February 15, 2000

EPA-SAB-CASAC-LTR-00-003

Honorable Carol M. Browner
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Subject: Review of the Draft Air Quality Criteria Document for Particulate Matter

Dear Ms. Browner:

The Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board, supplemented by expert consultants (together referred to as the "Panel"), met on December 2, 1999 to review the October 1999 draft document, *Air Quality Criteria for Particulate Matter* (EPA 600/P-99/002a), in a public meeting in Research Triangle Park, NC. This was the first review of the new draft Criteria Document (CD) for particulate matter (PM) of the new cycle for reviewing the National Ambient Air Quality Standard (NAAQS) for PM. The determination of the Panel regarding the draft is summarized below, and the attached report describes the Panel's views in more detail.

In his October 20, 1999 letter transmitting the draft PM CD to the CASAC Panel for review, Dr. Lester Grant, National Center for Environmental Assessment (NCEA), requested that the Panel focus primarily on the organization, structure, and presentation of material in the document. This approach acknowledged that additional information published before the document is finalized will be incorporated in subsequent drafts, and that there was no intent that the Panel might close on the document at this stage of its development. Accordingly, the attached report focuses on the structural and strategic aspects of the document, and does not comment on the details of the information presented therein. Panelists were encouraged to provide any detailed comments they might have in writing, and the individual written comments are appended to the summary report for that purpose.

The attached comments provide several points of guidance for improving the organization and structure of the CD. The greatest overall need is to develop a more explicit strategy for selecting the information to be included, distilling key new and pre-existing information into an updated statement of current knowledge, and integrating that knowledge within a recognizable risk assessment framework that flows through the entire document. It is important that the CD focus on information that will best inform decisions on the key elements of the PM NAAQS: the indicator(s) for PM; the concentration, or level(s); the averaging time(s); and the statistical form(s). NCEA faces a considerable challenge in

striking a balance between inclusiveness and selectivity in portraying the burgeoning information in this field. To successfully meet that challenge, it is critical that a strategy be developed and followed consistently to maintain a focus on the information that is most key to the assessment of risk to human health and ecosystems. Rigorous adherence to a well-focused strategy will also be very critical to the timely completion of both the PM CD and Staff Paper. We look forward to your response to our advice.

Sincerely;

/signed/
Dr. Joe L. Mauderly, Chair
Clean Air Scientific Advisory Committee

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U.S. Environmental Protection Agency
Science Advisory Board
Clean Air Scientific Advisory Committee
Particulate Matter Review Panel (FY2000)

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1. GENERAL COMMENTS

The Panel was generally complimentary about the content and quality of the document at this early stage of its development. Although this first draft is incomplete and requires considerable development both in structure and content, it was agreed that it serves as a good starting point for developing a high-quality CD.

There was a general sense that the draft CD attempts a review of existing knowledge that is too encyclopedic and that more attention should be given to selecting and presenting information within a framework reflecting its relevance for decision-making. The Panel recognizes the considerable challenge of presenting an update of current knowledge while also limiting the volume of the document by highlighting the information that is most key to decision-making, but this should be the goal. The information to be considered for inclusion is expanding continuously. To develop a cohesive document in a timely manner, therefore, it is critical to implement both a consistent framework for presentation and consistent criteria for selection. It is recommended that in all chapters, the information be selected and organized within a framework of key issues thought likely to bear most directly on the assessment of human health and ecological risks, and thus decisions regarding the PM NAAQS. These issues or themes should be outlined in the introduction (Chapter 2) and carried through the subsequent chapters in a recognizable manner.

Quantitative estimates of health and ecological risks should be the central theme of the CD. The first draft is oriented more toward a recitation of potential hazards than toward characterizations of risk. Although PM undoubtedly poses health and environmental hazards at some level of exposure, the NAAQS must ultimately focus on, and be defended on the basis of, limiting the level of risk. As the document progresses chapter-by-chapter, the information should be selected, organized, and evaluated in a cohesive manner that frames a statement of our current understanding of the quantitative, as well as qualitative, natures of sources, exposures, doses, effects, and the apportionment of effects among PM, co-pollutants, and other factors. The strategy for developing the CD should focus on information that will inform decisions on the four key elements of the NAAQS: 1) the most appropriate indicator(s) for PM; 2) the PM concentration, or level(s) of the standard; 3) the averaging time(s); and 4) the statistical form(s) of the standard. Although it is the role of the Staff Paper, not the CD, to set forth the principles upon which options for the PM NAAQS are developed, the need to identify and defend those principles should underlie the strategy for focusing the CD.

2. COMMENTS BY CHAPTER

Only general comments regarding the structure and content of the individual chapters are summarized here. The individual Panelist's written comments, which are appended to this report, should be reviewed for detailed comments on these issues and other detailed suggestions.

2.1 Chapters 1: Executive Summary, and 2: Introduction

Discussion was not directed specifically toward these chapters. It is expected that Chapter 2 will describe, in an explicit manner, the strategy followed in developing the document. This will be an important prelude to the selection and interpretation of information presented in subsequent chapters.

2.1 Chapter 3: Physics, Chemistry, and Measurement of Particulate Matter

There is a general need to update the information in this chapter. As one example, the portrayal of size distributions does not reflect the most recent information.

There is too much emphasis on the Federal Reference Method and too little portrayal of alternate sampling techniques and their comparative interpretations and advantages. Data acquired using other sampling methods are also useful and in some cases may be superior.

It would be useful to include a discussion of potential alternate ways of categorizing PM. PM is presently categorized by size (coarse, fine, etc.), but a discussion of the feasibility and utility of alternate categorizations, such as by composition or source, would be valuable.

2.2 Chapter 4: Concentrations, Sources, and Emissions

There is insufficient emphasis on modeling and on the combined use of modeling and measurements to estimate the regional concentrations that, together with local sources, result in local urban PM concentrations.

A better discussion of the speciation of organic PM and the organic fraction of combined organic-inorganic PM is needed.

Trends in emissions need to be discussed more thoroughly. The chapter needs a better discussion of trends in the contributions both primary PM emissions and emissions of precursors of secondary PM formation. The information on vehicle emissions is badly out of date.

2.3 Chapter 5: Human Exposure

This chapter could be organized in a more focused manner that allows a reduction in length.

The information in this chapter overlaps with that in the following chapter (Chapter 6, Epidemiology), and needs to be better linked to the following chapter. For example, measurement error should be discussed in this chapter, to set the stage for discussion of the implications of those errors in interpreting epidemiological data. As another example, the term "confounding" is used in different contexts in the two chapters.

The information linking outdoor and indoor exposures needs better integration. The general view presently conveyed that the only important issue is the correlation between indoor and outdoor concentrations is misleading. Other perspectives, such as the influence of indoor exposures on susceptibility to outdoor exposures, potential synergisms between indoor and outdoor exposures, and the contribution of precursors generated outdoors to formation of PM indoors should be mentioned. The extent to which observed health effects are due to outdoor vs indoor exposures is not argued convincingly.

The extent to which data from central outdoor monitors represent personal exposures of individuals and populations needs a more critical discussion. There appears to be an underlying assumption that a good correlation between central monitoring data and personal exposures is an established fact accepted from the last CD, when that is not necessarily true.

2.4 Chapter 6: Epidemiology

The organization and integration of the large amount of information in this key chapter need attention. The daunting nature of the task of extracting key conclusions from such a large number of disparate studies requires that a disciplined approach be developed, stated, and followed. The arrangement of the chapter into review and discussion sections may be appropriate, but the lack of a transparent strategy for selecting and summarizing the information results in the chapter appearing as somewhat of a "patchwork" of information. The chapter should be introduced by an explicit statement of the strategy used to select the information (studies) to be included, the criteria applied in evaluating the studies and their results, and the process followed in synthesizing the information. Having developed and stated such a strategy, the information can then be presented and summarized in tables in a more consistent, readily-understood manner. A more explicit strategy will also help reduce the repetition of the descriptions of studies and their results in the latter portion of the chapter.

Several issues bearing on the conclusions need to be dealt with in a more rigorous manner. Care should be taken to avoid presenting the information in a manner that appears focused on supporting conclusions drawn in the last review of the PM NAAQS. As noted in the preceding section, better descriptions of exposure issues in chapter 5 should set the stage for a better treatment of exposure assumptions, as well as other potential sources of measurement error, in this chapter. The extent to which co-pollutants are sometimes equal or stronger predictors of effects is not given sufficient

treatment. The potential role of the coarse fraction of PM_{10} is given insufficient attention. The heterogeneity of results across studies and the likely impacts of particle characteristics, meteorology, measurement error, and modeling techniques on differences among results should be given more attention. Based on a more explicit consideration of these factors, the Agency should provide an assessment of whether or not current views of the strength and consistency of relationships between PM and health differ from those at the time of the last review of the PM NAAQS.

The chapter needs a more consistent definition and terminology for statistical significance, and a more analytical treatment of the likely precision of effects estimates in the various studies.

Caution should be exercised in using meta-analyses to integrate information from multiple studies. Meta-analysis is most appropriately applied to studies that are similar in design. The present approach tends to ignore the considerable heterogeneity among studies in lag structure, accounting for confounders and co-variates, stratification of data, and smoothing techniques. Integrating the findings by simpler graphic techniques may be preferable to meta-analysis.

The discussion of causal inferences and the drawing of conclusions regarding causality might better be moved to Chapter 8 (integrative synthesis). Either in this chapter, or in Chapter 8, the issue of exposure-response relationships must be discussed in a straightforward manner. The extrapolation of responses from high exposures to low and the potential existence of thresholds in responses, will undoubtedly be a focus of discussion regarding the Agency's ultimate decisions on the NAAQS.

2.5 Chapter 7: Dosimetry and Toxicology

This chapter should be divided into separate chapters on Dosimetry and Toxicology. Dosimetry does not pertain solely to toxicological studies. Both dosimetry and toxicology are important topics that cut across human and animal studies and warrant individual attention. Combining them neither reduces the total verbiage required nor adds clarity to the presentation.

Both the dosimetry and toxicology material would be enhanced by providing an introductory, or "road map" paragraph at the beginning of each section. Much of the information is presented as paragraph-by-paragraph descriptions of studies with little apparent framework.

Dosimetry

This material would be enhanced by adding a figure depicting regional deposition vs particle size, rather than just depicting total deposition. A figure comparing total deposition in normal lungs with that in abnormal lungs would also be useful, and information on regional deposition in abnormal lungs should be added if available at the time of revision. An important new dimension has been added by studies showing markedly increased deposition in human subjects having lung abnormalities.

The section on comparative dosimetry from inhalation vs instillation exposures could be shortened. A paper from the Society of Toxicology discussing this subject in detail is in press, and the issue needs only to be summarized as it applies to the interpretation of results in this chapter.

Section 7.2.7 would be enhanced by figures or tables showing representative results from models predicting the amount of material retained in the lung. In portraying the available models, it should be kept in mind that the overall purpose of the chapter is to provide a basis for linking exposure, dose, and effect in view of regulatory decisions.

When discussing deposition, it is important to distinguish among total deposition, regional deposition, and the amount of retained material (which differs from the deposited dose).

Toxicology

An alternate approach to organization might enhance the presentation of the information on toxicology. One alternative is to begin with an introduction, and then to discuss inhalation studies (human and then animal), and then instillation studies (human and then animal), and then in vitro studies, all to be followed by a summary of key findings and conclusions. Another approach would be to arrange the information according to key research question, such as important PM characteristics, mechanisms, dose-response, and susceptibility, integrating across study types for each category.

Throughout the toxicology chapter, it is important to maintain a focus on exposure-dose-response relationships, and to put the findings in context regarding ambient inhalation exposures. No findings should be discussed without mention of the exposure mode and dose. Premium should be placed on conveying an understanding of effects, mechanisms, susceptibility, etc. in the context of the likelihood that the phenomena would occur at current ambient exposures. Information on particle size is also important.

The toxicology chapter should incorporate information on diesel particulate matter. Regardless of the separate document dealing specifically with diesel, it is a common component of ambient PM, and cannot be disregarded in the PM CD.

2.6 Chapter 8: Integrative synthesis

Much of this chapter consists of summaries of the foregoing chapters, and it presently functions much better as a summary than as a synthesis. The chapter needs greater focus on integrating across the preceding chapters to answer key questions and form conclusions that will lead into the Staff Paper. Examples of key issues include: 1) advances since the last CD in our understanding of the nature, magnitude, significance, and timing of PM-related effects; 2) subpopulations at risk and the levels of exposure posing substantive risk; 3) the types and sources of PM posing greatest risk; and 4) the specificity of the risks to PM (among co-pollutants and other factors).

The focus should be on the nature and exposure-response relationships for human responses to PM; other types of information are important only as they support or clarify the human findings.

2.7 Chapter 9: Environmental Effects

As for the preceding chapters on human health, the material on ecosystems should have a "risk assessment" orientation. For example, the information could be better focused on answering the question, "are ecosystems and their linkages to human health and welfare at risk from PM deposition?" The present organization does not link plants and ecosystems to humans very well, and appears to draw few conclusions. The information in the chapter does not seem to provide a very strong basis for considering the need for, or the cost-benefit characteristics of alternate levels of, a secondary PM standard that differs from the primary standard.

The long-term trends in deposition and accumulation of PM in ecosystems should be discussed. The times over which ecosystems can be burdened by deposition of PM can be longer than the lengths of human exposures.

The issue of the deposition and potential effects of the several different classes of PM-borne organic compounds is largely missing from the chapter. This issue needs to be discussed, and several relevant references were provided by the Panel.

The impacts of nitrates and other PM-borne species on watersheds are not discussed sufficiently. PM is deposited in waterways by both direct deposition of PM in water and indirect deposition by storm runoff in urban areas. Nitrate of PM origin may stimulate increased growth of biomass in the water.

Studies examining the effects of several types of particles on plants are summarized, but this review does not comprise a coherent picture of the nature and magnitude of impacts on different classes of vegetation. Some classes, such as fast vs slow-growing plants and coniferous vs deciduous trees, are mentioned, but there are a number of other plant characteristics that may be related to vulnerability to PM. Although the present literature does not allow a comprehensive analysis of the issue, the literature should be discussed with this focus and information gaps should be acknowledged. It will be necessary to integrate across the scientific disciplines represented in the chapter to develop and adequate synthesis of the impact of PM on plants.

The purpose of the section on economics is not presently clear. This draft gives a very limited introduction to the potential benefits of air quality improvements. Because both costs and benefits may be considered in setting the secondary standard, there should be a better discussion of costs.

APPENDIX A
DETAILED COMMENTS OF INDIVIDUAL PANELISTS

The following are the original, unedited written comments provided by individual Panelists prior to or at the December 2, 1999 meeting. They do not reflect consensus of the Panel and, in some cases, may have been revised subsequent to the meeting as a result of discussion. They were provided to the Agency following the meeting so that Agency staff would have detailed editorial comments as well as individual responses to the Charge. The material in this Appendix, along with the discussions at the December 2nd meeting form the basis for this written report. (Note: these comments may contain uncorrected typographical errors that result from electronic translation).

Joe L. Mauderly

Summary Comments

Overall this draft is a very good start on the next PM Criteria Document. For the most part, it provides a suitable framework on which the CD can be built. From a structural viewpoint, I think the dosimetry and toxicology sections should be different chapters.

This document falls far short of describing the current situation with diesel exhaust and its relationship to ambient PM. Despite the fact that a separate health assessment for diesel is being written, you can't ignore diesel here. It constitutes up to 30% of the fine PM, and you certainly spend time and verbiage on materials that constitute less. One of the long-term effects of concern is cancer. That isn't treated adequately in the present draft. That is one place that the diesel issue must be brought in. Allergenicity is another. Deciding what to do with diesel in this document is problematic. The one option that won't work, however, is to ignore it.

Chapter-by-Chapter Comments

Chapter 1: Executive Summary

1-7, Section 1.2.5: Something should be mentioned here about national trends in PM levels as well as regional trends. Actually nothing of significance is really said about either.

1-10, 3-6: The speculation about long-term effects being caused by retained dose doesn't belong here. The speculation doesn't make much sense in the first place. The following sentence makes a lot more biological sense.

1-12, Table 1-1: Why not add qualifiers to the first row as you did the others (e.g., likely, not likely, etc.)?

1-12, 13-14: This sentence doesn't make much sense and its value is not clear, although it may be true.

1-13, 23: Don't you mean particles containing metals instead of "metal particles"? There are very few "metal particles" in the environment.

1-13, 28: Here, you equate "urban air particles" to "combustion-related particles". Surely you don't think that all PM in urban air are the result of combustion.

Chapter 7: Dosimetry and Toxicology

There is not need to combine these two topics. It would be better to have separate chapters.

7-2, 12: Insert "fully" after "cannot". Animal models can not FULLY duplicate all the features of human diseases, but they certainly can duplicate selected features of human diseases, as the sentence goes on to state.

7-13, 17: The authors looked at particles retained on carinal ridges of cadavers, but they had no idea what portion of the particles were deposited there. Particles also reach those locations by clearance.

7-14, 10: Do you mean that females had higher total or fractional deposition?

7-16, 24: I think you mean that children have a higher size-specific dose. They may not have a higher absolute dose.

7-22, 26: Throughout the chapter, "soluble" and "insoluble" are used to denote particles that do or don't dissolve significantly compared to normal clearance time. I understand the point and its importance, but I think this is sloppy terminology. We ought to be more precise, or someone will take us literally. Few, if any, particles are truly "insoluble", and to say that a particle that is dissolving or leaching material is insoluble just because most of them may clear before much dissolution takes place seems misleading.

7-35, 7: Here, we have the term "poorly soluble". Is this different from the meaning intended by your earlier working definition of "insoluble"?

7-43, 27: The Nikula study included monkeys as well as rats.

7-45, 25: 100 micrograms per cubic meter isn't "fairly low".

7-45, 30-31: What animal species was this?

7-46, 1st paragraph: Again, what animal species are we talking about here?

7-47, 24: What is your definition of "macrophage clearance" here? Are you inferring "macrophage clearance" from a lack of infectivity, or was there some more specific measure?

7-49, 8: Do you really mean "90-fold"? I haven't heard of one going that high.

7-50, 3rd paragraph: What would the "human equivalent" concentrations be? When comparing responses of human and animals, comparative dose should be considered.

7-50, Section on Human Exposure to Other Particles: Why not cite the studies of Anderson et al. At Rancho Los Amigos in which they exposed humans to carbon particles and acid, separately and together?

7-54-60, Table 7-2: Abbreviations are not used consistently in this table.

7-61, 30: I doubt that even the most ardent ROFA fans would posit that ROFA is a "surrogate for ambient particles" – at least not in the sense of total ambient PM. There may be a few particles in the air that resemble ROFA, and indeed a few ROFA particles themselves, but ROFA isn't much of an ambient PM surrogate.

7-77, 23: Why not use the abbreviation "ROFA" here? ROFA is being used for a whole collection of different oil fly ash particles collected in different ways, so why not just stick with it?

7-78, 30: I think you should qualify your use of "adult" here. I suppose that most studies have used animals that have reached breeding age, and in that sense, might be considered "adults". However, by far the most studies have used rodents with lungs that are still growing rapidly, and representing lungs of older children and adolescents more than they do adults.

7-86, 15: Very little of the diesel particulate in urban PM is in the ultrafine mode. Although diesels (and other engines) do emit ultrafines, it is misleading to equate diesel particulate with ultrafines.

7-88, 18: Was this fuel oil fly ash "ROFA"?

7-89, Table 7-5: Again, was there really a difference between FOFA and ROFA? I recognize that the authors may have used different terms, but using different terms here implies that the reader should not equate them. Is that your intent?

7-97, 7: I think you might want to re-word the sentence. It is not true that characteristics that <u>may</u> be responsible for lung injury are not known – lots of them are known.

7-98, 9: Who cares if the centrifuge was on a bench? Just give the gs.

7-98, 20: I didn't notice where "POBN" was defined.

7-101, 26: What kind of PM was the "charged" PM?

7-103, 26 and 28: Give the ages. "Young" and "old" rats don't tell us much – those terms are used in a variety of ways, and often inappropriately.

<u>Chapter 8: Integrative Synthesis</u>

8-3, 14: In table 8-1, it is stated that the nuclei mode can be translocated 10s of km. That's not "near".

8-5, 24: It should be "ensures" instead of "insures".

8-7, 20: Elemental carbon also comes from gasoline engines.

8-7, 30-31: The biogenic material isn't listed under "fine" in table 8-1. Should it be?

8-8, 28: The statement that fly ash is similar in composition to soil is misleading. Although many elements are the same, fly ash is typically enriched in some elements, such as metals.

8-16, Figure 82-: Why doesn't PM10 cross the 50% line at 10 microns?

8-18, 14: I disagree that the retained dose is likely to be more important for chronic effects. The hypothesis seems to have a certain logic, but there are so many factors determining retained dose that it'' probably not a very useful concept. As an extreme example for illustrative purposes, this means that continued or repeated exposures to acids and other soluble materials can't have any chronic effects because they aren't measurable as a "retained dose". I think that repeated deposition of toxic materials will prove to be more important than the retained dose of some old particle "skeletons".

8-21, 6: Somehow this statement doesn't seem right. Even if you don't know the mechanism, there could be a quantitative relationship between dose and effect can't there? I think I know what you mean, but the wording isn't quite there yet.

8-22, Table 8-4: Why not use the same terminology established elsewhere in the document (e.g., ET, TB, A)?

8-23, 11: I'd argue that the newer information doesn't "complicate" our situation at all, it just point out how complicated the situation always was. The tone of the sentence implies that we'd be better off without new knowledge, a meaning I don't think is intended.

8-27, 28: What does "non-effects" mean?

8-47, 21: Are you really talking about just the UF fraction of diesel particulate (which is a very small portion of total diesel particulate) or are you making the mistake of calling diesel particulate ultrafine, which it mostly isn't?

John Elston

I was surprised that this Chapter did not contain any review of the impacts of nitrates, and other particulates (including air toxic particulates) on waterways. It is becoming increasingly apparent that particulate matter either directly entering waterways by air deposition or indirectly, through storm runoff in urban areas contribute significant loading to estuaries and other waterways. In the case of nitrates, I was told, the environmental effects are contained in the criteria document for nitrogen dioxide. From the standpoint of the organization, it seems to me, that separating environmental effects within specific criteria documents may lead to an underestimation of these effects. I would therefore suggest that one way to handle this is to develop Volume 3 as a stand alone document and include in it all of the particulate environmental effects, including those covered in other criteria documents.

Philip Hopke

My major comment with respect to the Criteria Document is that it is still too encyclopedic and is not sufficiently integrative and evaluative in nature. There needs to be a sufficient summary of the information in the literature, but there should be a greater effort to summarize that information into conclusions that indicate what we know with reasonable certainty, what we know with greater uncertainty, and that which we are quite unsure of. This then needs to be integrated into how this relates to the subject at hand which is the exposure to
airborne particulate mater and effects arising from that exposure. These effects include both health and welfare effects. The atmospheric chemistry is really only relevant to the estimation of exposure and it would be better to focus it more on where we need to go with measurements and models of particle emission, production in the atmosphere, transport, deposition, etc and resulting exposures of people and things that result in adverse effects. We have too much material in the document discussing what we know without clear direction toward the ultimate goal which is to provide a scientific basis for determining if the current regulatory environment is providing adequate protection of public health and welfare. This information is interesting and important for a variety of purposes, but not necessarily for determining public policy toward air pollution. I think that the writers need to keep firmly fixed on the purpose of the document and at each step in the process ask if the material being presented is directly relevant to the adequacy of scientific basis for taking regulatory action. If it is, then make sure that relationship is clearly articulated. If it cannot be succinctly and clearly articulated, then it really does not belong in the CD.

I verbally indicated that I thought there needed to be some additional material on modeling, dispersion, source, and exposure. The context again would be on how the atmospheric models combined with measurements could provide estimates of regional concentration upon which the urban concentrations are built through the emission of local sources. Receptor and dispersion models might provide an initial basis for beginning to examine the relationships between adverse effects and source emissions. Given the high level of composition complexity in PM, we have to look at ways to aggregate exposures. It does not seem likely that we can look at effects on a component by component basis, but we might be able to look on a source by source basis. The only way one would get the data needed to begin to statistically look at such relationships will be through a combination of both models and measurements. It seems to be we have to start moving away from the paradigm that it is some specific component or limited number of components that will be the causal agents of adverse effects. Rather it may be the collection of particles from particular source types (cars, diesels, oil-fired power plants, etc) that needs to be the point of focus. We will not have sufficient data at this time to be able to make any sensible changes in what we do about particles and this may not be the right way to aggregate the problem, but if we do not start looking at a variety of other approaches, we may not be able to come up with a sensible approach to improving air quality that really makes a difference with respect to health and welfare effects. We need to start looking at a number of ways of carving up the problem and this document is an appropriate place to raise these issues.

Eva J. Pell

Comments on Chapter 9

Chapter 9 provides review of direct and indirect effects of a variety of particulates on vegetation, both through direct and indirect effects. Impacts on ecosystems are also considered. The review reflects the literature which is a collection of studies examining different particulates and different plant species. There is neither a way from this review, nor from the literature in general, to predict impacts on classes of vegetation. There is some effort to speak of fast versus slow growing plants, and deciduous versus coniferous tree species, but there are many other distinguishing plant characteristics that could dictate vulnerability to particulates. The subject is massive, and in contrast to better studied pollutants like ozone, there really is still a lot to learn. Below are some specific comments.

Page 9-6 line 6-8 The statement that the majority of stomates are on the undersurface of leaves is true for some plants but there are many plants with equal numbers of stomates on both leaf surfaces.

9-8 12 The paper of Cape (1993) is about forest trees and the reference refers to crops. A long list is ascribed to Cape. It is hard to know what to make of this.

9-10 12 On what effects is the statement suggesting that the Marchinska and Kucharski (1987) study found little effect of SO_2 and heavy metals containing PM on beans, carrots, and parsley based? What kinds of effects were not detected?. This sounds very global.

9-10 15 Reduction in lichen species abundance is a characteristic of lichen populations in response to many things including toxic metals, SO_2, O_3, and other environmental assaults.

9-10 19-20 On what effects is the Nash, 1975 observation that lichen tolerance fall between 200 and 600 ppm based?

9-18 27 The statement that "Chronic pollutant injury to a forest community may result in the loss of sensitive species..." is a dangerous statement. It infers that genes are lost. There is significant evidence that sensitive individuals can become scarce but when pollution abates the sensitive individuals reappear suggesting suppression rather than loss of genes. The statement does not say genes are lost but that could be inferred.

9-20 24 The statement "Dust accumulation favored some species and limited others." Does this statement suggest a direct differential response of species to dust, or is it a matter of competitive advantage of the less tolerant individuals? This seems to be the interpretation put forward on the next page.

9-21 21 Are the authors sure that “early needle senescence and abscission in the San Bernardino Forest...” can be attributed to particulates? Early senescence of needles on ponderosa pine in this forest has long been associated with O_3.

9-38 21 Emphasis is placed on “Toxicity from cadmium, cobalt, and lead...” only being seen under unusual conditions. I have always been of the impression that toxicity is quite unusual. But what is really relevant is bioaccumulation and implications to other members of the food chain.

Sverre Vedal

Epidemiology (Chapter 6)

1. The organization of this part of the draft CD into study review sections (sections 2 and 3) and discussion sections (sections 4, 5 and 6) is reasonable. However, more attention should be given to restricting the Discussion to brief reviews of the major findings reported since the previous CD for each topic without presenting new epidemiological data or repeating much of what was presented in the review sections. New presentations, such as the detailed review of the Burnett hospitalization studies, would be best included in the study review section. There is generally too much repetition in the discussion sections of the material of the study review sections. It was valuable, however, to present findings of simulation and methodologic studies in the discussion sections where indicated.

2. One must sympathize with those who attempt to compare effects from a multitude of studies whose analysts have had to make independent decisions regarding the specification and reporting of the final models used in the analyses. Some difficulties that result that complicate comparisons and meta-analysis include:

a) the variable specification of lag structure, either choosing various individual lags (sometimes only the "best" lag, with the obvious bias that this entails) or different ways of averaging a variable number of lags (see point 1 under the Mortality section below),

b) the different lag structures for meteorological and co-pollutant covariates,

c) use of different smoothing techniques and smoothing "windows",

d) the inclusion of various combinations of co-pollutants and meteorological covariates in the regression models,

e) the variable stratification by age group or season.

Those who attempt to synthesize data exhibiting such differences across study have a difficult time. Difficulties in doing this, and some oversimplifications that the syntheses must make, should be acknowledged by those who attempt it and the findings appropriately qualified. Given these complexities, and given that some sort of summarization or synthesis is needed, some unified approach would be ideal. However, traditional meta-analysis techniques would not appear to be up to the task of handling such complexity. Probably the most reasonable approach would be to keep the meta-analyses to a minimum unless a point needs to be made about a group of relatively homogeneous studies.

3. Since the last CD, a great deal of new epidemiological data have accumulated on PM health effects. The current draft comes across as attempting to shore up the "party line" rather that seriously

addressing some interesting features of the new data. I have been struck by some features of these new data that are not well reflected in the current draft.

First, as more studies have seriously addressed the roles of co-pollutants, either as confounders or as independent predictors, it has become clear that the associations with PM are sometimes confounded by co-pollutants, and that co-pollutants are often equally strong predictors, and sometimes stronger predictors, of the outcomes of interest as PM.
Second, as more data specifically addressing the role of the coarse fraction becomes available, it is becoming less certain that effects are primarily due to the fine fraction (with possible implications for the pathogenicity of non-combustion particles).
Third, there is a significant amount of heterogeneity in PM effects across study, and therefore across city. The argument regarding consistency of effects has been weakened by these newer data. This presents opportunities for investigating sources of heterogeneity, such as particle sources/characteristics, meteorology, measurement error, population susceptibility, and adequacy of modeling.
A much more balanced and open approach is expressed in part of the Integrative Synthesis (section 8.4.1 and 8.4.2, pp. 8-21 through 8-28), an approach that I would recommend as an example for chapter 6 as well. These sections will hopefully not be lost as the document is modified.

Introduction (section 6.1)

6-2 -Table 6-1 is difficult to figure out and therefore of little help here. However, it does become clearer when the same table is presented in the Conclusion. More effort to be clearer in the Introduction is needed, or else you risk putting readers off at the outset.

minor points:

6-2 -(line 14) random variability or population susceptibility are other possibilities.

Morbidity (section 6.2)

1. It is difficult to discern a rationale for including some studies in more focused tables while other studies are relegated to an "other studies" table (e.g., p.6-27, table 6-18).
2. There are attempts in this section and elsewhere to single out both sulfate and acid aerosol (p.6-55, line 28) as showing stronger effects than other particle measures. This is by no means a consistent finding and should not be emphasized.

minor points:

6-21 -section 6.2.1.2. additional studies relevant to non-asthma need to be included here

6-36 -(line 20-22) or due to less exposure misclassification.

6-43 -(1st and 2nd para) interpreting the Burnett study 1997 as a positive study and supportive of the role of acid aerosol is dubious, both of which could be debated.

6-55 -(line 4) I would not single out elderly with COPD since the point is not convincing (table 6-22).
-(end of 1st para) there is little sense in comparing SO4 and PM effect sizes.

Mortality (section 6.3)

1. The issue of using "best" lags surfaces here. Figure 6-2 (p.6-84) is misleading in attempting to show a lag structure incorporating the findings of many studies. Some studies did not evaluate lags beyond 2 days. Others only reported "best" lag which may not have been much different from other lags. A different approach is required if this point is to be addressed.
2. Given the very small number of lung cancer cases that form the basis of the findings reported in the Abbey and Beeson papers, is it necessary to dedicate the number pages (6-113 and beyond) and tables to a discussion of these findings? As a minimum, the discussion should be qualified by noting the small number of cases.

<u>minor points:</u>

6-83 -(line 10-11) "best" lag issue again.
6-95 -(line 30) "relative error' needs better explaining - is this misclassification?
6-102 -(line 22) measurement error and its effect on dose-response I do not believe was discussed in the text prior to this point, so that this summary is the initial presentation of this topic.
-(line 12) discounting the effects of the coarse fraction is one-sided.
6-105 -(line 2) reference in these cohort studies to effects being due to "long-term exposure" is speculative.
-2nd para - comparing effects of cohort and time series studies remains problematic. Line 22 is a clear overstatement.
6-111 -Table 6-28, male LCL and UCL for SO4 must be wrong.
6-126 -Section 6.3.3.7 discusses only particles and not co-pollutants as the title would suggest.
6-128 -Table 6-37 essentially duplicates what has already been presented in Table 6-32 (p.6-118).
6-129 -This Discussion section is too focused on responding to 2 critiques rather than taking a broader perspective.
6-131 -Line 25 - be careful about claiming biological plausibility.

Discussion (section 6.4)

This section should begin with discussions of the "big" issues needing to be addressed since the last CD: particle size, study consistency, confounding and other issues relating to co-pollutants. The begin with a discussion of effect sizes and significance testing, while relevant, makes the document lose focus. The discussion also gets bogged down in repetition and detail (e.g., tables 6-39 to 43, pp.6-149 to 155) of findings already presented in detail in the study review sections.

6-135 -All of section 6.4.2 relating to specific diseases and age groups is generally not very helpful in its present form. First, it should come later since it distracts from the "big" issues. Second, it is repetitious of material presented earlier and does not take a sufficiently fresh tack to merit the repetition (especially last para, 6-138 and last para, 6-139 which is repeated better on the next page).

6-157 -In this sub-section on "PM size distribution and composition", studies that do not make use of the coarse fraction (that is, only have PM2.5) are much less informative. I would recommend dividing up this discussion into 2 sections, the first including discussion of the few studies that analyze effects of the coarse fraction, the second discussing the more numerous but much less valuable studies that do not.

-Also, there is too much detail on the specific studies for a discussion section (e.g., a very detailed presentation of the Burnett studies is included); this should be in the study presentation sections.

minor points:

6-136 -(line 9) Why are visits and hospitalizations better outcomes for more severe asthma?

6-142 -Comparing effect sizes from cohort vs. time series effects still has an "apples and oranges" flavor.

6-145 -The goal of using harvesting resistant estimators should be separated from that of assessing effects of long exposure periods, regardless of attempts to link them by original authors; the latter issue has not been pursued with adequate rigor to this point and is still very open to interpretation. That is, harvesting resistant estimators do appear to show that the effects are not merely due to harvesting. The interpretation of what these estimators are in fact showing is not known. For example, do the longer time windows adequately remove temporal cycles from the data? Your bias is showing when the cohort studies are brought up in this context (line 3), since even effects on the time scales assessed in the harvesting studies would likely not show up as differences in mortality rates in the cohort studies.

Epidemiology and causal inference (section 6.5)

A discussion here of the validity of the parallel time series study as a study design, a topic that has not received much attention, would be very helpful. Other study designs (case-control, cohort, even case-crossover) have received a good going over with respect to validity. This has not been the case for the time series design, or if it has, I am not familiar with it. This is a failing given the amount of data that has been generated in the air pollution epidemiology field using this design, and the great weight that is necessarily placed on the findings of such data.

Conclusions/Discussion (section 6.6)

minor points:

6-207 -"Negative" does not normally mean that the effect estimate is in the non-hypothesized direction, merely that it is not "positive".

6-213 -(line 11) singling out effects of SO4 and acid aerosol again, when this is probably not supported by the data.
-Table 6-57 helps to interpret initial presentation of this table in the Introduction (6-2) where it was difficult to understand.

Synthesis (Chapter 8)

This is overall an excellent chapter. It functions well as a summary, but probably less well as a synthesis. Again, I would hope that sections 8.4.1 and 8.4.2 do not get lost in the revision process, since they were excellent.

Warren H. White

Though it has by now achieved the status of "boilerplate" prose, it remains nonetheless true that "[a]irborne PM is not a single pollutant, but rather is a mixture of many subclasses of pollutants with each subclass containing many different chemical species" (CD page 1-3). We would never consider setting a NAAQS for "the mass concentration of trace gases", or even "the combined concentration of sulfur dioxide plus ozone", but we have historically approached the regulation of particulate matter in just this way. The new (1997) PM NAAQS represented a significant advance by resolving PM into two distinct classes of PM, fine and coarse, but otherwise continued the historical approach. In particular, the 1997 NAAQS employed the gravitational mass of particles in a given size range as their indicator of biological potency.

PM can be partitioned into distinct classes along many different dimensions: in addition to particle size there are morphology (e.g. droplets *vs*. particles *vs*. crystalline fibers), chemical composition (e.g. acid sulfates *vs*. sulfate-nitrate-ammonia salts *vs*. organics *vs*. metals), and source type (e.g. diesel exhaust *vs*. coal smoke *vs*. mineral dust *vs*. photochemical haze). Similarly, PM levels can be specified in terms of various measurable indicators besides gravitational or chemical mass, including particle count, surface area, and light scattering coefficient. The 1996 PM CD and its SP focused on establishing a vocabulary for the classification of PM by aerodynamic particle size, generally converting other indicators of PM concentration into their estimated equivalents as PM10 or PM2.5 mass. They did not undertake to articulate a rationale for selecting PM2.5 mass over other possible indicators for "fine-mode" or "combustion" aerosol.

I am disappointed to find that the draft 2001 CD largely contents itself with the fine/coarse dichotomy bequeathed by its predecessor. At our present state of knowledge, the distinction between fine and coarse mass may indeed be the best framework available for regulations. But if its Staff Paper is to make that argument, the Agency will need at least the vocabulary to discuss alternative taxonomies. I would like to see this CD lay the groundwork for that discussion. To illustrate the issues that need to be considered, I offer below a comparison of the 1997 NAAQS indicators with an example alternative. The point I mean to make is not that "my" alternative is better than the Agency's,

but that alternatives are conceivable. We are still at an early enough stage in our approach to PM that the Agency is not yet bound by precedent and tradition. I suggest that the CD maintain at least the appearance of an open mind toward the characterization of PM, unconstrained by the anticipated recommendations of the SP.

To illustrate the case that needs to be made for PM2.5 and PM10 (or PM10-PM2.5) mass as PM indicators, let us consider an alternative pair: sulfate and soot. A NAAQS for sulfate and soot would have implications for PM monitoring and management, and would have to be justified in terms of public health. I will consider each of these issues in turn.

Reference measurement methods. The reliable measurement of PM2.5 mass poses vexing challenges. Sample collection requires careful attention to the aerodynamic rejection of particles greater than 2.5 um in diameter, particularly in dusty areas. Sample collection and handling can produce poorly characterized losses of volatile particulate matter. Gravimetric analysis must be repeated before and after sampling, and requires equilibration of the sample. Sulfate and soot could be determined on the same Teflon filter now used for mass, and samples could be collected with no change in the existing FRM. For these predominantly fine species, however, there would be little need for aerodynamic sizing, and requirements for the maintenance of precise inlet specifications could be relaxed. Since sulfate and soot are both stable constituents, concerns for losses in sampling and handling would be similarly eased. The sulfate background of a Teflon filter is negligible, so blank determinations and corrections could be eliminated. The measurement of soot on Teflon by light absorption is more problematic, involving both blank corrections and controversial calibrations. Nevertheless, it is routinely carried out throughout the IMPROVE network, and yields one of that network's most precise outputs. (Alternatively, of course, one could add an aethelometer or similar instrument for a real-time soot measurement.)

Air quality management. The indicators selected for NAAQS influence attainment strategies in indirect as well as direct ways. As an example, some would argue that an excessive focus on secondary ozone in the battle against photochemical smog encouraged an over-reliance on mathematical modeling to the neglect of observational programs to verify claimed reductions in primary emissions. PM2.5 mass, with a typically substantial secondary component and no one dominant primary source category, prompts similar concerns. Sulfate and soot are more directly related to identifiable source categories: combustion of coal and oil in the first case, diesel engines and open burning in the second. These linkages would improve our ability to maintain the accountability of future control programs.

Health effects. Perhaps the strongest case for mass rather than chemical species as the PM indicator is that mass covers all possible toxins. "What if we control sulfate and soot and it turns out to be (e.g.) the metals that actually kill people?!" There are several responses to this legitimate question. 1) Metals (or ultrafines, or bioallergens, or ...) are trivial contributors to fine particle mass. Attacking metals by regulating fine mass is analogous to attacking sulfate by regulating TSP. To attack specific minor constituents, we must go after them rather than aggregate mass. 2) Indicators can be revised in future

NAAQS, just as TSP evolved to PM10 and then to PM10 plus PM2.5. We can add new indicators in addition to subdividing existing ones in the light of new knowledge. And we already have the Teflon filter samples -- we can just add metals to the analyte list. 3) In practice, major reductions in sulfate and soot would entail lessened emissions of some other pollutants, including metal-bearing fly ash and some nasty organic species. And in any case, evidence implicating metals would not necessarily exonerate sulfates and soot!

Sulfate and soot are just one set of indicators out of many that can be proposed, including some suited to continuous, real-time monitoring. Once again, the point is not that they or any other indicators are superior, only that we need an articulated rationale for preferring fine and coarse mass.

Paul J. Lioy

General Comments

The Chapter has been designed specifically to support use of ambient PM levels as an indicator of exposure within epidemiological studies for the general population. From this specific vantage point, the National Center for Environmental Assessment of EPA has completed a reasonable analysis; however, the Chapter is overly long, and the analyses and text can be reduced by 40%.

The Chapter's emphasis on associations which supported the cited epidemiological investigations; however, has seriously diminished its usefulness by the scientific and regulatory community. The basic problem is the lack of any information that establishes the critical role of exposure analysis in defining the dose of the contaminants of concern which is essential for explaining the etiology of PM related disease outcomes. This is a fundamental error.

The document must move beyond specific issue of associations in epidemiological studies, begin laying the foundation for quantifying exposures, and then quantifying or estimating the dose (acute or chronic) which may cause an effect. This is a well established scientific paradigm, one which is available in at least three articles by Lioy, and articles by Sexton and Zartarian. A balanced discussion on the issue of PM exposure and dose can then be used in combination with emerging dosimetry and toxicology data to examine PM ($PM_{2.5}$, PM_{10}, $PM_{0.1}$, and PM composition) cause and effect relationships.

It must be remembered that the effects of PM air pollution at current levels are suspected to be experienced by subgroups of the general population. These individuals may have sensitivities, pre-existing conditions, or participate in a variety of activities that can lead to exposures of concern.

Therefore, it is incumbent upon the EPA to recognize that individuals within a subgroup at risk have a variety of simple and complex exposures to PM, (e.g. PM size fractions, PM sources, and PM

chemical constituents). Further, exposures to the lung cannot be neatly differentiated or segregated into ambient PM, indoor PM and personally generated PM, as well as, gaseous species. Acknowledgment of these scientific realities, and then using the results of current and future studies will define the opportunities to establish cause and effect relationships, and ultimately the selection of the sources and situations to control and/or mitigate.

Specific Recommended Changes

One obvious, but important first step is to separate discussions of air pollution PM exposure by "size fractions": Total mass, PM_{10}, $PM_{10-2.5}$, $PM_{2.5}$ and ultrafine. Some of these are currently not available, but at least you will not mix information and statements and observations for one size fraction with those associated with other size fractions.

The authors must make an effort to define the chemical composition for outdoor, indoor and personal PM in different size fractions. For indoor air, the chapter must move beyond primarily tobacco smoke (especially direct inhalation), and for outdoor air, the chapter must move beyond $S0_4^{-2}$ ion.

Ambient PM_{10}, $PM_{2.5}$ and ultrafine particles are associated with two main dynamical and mechanistic schemes: fall and wintertime sources and processes, and summertime sources and processes. Ambient exposure must be characterized within these two schemes and information provided on potential contact, activity patterns, penetration indoors (e.g. AC/no AC, type of home). Plausible chemical and physical components of concern need to be simply characterized, and coupled, if possible to the exposure and dose that may cause health effects. At this time the latter may not be much more than a list of hypotheses and any information on the presence of specific species in outdoor air.

The authors strongly discounted indoor air pollution by making any relevance tightly bound to the variability needed to explain the epidemiological studies. However, the approach is flawed because we must begin to acknowledge that an exposure must lead to a dose of concern for PM and/or its physical and chemical components before it can cause an effect in individuals at risk. If all the toxicity could be ascribed to outdoor air this discussion would not be necessary. Unfortunately, we know little about the "silver bullet," and in some cases it may be a mixture derived from various origins, with outdoor air being the indicator for this potentially toxic mixture. Thus, the document needs to be able to provide a framework that can at least be used to develop plausible hypotheses about exposure/dose, and then exposure/dose-response relationships as new toxicological information becomes available, and as we identify the individuals or subpopulations who suffer and situations that cause adverse health outcomes.

The following are critical issues, and there may be more:

Indoor air pollutants are a heterogeneous mixture that will change based upon personal life style, socio-economic issues, and housing etc. However, for a given individual or group of individuals the

exposures may be relatively constant or consistent (e.g. smoking, indoor combustion or indoor chemistry). Thus, indoor PM can provide individuals at risk with a baseline dose that can be added to by outdoor air pollution. Relatively constant indoor levels can provide a baseline dose to the lung, which if exceeded by individuals at risk by outdoor air could lead to adverse outcomes.

Indoor air pollution, both gases and particles, may act synergistically with outdoor air pollution to cause serious health outcomes. An obvious example is asthma. Do indoor irritants leave individuals at greater risk to an outdoor pollutant known to produce an asthma attack? Could other indoor air pollutants act with outdoor air pollutants found during high winter or summertime PM episodes to cause an affect? The latter is un-studied therefore, unknown.

Indoor air chemistry, the vast frontier of unknowns with respect to enhancing or decreasing the toxicity of outdoor pollutants as they penetrate indoors. Most people spend vast amounts of time indoors, and we do not know if the individuals truly at risk to a disease outcome have been affected by outdoor air modified by indoor air chemistry. Thus, the toxic substances and toxic dose that may be produced must be defined clearly. In some cases the importance of indoor air chemistry will be of concern primarily on high air pollution days. Specific, obvious examples which cannot be ignored in the criteria document include:

Synergistic property of indoor air ammonia reacting with outdoor H_2N0_3 to form particulate $NH_4\ N0_3$ indoors.

Antagonistic property of indoor air ammonia reacting with outdoor air $H_2\ S0_4$ and $NH_4\ HS0_4$ to form the less toxic compound $(NH_4)_2\ S0_4$ (Waldman et al, and Su et al).

Synergistic property of the formation of organic species (e.g. aldehydes and organic acids) and ultrafine particles by reactions of ozone from outdoor air with indoor generated limonene from typical household air fresheners (e.g. Weschler et al, 1999 and Zhang et al, 1994). You will be surprised about the quantity of limonene emitted per year!

Synergistic property of the deposition of ultrafine elemental carbon, organic carbon and metals (emitted at ~10^9 particles/min.) generated by motors (Lioy, 1999) and fossil fuel combustion (wood) on accumulation mode outdoor air particles.

None of the above negate the associations established by the outdoor air epidemiological studies. However, they begin the process of realistically defining plausible complex mixtures that need to be examined, and characterized as we begin to develop exposure-dose-response relationship, and then consider control strategies for reduction of health effects. Remember, as I stated in my presentation, associations may lead to correlation, but they do not make the poison. Exposures leading to a biologically effective dose(s) will make the poison.

Specific Comments

Chapter 5

Pg. 5.4. EQ 5-1 Missing interaction terms – for generation of particles indoors by chemistry with outdoor pollutants.

Pg. 5.7. Lines 9-15. But $S0_4^{-2}$ is only a cation – the anion H_2 is potent, NH_4H is less potent, and $(NH_4)_2$ is even less impotent at ambient levels. So $S0_4^{-2}$ variability is not representative of the toxic exposure because, it is easily neutralized indoors.

Pg. 5.7. Line 25 Also, if the wind is blowing and you do not have AC.

Pg. 5.7-5.8. Last and First lines Motors generate ultra fine particles (e.g. Vacuum cleaners and can redistribute on both coarse and fine particles.) Lioy et al, 1999.

Pg. 5.9. Lines 29-31 Yes! Because different effects are based on more than variability.

Pg. 5.10. Lines 11-19 This is a manifesto that ignores the need for mechanistic studies of exposure which can be used to estimate dose. Please eliminate or modify.

Pg. 10. Lines 28-29 And, if one has a constant source of indoor PM and other chemicals they may react with outdoor PM to make the mixture more or less toxic in indoor air. Or, these can set up the lung for insults that are additive (threshold) or synergistic/ antagonistic –a hypothesis yet to be explored.

Pg. 9-11 Indoor air does not have to vary to be related to effects, just could increase to baseline dose.

Pg. 11. Lines 8-14, 26-31 Also, must consider Indoor x Outdoor chemistry, so please add a term.

Pg. 17. Not only ammonia outdoors, Waldman showed ammonia neutralization indoors.

Pg. 17. Also, there are ultrafines indoors emitted from vacuum cleaners and other motors, and wood combustion.

Pg. 18. But, what about variability around the mean?

Pg. 18-19. Never once mention neutralization processes that occur indoors.

Pg. 20- 23. No indoor air chemistry – although I provided the author with a conceptual diagram in April, 1999.

No terms in the equation provide for particle formation by chemistry.

Pg. 24. Disagree. Chemistry of O_3\ turpines, combustion products will yield similar compounds indoors and outdoors. Further, you can have a baseline level (irritants, carcinogens, mutagens) indoors/and then you add on the variable concentrations of outdoor PM. The Dose = Dose Indoor (baseline) + dose outdoor (variable).

Pg. 25. Lines 14-23 Also, emissions of fine and ultra fine particles from motors. May not be large amounts of mass, but contain large numbers of particles with elemental carbon and metals.

Pg. 5.30. Good points about distribution of personal exposure, but not represented in figures, you only "chase" the mean. Requires box and whiskers plots around mean of personal exposure values.

Pg. 5.30. Equation needs the interaction term $C_{og} \times C_{Ig} = C_{oI}$ PM, o= outdoor, I= indoor, g=gas.

Pg. 5-32-34. You keep focusing on the mean, but the mortality accounts of $\leq$ 1% of all annual deaths. You should consider the 95% tile of exposures.

Pg. 5-40. Indoor levels increase the intercept, and baseline PM exposure for a population. Variability, unless levels are extremely high, would still be dominated by outdoor PM (confusing statements on lines 7-11).

Pg. 15-41. Are you saying that the dose from indoor source can be infinite and only that PM which contributes to outdoor variability will contribute to toxicity – a new concept in toxicology.

Pg. 5-41. Lines 18-25 But, neutralization alters the character of the aerosol.

Pg. 5-41. Lines 27-31 Totally agree – needed to establish these individuals actually at risk. Once we know who is at risk, we need to define the distribution around the mean of personal exposures and how individual exposures are correlated to outdoor exposure. Caution: We may not be able to separate the synergisms.

Pg. 43. Lines 10-15 This is a hypothesis that is not proven, needs to be studied. What about the intercept? Need to look at additive and synergistic doses. Composition is also a problem. This is too rigid a hypothesis based on current data.

Pg. 44. Lines 4-15 Should be the starting point for this Chapter.

Pg. 5-44. Line 18 The assumption of linear dose – response relationship is really just a hypothesis.

Pg. 5-45. Lines 16-20 Yes, and these may be the people with the dose of concern when looking for populations at risk. The authors also need to include ambient/indoor chemistry, and synergisms among indoor/outdoor particles.

Pg. 5.52-53. Shows the problem you get when you study homes that have large indoor sources which may use combustion sources. A PAH is a PAH!

Pg. 5-55. Lines 14-18 Rethink your interpretation. Invariate and consistent indoor sources provide a baseline for incremental dose.

Figures 5-8, 5-9, 5-10, 5-11 A/B No variation has been reported. No explanation for the intercept. You see the information in provided tables, but reader cannot easily visualize. The Figures require box and whiskers for personal data

Pg. 5.66 Lines 20-27 So what. Do these people represent reality in US? Or did they just obtain sedentary people who were probably very interested in having something to do!

Pg. 5-67. Are the people in Baltimore's elderly home truly representative of a population at risk? They did not do anything. In contrast, the COPD patients showed very large variability and low correlation with ambient monitors. Why wasn't the COPD data shown graphically?

Pg. 5-72-76. You left with the feeling that $S0_4^{-2}$ is the only component of the PM that matters. Do not ever talk about neutralization indoors. Waldman, Sa., separate references – Reduction in toxicity. There is absolutely no discussion on organic particles – generated outdoors or indoors.

Pg. 79-80. The mean in PTEAM had large variability because of indoor sources and the mix of people. Need to report the variability around the personal mean concentration (Figure 5-10).

Pg. 88-96. Needs to be rethought; include more hypotheses. Also, the idea that eventually the dose of the mixture may make the poison, and not variability of PM or specific fractions, needs to be added to the discussion.

Mort Lippmann

General Comments

I have reviewed Chapters 3, 4, 5, and 6 and believe that the draft materials generally provide a suitable level and depth of coverage of a rapidly growing literature. I had the most trouble with Chapter 5, which I found to be too diffuse and pedantic. It needs to be tightened up and focused on the issues relating to human exposures to PM of outdoor origin.

While reviewing the draft CD, I noted a large number of needed editorial and/or technical changes that may be useful to Dr. Grant and his team. These are listed below.

Chapter 3

p. 3-2, l. 5: change "is" to "can be". The first definition is inconsistent with Friedlander's definition on lines 9-11. (also on p. 3-83, l. 8)

p. 3-3, l. 4 cite other recent texts as well, e.g., Hinds (2nd Ed.), Vincent, and Willeke and Baron.

p. 3-3, l. 13: add "and cyclones" at end of line.

p. 3-3, l. 14 insert "distribution" after "diameter"

p. 3-14, Fig. 3-7: explain large discrepancy between the curves labeled "manufacturer" and "Tsai and Cheng (1996)"

p. 3-19, l. 25: insert "ultrafine" before "particles"

p. 3-31, l. 28 & p. 3-32, l. 3: HNO3 dissolves into water droplets, which are carried into small lung airways and alveoli (See: Schlesinger et al., Inhal. Toxicol. 6:21-41, 1994).

p. 3-32, l. 11: add "also" at end of line

p. 3-55, l. 17: change "mm" to "μm" (same error at many other places in text)

p. 3-68, l. 4: delete "all" and clarify the message here

p. 3-80, l. 7: change "were" to "where"

p. 3-81, l. 5,6: totally confusing text

p. 3-84, l. 6,7: delete "generated in the laboratory"

p. 3-84, l. 13: insert "hygiene" after "occupational"

p. 3-87, l. 8,9: change "effects" to "risks"

p. 3-87, l. 22: replace "volatile" with "gas-phase molecules such as water and semivolatile organic compounds"

p.3-87, l. 23: delete "species"

Chapter 4

p. 4-2, l. 26: update reference to later year's annual report

p. 4-3, Figs. 4-1a,b,c: The text must comment on, or explain how 22.8% of PM2.5 can "unknown" in the eastern U.S., while the sums of the knowns in the central and western U.S. exceed 100%.

p. 4-7, l. 9: insert "Peak" before "twenty-four hour"

p. 4-17, Fig. 4-10: shouldn't the last line of the figure legend refer to Fig. 4.9?

p. 4-39, l. 20: insert “design,” before “age”

Chapter 5

p. 5-2, l. 23: insert “for convenience” after “zone”
p. 5-2, l. 26: delete “personal monitoring”
p. 5-3, l. 1: change “adequately characterized” to “estimated”
p. 5-7, l. 10: insert “somewhat” before “lower”
p. 5-9, l. 29: change “is the most” to “may be an”
p. 5-9, p. 31: add “, provided that one assumes comparable biological responses to PM components of indoor and outdoor origin” at end of line
p. 5-10, l. 9-21: This text is more of a sermon than a fair discussion of the limitations of the available literature. It should be rewritten or deleted.
p. 5-11, l. 21-25: The inclusion of “Outdoor PM” as a separate category is rather odd. It may warrant inclusion as a footnote at best in a more general note that special circumstance sampling does not fit into the general discussion in the text.
p. 5-13, l. 16-28: The inclusion of a category of “Exposure to Personal PM” is also rather odd. It should also be treated as a footnote, if at all.
p. 5-13, l. 12: What do the “r2 = 0.50 and 0.01” refer to? Clarify or delete.
p. 5-21, l. 15: insert “some” before “PM”
p. 5-28, l. 3-31: This discussion is not relevant. It should be deleted.
p. 5-24, l. 18: change “little” to “few”

Chapter 6

p. 6-97, l. 19: insert “and” at end of line
p. 6-102, l. 24: replace “in” with “confined to”
p. 6-104: The fact that the ACS population was of generally higher SES than that of 6-cities should also be discussed.
p. 6-135, l. 7: change “hospitalized” to “hospitalization rate”
p. 6-135, l. 16: change “which yielded” to “and reported”
p. 6-136, l. 25: change “more” to “greater”
p. 6-137, l. 5: change “conducted analysis” to “analysis that was conducted”
p. 6-137, l. 17: insert “an exposure on” after “from” and a common after “before”
p. 6-138, l. 12: change “for” to “associated with”
p. 6-139, l. 3: change “yields” to “reported”
p. 6-139, l. 13: change “possible” to “likely” and “may” to “would”
p. 6-181, l. 27: explain how nitric acid vapor can be a component of PM2.5
p. 6-211, l. 15,26: “SO4” has wrong font

Robert Rowe

Unfortunately I was unable to attend the December 2, 1999 review meeting due to illness and have missed the benefits of the informed comments of the presenters and other reviewers. As a result, my comments are focused only on the environmental effects and economics discussions in Chapter 9.

General Comments

From my review of the documents, the draft CASAC letter appropriately covers most of my key recommendations regarding the structure and presentation in the overall Draft Criteria Document. On the health side, in addition to the issue of "understanding the nature and magnitude of PM-related effects" (as identified in the draft CASAC letter comments on Chapter 8), add the "significance" and "timing" of these effects (we are to prevent against adverse health effects). I also recommend that the draft CASAC letter comments on Chapter 9 make clear that the same issues discussed for the environment "effects" presentation also apply to the "economics" presentation.

Additional Specific Comments on Draft Chapter 9

Generally, the purpose of Chapter 9 is unclear, and the level of detail (issues, models, data, literature citations) varies considerably across the sections. I am left with the feeling that the Chapter shows that PM, at some levels, impacts environmental endpoints and has some economic values, and some of these impacts and values may be measurable, and others may not. While a useful introduction, the chapter provides little to evaluate the significance of these impacts at alternative levels that may be considered for the secondary standard. Are there measurable and environmentally, or economically, adverse impacts at ambient concentrations, at current standards if met, or at alternative potential standards? Would some of this information provide insight into the form of the secondary standard (e.g., particle size, duration of elevated concentrations, thresholds).

What is the purpose of the economics section. As it presently stands, it presents a very limited introduction that there are potential economic benefits of air quality improvements. The inclusion of economics, and its coverage, needs to be made clear. As the secondary standard can consider both benefits and costs, why are costs not also addressed? The presentation of benefits is very limited and does not help address the standard setting questions at all. How will this tie into the RIA – what is the role of information here versus information in the forthcoming RIA? Presently the economics section is rather scant compared to other sections in the Draft Criteria document, to the CAA retrospective and prospective benefit-cost studies, or to other similar reviews (Chestnut et al., 1999 assessment for Title 4 for the U.S. EPA; Rowe et al. 1995 on environmental impacts of electricity generation in New York, and many other similar documents). The introduction of concepts and methods is particularly limited.

Other editorial comments include:

Page 9-14 mentioned Constanza et al (1997). Perhaps this should be carried into the economics section, with the Freeman et al. response article also cited (I do not have that reference readily available. Dr. Freeman can be contacted at Bowdoin College).

Page 9-78 discusses the deciview index and perceptible changes. It would be appropriate to also mention the L.R. Richards paper in AWMA, October, 1999 on this topic.

In terms of visibility, how does the regional haze rule overlap with or influence the considerations to visibility in the PM secondary standard?

Many of the references are incomplete.

In the economics section:

Page 9-101 paragraph 1 discusses the economic significance of effects in monetary terms of "costs". This is incorrect. Use "benefits (or damages avoided) from reduced ambient concentrations. " This will also clean up the jump between benefits and damages in paragraph 2. Later (page 9-105) the text jumps without explanation to "consumer surplus" as a value measure. The concept of economic surplus is poorly addressed in this introduction.

I seldom see the term "contingent choice" (page 9-101) in favor of conjoint and other choice based methods (e.g., a movement away from use of the term "contingent").

Hanley and Splash seems like an unusual reference when there are many North America studies and compendiums available since 1993, and there are other studies on ecosystem functions (including the Constanza and Freeman papers).

The agriculture and forestry section identifies there are models, but nothing about the potential economic significance of these impacts at current ambient concentrations, or at current or proposed standards. What is the point of the section (a point that generally applies to all of the subsections under economics).

The presentation should consistently refer to groups of methods as (1) Revealed preference methods and (2) Stated Preference methods. RP methods use market prices and methods based on actual behaviors and associated costs and values, all of which are used to reveal values. There are many methods here – avoided costs, mitigation costs, wage and price hedonics, factor price methods, production models, math programming models (e.g., Ag. models), as well as the simple use of market prices per unit of injury. The SP models are used to directly elicit values …..

9-102 just before 9.7.4 (and anywhere else) should consistently use "stated preference valuation methods" to refer to a group of methods of which CV is one (rather than referring to CV as the approach).

Under valuation methods for materials damages, I find the term "damage function approach" (DFA) likely to be confused with that term as more generally used in the literature and would avoid it used here as is done. More generally, the DFA term refers to the chain of assessment from emissions to impacts and values, much like as presented on the top of page 9-105, but does not mean the valuation is based on a cost based method. Usually, in a DFA one is measuring benefits with the best available measure with costs only used as a poor proxy for values (benefits and costs are different). So, rename this to a replacement/repair cost approach, or explicitly alter the last step to be "value the impacts" with costs used as a proxy for value (recognizing that costs and damages are different.

The materials damages section does not provide information useful to selection of a secondary standard. Losses from studies are not related to current or proposed levels.

Under cultural materials, there are valuation studies by Morey and co-authors, the papers and references for which can be obtained from http://spot.colorado.edu/~morey/discuss.html (note, there are about three different papers/reports for the same work on this web page). From these papers, there are many other references available.

Page 9-107, I don't find NAPAP 1996 in the references. Is this to refer to the SOS paper on visibility valuation done earlier. I think the whole literature on urban visibility valuation and on property values studies is missing here as are many individual studies and any understanding of the relationship of the studies to current or proposed standards, which again begs the question of what is the intent of this section?

Page 9-107 raises the issue of perceptible changes. This has long been a bothersome issue. If many sources each contribute an imperceptible impact, the sum of individual damages should not be assumed to be zero because the sum of the impacts may be perceptible (and even significant).

Page 9-109 third line from the bottom. If we have more specific impacts we may be able to do "valuation studies" rather than just "CVM studies" as stated. For instance, if fish populations (and fish catch rates) are impacts, methods other than CVM are readily available. Even for other impacts, other stated preference methods may be preferred to CVM.

Jonathan Samet

Overall Comments

This draft Criteria Document is evaluated for its structure and for the approach of the authors, rather than offering specific, detailed comments about the contents. It is understood that the draft is in-progress and that further changes will be made as further data become available. Nonetheless, a number of general issues related to approach are already evident, as follows:

The approach taken for development of the evidence included in the Criteria Document needs to be better specified. The process should be "transparent" and "replicable", in each of the chapters. A lack of transparency and potential replicability is evident in chapter 6, which presents the findings of the epidemiologic results. Here, the authors state "Those papers selected here as being most clearly relevant to this NAAQS review are described in greater detail in the text of this chapter, and the other are included in tables where appropriate." The generality of this approach is not sufficient. For each body of evidence, the chapter authors should indicate the search approach and selection criteria.

There is substantial inconsistency across key chapters, particularly those related to exposure and health effects in handling statistical significance and in using attainment of statistical significance as a decision-making criteria for accepting or rejecting evidence. This inconsistency in approach needs to be resolved. In my view, statistical significance has little role to play in determining whether a finding is useful; rather, emphasis should be placed on the amount of evidence available or on the precision of estimates.

Better integration might be achieved across the elements of the Criteria Document by using the source-health effect paradigm, which has long been used for air pollution and was most recently the basis for the research portfolio of the National Research Council's Committee on Research Priorities for Airborne Particulate Matter.

Chapter 1

This chapter serves as the Executive Summary. It might appropriately be used for integration of the evidence and evaluation using the causal criteria, now in chapter 6. A review of the evidence based around the causal criteria should not be left to the epidemiology chapter, but rather viewed as overarching and drawing on all relevant information.

Chapter 5

This chapter addresses human exposure. There is substantial overlap with chapter 6, but the chapters have not been adequately integrated or cross-referenced. This chapter should play the role of setting up a consensual framework for the material presented in chapter 6. In particular, it should consider the types of exposure measures used in the epidemiologic studies and their validity. Measurement error might be discussed in this chapter, setting out our understanding of errors in

exposure estimates, while chapter 6 might consider the implications of these errors for data interpretation. Regardless, better integration of chapters 5 and 6 is needed. There is also excess reliance on the concept of statistical significance (see page 5-95) for example.

In this chapter, the term "confounding" is used in reference to indoor PM. The authors of each chapter should be consistent in their use of the term "confounding" and recognition of the assumed causal model. In epidemiologic parlance, a confounding arises when the exposure of interest (in this case, outdoor particulate matter) is itself associated with another exposure that is linked to the health effect of interest. There is a distinction between the possibility of confounding and actual confounding, which is consistently lost throughout the document. Additionally, the assumption of "confounding" assumes independent causal effects.

Chapter 6

This massive chapter reviews the epidemiological evidence. In its current structure, it is a patchwork that is clearly the product of multiple authors. Careful and thoughtful revision is needed. I have the following suggestions:

The chapter should begin by setting out a framework for the evaluation of the evidence. I suggest the following: 1) describe the criteria used to select the included studies; 2) describe the criteria used to evaluate the evidence; and 3) describe the process used to synthesize the findings. The document currently lacks any such framework.

1. This initial framework should replace the present Introduction. I found Table 6-1 to be too difficult and arbitrary in its classification of potential hypotheses. It fails as an approach for organizing the chapter and should be removed.

2. The document would be improved by tables that provided evaluations of the individual studies in some systematic study, rather than study descriptions which are quite variable from study to study.

3. There is substantial inconsistency across this chapter in terminology and approach with regard to the statistical significance of findings and the precision of effect estimates. Principles are stated and then disregarded, repeatedly.

4. Meta-analysis is used as a summary approach. I am not sure that providing a summary estimate is either helpful or appropriate. There has not been an attempt to systematically gather all relevant data, so that any summary estimate may be biased. Additionally, there is substantial heterogeneity that is ignored with a simple quantitative summary. I am in favor of using more graphic approaches for providing the results of the individual studies. With more complete characterization of the studies, effect estimates might then be provided by relevant categories, i.e., location within the United States.

5. As noted, the material on causal inference might best be placed in the Executive Summary.

George T. Wolff

Chapter 3

p. 3-17, lines 21 – 25 – I think this should be labeled *Sulfates and Nitrates* and a sentence about nitrates should be added.

p. 3-20, line 5 – Insert "peroxy radicals" after hydroxyl radical.

P. 3-20, lines 12 – 13 – This sentence is not true for nitrates especially in the East.

P. 3-85, line 9 – insert "peroxy radicals."

Chapter 4

p. 4-3, Figure 4-1a – Isn't there any new data that can be used to make these graphs?

p. 4-25, Table 4-3 – Under anthropogenic organic carbon "any source of VOCs" should be added.

P 4-33 – A section on emission trends of primary and secondary processors is needed. This section should also tie the emission trends to the ambient concentration trends.

P. 4-42, lines 1 – 4 – There is a disconnect between this section and the section earlier on natural sources.

P. 4A-11, discussion on vehicle emissions – This is out of date. EPA should query their own Office of Mobile Sources and their R&D group to update this. Not only are the numbers old, they are obsolete.

Chapter 5

General Comments – The arguments that were made in the previous CD about the validity that a central monitor is a representative measure of exposure of everyone in the area were not convincing to a number of the previous CASAC PM panelists. Therefore it cannot be assumed by the agency that EPA that their conclusions in the previous CD are now accepted as gospel. A balanced discussion is needed here. As presently constituted it is written to attempt to justify the Agency's previous claims. Some critical discussion is needed.

Chapter 6

General Comments - I agree with many of the criticisms offered by Drs. Lipfert, Samet and Vedal. There must be someway to provide an objective, unbiased process to the evaluation of these studies. The uncertainties associated with the conclusions drawn from these studies must be critically and objectively evaluated. I question whether the present authors are capable of doing this as the citations

include their own work as well as the work of others who came to similar conclusions. A critical evaluation of the work of others would call into question their own conclusions. The recent CO CD was much more objective and they came to the opposite conclusion.

Chapter 8
Table 8-2 – See chapter 4, comment 2.

p. 8-13 – line 31 – What about the studies that identify $PM_{(10-2.5)}$ as the most significant culprit?

My comments on chapter 6 apply to the epidemiology discussions in this chapter as well.

Chapter 9
Section 9-3 – What is missing from this section is a discussion of deposition trends in the US.

2. Section 9.5.8 – This section devotes only 1 sentence to the fact the in most of the U.S. visibility is presently improving. It devotes most of the remainder of the section to those places where visibility may not be improving. This is unbalanced.

The remaining comments are not available in electronic format and must be obtained in hard copy from the SAB staff.