratory <u>D</u> isease <u>S</u> yndrome
CAA	<u>C</u> lean <u>A</u> ir <u>A</u> ct
CAACAC	<u>C</u> lean <u>A</u> ir <u>A</u> ct <u>C</u> ompliance <u>A</u> nalysis <u>C</u> ouncil (U.S. EPA/SAB)
CAACACPERS	<u>C</u> lean <u>A</u> ir <u>A</u> ct <u>C</u> ompliance <u>A</u> nalysis <u>C</u> ouncil, <u>P</u> hysical <u>E</u> ffects <u>R</u> evue <u>S</u> ubcommittee (U.S. EPA/SAB)
CASAC	<u>C</u> lean <u>A</u> ir <u>S</u> cientific <u>A</u> dvisory <u>C</u> ommittee (U.S. EPA/SAB)
CD	<u>C</u> riteria <u>D</u> ocument
CNS	<u>C</u> entral <u>N</u> ervous <u>S</u> ystem
CO	<u>C</u> arbon <u>M</u> onoxide
COHb	<u>C</u> arbon <u>M</u> onoxide <u>B</u> ound to <u>H</u> emoglobin
COH	<u>C</u> oefficient of <u>H</u> aze
COI	<u>C</u> ost of <u>I</u> llness
COPD	<u>C</u> hronic <u>O</u> bstructive <u>P</u> ulmonary <u>D</u> isease
CV	<u>C</u> ontingent <u>V</u> aluation
d	<u>d</u> eci- (1/10 of a particular unit of measure)
dl	<u>d</u> eci- <u>l</u> iter (1/10 Liter)
EC	<u>E</u> xecutive <u>C</u> ommittee (U.S. EPA/SAB)
EFC	<u>E</u> nvironmental <u>F</u> utures <u>C</u> ommittee (U.S. EPA/SAB/EC)
EPA	<u>U</u> .S. <u>E</u> nvironmental <u>P</u> rotection <u>A</u> gency (U.S. EPA, or "The Agency")
F	<u>D</u> egrees <u>F</u> ahrenheit
FEF	<u>F</u> orced <u>E</u> xpiratory <u>F</u> lowrate
FEV	<u>F</u> orced <u>E</u> xpiratory <u>V</u> olume
FEV ₁	<u>F</u> orced <u>E</u> xpiratory <u>V</u> olume (in one second)
FVC	<u>F</u> orced <u>V</u> ital <u>C</u> apacity
g	<u>g</u> ram
HERL	<u>H</u> ealth <u>E</u> ffects <u>R</u> esearch <u>L</u> aboratory (U.S. EPA/ORD)
H ₂ SO ₄	<u>S</u> ulfuric <u>A</u> cid
IQ	<u>I</u> ntelligence <u>Q</u> uotient
JAMA	<u>J</u> ournal of the <u>A</u> merican <u>M</u> edical <u>A</u> ssociation
K&O	<u>K</u> inney and <u>O</u> zkaynak
LA	<u>L</u> os <u>A</u> ngeles (California)
LOAEL	<u>L</u> owest <u>O</u> bserved <u>A</u> dverse <u>E</u> ffect <u>L</u> evel
m	<u>M</u> oles, also <u>m</u> eters
m ³	<u>C</u> ubic <u>M</u> eters
Micron (µm)	<u>A</u> unit of length equal to one thousandth of a millimeter, or about 0.000039 inch
MRADS	<u>M</u> ultiple <u>R</u> estricted <u>A</u> ctivity <u>D</u> ays
N/A	<u>N</u> ot <u>A</u> pplicable
NAAQS	<u>N</u> ational <u>A</u> mbient <u>A</u> ir <u>Q</u> uality <u>S</u> tandard
NAPAP	<u>N</u> ational <u>A</u> ir <u>P</u> ollution <u>A</u> ssessment <u>P</u> rogram
NESHAPS Pollutants	<u>N</u> ational <u>E</u> mission <u>S</u> tandards for <u>H</u> azardous <u>A</u> ir
NY	<u>N</u> ew <u>Y</u> ork

NO_x Nitrogen Oxides
 O₃ Ozone
 OPA Office of Policy Analysis (U.S. EPA)
 OPAR Office of Policy Analysis and Review (U.S. EPA)
 OPPE Office of Policy, Planning and Evaluation (U.S. EPA)
 ORD Office of Research and Development (U.S. EPA)
 ORNL Oak Ridge National Laboratory (A U.S. Department of Energy Facility)
 PERS Physical Effects Review Subcommittee of the CAACAC (U.S. SAB/CAACAC)
 Pb Lead
 PM Particulate Matter
 PM_{2.5} Particulate Matter (<2.5 μm in aerodynamic diameter)
 PM₁₀ Particulate Matter (<10 μm in aerodynamic diameter)
 PM₁₅ Particulate Matter (<15 μm in aerodynamic diameter)
 ppb Parts per Billion
 ppm Parts per Million
 PSD Prevention of Significant Deterioration
 RADs Regional Acid Depositions
 RADM Regional Acid Deposition Model
 RHA Respiratory Hospital Admission
 SAB Science Advisory Board (U.S. EPA)
 SO₂ Sulfur Dioxide
 SO₄ Sulfate (as in H₂SO₄)
 SO_x Sulfur Oxides
 TSP Total Suspended Particulates
 U.S. United States
 μ micro (10⁻⁶) in combination with specific units
 vs Versus
 VSL Value of Statistical Life
 WHO-EURO World Health Organization, European Region
 WTP Willingness to Pay

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APPENDIX C - DETAILED EDITORIAL COMMENTS

Overview Document

1. On page 3-line 3: say "human welfare instead of "human health."
2. Page 8 - next to last line - It is not clear what is meant by the "likely magnitude of the decision will have..." Do you mean whether the benefit component will disappear in the rounding process?
3. Page 10 - Discussion of thresholds in first paragraph has it backwards.

On page 6, it should say "cerebrovascular" rather than "cardiovascular" strokes.

Paragraph 3 begins with a sentence alleging, simultaneously, two views (relevant and irrelevant) about health effects research.

Paragraph 3 on page 12 is potentially confusing. It seems to state that only positive studies are included in meta analyses. The results of meta analyses are useful only when all valid data are included.

Some uncertainties (page 13) are not addressed. For example, exposure might modify other conditions or behaviors. Ozone might reduce the inclination to exercise, which is assumed to provide health benefits.

When describing epidemiological studies, distinguish prospective from retrospective designs and studies with lots of data on individuals from those with almost no data on individuals. Discuss the role of sample site.

Confounding is a problem with all studies, not just statistical ones. Often it is not possible to control all confounding factors, even all important confounders.

The discussion of thresholds is better phrased in terms of dose-response relationships. A dose-response threshold for a population is different from a dose-response threshold for an individual. Populations have sensitive individuals.

Specific Comments

p.4, 1 4, lines 8-10:

Clinical evidence has been "a" basis for some NAAQS, notably O₃ and CO- It has not been used to set the NAAQS for PM, Pb, or SO₂.

p.4,5, line 5:

Clinical studies have never been used to study asthma "attacks".

P.5,4, line 3:

Differences extend beyond physiology to anatomy, cell distributions and metabolism.

p.7, 1, line 3:

Add at end of sentence "as a primary basis for standard setting".

P. 18,2, line 8:

Reference is made to "two others", but only sulfur oxides is cited. Extend citation to include nitrates.

Page 1-1: (Section 1.1). The interference with macrophage function, mentioned later in section 1.2.8, should be noted here. A recent reference to this effect is not quoted [1].

Page 1-14: first two paragraphs. This quotes the work of Linder *et al.* (1988) and then dismisses it. Linder found that ozone exposure at low levels (less than 120 ppb) altered the anaerobic threshold in athletes performing heavy exercise. Paragraph 1 on this page refers to "a number of concerns" which have been raised about this work. These concerns should be described.

Page 1-56: Table: Under the Health Effect Heading Emergency room visits and hospital admissions - (which now has "N/A" under Functional Form), should appear an estimate of health impact based on the papers I have cited.

COMMENTS - Section 1

p. 1-1, #1, lines 7-8

Delete "is photochemically and".

p. 1-1, #3, line 2

Change "PHYSIOLOGY" to "CHEMICAL REACTIONS".

p. 1-1, #3, Line 2:

Change "decreases" to "can decrease".

p. 1-1, #3 line 6:

Change "(FEF)" to "rates".

p. 1-1, #3, line 11 and p.1-3, #3, line 1:

Insert "laboratory" before "studies".

p. 1-3, #1, line 1:

Insert "controlled laboratory exposure studies in" before "resting".

p. 1-3, #1, line 2:

Change "as low as" to "above".

p. 1-6, # 2, lines 1,2.

p. 1-6, #2, line 11:

Add a review of the recently published paper. "Respiratory effects of low-level photochemical air pollution in amateur cyclists" by Brunekreef, B. *et al.*, *Am. J. Respir. Crit. Care Med.* 150: 962-966, 1994, to the discussion, and the Table 1-2.

- p. 1-6, #5, lines 6-8:
Delete this sentence. It is not based on the cited studies.
- p. 1-10, Table 1-3, entry on Thurston *et al.*, 1993a:
The Results and Comments column should cite the findings that medication usage and symptom frequencies were increased with increasing O₃ conc.
- p. 1-13, Table 1-4:
Add comparable summary information on the Horvath *et al.*, 1991 study
- p. 1-15, #2, lines 7-8:
The statement that "interpretation is difficult" may be true, but the reasons given, i.e., "small sample size", "number of covariates", and lack of "individual exposure data" are not credible.
- p. 1-15, #3, line 11:
What is the basis for the judgement that "other pollutants such as SO₂ and particulate may have been involved" as confounding factors for the responses reported?
- p. 1-16, #2, line 2:
Insert "physician approved use of" before "bronchodilator".
- p. 1-16, #2, line 4, and Table 1-5:
Table 1-5 does not include the study discussed in this paragraph.
- p. 1-18, following #3:
Insert the second # from p.1-19 here.
- p. 1-19, #3, line 3:
Insert "laboratory studies" before "conducted".
- p. 1-22, #2 lines 6, 7:
The statement "However, these results have not yet been fully evaluated." is unacceptable considering the potential crucial importance of these findings. This is especially so considering that they were published in 1988 and 1991!
- p. 1-22, #4, line 4:
Delete "recent". Five-year-old data are not recent.
- p. 1-22, #4, lines 11-14:
Retreat behind a quote from the 1986 CD is not acceptable.
- p. 1-22, #5, line 3:
Change "with" to "following".
- p. 1-23, #4, line 11:
The real-world exposure is always "intermittent".
- p. 1-26, #3, lines 4, 5:
Presumably the reference should be to 96F
- p. 1-27, #1:
A recent paper by Kinney *et al.* (1995) using TSP data and O₃ in a multiple regression analysis suggested that PM was more closely associated with mortality in L.A. than O₃.
- p. 1-28, #2 lines 5-17
As noted earlier, in reference to the discussion in #2 of p. 1-6, the most relevant exposure-response data for the effects of ambient O₃ on pulmonary function are the field study data, and the controlled lab study data provide mechanistic support rather

than the most appropriate primary resource. Thus, everything that follows in this section needs to be revised.

p. 1-28, #4, lines 3-5:

The exclusion of the Avol et al. (1984) data from these analyses is arbitrary and inappropriate.

p. 1-31, #2, lines 1-2:

Shouldn't "decreasing" be inserted before "ventilation"?

p. 1-31, Table 1-10:

An O_3 conc. factor for all outdoor-near road situations is not credible.

p.1-37 #2, lines 4-6:

This interpretation of the asthma epidemiology is both poorly stated and unsupported.

p. 1-38, Section 1.4.6, lines 9,10:

Quantitative dose-response relationships are available now!

p. 1-39, #2, line 5:

As noted earlier (in regard to P. 1-22, # 2) these results are hardly "preliminary".

p. 1-39, #2, lines 8-10:

The recommendation to not use bronchial reactivity and inflammation in the current assessment is not adequately justified.

COMMENTS - Section 2 - Sulfur Oxides

p. 2-1, #1:

The stated goal of restricting this section to the gaseous sulfur oxides has not been explained or justified. It could be justified by a disclaimer that the particulate sulfur oxides would be fully covered in Section 3 - Particulate Matter, if that were indeed the case. This issue will be addressed further in my comments on Section 3. In any case, the stated goal is not followed either, since there is an extensive, if inconclusive text in this section on the historic PM-SO₂ epidemiology.

Most importantly, this section does not come to grips with the role of SO₂ as a precursor of acidic aerosol formation and the ways in which SO₂ emission controls have affected exposures to acidic sulfate aerosols and the health, ecological and visibility benefits of the reductions in such exposures. Without such considerations, this section is woefully inadequate.

P. 2-1, #1, line 5:

What is "a significant concentration"?

p. 2-1, #1, lines 9, 10:

Saying that "SO₂ can be transformed into sulfates (SO₄)", without mentioning H₂SO₄ at all is quite revealing of the superficiality of this discussion.

p. 2-1, #2, lines 7, 11:

Referring to SO₂ here as "sulfur particles" instead of "vapor" or "molecules" shows a woeful degree of ignorance.

p. 2-1, #2, line 13:

- SO₂ cannot be cleared by mucociliary transport.
- p. 2-2, #8:
The sequential discussion of the pre-1986 data and later studies is confusing and ineffective. The discussion needs to be integrated and Table 2-1 needs to include all of the cited studies.
- p. 2-11, #1, line 5:
Add note to refer to Section 3 for further discussion on this issue.
- p. 2-12, #2, line 4:
What is a trivial exposure? Define!
- p. 2-12, #3, line 7:
Does pollution refer to SO₂?, SO₄⁼?, other index?
- p. 2-12, 2-13:
What does this discussion have to do with SO₂?

Specific Comments on SO_x Section #2

- p.21 first # specify averaging time of SO₂ concentrations "sulfur particles" should read "sulfur molecules"
- p. 2-9 specifically refer to the 0.6 to 1.0 ppm studies in the discussion here
- p. 2-17 reference to Table 2-4 mentions a breakdown of the FEV response (exercise versus no exercise), but the Table does not show this.

COMMENTS - Section 3 - Particulate Matter

- p. 3-1, #1, line 5:
Add "soil dust, trace metals, construction and demolition debris, sea salt, etc."
- p. 3-1, #2, lines 2-3:
Insert "aerodynamic" before "diameter"; change "2.5 to 35" to "2.5".
- p. 3-1, #2, line 2 and numerous other places:
Change "µM to "µm".
- p. 3-1, #2, line 6:
Change "condense out of" to "form from".
- p. 3-1, #3, lines 4, 5:
Change "as PM15, British smoke and black smoke (in Spain)" to "black (British) smoke".
- p. 3-1, #4, line 7:
Change "more" to "less".
- p. 3-1, #4, line 10:
Change "is" to "can be"
- p. 3-2, #1, Line 1:
Change "inhaled" to "deposited in the conducting airways".
- p. 3-2, #3, line 3:
Change "this comes at the expense of" to "a disadvantage is that they lack the".
- p. 3-2, #3, line 5:

Insert "some indicator of the concentration of a" before "complex".

p. 3-2, #3, line 17:
Add "factors to contribute to the observation or misapportionment of risk" after "confounding".

p. 3-3, #4, lines 10-12:
Delete sentence. It is redundant with previous text.

p. 3-p. 3-6, Table 3-1, last entry (Brazilian children):
Insert "School Year" under column headed "Other Factors".

p. 3-7, #2, line 2:
A 1989 paper is hardly "recent".

p. 3-7, #3, line 6:
Define "noticeable".

p. 3-10, #2, line 1:
Move "More recently," to the beginning of the second sentence.

p. 3-10, #2, lines 2, and Table 3-3:
The Damokosh *et al.* study should not be cited as an independent study unless reference to Dockery *et al.* (1989) is deleted. The latter includes results from 6 of the 7 cities summarized in the former.

p. 3-13, Table 3-4:
Update the "Authors" refs.

p. 3-14, Figure 3-1:
Needs figure captions and legend for symbols.

p. 3-2, line
Change "particulate air pollution" to "black smoke".

p. 3-15, #1, line 3:
Change "to" to "in".

p. 3-16, #2, line 2:
What does "essentially no SO₂" mean?

p. 3-16, #5, lines 3, 4:
PM₁₀ is essentially equivalent to "thoracic" particles. "Inhalable" refers to a larger upper cut-size, i.e., those aspirated into the nose or mouth.

p. 3-17, #2, lines 10-12:
"low concentrations" is not equivalent to "absence of".

p. 3-17, #3, line 3:
"was present" is incorrect. It could be replaced by "would have exceeded the detection limit" if any comment at all was warranted.

p. 3-17, Table 3-3, Heading:
Insert "ACUTE" before "MORTALITY".

p. 3-17, Table 3-5, Schwartz & Marcus, 1990 entry:
Season was not another factor considered. Their analysis was confined to winters.

p. 3-17, Table 3-5:
Insert summary entries for more recently published papers on mortality in Chicago (Ito *et al.*, *Inhal. Toxicol.* 7: --- 1995) and Los Angeles (Kinney *et al.* *Inhal. Toxicol.* 7: 59-65, 1995).

p. 3-19, #1, lines 5, 6, 9, 18:

- Change "g/m₃", to "µg/m₃".
- p. 3-19, #2, line 1:
Insert "of acute mortality" after "time-series studies".
- p. 3-19, #2, line 6:
Insert "annual" before "mortality".
- p. 3-19, #2, line 10:
Change "Similarly," to "By contrast,".
- p. 3-19, #2 line 13:
What does "similar" mean, in this context?
- p. 3-19, #3, line 2:
Insert "by more than" after "forward".
- p. 3-20, #2, line 8:
Change "long-term" to "monthly average".
- p. 3-20, #4, lines 2, 3:
Wording is wrong.- It should say "2.4% lower FVC for PM₁₀ of 18 µg/m₃, compared to a community with a PM₁₀ of 35 µg/m³".
- p. 3-30, #2, lines 2, 3:
If a conversion to PM₁₀ is needed, it should use the PM₁₅ as a starting basis, rather than TSP whose ratio to PM₁₀ is much more unstable.
- p. 3-31, Section 3.4.1.5
It must be noted that the Burnett *et al.* and Thurston *et al.* papers referred to "total respiratory admissions", "not total admissions". Furthermore, they provided data on asthma admissions separately, and this is a separate category of great interest which should also be considered in any benefits analysis. In fact, it should be further divided into pediatric and adult asthma.
- p. 3-33, Section 3.4.1.6:
This section is incomplete as it stands in several respects. First, it needs to account (as does Table 3-13) for other acute mortality studies that satisfy the selection criteria. Second, it must address the nature and significance of acute mortality in terms of the extent of life-shortening for those who die in excess on polluted days.
Finally, the biggest deficiency is that it ignores the differences in annual average (cross-sectional) mortality rates among communities.
It is difficult to understand what the final # of this section is saying. On the face of it, it seems to be more relevant to cross-sectional mortality than to time-series based daily mortality rates, but the implication of its inclusion here suggests that it is somehow relevant to acute mortality.

Overall Comment

As noted with reference to Section 2 and 5, which were largely restricted to sulfur oxide and nitrogen oxide vapors, it becomes important that Section 3 give adequate consideration to the aerosol forms of the sulfur and nitrogen oxides. While the NO_x aerosol literature is meager and inconclusive, the SO_x aerosol literature is not. There is extensive evidence of health

effects associated with exposure to strong and aerosol (H+), and even more literature relating exposures to health effects associated with exposures to SO_4^- . Whether SO_4^- itself produces such effects is an open question, because ambient SO_4^- may simply indicate the presence of H^+ . In any case, SO_4^- often shows closer associations to the effects than simultaneous measurements of PM_{10} or $\text{PM}_{2.5}$, and is often a major mass fraction of $\text{PM}_{2.5}$.

The SO_x aerosol health effects literature cannot be ignored in the benefits analysis. Either it must be included in a revised Section 3, or alternatively in a revised Section 2.

The other glaring omission in Section 3 is the absence of any discussion of lost-time associated with PM exposures. This is curious, because this has been the topic of previous cost-benefit analyses.

Specific Comments on Particulate Matter Section # 3

p. 3-2 PM can be rapidly cleared by nose-blowing..." or to the pulmonary system by different mechanisms."

p. 3-14 Fig. 3-1 needs a legend

P. 3-25 Remove "Should"

p. 3-26 Must be an error in random-effects slope (does not lie between 0.85 and 1.19)

p. 3-34 Fixed coefficient should be 0.00096 not 0.000096

Specific Comments on Particulate Matter Section # 3

p. 3-30 Is Table 3-10 Annual or Daily PM_{10} ? If Annual, it should be in chronic effects section.

1. Review and evaluate the data on asthma (Whittemore, Alkorn, Ostro et. al., 1991.),

2. Incorporate or compare symptoms in meta study with RADs (Ostro, 1987; (?) Rothschild, 1989) and acute respiratory symptom days (Krupnick et. al., 1990),

COMMENTS - Section 4 - Carbon Monoxide

This Section seemed on track and well done until it concluded (p. 4-14, #1 lines, 5, 6) that: "A concentration-response function based on the Allred *et al.* data will not be developed and used in the current assessment".

Why not? Much weaker and less conclusive results were used for this purpose in other Sections. If the lower-bound estimate includes zero, then say so; but there is no excuse for bailing out on this one.

COMMENTS - Section 5 - Nitrogen Oxides

As noted for Section 2 on Sulfur Oxides, it is defensible to exclude aerosol forms of NO_x from this Section if they are

adequately discussed in Section 3 on particulate matter. However, such an exclusion needs to be made more explicitly. In the case of NO_x, that still leaves all of the vapor-phase nitrogen oxides to be discussed in this Section. Thus, this Section is obligated to review the health effects associated with nitrous acid, and possibly nitrous acid (as well as those associated with NO₂). It fails to do so, and is, therefore, deficient.

Page 5-1: Section 5-1:

This section should stress the effect of NO₂ on macrophage function; if there is a significant relationship between NO₂ exposure and increased lower respiratory illness (as is stated later), it is probably through this mechanism of action. There is also no mention of the relative insolubility of NO₂ - hence its deeper penetration (than occurs with SO₂ for example).

Page 5-3: Section 5.2.1:

The first two paragraphs need tightening and re-drafting. Palmes tubes have been used in recent studies of children's exposure to NO₂ (3). The Neas *et al* study (which is quoted) went to considerable lengths to measure average NO₂ exposure levels of the children being studied. The very detailed Samet study (4) is not quoted in full; but it established that respiratory events in the first eighteen months of life were not associated with indoor levels of NO₂ in houses with no smokers in them.

Page 5-9: Section 5-4.1:

Note four lines from the bottom: "First, Neas *et al* verifies the conclusions reached by earlier researchers. Consequently, greater weight can be attached to the results". What, exactly, is implied here?

COMMENTS - Section 6 - Lead

p. 6-1, Heading 6.1:

"PHYSIOLOGY" is not descriptive of the content of this Section. It covers metabolism, distribution, translocation, and biological effects.

p. 6, # 2, lines 2, 3:

Why is there a 1 µm limit? Suggest changing "greater than 1 µm may be swallowed after clearance from the respiratory tract by mucociliary action" to "may -be deposited on conductive airways in the respiratory tract and cleared by mucociliary action to the gastrointestinal tract".

Section 6.2.2.2 (Effects in Children) is not clearly written. Children are not more susceptible because of their "rapid rate of development." They are more vulnerable because of critical processes, such as synaptogenesis, occurring early in life. Absorption and retention kinetics also play a role. The word, "neurological," is used inaccurately. That term implies overt

disease. This error is repeated elsewhere in the document. Neurobehavioral is more descriptive because of the dependence on population IQ shifts to describe risk. The last sentence is missing a phrase; I assume the relationship is between exposure levels during the early postnatal years and later intellectual performance.

p. 6-1, Heading 6.1:

"PHYSIOLOGY" is not descriptive of the content of this Section. It covers metabolism, distribution, translocation, and biological effects.