UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

March 27, 2007

EPA-CASAC-07-003

Honorable Stephen L. Johnson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Subject: Clean Air Scientific Advisory Committee's (CASAC) Review of the 1st Draft Lead Staff Paper and Draft Lead Exposure and Risk Assessments

Dear Administrator Johnson:

The Clean Air Scientific Advisory Committee (CASAC or Committee), augmented by subject-matter-expert Panelists — collectively referred to as the CASAC Lead Review Panel (Lead Panel) — completed its review of the Agency's 1st Draft Lead Air Quality Criteria Document (AQCD) in September 2006 (EPA-CASAC-06-010). On December 7, 2006, Mr. Marcus Peacock, the EPA Deputy Administrator, issued a memorandum providing his final decisions on revisions to the process by which the National Ambient Air Quality Standards (NAAQS) are reviewed. In this memo, Deputy Administrator Peacock directed that this revised NAAQS review process should begin with the current, ongoing review of the NAAQS for lead. (See URLs: http://www.epa.gov/ttnnaaqs/memo_process_for_reviewing_naaqs.pdf and http://www.epa.gov/ttnnaaqs/naaqs_process_report_march2006_attachments.pdf).

On February 6–7, 2007, the CASAC's Lead Panel conducted a peer review of EPA's *Draft Review of the National Ambient Air Quality Standards for Lead: Policy Assessment of Scientific and Technical Information* (1st Draft Lead Staff Paper, December 2006) and a related draft technical support document, *Lead Human Exposure and Health Risk Assessments and Ecological Risk Assessment for Selected Areas: Pilot Phase, Draft Technical Report* (Draft Lead Exposure and Risk Assessments, December 2006). In addition, on March 9, 2007, the Lead Panel held a public teleconference to review the CASAC's draft letter to the Administrator resulting from its February meeting. The CASAC roster is found in Appendix A of this report, and the Lead Panel roster is attached as Appendix B. The charge questions provided to the Lead Panel by EPA staff are contained in Appendix C to this report, and examples of population-based approaches to lead risk assessments for the primary Lead NAAQS are found in Appendix D. A discussion of issues related to setting of the secondary Lead NAAQS is attached as Appendix E, and Panelists' individual review comments are provided in Appendix F.

At the February 6–7 public meeting, the Lead Panel expressed serious concerns both about the EPA documents to be reviewed and the Agency's proposed rulemaking schedule for the Lead NAAQS, as follows:

- 1st Draft Lead Staff Paper had no staff-derived options for keeping or altering the current Lead NAAQS.
- The Draft Lead Exposure and Risk Assessments document did not have a full discussion of the risk associated with different options for keeping or altering the Lead NAAQS. The Lead Panel judges that, while the latter document represented a good first effort, it was nowhere near completion.
- Under the Agency's new NAAQS review process, EPA's Staff Paper for lead will no longer be prepared but will be replaced by a Policy Assessment (PA) for lead, to be issued in the form of an Advance Notice of Proposed rulemaking (ANPR). However, the Agency's proposed schedule for the Lead NAAQS review calls for completion of the Lead Exposure and Risk Assessments document *after* the PA for lead is issued via the ANPR. Thus, it was not planned for the CASAC to be given an opportunity to review a more fully-developed, second-draft version of the Risk/Exposure Assessment (RA) prior to the ANPR, so that the PA would not be informed by the science assessments of the Lead Panel.

Subsequent to the February 6–7 meeting of the CASAC Lead Review Panel, Agency officials, managers and staff held administrative discussions with the chartered members of the CASAC to learn directly from these seven members their specific concerns with the schedule for review of the lead standards and the revised NAAQS review process in general. The Lead Panel is pleased to have been briefed by Agency staff during the Panel's March 9 teleconference that EPA has modified its timeline both for the generic NAAQS review process and the current Lead NAAQS review in particular, such that the Lead Panel will now review the 2nd draft of the Agency's Lead Risk/Exposure Assessment this summer, prior to the issuance of the associated PA document in the ANPR.

The CASAC Lead Review Panel used the scientific information found in the Agency's Final Lead AQCD, which was also reviewed by the Lead Panel, in its review of EPA's 1st Draft Lead Staff Paper and the Draft Lead Exposure and Risk Assessments document. The Lead Panel's recommendations and the associated scientific basis for these recommendations are presented below. *The unanimous judgment of the Lead Panel is that lead should not be de-listed as a criteria air pollutant, as defined by the Clean Air Act, for which primary (public-health based) and secondary (public-welfare based) NAAQS are established, and that both the primary and secondary NAAQS should be substantially lowered.* It is also recommended that future monitoring of lead exposure be conducted with low-volume PM_{10} samplers rather than with total suspended particulate (TSP) samplers, and that the averaging time be decreased from quarterly to monthly.

The reasons for these recommendations are given below.

Introduction

Over the past three decades, blood lead (PbB) concentrations in the U.S. population have plummeted (1). This decline was largely due to the elimination of leaded gasoline (2). In 1976, the Consumer Products Safety Commission restricted the allowable amount of lead in residential paints to 0.06 percent (600 ppm) (3). Lead solder used in canned foods was also decreased — from over 90% in 1978 to less than 5% in 1988 (4). Finally, there was a decrease in the abundance of residential housing in which lead-based paints had been used (5). Although it is difficult to quantify the extent of decrease in blood lead concentrations attributable to specific sources, the 1978 NAAQS for lead were undoubtedly among the major reasons for the rapid and widespread decrease in PbB levels in the U.S. population (6).

Despite the dramatic decrease in environmental lead exposure, lead toxicity remains a major public health problem. Environmental lead exposure in children has been associated with increased risks for reading problems, school failure, Attention Deficit Hyperactivity Disorder (ADHD), delinquency, and criminal behavior (6–10). Among U.S. children, eight to fifteen years old, those in the highest quintile (> 2 μg/dl) of lead exposure were four times more likely to have doctor-diagnosed ADHD (11). Moreover, there is no evidence of a threshold for the adverse consequences of lead exposure; studies show that the decrements in intellectual (cognitive) functions in children are proportionately greater at PbB concentrations < 10 μg/dl, the concentration considered acceptable by the Centers for Disease Control (11–14).

Lead's effects extend beyond childhood. In adults, lead exposure is a risk factor for some of the most prevalent diseases or conditions of industrialized society, including cardiovascular disease and renal disease (16–20). There is also compelling evidence that the risks for mortality from stroke and myocardial infarction are increased at PbB concentrations below 10 μg/dl, which is considerably lower than those considered acceptable for adults (19). Finally, although less definitive, there is also evidence that lead exposure during pregnancy is a risk factor for spontaneous abortion or miscarriage at PbB concentrations < 10 μg/dl (21). (It should be noted that references 11 and 19 above were not cited in EPA's Final Ozone AQCD.)

Scientific Basis for Continuing or De-listing the Lead NAAQS

The CASAC Lead Review Panel considered the implications of present scientific understanding regarding the need for protection of public health and public welfare from exposure to lead in the environment. One of these implications relates to the question of whether the current science continues to support the need for lead to be listed as a criteria air pollutant for which a NAAQS is established, or might warrant the de-listing of lead, as presented as a policy option in the 1st Draft Lead Staff Paper. In addressing this question, the Lead Panel examined several scientific issues and related public health and public welfare issues that are essential in determining whether or not a pollutant such as lead should be de-listed or maintained as a criteria air pollutant.

1. *Does new scientific information accumulated since EPA's promulgation of the current primary Lead NAAQS of 1.5 μg/m^3 in 1978 suggest that science previously overstated the toxicity of lead*? Here, the Lead Panel's answer clearly is No. The data accumulated over the past three decades make it apparent that adverse health effects on both humans and

other species appear at blood lead concentrations and environmental exposures well below those previously thought to pose important risks. Indeed, if anything, this improved scientific understanding indicates that scientific studies previously underestimated the toxicity of lead.

2. *Have past regulatory and other controls on lead decreased PbB concentrations in human populations so far below levels of concern as to suggest there is now an adequate margin of safety inherent in those PbB levels*? Again, the Panel's answer here is No. The Nation can take great pride in the extent to which exposures to lead have been decreased, leading to laudable decreases in PbB concentrations to an average approaching 2 µg/dl. However, there remains a significant segment of the population with blood-lead concentrations above 5 µg/dl — and some even above 10 µg/dl — and scientific evidence supports the contention that these PbB concentrations do not provide an adequate margin of safety. In fact, this evidence suggests these blood lead concentrations below 5 µg/dl are associated with unacceptable adverse effects.

3. *Have the activities that produced emissions and atmospheric redistribution of lead in the past changed to such an extent that society can have confidence that emissions will remain low even in the absence of NAAQS controls*? Here, the Lead Panel concludes that the answer, once again, is No. While there have been major decreases in emissions of lead from use of leaded gasoline, industrial and other activities, even the current air emissions from some lead mining and reprocessing facilities produce considerable environmental exposures once the concentrations of lead in environmental media equilibrate. The Lead Panel concludes that past success in decreasing PbB concentrations in human populations are due in part to NAAQS controls, and that in the absence of such controls, there will be a significant possibility that blood-lead concentrations would begin to rise again.

4. *Are airborne concentrations and amounts of lead sufficiently low throughout the United States that future regulation of lead exposures can be effectively accomplished by regulation of lead-based products and allowable amounts of lead in soil and/or water?* Lead Panel concludes that the answer to this question is No. While airborne lead concentrations have been decreased throughout much of the United States, airborne lead remains a primary vehicle for movement of lead between different environmental compartments. While control of airborne lead is not sufficient by itself to control exposure to lead, it is an essential component of a successful control strategy. Maintaining appropriate Lead NAAQS is considered by the Lead Panel to be an essential component of a national program to decrease the ongoing adverse effects of lead in children, adults, and in both terrestrial and aquatic ecosystems.

5. *If lead were de-listed as a criteria air pollutant, would it be appropriately regulated under the Agency's Hazardous Air Pollutants (HAP) program?* The Panel's answer is again No. The HAP program, which regulates according to use of maximum achievable control technology (MACT), followed by an analysis of residual risk, is appropriate for point sources. However, the most widespread source of airborne lead throughout the

nation is the historically-deposited lead along roadways. Thus, this source of airborne lead could not be regulated under the HAP program.

As a result of the CASAC Lead Review Panel's own answers to these scientific and public health issues, the Panel concludes that the existing state of science is consistent with continuing to list ambient lead as a criteria air pollutant for which fully-protective NAAQS are required.

Additional Analyses to Inform Decisions About a Primary (Health-Based) NAAQS for Lead

Despite the dramatic decreases in amounts of airborne lead exposures and human-population blood-lead concentrations following the phase-out of leaded gasoline, lead toxicity remains a major public health problem. As discussed above, there is increasing evidence of lead-induced toxicity at the lowest contemporary exposures to lead — resulting in significant IQ deficits in children (11–14), and increased frequency of ADHD (11) and cardiovascular disease (16–19). Although less definitive, there is evidence that lead exposure is a risk factor for spontaneous abortion and renal disease (20–21).

Although relatively few counties in the United States are out of compliance, the greatest benefit to public health will be realized by broad decreases in airborne lead concentrations across the U.S. population because:

1. The adverse consequences are proportionately greater at the lowest increments of lead exposure;
2. Lead exposure is cumulative; and
3. Airborne lead exposure, in contrast with exposure to lead-based paint, is more widely dispersed. Thus, reducing exposure from air lead will broadly reduce population blood lead levels.

In 1978, EPA established a primary Lead NAAQS of 1.5 $\mu g/m^3$ to ensure that 99.5% of the public did not exceed a blood-lead concentration of 30 µg/dl, with the 99.5% figure being the Agency's risk management (*i.e.*, policy) choice at that time. In addition to the separate Federal regulations that had been adopted in 1973 that requiring the phase-out of leaded gasoline, the 1978 Lead NAAQS was instrumental in helping to produce the dramatic decreases in air lead and blood-lead concentrations over the last 30 years. However, these primary and secondary Lead NAAQS are totally inadequate for assuring the necessary decreases of lead exposures in sensitive U.S. populations below those current health hazard markers identified by a wealth of new epidemiological, experimental and mechanistic studies.

Consequently, *it is the CASAC Lead Review Panel's considered judgment that the NAAQS for Lead must be decreased to fully-protect both the health of children and adult populations.*

The EPA pilot-phase human health risk assessment focused on three case study locations (*i.e.*, primary lead smelter, secondary lead smelter, and near-roadway urban). While the case study approach undertaken in the risk assessment is enlightening and provides a potentially

useful framework for understanding lead exposure for some discrete populations within the U.S., there are some additional considerations and analyses that the Lead Panel strongly feels could help inform a scientifically-defensible NAAQS for lead. In particular, the Panel believes that the risk assessment would be better informed with a *"population-based"* risk assessment to supplement the current case study approach. A population-based risk assessment would typically include two key components:

1. A quantitative description of the relationship between concentrations of lead in national ambient air and distributions of resulting blood lead concentrations; and
2. A quantitative description of the relationship between blood lead concentrations and impacts on IQ.

There are multiple ways in which EPA could conduct a population-based analysis, and the Panel illustrates some possibilities in Appendix D attached to this letter. Please note that this work does not represent a complete analysis on the part of the Lead Panel; rather, it is meant to illustrate the Panel's thinking in this area. It will be important for EPA to consider these approaches and to fully evaluate their pros, cons, and associated uncertainties. An adequately comprehensive analysis should characterize the uncertainty, preferably in a quantitative manner, in two key areas: (1) the relationship between a change in the NAAQS for lead and the distribution of population blood lead concentrations; and (2) the relationship between blood lead concentrations and the risk of adverse health effects. This type of review by the Agency will be necessary to highlight the strengths and weaknesses of the available approaches and help to elucidate a primary Lead NAAQS that is scientifically-defensible and adequately-protective of public health.

As described in Appendix D, the Lead Panel considered three separate, but related, population-based analytical approaches as examples to be considered in deriving an acceptable range of alternative levels, on the basis of the scientific evidence, for setting a new level for the primary Lead NAAQS, as follows: Approach A relates air lead levels to blood lead levels using the approach used in previous lead NAAQS calculations; Approach B uses an epidemiologic approach to derive an adjusted slope factor relating air lead levels to blood levels taking into account all exposure pathways between air lead and blood lead; and Approach C relates air lead levels to blood lead levels and then to IQ loss in children. In addition, *the CASAC Lead Review Panel considers that a population loss of 1-2 IQ points is highly significant from a public health perspective (22). Therefore, the primary lead standard should be set so as to protect 99.5% of the population from exceeding that IQ loss.*

The three approaches provide comparable results. Given the Panel's assumptions and preliminary analysis conducted for the three approaches, *the resulting analyses indicate to the CASAC that there is a need for a substantial reduction in the primary Lead NAAQS, to a level of about 0.2 µg/m^3 or less*. CASAC recognizes that these preliminary calculations are dependent upon the results of EPA's forthcoming uncertainty analyses and the current risk management choice for the percentage of the population left at risk, as well as acceptable blood levels, IQ loss and slope factor — the appropriateness of which all depend on certain scientific assumptions and the risk management criteria that are chosen. Imposing more stringent criteria would result in a lower (that is, more stringent) range of primary Lead NAAQS levels, whereas less stringent

criteria would result in the calculation of a higher (*i.e.*, less stringent) range of primary lead standards.

Possible Revision to Lead Indicator from TSP to Low-Volume PM_{10}

As revisions to the level, form, and averaging time of the Lead NAAQS are considered, CASAC also recommends that EPA revise the indicator. Currently, Lead NAAQS monitoring is predominantly based on atomic absorption analysis of fiberglass filters run on hi-volume total suspended particulate (TSP) samplers. Most other TSP sampling was discontinued after PM_{10} standards were promulgated in 1987. TSP samplers capture particles with an imprecise and variable upper particle cut size in the range of approximately 30 to 50 microns on fiberglass filters which are not well-suited for analysis by inexpensive, multi-elemental surface beam techniques like particle-induced X-ray emission (PIXE) or X-ray fluorescence (XRF). Consequently TSP sampling by imprecise samplers is primarily conducted only for lead analysis and these filters are rarely analyzed for other species.

If Lead NAAQS monitoring was based on (low-volume) PM_{10} sampling on Teflon filters, the resulting data would be correlated with TSP lead, as suggested by limited data in the 1st Draft Lead Staff Paper, but would have substantially improved sampling precision. The Lead Panel recognizes that either monitoring system would be subject to variability based on location, particularly near sources. Other advantages of low-volume PM_{10} sampling include:

1. Focus on those biologically-relevant particles that, when inhaled, are deposited in the thoracic region;
2. Larger spatial-scale representativeness for population exposures to monitored particles which remain airborne longer;
3. Could utilize more widespread PM_{10} and "air toxics" metals sampling networks, leading to collection of more data at lower costs;
4. Potential for inexpensive multi-elemental analysis by XRF or PIXE would provide useful supplemental metals information for health effects studies and source apportionment;
5. Potential for automated sequential PM_{10} samplers (not available for TSP) would be especially useful if sampling frequency is increased from once every six days; and
6. Weighing filters would provide useful information on PM_{10} mass; and, if collocated with $PM_{2.5}$ Federal Reference Methods (FRM), could provide needed information on $PM_{10-2.5}$ mass and speciation.

Reasons for retaining the current TSP lead indicator include: preservation of a long-term historical record at some sites; and inclusion of very coarse (> 10 micron particle) lead which may deposit in upper regions of the respiratory tract and ultimately be ingested, or which may deposit on surfaces and be ingested via hand-to-mouth activity of children. Some such coarse particles might be missed by PM10 samplers. Presumably a downward scaling of the level of the Lead NAAQS could accommodate the loss of very large coarse-mode lead particles, and some short period of concurrent PM_{10} and TSP lead sampling could help develop site-specific scaling factors at sites with highest concentrations where long-term historical records are important.

Given the advantages of using PM_{10}, the CASAC Lead Review Panel recommends that the Agency revise the lead indicator to utilize low-volume PM_{10} sampling, and also develop equivalent analytical methods to allow use of XRF and Inductively-Coupled Plasma Mass Spectrometry (ICP-MS) analysis.

Possible Revision to Averaging Time Used for the Lead NAAQS

A second change that should be considered with a change in the Lead NAAQS is possible use of a different averaging time. Currently, quarterly averaging is used. However, studies suggest that blood lead concentrations respond at shorter time scales than would be captured completely by quarterly values. Here, the Lead Panel recommends that the Agency conduct *monthly* averaging instead of quarterly.

One consideration involved in using a shorter averaging period is sampling frequency. Currently, many of the samplers operate with sampling frequencies less than once per day, and as infrequently as every sixth day. In the most extreme case, as few as four samples may be involved in determining a monthly average (assuming no samples are considered invalid). This may make the average susceptible to anomalously-high events. On the other hand, this may motivate more frequent sampling in those areas whose air concentrations are near the level of the Lead NAAQS, which would increase the protection of public health and significantly decrease the impact of a single high lead exposure event. One could also consider having the lead standards based on the second highest monthly average, a form that appears to correlate well with using the maximum quarterly value.

The CASAC Lead Review Panel recommends adopting monthly averaging as being more protective of human health in light of the response of blood lead concentrations that occur at sub-quarterly time scales, and further recommends that the most protective form would be the highest monthly average in a year. An area could choose to increase sampling frequency to make the monthly average less susceptible to more extreme events. Such a change is consistent with either using TSP or PM_{10} sampling.

Secondary (Welfare-Based) NAAQS for Lead

An extended discussion of issues related to setting the secondary Lead NAAQS can be found in Appendix E. Chapter 6 of the 1st Draft Lead Staff Paper and Chapter 7 of the "Pilot Phase" Draft Lead Exposure and Risk Assessments technical support document present compelling scientific evidence that current atmospheric lead concentrations and deposition — combined with a large reservoir of historically-deposited lead in soils, sediments and surface waters — continue to cause adverse environmental effects in aquatic and/or terrestrial ecosystems, especially in the vicinity of large emission sources. These effects persist in some cases at locations where current airborne lead concentrations are below the levels of the current primary and secondary lead standards.

Thus, from an environmental perspective, there are convincing reasons to both retain lead as a regulated criteria air pollutant and to lower the level of the current secondary standard.

Since concentrations of historically deposited lead in soils throughout the U.S. (averaging 0.5 to 4 g/m^2 of land area) are changing only slowly — with a half-life exceeding a century — these concentrated deposits of lead are expected to remain accessible for exchange with the atmosphere and the rest of the biosphere into the foreseeable future. Fires, changes in land use, or climatic events such as regional dust storms could mobilize significant quantities of lead that would be harmful both to human health and ecosystems downwind. This potential for harm is not adequately recognized in the 1st Draft Lead Staff Paper and the Draft Lead Exposure and Risk Assessments technical support document, but is a concern that warrants careful continued monitoring in the future.

In addition, while neither the 1st Draft Lead Staff Paper nor the Draft Lead Risk/Exposure Assessments document provide a clear quantitative basis for identifying a specific lower level at which a more protective secondary (welfare- or environmental-based) Lead NAAQS should be set, there are no reasons to expect that humans are uniquely sensitive to lead pollution among the millions of animal and plant species.

Therefore, at a minimum, the level of the secondary Lead NAAQS should be at least as low as the lowest-recommended primary lead standard. The EPA is also encouraged to identify the necessary funds to support needed continuing research on the ecological effects of airborne lead pollution and to consider developing alternative secondary standards such as critical loads *for lead, which may be different from primary standards in indicator, averaging time, level or form.*

The CASAC continues to be pleased to provide advice to you concerning the scientific basis for the setting of the primary and secondary Lead NAAQS. In addition, the CASAC looks forward to continued dialog with Agency officials and staff aimed at improving EPA's NAAQS review process in a manner that enhances the efficiency of the process while maintaining its integrity and adherence to the stipulations of the Clean Air Act. Finally, the Committee also looks forward to reviewing the 2nd draft of the Agency's Lead Risk/Exposure Assessment this summer. As always, we wish Agency staff well in this important task.

Sincerely,

/Signed/

Dr. Rogene Henderson, Chair
Clean Air Scientific Advisory Committee

Appendix A – Roster of the Clean Air Scientific Advisory Committee

Appendix B – Roster of the CASAC Lead Review Panel

Appendix C – Agency Charge to the CASAC Lead Review Panel

Appendix D – Population-Based Approaches to Risk Assessment Analyses for the Primary Lead NAAQS

Appendix E – Issues Related to the Setting of the Secondary Lead NAAQS

Appendix F – Review Comments from Individual CASAC Lead Review Panel Members

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Appendix A – Roster of the Clean Air Scientific Advisory Committee

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Science Advisory Board (SAB) Staff Office
Clean Air Scientific Advisory Committee (CASAC)

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Dr. Rogene Henderson, Scientist Emeritus, Lovelace Respiratory Research Institute, Albuquerque, NM

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Appendix B – Roster of the CASAC Lead Review Panel

U.S. Environmental Protection Agency
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Clean Air Scientific Advisory Committee (CASAC)
CASAC Lead Review Panel

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Dr. Robert Goyer [M.D.], Emeritus Professor of Pathology, Faculty of Medicine, University of Western Ontario (Canada), Chapel Hill, NC

Mr. Sean Hays, President, Summit Toxicology, Allenspark, CO

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Appendix C – Agency Charge to the CASAC Lead Review Panel

Charge to the CASAC Pb Panel

Within each of the main sections of the first draft Staff Paper, questions that we ask the Panel to focus on in their review include the following:

Ambient Pb information and analyses (Chapter 2):

1. To what extent are the emissions and air quality characterizations and analyses clearly communicated, appropriately characterized, and relevant to the review of the primary and secondary Pb NAAQS?

2. Does the information in Chapter 2 provide a sufficient ambient Pb-related basis for the exposure, human health and environmental effects, health risk assessment, and environmental assessment presented in later chapters?

Pb-related health effects (Chapter 3):

1. To what extent is the presentation of evidence from the health studies assessed in the Pb AQCD and the integration of information from across the various health-related research areas drawn from the Pb AQCD technically sound, appropriately balanced, and clearly communicated?

2. What are the views of the Panel on the appropriateness of staff's discussion and conclusions in Chapter 3 on key issues related to quantitative interpretation of epidemiologic study results, including, particularly, the form of a blood Pb-response function for neurocognitive effects, and the form of the associated blood Pb metric?

3. What are the Panel's views on the adequacy and clarity of the discussion of potential thresholds in concentration-response relationships presented in Chapter 3?

Human Exposure and Health Risk Analysis, Pilot-Phase (Chapter 4):

1. To what extent are the assessment, interpretation, and presentation of the results of the pilot exposure analysis, including characterization of Pb concentrations in media, the modeling of multi-pathway Pb exposure and application of biokinetic blood Pb models, as presented in Chapter 4 technically sound, appropriately balanced, and clearly communicated?

2. Are the methods used to conduct the pilot exposure analysis, including the modeling of population-level distributions of total blood Pb levels and the pathway-apportionment of those blood Pb levels (*e.g.*, air-inhalation, versus soil-ingestion versus dust-ingestion, versus

background) technically sound? Does the Panel have any suggestions for improvements in the methods used?

3. What are the Panel's views on the staff interpretation of the performance evaluation completed for the pilot analysis (and described in Chapter 4) with regard to the representativeness of individual modeling steps completed for the analysis (*e.g.*, characterization of ambient air and outdoor soil Pb levels and the estimation of blood Pb levels for specific case studies)?

4. In general, are the concentration-response functions and blood Pb metrics (*i.e.*, lifetime average, concurrent blood lead) used in the pilot analysis appropriate for this review?

5. Are the methods used to conduct the pilot health risk assessment, including the application of the cutpoints in relation to the concentration-response functions employed, technically sound? Does the Panel have any suggestions for improvements in the methods used?

6. To what extent does the sensitivity analysis completed for the pilot analysis (and described in Chapter 4) identify key sources of uncertainty and provide an assessment of their impact on risk results?

7. As part of the NAAQS review, there is interest in attempting to differentiate Pb exposure and health risk impacts for modeled populations between: (a) historically-deposited Pb (*e.g.*, near-roadway dust/soil lead from leaded gasoline); and (b) newly-emitted Pb. Does the Panel have specific recommendations regarding approaches that might be employed in the full-scale assessment for this purpose?

8. What are the Panel's views on the most important issues to be addressed in the subsequent full-scale human exposure and health assessment that will be presented in the revised documents?

<u>The Primary Pb NAAQS (Chapter 5)</u>

1. What are the Panel's views on the adequacy and clarity of the presentation of the basis for the existing standard and conclusions reached in the last review?

2. Based on the information contained in the first draft Staff Paper, as well as the AQCD, does the Panel have recommendations with regard to specific aspects of the standard to be considered in developing policy alternatives? For example, considering the prominence of the soil and dust pathways for ambient Pb exposures, and the evidence regarding environmental response times, is there reason to give more emphasis to consideration of an alternative (shorter or longer) averaging time; and, how might this be considered in the full-scale risk assessment given current capabilities?

Pb-related welfare effects and screening level ecological risk assessment (Chapter 6):

1. To what extent is the presentation of evidence from the ecological studies assessed in the Pb AQCD and the integration of information from across the various ecologically-related research areas drawn from the Pb AQCD technically sound, appropriately balanced, and clearly communicated?

2. Given the lack of quantitative information on Pb-related ecosystem effects, what are the Panel's views on the presentation of this topic in chapter 6?

3. What are the Panel's views of the data sources and models used to estimate current levels of Pb in soil, freshwater, and sediment for the case study locations?

4. To what extent are the methods used to conduct the exposure assessment and the interpretation and presentation of the results technically sound, appropriately balanced, and clearly communicated?

5. What are the Panel's views of the approach for addressing uncertainty in apportionment of Pb contributions in the national-scale screen by factoring out those locations with known non-air sources (*e.g.*, mining, point discharges)?

6. To what extent are the assessment, interpretation, and presentation of the results of the screening-level risk analysis, including characterization of lead concentrations in media and the comparisons to ecological screening values, as presented in Chapter 6 and the risk assessment report technically sound, appropriately balanced, and clearly communicated?

7. Does the Panel feel that adequate screening criteria (ecotoxicity screening values) were selected for each of the media?

8. What are the Panel's views on the derivation of the soil screening values for birds and mammals (*i.e.*, using the Eco-SSL methodology)? Do the resultant values adequately reflect current information on exposure characteristics of these organisms?

9. To what extent are the uncertainties associated with the exposure analysis clearly and appropriately characterized in Chapter 6 and the risk assessment report?

Appendix D – Population-Based Approaches to Risk Assessment on Analyses for the Primary Lead NAAQS

The CASAC Lead Review Panel considered three separate, but related, population-based analytical approaches as examples to be considered in deriving an acceptable range of alternative levels, on the basis of the scientific evidence, for setting a new level for the primary Lead NAAQS, as follows:

- **Approach A** relates air lead levels to blood lead levels using the approach established in previous lead NAAQS calculations;
- **Approach B** uses an epidemiologic approach to derive an adjusted slope factor relating air lead levels to blood levels taking into account all pathways between air lead and blood lead; and
- **Approach C** relates air lead levels to blood lead levels and then to IQ loss in children.

These approaches consider existing information and the following assumptions:

- the population to be protected (99.5% of the population of children);
- the *maximal acceptable blood concentration* (up to 5.0 μg/dl);
- an *appropriate geometric standard deviation (GSD)* for the blood lead levels in children exposed to a given level of air lead (range 1.3–2.0);
- the *non-air background* (1.0–1.4 μg/dl or lower range should be considered);
- the *slope factor* for the relation between air lead and blood lead for levels of blood lead below 10 μg/dl, with the candidate values considered being 2.0 μg/dl per μg/m^3 (m^3/dl) used in 1978, 5.0 m^3/dl used by the World Health Organization (WHO) in 2000, 10.0 m^3/dl noted in recent studies (see the discussion in Approach B below), and assuming 20.0 m^3/dl as a maximum; and
- the most sensitive toxicity endpoint (*i.e.*, IQ loss in children).

In addition, the CASAC Lead Review Panel considers that a population loss of 1-2 IQ points is highly significant from a public health perspective (22). Therefore, the primary lead standard should be set so as to protect 99.5% of the population from exceeding that IQ loss.

The three approaches provide comparable results. Given the Panel's assumptions and preliminary analysis conducted for the three approaches, *the resulting analyses indicate to the CASAC that there is a need for a substantial reduction in the primary Lead NAAQS, to a level of about 0.2 μg/m^3 or less*. CASAC recognizes that these preliminary calculations are dependent upon the results of the Agency's forthcoming uncertainty analyses and: the values chosen for the percent of the population left at risk; acceptable blood levels and IQ loss; and slope factor — the appropriateness of which all depend on certain scientific assumptions and the risk management criteria that are chosen. Imposing more stringent criteria would result in a lower (that is, more

stringent) range of primary Lead NAAQS levels, whereas less stringent criteria would result in the calculation of a higher (*i.e.*, less stringent) range of primary lead standards.

Approach A

The first approach (A) relates air lead levels to blood lead (PbB) levels using a simplified and modified empirical-deterministic approach that is essentially the same approach used in previous EPA NAAQS (1978) and World Health Organization (WHO, 2000) guidance documents. This approach begins with selection of a "not-to-be-exceeded" PbB value or values based on scientific evidence. These "not-to-be-exceeded" PbB values for beginning the 1978 NAAQS and the 2000 WHO uses of the approach were 30 and 10 µg/dl respectively. The current scientific evidence reviewed by the Panel, per the Agency's Final Lead AQCD indicates that the concentration of lead in blood shown to be harmful has declined substantially below those levels, to around 5 µg/dl or less.

For example, based on current evidence, one might consider two "not-to-be-exceeded" PbB values, such as 5.0 and 2.5 µg/dl. These are not to be exceeded at the 99.5 percentile and, for an illustrative GSD of 1.3, produce geometric mean values of 2.5 and 1.3 µg/dl, respectively. The non-air portion of these two means must be subtracted to give the air Pb-based contributions to PbB. Panel member Dr. Paul Mushak (Appendix F) calculated the non-air portion using the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK).

The use of a "not-to-be-exceeded" PbB value of 5 µg/dl in Approach A and slope factors of 5, 10, or 20 produce corresponding suggested NAAQS values of 0.22, 0.11, or 0.06 µg/m^3. Use of a "not-to-be-exceeded" value of 2.5 µg/dl and the same slope factors produce air lead values half as high, *i.e.*, 0.11, 06 and 0.03 µg/m^3, respectively. (Note that, for the 1.25 mean PbB scenario, the non-air PbB contribution is the dominant source and modeling does not provide an exact value; see Dr. Paul Mushak's detailed derivation comments in Appendix F, pp. F-55 through F-57.) Based on these values alone, the current primary Lead NAAQS set in 1978 should be lowered by at least a factor of seven and by as much as 50, depending on the slope factor used (see, in particular, the individual comments of Lead Panel members Dr. Mushak and Dr. Ian von Lindern found in Appendix F).

Approach B

Approach B is a "top-down" approach. That is, instead of estimating the effect of inhalation alone, the effect of air lead on deposition into dust, food, *etc.* and the uptakes from those pathways, an epidemiologic approach should be used to derive an adjusted slope factor taking into account all pathways between air lead and blood lead. This is based on the changes in blood lead observed when lead began to be phased-out of gasoline. This analysis relies on the results of Schwartz and Pitcher (23).

The Schwartz and Pitcher analysis showed that in 1978, the midpoint of the National Health and Nutrition Examination Survey (NHANES) II, gasoline lead was responsible for 9.1 µg/dl of blood lead in children. Their estimate is based on their coefficient of 2.14 µg/dl per 100 metric tons (MT) per day of gasoline lead use, and usage of 426 MT/day in 1976. Between 1976 and when the phase-out of lead from gasoline was completed, air lead concentrations in U.S.

cities fell a little less than 1 μg/m^3 (24). These two facts imply a ratio of 9–10 μg/dl per μg/m^3 reduction in air lead, taking all pathways into account.

Under this scenario, a decrease of mean air lead concentrations of 0.1 μg/m^3 could be expected to produce a further decrease in average blood lead concentrations of 0.9–1.0 μg/dl. Assuming a slope of three IQ points per μg/dl reduction in blood lead, which is indicated by the pooled analysis of low concentration lead exposure (13), this further decrease would be expected to raise the average IQ of children in U.S. cities by approximately three IQ points — a significant positive health impact. Put another way, the derivation above empirically justifies the use of the slope factor of 10 in Approach C, and the resulting estimates that an air quality standard of 0.11 μg/m^3 (that is, a 13-fold reduction) would be required to keep 99.5% of the children below a blood lead of 5 μg/dl.

Approach C

Approach C is more sophisticated, starting with an air lead level and a blood lead level produced only by airborne lead, and relates that air level to IQ point loss (see Table 2). A linear model between the ranges of 1-7.5 μg/dL PbB for both concurrent and lifetime exposures suggests a three-point decrement in IQ for each unit change in PbB (13). Approach A and C are in agreement on the relationship between air lead levels associated with PbB, dependent on the slope factor used. (Approach A does not consider IQ loss or any other health effect.)

These considerations are summarized in Tables 1 and 2, and are also contained in the individual review comments from various Lead Panel members attached as Appendix F. Depending on the slope factor selected between 5 and 20 m^3/dl, the estimate of blood lead concentrations from various air lead concentrations varies by a factor of four (Table 1). For example, using the linear estimate of IQ loss associated with PbB below 7.5 μg/dl (13), the Lead Panel estimated that, over the range of PbB from 0.5–4.0 μg/dl (*i.e.*, an eight-fold range), the loss of IQ would similarly increase from 1.5 to 12 IQ points (Table 2).

Since the Lead Panel considers a population loss of 1-2 IQ points to be highly significant from a public health perspective, the Lead Panel therefore considers this extent of loss in IQ as a "change in IQ not to be exceeded." Depending upon the slope factor selected, this results in a range of 0.025–0.200 μg/m^3 (*i.e.*, about a 7.5- to 60-fold decrease from the current primary Lead NAAQS) as the estimated air lead concentration to consider under Approach A.

References

23. Schwartz J, Pitcher H. The relationship between gasoline lead and blood lead in the United States. 1989 *J. Official Stat.* 5: 421-431.

24. U.S. Environmental Protection Agency. (1986) *Air Quality Criteria for Lead*. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no. EPA/600/8-83/028aF-dF. 4v. Available from: NTIS, Springfield, VA; PB87-142378, p. 1-21.

TABLE 1. Relationship of Blood Lead (PbB) to Air Lead (Pb-Air) by Differing Slope Factors

Pb-Air (μg/m$^{3)}$	PbB (μg/dl)		
	S.F.* = 5	S.F.* = 10	S.F.* = 20
0.010	0.05	0.10	0.20
0.025	0.13	0.25	0.50
0.050	0.25	0.50	1.00
0.100	0.50	1.00	2.00
0.200	1.00	2.00	4.00

*S.F. = slope factor (m^3/dl) = PbB/Pb-Air; S.F. value varies with increasing impact of indirect Pb-Air pathway (Dust Pb + Soil Pb)

TABLE 2. Relationship of IQ Point Losses to Increases in Pb-Air and Pb-Air-Based Blood Lead (PbB) Values Above Zero [a,b,c]

Pb-Air ($\mu g/m^3$)	S.F. = 5		S.F. = 10		S.F. = 20	
	PbB [d]	IQ Loss [e,f]	PbB	IQ Loss	PbB	IQ Loss
0	0	0	0	0	0	0
0.010	0.05	< 1	0.10	< 1	0.20	< 1
0.025	0.13	< 1	0.25	< 1	0.50	1.5
0.050	0.25	< 1	0.50	1.5	1.00	3.0
0.100	0.50	1.5	1.00	3.0	2.00	6.0
0.200	1.00	3.0	2.00	6.0	4.00	12.0

a Pb-Air-related increases affecting IQ point loss through calculated PbB values using 3 slope factors per Table 1
b IQ vs. PbB dose-response relationships based on Lanphear *et al.*, 2005 (13): sub-7.5 µg/dl linear segment, average slope = 3.0, combining slopes of 2.9 and 3.1 for concurrent and lifetime average dose metrics, respectively
c Slope factors as defined in Table 1 and text
d PbB as derived in Table 1
e Rounding for values < 1 IQ point
f Population, not individual, IQ loss/gain projections; U.S. CDC 2007 estimates 23,380,860 U.S. children 0-71 months of age. Source: U.S. Centers for Disease Control, 2007. CDC Surveillance Data. (Last updated 2/16/2007). URL: http://www.cdc.gov/nceh/surv/stats.htm [accessed 3/8/2007]

Appendix E – Issues Related to the Setting of the Secondary Lead NAAQS

Chapters 6 of the Agency's 1st Draft Lead Staff Paper and Chapter 7 of EPA's Draft (Pilot-Phase) Lead Exposure and Risk Assessments document summarize a very large body of scientific knowledge about environmental and ecological effects of atmospherically deposited lead on the biota, soils, sediments, and surface and ground waters of terrestrial and aquatic ecosystems in various parts of the U.S. and nearby parts of the world. This significant body of scientific knowledge includes environmental effects of both historically-deposited lead and continuing air dispersal or reentrainment of lead compounds from primary and secondary lead smelters, along roadsides, and in ecologically-sensitive areas such the Hubbard Brook Experimental Forest as described in Chapter 7 of the pilot-phase technical support document.

Although lead is recognized in these two chapters as one among a longer list of heavy metals in the environment (including cadmium, zinc, and mercury), these two chapters do not contain adequate discussion of the special characteristics of lead or its ecological effects in the context of these other metals — or other air-dispersed criteria pollutants. Also, little of the information about specific environmental effects of lead is presented in a way that is directly relevant to the issue of whether the EPA Administrator should retain, increase, or decrease the present primary and secondary National Ambient Air Quality Standard (NAAQS) for lead. These identical standards were established in 1978 and have been maintained ever since at a level of 1.5$\mu g/m^3$ as a quarterly average (maximum arithmetic mean averaged over a calendar quarter).

The Lead Panel believes that especially Chapter 6 of the draft Lead Staff Paper (and perhaps also at least parts of Chapter 7 of the pilot-phase risk-exposure assessment document) would be much improved in their intended purposes if they were to contain a concise summary of:

1. The knowledge available and (as best they can discern) the rationale used by the Administrator in promulgating the original NAAQS for Lead in 1978;
2. The knowledge available and rationale used in the decisions made in 1989 and 1990 to retain unchanged the identical primary and secondary Lead NAAQS established in 1978; and
3. The knowledge available and rationale used by the Administrator in establishing and maintaining identical primary and secondary standards for criteria air pollutant — not only for lead — but also for most of the other criteria pollutants for NAAQS since 1970.

Despite the limitations mentioned in the last two preceding paragraphs, the Lead Panel believes that the body of scientific knowledge summarized in the Agency's Final Air Quality Criteria Document (AQCD) for lead, and further presented in the aforementioned chapters in the 1st Draft Lead Staff Paper and the Draft (Pilot-Phase) Lead Exposure and Risk Assessments documents, provide compelling scientific justification for both:

1. The original (1978) decision by EPA to regulate lead as a Criteria Air Pollutant and to establish what was then considered to be an appropriately-designed primary (public-health based) NAAQS for lead, with a secondary (public-welfare based) standard set at the same level and form, and

2. Maintaining for the foreseeable future similarly well designed (but contemporarily scientifically well-informed) primary and secondary NAAQS for lead — standards with levels and forms that may be different from, rather than identical to each other.

There are several features of the environmental and ecological effects of lead, and both the chemical and physical properties of lead in the environment, that make lead distinct from the other four criteria pollutants for which NAAQS have been developed by the EPA. These distinctive properties include:

1. The widespread use of lead as an ingredient in decorative paints, lead-acid batteries, as an additive for gasoline used in motor vehicles, and even in some pesticides used earlier to protect some horticultural crops from plant pathogens;
2. The persistence of lead in soils, surface and ground waters, sediments, and in both the structural- and some biologically-active tissues of plants, animals, insects, and microorganisms;
3. The well-known toxicity and interference in development of cognitive functional capacity in humans (especially children) and the much less well-known toxicological and other effects of lead on all the other different types of animals, plants, insects, and microorganisms in managed and natural terrestrial and aquatic ecosystems of the Earth — some of which are undoubtedly even more sensitive to lead than human infants;
4. The very substantial decreases in current air concentrations and atmospheric deposition of lead into the environment that were achieved in recent decades through:

   (a) The Phase-out of lead additives in gasoline during the 1970s, ‘80s, and ‘90s;

   (b) Severe limits on air emissions from lead smelters during earlier decades; and

   (c) Decreases in air emissions from lead battery processing facilities in more recent years.

   Thus, most current exposures of living organisms in natural and managed terrestrial and aquatic ecosystems are caused primarily by redistribution of environmentally persistent airborne lead compounds deposited in soils, sediments, and surface waters during the latter earlier decades of the 20th century.

5. The continuing airborne resuspension and dispersal of lead that persists in soils, fugitive dusts, sediments, and surface waters and are transported and deposited once again from air in both fine and coarse particulate matter and aerosols — especially along roadways.

These distinctive properties of lead suggest to some policy makers that ecological and environmental effects of lead might be managed by other means than maintaining both primary and secondary Lead NAAQS. In the Lead Panel’s considered judgment, the limitations of the other methods of management now available to the EPA are such that none of these alternative

methods would be anywhere near as effective in protecting public health or welfare as maintaining *for the foreseeable future* as appropriately well-designed (and contemporarily scientifically well-grounded) primary and secondary NAAQS for lead.

As indicated in the body of the letter to the EPA Administrator, the members of the CASAC Lead Review Panel have provided a consensus scientific judgment that the present level ($1.5 \mu g/m^3$) of the primary Lead NAAQS should be decreased substantially and that appropriate adjustments probably also should be made in the indicator, averaging time, and statistical form of the primary NAAQS for lead.

The scientific evidence on ecological and environmental effects of lead summarized in the Draft Lead Staff Paper and the Draft Lead Exposure and Risk Assessments documents indicate that any significant decrease in the present level of the primary Lead NAAQS will very likely have similarly significant beneficial effects on the magnitude of lead exposures in the environment and lead toxicity impacts on natural and managed terrestrial and aquatic ecosystems in various regions of the U.S., Canada, Mexico, the Great Lakes, and also in the open-water regions of the Atlantic Ocean.

Since concentrations of historically deposited lead in soils throughout the United States (averaging 0.5 to 4 grams/m^2 of land area) are changing only slowly — with a half-time exceeding a century — these concentrated deposits of lead are expected to remain accessible for exchange with the atmosphere and the rest of the biosphere into the foreseeable future. Fires, changes in land use, or climatic events such as regional dust storms could mobilize significant quantities of lead that would be harmful both to human health and ecosystems downwind. This potential for harm is not adequately recognized in the present Draft Lead Staff Paper and the Draft Lead Exposure and Risk Assessments documents.

Considering the magnitude of important ecological effects of lead in the environment, as described in these document, it is very disappointing to note that the EPA apparently lacks (or chooses not to expend) funds for any additional ecological risk assessment work for this current (2006–2008) review of the NAAQS for lead. This disappointment also is increased by the very welcome attention given in the Final Lead AQCD to the alternative concepts of critical loads, critical limits, target loads, and target times that have been developed in Europe and Canada to guide the processes of decision making regarding both environmental and public health effects of airborne chemicals.

Although these alternative concepts and processes of analysis of multiple pollutant/ multiple effects have not been carefully considered for use in the U. S., the CASAC Lead Review Panel — together with the authors of the National Research Council (NRC)/National Academy of Sciences (NAS) 2004 report on "Air Quality Management in the United States" — believes that these alternatives should be considered very carefully in the future as air quality management tools for use in this country as well as in other countries around the world.

Appendix F – Review Comments from Individual CASAC Lead Review Panel Members

This appendix contains the preliminary and/or final written review comments of the individual members of the Clean Air Scientific Advisory Committee (CASAC) Lead Review Panel who submitted such comments electronically. The comments are included here to provide both a full perspective and a range of individual views expressed by Panel members during the review process. These comments do not represent the views of the CASAC Lead Review Panel, the CASAC, the EPA Science Advisory Board, or the EPA itself. The views of the CASAC Lead Review Panel and the CASAC as a whole are contained in the text of the report to which this appendix is attached. Panelists providing review comments are listed on the next page, and their individual comments follow.

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Dr. Ellis Cowling

Dr. Ellis Cowling
North Carolina State University
January 29, 2007

Individual Comments prepared in advance of the February 6-7, 2007 Peer Review of the Ecological Risk Assessment chapter (Chapter 7) within the Pilot Phase External Review Draft Technical Report titled "Lead Human Exposure and Health Risk Assessments and Ecological Risk Assessments for Selected Areas"

This "Pilot Phase" technical support document is intended to provide background scientific information for possible revision of the existing primary and secondary National Ambient Air Quality Standards for lead. After review by CASAC, this document will be used to develop a "Full Scale" technical support document that will be used as further background for what traditionally has been called the "EPA Staff Paper" on lead and in the future will be called a "Policy Assessment of Scientific and Technical Information" document for lead.

This Pilot Phase document is based on a series of case studies that have been selected to provide further information that will be summarized in the "Policy Assessment" document which in turn will be used by the Administrator of EPA in making final decisions about whether to retain or change one or the other, or both, of the presently existing (since 1978) identical primary (public-health based) and secondary (public-welfare based) National Ambient Air Quality Standards for lead.

The "case studies" chosen for inclusion in this Pilot Phase report are intended to illuminate the lead-exposure and lead public-health effects and lead eco-system effects relationships in a variety of locations and situations where both current and historical air emissions of lead have resulted in significant gradients in both human exposures and ecosystem exposures to lead. The case studies included in this Pilot Phase report were developed on the basis of studies at the following locations:

1) A primary lead smelter near Herculeaneum, MO where lead ores has been mined and smelted for most of the past 100 years — and where recent measurements have been made of lead concentrations in air, soil, indoor dust, surface water, and sediment. This location was used for both the human health risk assessment and the ecological risk assessment studies in the Pilot Phase report.

2) A secondary lead smelter near Troy, AL was selected as representative of 15 secondary smelters in the US where scrap metal and lead-acid motor vehicle batteries are processed to recover molten lead metal, spent acid, and slag waste materials. At the Troy, AL site, recent measurements of lead air concentrations were available. Estimates of air emissions were used to provide mathematical-modeling estimates of various types of human and ecosystem exposures and effects.

3) A near-roadway urban site in Houston, TX was used for the human health risk assessment. A single air-quality monitoring instrument about 115 meters from this roadway was used to measure lead air concentrations but no direct measurements of human exposures or records of health impacts were available. Thus, mathematical models were used to estimate the probable human exposures and health effects at this near road-roadway urban location and in another case study near Corpus Christi, TX.

4) Two different non-urban near-roadway locations were selected for analysis in the ecological risk assessment part of this Pilot Phase study: one site was along Interstate 37 near an oil refinery in the vicinity of Corpus Christi, TX and the other site was near exist 86 on Interstate 95 north of Richmond, VA. At both of these locations, soil lead measurements were made at various distances from the roadway. These data were then used to estimate expected uptake rates which then were used to calculate expected effects of soil lead on growth, development, reproduction, etc of various species of trees, aquatic plants, invertebrates, and birds.

5) The Hubbard Brock Experimental Forest in the White Mountains of New Hampshire was selected to serve as an "ecologically vulnerable case study." Although a few measurements of soil lead concentrations at various depth in the soil on different watershed areas within the Hubbard Brook Experimental Forest have been made, very few analyses have yet been made to determine probable effects of historically deposited lead or current lead deposition rates on the health, productivity, reproduction, or other characteristics of the trees and other species of plants within the Hubbard Brook Experimental forest.

6) Using the National Water Quality Assessment (NAWQA) Database developed by the US Geological Survey, a National-Scale Surface Water and Sediment Screening Assessment was made using lead concentrations measurements in water columns within 47 river basins and sediments with 12 river basins at various locations across the United States. This data base was used to develop a rough screening assessment that is relevant mainly to the current concentration of lead in water columns and sediments which appears mainly to be the result of redistribution of lead from soils, sediments, and other reservoirs that were contaminated by atmospheric deposition of lead in the later decades of the 20th Century.

As I completed this very short description of the case studies used in the Pilot Phase study, my intention was to go on to consider the limitations of both the available data and the conceptual frame of reference for evaluating the results of each case study by making some recommendations for what might be included in the "Full Scale" technical support document. But as I contemplated these recommendations, I could not help but remember [and be inspired by] the following four assertions made in the transmittal letter and attachments to our July 26, 2006 CASAC report to Administrator Johnson:

1) Many of us in CASAC continue to believe that:

   "The principal goal of the NAAQS review process is to answer the following policy question: 'What scientific evidence is there since the last review to indicate if the current

NAAQS standards are satisfactory or need to be revised or if additional standards needs to be implemented to protect public health and public welfare and the environment.'"

2) The most impressive general conclusions in the First and Second External Review Drafts of the Lead Criteria Document are the very substantial decreases in air concentrations and atmospheric deposition of lead into the environment that were achieved in recent decades– especially as the result of the phase-out and almost complete discontinued use of lead as a motor fuel additive. The amounts of lead that were emitted into the air by human activities, transported through the atmosphere, and deposited onto vegetation, surface waters, soils, and accumulated into sediments during earlier decades of the past century were very substantial indeed.

Total lead cumulative deposition in the United States during the 20th Century is estimated to be 0.5-4 grams per square meter of land and water surface area – depending on elevation and proximity to urban areas and lead smelting and processing facilities.

Contemporary loadings to terrestrial ecosystems are now about 1-2 milligrams per square meter per year – about three orders of magnitude smaller than the cumulative loading from all atmospheric sources during the past century.

Thus, with rare exceptions in the immediate vicinity of some lead processing facilities, most contemporary exposures of living organisms (and consequent risks to the health and productivity of natural and managed ecosystems in the United States) are not caused by contemporary air concentrations and exposures to airborne lead compounds, but rather are caused primarily by redistribution of environmentally persistent lead compounds deposited in soils, sediments, and surface waters during the past century.

3) Multi-Media Nature of Lead

More than any other of the five Criteria Pollutants which CASAC has been charged to review in recent years, lead crosses more if not all of the "media of concern" to USEPA. These multi-media aspects include: 1) air emissions and deposition of lead from transportation vehicles, metal smelters, and battery production and recycling facilities, 2) lead content of drinking water, 3) lead containing paints, 4) lead containing pesticides involved in food production, 5) soil contamination with lead, 6) lead in municipal and solid waste management, 7) lead contamination of superfund sites, etc. The multi-media nature of this pollutant is touched upon in several different parts of this the First External Review Draft [of the lead Criteria Document]. It may be worthwhile to draw these multi-media aspects of lead together in a single part of the Criteria Document, probably in Chapter 1 [and also in the Pilot Phase study and the soon-to-be-completed Final Phase technical support documents for the 2007 Policy Assessment document].

4) ... it was very encouraging to see the following statements prepared by my CASAC colleagues Jim Crapo and Paul Mushak, in their individual comments after the CASAC meeting on the lead Criteria Document and Staff Paper.

Jim Crapo's very brief statement was as follows: "It is recommended that the introduction include a more detailed discussion of the history of EPA Lead NAAQS revisions including recommendations of previous CASAC groups. It is recommended that this section also include the chronology of international policies on lead air quality standards."

Paul Mushak's more detailed statement was as follows: "CASAC member Dr. Cowling recommended acceptance of the document but only so long as the history of past efforts by EPA and others, post-1978, to evaluate and make recommendations on air lead standards or guidelines be included. Similar sentiment was expressed by others. I agree. I particularly agree with the need for inclusion of discussion of past CASAC actions, post-1978, as part of the review record.

Members of the current CASAC Panel may or may not be aware that, in the 1989-90 timeframe, a former CASAC Panel presented a set of quite clear recommendations to Administrator William K. Reilly regarding that Panel's review, conclusions and recommendations for the EPA/OAQPS Staff Paper on NAAQS evaluation dated March, 1989. I was a member of the CASAC Panel preparing the 1/90 report (and also a member of the two WHO-Europe panels noted below who presented WHO-Europe air lead guidance values in 1987 and again in 2000).

The 1990 CASAC Report on the NAAQS. The most significant parts of EPA's former SAB/CASAC Committee on NAAQS review for Pb, in its January 3, 1990 transmittals to EPA Administrator Reilly, were specific conclusions and recommendations deriving from its review of the OAQPS March, 1989 Staff Paper. I would urge that the current CASAC Chair include, in any near-future transmittals to Administrator Johnson, complete copies of both the January 3, 1990 transmittals and the March, 1989 OAQPS/EPA Staff Paper as part of the Administrative Record. The subject 1/90 CASAC transmittal to Administrator Reilly included two paragraphs among the conclusions and recommendations that captured the essence of the CASAC Panel's efforts. I strongly recommend that these two paragraphs be quoted in the current AQCD and any new OAQPS Staff Paper so as to provide important context. These two paragraphs are presented verbatim below:

> [1990 CASAC Report, p. 1, 2nd Par.] "In discussing blood lead levels used to assess alternative standards, it is the consensus of CASAC that blood lead levels above 10 μg/dl clearly warrant avoidance, especially for development of adverse health effects in sensitive populations. The value of 10 μg/dl refers to the maximum blood-lead level permissible for all members of these sensitive groups, and not mean or median values. The Committee concluded that the Agency should seek to establish an air quality standard which minimizes the number of children with blood lead levels above a target value of 10 μg/dl. In reaching this conclusion, the Committee recognizes there is no discernible threshold for several lead effects and that biological effects can occur at lower

levels. In setting a target value for blood lead (matched ultimately to air lead level) the Committee emphasized the importance of always being mindful that blood lead levels and health outcome measures are best characterized as a distribution of values about mean or median values. The importance of considering the distribution of values about the mean or median is apparent from consideration of the influence of lead exposure on I.Q. A seemingly modest decrease in the mean or median I.Q. may result in significant changes at the outer limits of the distribution with both a reduction in the number of bright children (I.Q. > 125) and an increase in the number of children with I.Q. < 80."

[1990 CASAC Report, p. 3, 1st Par.] "The EPA Staff recommended in the Staff Position Paper that the lead NAAQS be expressed as a monthly standard in the range of 0.5 to 1.5 $\mu g/m^3$ not to be exceeded more than once in three years. The Committee concurs with the EPA Staff recommendation to express the lead NAAQS as a monthly standard not to be exceeded more than once in three years. The Committee strongly recommends that in selecting the level of the standard you take into account, the significance and persistence of the effects associated with lead as well as those sensitive population groups for which valid quantitative exposure/risk estimates could not be made at this time. The Committee believes you should consider a revised standard with a wide margin of safety, because of the risk posed by lead exposures, particularly to the very young whose developing nervous system may be compromised by even low level exposures. At the upper level of the staff paper range (1.0-1.5 $\mu g/m^3$) there is relatively little, if any, margin of safety. Therefore, the Committee recommends that in reaching a decision on the level of the standard, greater consideration be given to air lead values below 1.0 $\mu g/ m^3$. To provide perspective in setting the NAAQS for lead it would be appropriate to have the EPA Staff compute the distribution of blood- lead levels resulting from a monthly standard of 0.25 $\mu g/ m^3$ for comparison with the values already computed for higher levels. In setting the NAAQS for lead it is important to recognize that airborne lead serves not only as a source of inhalation exposures, but that lead in air deposits on soil and plants becoming a potential source for intake into the body."

The WHO-Europe Air Lead Guidelines. The 1987 (first edition) WHO-Europe "Air Quality Guidelines for Europe" developed an air lead guideline for Europe consisting of a level in the range of 0.5 to 1.0 $\mu g/m^3$. The process for development of the 1987 air Pb guideline is contained in Chapter 23. The key elements in that development included, but were not limited to, the fact that both adults and very young children are affected; children are affected at lower exposures than adults; and air lead enters the body directly through inhalation but also subsequently via ingestion of dusts and soils produced from air lead fallout.

World Health Organization. 1987. Air Quality Guidelines for Europe. Lead. Ch. 23.WHO Regional Bureau for Europe, Copenhagen, pp. 242-261.

In Conclusion:

In light of these four assertions — all of them made in the context of our earlier CASAC reports to Administrator Johnson — I recommend that our CASAC discussions on February 6,

2007 regarding at least the Ecological Risk Assessment chapter (Chapter 7) — if not the whole of the Pilot Phase External Review Draft technical support document — begin with a careful reexamination of these four (and perhaps other) already existing CASAC conclusions and recommendations.

I say this because I believe it is important that CASAC be very clear about:

1) the scientific and policy purpose of these periodic reviews of National Ambient Air Quality Standards,
2) our Congressionally mandated role in the (now recently revised) NAAQS review processes and procedures, and
3) many of the unique multi-media features of lead as a Criteria Air Pollutant,

before discussing:

A) the details of the case studies in this Pilot Phase technical support document, and/or
B) the special features of these case studies – and both their relevance to and adequacy (or inadequacy) – as a foundation for current decisions about management of both public-health risks and ecological risks associated with both current and historical atmospheric deposition of lead in various parts of these United States.

Dr. Douglas Crawford-Brown

Comments on Chapter 4: Characterization of Health Risks

Doug Crawford-Brown, January, 2007

The following comments apply to Chapter 4 as a self-contained document. I provide related comments in my review of Chapter 5. I end with bulleted comments summarizing my main points.

Overall, I am impressed with the analysis performed, especially the general structure of the analysis. I begin here, however, with consideration of the reason for conducting the assessment in the first place. As I understand it, the goal was to determine whether Pb should still be considered under the NAAQS program, or might be "de-listed" because it is no longer a significant component of environmental risk. Given this goal, I am not fully convinced that the three case studies fully answer that question.

In saying this, I am assuming that there are times when the ubiquity of an issue requires a national approach such as NAAQS, and other times when the problem is so isolated (in space and/or time) as to suggest a more targeted risk management strategy. The fact that one can find an example where residual risks are high despite existing regulations, such as at the primary smelter, is not in itself evidence that a NAAQS approach remains valid. It simply means there is continued need to apply risk management strategies in some parts of the country. As a result, I am not sure whether I buy into the idea that this assessment allows the EPA to determine whether continuation of Pb within the NAAQS program is justified. But having said that, I still believe the assessment that was conducted provides a reasonable picture of the risks in the population living within the three case study areas. What is missing is a clear statement of the relationship between these three cases and the more general national picture.

The structure of the assessments performed, both in terms of the treatment of spatial variation and the modeling of exposure routes, seems to me to be sound, or at least in keeping with best scientific practice. The EPA and its contractors have established these methods over the past decade, and so they have become fairly routine and accepted as a reasonable approximation to the intersubject variability distribution for risk. I might quibble with specific parameter values (and this would be only a quibble, since my preferences wouldn't change the assessment significantly), but the general approach is sound.

I am comfortable with the decision to use modeling approaches for the smelters and monitoring data for the road scenario. I am not that well versed in the monitoring programs, and so I can't attest to their ability to yield results that are suitable for a spatially-gridded risk assessment. But it seems to me the authors have used the results correctly. And the choice to focus on a geographic area where the data are plentiful was wise, with one caveat: plentiful data at times mean an area was monitored more extensively because of a finding that it is unusually high in some sense. This would call into question whether it is a representative case study. I can see no

discussion of this issue in the document, and will look for some clarification in the upcoming meeting.

I cannot follow the reasoning at the top of Page 4-11 justifying the focus on a particular roadway location. I can think of a number of reasons why monitoring might exceed predicted ambient concentrations, such as incorrect monitoring results, poor modeling assumptions, incorrect alignment of modeling predictions (which tend to be spatially averaged) with particular monitoring locations, etc. The fact that this ratio is above one does not seem to me strong evidence that re-entrainment is important there.

As I mention in my review of Chapter 5, I am comfortable with the treatment of inter-subject variability as a post-processing step using a lognormal distribution of adjustment factors. I remain unconvinced that the particular characteristics of this distribution, such as the GSD, can be determined from data such as measurements in NHANES because I believe the data cited by the authors are produced by BOTH inter-subject variability of exposure and inter-subject exposure and pharmacokinetic properties. As a result, the dispersion in the measured blood Pb levels will overstate the dispersion in the intake-to blood Pb level distribution.

At several points in the document, it would have been useful to have a picture showing the spatial template for the dispersion calculations for both the smelter and road scenarios. A reader familiar with the modeling process can guess what these must have been, but a picture would have been better. This would also have been a good place to show how the census blocks/tracts were overlain. I assume the centroid of a block or tract was used as the point of exposure, but can't be sure given the discussion.

On Page 4-20, I cannot follow the justification for what seem to me thresholds in blood Pb levels. I know the authors state that these are "cut-points" below which calculations are not performed, but they function as thresholds if a reader then interprets later tables of results as indicating no risk at the lower percentiles. We can discuss this at the meeting, as there is a need to be clear in the document as to the interpretation of a dash (rather than an IQ loss value) in the summary tables.

On Page 4-22, the authors state that the sensitivity (elasticity) results can provide a "semi-qualitative feel" for the magnitude of uncertainty. "Semi-qualitative feel" strikes me as about the softest statement one can make about the quality of an uncertainty analysis. I am not even sure I know what it means. I remain unconvinced that any of the sensitivity results presented give much in the way of information on the degree of uncertainty. I doubt a more rigorous uncertainty analysis is justified, especially since this is a series of case studies and not a representative sample of locations throughout the country, so I may not push this issue too strongly.

On Page 4-24, the authors say that a block group was included in the study area if "the majority of their air concentration would come from the study area". I have no idea what this means. I assume the authors included a block group if more than 50% of the geographic area of the block group fell within the study area. It makes no sense to speak of the "majority of the air concentration".

I am comfortable that the particular models chosen throughout are appropriate for the purpose of this assessment, and so won't comment on these further here. The one issue I would raise is that the authors have assumed that the ratio of the exposure concentration to the ambient concentration can be obtained from NATA. This is valid if the particle size distribution is about the same, but invalid if this is not true. Some assurance that the assumption is valid seems warranted here.

On Page 4-34, the authors state that their justification for particular regression equations was based on goodness-of-fit and "other considerations". The latter criteria are much too vague and need to be specified to provide reasonable confidence in the regressions. But I do believe the regressions they developed are reasonable approximations to the data. They simply need to display this goodness-of-fit in exposure-response figures showing the data and model fits.

I commented in Chapter 5 on the issue of the validation and verification of the Leggett model, and so won't repeat that here. Again, I am surprised that the Leggett model is now predicting blood Pb concentration below that of the IEUBK model, and so it would be useful for the authors to explain why this change has appeared. But it is clear from the validation/verification study conducted in this chapter that the models are predicting reasonable values when compared against the NHANES data (see Table 4-12).

Once the assessment methods are set up, the authors have done a good job of summarizing the results in the remaining tables, including the one on sensitivity. So, I have no qualms about these tables or that they accurately represent the results from the model runs. And I find the tables to include precisely the information needed to draw policy conclusions.

As a final comment, section 4.4.3.3.2 on Qualitative Discussion of Uncertainty contains very little useful information. I left this chapter with little appreciation for what the magnitude of uncertainties might be. As I said before, I am not convinced that any more rigorous analysis of uncertainty would be warranted given the non-random nature of the three case study sites and the inability to do a full nested variability-uncertainty analysis. But the authors should try to give at least some subjective estimate of the uncertainty (accurate to within a factor of 2, factor of 4, etc).

Overall, an impressive study that is probably as informative as one can find for these case studies.

Summary Comments:

- Even after several readings, I am unclear as to how the case studies will be used to decide any particular issue related to delisting or an appropriate regulatory limit. There must be a discussion in the report of the rationale behind application of the results to specific risk questions that will inform policy. Absent this discussion, it is impossible to judge whether a policy decision is warranted by the scientific analysis.

- The case studies themselves are well developed in terms of the computational steps. There are a number of key points in estimating exposures, however, where data are scarce, and this

affects the reliability of the risk estimates. I am particularly concerned that exposures due to contact with surfaces may be underestimated, and that exposures due to re-entrainment in indoor and outdoor air may be underestimated.

- The EPA should consider looking at all three ways of estimating blood Pb levels from exposure, and weight these equally in a form of uncertainty or sensitivity analysis.

- Overall, the analysis of uncertainty in the document is too qualitative and fails to adequately characterize that uncertainty. Variability is handled better. I especially like the use of the post-processing approach to generating variability distributions from central tendency estimates.

Comments on Chapter 5: The Primary Lead NAAQS

Doug Crawford-Brown

My comments here apply primarily to the issue of Statistical Form, as I will be the one guiding that discussion. Before providing those comments, I do have several other minor issues to raise with the current chapter. I end with bulleted comments summarizing my main points.

1. On Page 5-2, I don't understand the distinction being drawn between "evidence-based" and "quantitative exposure – and risk-based considerations". The last time I looked, evidence was being used in "quantitative exposure – and risk-based" assessments. I suppose the authors have some further distinction in mind, such as a distinction between (i) direct epidemiological studies of the impact of a policy and (ii) predictions of this impact based on separate exposure estimates and exposure-response relationships. But to call one evidence-based in distinction with the other isn't correct.

2. On Page 5-5, there is a discussion of the target level (99.5%) of the population to be brought below a maximum safe blood lead level. While I agree with the sentiment here, the ability to reliably estimate the upper tail of this distribution, especially a value (99.5%) so far into that tail, is severely limited at present. I haven't done a formal uncertainty analysis of this estimate, but my guess is that the confidence interval would be quite large.

Now to the issue of Statistical Form. The current form is to use a strict average over a calendar quarter. The Agency appears to have considered a variety of alternatives, ranging from a violation if ANY measurement is above the limit (1.5 μg/L), to monthly, to even annual averages. In the end, the quarterly average was selected based in part on the larger number of samples that would be averaged (reducing statistical variability due to small sample size) and the fact that the human body in a sense "integrates" or "smoothes" exposures through storage in body tissue.

The quarterly average appears to me a reasonable compromise scientifically. Given the possibility of windows of vulnerability during development, it would not have been wise to use an annual average, as these windows probably contain developmental processes that are on a scale closer to months than years. And I agree with the authors that both the issues of sample size and storage in tissue make any statistical form on the scale of individual measurements or even weeks subject to too many false negatives and false positives.

There is some ambiguity in at least the current chapter as to what is meant by a "maximum arithmetic mean". Consider a single year, with weekly measurements (and, hence, 52 measurements). I could imagine that the measurements for January, February and March are averaged to yield the first quarterly result. Then the same is done for April, May and June. And so on to yield four quarterly values. The rule then might be that the site is in violation if ANY of these four values exceeds 1.5 μg/L.

But we could also imagine a "rolling" quarterly average. One would use the first 13 measurements and compute an average. Then one would use measurements 2 through 14 and compute an average. Then one would use measurements 3 through 15, and so on. This would yield about 52 such "quarterly averages" in the year. These are sorted and the highest examined. If THAT one is above 1.5 μg/L, there is a violation. I simply am not sure which of these approaches the authors intend. I believe the latter is the more justified approach.

On a related note, there is also the issue of the sampling schedule. I am completely comfortable with the Statistical Form selected if the samples are on a fixed schedule, or are randomly scheduled throughout the year. We just need to be sure there is no chance that the monitoring includes deliberate over-sampling during periods of high concentration. I realize that such over-sampling is not supposed to be part of a sampling program for compliance monitoring, but there is always the possibility that a manager somewhere will want to deviate from the sampling schedule when problems are suspected. I just want to be sure this issue is built into the sampling used for the compliance monitoring.

Other than these points, I am comfortable with the Statistical Form selected by the EPA.

Summary Comments:

- I am not confident that the 99.5 percentile of the variability distribution for blood Pb levels can be estimated reliably, and so don't believe it is appropriate to use such a percentile in establishing any sort of limits on exposure.

- On Statistical Form, this issue must be discussed by the EPA after the Averaging Period is established. The appropriate form to be used depends on the averaging period, the frequency of sampling needed for such a period, and on the monitoring methods appropriate to such a frequency. It is clear that, whatever Statistical Form is used, it must be chosen based on the rates of false positives and false negatives for the particular monitoring methods, sample frequency and averaging time.

- I doubt there is a biological justification for going to a monthly rather than quarterly averaging time, as this would presume a rather narrow developmental window. And I would be particularly concerned about an attempt to choose a short averaging time simply as a way to get more stringent controls on exposures. The averaging time needs to be based on biological considerations (e.g., developmental windows) and not on broader policy goals at this stage of the NAAQS process.

Comments on Chapter 5: Human Exposure Assessment and Blood Pb Estimation

Doug Crawford-Brown

As a general comment, I am supportive of the overall approach being taken in this assessment, which has several aspects:

1. Modeling of the variability of health risk from Pb exposures in a series of case studies that include primary and secondary smelters and exposures near roads.

2. Use of the biokinetics models as the basis for converting exposure information to estimates of blood burden. I also support the idea of examining the variation between the two models proposed to provide a sort of sensitivity analysis examining the robustness of the results.

3. Use of inter-subject variability distributions to be applied in post-processing to central tendency values generated by the exposure and biokinetics models.

4. Performing the variability analysis in part by modeling the exposures in individual blocks within the geographic area, and sampling individuals from these blocks (followed by the post-processing treatment of inter-subject variability in the relationship between exposure and blood lead levels).

Having said this, I have some specific issues to raise:

1. On Page 5-2, I can't begin to unpack that first paragraph. Two dose metrics are mentioned: a lifetime average, which is said to be between age 6 to 84 months, and a concurrent concentration, defined as the average over ages 73 to 84 months. What is the difference here? Both seem to focus on one year immediately starting at age 6. We aren't given the code, so I can't determine whether there is an actual difference between these two metrics, perhaps with poor wording here. We can discuss this.

2. In that same paragraph, the claim is made that these models yield central tendency estimates of blood lead. I am not sure that this is strictly true, given that the parameter values used don't seem to me to be all central tendency values, if by this the authors mean median values. And in any event, the model is so complex that I doubt the use of all central tendency values yields a final

estimate of burden that is itself the central tendency. But perhaps I am being too picky on this point, as the science isn't good enough to develop fully probabilistic versions of the models yet,

In any event, this issue is important because the post-processing approach of multiplying the central tendency estimate by a randomly selected value from a lognormal distribution with geometric mean (median) of 1 depends critically on the model result being interpreted as a median (and not only the looser concept of a central tendency value).

3. On Page 5-3, I am not sure I would agree that the Leggett model is widely used and subject to a lot of validation and verification. At least in the original version of the All Ages Lead Model I reviewed for the EPA, there was a significant difference between the Leggett and IEUBK models, with the former predicting higher concentrations than the latter. This is even mentioned on Page 5-7 of the current document. I am interested in the fact that in this new assessment, the pattern is reversed, with Leggett predicting under the IEUBK model. Perhaps this indicates some error in the original use of the Leggett model was uncovered and corrected (the authors of the current document suggest the Leggett model was "tailored" for the current application – I am not sure what they mean by "tailored"), but I suggest the CASAC determine whether this is the case. The authors of the current document state in the footnote that they are doing additional model evaluations, and it will be interesting to see how these turn out. In any event, the current agreement between the IEUBK, Leggett and NHANES results, shown later in the chapter, seems reasonably good at present, and certainly within the error bounds of both the models and the data.

4. On Page 5-12, second paragraph, the authors say that they have "added" a lognormally distributed term. I hope by "added" they mean "included" in the calculation. My understanding is that the randomly sampled term should be multiplied by the calculated median blood concentration to obtain an individual-specific blood concentration.

5. I am generally uncomfortable with the use of a generic water concentration and food concentration, rather than a distribution. But part of my discomfort comes from the fact that I am not clear from the document whether the post-processing inter-subject variability distribution correction was applied only to the component from air/soil, followed by addition of the non-air/soil contributions, or whether all pathways were first summed and THEN the post-processing adjustment applied. If it is the latter, I am less concerned, but I can't be sure from the document

The reason this issue arose in my mind is Table 5-15, which shows the average proportion of exposure from policy-relevant sources increasing from about 50% at the median of the inter-subject variability distribution to 98% at the upper tails of the distribution. To get to 98%, it must be the case that there is a MUCH wider variation in blood Pb levels from policy-relevant sources than from the other sources (such as water). But that may be because everyone who is sampled is given the same water concentration. I worry that there may be significant under-dispersion of the variability distribution for the non-policy-relevant sources than for the policy-relevant ones, which would be increasingly apparent out in the tails of the aggregate exposure distribution. This would tend to over-state the case that the policy-relevant sources are the primary ways to control exposures and risks.

Having said this, I understand the large problems with incorporating inter-subject variability in the non-policy-relevant sources. It would require developing correlations between the sources, since I doubt that the sources are fully independent. For example, someone living in an old home with lead paint is probably more likely to live near a smelter, and also more likely to have elevated lead in the water due to aging pipes and fixtures. So, this issue isn't trivial to fix.

6. Finally, I believe the authors have used measured inter-subject variability in blood Pb levels within a study population to develop the lognormal distribution I mentioned previously (used in post-processing). The authors argue that the sample population consisted of individuals with equal exposures, and so the variation in blood Pb level is also the variation in the ratio of blood Pb over exposure. But I am not convinced this assumption is completely true. To the extent it is not, it will produce over-dispersion in the inter-subject variability adjustment factor used in the current assessment.

Summary Comments:

- There remains significant work to validate and verify any of the biokinetics models, but particularly that of Leggett.

- There is difficulty comparing any specific blood Pb level predictions in the current study against NHANES values due to significant differences in the composition of the populations and the levels of exposure. I am unconvinced, therefore, by the model-data comparisons in the document.

- The contribution of water and soil and food to the variability in exposure is underestimated since the same values are applied to all individuals in the region. However, I don't think it will be possible to obtain data needed to improve this situation significantly.

Dr. Andrew Friedland

Andy Friedland, Dartmouth College
1 February 2007

Preliminary Individual Review Comments on OAQPS "Staff Paper" First Draft
Chapter 6 "Policy Relevant Assessment of Welfare Effects" and TSD Chapter 7 "Ecological Risk Assessment"

In general, the staff paper Chapter 6 is a reasonable condensation and interpretation of the Air Quality Criteria Document for Pb (the "CD").

I think the most important information that needs to be taken away from this chapter that will inform both health and welfare discussions is the material on the legacy of the leaded gasoline era preserved in soils both near roadways and urban areas and in remote locations.

Page 6-8: "The deposition of gasoline-derived Pb into forest soils has produced a legacy of slow moving Pb that remains bound to organic materials despite the removal of Pb from most fuels and the resulting dramatic reductions in overall deposition rates."

Page 6-2 discusses mobilization of previously stored Pb. However, the fullest discussion in the Staff Paper occurs on page 2-7 which points out the lack of estimates for Pb emissions from re-suspension of Pb residing in roadway dust and soil and from burning of biomass materials. The Staff Paper describes that the effects of emissions from forest fires or mechanical disturbance of soils are unknown. Finally, it states: "As described in the CD (Section 8.2.2), re-suspension of soil bound Pb and contaminated road dust may be a significant source of airborne Pb…."

These and other sections establish that: (1) soils are an important potential source of Pb for the foreseeable future and; (2) this is anthropogenic Pb stored in a natural substrate. Although it appears that this source is not particularly mobile at present, it still merits short- and long-term monitoring due to its potential to contaminate other components of ecosystems and human systems long after more typical sources such as the atmosphere are no longer monitored due to lack of need.

Because the Pb is moving very slowly (i.e., hardly at all), it is actually present as a very concentrated deposit of Pb. Fires, changes in land use, or climatic events such as regional dust storms could mobilize significant quantities of Pb which would be harmful to humans and biological organisms downwind. This potential for harm needs to be kept in mind throughout various aspects of the Staff Paper and the Risk Assessment document.

Dr. Robert Goyer

Comments by Robert Goyer on QAQPS Staff Paper – First draft and HRT-pilot phase January 29, 2007

Chapter 5: The Primary Lead NAAQS

This Chapter addresses the general approach used to derive the current Pb NAAQS. An objective is to determine the adequacy of the current standard and to provide background for revision of the standard. The chapter systematically reviews the basis for the standard as established in 1978. and provides a number of charge questions that will assist in making the policy decision. My comments are in response to charge questions.

There is considerable new information that reinforces the association of exposure to lead with various effects supporting the 1978 standard. The critical effects then were neurocognitive effects on the developing nervous system particularly in children ages 1 to 6 years. The new evidence, both experimental and from human studies strengthen this association and reduces uncertainty regarding the quantitative relationship. Much more is known about the mechanisms responsible for these effects and their persistence with continued exposure

In terms of new information about exposure and effects, risk of neurological effects from exposure to the fetus in utero due to transplacental transfer to fetus from the mother and possible developmental effects during the neonatal period from lead in maternal milk have been identified. The quantitative relationships of the early in life exposure <1 yr have a high degree of uncertainty. Nevertheless the evidence identifying children in early childhood as the most sensitive population has been strengthened.

What extent does new information affect uncertainties?

New information does provide greater uncertainties regarding factors that influence host responsiveness. These include role of nutrition, (dietary iron, calcium) and polymorphisms, (e.g. alleles for ALAD, and vitamin D metabolism).

Are there questions regarding basic elements of current standard?

The new evidence suggests that the maximum safe level blood lead is below the level of 10-15 μg/dl on which the current standard is based. Again, in terms of uncertainty, the lower the level of the dose- effect relationship, the greater the risk if uncertainty. The new information reinforces the nature of the exposure-effects relationships and provides additional information about the quantitative aspects of these relationships.

There is no reason for questioning or calling into question the nature of the lead-exposure relationships considered at the last review (effects on the CNS, hematological and cardiovascular systems and kidneys) but the association of these effects with lead exposure has been

strengthened and newer studies provide additional information about their quantitative relationships particularly at lower levels.

New information about lead effects on other organ systems since the last review such as the immune system, skeletal system and reproductive system provide strength to the potential toxicity of exposure to lead particularly

The new information cited above and presented in detail in the 2006 AQCD does suggest that it is appropriate to consider revision of the current standard at least in terms of maximum safe levels of blood lead.

Are exposures of concern and health risks estimated to occur in areas that meet the current standard?

Cannot comment

Do causal associations extend to air quality levels that are as low or lower than had previously been observed?

I do not know

Does the new evidence provide support for a different lead indicator?

No, lead in blood is in close proximity (equilibrium) to target organs and cells.

Averaging Times?

I am not aware of any evidence to suggest any other time is better than the calendar quarter.

Suggestions for alternative standards?

Only that the standard for blood lead levels should be lower but do not have any comments for changing the air standard.

Comments on Lead human Exposure and Health Risk Assessment — Pilot Phase

Chapter 2. Overview of Risk assessment

The basis for the risk assessment is an estimate of neurological effects on young children from exposures to lead emitted into ambient air for three case studies, a primary Pb smelter, a secondary Pb smelter and a near roadway urban location. A question might be whether these three examples are appropriate basis for risk assessment but they do represent real world situations.

The pathways shown in the conceptual model Exhibit 2-1 seem appropriate. The model correctly includes contribution of air lead to pathways resulting in ingestion as well as inhalation. Blood lead levels were predicted for a child population at each case study using the IEUBK and Leggett models. The cutpoints of 2.4 and 6.1 μ/dl are based on results of the models as blood lead levels below which IQ loss was not to be predicted. These cutpoints are dependent on the validity of the models. I cannot comment on application and interpretation of the models.

Mr. Sean Hays

Comments on EPA's Lead Human Exposure and Health Risk Assessments and Ecological Risk Assessment for Selected Areas
By: Sean Hays, Summit Toxicology
February 22, 2007

EPA's exposure and risk assessment should help inform how children and the general population are being exposed, especially at the higher exposure scenarios. These exposure and risk assessments should help give insights on how the children with the highest blood lead levels are being exposed and what can be done to lower their blood lead levels via lowering the NAAQS for lead. In this vain, it is instructive to see how the EPA's risk assessment predicted blood lead levels for their various scenarios (primary lead smelter, secondary lead smelter and living near roadways) compares to the blood lead levels observed in the US population from the most recent CDC NHANES biomonitoring survey. The following figures show these comparisons. Figure 1 is a cumulative probability plot of the CDC NHANES results from the most recent survey (representing blood samples collected during 2000 and 2001) of children ages 1 – 5 years (CDC, 2005).

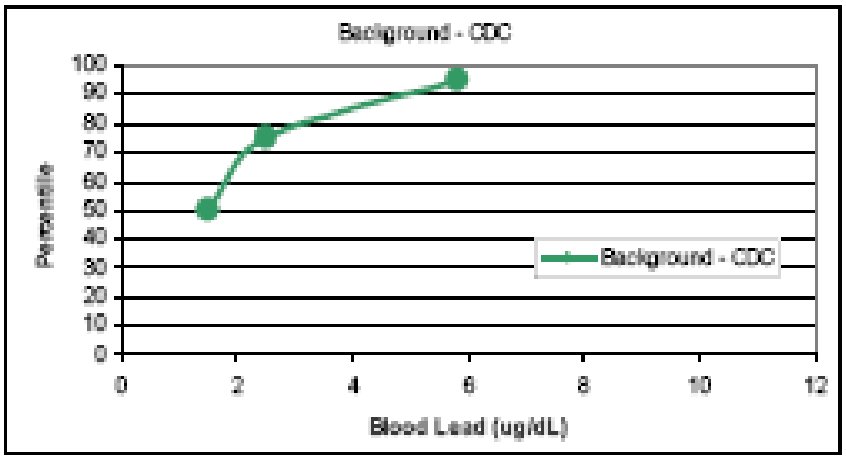


Figure 1: Cumulative probability distribution for blood lead levels among children ages 1 – 5 years of age from samples collected in 2000 and 2001 (CDC, 2005).

It is instructive to compare these results with those of the predicted distributions of blood lead levels from EPA's risk assessment (Figure 2).

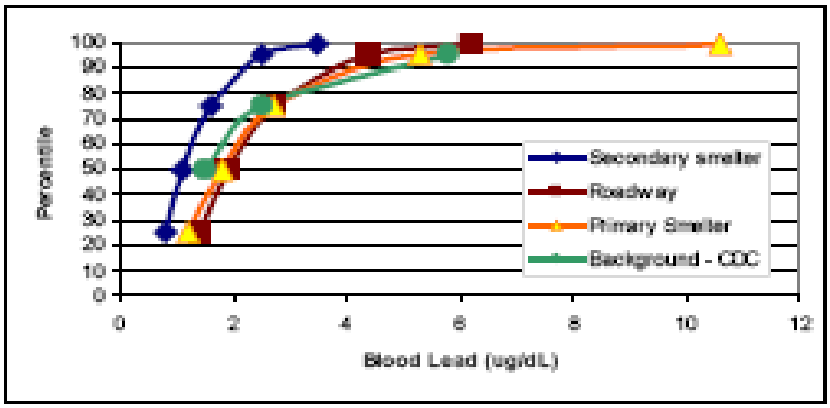


Figure 2: Cumulative probability distributions for blood lead levels from the general population (from Figure 1) and from EPA's risk assessment scenarios.

EPA's Risk Assessment Does Not Inform How the Highly Exposed Children are Being Exposed

As can be seen from Figure 2, the results from EPA's risk assessment predict blood lead levels among children that are either consistent with the distribution of current blood lead levels among children in the US or slightly lower for their various scenarios. As a result, EPA's risk assessment has not informed the reviewer of this risk assessment how the most highly exposed children are being exposed and how lowering the NAAQS might help to lower their blood lead levels. As a result, as is currently written, this risk assessment has not done its job of informing the CASAC how a change in the NAAQS might help lower blood lead levels among children in the US.

Potential Problems With EPA's Risk Assessment

Potential problems with EPA's risk assessment include:

1) *Wrong scenarios*: It is possible that the scenarios chosen by EPA do actually result in their predicted blood lead levels, but are not the scenarios that are actually leading to the higher blood lead levels among children in the US. In which case, the most logical explanation is that the source of lead among the children in the US with the most elevated lead levels are in fact due to lead based paint in their homes, etc. EPA should provide an analysis that is compelling in terms of how the most highly exposed children are in fact getting their elevated blood lead levels. *If this cannot be done, then EPA should explore alternative risk assessment approaches to support this lead NAAQS.*
2) *Wrong Assumptions About Exposure Scenarios*: It is possible that EPA has corrected guessed the scenarios that cause elevated blood lead levels among children in the US, but in fact their assumptions about exposures in the scenarios are biased low. A simple comparison of EPA's modeling results with some data from a community that lives near a primary smelter helps to inform this issue. Figure 3 presents the same results as in Figure 2 with the addition of some blood lead level results from residents living in the community of Herculaneum. It can be seen that EPA's risk assessment results predict lower blood lead levels than were measured in a limited survey of residents in Herculaneum (data obtained

from http://www.cdc.gov/nceh/publications/statefacts/mofactsheet.htm). *EPA should explore how their exposure scenarios may be biased low.*

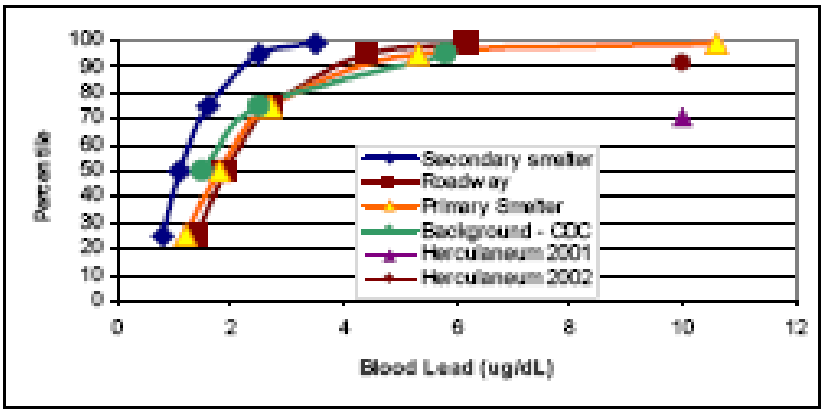


Figure 3: Cumulative probability distribution plot of blood lead levels predicted in EPA's risk assessment, from the CDC, and from children living in Herculaneum.

3) ***Unreliability in Model Results***: It is possible that EPA has chosen the appropriate scenarios for elevated blood lead levels among children in the US and the exposure scenarios are correct, however, the blood lead kinetic models and empirical relationships are not accurate for predicting "**absolute**" blood lead levels for a population. There is considerable uncertainty associated with predicting absolute blood lead levels among a population when there is considerable uncertainty and variability in the sources of exposure, the media lead concentrations, contact rates, and bioavailability. The models used by EPA for predicting blood lead levels among children are based on data that have shown relative changes in blood lead levels associated with changes in various exposure scenarios and/or were parameterized using data on changes in blood lead levels resulting from controlled lead dosing studies in rodents, non-human primates and in humans. For this reason, the blood lead models are much more reliable for predicting relative changes in blood lead levels for a delta in exposure scenarios. For this reason, *EPA should explore conducting a delta or relative risk assessment.*

Recommendations

To provide a risk assessment that helps the CASAC make an informed decision about how to advise the EPA on how the lead NAAQS should be updated, the EA needs to provide the following:

- Evidence for how children in the US with the highest blood lead levels are being exposed to lead.
- How a change in the NAAQS will help to lower both the national average and especially the upper quartile in blood lead levels among children in the US.

With this information, the CASAC can make a more informed decision on how to advise the EPA on how the lead NAAQS should be updated or modified.

References

CDC (2005). Third National Report on Human Exposure to Environmental Chemicals. National Center for Environmental Health Division of Laboratory Sciences Atlanta, Georgia 30341-3724 NCEH Pub. No. 05-0570

Dr. Bruce Lanphear

Characterization of Health Risks (Chapter 4) – Lanphear

1. To what extent are the assessment, interpretation and presentation of the results of the pilot exposure analysis technically sound, appropriately balanced and clearly communicated?

The case studies and scenarios used by US EPA were based on extremely limited data and therefore they were not technically sound. Nor was it obvious how the assumptions and modeling led to the results; thus, it was not clearly communicated.

It was not clear why there were only two scenarios; estimated IQ deficits at current ambient air lead levels and estimated IQ deficits if NAAQ standards were achieved. It is essential to thoroughly estimate the impact of lowering the lead NAAQS to various intervals, such as 0.5 $\mu g/m^3$, 0.25 $\mu g/m^3$ and 0.01 $\mu g/m^3$, depending on the distribution of ambient lead exposure in the United States. What percent of the population is exposed to air lead levels below each of these cut-offs? What is the estimated reduction in IQ deficits (or IQ benefits) from reductions in the lead NAAQS for each of these cut-off values?

Why didn't The Report consider what air lead standard would be required to achieve >99.5% of children to have a blood lead concentration < 2.4 $\mu g/dL$, < 1.0 $\mu g/dL$ (and lower), analogous to the approach that was used in the 1987 document?

2. Are the methods used to conduct the pilot exposure analysis … sound? Does the Panel have any suggestions for improvements in the methods used?

The methods are not sound. This absence of any data ultimately raises questions about the ability to define this Report as having any basis in science. The modeling relies on data that was largely "made-up".

On page 4-7, line 32: The Report provides a rationale for selection of case studies, including availability of site-specific monitoring data. Yet it was troubling how little data existed for any of the case studies. In the vast majority of cases, there was inadequate data on blood lead levels, soil lead concentrations, dust lead levels or water lead levels. As such, there is no evidence presented to allow the Committee to have any confidence that the estimated benefit of reducing (or not reducing) are reasonable; indeed, as noted in the report Page 4-80, line 14-16), the risk estimates for the 90^{th} to 99.9^{th} percentile could vary by up to several hundred percent.

Page 4-8: The Report indicates that they were unable to a satisfactory location for examining a multiple-source case study for inclusion as a case study. This is incorrect and unsatisfactory for a public health standard. In the last CASAC advisory meeting, I recommended that The Report provide parallel evidence that any data generated using biokinetic models was consistent with epidemiologic data. This is essential given the paucity of empiric data used in the Case Studies.

I also indicated that there were two data sets available from Rochester, NY with extensive measures of environmental lead exposure (paint, dust, soil and water) and children's blood lead levels. In the first study, which was a random sample, there were 204 children 1who were 2 to 31 months of age with extensive measures of dust lead (troughs, sills and floors), paint lead, soil lead and water lead. Air monitoring data are available from the local health authority.

In the second study, a prospective cohort involving 248 representative children followed from 6 months to 24 months of age, blood lead levels and dust lead levels were available at 6-month intervals. Soil lead, paint lead and water lead were available at baseline or if children moved to a different housing unit. Although there are the usual caveats that the results may not be generalizable, there are at least actual data to test the numerous assumptions inherent in the case studies. This latter study has the added value of having IQ results linked with the exposure data.

3. What are the Panel's views on the performance evaluation?

Once again, given the lack of any data, it was of limited value.

4. In general, are the concentration-response functions and blood lead metrics (i.e., lifetime average and concurrent blood lead) used in the pilot analysis appropriate for this review?

They are reasonable metrics to select based on the existing evidence, which generally indicates that chronic measures of exposure are stronger predictors of lead-associated deficits than peak or early childhood.

However, on page 4-20, the Report relied on 2.4 μg/dL (concurrent blood lead concentration) as the lower cut-point for estimated decrements in QI scores. This cut-off, which was reported in the pooled analysis to ensure that we didn't overestimate the adverse consequences of lead exposure, doesn't provide any margin of safety. A lower cut-off — including levels below 1 μg/dL — should be examined for additional benefits because the NAAQ standard is intended to provide a margin of safety.

5. Are the methods used to conduct the pilot health risk assessment, including the application of the cutpoints in relation to the concentration-response functions employed technically sound? Does the Panel have suggestions for improvement in the methods used?

As noted above, a lower cut-off, such as blood lead levels below 1 μg/dL, should be examined for additional benefits because the NAAQ standard is intended to provide a margin of safety.

It is also important to essential to estimate the impact of lowering the lead NAAQS to various intervals (e.g., 0.5 $\mu g/m^3$, 0.25 $\mu g/m^3$ and 0.01$\mu g/m^3$). What percent of the population is exposed to air lead levels below each of these cut-offs? What is the estimated reduction in IQ deficits (or IQ benefits) from reductions in the lead NAAQS for each of these cut-off values?

6. To what extent does the sensitivity analysis completed for the pilot analysis identify key sources of uncertainty and provide an assessment of their impact on risk results.

It does provide some evidence of the uncertainties and their impacts. In particular, on page 4-80, line 14-16, the Report indicates that the risk estimates for the 90th to 99.9th percentile could vary by up to several hundred percent. This conclusion, along with the fact that the vast majority of the blood lead and exposure variables were contrived, confirms that parallel epidemiologic studies are critical to have any confidence in the revised NAAQ lead standard.

7. Does the Panel have specific recommendations about approaches that might be employed in the full-scale assessment for this purpose?

Yes. Use epidemiologic data.

8. What are the Panel's views on the most important issues toi be addressed in the subsequent full-scale human exposure and health assessment?

It is essential to examine additional case studies and additional air quality scenarios. These additional case studies and air quality standards must use empiric data; additional case studies that rely on populations without adequate blood lead or exposure variables are inadequate.

Page 4-13, line 6: The Report indicated that CASAC requested recognition of "the importance of dust lead loading". But there was no recognition or estimates for dust lead loading; instead all modeling appeared to rely on dust lead concentration. It is widely recognized that dust lead loading is a significantly stronger predictor of children's blood lead concentration. Moreover, dust lead loading is the metric used to estimate deposition of airborne lead and subsequent risk to childhood lead intake.

4-34: The inverse relationship of soil lead levels with interior dust lead levels only makes sense if remediation had occurred, which is indeed the case in the primary smelter site. This finding, while casually brushed aside (soil was subsequently excluded from the model), raises serious questions about the extremely limited data sets used in the Case Studies in the Report. Why weren't more of the existing data considered? Why did the Report fail to examine existing epidemiologic studies — as previously recommended by this Committee — to test results obtained from the case studies that lack any data?

Page 4-18, line 32: The Report indicated that, to predict ambient lead in indoor dust, they used empirical (regression) models that relate indoor dust to outdoor air lead and/or outdoor soil lead. It would be helpful, as repeatedly requested by Paul Mushak, to have these data tabulated. If they are tabulated elsewhere in the Report, it would be useful to describe where it can be found in this paragraph.

Page 4-12: It is disturbing, to say the least, that the vast majority the blood lead and environmental blood lead data were "made up." This makes the performance evaluation of limited value.

Page 4-37: The Report indicates that modeling of blood lead levels is required for the pilot analysis for a number of reasons (e.g., measured blood lead levels were only available for a small

fraction of the study population in the primary smelter study and entirely absent for the other two case studies; exposure needs to be apportioned between policy-relevant and background exposures and; potential changes in existing blood lead levels distributions need to be predicted given reductions in ambient air lead levels). But these blood lead levels are available for at least two epidemiologic studies — along with other exposures and ambient air lead levels. Moreover, the effect of lowering the air lead standard can be quantified and the various policy and background exposures apportioned.

The failure to use existing epidemiologic studies to test the reality of the biokinetic models is unacceptable, especially given the lack of any objective exposure data and blood lead levels for the case studies.

Page 4-37: The Report indicates that it is unacceptable to use regression models for populations and exposure scenarios other than those used in their derivation. This is a false argument. First, the authors used contrived data from three case studies to "quantify" the effects of lowering the air lead standard to populations and exposure scenarios that will ultimately affect populations other than those used in their derivations. The only other rationale is that mechanistic models have greater flexibility in their application. This may be true. It is also true that it is easier to make up data than to use actual data, but that doesn't justify making up data to promulgate standards that are essential to protect the public's health. Conducting good science is rarely easy.

Page 4-39: The Report indicates that "a number of input parameters for both models have been adjusted to reflect the latest data on behavior, biokinetic models and lead exposure. This sounds reassuring, but it is not; there is no exposure data.

4-42, line 24: and 4.45, line 5-22: It would be quite important to understand the impact of using a GSD=2.0-2.2 (which is found in the NHANES data) versus the GSD=1.6, used in the exercises. On the surface, it would seem more relevant to use the GSD=2.1 for a national air quality standard.

Page 4-49, line 19-24: The Report is incomplete. It is essential to thoroughly estimate the impact of lowering the lead NAAQS to various intervals, such as 0.5 $\mu g/m^3$, 0.25$\mu g/m^3$ and 0.01 $\mu g/m^3$, depending on the distribution of ambient lead exposure in the United States. What percent of the population is exposed to air lead levels below each of these cut-offs? What is the estimated reduction in IQ deficits (or IQ benefits) from reductions in the lead NAAQS for each of these cut-off values?

Page 4-59, line 22-25: It is difficult to believe that there wouldn't be additional IQ benefits with further reductions in ambient lead exposure. This conclusion — which is in conflict with the prevention principle — and the fact that most of the data used in the simulated models were also simulated, raises serious questions about the entire Report.

Page 4-61, lines 1-9: It is implausible, from my recollection of participating in an ATSDR workshop on Herculaneum that included extensive measures of children's blood lead, that only 1% to 10% of children in this primary lead smelter case study would experience IQ point losses

at current or proposed attainment of 1.5 $\mu g/m^3$ from ambient air lead exposure. The only way this makes sense is if you limit the range of exposure to such a small increment that any change is inconsequential.

Page 4-72, footnote: The footnote "it was necessary to convert dust lead concentrations derived for each study site into dust lead loading" is inaccurate. Many of the dust lead measures were measured only as dust lead loading. Moreover, the investigators of the pooled dust lead study, which included many of the national experts in lead exposure, unanimously concluded that it was not useful to convert dust lead loading into a dust lead concentration (or vice versa).

4-77, lines 20-40: The report indicates that the Performance Evaluation involved comparison of modeled results with available empiric data to characterize potential uncertainty. There was not, however, sufficient comparison of the mechanistic models with empiric (i.e., epidemiologic) data.

Dr. Samuel Luoma

Comments on Lead Staff paper and Risk Assessment
February 2007
Samuel N. Luoma

The staff paper must meet the challenge of linking atmospheric inputs to water, sediments and soils to ecological risks. The measures required are concentrations in air, concentrations in water, sediment and soils and the effects concentration (measures of ecological risk) in each media. The greatest uncertainty lies in the measures of risk. The in-depth evaluation and synthesis is insufficient for the reader to get a grasp of the very great uncertainties in all the standards. For example,

1. the Eco-SSLs can only be described as wildly incoherent; 11 mg/Kg for birds (probably below most background Pb concentrations) and 1700 mg/Kg (typical of the worst mine-contaminated soils – i.e. "slickens") for invertebrates simply do not make biological or ecological sense. The agency must be commended for using good professional judgment in choosing more a more coherent set of Eco-SSL's in the risk assessments. But those choices do eliminate correction factors built in to correct for the great uncertainty in toxicity tests relative to the environment. More science is needed to make these standards more defensible. On the other hand, the very large HQs that occur in some instances in the case study suggest the high end tail of the effects distribution is not being eliminated by implementation of the current standard.

2. The agency must also be commended for choosing the NOAA/MacDonald statistical approach in evaluating risks from sediment contamination. This approach has many limitations, but its great advantages are simplicity and the way it constrains different levels of uncertainty about effects from Pb contamination in sediment. These are the most informative analyses in the risk assessment and the case study. Using these risk levels with the NAWQA data set was a way to address the question of risks from widely distributed lead concentrations. The several very HQs relative to the PEC in the NAWQA data were somewhat surprising, again raising questions about existing standard as implemented. However, it was noted that most of these effects were not from modern atmospheric inputs (historically deposited atmospheric Pb was not eliminated, however). The core data presented from the Great Lakes does allow an analysis (Fig 2-22 from Yohn et al 2004). It suggests that there were substantial ecological risks from at least some sediments in the Great Lakes when inputs were at their highest, even though the 90th percentile of atmospheric concentrations at that time were ~1 $\mu g/m^3$. This suggests that if a standard were set at 1 $\mu g/m^3$ and atmospheric concentrations were allowed to go back to that level then it is likely that there would be risk for ecological damage in many aquatic environments (HQ>>1 based upon PEC). This logic suggests that a regression of historical, dated lake sediments against atmospheric lead might be a way to address the question of how atmosphere links to ecosystems. The TEC and PEC approach might be used to estimate risks based upon that curve. It is also notable that at the ~1 $\mu g/m^3$ 90^{th} percentile atmospheric Pb, dissolved concentrations in the open oceans rose to >30X

undisturbed Pb concentrations in the ocean. It is not possible to relate this to ecological risk to open ocean ecosystems (the toxicity testing data is probably irrelevant for this pristine environment); but from a common sense point of view one must ask if we want a standard that would allow us to go back to this condition?

3. The HQs developed from NAWQA data and the AWQC, unfortunately, do not take sufficient account of the predominance of bad analytical data (for dissolved Pb) in the literature and the limited value of standardized dissolved toxicity tests in establishing risk limits. One solution would be to limit NAWQA data to recent years. In any case the analytical uncertainties raise questions about the credibility of both high and low HQs. The report should be commended for using a multi-media approach (sediment and water) to evaluate situations where HQ>1; but should add a similar analysis for HQ<1 for water and/or HQ>TEC for sediment.

For the future, modeling efforts are needed to link any proposed standard to the eventual Pb concentrations in water, sediments and soils. This could go on after a standard is promulgated and monitoring data begin to come in. "Critical loading models" are being used in Europe to great effect in understanding trajectories in soil contamination in response to existing or forecast atmospheric concentrations of metals. The regulatory process and use of indicators would benefit from exploring use of these models.

An analytical, weight of evidence synthesis is not available of the existing literature (toxicology, geochemistry, processes and observations of nature) relevant to water, sediment and soil standards. This could be another way to reduce uncertainties in the standards against which atmospheric inputs are compared. Contradictions among such lines of evidence should be used to point toward needs for greater understanding. The sensitivity of different ecosystems subjected to atmospheric lead inputs might also be systematically recognized in such a review (e.g. are pristine systems like the open ocean and large lakes sensitive to atmospheric Pb inputs? Can the historic data be used to evaluate such questions?). Again, looking toward the future, approaches like Europe's water framework directive might lead us toward a more a unified view of atmosphere, soils, water and sediments with regard to inputs, concentrations and ecological risk. Unification could guide a research agenda that could vastly improve the justification for future atmospheric Pb standards.

Dr. Frederick J. Miller

Fred J. Miller, Ph.D.
February 16, 2007

Chapter 3 Policy-Relevant Assessment of Health Effects Evidence

General Comments

Overall, staff has done an excellent job of summarizing the health effects of Pb most relevant for policy and NAAQS standards issues. The chapter is well written and staff clearly describes the key studies from the CD that support the points they discuss. Organizationally, the chapter structure assists the reader in following the basis for which endpoints and subpopulations should be focused upon in the risk assessment that will be done later, as well as the Pb dose metrics that should be used.

CASAC's discussion at the Feb. 5-6 meeting was very useful relative to staff including a "hypothetical threshold or cutpoint" in the pilot quantitative risk assessment that is subsequently described in the document (i.e., a blood Pb value of 2.4 μg/dL for concurrent blood Pb level and 6.1 μg/dL for lifetime average blood Pb). The net effect of the selection of a threshold is to reduce resulting risk estimates for whatever biological endpoint is being examined. While the epidemiology studies conducted at low levels provide no clear cut evidence of a threshold, neither do they demonstrate that there is no threshold.

In view of the long-range public health importance of CNS cognitive effects in children, an endpoint that staff has selected for quantitative risk analyses, this reviewer is now of the of the opinion that the risk analyses should be done without incorporation of a threshold. The additional new studies circulated during the Feb. 5-6 meeting show effects on attention deficit below 2 μg/dL, effects on cardiovascular mortality and stroke below 10 μg/dL, etc. The importance of the level for a standard can be approached from a risk viewpoint by looking at the percentage of the population that would be at risk rather than by assuming there is some exposure level below which there is no risk.

Specific Comments

Page & line	Comment
3-2, 17	The use of "upper respiratory tract" is not correct here. Suggest replacing with 'head and in the conducting airways".
3-11, 13	Staff in this paragraph quote the CD for implying that Pb may play a role in the epigenesist of behavioral problems in inner-city children. This contention is very poorly supported by any data and this paragraph is too speculative. Suggest deleting this paragraph.
3-15, 14	The statement about the change of 1 millimeter mercury increase in systolic pressure per doubling of blood Pb is confusing. Suggest a specific example be included (e.g., a blood level increase from X to Y results in a systolic pressure increase of Z).

Dr. Paul Mushak

PRE-MEETING COMMENTS ON:

REVIEW OF THE NATIONAL AMBIENT AIR QUALITY STANDARDS FOR LEAD (Pb): POLICY ASSESSMENT OF SCIENTIFIC AND TECHNICAL INFORMATION OAQPS STAFF PAPER - FIRST DRAFT

Reviewer: Paul Mushak, Ph.D.

I have both general and specific comments about the referenced document. I confine comments to the first five Chapters, and defer comments on the welfare/ecological effects Chapter (6) to the Panel's ecotox experts. My overall critique of this draft document addresses clarity, organization, quality of analysis, validity of conclusions, etc.

Specific comments are offered across Chapters in addition to comments within the framework requested by OAQPS staff.

I. GENERAL COMMENTS ON THE DRAFT STAFF PAPER

This draft represents an enormous amount of work by OAQPS staff and contractors and they are to be commended for the effort. I do have general comments about the case study procedures and the results. My comments cover both scientific aspects and the editorial/organizational status of the draft.

This body of work suffers from some unclear writing and some verbosity. It can be tightened and clarified.

It is quite difficult to follow the writing in parts and there is considerable imprecision in what may be intended. These are complex topics and unclear writing makes them more complex. It's far easier to see all the devils in the details of these drafts when the details are clearly presented.

An example of seemingly garbled writing is data contained in Table 4.3, which deals with IEUBK input parameters for Pb-B modeling. The numbers and the text for Table 4.3, and the associated Table 5-11 in the Pilot risk assessment, need to be double-checked. Be sure that the bioavailability values given in the Table section for soil and dust Pb inputs are clear and correct for all three case scenarios. The bioavailability (uptake fraction) of Pb for the secondary smelter and roadway dust cases are the defaults typically used with the IEUBK model for dusts and soils, i.e., 0.3. However, the authors state for the secondary and near roadway values that the "USEPA (1989) reflects evidence that Pb in dust and soil is as accessible as dietary lead..." If the authors mean to use dietary uptake for the secondary smelter and roadway dust and soil uptakes, then the value is 0.5, not 0.3.

The discussion of values stated for the primary smelter soil and dust uptake seems even more confusing. Are the values of 0.48 for soil and 0.26 for dust relative bioavailability (RBA), i.e., "bioaccessibility" (which is system-specific solubility) or absolute bioavailability? Relative bioavailability is reduced by half to get absolute bioavailability (ABA), i.e.,

ABA = RBA x 0.5 (maximum uptake from the child's gut for fully soluble lead)

Check the original data for the dust versus soil Pb values. The dust versus soil values seem to be reversed.

There are some problems with the organization of the draft. Why is the Chapter on welfare and ecological effects placed after Chapter 5, the latter a sort of general schematic for how EPA regulates criteria pollutants such as lead to come up with a de-facto history of the current NAAQS. The same sequencing problem arose with the original AQCD draft. The NAAQS for lead has both primary and secondary rationales for the standards. One needs to have the material for both types before launching into discussions of NAAQS.

Should a chapter on NAAQS development appear in this first draft at all, since no proposed NAAQS options are presented? Is this simply a placeholder draft chapter? The NAAQS development chapter in general says little about the proposed "transitioning" process and little about the logic behind the proposed process. If it's historically useful for the draft, why have it as Chapter 5 instead of a Chapter 1 or 2, "History"? Past usually goes before present.

Might not the subject matter of Chapter 5 be expanded to clearly set forth the various regulatory schemes for lead, such as control of air lead through various parts of Sec. 112 of the 1970 CAA and 1990 CAA Amendments dealing with hazardous air pollutants (HAPs)?

General Comments on Chapter 4

This draft's functional innards for human population impacts of (generally) current condition ambient air lead impacts are to be found in Chapter 4, but preceding Chapters are important as well. Chapter 3 mainly presents the important adverse effects and dose-response data for the risk evaluation and quantification methodologies. The exposure approaches are summarized in Chapter 4 and more is provided in the supporting Pilot Study draft. Chapter 3 was generally well done.

<u>Exposure Modeling</u>

Utility of the biokinetic models described in the Pilot draft and summarized in Sec. 4.3 of the staff paper draft is of major importance, the models providing the exposure link between environmental lead inputs and resulting neurotoxic risks expressed as IQ point losses. I believe, however, that the authors should summarize the interim findings of the SAB Panel that is reviewing the all ages lead model (AALM) and various other biokinetic models for EPA lead regulatory use, especially how the supporting IEUBK, the Leggett (Pounds Leggett) and O'Flaherty models perform relative to each other. A number of members of this CASAC Panel are also on that AALM Panel.

The authors should summarize the typical performance of the models and the principal biokinetic and exposure inputs that drive the models. For example, those of us who routinely use the IEUBK model know that it can either overestimate or underestimate validly and accurately measured Pb-B levels depending on such factors as the site-specific and site segment-specific uptake fraction (bioavailability) and the daily intake quantities of environmental lead media (dusts, soils).

IEUBK models can both underestimate and overestimate measured Pb-B at a given locale depending on the spatial segment of the risk study area being examined. Panel member Ian von Lindern has shown this for the reaches of the Bunker Hill lead Superfund site impacted along the lower Coeur d' Alene River Basin. The manual for the AALM and the background document provided to the models Panel by EPA's NCEA can be consulted for details on how the different models perform.

The O'Flaherty model will either underestimate or overestimate measured Pb-Bs depending on where along the dosing-Pb-B curve the comparison is done. Relative to each other, the input parameters for Pb-B output differ. Leggett uses different gut uptake fractions than does the IEUBK.

I think it's important to keep a quantitative perspective on the models vis-à-vis other elements of variability or uncertainty in these case-study scenarios. For these case studies, the greatest source of variability and uncertainty are the environmental measures, especially the modeled air Pb levels.

The draft notes that consideration will be given to both biokinetic lead exposure modeling and ad-hoc lead exposure statistical modeling, but this draft only appears to directly use regression relationships for inter-media estimating of lead content. The Lanphear et al. statistical model for Pb exposures was only used in the sensitivity analysis.

Summary of General Conclusions:

A. The Precise, and Only, Purpose of the Case Study Approach is to Provide the Administrator With a Range of NAAQS-Relevant Exposures and Risks Versus Air Lead Levels for Illustrative Point Sources and Selected Conditions.

- The authors need to be absolutely clear and precise about what is the actual utility of results from these three case study assessments so that any reviewer or other interested reader will not misinterpret the results, get ambiguous and erroneous messages, or misuse the results. The data from the three studies provide a three-part illustrative tableau of the range of current Pb exposures and associated health risk (IQ point loss) for ranges of environmental media Pb that would and do vary across the three illustrative cases.

- For example, as I read the point of the Pilot and eventually the Full Assessment exercises, the Administrator may conclude that he/she must regulate air Pb to the lowest air Pb (in whatever averaging and statistical air lead form) that eventually produces <1 IQ point

loss in 99.9% of U.S. children < 6 years of age. These three sets of results theoretically would provide the wherewithal for making this determination via selection(s) among the three options presented. That selection may be a single air Pb that springs from the Chapter 4 Tables or it may be a set of air Pb levels from the three study areas that give the same risk criterion of there being < 1 point IQ loss.

- If one misinterpreted the exercises here to mean they serve for source characterization globally or historically, then this would imply that one of the toxicologically safest places in America for families to raise young children is in the general proximity of an operating primary lead smelter which has very rarely been in regulatory compliance — over almost 30 years — with a relatively high, 29-year old air lead standard, and that has had a long recorded history of adverse impacts on the adjacent community in such forms as high frequencies of elevated Pb-Bs in young children living in the Herculaneum, MO community.

- It would also be erroneous to conclude that the illustrative results for the secondary operating smelter in this draft in any way characterize the operating history of this particular secondary smelter, secondary smelters historically or currently globally. That would mean that another safe place toxicologically for families to raise young children is in the proximity of a secondary lead smelter that (1) has the third highest lead emissions of 15 U.S. secondary smelters, (2) has actual air monitoring data that place air lead from this secondary smelter within the highest 15% of secondary smelter air lead monitoring results nationwide, and (3) is located in a county (Pike County, AL) which CDC in 2003 found has 8% of children with Pb-B >10 µg/dl, a fraction four times higher than national results and over three times the Alabama-wide figure.

B. <u>"New" Lead Inputs for Defining Health Risk are not Trivial Compared to Historical Levels</u>

- Finally, there are technical reasons for why "current condition" results cannot be used to support any policy conclusion that new inputs of Pb in any of the three case studies are minor compared to levels already accumulated. First, there are limitations I note that are rooted in the three-case methodology. There are other important reasons as well.

- First, as I noted in Tables of dust Pb loadings at different very low air Pb levels in my earlier AQCD comments provided last year, hazardous dust Pb loadings for interior and exterior will rapidly accumulate on hard surfaces encountered by children. Refer to those tables for details, especially health risks for children at an air Pb of 0.1µg/m^3. Any air Pb above or around the very low levels employed in those calculations will pose a risk.

- Secondly, the "current conditions" scenario for dust and soil Pb do not provide a temporal estimate of how quickly air Pb at "current conditions" will result in soil and dust Pb that become hazardous in the fullness of time. Equally troublesome, dust and soil leads used in the environmental Pb Tables give soil and dust Pb levels that are greatly underestimated when based on modeled air Pb. Table 4-11, Performance Evaluation, says the secondary smelter case shows modeled soil Pb is three times lower than a reference measured Pb data set.

- The authors also state in **Table 4-11**, "Performance Evaluation..." for the primary smelter case that Pb in soils were calculated directly from measured data for the remediation zone and extrapolation results for the site remainder. But the remediation zone soil Pb data are difficult to link to current air Pb. They represent rapid recontamination in terms of average lead content for recontamination from the smelter (arguably from fugitive dust movement). They cannot be explained by the actual dispersion-modeled ambient air lead levels provided even though dispersion modeling of the 266 tons of facility Pb included fugitive dusts (Pilot draft, Appendix B). The median air Pb level in **[Table 4-4]** appears along with a "measured" soil Pb median value of 84 ppm **[Table 4-5].**

- Could the authors clarify and specify the extent to which the listed median and other air Pb values for the primary and secondary smelter air Pb values reliably predict the indicated soil Pb distributions in the Tables? If they cannot do that, then it is pointless to talk about distinguishing "new" air, soil and dust Pb from "historical" air, soil and dust Pb in terms of relative ranking for adverse impacts on lead-exposed populations.

- Current air Pb cannot be considered trivial in terms of producing the main exposure drivers, soil and dust Pb. Secondly, soil Pb and undisturbed dust Pb are never in equilibrium, i.e., never in "steady state" with air Pb. The relationship of the former to the latter is time-contingent and the longer air Pb deposits onto soils, the higher the soil Pb will be. The primary smelter community recontamination data for soils appears to say that air Pb (as direct stack and fugitive dust inputs) has to be much greater than the modeled values.

C. Design and Results of the Case Study Approach, Selected Areas

- The conceptual approach and its associated methodological rationales for the case-study approach in this draft staff paper remain to be laid out clearly and adequately up front, as does a comparative discussion of other approaches one might use. I urged this be done in my previous comments for the Panel consultation done last year but I still don't see a clear discussion. The closest to this is buried back in Chapter 2 (p. 2-46, Findings and Limitations).

- The authors, in 4.1, lay out a nice and clear contrast as to the conceptual distinctions between the 1989-1990 Staff Paper assessment for NAAQS selection and the current approach. As the authors note, modeling of risk was done here, versus modeling of exposure indexed as Pb-B elevations and frequencies in the 1990 assessment. Modeling toxicodynamics (effects), as opposed to modeling of conventional toxicokinetics (exposures), is something new for the Agency's approach to lead risk assessment.

- The Staff Paper analyses and the supporting pilot study draft consist of (1) a computational marathon of sequential modeling data mixed to some extent with measurement data but mainly modeled data, and (2) modeling data sequentially fed as inputs into one or more downstream models to get even more modeling data.

- All of these methodological elements are for three case-study scenarios, each of which contains within themselves a raft of questions about suitability, problems complicating reliable use of the study scenarios for national projections, and serious data gaps.

- Tandem use of (1) case studies with their peculiarities and their problematic utility for national scenarios and (2) compounded modeling methodologies have produced results that raise a number of questions.

- The sequential modelings with the accompanying intra-model and inter-model compounding of variability and uncertainty mean that a high potential for mis-estimation exists and that even limited parameter selection and assumption biasing well upstream can eventually produce large errors in the downstream risk assessments – child neurotoxicity (IQ loss) risk.

- For example, the secondary smelter scenario for current conditions produces (1) modeled risks of IQ loss in young children from (2) biokinetically-modeled Pb-B levels that are based on the combination of (3) modeled dust and partially modeled soil lead from measurements done for other than the location of the smelter, which in turn were based on (4) dispersion-modeled ambient air lead levels that in turn are based on (5) only three stack test results done 7 to 10 years ago at the facility. A further concern for this case study is that air monitor data near the smelter indicates that 2003 ambient air lead, not used in the modeling, is likely much higher than the air leads found 1997-2000 (see Table 2-6 of the draft Staff Paper).

- This draft paper describes methodologies which operate in the dimensions of time and space. The dispersion modeling used a spatial reach of 10 km for the air Pb distributional statistics. However, their calculations indicate that 50% of background, from the primary smelter, is not reached closer than 50 km. Restriction to 10 km was dictated by run times.

- The authors indicate in Table 1 the cumulative time periods for testing air, soil and dust Pb as 2000 to 2005 for the primary smelter, 1997 to 2000, and circa 2001 for the roadway dust Pb scenario. At the same time, the authors for the primary smelter case make use of dust and soil Pb data that had accumulated for decades for the no remediation zone. Similarly, the secondary smelter "hybrid" scenario uses long-impacted soil Pb values.

- Assumptions and parameter selections used in this draft for the above evaluations frequently serve to underestimate exposure and associated risks in affected populations. For example, the soil lead results presented in **Table 4-5** for the projected soil Pb around the operating primary lead smelter represent "current condition" Pb that may or may not capture the significant role of fugitive dust Pb contributions in the remediation zone and to some distance beyond that.

- The combined impact of stack Pb and on-site fugitive dust sources for off-site Pb levels must be sizeable. I am not sure that one can get a stable soil Pb level near this smelter. The soil was remediated out to 1.5 km but the MO DNR has reported that there is rapid recontamination. EPA in follow-up has also shown serious recontamination for the 1.5

km distance corresponding to the remediation zone. Areas that were remediated with removal and use of low-lead soils were shown by the MO DNR to have recontamination with an average of 600 ppm lead. The highest recontamination level was 2400 ppm.

Ref:

Missouri Department of Natural Resources. 2002. Doe Run Herculaneum Smelter – Residential Yard Data on Historic Recontamination. February. Internet: www.dnr.state.mo.us/deq/herc.htm

Equally troubling is that remediated soil surfaces appear to have continued being recontaminated through May, 2006.

Ref:

U.S. EPA. 2006. Lead soil trend analysis through May 2006. Evaluation by individual quadrant. Herculaneum lead smelter site, Herculaneum, MO. Kansas City, KS: Region 7, US Environmental Protection Agency.

- One can readily calculate that to recontaminate from "clean" replacement soil at a low background Pb content (50 ppm Pb) to the above reported recontamination levels in a few years would require quite high direct stack and fugitive dust emissions. See USEPA 1996 for the calculations.

Ref: U.S. EPA. Urban Soil Lead Abatement Demonstration Project. Vol. I: Integrated Report. Report No. EPA/600/P-93/001aF Washington, DC: Office of Research and Development. 1996.

- Equation 2-3 in that document can be used to calculate that for a 1.0 $\mu g/m^3$ air Pb level, and an ultra-conservative deposition rate of 0.2 cm/sec, receiving soils collect lead at the rate of 3 ppm/year. The very rapid reaccumulation of lead in soils to an average of 600 ppm Pb over, say, four years or so, using the above indicates an annual recontamination rate of ca. 140 ppm (600 - 50 background) and an annual air Pb level of ca. 40-50 $\mu g/m^3$ from combined stack and fugitive dust releases.

- This air Pb figure can be compared to findings of air Pb levels proximate to the smelter ranging up to 85 $\mu g/m^3$ (noted in Ref. 4 of the ATSDR Consultation, Jan. 15, 2004, and discussed in that Consultation). A house with remediated soil showed, after two years, a soil lead level of 300 ppm, indicating 250 ppm of contamination (300 - 50 ppm low-Pb fill soil), at a rate of 125 ppm/year. This jibes with the above estimate from calculations. This corresponds to about 40 $\mu g/m^3$ air Pb as an average.

Performance Evaluations, Uncertainty Analyses, Etc.

- The authors need to use reality checks to do a full conceptual and methodological audit of their results at both the macro level of collective scientific conclusions and weight-of-

published-evidence and the more micro level of, for example, performance evaluations and sensitivity analyses. It is not useful to know one has a valid and reliable way to get to Union Square in New York if one actually has to be at Union Square in San Francisco.

- A critical point to keep in mind is that the exposure assessment and health risk determinations in the form of IQ losses laid out in Chapter 4 Tables in the draft Staff Paper are only for current conditions (see related discussion above) and the associated dust and soil lead increases reflect this current state. Percent frequency of measured children's Pb-Bs ≥ 10 in the Herculaneum area, especially for areas closer to the smelter, accord with exposures that appear much higher than what is modeled in the upper tails of Pb-B here.

- The 2002 screening of Herculaneum children's Pb-Bs by ATSDR showed 14% ≥ 10 μg/dl.

Refs:

ATSDR Health Consultation.2002. Herculaneum Lead Smelter Site, Herculaneum, Jefferson County, MO. Blood Lead Results for 2001 Calendar Year.

ATSDR Health Consultation.2003. Herculaneum Lead Smelter Site, Herculaneum, Jefferson County, MO. Blood Lead Results for 2002 Calendar Year.

- A second critical point is that the exposure and risk assessments are for the spatial reach of 10 km, while much of the Pb-B historical data have been gathered for child exposures close to the smelter, within a mile or so (1.5 km).

- The 2003 CDC sampling of Pb-B levels for children in the secondary smelter (Troy, AL) area needs to be evaluated for those results likely being driven by lead emissions from the smelter. Presumably, this secondary smelter community was selected in the first place because there were no other significant lead emitting point sources voiding the case study entry criterion that there be one principal lead source.

- The 2003 CDC Pb-B data for the secondary smelter community needs to be broken out via such devices as zip code exceedence frequencies and name-redacted Pb-B measurements for group-stratified Pb-B frequency dependence on proximity to the facility.

- The authors don't seem to connect with the fact that if a countywide Pb-B elevation frequency is 8%, 1-in-12, as determined by the 2003 CDC sampling, and this overall frequency occurs from and is driven by the secondary smelter emissions, the reported frequency is very likely a dilution of the higher frequency of Pb-B elevations closer to the facility, i.e., the latter is >8%.

- Overall, the Herculaneum projected soil Pb levels as medians are quite low **[Table 4-5]**, and approach typical background soil lead levels at many locales. The background soil

lead value for comparison with Herculaneum would be that of unimpacted agricultural soils distant from the primary smelter. This background is on the order of 50 ppm Pb or so. Soil recontamination and the likely elevated soil Pb levels for unremediated properties in the 8.5 km spatial interval from 1.5 to 10 km would clearly produce much higher soil Pb due to more than 100 years of lead depositions. A problem at this site is the relative absence of measured soil (and dust) lead levels beyond the immediate impact zone.

- This draft remains unclear about the flexibility of multi-layer sequential modeling to permit differentiation between current conditions, i.e., new air Pb inputs to compartments, versus the historical Pb levels already in place given the complexity and unwieldy nature of a multi-layered, multi-case study methodology. Results from this approach show sequential points of question in any differentiation of current from historical Pb impacts at the locales chosen. Pediatric blood lead levels were modeled as very low at these two smelter sites despite Pb-B screening data: ATSDR results for Herculaneum, CDC results for Pike County, AL **[Tables 4-7, 4-8: Leggett and IEUBK outputs]**. In some cases the modeled Pb-B values are actually approaching detection limits and typical laboratory measurement variability of Pb-B (1-2 µg/dl). There are the median Pb-B levels ranging from 0.7 to 1.8 µg/dl for the primary smelter case **[Table 4-7, current conditions and NAAQS attainment scenarios, both biokinetic models used in both modes]** and a primary smelter 95th percentile value of 2.0 µg/dl **[Table 4-7; concurrent Leggett, both current conditions exposures and NAAQS attainment exposures].**

- There are the median Pb-B levels in a range of 0.5 to 1.5 µg/dl for the secondary smelter case **[Table 4-8; model-only and hybrid cases, both models, both Pb-B metrics]** and a secondary smelter 99th percentile value as low as 1.4 µg/dl **[Table 4-8; model only, Leggett concurrent].**

- These very low Pb-Bs will give rise to IQ decrements that would be minimal or nonexistent **[Table 4-13, primary smelter, current exposures; Table 4-14, primary smelter, NAAQS attainment; Table 4-15, secondary lead smelter, modeled only; Table 4-16, hybrid]**. The primary smelter case results **[Tables 4-13, 4-14]** would have us believe that children in proximity to an operating primary lead smelter would have no more than a 1-in-100 chance (99th percentile) of having an IQ loss of as much as 3 points **[Tables 4-13 & 4-14]** and a loss of as little as <1 IQ point **[Tables 4-13 & 4-14, Leggett lifetime Pb-B metric, both current and NAAQS attainment scenarios]**.

- The secondary smelter case results **[Tables 4-15 & 4-16]** are equally lower than expected, given the weight of the empirical evidence. In particular, the modeling shows an operating secondary lead smelter has no likelihood of producing any IQ point loss in virtually all combinations of risk modeling depicted in these two Tables. In fact the highest modeled loss of IQ points is 2, for which there is only a 1-in-1000 chance (99.9th percentile) [**Table 4-16, hybrid scenario, concurrent IEUBK**]. The modeled soil approach for the secondary smelter shows **[Table 4-15, IEUBK concurrent Pb-B]** IQ point losses of 1, 1, and <1 for the 99.9, the 99.5 and 99th percentiles respectively.

- The authors would have the Panel believe that the authors could legitimately create a hypothetical threshold of either 2.4 or 6.0 µg/dl for whatever arcane purpose in Pb-B in the absence of any biological or toxicological evidence supporting this step. The literature and expert consensus documents support a conclusion of no thresholds to effects so far. The authors cannot simply argue that there's uncertainty below the range 2.4 - 6.0 µg/dl. Uncertainty cuts both ways. There is no more argument for setting a threshold for IQ decrements in this region than there is for not setting one. Setting a hypothetical threshold is not simply an arbitrary computational step. A threshold to lead toxicity indexed as blood lead has implications for multi-endpoint toxicity in children and implies a toxicological mechanism of action that has not been accepted for low-level lead neurotoxicity by the scientific and health communities.

- Besides scientific objections, an arbitrary setting of a Pb-B threshold for adverse effects would appear to violate the statutory language in Section 109 of the CAA. That section mandates use of an "adequate margin of safety" for those effects (or accompanying dose-response relationships) not fully amenable to quantification. Use of a purely arbitrary "hypothetical" threshold clearly violates the "adequate margin of safety" mandate in Sec. 109 while avoidance of any threshold accords with an adequate margin of safety and is therefore in accord with 109.

- Have the authors characterized the air Pb emissions for this primary smelter in terms of expected correlations with Pb production rates, and are NAAQS attainment achievements or failures artifacts of the lower or higher operating levels of the smelter in response to variable demands in the prevailing metal markets? The instability of smelter lead production rates due to market fluctuations means that "current conditions" have an economic component as well as environmental ones.

- The projected operating secondary lead smelter air lead levels are also low, but much lower than the primary smelter results. This relative ranking is expected, being five-fold lower at the median and 33-fold lower at the 95th percentile. However, the absolute levels seem lower than the bulk of the global literature on such facilities indicates and that the two site monitors would suggest.

- The Pilot support draft implies that this secondary smelter is not a particularly clean one, having lead emissions over 1997 to 2000 (three stack tests) that rank it as the third highest lead emitter of 15 remaining U.S. secondary smelters. Further evidence of a dirty facility are the two lead monitor results, which indicate ambient air levels that lie within the top 15% of air lead levels for secondary smelter releases nationally. Furthermore, information in the Pilot draft indicates that 2003 air lead monitoring data off-site may indicate this operation is getting dirtier via higher production rates or equipment aging and failures.

- There are other complications as well. The primary smelter owners in 2002 and before bought 160 of the homes closer to the facility. This block of purchased homes would not represent a typical household occupant distribution for purpose of census tract modeling

and would not fall within modeled dust and soil lead values. How have the authors dealt with these 160 smelter-owned properties?

- Herculaneum is also a community with a long history of exterior and interior dust lead levels and loadings. Dust lead levels in Herculaneum homes measured in 1992 were as high as 6000 ppm in children's bedrooms. An earlier set of interior dust Pb tests showed an average of ca. 1900 ppm and a maximum of 8,300 ppm. Exterior street and curb dusts measured in August 2001 showed lead content as high as 300,000 ppm on roads used to haul lead concentrate. The authors used measurement data for 17 homes to capture this accumulation.

- It is unlikely that the above interior dust, whatever the soil lead abatement efforts, would have ceased being released from interior reservoirs. It is also unlikely that road surfaces having 30,000 to 300,000 ppm would have been remediated enough by washing to void this as an exterior dust Pb reservoir. It is likely high external dust reservoirs as well as interior dust reservoirs remain. Also relevant to exterior dust levels, a March 2002 testing of a Thurwell St. address closer to the smelter showed a porch eave lead loading of 24,400 $\mu g/ft^2$ with associated high cadmium and arsenic levels. Co-occurrence of high arsenic and cadmium levels rules out porch lead paint and rules in a geochemical source.

<u>NHANES National Pb-B Data are no Validation of the Modeling Results for Pb-B in Three Specific Locales</u>

The authors seem to take comfort in noting that the modeling results are sort of in line with the latest NHANES survey data. The main problem with this is that one cannot disaggregate the statistical design and national snapshot numbers from an NHANES survey for the nation as a whole into site-specific Pb-B distributions. The 1988 ATSDR report to Congress on childhood lead poisoning in America makes clear that one cannot use data for the nation as a whole for comparisons to geographic areas smaller than the nation. The Executive Summary in this Congressional report notes on p. 4, top, that:

> "Valid estimates of the total number of lead-exposed children according to SMSAs [Standard Metropolitan Statistical Areas] or some other appropriate geographic unit smaller than the Nation as a whole cannot be made, given the available data. The only national data set for Pb-B levels in children comes from the National Health and Nutrition Examination Survey II (NHANES II) of CDC's National Center for Health Statistics. *The NHANES II statistical sampling plan, however, does not permit valid estimates to be made for geographic subsets of the total database* [Italics added for emphasis]."

This caveat to localized use of national NHANES II data was provided to me as the senior author of the Congressional report from the contributing authors representing the National Center for Health Statistics.

Alternative Approaches

- Given all the problems inherent or demonstrable in the case-study approaches in this draft, why is this approach at the end of the risk assessment day any more useful than any alternative that begins with use of actual environmental lead levels derived from measurement data at source-oriented monitoring sites. Specifically, the data for the 59 air Pb monitoring sites for nationwide industrial source air lead levels depicted in Figure 2-12 and text, p. 2-31??

- The driver for modeled lead exposure distributions (Pb-B) and modeled risk distributions (IQ loss) in any approach, and for this Pilot assessment, is simply the environmental lead inputs to the exposure models and the resulting Pb-B outputs being used for inputs to the IQ loss modeling. The exposure and risk results in the Chapter 4 Tables for the case studies are what they are mainly because the environmental lead inputs, especially air, dust and soil Pb, are what they are modeled to be.

- Based on these 59 source-oriented sites described in Fig. 2-12, measured air Pb levels for source-oriented monitors give air Pb distributions that are well above the air lead measurements and distributions for areas without lead source-oriented monitors. For example, medians for the former are 16X higher than for the latter. A key component of looking at Pb NAAQS for revision or other disposition is to first look at where the air lead is. Approaches that maximize use of measured data to show where air lead is do have merit.

- These 59 air lead monitor sites are known, their distances from the major lead sources are known for purposes of modeling soil and dust lead, their census unit child populations and demographics can be determined, etc., so statistical analyses and media Pb modeling from air lead can be done. It is not clear that policy-relevant partitioning of air lead levels from the 59 monitors pose any greater problems than do the assumptions and limitations in the case-study approach.

- Comparison of air Pb distributions for all the lead source-depicting air lead monitoring data on p. 2-31 with modeled data tabulated in Chapter 4 (Table 4-4) is done in my Comments Table 1 at the end of this commentary. The measured air Pb values greatly exceed the dispersion-modeled air Pb values, as seen with the median and 95th percentile values for the secondary smelter case study modeled air Pb levels given in **Table 4-4**. The ratio of measured vs. dispersion modeled air Pb for the secondary smelter scenario at the 95th percentile (where the Pb-exposed individuals would be those nearer to the secondary smelter), is 107, giving two full orders of magnitude underestimate when using modeling.

II. SPECIFIC COMMENTS

Chapter 1, Intro

pp. 1-3, 1-4 The discussion here of "adequate margin of safety" indicates that one cannot arbitrarily set a threshold (to effects) that actually voids an "adequate margin of safety."

Chapter 2, Characterization of Ambient Lead

p. 2-24, Figure 2-6. It is troubling that only 2 of 26 facilities (both lead smelters) having emissions of more than 5 tons/year have a NAAQS compliance monitor within one mile. This would appear to limit the reliability of the data depicted in Table 2-4 and Figures 2-2 and 2-3 for areas closer (<1 mile) to the emission sources. The actual amount of air lead measurement data for ambient air lead in the NEI 2002 must be assumed to be significant underestimates for the national air lead picture.

p. 2-28, Figure 2-10. It is also troubling that the air lead monitoring trend data were gathered for monitors that were principally set up as a function of (urban) population density, presumably areas with high vehicular densities and associated mobile lead source relationships. They were not set up for depicting point source-impacted air lead levels. While the trend curves in 2-10 say a lot about mobile source lead declines, it's problematic what they could say about the major source emissions depicted in Table 2-4.

p. 2-30, Figure 2-11 and text This discussion and the figure are important for comparisons with earlier text (p. 2-24, Figure 2-6). The discussion also offers some peculiar reasoning in terms of significance of air monitoring data that do and don't reflect capture of industrial emissions. The earlier text noted that only two air lead monitors were set up to more reliably capture air lead releases and these were for smelters. This Figure 2-11 says that data from 224 monitoring sites were used.

p. 2-31, Figure 2-12 and text Figure 2-12 requires further discussion in appropriate context. The median air lead value for the 59 source-oriented sites is 0.252 $\mu g/m^3$ and for the 95th percentile is 1.923 $\mu g/m^3$. These values greatly exceed the corresponding scenario modeling of air Pb given in Table 4-4 of Chapter 4. Comment Table 1 gives ratios of measured to dispersion-modeled air Pb. In the Comment Table, the ratio of the 95th percentile of recordings of air Pb levels for the 59 sites is over 100X higher than the corresponding modeled figure for the secondary smelter scenario.

Table 2-7, p. 2-37 This is an important Table in that it shows the impact of averaging times or forms for determination of fractional exceedence of standard levels and shows why expressions of the form and averaging time are as important a consideration as the level of the NAAQS itself. The maximum monthly average, as mathematically expected, always shows more exceedences than the other averaging and form combinations. As the reference air Pb levels increase, the ratio of maximum monthly average to 3-year annual average increases greatly.

p. 2-46, Findings and Limitations This is an important discussion, since it states the major limitations of this approach of using case studies.

p. 2-47, Air Quality Summary This summary correctly notes that the 30-day maximum average will capture more instances of elevated air lead excursions than the present quarterly maximum average. The reason for a 30-day average should also be expanded to include the fact that we know that air lead changes producing children's Pb-B changes are better captured over 30 days than a quarter. The authors should check the Pb AQCD for information on this.

p. 2-49, Sec. 2.6 The authors need to include exterior dusts on outside play surfaces, which can be a significant source of dust lead ingestion during play activities outside in warmer months and in warmer areas of the country.

p. 3-1, dose-response Authors need to point out that low-dose lead effects occur with no definable threshold indexed as Pb-B so far.

p. 3-14, Discussion of threshold cut points I have already stated criticisms of any use of threshold cut points in my general comments earlier.

pp. 4-1, 4-2 Ch. 4 Intro There is too little regarding the criteria for selection of case study locales. Also, there are no discussions on alternative general approaches to the case study approach. The primary operating smelter is necessarily confined to one choice, but there are 15 secondary smelters to choose from and an almost infinite set of road stretches with reentrained roadside dust lead.

On p. 4-1, What exactly are "buffered" populations noted in footnote 2 in terms of quantifying lead exposures?

p. 4-20, discussion of hypothetical threshold to Pb neurotoxicity See discussions on the topic in my general comments.

p. 4-38, IEUBK There seems to be some author confusion on application of the IEUBK model for childhood exposure. The model cannot be used for just community-wide expressions of lead exposure, and this proscription is stated in the IEUBK manual (1994). The model applies to a typical child or set of children, e.g., siblings, at a given lead exposure unit. EPA considers the exposure unit for regulatory actions at, say, Superfund sites to be the residential property, house plus yard. One can of course derive batch mode statistical comparisons for modeled vs. measured Pb-Bs etc. using all the individually modeled Pb-Bs derived for each residential exposure unit vs. their measured Pb-B. The broader the modeling net that's cast, the higher the required geometric standard deviation (GSD) and the higher the probability of more than one set of Pb-B distributions, for example a subset of children at lead "hot spots," being missed.

Table 4-3. IEUBK input parameters See earlier comments. The soil/dust bioavailability data are confusing. Dust Pb is stated to be about half that of soil lead, 26% vs. 48%. Typically dust Pb is more bioavailable than soil Pb because of smaller particles in dust, dust Pb in more bioavailable chemical forms, e.g., lead oxide dusts from smelter emission lead fallout from air, etc. The 48%

for the primary smelter case is virtually the same as bioavailability of fully soluble simple lead salts, e.g., lead acetate, which are considered to be 50% bioavailable. That is, the most one ever encounters as an uptake fraction from the human gut, which is the case for very young children, is 50%, based on available data. The assumption of only 30% absolute lead bioavailability for secondary smelter and roadside soil scenarios are the model default selections.

p. 4-45, GSD for Population. I follow the logic of Dale Hattis' estimates in the cited paper about use of a higher GSD nationally for current lead exposures compared to the national 1.3 to 1.4 used in the 1986 lead criteria document, Chapter 11 (Table 11-9, p. 11-30). I don't quite follow the logic of why a smaller GSD than that given by Hattis for a national increase in GSD is used for the primary and secondary smelter scenarios when historically separate and cumulative fugitive dust emissions may behave as being uncoupled from current stack emissions.

The Midvale case is not the only Western smelter/milling site with Pb-B data. Bornschein and colleagues at the U. of Cincinnati have done a number of these as have other groups such as local and state health departments. Some sites have produced GSDs greater than 1.6.

p. 4-46, Sec. 4.3.4 I would point out the need to discuss a bit more the "NAAQS attainment" assumptions as part of the modeling for the primary smelter case study. The validity of the scenario is predicated on some high expectations that attainment is a good probability. However, nonattainment has typified monitoring results from this operation. The noncompliance is current, is significant as to level of exceedence, and is likely to get much worse if a 30-day averaging vs. quarter averaging time is selected.

p. 4-48, Table 4-6 footnote The footnote says that outdoor soil has less of an impact the further away one is from the facility. That may be generally true, but unremediated soils outside the maximum impact zone would still have accumulated soil leads and internal dust reservoirs that are under-represented by modeled soils in these more remote quadrants that don't account for the 100-plus years of contamination.

Secondly, the authors need to keep in mind that soil lead remediation around the Herculaneum smelter was triggered by soil lead levels at or above the action level. Unremediated soil lead areas farther away from the smelter do not indicate that they were unaffected or not elevated above background, merely that they were below the selected action level.

The calculation I indicated earlier using EPA's own equations and data showed a soil lead accumulation rate of 3 ppm/year for each 1 $\mu g/m^3$ air Pb. That would say that an average soil Pb at more remote quadrants from the smelter that would have, for example, only 1 $\mu g/m^3$ would be 300-plus ppm for 100-plus years of lead emissions. The median projected soil Pb for the primary smelter in **Table 4-5** is 84 ppm.

p. 4-59, 4.4.1.2, 4.4.1.3 There is no basis for running the projections with the cut point, for reasons noted earlier. Please also run without the cut point or, preferably, don't use an arbitrary cut point that appears at odds with the CAA, Sec. 109 requirement for an "adequate margin of safety."

p. 4-62, Table 4-13. It is not convincing that a group with a 99th percentile Pb-B of 6.8 (Leggett, lifetime average) would show no impact at all on IQ (< 1 point) at the 99th percentile in view of the dose-neurotoxic response statistics presented in Lanphear et al. 2005.

p. 4-78, top half. This is a typical example of the authors flagging or highlighting variability or uncertainty that overestimates the risk results, but not the many elements of underestimating.

III. RESPONSES TO CHARGE QUESTIONS FOR CHAPTER 4

1. To what extent are the various aspects of the exposure analysis technically sound, appropriately balanced, and clearly communicated?

This overall effort, for reasons given in the above general and specific comments, has technically sound components and some problems. There is a mixed picture for balance. Clear communication is a challenge. See my general comments.

2. Are the methods used to conduct the pilot exposure analysis...pathway apportionment...versus background technically sound?

Overall, the exposure analysis in the pilot draft is sound only to the extent that the biokinetic models used are crunching on sound environmental Pb inputs to produce sound results. The biokinetic construction of the models is generally O.K.

3. What are the Panel's views on the staff interpretation of performance review...for the pilot analysis...with regard to representativeness of the individual modeling steps?

See my general and specific comments. Macroscopic reality checks and knowledge of the measured local and the measured global literature for these case study sites are needed.

4. Are the Pb-B metrics used in the pilot analysis appropriate...?

Yes.

5. Are the methods used to conduct the pilot health risk assessment...technically sound? Does the Panel have any suggestions for improvement?

The health risk dose-response functions for use later in the draft are generally sound. The use of a purely arbitrary cut point is not valid. See my general and specific comments. A recommended improvement is dispensing with the notion of using arbitrary cut points.

6. To what extent does the sensitivity analysis...identify key areas of uncertainty in this pilot analysis?

See my general and specific comments.

7. *As part of the NAAQS review...there is interest in ...differentiating...historical and deposited...newly deposited...air lead? Does the Panel have ...recommendations...that might be employed...?*

See my general and specific comments above. First, it is critical to keep in mind that "new" lead inputs to soils and dusts, the latter being the main exposure drivers, are continuous so long as air Pb input is continuous. "New" Pb input simply becomes a rate by which one will get to historical accumulations over the long term. The short term is not the ultimate question, as I made clear earlier, referring to modeled data in **Tables 4-4 and 4-5.** Secondly, it is equally or even more important to recognize that all sources of lead being ingested by children, i.e., the ingested sum of historical soil and dust lead plus newly produced dust and soil lead all contribute to absorbed lead. Toxicological dose-response relationships do not biologically differentiate between the two temporally different histories for the ingested lead.

Third, it is not necessary that newly emitted lead, inhaled directly or as lead newly entering as dust and soil lead for ingestion, is required to produce all the dose (exposure) for harmful effects. A minor amount of new lead can add enough to a large historical fraction of lead to push the dose metric into the next category of toxic risk. I prepared dust lead loading tables entered into the review record for the AQCD review early last year showing that dust lead on a hard surface rises to a hazardous level with an interior air lead level of 0.1 $\mu g/m^3$.

The seemingly endless dissection of components of this draft staff paper such as fractional or "policy-relevant" lead sources and fractional exposure contributions into regulatory salami slices strikes me as a somewhat statutorily Balkanized exercise with no apparent redeeming feature, toxicologically or epidemiologically.

There is little scientific evidence to force disaggregation for some ranking purpose of what toxicologically is a continuum of exposure producing significant risk down to very low exposures. The impact of even modest new air Pb entering the dose-response continuum to produce some risk impact argues for both air Pb as a criteria pollutant for regulation via the Pb NAAQS mechanism and revising the NAAQS to a much lower level than this current and indefensible 1.5 $\mu g/m^3$.

8. What are the Panel's views on the most important issues to be addressed...in the full-scale...assessment that will be presented in the revised documents?

One significant issue is that of a clear presentation of the uses and limits of the methodology and results for the three selected sites. A second issue is to establish the overall validity of the collective approaches used in this pilot draft. Unless this pilot approach can be shown to be scientifically sound, there is little argument for doing much more of the same with more case studies. Thirdly, there is the issue of how the current approach accurately addresses the relative importance of "new" versus "historical" lead inputs and what this means over time.

COMMENT TABLE 1. Measured and Modeled Air Lead Levels ($\mu g/m^3$)

Pb-Air Metric	Measured Air Pb [a]	Primary Smelter Dispersion Modeling [b] Current Condition	Secondary Smelter Dispersion Modeling [b]	Ratio (rounding) [c] of Measured to Modeled Data
5th percentile	0.019	0.008	0.001	19/8
25th percentile	0.081	—	—	—
Median	0.252	0.022	0.005	50/4
Mean	0.484	—	—	—
75th percentile	0.695	—	—	—
95th percentile	1.923	0.662	0.018	107/3

a = Air Pb distributions from data for 59 air Pb monitoring sites, p. 2-31.

b = Table 4-4, draft staff paper.

c = Ratio measured to secondary smelter case/ ratio measured to primary smelter case.

PRE-MEETING COMMENTS:

LEAD HUMAN EXPOSURE AND HEALTH RISK ASSESSMENTS AND ECOLOGICAL RISK ASSESSMENT FOR SELECTED AREAS

PILOT PHASE

EXTERNAL REVIEW DRAFT

Reviewer: Paul Mushak, Ph.D.

I comment on the human impact chapters and leave the welfare and ecotoxicity issues in Chapter 7 to others. Many general comments would be those already stated as to key points in the draft staff paper and are not repeated here. However, I have some additional ones. I mainly have specific comments across the Chapters.

I. GENERAL COMMENTS

The overall clarity of the draft is good.

The pilot draft seems to lag in its information relative to the staff paper draft. For example, discussions on determination of environmental lead in relevant media seem somewhat disconnected in the pilot document from the parallel section in the staff paper.

Chapter 2 (draft Sec. 2.1) does not spell out in any detail why the particular case study sites were chosen. It particularly does not rank the criteria for selection of the secondary smelter and roadway dust case studies. The primary smelter selection, of necessity, was the only one operating.

For the secondary smelter case study, it appears that the more important criterion was availability of more recent air Pb monitoring data and not neighboring population numbers. Both Herculaneum, MO, and Troy, AL are very small communities. I assume a second criterion ranking higher than impacted population size was the presence of the one principal (e.g., smelter) point source. This would steer toward more remote sites with few other lead point sources. There are about 15 secondary smelters and potentially endless stretches of roadways with reentrained dusts with Pb (from former use of leaded gasoline) as the point source.

I would add to this list, for purposes of performance evaluation, the availability of exposure data indexed as blood lead (Pb-B). These include the many reported Pb-B testings at Herculaneum done by ATSDR in 2001 and 2002 and earlier efforts, and the 2003 CDC Pb-B survey done for Pike County, AL, the site of the secondary smelter. For the latter, the authors need to do some spatial stratification of elevated Pb-B frequency versus distance from the secondary smelter.

As I also noted in comments for the draft staff paper, the overall driver for the results from these case study analyses are the environmental lead levels, in terms of the relative levels of variability and uncertainty. The exposure modeling uses reasonably understood biokinetic models and the dose-response relationships for modeling health risk, especially for IQ loss, are also well understood.

II. SPECIFIC COMMENTS

Chapter 2, Overview of Risk Assessment

p. 2-10, Table 2-4. The information for the primary and secondary smelter environmental media determinations is a bit confusing. For soil Pb, the secondary smelter case study used both modeled and "hybrid" data, the latter entailing use of some measured soil Pb from another site, Corpus Christi, TX. Here, the entry noted "modeled" soil Pb based on air deposition. Similarly, the indoor dust Pb at the secondary smelter site is referred to as being "estimated" from ambient air levels. My understanding is that the secondary smelter indoor dusts were modeled from modeled air Pb via the three stack tests.

The Table notes that indoor air levels were based on ambient air and soil levels. But the authors did use interior dust values from 17 homes as part of all the calculations, correct?

p. 2-11, 2nd par. The soils at the remediation zone in Herculaneum will continue to rapidly accumulate Pb from recontamination. See my comments for the draft Staff Paper.

p. 2-12, Secondary Pb Smelter, top The text indicates only modeled soil and dust PB. However, the actual soil lead values in the model arose from both "modeled" and "hybrid" approaches. Footnote 3 notes that the regression-based Pb-B model of Lanphear et al., 1988, was only used in the sensitivity analysis.

Chapter 3, Case Study Descriptions

Chapter 3 deals with the measured data sets available for environmental lead for the three selected sites. These data sets were used to characterize what has been measured over the years for lead in various media of interest to the documents: air, soil, dust, etc. These data sets were not the principal means for determining current condition lead levels in the media. The exercises used in the drafts to get these current levels entailed modeling, beginning with modeled air Pb using dispersion models crunching on stack emissions plus some level of fugitive dust burdens. Measurement data mainly appeared to serve for checking modeling performance.

p. 3.1, Sec. 3.1 Intro This par. should be clarified to show that both historical media lead accumulations and remediation soil lead were elevated and the rate of elevation of Pb in low-lead, post-remediation soils showed a rapid rate of recontamination. Appendix A contains the various stages of soil Pb sampling: pre-remediation, post-remediation (soon after clean-up) and recontamination.

pp. 3-4, 3-5 The various rounds of Pb-B testings are plagued by factors that tend to underestimate exposures indexed as Pb-B elevations and exceedence frequencies and both the authors and the readers should keep that in mind. First, the ATDSR samplings were not structured surveys of statistically valid design that would better reflect true distributions and exceedence frequencies. They involved voluntary efforts by mainly the MO health department in which children showed up for testing in each of two years: 2001 and 2002. Some showed up for both years.

The Pb-B levels rise to a peak in young children during the Summer, being at maximum around August. Collection of Pb-B samples throughout the year obscures the seasonal impact on the level of Pb-Bs, in that Pb-B testing in off-peak, colder months would yield lower results versus a structured survey done in, say, August.

The child data for 2001 and 2002 are only broken out for ages <72 months. However, within that larger risk band, there is a highest risk band for infants and toddlers. One would expect that prevalences of Pb-Bs 10 μg/dl for tested children 12-30/36 months would be higher. This relative increase was in fact seen and is noted in the July 13, 2001 ATSDR Health consultation for the Herculaneum area (see below). Other sites show this child age stratification, e.g., the Human Health Risk Assessment (HHRA, 2001) done for the lower Coeur d' Alene

River Basin in Idaho by Idaho and EPA Region X. The ID HHRA showed significantly higher Pb-B exceedences, within the overall young child age band, for children 24 months or less.

Other factors may or would reduce exposures indexed as Pb-B. The distribution of people showing up for testing in the 2002 year as a function of distance is not noted. We don't know if a lighter or heavier weighting is occurring by who shows up vs. where they live. That is, the participant selection bias is unknown but potentially troublesome. If the relative testing of older children entails more than younger children within the 71-month age band, the exposures for infants and toddlers are under-represented. Secondly, the smelter owners bought a number of residences closest to the smelter around the 2002 time frame. Where renting of these houses was permitted, the owners barred families with children. So, the newest data set versus older sets would have a reduced sampling population for the high risk zone.

The 2000 Herculaneum Pb-B Study showed there still was a major blood lead elevation problem for 0-6 y. preschool children in Herculaneum, especially for children closest to the smelter. It is not clear that the data were gathered in a way that they could be compared to past years in terms of past blood lead results. Each ad-hoc screening appears to have been done differently, which frustrates even simple or crude comparisons. We do not actually know how real or rapid the blood lead decline rate has been.

The August 2000 Pb-B survey results are tabulated in the ATSDR Health Consultation dated July 13, 2001. Table 7 shows the age dependence of Pb-B elevations for the Herculaneum community and Pb-B statistics for children when tested in August. For children 1 to 3 years of age, the highest risk subgroup, 4 of 13 children or 31%, had a Pb-B 10-20 µg/dl and 1 child of the 4 had a Pb-B >20 µg/dl. Earlier testings 1/95 to 6/99, recorded in Table 6 of this report also showed high frequency of elevated Pb-B. Table 6 notes, for 100 children 5 to 21 months old, 43% had Pb-B 10 µg/dl.

p. 3-6, Table 3-5 and Appendices A-6, A-7 Residential soil recontamination rates are significant for the residences tested, showing recontamination from 2002, when the MO DNR released their own recontamination figures, to 2006 (see the relevant citation in my comments on the draft paper. Table A-7, Appendices, indicates that many of the 31 remediated soil locations showed a 2 to 4-fold increase in lead 2002 to 2006.

p. 3-8, Pb deposition into soil boxes Soil lead in 10 boxes placed around the site indicated that over the course of one year, 2003 to 2004, mean Pb content doubled, from 49 to 97 ppm Pb, an increase approaching 50 ppm. At this rate, the EPA child Pb hazard standard of 400 ppm for contact soils would be achieved in about 8-9 years. It is not clear whether the ten soil test boxes were placed on any of the 31 properties also selected for testing total yard surface contamination. It would have made sense to do so.

Ibid, Indoor Dust The problem, as recognized in the draft, is that one has ongoing and variable dusting of residential interiors. Unless one is using undisturbed household surfaces or, better yet, dust plate deposition methods, the variable cleaning factor is difficult to separate from dust fall rate variability. Lead loadings, in any case, are less sensitive to housekeeping than dust

concentration, and this characteristic was first tested by Milar and Mushak, 1982, who presented lead loading methodology versus use of lead concentrations.

Ref:

Milar CR, Mushak P. 1982. Lead-contaminated house dust: Hazard, management, and decontamination. In: Proceedings of the Conference on Management of Increased Lead Absorption in Children: Management, Clinical, and Environmental Aspects. JJ Chisolm Jr, DM O'Hara, Eds. Baltimore, MD: Urban and Schwarzenberg, pp. 143-152.

p. 3-14, Sec. 3.2.3 It is not quite true that there were no Pb-B data around the secondary smelter. There was a 2003 CDC survey for the county containing the smelter. It would have been inconceivable that the Pb-B sampling would have ignored areas close to the secondary smelter. As I noted elsewhere, the authors need to break out the results via various means. Presumably, the AL health department has these data. I would have to assume that CDC did a Pb-B testing in Pike County in the first place because of the secondary smelter. The Pb-B elevation rate was over three times the AL rate, and was also likely due to the smelter.

Chapter 4, Estimates of Media Concentrations

p. 4-2, last par. Are these six census blocks mentioned those corresponding to blocks of properties purchased over the years by the smelter owner?

p. 4-5, Sec. 4.1.2.3 What exactly is the point of doing an "attainment" scenario for a case study involving an operating primary smelter which has rarely been in attainment. It's a hypothetical case. This strikes me as the triumph of EPA's modeling hopes over its regulatory experience with this particular site. Would it not be more realistic and protective of public health to assume some level of non-attainment in terms of air Pb and go from there?

p. 4-9, Table 4-4 This is a significant Table, in that it says that once one goes beyond 0.5 miles from the main stack using Region 7 HiVols data, for a total modeling reach of 10 km (6.3 mi.), the dispersion modeling markedly underestimates air Pb, up to 5.4 times, monitored vs. modeled. Similarly, for "AirData" results, distances beyond 1.7 mi., for a total modeling reach of 6.3 mi, the modeled air lead values underestimate the measurement data, as much as 7-fold lower.

p. 4-21, Sec. 4.2.1 More to the point, the 2003 monitoring data showing the exceedence of the current NAAQS (1.9 units) indicate that the modeling of air Pb for the secondary smelter site would be underestimating actual air Pb.

p. 4-27, Sec. 4.2.2.4 The underestimation of the secondary monitoring results when using modeled results is rationalized based on differences in actual site meteorology versus meteorology used. This is puzzling, since the significant differences in the primary smelter case did not encounter meteorological disparities.

p. 4-30, Sec. 4.2.3.1 The authors note that using just modeled soil levels, the results were three-fold lower than a comparison secondary smelter site, studied by Kimbrough and Suffet in a 1995 report. The smelter was in an urban area. Were other media levels measured as well?

Chapter 5. Human Exposure Assessment and Blood Lead Estimations.

p. 5-2, 1st full par. I have looked through both drafts (staff paper and pilot) and don't see actual counts of children by census block groups or any other Census Bureau metric. However, the authors have these numbers, since they say so on p. 5-2, and used such numbers in generating central tendency estimates from the models. Is there a place in one of these chapters to lay out child counts at least by distance from the smelters? Some of the other, earlier data sources in the case of Herculaneum do break out total children's representation in the town population by distance from the smelters. Counts of children are also useful for matching up areas of underestimation and overestimation of modeled data with the child count and the fraction of total counts impacted.

p. 5-9 to 5-11, 5.1.3.3 I am astonished that the significant changes in the construction of the original Leggett (Pounds Leggett) core (Fortran) construction, done for the specific needs of the pilot analyses, were done without any input from the SAB's All Ages Lead Model review Panel, who reviewed the various models and their usefulness for EPA's regulatory use. I do not see, in Chapter 8: References, any reference to the various reports from the Panel to EPA pointing out the usefulness of Leggett and other biokinetic models for modeling of risk. What validating steps were taken for these changes?

p. 5-21 to 5-22, Table 5-11 See also my comments for the draft Staff Paper. Since submission of my comments on the draft Staff Paper for questions of bioavailability for IEUBK model use, I have been in touch with Dr. John Drexler, University of Colorado-Boulder, who did the in-vitro, bioaccessibility testings for Herculaneum environmental samples. The in-vitro bioaccessibility testings were done in collaboration with Drs. Casteel and Brattin, who did the young swine in-vivo studies for Herculaneum site samples. I have not yet contacted the latter investigators, but Dr. Drexler's results point to their results as well.

First, the dust Pb and soil Pb values as indicated —0.48 soil, 0.26 dust— are correct for the indicated media and these fractions are absolute bioavailability figures and not relative bioavailability. The term "accessibility" (or "bioaccessibility") should be dropped in the present context, because it has a meaning different from bioavailability, the actual uptake rate of lead from the GI tract.

The anomalous uptake fractions for dust versus soil are rationalized from the Drexler speciation data, showing more oxidized (and more bioavailable) forms of the starting galenic (PbS) lead form in soils, but higher fractions of persisting galenic Pb dusts in the interiors of the tested residences. However, bioaccessibility in the Drexler in-vitro testing as indicated shows the small galena particles in these analyses translated to absolute bioavailabilities of .29 for dusts and .41 for residential soils, not materially different from the in-vivo young swine results (0.26 and 0.48). The dust bioavailability for galena at 26% is not materially different from the default value of 0.3 (30%). I would take these findings to mean that the dusts in the tested homes arose

from high-lead-concentrate fugitive dusts in the general proximity of the haulage roads and from direct deposition from atmospheric emissions from the facility.

The roadside dusts tested for lead levels as high as 300,000 ppm in 2001 testings (see my Staff Paper comments).The Drexler report notes roadside soils in that testing had Pb levels ranging from 16,085 to 132,218 ppm. The dust and soil Pb speciation results rule out lead paint.

Dr. Drexler, in his Final Report, indicated chemical speciation results showed interior dusts largely arose from atmospheric lead directly rather than via soil and subsequent tracking into the residences. The Report's findings of quite similar air Pb vs. interior dust Pb speciation profiles is consistent with this. The report notes "...also suggest that the pathway for household dust is dominated by recent air infiltration [into the residences] and not by foot-traffic from residential yards."

The bulk Pb content for the 10 residential dust samples ranged from 1272 to 24,651 ppm. These bulk values, to the extent they are derived from recent air Pb infiltration into the residential interiors, indicate rapid dust lead loadings and the Drexler report is consistent with my earlier calculations that atmospheric air lead entering residences can rapidly lead to hazardous depositional Pb loadings onto interior surfaces accessible to young children.

Ref:

Drexler JW. 2005. Final Report. The Speciation and Bioaccessibility of Anomalous Lead Concentrations in Soils from the Herculaneum Community-Herculaneum, Missouri. Boulder, CO: Laboratory for Environmental and Geological Studies. University of Colorado. Done for: U.S. Environmental Protection Agency Region VII, via Black and Veatch .

Related Refs:

Mushak P. 1991. Gastrointestinal absorption of lead in children and adults: Overview of biological and biophysico-chemical aspects. Chemical Speciation and Bioavailability 3: 87-104.

Drexler J, Mushak P. 1994. Health risks from extractive industry wastes: An approach to bioavailability of toxic metal and metalloidal contaminants. Seattle, WA, 1994 Annual Meeting of the Geological Society of America, October 24-27.

Drexler J, Mushak P. 1995. Health risks from extractive industry wastes: Characterization of heavy metal contaminants and quantification of their bioavailability and bioaccessibility. Paris, France, Third International Conference on the Biogeochemistry of Trace Elements, May 15-19.

Mushak P. 1998. Uses and limits of empirical data in measuring and modeling human lead exposure. Environ. Health Perspect.106 (Suppl 6) 1467-1484.

p. 5-22, 5-23 Sec. 5.1.4.4, Table 5-12 I provided chapter and verse in discussions for the draft Staff Paper regarding the importance of having the right GSD and the existence of various GSDs empirically generated from measured Pb-Bs.

p. 5-25, Sec. 5.2, Table 5-12. Again, one cannot use national NHANES data to compare with local geographical areas regarding Pb-B statistics. See the discussion in my comments on the draft Staff Paper.

Chapter 6: Risk Assessment

See my comments for this portion of the draft Staff Paper.

SUMMARY COMMENTS: APPROACHES TO DEVELOPMENT OF CASAC PANEL-DERIVED LEAD NAAQS NUMERICAL VALUES

Paul Mushak, Ph.D.
March 1, 2007

Below are bullet comments on preliminary derivations of numerical primary NAAQS values by the CASAC Pb Panel.

- The lead NAAQS CASAC Panel is presently evaluating EPA's OAQPS staff paper and its supporting documentation for derivations of a lead primary NAAQS. CASAC Panels on criteria pollutants are mandated by statute and qualified by scientific expertise to review any proposed EPA NAAQS and its scientific basis. It follows that this Panel is qualified to independently present a suggested range of NAAQS values for consideration by the EPA Administrator and information on other elements of a Pb NAAQS.

- The Panel has had a good opportunity to assimilate OAQPS' current draft approaches to Pb NAAQS options and to put them in interpretive perspective.

- There are concerns that the methodological approaches presented in the draft OAQPS documents for eventual generation of NAAQS options may not fully reveal national childhood lead exposures.

- Given present advisory concerns and process uncertainties, it is appropriate for the Panel to offer input on ranges and options for the Pb NAAQS at this time based upon CASAC's statutory mandate and scientific expertise

1. Status of the Current U.S. Pb NAAQS for Adequate Public Health Protection.

- Preservation of the current NAAQS for Pb of 1.5 μg/dl is indefensible in terms of the large amount of new health risk data for lead in the environment.

- Any allowed increases in the current low air Pb levels to near or at the current Pb NAAQS for the U.S. would be catastrophic to public health in general and children's health in particular. It would also be devastating to the ecological environment.

- Preservation of the current, obsolete air Pb NAAQS only because air Pb levels happen to be low at this time is logically as well as scientifically indefensible.

- Regulatory controls for lead or any other health hazards do not come or go based on single temporal snapshots but rather on the enduring need for the controls for protection of public and ecological health -- in this case, on the persistence of robust dose-toxic response relationships for lead. New data from credible sources invariably show lead is more toxic than was assumed and virtually never that lead is less toxic than was assumed.

- Anyone familiar with the checkered history of lead's entry into commercial channels and human environments in the 20th Century or before would likely not reject the future possibility of some rapidly introduced technology or industrial activity producing elevated air Pb levels.

2. Preliminary Approaches to Development of a Panel-derived Numerical Value for a Pb NAAQS

Three potential approaches have been considered by the Numerical NAAQS Value subgroup for Panel-recommended NAAQS air Pb concentrations. One or more of these can be hybrids. The various approaches are not mutually exclusive, but rather are complementary.

A. The Modified Empirical-Deterministic/Slope Factor Approach

The Derived NAAQS Values

Based on the current health risk literature, this approach produces two sets of NAAQS values based on two not-to-exceed child Pb-B values at the 99.5 percentile:

Not-to-exceed child Pb-B of 5.0 µg/dl: 0.22, 0.11, 0.06 µg Pb/m^3 NAAQS values corresponding to slope factors of 5, 10, and 20, respectively.

Not-to-exceed child Pb-B of 2.5 µg/dl: 0.11, 0.06, 0.03 µg Pb/m^3 NAAQS values corresponding to slope factors of 5, 10, and 20, respectively.

These two ranges are between seven (rounding) and 50 (rounding) times lower than the current NAAQS numerical value of 1.5 µg/m^3.

These suggested air lead values using Method A might be viewed as protective in terms of the indicated reductions below the current NAAQS. However, the use of Approach C would indicate that some of these estimated air lead values may not be fully protective in preventing IQ losses. For example, Table 2 in my Approach C indicates that the amounts of IQ point loss for 0.11 and

0.22 air lead values are too high. Table 2 shows IQ point losses of 6 and 12 (rounding) for air lead values of 0.100 and 0.200 μg/m^3.

Details for Derivation Rationale and Methods

- This approach is probably the most direct and simplified of the preliminary options that have been considered. It is, however, one that arguably engenders no more overall uncertainty for a NAAQS than the many and compounded uncertainties attending the macro multi-modeling approach presented by OAQPS in its draft documents for three case studies. Each of the case studies has multiple uncertainties that collectively pose added uncertainties in their translation to a NAAQS.

- This approach also allows accommodation of the contributions of air Pb to dusts and soils, a critical aspect in NAAQS derivations, by use of appropriate blood lead/air lead slope factors that derive from both direct and indirect pathways (air Pb deposited to dusts and soils).

- The conceptual premise of this approach is rooted in the valid use of a not-to-exceed blood lead value for any child in the upper tails of a dose-response distribution arising from the dose-risk response literature. The validity of this concept was essentially confirmed in the January 3, 1990 CASAC report to the EPA Administrator in its review of the 1986 and 1990 Pb AQCD and staff paper (p. 1, par. 2):

    - "...it is the consensus of CASAC that blood levels above 10 μg/dl clearly warrant avoidance, especially for development of adverse health effects in sensitive populations. *The value of 10 μg/dl refers to the maximum blood-lead level permissible for all members of these sensitive groups, and not mean or median values*" [italics added].

- This was the approach for both (1) the 1978 NAAQS promulgation and its survival after court challenges from regulated stakeholders, and (2) the WHO 1987 and 2000 guidelines, which were the basis for ministerial directives (standards) within the European Union and other international jurisdictions.

- The 1978 EPA NAAQS and the more recent WHO 2000 derivations are methodologically the same, differing only in the choice of input parameters. They began by setting, in the case of the 1978 EPA NAAQS, a not-to-be exceeded Pb-B as the then-current CDC guidance level of 30 μg/dl for 99.5% of U.S. young children, and, in WHO 2000, a not-to-exceed level of 10 μg/dl in 98% of young children.

- This produced a mean Pb-B value of 15 in the 1978 EPA case, and 5.4 in the WHO case. In the EPA NAAQS derivation, 12 μg/dl of the total came from diet and water. In the WHO 2000 approach, 3 μg/dl came from all non-air contributions. Non-air components of 12 μg/dl for the 1978 EPA standard and 3 μg/dl for the WHO 2000 case left 3 μg/dl for EPA's 1978 figure and 2.4 for the WHO derivation process. These non-air selections were based on non-air Pb contributions at those times.

- A Pb-B/ air Pb slope factor was then used, a factor of 2 for the EPA 1978 NAAQS and a factor of 5 for the WHO 2000 case. The latter took into account a more valid impact of air Pb depositing as dusts or onto soils than did the EPA 1978 selection. This produced the 1978 NAAQS of 1.5 μg/m^3 and the 2000 WHO value of 0.5 μg/m^3, respectively.

- For the present approach, we suggest use of the starting not-to-exceed values of 5.0 and 2.5 μg/dl for avoidance of risk of IQ loss at the 99.5 percentile, the percentile used by EPA 1978. The scientific logic of this selection as to risk is that the values are within the bounds of science-based protection of public and ecological health. While a percentile within the upper tail of a risk population distribution might be viewed as a policy parameter, there is no scientific basis for saying protection should be any lower in terms of that population fraction being protected.

- There are five supporting elements to the two choices as a bounding range for any child. They arise from (1) avoiding any significant IQ loss from Pb exposure in children; (2) the values represent the mid and lower portion of the linear segment, i.e., the sub-10 μg/dl segment, of the dose-response relationships in the Lanphear et al. (2005) report for IQ loss in Pb-exposed children; (3) the values are those for the sub-7.5 μg/dl linear segment having the steepest dose-response curve; (4) are one-fourth to one half the value the CASAC recommended in 1990 as the maximum to occur in any child; and (5) are within the range indicated in various current consensus treatises as a sub-10 μg/dl value of concern (e.g., CDC, 2005).

- For keeping 99.5% of U.S. young children below 5.0 and 2.5 μg/dl, respectively, mean blood Pb values of 2.50 and 1.25 μg/dl are required. These mean values reflect air and non-air Pb. Adjustment for the air portion working through direct and indirect pathways (soil, dust deposition) is needed.

- Modifications for any current use of this approach must recognize that the non-Air Pb levels contributing to current children's Pb-Bs are much lower than were the cases considered in 1978 and 2000. For example, the daily diet and tap water Pb intake estimates for the early 1970s used for the EPA 1978 NAAQS non-air Pb estimation were on the order of 200 μg Pb/day, producing 12 μg/dl, i.e., 6 μg/dl Pb-B/100 μg Pb intake. OAQPS estimates of current diet Pb intakes by children are only about 2-3 μg Pb/day, which are two orders of magnitude lower.

- To provide the estimate for the background, non-air (no direct or indirect, i.e., dust soil) Pb levels from air Pb in the above 2.50 and 1.25 μg/dl mean Pb-B selections, the IEUBK model for lifetime child exposures 0-72 mos. was used, without air lead, and with only typical geochemical non-air input levels for dust and soil, 50 ppm, and the dust default of 35 ppm, respectively (dust Pb = soil Pb x 0.70). The exposure and biokinetic inputs for this non-air Pb exposure modeling were those for non-air sources in Ch. 4, Table 4-3, in the OAQPS draft staff paper, with uptake factors of 0.3 for geochemical soil and dust lead.

- The modeling produces a background Pb-B of 1.4 μg/dl as a geometric mean, using all current non-air related and geochemical soil and dust inputs but no air Pb. For the mean of 2.5 μg/dl scenario, the non-air Pb-B for subtraction (1.4 μg/dl) produces an air-related contribution of 1.1 μg/dl. For the 1.25 mean Pb-B scenario, the modeling background was very close to the 1.25 μg/dl mean level. Modeling noise does not permit a non-air Pb calculation. Therefore, one-half the 2.5 mean Pb-B scenario was used.

- To convert adjusted Pb-B values to air Pb values, three different slope factors were used: 5, 10 and 20. The 5 slope factor gives 0.22 and 0.11 $\mu g/m^3$ respectively for the two risk scenario mean Pb-Bs. Use of the WHO 2000 Pb-B/ air Pb slope factor of 5, which was acceptable for that time and set of circumstances (the 1980s/1990s European picture) is currently insufficient to capture the growing contribution to Pb-B of dust and soil Pb generated from both present and past air Pb. Using a slope factor of 10 appears consistent with the discussion for Approach B presented by Panel member Joel Schwartz. A slope factor of 20 was selected to address need for an adequate "margin of safety." One cannot realistically approach what NAAQS value is protective without accounting for all of the dust and soil Pb present.

- Slope factors of 10 or 20 best reflect the mix of historical and ongoing input of air Pb. Using these slope factors, corresponding Pb NAAQS values of 0.11 and 0.06 $\mu g/m^3$ and 0.06 and 0.03 $\mu g/m^3$ for the 2.5 and 1.25 μg/dl mean scenarios are derived.

- The above computations and the IEUBK estimates of air-added and non-air "new" outputs indicate that very little additional or "new" Pb can be permitted in ambient air.

- Panel member Ian von Lindern has provided a more detailed version of this approach using different GSDs, non-air Pb backgrounds, etc. His comments should also be reviewed.

B. An Approach Using National Relationships Between Changes in National Child Pb-B Statistics and Changes in National Air Pb Values

The Derived NAAQS Value(s)

See the Approach described by Panel member Joel Schwartz.

Derivation

See the comments of Panel member Joel Schwartz for details of this suggested NAAQS value.

C. Modified Health Risk Estimation Approach

Derived Pb NAAQS Values

Relationships of a range of air Pb values to blood lead values are presented in Table 1. Relationships of a range of air Pb values to a range of IQ point losses are contained in Table 2.

Panel member Bruce Lanphear has also addressed dose-responses for air lead- IQ loss relationships. His comments should be reviewed.

Derivation

- This approach is a variation on the empirical slope-factor approach with some use of directly measured dose-response relationships for the risk relationships. Panel member Bruce Lanphear and I discussed this approach at some length. It was agreed I would provide one approach and he would look at providing something further.

- A set of Pb-B levels and their associated IQ point loss values can be generated directly from Lanphear et al. 2005 without inclusion or assumptions of thresholds. OAQPS' use of thresholds to these data means the current IQ loss risk tables in Chapter 4 of the OAQPS draft staff paper are of limited use. In fact, the biasing downward of the otherwise robust dose-IQ loss response is quite significant from the Ch. 4 Tables because of these threshold interpositions.

- My approach first generates a set of Pb-B values from direct and indirect air Pb inputs to Pb-B. These estimates are in Table 1. The air Pb and air Pb-generated Pb-B values are then connected to IQ point changes in Table 2. The IQ--Pb-B relationship chosen from Lanphear et al. 2005 is that for the sub-7.5 μg/dl linear segment of the dose-response curves. The sub-7.5 segment was chosen because it depicts the most robust relationship and the Pb-B range is more relevant to national Pb-B figures seen in current NHANES data.

- Table 2 estimates for IQ loss can be read bi-directionally. One can observe IQ point loss as Pb-B rises from a zero value. Conversely, one can observe estimates of avoided IQ point loss with reduction in air Pb-based Pb-B.

D. Caveats to the Approaches

In terms of the national childhood lead exposure picture embodied in NHANES IV data, one must consider relative impacts of lead sources on child Pb exposures.

- The subset of the U.S. child population impacted by paint lead exposures may not respond as quantitatively to protective NAAQS as those with air Pb-derived, non-paint sources. That has always been true. That subset would still require additional interventions. However, the need to implement lead exposure control X does not void the need to also implement lead exposure control Y.

- Subsets of U.S. children impacted by lead paint are simultaneously impacted, in many instances, by industrial and other air emissions in close proximity. These children would still benefit in aggregate exposure terms from the air emission control component, much as this subset of children also benefited from leaded gasoline removal.

- One needs to keep in mind that the relationship of leaded gasoline removal to falling Pb-B statistics through the 1990s was expressed across all of the demographic and socioeconomic strata used in NHANES survey compilations, including those strata linked to old, deteriorated urban housing with relatively more deteriorating Pb paint. Consequently, the Nation collectively received a huge preventive medical payoff across all strata from the phase-out of leaded gasoline in the 1980s and early 1990s.

- All subsets of U.S. children are impacted by interior and exterior dust lead. The interior dust values are summed across paint plus industrial inputs. It is atypical to have no air Pb input and only lead paint input to residential dusts encountered by many urban child subsets of the population. Relative contributions of the above to Pb-B are teased out by such methods as the form of multiple regression analysis known as structural equation modeling.

TABLE 1. Relationship of Pb-B to Pb-Air by Differing Slope Factors

Pb-Air ($\mu g/m^{3)}$	Pb-B (μg/dl)		
	S.F.* = 5	S.F.* = 10	S.F.* = 20
0.010	0.05	0.10	0.20
0.025	0.13	0.25	0.50
0.050	0.25	0.50	1.00
0.100	0.50	1.00	2.00
0.200	1.00	2.00	4.00

*S.F. = slope factor (m^3/dl) = Pb-B/Pb-Air; S.F. value varies with increasing impact of indirect Pb-Air pathway (Dust Pb + Soil Pb)

TABLE 2. Relationship of IQ Point Losses to Increases in Air Pb and Air Pb-Based Pb-B Above Zero [a,b,c]

Air Pb ($\mu g/m^3$)	S.F. = 5		S.F. = 10		S.F. = 20	
	Pb-B [d]	IQ Loss [e]	Pb-B	IQ Loss	Pb-B	IQ Loss
0	0	0	0	0	0	0
0.010	0.05	< 1	0.10	< 1	0.20	< 1
0.025	0.13	< 1	0.25	< 1	0.50	1.5
0.050	0.25	< 1	0.50	1.5	1.00	2.9
0.100	0.50	1.5	1.00	2.9	2.00	5.8
0.200	1.00	2.9	2.00	5.8	4.00	11.6

a Air Pb-related increases affecting IQ point loss through calculated Pb-B values using 3 slope factors per Table 1
b IQ vs. Pb-B dose-response relationship based on Lanphear et al., 2005: sub- 7.5µg/dl linear segment, slope - 2.9
c Slope factors as defined in Table 1 and text
d Pb-B as derived in Table 1
e Rounding values

Dr. Michael Newman

6. Policy Relevant Assessment of Welfare Effects

General

"This chapter presents information in support of the review of the secondary NAAQS for lead (Pb). Welfare effects addressed by the secondary NAAQS include, but are not limited to, effects on soils, water, crops, vegetation, man-made materials, animals, wildlife, weather, visibility and climate, damage to or deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being."

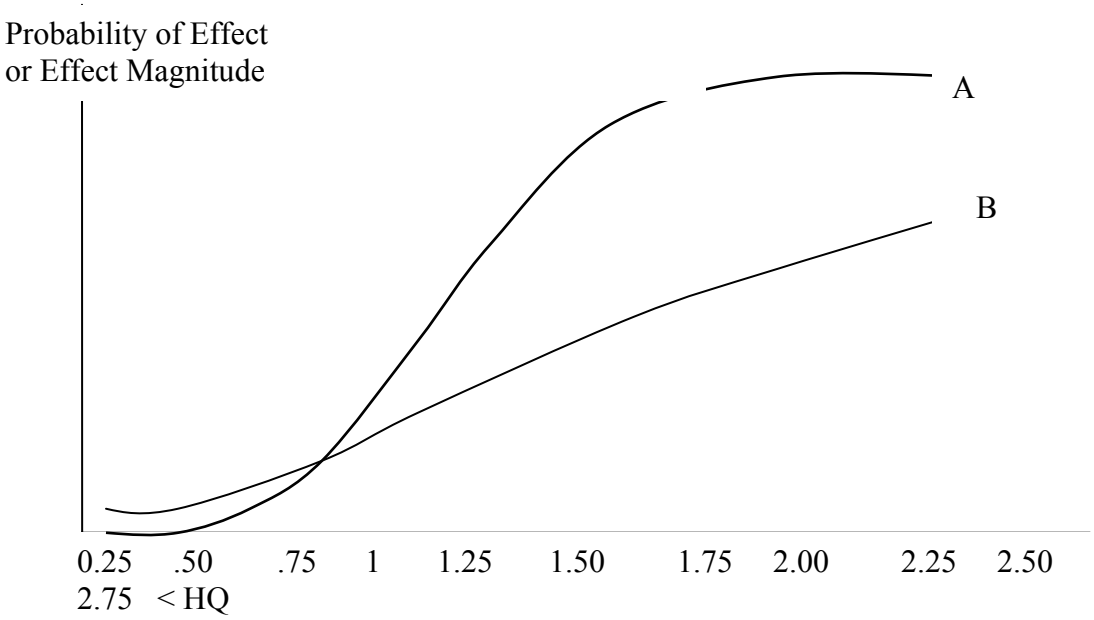
In my opinion, this section is well written and presents a sound approach. As stated in the associated Ecological Risk Assessment review, the points made below are intended to further enhance the value of the document, not to highlight significant shortcomings. Because the Ecological Risk Assessment (Section 7) details information also discussed in this section, the reader is referred to my review of that section for more comments.

Specific

1. In Section 6.2.5, Lines 2-5, Lines 25-30 and elsewhere. There does not appear to be any mention of exposure to dabbling ducks or swans from Pb shot or other birds using such solid Pb materials as grit.
2. Page 6-11, Lines 6-7. It is very helpful for the document to emphasize the importance of considering non-individual ecological entities. Unfortunately, the wording of this sentence is confused. Lead effects are stated to potentially harm organisms at the individual, population, community, and ecosystem level. What is meant is clear but ecosystems are not only organisms to be impacted. Effects can occur to interactions among species or to biogeochemical processes. I suggest a slight rewording of this important sentence.
3. Page 6-16, Lines 1-11. The purpose of the three cases studies is clear but that of the national-level screening assessment is not as clear. This was also the case in the Ecological Risk Assessment document. I suggest that the purpose or goal be stated more clearly or prominently. The text in Section 6.4.3.1 does not seem to do this (Page 6-20, Lines 21-27).
4. Table 6-3 on Page 6-27 and Table 6-4 on Page 6-28. The division of the arithmetic mean Pb concentration by the CCC (which is a predicted geometric mean) results in a biased HQ. The reasons are outlined in my review of the related Ecological Risk Assessment document and references are provided to reinforce this statement.
5. On Page 6-29 (Lines 3-5), the statement is made that "Five of the PEC-based HQs exceeded 1.0, indicating probable adverse effects. Three of these exceeded a PEC-based HQ of 10, indicating a very high probability of adverse effects, and possibly higher severity of effects than at the locations with lower HQ values." As much as I would tend to agree, the HQ values strictly indicate presence of potential danger/hazard, not the level of risk (probability of a specified adverse effect) or severity of the effect. (Elsewhere in this document, e.g., Page 6-35, Line 30, HQ-based inferences are also used imprecisely to judge risk.)

   An example can be used to show one situation (of many) in which this is true: risk is not reflected effectively with hazard quotients and a HQ only indicates the presence of a

potential danger or hazard. Assume that two toxicants have an HQ=1 but each has a different shaped concentration-effect curve. A specified multiple of HQ for A will not result in the same probability of effect or magnitude of effect as that same HQ multiple for B. Let both A and B have the same reference concentration (i.e., NOEC for A = NOEC for B = 4 ppm). They will have identical HQ values at different concentrations, i.e., as concentration increases, HQ values for both A and B will change by the same amount (see X-axis concentration and HG units); however, the probability of an effect or magnitude of the effect (Y-axis) could be different for the two toxicants at HQs > 1. The HQ is not a good measure of probability or magnitude of effect. It is a metric suggesting presence or absence of danger or hazard.



6. Page 6-36, Line 9. The authors may want to mention that the scenarios being considered were not for hydrological or seasonal conditions that could generate the highest dissolved Pb concentrations.
7. Page 6-36, Lines 10-15. What is the factual foundation for the judgment of "might easily be at least ten times higher than what is represented in the NAWQA database." What does "easily" mean in the context of these data? What is the basis for the estimate of "at least ten times higher?"
8. Page 6-36, Lines 24-26. The hardness adjusted concentration predictions are biased (see details in Ecological Risk Assessment review please) and this should be acknowledged.
9. Page 6-36, Line 28-29. May want to include a statement that hydrology- and season-linked variations were not considered directly although dissolved Pb concentrations often vary with season and hydraulic flow.

7. Ecological Risk Assessment

General

This section is well written and reasoned. It provides detail for gauging the potential hazard associated with each of the case studies. Therefore, the comments provided below are intended as suggestions to further enhance its obvious value.

This section begins as follows:

"The ecological risk assessment for this NAAQS review consisted of three case study screening assessments and a national-scale surface water and sediments screening assessment. The case study screening assessments were designed to estimate the potential for ecological risks associated with exposures to Pb emitted into ambient air for three case studies: primary Pb smelter, secondary Pb smelter, and near roadway non-urban location. The national scale screening assessment evaluated the potential for ecological risks associated with the atmospheric deposition of Pb released into ambient air at surface water and sediment monitoring locations across the United States."

Specific

10. The surface water and sediment screening involved culling of locations based on a series of criteria: minimum number of exceedances of chronic dissolved Pb screening values, dissolved Pb not sufficiently elevated relative to WQ-related levels, and presence of likely sources of Pb. As stated in 7-28 (lines 23-26), the selected sites "represent a small fraction if surface waters in the United States …" Consequently, it is important to define clearly what the population this sample of sites is thought to represent and how strong inferences are about that population based on this sample. Currently, the selection and results are clearly described but statements linking sample sites to population are not equally clear. Statements such as that on Page 7-28 (Lines 23-26) might be made more prominent or expanded.
11. The soil screening section also seems sound and appropriate.
12. Section 7.1.3.2 Water quality Criteria applies soon-to-be-released Pb AWQC that use a power model to adjust criteria for hardness, producing CCC or CMC values. Although the document does faithfully apply the Pb AWQC equations, it is an inconvenient truth that the equations are demonstrably biased. The downward bias becomes larger as the mean squared error increases for the regression technique/data set with which the model was generated. This fact has been documented in general by many authors (e.g., Koch and Smillie 1986, Miller 1984) and specifically for hardness corrections (i.e., Newman 1991). Minimally, this bias should be acknowledged as a source of uncertainty. Ideally, it should be adjusted for so that more accurate predictions result. Adjustment is easily done as described in the references above and other publications.

    An example of the confusion about what the hardness models predict is evident in Exhibit 7-10. The arithmetic mean dissolved Pb concentration is compared to the CCC. Strictly, the CCC derived from the model is the geometric mean, not the arithmetic mean.

Therefore, the HQ shown in the Exhibit 7-10 table and the other similar tables (Exhibit 7-13) is inherently inaccurate. (See references for detail.)

13. Page 7-13, Lines 19-20, page 7-14, Line 1, Page 7-17, Line 1, Page 7-23, Lines 12 and 16, Page 7-27, Line 19. The HQ is used to infer risk or even combined as the phrase, "risk HQs" here and in other places in the section. The Hazard Quotient (HQ) is a metric for hazard, not risk. Although the significance of the associated statements is clear, it would improve the presentation if the distinction was clearer in some places. As an example of some resulting confusion, Page 7-23, Lines 27 use HQ to infer probability of a specified type of effect, i.e., to infer risk. Minimally, the use of HQ to infer risk should be added to the list in "7.3. Limitations and Uncertainties" on page 7-25.
14. Perhaps, "Using a single value approach instead of a probabilistic risk assessment" could be added to "7.3 Limitations and Uncertainties" on page 7-25.
15. On Page 7-27 it is stated that "It is unlikely that there is significant avian and mammalian use of habitats within 2 meters of heavily traveled roads; therefore, HQs above 1 at locations as 2 meters might not be associated with ecological impacts beyond those associated with traffic on the road." Many species are drawn to edges (ecotones) so this statement could be questioned. Also some species, such as birds seeking pebbles/large sand grit from the roadside, raptors attracted to meadow rodents, or reptiles seeking the road's warmth at certain seasons/times of day, might be drawn to road edges. It would be better to remove the opinion that the Atlee, Virginia might be an overestimate.

References

Koch, R.W. and G.M. Smillie. 1986. Bias in hydrologic prediction using log-transformed regression models. Water Res. 22: 717-723.

Miller, D.M. 1984. Reducing transformation bias in curve fitting, Am. Stat. 38(2): 124-126.

Newman, M. C. 1991. A statistical bias in the derivation of hardness-dependent metals criteria. Environ. Toxicol. Chem. 10:1295-1297.

Mr. Rich Poirot

Review of the National Ambient Air Quality Standards for Lead: Policy Assessment of Scientific and Technical Information

OAQPS Staff Paper – First Draft

CASAC Pre-meeting Review Comments on Chapter 6, R. Poirot, January 30, 2007

Generally the authors of chapter 6 and the supporting ecological risk assessment have made a commendable first attempt to assess environmental risks from atmospheric lead pollution — considering that they have such limited quantitative information to work with. It is therefore disappointing to note up front that the Agency lacks (chooses not to expend) funds for any additional ecological risk assessment work for this review, especially considering that the 1978 decision to (simply and conveniently) set the secondary Pb standard equal to the primary standard was "due to a lack of relevant data at that time". Here we go again…

The information presented here is primarily focused on identifying the kinds of locations and environmental media where adverse effects from historical and continuing atmospheric lead emissions are most likely. These "screening" analyses do seem to indicate that there are adverse environmental effects from lead contamination in relatively limited areas near large point sources or along roadways with high historical accumulation where, in some cases, the contributions from current atmospheric Pb emissions are adding to the severity of the problem or, at least adding to the time for which injurious levels of Pb will persist in various environmental media (soils, sediments, surface waters). The chapter indicates that these areas most susceptible to adverse environmental effects could be subject to additional future analyses, although its not clear what the intended future analyses would entail or whether such analyses would support considering a revised secondary standard equal to or different from the current standard(s).

Several key questions that are not directly addressed in the current document include the extent to which current environmental effects would be remedied if current standards were attained everywhere, or whether there are environmental effects occurring in locations that currently meet the primary & secondary NAAQS. No information is provided to relate the current indicator (particulate Pb on TSP filters) to total (wet & dry) deposition and accumulation of airborne Pb to the environment, nor is there any discussion of other more environmentally relevant indicators — such as deposition or exceedance of critical loads. Much of the staff paper chapter is a repetition of results from the (fairly brief) risk assessment. There is no discussion of alternative indicators, averaging times, forms and levels of possible secondary (or primary) standards - which typically form the central organizing concepts for NAAQS "staff papers," and only minimal information is provided on the planned approach for the upcoming "policy assessment". This makes for an awkward and inefficient science review process.

It would be useful to more closely consider the ways and locations where rates of current atmospheric Pb deposition are greater than or less than the rates at which historically deposited Pb is removed from the "biologically active layers" of soils and sediments (or perhaps buried under newly deposited layers of cleaner material). In remote deciduous forest soils, for example,

its estimated that historically deposited Pb may be removed from the biologically active layer within 60 years. How does this removal rate slow or reverse as we move from remote areas to suburban or urban areas or to the vicinity of large point sources where lead concentrations in biologically active environmental media continue to accumulate.

1. To what extent is the presentation of evidence from the ecological studies assessed in the Pb AQCD and the integration of information from across the various ecologically-related research areas drawn from the Pb AQCD technically sound, appropriately balanced, and clearly communicated?

Given the relative absence of quantitative research data — especially for relating ecological effects from accumulated lead pollution to current airborne lead contributions, the presentation of "evidence" seems sufficiently sound, balanced and clearly communicated. I thought the discussion of "critical loads" and there potential applicability to Pb in the CD was especially useful, even though the concept did not seem to be quite ready to apply to Pb in the current review cycle. I think it would be useful to at least mention the concept in the staff paper and identify some of the research needed to make the technique more applicable for Pb NAAQS review.

2. Given the lack of quantitative information on Pb-related ecosystem effects, what are the Panel's views on the presentation of this topic in chapter 6?

I don't see how you could have done this much more effectively. An inherent difficulty is that in specific locations — such as in the vicinity of large mining or smelting sources — where adverse ecological effects are most evident, lead is typically only one of multiple metal, gaseous and/or acidic stressors, and its also often difficult to separate the contributions from current and historical airborne contaminants from past or present direct discharges to water or soil.

3. What are the Panel's views of the data sources and models used to estimate current levels of Pb in soil, freshwater, and sediment for the case study locations?

The data sources and models generally seem appropriate. For the secondary smelter case study, the logic of increasing the soil Pb by a factor of 3 to better reflect measured soil Pb in the vicinity of "a similar source" would benefit from additional discussion (in the risk assessment or SP). Can you at least offer some explanation of why your atmospheric model calculations might be way too low? For the soil and sediment data, an important detail is the depth to which or at which the samples are taken. I couldn't find any indication of the depth of the sediment samples. The soil lead at the Corpus Christi site shows large variation by depth, although it's not clear if a sample depth of 10 cm means "averaged over the top 10 cm" or "specifically at the 10 cm depth." I assume it must be the latter since Pb concentration for 10 cm is >40x concentration for 20 cm. At the Virginia site, it's not clear whether the indicated "sampling depths of 0 to 15 inches" (38 cm) represent the range of variability in the sampling or the total depth of sample. Neither seems right. The NAWQA water quality data are unfortunately very limited, although I don't know of alternate data that would be more useful, and also think you do a good job pointing out the limitations of these data in your discussions of uncertainties.

4. To what extent are the methods used to conduct the exposure assessment & the interpretation and presentation of the results technically sound, appropriately balanced, and clearly communicated?

As indicated above, some additional explanation for the discrepancy between modeled & measured soil lead for the secondary smelter seems warranted. The discussion of the national water quality screening methods is difficult to follow in the risk assessment. Is there a way you could provide a graphic plot of the equations used to calculate the CCC and CMC values to more clearly communicate the critical importance of water hardness as it affects lead bioavailability and toxicity?

5. What are the Panel's views of the approach for addressing uncertainty in apportionment of Pb contributions in the national-scale screen by factoring out those locations with known non-air sources (e.g. mining, point discharges)?

I think you point out the uncertainties associated with this approach reasonably clearly. Unfortunately, many of the large sources likes mining activities that have substantial non-air discharges also have substantial air discharges, and over time with changing climates, water-deposited contaminants or tailing piles can become important air emission sources. Furthermore these kinds of locations are likely to have very high accumulated levels of lead and other metals — to which any additional atmospheric deposition exacerbates an existing adverse condition. It's important to communicate that current airborne lead adds to the problem even though it may be difficult or impossible to quantify the incremental effect of current air emissions. We are least able to quantify air contributions in locations where we suspect total current and historical contributions are most injurious.

6. To what extent are the assessment, interpretation, and presentation of the results of the screening-level risk analysis, including characterization of lead concentrations in media and the comparisons to ecological screening values, as presented in Chapter 6 and the risk assessment report technically sound, appropriately balanced, and clearly communicated?

It's important to indicate that these are screening results for a limited number of terrestrial & aquatic species at a limited number of locations where relatively recent environmental lead data are available. As such, I think you make and justify reasonable adjustments to some of the most extreme eco-SSLs, develop reasonable estimates of lead concentrations in various media and clearly present the results with appropriate caveats. For the water quality criteria, I get the impression that the current CCC and CMC calculations may not work well for very soft water environments, and that these equations may be further modified within the year. It might be useful to explore this more closely. Conversely, I note that the hardness of the selected surface waters near the Herculaneum smelter is exceptionally high and wonder if there may be lower alkalinities at other relatively nearby locations – or in the vicinity of secondary smelters or roadways.

7. Does the Panel feel that adequate screening criteria (ecotoxicity screening values) were selected for each of the media?

I'm not aware of other options. The relatively small upward adjustments you made to the eco-SSLs for birds and mammals seems reasonable. Surely we expect some additional Pb burden to these terrestrial species from direct inhalation — just as there is for humans — especially at locations near sources where atmospheric lead continues to add to soil contamination levels.

8. What are the Panel's views on the derivation of the soil screening values for birds and mammals (i.e., using the Eco-SSL methodology)? Do the resultant values adequately reflect current information on exposure characteristics of these organisms?

I don't have sufficient expertise to comment — other than to indicate that you present reasonable arguments for these modifications.

9. To what extent are the uncertainties associated with the exposure analysis clearly and appropriately characterized in Chapter 6 and the risk assessment report?

(Unfortunately) I think your discussion of uncertainties is one of the stronger sections of the chapter. As always it would be useful where possible to point out approaches for reducing these uncertainties.

Minor Comments on other Chapters 1-5

P. 2-27 lines 16-20: It might be appropriate to indicate that absence of these road dust and soil emission factors likely results in underestimates of lead exposures in the vicinity of these sources.

P. 2-9, Table 2-3: After seeing several different source categories listed as the predominant current Pb emitters, its interesting to se yet another new #1 category (aviation fuel) here in the staff paper. Does this suggest a need to consider possible current effects on health or environment near airports?

P. 2-18, Figure 2-3: There looks like an indication of applying different emissions factors in the clear step-function change along the PA/MD border.

P. 2-21, line 3: Delete "a" or "es" from "approach(es)".

P. 2-21, lines 27-34 & page 2-22, lines 1-4: Might be worth mentioning that while TSP samplers do collect larger particle sizes than PM-10, they are consequently extremely sensitive to distance from source, height above ground, wind condition, etc., have relatively poor precision and limited spatial representativeness. Also the fiberglass filters used for TSP sampling are generally elementally dirty, subject to artifacts for acidic gasses, not amenable to surface beam analysis techniques, and poorly suited for analyses for anything but mass and Pb. Low-vol PM-10 samplers would capture somewhat less coarse Pb, but with much greater precision, and with much more useful application for other speciation analysis. They are increasingly being used for "air toxics" metals sampling and would provide needed information on coarse PM and its speciation. A Pb standard could always be tightened to account for the very coarse Pb missed by PM-10 heads.

P. 2-28, lines 14-16: Should be “decline…has” or “declines…have”.

P. 2-47, lines 25-26: Would like to see some additional discussion of “significant ‘under-monitoring’ near known Pb emission sources”. This certainly implies that the highest air exposures are not well-characterized at all – and with the Agency’s knowledge & approval.

P. 2-47, line 31: “15.” Should be “1.5.” Also this might be a good place to mention numbers of sites that exceed other lower thresholds – like 1.0 0.5, 0.25 — despite significant ‘under-monitoring’ near known Pb emission sources.

P 2-51, lines 10-20: It’s not entirely clear what these reported re-suspension rates (10^{-11} per second) refer to. Is this the fraction of total soil Pb (within a certain depth of what?) that’s re-suspended? Need some explanation.

P. 2-55: lines 11-13: Its not at all clear what “as compared to a mean of 109 ug/g…” refers to.

P. 4-11, lines13-21: Does this observation that measured concentrations are higher than modeled relatively near to a large airport indicate that aviation fuel emissions were not included or were underestimated? Also the later indication that air measurements were based on only 3 days in February 2001 doesn’t provide much confidence in the measurements (sounds like “significant under-monitoring near large Pb emission sources…”). I would imagine Pb emissions from roadside soils could be extremely episodic – high wind speeds, etc.

P. 4-27, lines 25-31: I’m not sure this adjustment of reducing only the above 1.5 concentrations to 1.5 ug/m3 is a reasonable way of evaluating benefits of attaining current standard. Emission reductions to achieve this would shift the whole distribution downward downwind of the source. Conversely, its sort of a silly paper exercise since the current (non-protective) standard has not been attained near this source since 1978, so setting a standard at any level does not appear to reduce ambient concentrations to any predictable level (even at a significantly under-monitored location). Possibly there would be some logic to setting a standard at lower levels since certain sources seem likely to chronically exceed standards by X %. Since there are not PSD increments for Pb, would there be some logic to including additional modeled impacts from new utility, smelting or mining sources up to allowable emission limits?

P. 4-31, Footnote 13: First sentence needs a verb or something. Maybe “…soil Pb levels for this case study (were) modeled”.

P. 4-47, Table 4-4: As indicated previously, just attaining current standard would obviously require reductions in emissions that will shift the whole distribution – not just the maximum-downward.

P. 4-51, Table4-7: This seems inconsistent with Table 4-4 (see previous comment) in that there are changes in blood Pb at all percentiles from the attainment scenario. Also how can blood Pb at some percentiles (for example 99^{th} & 50^{th} for IEUBK concurrent) increase with NAAQS attainment?

P. 4-54, Table 4-10, lower right box: Interesting that these very nearby monitors are not downwind of the facility. What was their purpose, background?

P. 5-2, lines 30-32: Not quite stated correctly. You could add something like "those at which effects" between "than" and "had" in line 31.

Dr. Michael Rabinowitz

Comments by Michael Rabinowitz January 2007

1st Draft Lead Staff Paper

Chapter 2: Characterization of Ambient Lead

Charge Questions:

1. *To what extent are the emissions and air quality characterizations and analyses clearly communicated, appropriately characterized, and relevant to the review of the primary and secondary Pb NAAQS?*

Generally, current ambient airborne lead has been presented with sufficient clarity and relevance to serve well. However, I would offer:

Lead is a multi-media pollutant. Airborne lead is only one source. Somewhere in the document, it needs to be stressed that lead exposure is from not only the air, but is a combination or summation that also includes the diet, drinking water, and any other sources in the household, neighborhood, or occupational environment. A small table, or pie chart, might be offered about the role of airborne lead, diet, water … for one or just a few examples (typical urban adult, child, exposed person…). A portion of the diet could necessarily be assigned to having been airborne. The reader could see airborne lead in the context of these other sources, and would see the futility of regulating only airborne lead without addressing these other sources.

If such a pie chart were compared to a chart from the time of the first Lead Criteria Document, the Agency could show real success in terms of the magnitude of lead exposure (the size of the pie) but also changes in the relative importance of the remaining sources. This topic is touched on in Chapter 5, but could be introduced more in this chapter and even expanded in Chapter 5. It is among the basic important aspects of lead exposure.

2. *Does the information in Chapter 2 provide a sufficient ambient Pb-related basis for the exposure, human health, and environmental effects, health risk assessment, and environmental assessment presented in the later chapters?*

Yes, I suppose it does. Fortunately, now we are in a situation where generally, nationwide, current air lead levels are much lower than most proposed standards, for example as offered in Chapter 5. Setting a standard at a level higher than current levels might result in increasing allowed emissions. Somehow we should guard against that.

Other Comments on Chapter 2

2-5 L7 Consider adding here: The particle size of the re-suspended lead would be expected to strongly influence its environmental transport and human uptake, with larger particles being less mobile and respirable.

2-50 L12 Suggest changing "low" to "slow" for a more active phrasing.

2-51 L20 Does this model involve this mechanism (re-suspension) only or does it include the other known mechanisms by which soil lead concentrations decrease slowly over time?? If the former, maybe add at end of sentence…"..by this mechanism alone ."

I suppose that means we can't wait for re-suspension to be of much assistance in remediation.

I mention this because often revisiting known old lead works yields new landscapes, including new land use patterns.

2-52 L20 Really <1 ppm as a background value? I might have expected a higher value, more like 25, like agricultural or suburban soils. Or is that peculiar to forests, because forest floor litter and the uppermost soils are so rich in organic matter?

A Few Comments on Chapter 4:

4-34 4.2 Would like to see a comparison of fugitive lead emissions with total factory through-put (annual production tonnage) for primary and secondary smelters. Which process is less contained? The one that's cleaner could be held up as an example for the other.

4-9 L 10 That's a lot of ingots. 130+ lbs daily.

4-34 L 17 Using 5 significant figures I found jarring. Why put down more figures than the uncertainties permit. This format enshrines computer output suggesting a level of accuracy which it lacks. So I suggest 2 or 3 significant figures and a statement of uncertainty such as 8.4± .8 and 0.74 ± 0.1 (I made up these standard errors, but trust that the Risk Assessment Report contains them.)

Comments by Michael Rabinowitz January 2007

On Lead Exposure/Risk Assessment Technical Support Document

Comments on Chapter 5:

I found section 5.1 to be clearly written.

Section 5.2, Exhibit 5-12. Is it possible to add to the Results Column values for the observed range and average values of blood lead for comparison with the models, as was done for the Primary Pb Smelters as shown on the following page of the same Table?

In the Implications Column the Leggett Model's performance was described as underestimating "somewhat". Could specific values be provided? Percentage or absolute amount would be helpful.

On page 5-26, line 14-16 makes a general statement about the agreement between the 2 models and the measured values. This is shown above in the Results Column. I was struck by how the 2 models differed. One gives a 95th percentile as 20 to 29, but the other generated a value of 5. If other panel members, who are more comfortable with is sort of statistical comparison, see no reason for my concern, then I'll go along. If this is as good an agreement as modeling can provide at this stage of their development, then we need to consider other means of setting a standard.

Page 5-32. I was struck by the good agreement between the fraction of lead from Policy-Relevant Sources across the wide range of blood lead levels for both the Current Conditions and the Attainment Exposure Scenarios. I concur with the observations expressed on lines 6 to 10.

Generally from Section 5.4 The predicted median blood lead between the two models differ by only about 0.3 μg/dL for the median values of lifetime average blood lead, and 0.6 for concurrent values. Compared to the distribution of blood lead values, it appears that the models agree for lifetime average predictions much more closely than for the concurrent values. This may be because the IEUBK model gives a broader distribution of lead values, so differences near the mean affect smaller fractions of the population. I take this general agreement as support for the overall approach of using these models to predict blood lead levels sufficiently well for policy discussions, if their limitations are presented.

Reflecting on these developments, I imagine that the two models could be forced to concur (at the median) by arbitrarily adjusting some of the main parameters. For example, values used for fractional gut absorption could be set to make the blood values aggress. At least the blood lead distribution would be scaled up or down, without changing its shape. Similarly, varying the value for the fractional excretion of lead into urine from blood plasma would proportionately change the predicted blood lead level. I would predict that the amount by which the variable would be tweaked is well within the range of our uncertainty for this variable, based on clinical measurements. The values used for those biokinetic parameters often are based on just a few subjects, with higher lead intakes and higher blood levels, closer to 20 μg/dL, than currently prevail. Adjusting these biokinetic coefficients to match the models' blood value outputs would likely improve our estimates for these biokinetic variables. Try not to think of this suggestion as cheating but as calibrating.

<u>Comments on Chapter 6:</u>

Overall I applaud the overall approach of taking a few "case studies" of contemporary American lead exposure. It should be useful for teaching and standard setting.

P6-17, line 36. Last sentence is confusing for me. Does it mean: "The predicted IQ losses at difference ages for a given blood lead level might all be equivalent, but the blood lead for a given environmental lead is known to be age dependent." Or some permutation of that? It's not clear to me how or why the persistency issue is required here. Or drop the whole sentence.

In Section 6.4, the graphs in Exhibits 3-3, 3-6, 3-8.
I had difficulty in my copy matching the symbols with the lines. Perhaps larger or more extreme symbols could be used or putting the order of the symbols in the key box to same as the order they appear on the vertical axis. It might help those of us who were not able to attend the presentation.

Exhibit 6-24.
I wish I was in RTP to sympathize with you over these findings: Both for blood and IQ, of the eight issues you explored, using reasonable values for what we know, you found very often ranges of over 100% or 200% between the highest and lowest number of people in the highest risk category for a given environmental lead load. For example, if I'm reading this right, our uncertainty in gut absorption factors impacts our ability to estimate IQ loss among the most effected 10% of our children over an error range of -100 to +50%.

Looking at the many ranges in Exhibit 6-24, does it matter being zero-centered, and others have a non-zero bias?

What to do to decrease these ranges of uncertainties? We could identify research needs for next rounds decades from now? Maybe if we knew more, we could refine our estimates. But I suspect that human variability is such that we will always have a wide blood lead GSD and a wide range in our responses to lead, hence we will always be faced with a wide range of risk estimates. But at least it is not a thousand or ten-thousand percent.

For now, we recognize this level of predicative imprecision, with some estimates being off by 100%. We can use the size of this range in setting appropriate safety factors in the setting of standards. The size of any safety factor needs to accommodate the magnitude of these unknowns.

Dr. Armistead (Ted) Russell

Review of Chapter 2 of the Lead Staff Paper: Characterization of Ambient Lead
Ted Russell

This chapter has a plethora of information about ambient lead concentrations, along with emissions, loss processes and physical characterization. If one reads though it, you can get a very good idea of what is out there, its physical why and where it is going. In that regard, the chapter succeeds. However, it does not do so very efficiently, and does not seem to capture the real issues as well as it might. To me, given the general trend in lead, the real issue is best portraying the high end concentrations, providing trends at such locations, along with the more typical. I would also want a good concentration distribution plot (e.g., like a pdf), which I think provides much more information than the box and whisker plots, particularly when the box plots do not also show the high outliers. For example, they could plot the maximum quarter average for each site on the distribution. They might compare this with, say, a similar plot of the second highest monthly average.

I was also disappointed in the modeling estimates section. I started reading it, thinking that there would be some actual results represented. Without some results, the section raises more questions than it answers, and falls far short of the information that could be provided. First, as noted beforehand, a number of the larger sources do not have monitors nearby. Models can provide some idea of hot spot levels and locations. Second, it is noted that the modeling results generally are below the observations. They proffer some explanations. A key one is that the source strengths are underestimated. This feeds back to the prior sections of this chapter, and significantly impacts any uncertainty assessment. It would be good if they were to take the model results and also develop a concentration distribution for direct comparison to the one developed based on the observations (thus use the same metric in both cases). Again, specific attention should be paid to locations with higher concentrations. In doing this, they should look to adjust the modeled distribution to account for the low bias in the simulations.

At this point, the analysis should be able to provide a good characterization of the likely levels of lead around the country, even where no monitoring is being conducted. From this, a much better view of the severity and extent of the problem is achieved. In the process of getting to this point, uncertainties should be characterized.

Specific points.

2:21:6 (Chapter:page:line): Not all measurement approaches require chemical extraction (e.g., XRF).

2:23:14-16. These lines suggest (somewhat repetitively) that the reduction in Pb monitoring was due to the need to fund PM2.5 and ozone monitoring. Given when PM2.5 monitoring was initiated over most of the US (early this decade), that PAMS came on line starting in about 1993

and the trend in ozone monitoring, does this gel with what is shown in Figure 2-5? This should be documented.

2-28 Figure 2-10. This figure could be greatly improved. First, why just 42 sites? Second, go back before 1983 (n.b., the figure caption mistakenly says 1980). Why is it flat from 83-84. Seems unlikely at best. This should also show the outliers at the upper end, e.g., all sites that had levels above the NAAQS. It also brings up an interesting question: why did the average Pb go up by about 2x in 2001? Bottom line: Can this figure be extended back in time and also show the sites over the NAAQS?

2-30 Figure 2-11: A better figure caption is warranted.

2-31 Figure 2-12: The table in this figure appears inconsistent with the same information in Figure 2-11. It looks like the table is summarizing Max quarterly average.

2:34:4. It would be of interest to note Pb levels for East Helena as they are not given in the tables below. What do the Pb levels look like in such an area that is out of compliance, but would appear to have dropped to the point that the site would now be in compliance (with an explanation of why).

2:37:2 I very much agree with the statement that shorter term averaging than quarterly, and that monthly would appear to provide a balance between the weight given any one observation and the need to assess variations in [Pb].

In direct response to the Charge:

To what extent are the emissions and air quality characterizations and analyses clearly communicated, appropriately characterized and relevant to the review of the primary and secondary lead NAAQS?

As noted above, there is a plethora of information on these topics in this chapter. However, I view it as inefficiently presented for the charge at hand. More concentration and details should be provided relating to the areas that continue to have Pb levels near or above the current, and somewhat tighter NAAQS. To adequately assess what will be the population risks and decreases in those risks, associated with various standards, the high end is where the action is. Further, as noted, there are many areas near major sources that are not well monitored. Modeling results are required in such a case, and the level of information provided from the modeling is inadequate. If there are but one or two areas that might even be affected by a change in the standard, this is important to know.

Does the information in Chapter 2 provide a sufficient ambient Pb-related basis for the exposure, human health and environmental effects, health risk assessment and environmental assessment presented in later chapters?

Similar to above, it is lacking in terms of providing a more comprehensive characterization of lead levels nationally and the high-end concentrations. This may be particularly true if other health endpoints are examined besides IQ loss, e.g., more acute impacts.

A further limitation is that one needs to look in to the future as to what various standards will do to risks associated with Pb exposure, particularly given the increased evidence of risks below 10 ug/dl. I was hoping to see some more concrete analysis of how future Pb levels and exposures across media will evolve, and how those relate to different standards. While such data may be scarce, the current SP could provide some more quantitative information. Along these lines, they provide a graph for resuspension rates, and say that for SoCal conditions, this will lead to little change in soil Pb for quite a while. How about other areas?

Review of Chapter 2 of the Lead Human Exposure and Health Risk Assessments and Ecological Risk Assessment for Selected Area

This chapter presents an overview of the way the Risk Assessment will be conducted, in particular identification of selected case studies and models to be used. The first section notes that they will present the human and ecological associated with two scenarios: current conditions and attainment of the NAAQS. This is a very good start on providing the type of information needed, but is insufficient to provide the information necessary for CASAC or the administrator to do the job at hand. What is also required is information on risks associated with levels, and forms, averaging times, etc., other than the current NAAQS. Further, it took a bit to find the results of those two cases later on in the chapters, in part because only one of the cases had a non-attainment vs. attainment comparison. This document could benefit from a two page summary of the key approaches and results. In particular, they should provide the change in each of the cases considered.

In setting up how they plan to assess the reductions in risk associated with meeting the current standard, they make a mistake in the approach. Unlike the ozone SP, to simulate attainment, they essentially cap ambient levels at the standard, leaving all other concentrations unchanged. This is not what will happen in real life. Controls implemented to meet the standard will decrease Pb at other times as well (or else they should consider tighter standards). **Thus, a more real life attainment scenario would be to decrease Pb concentrations at all times, rolling the levels back towards the background.**

While I like the idea of case studies, they do not replace an assessment for the population as a whole. While there are always uncertainties associated with scaling things up (or down), they are instructive and provide the type of information needed to set a national standard. For one, it is important to know what are the risks and estimated impacts, nationally, at the current level of exposure. While this may be viewed as being provided by the NATA assessments, they are

insufficient for the reasons spelled out in the SP, and also they do not go far enough, e.g., considering alternative standards. A more comprehensive national-level analysis should be added. Further, this lays the groundwork for assessing the change in risks, nationally, associated with different standards.

I am somewhat perplexed by Exhibits 6-1 and 6-2. Looking at the column IQ Loss under IEUBK in 6-1 and 6-2, the first thing that struck me was that in 6-1, a value of <1 is used, and in 6-2, a value of 0.5 is reported. Why the difference in how the values are reported? Are they different? Second, given how the attainment scenario is run, i.e., that air concentrations are truncated, how can the impacts be greater in the attainment scenario for any of the %'iles? The results from this case study also reveals a problem when only taking a snapshot in time: given that the inhalation pathway is so small, there will be virtually no immediate impact from meeting (or tightening) the standard as only the inhalation and how dust exposures respond, and the dust response is muted as it does not have a factor that integrates over time and is dominated by other terms in the functional representation. It would be useful in Chapter 4 (e.g., around page 4-16) to show the fraction of dust from air. While I can only ball park this at present, it would appear that significant reductions in lead in the air, using the H6 model, would lead to very significant reductions in dust-Pb, and hence a major reduction in the highest IQ loss individuals. True? I would like to see this case run, e.g., tightening the standard to, say, 0.5 ug/m^3. Would this push all of the blood lead levels to below 10 ug/dl?

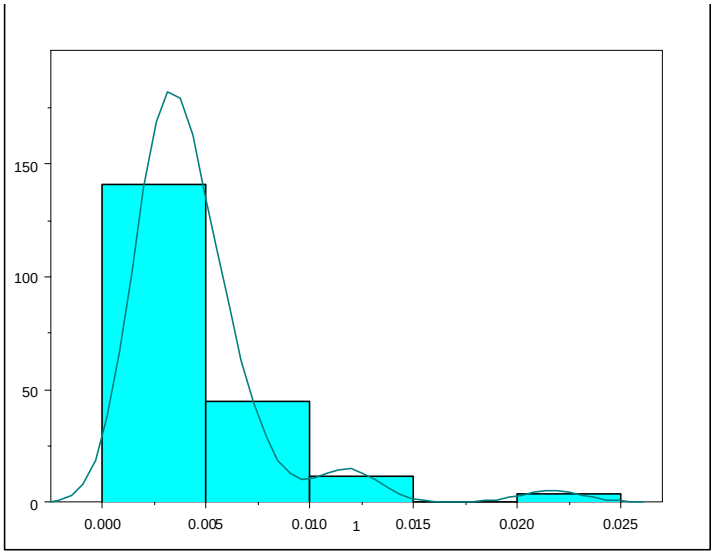
Dr. Joel Schwartz

Comments on Lead Staff Paper

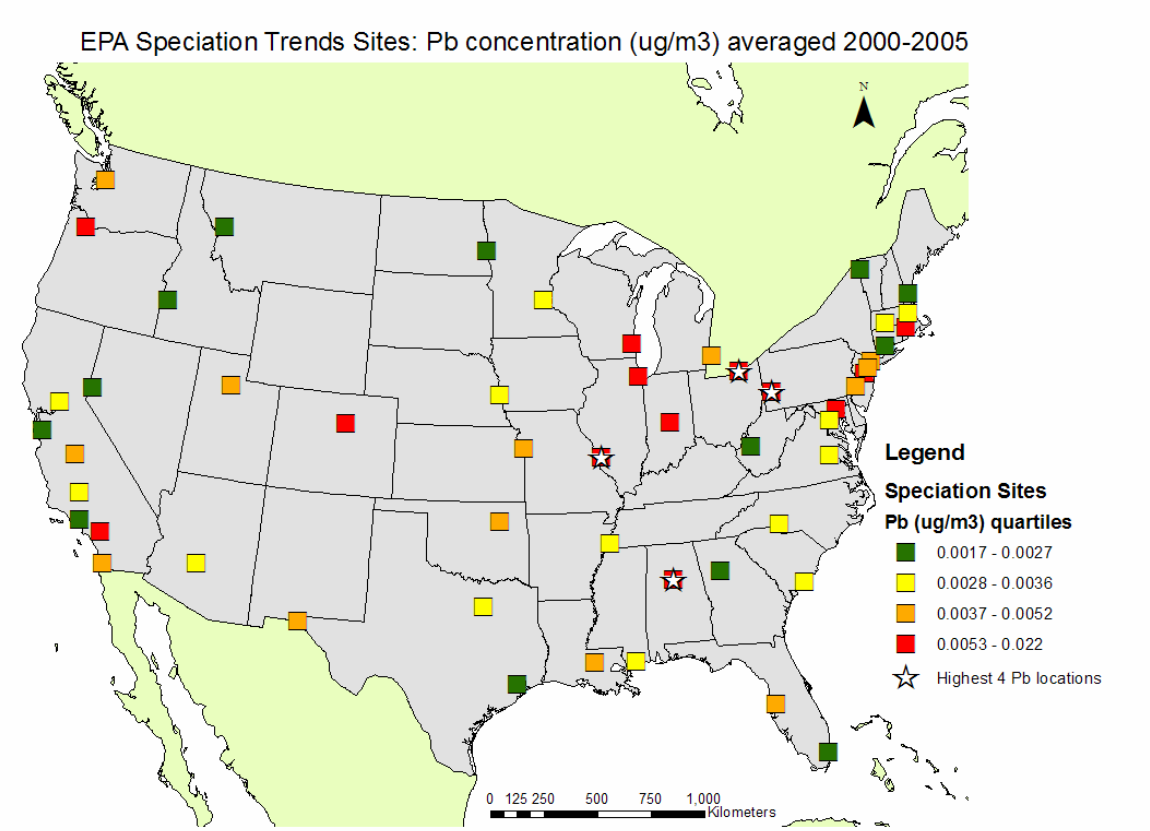
Joel Schwartz

First I would like to address the issue of whether there is anything to be gained by a national (i.e. Ambient Air Quality Standard) approach Vs a site by site (Air Toxics) approach to controlling lead. The argument for dropping a NAAQS is that there is little to be gained, except in a few places that could be addressed by MACT. To examine this, I took the data from the STN network, took the average by location, and plotted a histogram of the distribution below. What is clear is that about 60% of the locations have levels below 0.005, but that the remaining locations have concentrations that range up to five times as much. Clearly then, there is a range of exposures that our urban citizens suffer from, and that a good third of them have exposures considerably higher than the least exposed half of the urban US. This suggests that there is exposure above what is typical in a large number of US cities, and that a national approach to get those cities to identify what needs to be done to reduce exposure is appropriate.

Figure 1. Distribution of Long Term Mean Pb from the STN urban sites



The following figure shows the locations of the sites, by long term average concentration. It is noticeable that one of the four highest urban locations is close enough to Herculaneum to potentially be impacted by long range transport. This raises the question of whether the risk assessment, by not including exposure outside a defined radius around the sites, is missing some health impacts. Certainly, if one assessed the impacts of coal-burning power plants within 100km of the plant, one would miss most of the particle health effects.



While I recognize that there are limits to what OAQPS can do with its modeling, I am concerned that the re-suspension of road dust issue is not being quantitatively addressed. In some source apportionment models we have done of elementals on PM2.5 filters, we have seen Pb load on a factor that seems to be traffic related, a combination of road dust, brake lining, etc. That means, at our monitor on the roof top of the Countway Library, a major source of lead is road dust. This is likely to be true for many children in Boston as well.

While the tables present sources in order of emissions this does not include the important concept of intake fraction. For example, recent PM related studies have shown that roadside emissions are much more likely to make it into a lung than emissions from distant power plants or factories. Are we missing important exposures by ignoring this? One suspects that the traffic related sources, including re-suspension, are under appreciated when ranked simply by emissions.

Specific Comments:

3.3 While bone resorption is higher in women, this is an issue for older men as well.

3.13-14 I agree that the evidence points to a higher slope at lower doses, and that a log-linear curve is a reasonable choice. I am therefore mystified by the latter choice of several thresholds to examine. Yes, it is true that there is little data below 2.5 ug/dL. But the available data suggests the slope is steepening, not getting less steep, so why is it reasonable to argue the uncertainty

suggests a sensitivity analysis with a slope of zero? A more reasonable sensitivity analysis would have the slope double below 2.5 ug/dL. Why was this not tried? Given asymmetric evidence in favor of steeper slopes, sensitivity analyses that are only asymmetric in going in the contradictory direction seem unjustifiable. Do the sensitivity in both directions, or not at all.

4.7 While cognitive health benefits in children may be the most sensitive endpoint, we are talking about an ambient standard, not setting a reference dose. That is, we can quantify health benefits for multiple outcomes, and not just choose one. While there is a finite amount of time (and labor) available, the health benefit analyses for both the lead phase-down and the drinking water lead standard included other outcomes. So did the CDC benefit analysis for housing abatement. This would be a minor point except that in those analyses, the adult benefits were about the same size as the ones for children, so this could well impact the choice of the standard.

TSD Comments

1: Uncertainty. The TSD assumes a GSD of 1.5. This is considerably smaller than that seen in NHANES IV. The authors argue that since they are looking at small areas, their GSD should be smaller. While directionally true, it is not clear that this justifies the drop from 2.2 to 1.5, which is a great deal. The use of localized site data from the 1990's to justify 1.5 is unsatisfying, since as the authors note, as the mean blood lead falls, the GSD increases. The mean blood lead in the locations they are examining is lower than was seen in the sites from the 1990's which provided a GSD of 1.5. I think this is too low, and should be increased.

Secondly, the authors are somewhat schizophrenic in their modeling. They reject epidemiologic studies relating dust and soil lead to blood lead, and insist on only using the biokinetic model to estimate means. They then reject the biokinetic model and only use epidemiologic data to estimate variances. The reason everyone does not have the same blood lead in the same block group is, in part, that intake varies, absorption varies, etc. Hence, a Monte Carlo simulation varying parameters for the IEUBK model should produce estimates of variance. These could be compared to the variance estimates from the epidemiology studies. Large differences from the empirical data would suggest changing the range of some of the parameters. Deviations between means estimated from the epidemiology and IEUBK model could likewise be useful. I think both approaches need to be used for both mean and variance.

2. The assumption that no improvement in IQ will be seen if blood lead levels fall below the 5th percentile of the Lanphear distribution is unjustified. Recent data from NHANES show effects below levels of 2.4 to 6 ug/dL, and there is no data that supports a threshold. The baseline approach should assume no threshold in the range of exposure seen in the population, and sensitivity analyses should be done around that baseline. To argue that uncertainty in an association justifies assuming it is zero is bad science and bad risk assessment. The expected value of uncertainty is not zero.

There is no need to include the intercept in the analysis; the risk assessment is predicting IQ loss, not IQ. Hence that issue need not be dealt with.

Dr. Frank Speizer

Pre-meeting Comments on Lead Staff Paper dated Dec. 4, 2006

Submitted by Frank Speizer, MD
1/29/07

I have reviewed Paul Mushak's comments and in large part I agree with much of what he has done in a thoughtful outline of the details with which I believe Staff needs to promptly consider. Many of my comments simple express frustrations as to where I see inconsistencies and concerns about lack of completeness to this point. I am sure much will be handled in subsequent drafts, but those drafts will be under time pressures and I would predict will lead to frustration by CASAC as well as the public in have adequate time for review. (I may be wrong, but past history does not offer much hope).

From Chapter 1, I note in the footnote on page 1-5 that the agency indicates that it does not anticipate having funding to perform additional quantitative ecological risk assessment work for this review. This seems inappropriate since we indicated at the last review that the progress on information to inform the secondary standard had not progressed in the last 10 years. Why are we still in this same state and seem to not be able to move forward on it?

Chapter 2

Section 2.3.2, page 7. Sources other than air not yet evaluated. Will this be done? Seems like an important source would otherwise be overlooked. Page 2.52 section 2.6.2.2 suggests that soils can contain up to 50-100x background and somehow this needs to be at least discussed as to how this might alter policy relevant background.

Chapter relevant questions: It would appear that at least for lead the decline in monitoring of TSP to PM10 and PM2.5 results in an uncertainty as to the trends and less capacity to follow trends. Although TSP and PM10 are highly correlated this breaks down for PM2.5. Although this is described in this chapter it seems not to have been resolved as to how it will be dealt with. Unless this comes up later I think a "policy relevant statement" is required.

Chapter 3

Section 3.2 leads to a conclusion that seems appropriate but incomplete. If we were only worried about "acute" exposure then blood lead seems appropriate but this certainly is not the case. Therefore need to at least the discuss the role of other biomarkers of lead that might be considered as alternatives or in addition to blood lead for more chronic exposures. These would include both other markers measured in blood as well as bone lead (that can be measured non-invasively.

Table 3.1 and 3.2 I will need to go back and check but I would have thought if not updated this figure would have been reproduced in the current CD and reference to it rather than the 1986 CD would have been more appropriate. Ditto table 3.2.

Summary of staff questions: The presentation seems to have integrated the material from the CD reasonably well. There seems however, presumably for reasons of simplicity to have ignored the potential importance of chronic vs. acute exposure as expressed by focusing on blood lead as the parameter of interest. With regard to the conclusions I find it hard to focus on the form of the blood Pb metric as the only value to consider. This clearly is important for the children but does not do well for adult effects. With regard to the threshold argument, it seems inappropriate to have focused on it. Clearly both human and animal toxicology do not justify any threshold and although this is stated it is muddled in the discussion by suggesting a hypothetical level for individuals when in fact this would be the specific case where no threshold would exist and if there is a "threshold" it would be for a subgroup.

Chapter 4

Page4.5 and 4.6, Figure 4.1

I am concerned about putting all exposures other than ambient as "Background" for *Pathways*. As indicated in *Routes* the ingestion route is likely to be considerably higher than ambient and thus will make Background overwhelm other sources. This may all be semantic but may have important relevance to what might be considered policy relevant. Clearly this is more complicated and cannot be simply managed as background. (I suggest a separate term in the modeling will be necessary.)

Page 4.9-10:The fact that this area is in non compliance for Pb NAAQS and this is based on 9 TSP monitors it is not clear how this might relate to standards going forward, particularly as we have moved away from TSP for particles. This should be discussed in greater detail (even if it is only to say we are staying with (or changing from) a TSP standard.

The three selected sites will each have to be considered separately as each has measures which do not overlap. Eg. No measure of alternative sources, PM10 vs. TSP measures.

Page 4-24, section 4.3.1.3: Further discussion or documentation is needed on the difference in particle size distribution for lead from presumed mobile sources vs. diesel particle distribution.

Page 4.26, Table 4.2: For ambient concentration estimates may need a footnote fact that dispersion model based on TSP (if it is) and Monitoring data based on PM10.

Page 4.27, line 29: Should this be "less than" rather than greater!!!!

Table 4.10: This seems unacceptable. Caveats are given for the above two models that suggest potential biases in estimates. Performance evaluation for perhaps the most relevant component to the general population must be completed (along with what ever caveats are necessary).

Page 4.78: Must keep in mind: "Consequently, the risk results associated with the pilot are not intended to reflect our best estimates of risk associated with these case studies. Rather, they reflect preliminary estimates, limited by the initial application of our modeling tools and information. We intend to build on our experience and findings associated with the pilot assessment in designing and implementing the full-scale risk assessment, the purpose of which is

to inform the Agency's development of and consideration of NAAQS policy options with regard to policy relevant sources of ambient Pb."

4.81 comment on summary of plans: It is not clear which of the suggested additional activities to be carried out will, in fact, be done. It would have been better to have seen these bullets as a list of work scope factors. The past experience has been that the agency will run out of time and the work will not be completed. Worse would be to do partial work on each topic that to have set out a set of priorities of what would be accomplished and in what order.

General comments on questions:

There seems to be incompleteness in this chapter that permeates throughout. Consideration of the sites, seem to have been selected as a matter of convenience. Suggestions in the final analyses that other sights will be added are made yet no details are provided and one wonders where these will be, how they will be selected, how much less or more the completeness of data will be and how useful the additional results will be. Although uncertainty is considered, it is not clear how much is a statistical exercise vs. real lack of data used or available to make a more objective assessment of uncertainty. Given that most of the population does not live near primary or secondary smelter sources, it seems to me that more work needs to be devoted to finding data that might be used to do better modeling of roadway or urban exposure and analyses devoted to this segment of the risk assessment.

Chapter 5

Page 5.9: The current data would suggest that this figure would need to be reduced by a factor of 3 (5ug/dl).

General comments on questions:

The entire chapter reads like a promissory note with out much expectation that the promise will be met. By simply discussing what was done in the past the authors have not told us much of what they plan to do. In addition, the "facts" that lead to the previous conclusions, though stated do not appear to take into account current knowledge. For example the indication that 30ug/dl is the 99.5% level to shoot at may have been ok in 1977 but is clearly irrelevant today (as indicated by various footnotes that bring the level down to 10ug/dl). Somewhere it might be indicated that the all these level may be as much as three time higher than the science today would indicate, and therefore NEW CALCULATIONS WILL BE PERFORMED

Chapter 6

General comment: This chapter seems to be as incomplete as the preceding one. Will Staff have time to do the additional analyses suggest that must be done and will CASAC see them before the work goes to the administrator? If not than the purpose of CASAC being in the loop is clearly inappropriately relegated to an after thought. Even in light of a change in the approach being used to the setting of NAAQS this seems to be violation of *de facto* if not *de jure*. I for one will not be part of it and will not sign off in any way on draft without the work being reviewed.

Dr. Ian von Lindern

CASAC Review of First Draft Lead Staff Paper and Related Analyses

Pre-Meeting Comments of Ian von Lindern

My comments address both the OAQPS Staff Paper and the Pilot Analyses and are arranged in three main sections – i) Introductory Comments that provides general observations and policy related comments; ii) Answers to the Charge Questions: and iii) Technical Comments that follow the structure of the analyses.

Introductory Comments

Overall Strategy and Presentation: The Staff Paper and the underlying Pilot Risk Assessment Methodology represents a substantial and comprehensive effort to quantify the pathway-specific effects of childhood lead exposure in typical scenarios that might be considered under a revised NAAQS. The logic and sequencing of the modules that have been combined to characterize lead from source to adverse health risk represents a thorough understanding of important factors and mechanisms that influence childhood lead poisoning and the consequent effects.

It is a commendable effort and appropriately captures both the knowledge and advancement in assessing environmental mechanisms and lead health risks that have occurred in the last twenty years. It also is indicative of the level of sophistication in the science of risk assessment that has been accomplished; and the data presented and used largely captures the environmental improvements and consequent benefits of lead regulation strategies employed since 1991.

In producing these documents the staff has done an excellent job of assembling and developing a series of empirical and mechanistic models to assist in quantifying lead health risk. However, the emphasis to this point reflects an understandable bias toward development of the methodology that comes at the expense of critiquing how it can assist in developing effective policy, and in performing such analyses. There is some confusion, at least for this reviewer, as to how these analyses will be used in policy recommendations. The corollary risk and exposure assessment analyses conducted for the last review in 1990 were presented to CASAC members as a proto-type methodology for assessing risk to lead in multi-pathway environments that could then be used to assist in developing remedial and compliance strategies for the agency programs. Notably, that methodology was subsequently used and constantly upgraded in several Agency programs that ultimately led to the successes in multi-media exposure reductions noted in this Staff Paper. The 1990 Committee reviewed those analyses in the context of its scientific and technical appropriateness for assessing risk, specifically for the Air Programs, but also was charged by then Administrator Riley to assess lead risk assessment protocols across several Agency programs.

NAAQS Policy Implications: However, there is in this draft report a seeming, but less understandable, bias toward supporting a revocation of the NAAQS. This seems premature, as

there is little in the analyses, to this point, suggesting what might be a "safe" or, at least, an acceptable ambient air lead level. There also seems to be no obvious route laid out to make such a determination. More skeptical reviewers might view the document as tending toward a conclusion that because we see no problem, there is no problem and no need to regulate. This conclusion, it seems, could only be supported if we were sure that that we had adequately monitored for potential problems and there are sufficient guarantees and safeguards that no problems could occur in the future. In this regard, the monitoring record is shallow, at best, and, as yet no determination of what is an acceptable ambient level, if there were a record to compare to it. And most importantly, what mechanisms are in place to ensure that any new source developed would not produce emissions that would exceed this critical threshold, whatever that " safe or acceptable" level might be.

A clear and transparent understanding of the policy questions that these analyses will assist in answering is of critical importance to entire procedure. This is important to any reviewer to help focus comments and to any stakeholder to critique and weigh in on the decisions. However, for this case it is paramount that the hypotheses be clearly stated. The inherent nature of the structure of the models and the lack of precise input data regarding the sources of pollution, the receptor populations, and the specification of parameters and co-factors require that the problem be clearly stated and the uncertainty and sensitivity analyses address that hypothesis.

Charge Questions to the CASAC Pb Panel

Ambient Pb information and analyses (Chapter 2):

1. To what extent are the emissions and air quality characterizations and analyses clearly communicated, appropriately characterized, and relevant to the review of the primary and secondary Pb NAAQS?

It is a commendable effort and appropriately captures both the knowledge and advancement in assessing environmental mechanisms and lead health risks that have occurred in the last twenty years. It also is indicative of the level of sophistication in the science of risk assessment that has been accomplished; and the data presented and used largely captures the environmental improvements and consequent benefits of lead regulation strategies employed since 1991. However, little in the analyses, to this point, suggesting what might be a "safe" or, at least, an acceptable ambient air lead level. There also seems to be no obvious route laid out to make such a determination. A clear and transparent understanding of the policy questions that these analyses will assist in answering is of critical importance to entire procedure. This is important to any reviewer to help focus comments and to any stakeholder to critique and weigh in on the decisions.

2. Does the information in Chapter 2 provide a sufficient ambient Pb-related basis for the exposure, human health and environmental effects, health risk assessment, and environmental assessment presented in later chapters?

The Staff Paper provides significantly more information in this regard than was presented in the AQCD and the information seems to be a good and fair representation of the state of knowledge

in the U.S. at this time. It does indicate, however, that the database is a poor representation of true emissions and ambient air lead levels in the U.S. today. This lack of appropriate data should be dealt with in a more comprehensive sensitivity analyses and uncertainty discussions.

Pb-related health effects (Chapter 3):

1. To what extent is the presentation of evidence from the health studies assessed in the Pb AQCD and the integration of information from across the various health-related research areas drawn from the Pb AQCD technically sound, appropriately balanced, and clearly communicated?

These analyses seem to suggest the risk metric is whether there is significant potential for IQ loss among children exposed to the policy relevant sources? This, however, doesn't seem to be explicitly stated and it is unclear whether the Agency considers IQ loss an unacceptable outcome. There is little in the current documents regarding what the Agency considers regarding the significance of IQ loss and whether there is a threshold of concern that will be a benchmark in setting a new standard or developing an alternate protective risk management strategy. This discussion needs to be clearly laid out in future drafts.

2. What are the views of the Panel on the appropriateness of staff's discussion and conclusions in Chapter 3 on key issues related to quantitative interpretation of epidemiologic study results, including, particularly, the form of a blood Pb-response function for neurocognitive effects, and the form of the associated blood Pb metric?

The discussions are well developed and clearly presented. The appropriateness of the blood lead metrics seem to be in question because the use of lifetime value and the concurrent (6-7 year old) blood lead level tends to moderate the observation that both the maximum blood lead tends to occur at the most vulnerable age. Presumably, this is accounted for in the structure of the empirical relationship used, but I must defer other committee members more familiar with these results regarding the appropriateness of the quantitative estimations associated with the health effects.

3. What are the Panel's views on the adequacy and clarity of the discussion of potential thresholds in concentration-response relationships presented in Chapter 3?

There is some concern with the treatment of the "cut points" in these analyses. Assuming that there is no effect below the cut point eliminates the possibility of a Type I error as no false positive is possible below the cut point. The only possible error result is a false negative. This should be pointed out in the discussions and considered in determining an acceptable level of risk or margin of safety in recommending a standard.

Human Exposure and Health Risk Analysis, pilot phase (Chapter 4):

1. To what extent are the assessment, interpretation, and presentation of the results of the pilot exposure analysis, including characterization of Pb concentrations in media, the modeling of

multi-pathway Pb exposure and application of biokinetic blood Pb models, as presented in Chapter 4 technically sound, appropriately balanced, and clearly communicated?

The Staff Paper and the underlying Pilot Risk Assessment Methodology represents a substantial and comprehensive effort to quantify the pathway-specific effects of childhood lead exposure in typical scenarios that might be considered under a revised NAAQS. The logic and sequencing of the modules that have been combined to characterize lead from source to adverse health risk represents a thorough understanding of important factors and mechanisms that influence childhood lead poisoning and the consequent effects. The report recognizes that there are several difficulties inherent in both estimating and quantifying outcomes within each step and in utilizing those results in subsequent steps. However, the report fails to recognize the effects are often multiplicative, meaning that, although uncertainty increases, the consequences of under-estimation or over-estimation can be dissimilar, and are dependent on the status of other variables in the formulations.

2. Are the methods used to conduct the pilot exposure analysis, including the modeling of population-level distributions of total blood Pb levels and the pathway-apportionment of those blood Pb levels (e.g., air-inhalation, versus soil-ingestion versus dust-ingestion, versus background) technically sound? Does the Panel have any suggestions for improvements in the methods used?

Several assumptions made in individual steps in the methodology tend to possibly underestimate of the policy relevant background contributions to intake, ignore the potential impacts of aggregate soils in the community, and underestimate dust lead concentrations. This likely results in minimally low intake estimates for soil and dust that translate to low blood lead predictions and health impacts in the primary and secondary smelter situations. These in turn can result in false negative findings for children near the threshold concentrations. Additionally, the lack of air quality data may dictate that determining an appropriate standard will likely rely on modeled concentrations, emphasizing the importance of conducting appropriate sensitivity analyses.

3. What are the Panel's views on the staff interpretation of the performance evaluation completed for the pilot analysis (and described in Chapter 4) with regard to the representativeness of individual modeling steps completed for the analysis (e.g., characterization of ambient air and outdoor soil Pb levels and the estimation of blood Pb levels for specific case studies)?

See the response to the previous question and the expanded discussion in the text of the comments.

4. In general, are the concentration-response functions and blood Pb metrics (i.e., lifetime average, concurrent blood lead) used in the pilot analysis appropriate for this review?

The discussions are well developed and clearly presented. The appropriateness of the blood lead metrics seem to be in question because the use of lifetime value and the concurrent (6-7 year old) blood lead level tends to moderate the observation that both the maximum blood lead tends to occur at the most vulnerable age. Presumably, this is accounted for in the structure of the

empirical relationship used, but I must defer other committee members more familiar with these results regarding the appropriateness of the quantitative estimations associated with the health effects.

5. Are the methods used to conduct the pilot health risk assessment, including the application of the cut points in relation to the concentration-response functions employed, technically sound? Does the Panel have any suggestions for improvements in the methods used?

There is some concern with the treatment of the "cut points" in these analyses. Assuming that there is no effect below the cut point eliminates the possibility of a Type I error as no false positive is possible below the cut point. The only possible error result is a false negative. This should be pointed out in the discussions and considered in determining an acceptable level of risk or margin of safety in recommending a standard.

6. To what extent does the sensitivity analysis completed for the pilot analysis (and described in Chapter 4) identify key sources of uncertainty and provide an assessment of their impact on risk results?

The inherent nature of the structure of the models and the lack of precise input data regarding the sources of pollution, the receptor populations, and the specification of parameters and co-factors require that the problem be clearly stated and comprehensive uncertainty and sensitivity analyses address that hypothesis. Extensive sensitivity analyses should be conducted by first (as indicated in the reports) identifying an appropriate baseline or typical model run, and then varying key components while holding others constant. However, the sensitivity runs should be accomplished using both lower and upper limit analyses. It would enhance the uncertainty analyses by framing the discussions in terms of Type I (false positive) and Type II (false negative) errors for the final risk assessment.

7. As part of the NAAQS review, there is interest in attempting to differentiate Pb exposure and health risk impacts for modeled populations between (a) historically deposited Pb (e.g., near roadway dust/soil lead from leaded gasoline) and (b) newly emitted Pb. Does the Panel have specific recommendations regarding approaches that might be employed in the full-scale assessment for this purpose?

The document fails to recognize that there is significant variance in the background levels and certain populations and individual members of all populations will be at greater risk as a result of these background sources. It is as important to identify these at-risk populations and assess the possibility for Type II error (false negative) with these groups as it is to assess uncertainties based on statistical extrapolations of mean or baseline characterizations.

8. What are the Panel's views on the most important issues to be addressed in the subsequent full-scale human exposure and health assessment that will be presented in the revised documents?

The intended use of the exposure and risk analyses as these relate to standard setting or alternate risk management strategies should be clearly, explicitly and transparently stated. The baseline

concentrations used in the initial model runs should reflect typical levels and corresponding reasonable maximum and likely minimum values should be identified and supported by references, if available. Sensitivity analyses should be structured to specifically address the likelihood of false negative or false positive conclusions in the uncertainty analysis. Values in the current draft for soils and dust estimates are likely low and should either be adjusted higher in the baseline runs, or higher values should be specifically addressed in sensitivity analyses and subsequent uncertainty discussions.

The Primary Pb NAAQS (Chapter 5)

1. What are the Panel's views on the adequacy and clarity of the presentation of the basis for the existing standard and conclusions reached in the last review?

The document accurately captures the history of the NAAQS process for lead, but does little to indicate any policy implications or conclusions for today.

2. Based on the information contained in the first draft Staff Paper, as well as the AQCD, does the Panel have recommendations with regard to specific aspects of the standard to be considered in developing policy alternatives? For example, considering the prominence of the soil and dust pathways for ambient Pb exposures, and the evidence regarding environmental response times, is there reason to give more emphasis to consideration of an alternative (shorter or longer) averaging time; and, how might this be considered in the full-scale risk assessment given current capabilities?

The report needs to comprehensively consider and address the relationship between ambient air lead concentrations and resultant soil and, particularly, dust lead concentrations. There then needs to be a best effort made to determine an acceptable air lead concentration. Once that is determined the Agency could assess whether there are significant national exposures that require an NAAQS to address. There is in this draft report a suggestive bias toward supporting a revocation of the NAAQS. This seems premature, as there is little in the analyses, to this point, suggesting what might be a "safe" or, at least, an acceptable ambient air lead level.

Pb-related welfare effects and screening level ecological risk assessment (Chapter 6):

I defer to other Committee members comments regarding Chapter 6.

Technical Comments

Consideration of Uncertainty: The basic elements of the risk assessment methodology presented can be described by the breakdown of the procedures employed in the documents. Those generally include i) identifying and quantifying the sources, ii) using empirical models to estimate media concentrations, iii) estimating exposures via intake calculations, iv) predicting typical blood lead levels, v) estimating outcome blood lead distributions, vi) predicting the health effect, and vii) partitioning the effect between "policy relevant and background" sources.

The report recognizes that there are several difficulties inherent in both estimating and quantifying outcomes within each step and in utilizing those results in subsequent steps. These models also encompass a combination of non-linear relationships that provide input to the following steps. As a result, errors can propagate and this must be recognized in the uncertainty analyses. However, the effects are often multiplicative, meaning that, although uncertainty increases, the consequences of under-estimation or over-estimation can be dissimilar, and are dependent on the status of other variables in the formulations.

As a result, it is important to thoroughly examine and discuss uncertainty and the potential for error in developing and assessing these models. It could enhance the uncertainty analyses by framing the discussions in terms of Type I (false positive) and Type II (false negative) errors for the final risk assessment. Uncertainty in the risk assessment process basically produces the potential for two types of errors. Type I error is the identification of a specific chemical, area, or activity as a health concern, when in fact it is not (false positive). Type II error is the elimination of a chemical, area or activity from further consideration, when in fact, it is a potential health hazard (false negative). In risk assessment, uncertainties are generally handled conservatively (i.e., health protective choices are preferentially made) to avoid Type II errors.

For these analyses, it seems the question is whether there is significant potential for IQ loss among children exposed to the policy relevant sources? This presupposes that the Agency considers IQ loss an unacceptable outcome. There is little in the current documents regarding what the Agency considers regarding the significance of IQ loss and whether there is a threshold of concern. This discussion needs to be clearly laid out in future drafts. The Type I error (false positive) would be predicting IQ loss for children that in fact suffer no such loss. The Type II error (false negative) would be predicting no loss for children that do indeed suffer a decrement. The consequences of the Type I error is requiring unnecessary regulation for the source(s) and damaged children is the outcome for the Type II error.

Each step in the risk assessment process can be evaluated in the context of the Type I/Type II error format and the sensitivity analysis conducted should be specified to provide insight into the likelihood of each error occurring. As a result my comments are provided by examining the main steps used and noted above:

i) identifying and quantifying the sources,

The Staff Paper provides significantly more information in this regard than was presented in the AQCD and the information seems to be a good and fair representation of the state of knowledge in the U.S. at this time. It does indicate, however, that the state of knowledge is a poor representation of true emissions and ambient air lead levels in the U.S. today. This seems particularly true around the majority of smaller source types that constitute a significant segment of overall emissions. This shortage of representative emissions data becomes acutely evident in the Pilot Risk analyses, where in several instances there are no site-specific data and there is reliance on surrogate data, which also seems in short supply. As a result, it seems inherent on the Agency to develop as complete an evaluation of the range of potential emissions associated with the types of facilities assessed in the Pilot. In the absence of real data, the Agency could

draw on its experience with regulating and evaluating emissions in similar facilities over the past thirty years.

Once this determination is made, an appropriate baseline emission rate should be identified. It may be that the current document does provide the best baseline values that can be developed. The sensitivity analysis, however, should then be conducted using some reasonable maximum estimates and a similar reduced rate, while holding all other inputs constant as was accomplished in the draft. However, it is important that both upper and lower limits be accomplished as the effects may differ in each direction due to the non-linear relationships in the models. It is important to remember that these multiplicative effects can be real and are not artifacts of the mathematical structure of the models. In the absence of "better" emissions estimates, a factor of 2 analysis might be appropriate, i.e., ½ and 2 times the baseline.

ii) using empirical models/observed data to estimate media concentrations,

There are two model efforts that are ultimately most important in estimating risk outcome and subsequently relating outcomes to a critical ambient air lead level. Those are the ambient air lead estimates and dust lead concentration models. There is a lack of air quality data, at least, in comparison to the previous NAAQS reviews and the monitors indicated in the Pilot analyses seem to be disadvantageously located. As a result, determining an appropriate standard will likely rely on modeled concentrations. The document does indicate that current models are thought to be accurate to factor of 2. This might be an appropriate ratio to use for the sensitivity analysis, by substituting increased and decreased air lead estimates into the subsequent risk assessment steps and relating the outcomes to those levels. This is as opposed to doubling and halving the dispersion model inputs. Again it is important to conduct sensitivity runs using both upper and lower limits as the outcome must be related to this input parameter.

Perhaps, most critical is the estimation of dust lead concentrations. It is interesting that despite the immense amount of work accomplished with lead dust in the last sixteen years, the same relationships developed for the 1990 analyses are employed today. These models are likely appropriate in the absence of any data, as they relate to estimating the increases in dust lead concentrations associated with increasing air and soil concentrations. These models however, at least in this reviewers experience, tend to underestimate dust lead concentrations at the much lower air and soil levels seen today. The empirical relationships were developed from databases in which the dust concentrations were overwhelmed by high air and soil lead levels associated with the point sources. Although lead associated with other sources, such as paint, was always present, it seldom showed up as significant variable in these equations. This resulted in intercept terms that failed to reflect "policy-related background sources" and under-prediction in dust lead levels as soil and air lead approached background concentrations. At the primary smelter site in Idaho (Bunker Hill Site) that underwent a similar remediation to the Missouri site, dust lead levels reached a plateau at 300-350 μg/g after completion of soil cleanup (mean soil concentrations were less than <200 μg/g post-remediation with no significant air lead remaining after smelter demolition). Dust lead surveys conducted in socio-economically similar communities with no history of lead emissions showed house dust lead levels of about 50 μg/g in homes built after 1978 and 200 μg/g in older homes. It is unlikely, at the Bunker Hill site that mean dust lead levels will decrease to below 200-300 μg/g due to residual soil and dust

contamination and the age of the housing stock. The low dust lead levels estimated for both the active primary and secondary smelters seem low to use as the baseline, but might be appropriate for the lower limit sensitivity run. The Agency should consider adjusting the baseline run to reflect higher dust concentrations and a background component indicative of older housing, and then performing sensitivity analyses at higher and lower concentrations based on a review of available data, or a "factor of 2" type analysis. If not, increasing and decreasing this background component should be one of the sensitivity analyses.

iii) estimating exposures via intake calculations,

Various intake estimates are developed as input to the bio-kinetic models. These intakes depend on the media concentrations developed above and exposure variables that ultimately determine how much lead enters the body. It is clear that under typical conditions in the U.S. today, the majority of that lead comes via the ingestion of soils and dusts, and significant efforts are directed at determining these parameters for the Pilot and Staff Paper, as discussed below. However, this should not discount the importance of other routes, i.e. diet, drinking water, maternal contribution, and other "policy relevant background" sources. In a direct corollary to how the current NAAQS was established in 1978, these sources set a foundation blood lead level. Any "policy relevant air sources" can only be accommodated between this baseline and whatever unacceptable threshold is determined for the NAAQS or other critical risk management criteria. Because typical baseline blood lead levels are of the same order of magnitude as the effect levels, (i.e., 1-3 μg/dl vs. 2-5 μg/dl) it is equally important to appropriately identify the typical values and variability in "background" levels as it is in the "policy relevant source". This seems particularly important to accomplish in the primary and secondary smelter pilot analyses, as there are reasons to believe that blood lead levels would be elevated over "non-industrially" lead impacted communities. This could be due to the age of housing stock, residual lead from historic operations impacting "baseline dust and soil concentrations", increased dietary components due to contamination of local food sources, preparation, and so on. For example, at the Bunker Hill site, despite remediation of 97% of homes in some communities, house dust and blood lead levels both remain somewhat greater than the typical "national" values reflected in the default input parameters used in these analyses. Although "post-remediation" blood lead testing and risk analyses conducted at this site show compliance with CDC criteria at Bunker Hill, these communities could not safely accommodate significant emissions from a "new" smelter, as most of the acceptable exposure is accommodated in the "post-remedial baseline". Similarly, at least the primary and, likely, the secondary smelter pilot studies should consider that higher than "typical U.S." background blood lead levels are prevalent in the communities. This possibility should be addressed either by increasing the default input values for the baseline analysis, or investigating the potential effects of this uncertainty in the sensitivity analyses.

The most critical intake estimates with respect to the "policy relevant sources" are related to the dust ingestion rates (as the uncertainty in concentration was discussed above). The overall soil and dust ingestion rate is set in the IEUBK and comparable Leggett model inputs and are appropriate values. There is uncertainty however, in the partition factor to employ (default 55:45 dust:soil). The default is appropriate for the baseline, but the use of the Bunker Hill Site partition values (von Lindern et al., Please note the lower case v) in the sensitivity analysis bears some discussion. The 40:30:30 dust:yard:community partition at Bunker Hill was empirically derived

and most critically differs from the IEUBK default assumption, in that it reflects a significant contribution from soils outside the home yard, (i.e. other soils in the community). Other yards, commercial properties, parks, playgrounds, roadsides, streets, etc. were found to contribute as much to soil ingestion and house dust lead content, as the home yard soil. This became a more obvious problem with the IEUBK default guidance as the soil remediation program advanced. Use of the clean soil values established at the child's home to describe the soil intake and soil contribution to house dust, ignored the more important (after remediation) contribution by other soils in the community to both soil ingestion and dust lead. In the Pilot Analysis and Staff Report, there are two important omissions in the determination of soil and dust intake calculations. The first omission is use of the remediated home yard soil concentration as the only soil contribution. This limits the soil contribution to the lowest possible value in the site, and ignores potential contributions to soil ingestion from other unremediated soils throughout the community. Secondly, this has a double impact in reducing the dust contribution. This minimal concentration is used in estimating dust concentrations and it is possible that the impact of community soils as portrayed in von Lindern et al. is manifested through house dust. The treatment in the pilot analysis limits this contribution to the absolute minimum.

Combined with low concentrations predicted above for dust, possible underestimation of the policy relevant background, ignoring the potential impacts of other soils in the community likely results in minimally low intake estimates for soil and dust to blood lead in the primary and secondary smelter situations.

iv) predicting typical blood lead levels,

There have reportedly been some difficulties encountered in the application of the Leggett model. As a result, any critique of the Leggett methodology will be reserved for the following draft. With respect to the IEUBK estimates, most of the factors that influence outcome blood lead levels are associated with the input variables discussed above. However, there are important considerations with respect to the absorption or bioavailability utilized. The baseline runs should use the default bioavailability unless there is compelling site-specific information to substitute. The use of the Midvale results for the Missouri site is a professional judgment and is likely appropriate in that it is similar to the default values for dust. The 42% value for soil could be applicable for a smelter situation, but perhaps high if mine wastes were predominant. It is curious, however, why dust bioavailability would be lower than soils if the sources of lead to both media are similar. The use of 18% from von Lindern et al. for the sensitivity analysis also bears some discussion. The 18% value was also derived empirically from some 5000 paired blood lead:soil/dust concentration observations over several years. It had long been noted at the site the overall dose-response relationship at Bunker Hill was reduced from that indicated by the IEUBK default parameters. However, the underlying reason for the reduced response was never clearly determined. The most plausible explanations were either lower than default bioavailability and reduced ingestion rates associated with the health intervention efforts aggressively pursued with community families. The analyses in the von Lindern et al. paper attribute the full effect to reduced bioavailability and acknowledge that decreased ingestion rates are also a likely explanation. As a result, the 18% absorption estimate is an absolute minimum value and should be regarded as the lower limit in the sensitivity analysis and uncertainty discussion. The upper limit for sensitivity analyses should likely be near 40%, reflecting the

likely predominance of lead oxides and small particles associated with the smelters and paint contributions.

v) estimating outcome blood lead distributions,

Estimating the blood lead distribution depends on the selection of the GSD value. The 1.6 selected is correctly described as appropriate for a group of children with uniform exposures. However, it is not likely that all of the variance should be ascribed to differences in individual response. The GSD reflects both individual variation and some degree of variation in the exposure variables, as well. The 1.6 value is recognized as being appropriate to situations where the degree of variation in exposure is typical of the types of scenarios being evaluated. It becomes important that in the discussion of uncertainties pursuant to the sensitivity analyses, the variances typically associated with realities of individual response and distributions of exposures, not be confused with uncertainties related to the lack of characterization data. That is, there are real variances associated with individual response and the source and exposure parameters that should be captured in the 1.6 GSD. There are unknowns associated with using average emission factors that should be addressed in the sensitivity analyses and uncertainty discussions. Some alternate GSDs, perhaps, 1.5 and 1.7 should be tested to assess the effect on outcome variables due to this selection.

vi) predicting the health effect,

There is some concern with the treatment of the "cut points" in these analyses. Assuming that there is no effect below the cut point eliminates the possibility of a Type I error as no false positive is possible. The only error result would then be a false negative. This should be pointed out in the discussions and considered in determining an acceptable level of risk or margin of safety in recommending a standard.

vii) partitioning the effect between "policy relevant and background" sources.

The methodology developed does allow determination of the relative contributions to absorbed lead and will be useful in developing a new standard or alternative risk management strategy. However, it must be recognized that there is significant variance in the background levels and certain populations and individual members of all populations will be at greater risk as a result of these background sources. It is as important to identify these at-risk populations and assess the possibility for Type II error with these groups as it is to assess uncertainties based on statistical extrapolations of mean or baseline characterizations.

Summary: A clear and transparent understanding of the policy questions that these analyses will assist in answering is of critical importance to entire procedure. There is little in the analyses, to this point, suggesting what might be a "safe" or, at least, an acceptable ambient air lead level. There needs to be a best effort made to determine an acceptable air lead concentration. Once that is determined the Agency could assess whether there are significant national exposures that require an NAAQS to address. The report needs to acknowledge the poor state of knowledge with respect to emissions and ambient air data relative to the potential standard level, and the necessity of depending on modeling and surrogate data. The report needs to complete an

evaluation of the range of potential emissions associated with the types of facilities assessed in the Pilot. The report needs to comprehensively consider and address the relationship between ambient air lead concentrations and resultant soil and, particularly, dust lead concentrations. The current methodologies likely underestimate both background and policy related house dust concentrations. All of these factors combine to make the sensitivity and uncertainty analyses an essential component of the procedure. These analyses should pay special attention to distinguishing between variability and uncertainty and discussing the potential for false positive and false negative conclusions relative the policy decisions under consideration.

Post-Meeting Comments of Ian von Lindern

Following the February 5-7 meetings, it seems that the OAQPS staff will continue to assess potential NAAQS revisions and present those in *Advanced Notice of Proposed Rule* (ANPR) process. The technical aspects of potential NAAQS development and specification, or de-listing, will be supported by the Pilot Risk Assessment activities. This differs in several respects from the original NAAQS process. Although the new procedure is endeavoring to provide the safeguards and opportunity to provide scientific advice, there seems to be two notable shortcomings that are not addressed in the current Pilot Studies.

Both of these shortcomings and the problems introduced are traced to the lack of sufficient monitoring data for sources, emissions, ambient air levels and impacts. This lack of data has evolved because EPA has failed to monitor lead in situations that were at levels considerably lower than the 1978 NAAQS. However, it seems that health impact studies conducted over the last two decades suggest that this strategy may not have been health protective. This may not have been the case had the Agency diligently monitored at the levels suggested in the 1990 proposed revision to the NAAQS. Nevertheless, the result is that the risk assessments now requisite to establish a protective strategy must rely on modeling and substantial inputs of professional judgment, experience, and protective policies. It is ironic that EPA's attempt to streamline the process may discourage, rather than solicit, this type of input.

The first deficiency considers the lack of national perspective with respect to populations impacted by potentially significant air lead levels. The reliance on the three case studies does address examples of the country's primary smelter, major secondary point sources, and emissions from roadside re-entrainment. However, it seems likely that the new level of concern will extend to lead TSP values of .25 μg/m3, or below. There are large urban areas and numerous smaller sources that are of potential significance, and poorly characterized, at these levels. Moreover, there is little presented in the AQCD, Draft Staff Paper or the Pilot Study regarding the source-receptor relationships between these sources, ambient air lead levels and populations potentially impacted. This seems to be a major omission that, if addressed in the ANPR, will not receive CASAC critique prior to vetting by policy and management. That could be remedied by including such a "national view" risk assessment as an additional analysis, but at this point it seems this most important issue will not benefit from critical scientific review.

The CASAC committee is addressing this situation by trying to provide pro-active advice regarding a critical assessment of the current standard and potential strategies for determining a protective standard for implementation. My comments in this regard are included in the sub-committee recommendations.

The second critical shortcoming has to do with developing the risk assessment with the current population residing in the Pilot Site impact areas and extending those conclusions to a national standard protective of human health. This is because, although the Pilot studies, with some modifications, may be representative of current conditions at those sites; these analyses may not reflect potential exposures that could apply at these or other locations in the future.

My comments in this regard are included below as recommendations as to how the Pilot Risk Assessment could be modified.

Potential modifications to the baseline characterizations:

Several assumptions made in individual steps in the methodology tend to underestimate background contributions to intake. The Agency should consider adjusting the baseline run to reflect higher dust concentrations and a background component indicative of older housing. Similarly, at least the primary (and possibly the secondary) smelter pilot studies should consider that higher than "typical U.S." background blood lead levels are prevalent in the communities, or would be in the absence of active health intervention programs. In these cases, the draft analyses ignore the potential impacts of aggregate soils in the communities, and underestimate dust lead concentrations through the use of regression models that likely don't extrapolate appropriately to levels that are lower than the parent database.

Potential Sensitivity Analyses

Extensive sensitivity analyses should be conducted by (as indicated above) identifying an appropriate baseline or typical model run, and then varying key components while holding others constant. However, the sensitivity runs should be accomplished using both lower and upper limit analyses. It would enhance the uncertainty analyses by framing the discussions in terms of Type I (false positive) and Type II (false negative) errors for the final risk assessment.

Baseline Blood Lead Levels: It should be recognized that there is significant variance in the background levels and certain populations and individual members of all populations will be at greater risk as a result of the background sources. It is as important to identify these at-risk populations and assess the possibility for Type II error with these groups as it is to assess uncertainties based on statistical extrapolations of mean or baseline characterizations. The potential effects of increasing and decreasing the background components should be one of the sensitivity analyses.

Baseline Emission Rates: The current analyses rely on average emission rates that may be out of date. In several instances there are no site-specific data and there is reliance on surrogate data, which also seems in short supply. It may be that the current document does provide the best baseline values that can be developed. However, the range of potential emissions associated with

the types of facilities assessed in the Pilot should be developed. The sensitivity analysis should then be conducted using some reasonable maximum estimates and similar reduced rates.

Soil Lead Levels: Possible underestimation of background soil levels for the point source communities, as discussed above, and ignoring the potential impacts of other soils in these communities could result in minimally low intake estimates for soil. As a result, soil concentrations should be varied as one of the sensitivity components.

Dust Lead Levels: Perhaps, most critical is the estimation made in the entire methodology is dust lead concentration from soil and air lead concentrations. Possible underestimation of background dust lead levels and ignoring the potential impacts of other soils in the community likely results in minimally low input variables for soil contribution to dust. Additionally, the air variable that determines this route's contribution to dust has particular uncertainties. As a result, it will be important not only to assess the effect of dust on outcome health indices, but the agency should also consider additional sensitivity analyses of the soil and air selections on dust lead.

Bioavailability: The 18% absorption estimate is an absolute minimum value and should be regarded as the lower limit in the sensitivity analysis and uncertainty discussion. The upper limit for sensitivity analyses should likely be near 40%, reflecting the likely predominance of lead oxides and small particles associated with the smelters and paint contributions.

GS*D:* Alternate GSDs, both higher and lower than the baseline 1.6 should be tested to assess the effect on outcome variables due to this selection. A substantially higher level, perhaps 2.0 should also be considered.

Alternative Standard levels: The overall process envisioned in the Pilot Assessment suggests that the final runs will be accomplished at candidate NAAQS revision concentrations. This will be a convenient method to examine prospective standard levels. However, due the lack of pertinent air quality data at critical exposure locations, these analyses ultimately rely on modeled data. Because the accuracy of model predictions is (at best) a factor of two this will present "special" uncertainty questions. The predictions drive much of the internal concentrations derived in the model will ultimately be used to determine risk. However, those same model predictions will presumably be used to determine the protective concentration for the most exposed location and then at the property line. This value will, presumably be a driver with an appropriate margin of safety as the proposed NAAQS, if adopted. As a result, it may be advisable to conduct a "last" uncertainty discussion relative to the selection of the appropriate air concentration value.

TSP/PM10 Conversion: This problem is compounded by the possible need to convert from a TSP-based risk assessment analysis to a proposed PM-10 standard. If it is proposed to develop a PM10 standard, the sensitivity to the conversion equation assumptions should be tested in a sensitivity analysis.

Prepare Intake and Uptake Tables and Partition those by Pathway

The risk assessment documents would be considerably more convenient to review if summary tables were prepared for pathway specific intake and uptake for the various model runs. These could be provided as an appendix for interested parties.

Property Line Implementation

The Standard, if and when modified, will be required to apply at the facility property lines in relation to point sources. However, if the lowest acceptable air concentration determined to protect the surrounding population, for example, is found to be .25 µg/m3 for highest exposed current resident, the air value at the property line would be considerably higher. According to the manner in which the current standard should be enforced, the least protective level that would qualify as a result of the risk assessment would apply at the property line. This would result in a margin of safety for those currently most exposed after implementation. However, this analysis may not be protective because of the non-linear inter-dependencies of air soil and dust exposures accounted for in the risk assessments. This presents the basic question, of why don't the Pilot assessments consider potentially exposed children at the property line, and use the existing population data to assess model performance. The agency should consider model runs with population blocks filled with a hypothetical childhood population.

Long-term Air-Soil-Dust Equilibrium

Determination of recontamination rates and equilibrium media concentrations for active source areas remains a major shortcoming in the overall analysis.

Comments of Ian von Lindern Regarding the Primary NAAQS

February 22, 2007

Standard Setting Considerations

Proposing a primary NAAQS for lead is a sophisticated and complex undertaking that must reflect numerous scientific, societal, technical and policy considerations. These must be integrated into comprehensive policy that reflects the best science available. In the past it has been CASAC's job to i) identify the appropriate science and ii) provide advice to EPA as to how to interpret those findings in applying policy considerations and developing a protective health strategy. It seems the CASAC's fundamental problem is that the "new" process shortcuts and endangers the effectiveness of the latter portion of this protocol. EPA managers and political appointees will now combine policy directives with their own interpretation and application of the science in a composite document that will be presented "after the fact" for scientific appraisal by CASAC concurrent with public review. This limits CASAC's role to critiquing the AQCD to identify and describe the pertinent science, but diminishes the committee's ability to judge whether the science is appropriately applied in developing the proposed NAAQS, or de-listing.

CASAC's immediate challenge, it seems, is to send a clear message to EPA regarding the perceived health significance of ambient air lead, the adequacy of the current standard, the need for revision, and how the available scientific information can and should be used. The Chair's proposal to take a pro-active stance and forward recommendations on a primary NAAQS is a positive strategy to achieve this end. This sub-group has been assigned the task of undertaking the steps of developing and proposing a revised primary standard. In going through this exercise, those issues that must be addressed in developing the standard, and the CASAC's opinion of how the science identified in the AQCD can be applied, will be illuminated for the Administrator.

There are some clear conclusions that the CASAC seems to have reached consensus on. Paul Mushak did a good job of identifying those in his earlier transmittal. A candidate list includes:

- The current NAAQS is not protective, allowing these concentrations would be poor public health policy and likely cause irreversible harm to children.

- This finding is not new. The NAAQS was regarded by CASAC in 1990 to be insufficient to protect public health. The EPA elected to pursue a policy of multi-media exposure reduction that was, given the understanding of potential health effects at that time, in large part successful in reducing childhood lead absorption throughout the country.

- Since 1990, new findings suggest that adverse effects, in particular those associated with neuro-cognitive deficits in young children, occur at lower blood lead levels than previously understood.

- Dose-response and exposure/bio-kinetic studies suggest that current ambient air lead levels in portions of the U.S. can result in elevated blood lead levels among children that are of concern. *Analyses conducted by this sub-group suggest that ambient air concentrations of .25 µg/m3 or lower could be of concern.*

- Lead is a ubiquitous, multi-media contaminant that has been traditionally regulated as a priority pollutant. Both ambient air and background concentrations in other media of sufficient magnitude to increase children's blood lead to levels of concern.

- Although air quality and emissions were appropriately monitored for the current NAAQS over the last two decades, the existing national database is insufficient to identify those populations at-risk in the country today. Failure to lower the NAAQS in 1990 resulted in abandonment of numerous monitors. As a result, the extent of exposures exceeding likely revised NAAQS levels is unknown.

- OAQPS has elected to conduct a risk assessment analyses that focus on impacts around "pilot" point and area sources. This information will be useful in estimating specific impacts in such locations, and presumably in evaluating control strategies to reduce excess exposures. However, these analyses do not address the potentially large number of children in the U.S. exposed to air lead concentrations in the potential range of a revised NAAQS.

- As a result, the CASAC is concerned that the EPA does not have a sufficient database to evaluate the efficacy of regulating lead emissions as a hazardous air pollutant and should exercise great caution in considering delisting.

Paul Mushak has laid out three potential methods to develop a primary NAAQS. This is obviously a substantial undertaking and it is unlikely that the CASAC, in its limited capacity can fully develop a revised NAAQS, particularly considering the need to integrate policy concerns. However, the exercise can illuminate those issues that require responsible consideration of the science and illustrate CASAC's understanding and recommendations regarding the use of the science in resolving those issues.

Method A

As Paul Mushak points out, this is the method employed in 1978 to set the current NAAQS and the subsequent WHO standard. It is simplified and nearly 30 years old, but has the particular advantage of being in a familiar structure and having addressed the several issues that must be considered in developing a standard. EPA's considerations were documented in the Federal Register, Vol. 43, No. 194 (46252) and the methodology essentially condensed the procedure into the selection of five key parameters. Discussing the scientific considerations that go into selecting these parameters provides a vehicle for CASAC to convey an opinion on the issues and the science that should be relied upon. Doing this in parallel to EPA's 1978 strategy, and in the context of new information developed in the AQCD, is an opportunity to convey scientific advances in each of these areas, and simultaneously provide, at least, a range of potential primary NAAQS values to support the above conclusions.

Selection of Key Parameters

In selecting values for the key parameters it is important to note that these are interrelated. Similar factors are considered in each parameter and the effects are multiplicative. The overall objective of the procedure is to accurately predict the "tail" of the population. As a result, failure to "balance" the selection, or choosing all conservative or protective parameters, will increase the probability of error.

The three attached Tables show various permutations of parameter selections in the ranges indicated by Paul Mushak and Bruce Lanphear's earlier transmittals. Their specific numbers, I believe, are highlighted in the first table along with EPA's 1978 calculation.

Maximum Blood Lead Level – This is related to lowest observable effects of concern, and previously cited CDC criteria that was 30 µg/dl in 1978 and has been 10 µg/dl since 1991. The weight of evidence in the AQCD seems to suggest that deleterious effects extend to 5 µg/dl, or below. CASAC will need to justify these values and address the applicability of CDC's current recommendations. Current candidate values are 2.5 and 5.0 µg/dl.

Protectiveness – This represents the percent of population can be protected by a reasonable standard. In 1978 EPA selected 99.5% (and a GSD of 1.3) in order to accommodate "special high risk groups within the overall population," that was large and estimated to exceed 20

million children at that time. This parameter is likely the most "policy" driven selection in the process, but there is little scientific reason to advocate a lessening of the degree of protectiveness today. However, there are, at least two additional considerations that are both related to why those children are observed in the "unprotected" portion of the tail of the blood lead distribution. The first consideration relates to factors that can't practicably be addressed by the NAAQS strategy. These children may show high blood lead levels due to exposures and sources unrelated to the air lead input, or may be the result of extreme co-factor considerations (i.e., pica-type behavior or physiological pre-disposition). With regard to the second consideration these children may be in extreme situations where several factors multiply to result in excessive blood lead levels. This is the special population that EPA elected to protect with a combination of the 99.5 % /GSD=1.3 combination in 1978. These two parameters must be selected in concert in order to encompass those groups that can be effectively addressed with the control strategy. EPA faced a similar decision in the last decade in other programs and elected to pursue a 95%/GSD=1.6 selection as a risk management goal in CERCLA programs. Based on this precedent, it seems the candidate values for protectiveness range from 95-99.5%, and include the 98%-tile WHO value.

GSD – This parameter, in concert with protectiveness, is the most important to define. The selection of the GSD is the likely reason for the difference in values presented by Bruce Lanphear and Paul Mushak in their transmittals. There are, at least, two particular problems in selecting an appropriate GSD. Those are first, defining what variance the GSD accommodates and second, the GSD for a similarly exposed population likely increases at lower blood lead levels. I find it convenient to consider the elements of variance that make up the GSD for the blood lead distribution. The GSD, as used by EPA, encompasses a basic individual response variance for those with similar internal exposure, additional variance associated with absorption and excretion that can be due to differences in pre-disposition such as nutritional status, etc.; variance due to differences in intake associated with behavior; and differences due exposure gradients. The GSD found in typical population surveys includes to some degree all of these (and perhaps others), and uses this single parameter to account for several elements of variance in blood lead response. The 1.3 value selected in 1978 was reported for the epidemic at the Bunker Hill site in Idaho and likely represents the individual response differences and lowest reasonable value. This GSD is also reflective of the dose-response relationship and the mechanisms of absorption. Exposure, pre-disposition, behavioral and absorption mechanisms in these severely exposed children were possibly saturated, masking out several potential contributors to variance, leaving individual response. Slightly higher (1.4) GSDs were noted for highly (but no so severely) exposed children in urban environments (New York City) at that time. As these high exposures and blood lead levels decreased GSDs were noted to increase, arriving at a consensus value of 1.6 for risk assessment in the 1990s, although this value is often disputed. More recent surveys, notably NHANES, show GSDs of 2.0 and greater. These large surveys likely encompass several elements of overall variance and includes those "special populations" that should be considered (in or out) in selecting the level of protectiveness. GSDs are probably higher now than in 1978 (possibly excepting the individual variance), both because lower blood lead levels can show more variance as means decrease and fluctuations are relatively more significant; and because the dose-response relationship is "steeper" at lower blood leads. This becomes important in the selection of the slope factor relating blood lead levels to air lead levels below. The selection of the slope factor must consider the shape of the dose response relationship

at current blood lead levels, as opposed to typical levels in 1978. As a result, the GSD must be selected in concert with both the level of protectiveness described above and the slope factor discussed below. Candidate values likely range from 1.3 to 2.0.

These first three parameters are largely related to protectiveness and the distribution of at-risk children in the "tail" of the population. The next two parameters are related to the mean blood lead response, and the dose-response relationship at the mean. However, the GSD must be selected to accommodate those factors used in selecting the other parameters in both categories.

Non-air Background – This parameter is most important to select as it represents the baseline or background component of lead exposure that is not amenable to reduction through the NAAQS. This value was selected as 12 µg/dl in 1978, and was believed to be based on blood lead levels in supposedly "non-air" exposed areas. It is important to note that this background level has decreased to near 1 µg/dl in the interim due to EPA and other agencies multi-media exposure reduction programs. It is also true that much of what was considered non-air in 1978, subsequently decreased due to the air lead reductions. These were likely related to secondary dust exposures resulting from what EPA acknowledged as the poorly understood air-soil-dust relationship at that time. That relationship is no better understood at today's media concentration levels. It is also important to note that the blood lead levels ascribed to background sources in 1978 were of similar or greater magnitude than the air, and in some cases exceeded acceptable criteria in the absence of significant air lead exposure. This was cited as a reason for implementing an NAAQS strategy that accounted for contributions from other sources in favor a technology-based or hazardous air pollutant standard. The same situation exists today at about $1/12^{th}$ the overall exposure level. This parameter should be carefully selected to include those typical background sources, but not those "special exposure populations" considered in the protectiveness and GSD selection. Candidate values are 1.0, 1.2, and 1.4 µg/dl. A lower level could be considered.

Blood/Air Slope – EPA selected a slope value of 2 µg/dl blood per 1 µg/m3 air lead in 1978. This was based on three studies, two controlled adult inhalation studies and one childhood epidemiologic study that was the same Bunker Hill site in Idaho study used in selecting the GSD. For reasons discussed above, both of these parameter values were derived from a severely exposed population that had absorbed almost all the lead it could accommodate. It is likely that these values represent minimums today, as the blood lead levels are much lower and the dose-response rate seems to increase as blood lead levels go below 10 µg/dl. However, in selecting the value to use in subsequent analyses, it should accommodate, and not "double count", any effect of increased dose response accounted for in the GSD selection. Candidate values are 2 used in 1978, 5 used by WHO, 10 noted in recent studies, and 20 as a maximum.

The attached Tables are my attempt to calculate the range of a revised NAAQS using the 1978 methodology and the candidate values discussed above for the five parameters. This may be of use in identifying likely values for a health protective standard. I have not had the opportunity as yet to review **Methods B and C** in similar detail. I do, however endorse the approaches and believe we should develop those, as well. In that way we may arrive at a consensus value (or range of values) from all three methods that we, as a committee, may consider protective of

public health. We can then, in our best scientific judgment, convey to the Administrator the CASAC's view of the application of science in developing this most important health protection.

Table 1: NAAQS Calculations Using the 1978 methodology and a Non-air Background of 1.0 µg/dl

NAAQS Calculations Using the 1978 methodology and a Non-air Background of 1.0 ug/dl

	Protecting 99.5% of children below 5ug/dl					Protecting 99.5% of children below 2.5ug/dl					
gsd	1.3	1.4	1.5	1.6	2	1.3	1.4	1.5	1.6	2	
mean	2.54	2.10	1.76	1.49	0.84	1.27	1.05	0.88	0.74	0.42	
air allowable	1.54	1.10	0.76	0.49	-0.16	0.27	0.05	-0.12	-0.26	-0.58	1 Background non-air
NAAQS(sl=2)	0.77	0.55	0.38	0.24	-0.08	0.14	0.02	-0.06	-0.13	-0.29	2 slope=2
NAAQS(sl=5)	0.31	0.22	0.15	0.10	-0.03	0.05	0.01	-0.02	-0.05	-0.12	5 slope=5
NAAQS(sl=10)	0.15	0.11	0.08	0.05	-0.02	0.03	0.00	-0.01	-0.03	-0.06	10 slope=10
NAAQS(sl=20)	0.08	0.05	0.04	0.02	-0.01	0.01	0.00	-0.01	-0.01	-0.03	20 slope=20

	Protecting 99% of children below 5ug/dl					Protecting 99% of children below 2.5ug/dl					
gsd	1.3	1.4	1.5	1.6	2	1.3	1.4	1.5	1.6	2	
mean	2.72	2.29	1.95	1.68	1.00	1.36	1.15	0.98	0.84	0.50	
air allowable	1.72	1.29	0.95	0.68	0.00	0.36	0.15	-0.02	-0.16	-0.50	1 Background non-air
NAAQS(sl=2)	0.86	0.65	0.48	0.34	0.00	0.18	0.07	-0.01	-0.08	-0.25	2 slope=2
NAAQS(sl=5)	0.34	0.26	0.19	0.14	0.00	0.07	0.03	0.00	-0.03	-0.10	5 slope=5
NAAQS(sl=10)	0.17	0.13	0.10	0.07	0.00	0.04	0.01	0.00	-0.02	-0.05	10 slope=10
NAAQS(sl=20)	0.09	0.06	0.05	0.03	0.00	0.02	0.01	0.00	-0.01	-0.02	20 slope=20

	Protecting 98% of children below 5ug/dl					Protecting 98% of children below 2.5ug/dl					
gsd	1.3	1.4	1.5	1.6	2	1.3	1.4	1.5	1.6	2	
mean	2.92	2.51	2.18	1.91	1.21	1.46	1.25	1.09	0.95	0.60	
air allowable	1.92	1.51	1.18	0.91	0.21	0.46	0.25	0.09	-0.05	-0.40	1 Background non-air
NAAQS(sl=2)	0.96	0.75	0.59	0.45	0.10	0.23	0.13	0.04	-0.02	-0.20	2 slope=2
NAAQS(sl=5)	0.38	0.30	0.24	0.18	0.04	0.09	0.05	0.02	-0.01	-0.08	5 slope=5
NAAQS(sl=10)	0.19	0.15	0.12	0.09	0.02	0.05	0.03	0.01	0.00	-0.04	10 slope=10
NAAQS(sl=20)	0.10	0.08	0.06	0.05	0.01	0.02	0.01	0.00	0.00	-0.02	20 slope=20

	Protecting 95% of children below 5ug/dl					Protecting 95% of children below 2.5ug/dl					
gsd	1.3	1.4	1.5	1.6	2	1.3	1.4	1.5	1.6	2	
mean	3.24	2.87	2.56	2.30	1.59	1.62	1.43	1.28	1.15	0.80	
air allowable	2.24	1.87	1.56	1.30	0.59	0.62	0.43	0.28	0.15	-0.20	1 Background non-air
NAAQS(sl=2)	1.12	0.93	0.78	0.65	0.30	0.31	0.22	0.14	0.08	-0.10	2 slope=2
NAAQS(sl=5)	0.45	0.37	0.31	0.26	0.12	0.12	0.09	0.06	0.03	-0.04	5 slope=5
NAAQS(sl=10)	0.22	0.19	0.16	0.13	0.06	0.06	0.04	0.03	0.02	-0.02	10 slope=10
NAAQS(sl=20)	0.11	0.09	0.08	0.07	0.03	0.03	0.02	0.01	0.01	-0.01	20 slope=20

Table 2: NAAQS Calculations Using the 1978 methodology and a Non-air Background of 1.4 µg/dl

NAAQS Calculations Using the 1978 methodology and a Non-air Background of 1.4 ug/dl

	Protecting 99.5% of children below 5ug/dl					Protecting 99.5% of children below 2.5ug/dl					
gsd	1.3	1.4	1.5	1.6	2	1.3	1.4	1.5	1.6	2	
mean	2.54	2.10	1.76	1.49	0.84	1.27	1.05	0.88	0.74	0.42	
air allowable	1.14	0.70	0.36	0.09	-0.56	-0.13	-0.35	-0.52	-0.66	-0.98	1.4 Background non-air
NAAQS(sl=2)	0.57	0.35	0.18	0.04	-0.28	-0.06	-0.18	-0.26	-0.33	-0.49	2 slope=2
NAAQS(sl=5)	0.23	0.14	0.07	0.02	-0.11	-0.03	-0.07	-0.10	-0.13	-0.20	5 slope=5
NAAQS(sl=10)	0.11	0.07	0.04	0.01	-0.06	-0.01	-0.04	-0.05	-0.07	-0.10	10 slope=10
NAAQS(sl=20)	0.06	0.03	0.02	0.00	-0.03	-0.01	-0.02	-0.03	-0.03	-0.05	20 slope=20

	Protecting 99% of children below 5ug/dl					Protecting 99% of children below 2.5ug/dl					
gsd	1.3	1.4	1.5	1.6	2	1.3	1.4	1.5	1.6	2	
mean	2.72	2.29	1.95	1.68	1.00	1.36	1.15	0.98	0.84	0.50	
air allowable	1.32	0.89	0.55	0.28	-0.40	-0.04	-0.25	-0.42	-0.56	-0.90	1.4 Background non-air
NAAQS(sl=2)	0.66	0.45	0.28	0.14	-0.20	-0.02	-0.13	-0.21	-0.28	-0.45	2 slope=2
NAAQS(sl=5)	0.26	0.18	0.11	0.06	-0.08	-0.01	-0.05	-0.08	-0.11	-0.18	5 slope=5
NAAQS(sl=10)	0.13	0.09	0.06	0.03	-0.04	0.00	-0.03	-0.04	-0.06	-0.09	10 slope=10
NAAQS(sl=20)	0.07	0.04	0.03	0.01	-0.02	0.00	-0.01	-0.02	-0.03	-0.04	20 slope=20

	Protecting 98% of children below 5ug/dl					Protecting 98% of children below 2.5ug/dl					
gsd	1.3	1.4	1.5	1.6	2	1.3	1.4	1.5	1.6	2	
mean	2.92	2.51	2.18	1.91	1.21	1.46	1.25	1.09	0.95	0.60	
air allowable	1.52	1.11	0.78	0.51	-0.19	0.06	-0.15	-0.31	-0.45	-0.80	1.4 Background non-air
NAAQS(sl=2)	0.76	0.55	0.39	0.25	-0.10	0.03	-0.07	-0.16	-0.22	-0.40	2 slope=2
NAAQS(sl=5)	0.30	0.22	0.16	0.10	-0.04	0.01	-0.03	-0.06	-0.09	-0.16	5 slope=5
NAAQS(sl=10)	0.15	0.11	0.08	0.05	-0.02	0.01	-0.01	-0.03	-0.04	-0.08	10 slope=10
NAAQS(sl=20)	0.08	0.06	0.04	0.03	-0.01	0.00	-0.01	-0.02	-0.02	-0.04	20 slope=20

	Protecting 95% of children below 5ug/dl					Protecting 95% of children below 2.5ug/dl					
gsd	1.3	1.4	1.5	1.6	2	1.3	1.4	1.5	1.6	2	
mean	3.24	2.87	2.56	2.30	1.59	1.62	1.43	1.28	1.15	0.80	
air allowable	1.84	1.47	1.16	0.90	0.19	0.22	0.03	-0.12	-0.25	-0.60	1.4 Background non-air
NAAQS(sl=2)	0.92	0.73	0.58	0.45	0.10	0.11	0.02	-0.06	-0.12	-0.30	2 slope=2
NAAQS(sl=5)	0.37	0.29	0.23	0.18	0.04	0.04	0.01	-0.02	-0.05	-0.12	5 slope=5
NAAQS(sl=10)	0.18	0.15	0.12	0.09	0.02	0.02	0.00	-0.01	-0.02	-0.06	10 slope=10
NAAQS(sl=20)	0.09	0.07	0.06	0.05	0.01	0.01	0.00	-0.01	-0.01	-0.03	20 slope=20

Table 3: NAAQS Calculations Using the 1978 methodology and a Non-air Background of 1.2 µg/dl

NAAQS Calculations Using the 1978 methodology and a Non-air Background of 1.2 ug/dl

	Protecting 99.5% of children below 5ug/dl					Protecting 99.5% of children below 2.5ug/dl					
gsd	1.3	1.4	1.5	1.6	2	1.3	1.4	1.5	1.6	2	
mean	2.54	2.10	1.76	1.49	0.84	1.27	1.05	0.88	0.74	0.42	
air allowable	1.34	0.90	0.56	0.29	-0.36	0.07	-0.15	-0.32	-0.46	-0.78	1.2 Background non-air
NAAQS(sl=2)	0.67	0.45	0.28	0.14	-0.18	0.04	-0.08	-0.16	-0.23	-0.39	2 slope=2
NAAQS(sl=5)	0.27	0.18	0.11	0.06	-0.07	0.01	-0.03	-0.06	-0.09	-0.16	5 slope=5
NAAQS(sl=10)	0.13	0.09	0.06	0.03	-0.04	0.01	-0.02	-0.03	-0.05	-0.08	10 slope=10
NAAQS(sl=20)	0.07	0.04	0.03	0.01	-0.02	0.00	-0.01	-0.02	-0.02	-0.04	20 slope=20

	Protecting 99% of children below 5ug/dl					Protecting 99% of children below 2.5ug/dl					
gsd	1.3	1.4	1.5	1.6	2	1.3	1.4	1.5	1.6	2	
mean	2.72	2.29	1.95	1.68	1.00	1.36	1.15	0.98	0.84	0.50	
air allowable	1.52	1.09	0.75	0.48	-0.20	0.16	-0.05	-0.22	-0.36	-0.70	1.2 Background non-air
NAAQS(sl=2)	0.76	0.55	0.38	0.24	-0.10	0.08	-0.03	-0.11	-0.18	-0.35	2 slope=2
NAAQS(sl=5)	0.30	0.22	0.15	0.10	-0.04	0.03	-0.01	-0.04	-0.07	-0.14	5 slope=5
NAAQS(sl=10)	0.15	0.11	0.08	0.05	-0.02	0.02	-0.01	-0.02	-0.04	-0.07	10 slope=10
NAAQS(sl=20)	0.08	0.05	0.04	0.02	-0.01	0.01	0.00	-0.01	-0.02	-0.03	20 slope=20

	Protecting 98% of children below 5ug/dl					Protecting 98% of children below 2.5ug/dl					
gsd	1.3	1.4	1.5	1.6	2	1.3	1.4	1.5	1.6	2	
mean	2.92	2.51	2.18	1.91	1.21	1.46	1.25	1.09	0.95	0.60	
air allowable	1.72	1.31	0.98	0.71	0.01	0.26	0.05	-0.11	-0.25	-0.60	1.2 Background non-air
NAAQS(sl=2)	0.86	0.65	0.49	0.35	0.00	0.13	0.03	-0.06	-0.12	-0.30	2 slope=2
NAAQS(sl=5)	0.34	0.26	0.20	0.14	0.00	0.05	0.01	-0.02	-0.05	-0.12	5 slope=5
NAAQS(sl=10)	0.17	0.13	0.10	0.07	0.00	0.03	0.01	-0.01	-0.02	-0.06	10 slope=10
NAAQS(sl=20)	0.09	0.07	0.05	0.04	0.00	0.01	0.00	-0.01	-0.01	-0.03	20 slope=20

	Protecting 95% of children below 5ug/dl					Protecting 95% of children below 2.5ug/dl					
gsd	1.3	1.4	1.5	1.6	2	1.3	1.4	1.5	1.6	2	
mean	3.24	2.87	2.56	2.30	1.59	1.62	1.43	1.28	1.15	0.80	
air allowable	2.04	1.67	1.36	1.10	0.39	0.42	0.23	0.08	-0.05	-0.40	1.2 Background non-air
NAAQS(sl=2)	1.02	0.83	0.68	0.55	0.20	0.21	0.12	0.04	-0.02	-0.20	2 slope=2
NAAQS(sl=5)	0.41	0.33	0.27	0.22	0.08	0.08	0.05	0.02	-0.01	-0.08	5 slope=5
NAAQS(sl=10)	0.20	0.17	0.14	0.11	0.04	0.04	0.02	0.01	0.00	-0.04	10 slope=10
NAAQS(sl=20)	0.10	0.08	0.07	0.06	0.02	0.02	0.01	0.00	0.00	-0.02	20 slope=20

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Comments on Chapter 4 of the Draft Technical Report: Estimates of Media Concentrations

Overall, this is a very well written and informative chapter that describes the results of modeling of media concentrations at three locations and compares them with measurements. The question one may ask is if the selected locations do represent the general population exposures. While the case of primary Pb smelted represents the upper level of exposure (as this is the only location exceeding the current Pb standard), the exposure near roadways is far more common in urban and suburban areas and perhaps it should be expanded further. Although TSP lead data are rather sparse, the general population exposure is probably better characterized by PM_{10} lead (especially close to roadways) and I believe that there are data available (beside Houston) that can support such evaluation. It would be also desirable to extend the analysis beyond attainment of the current NAAQS and include much lower levels, down to approximately 0.1 ug/m^3.

Specific comments/questions:

1. Section 4.1.2.4 – Air Modeling Performance Assessment for Primary Smelter (p. 4-8 to 4-10).
    a. It is evident from Table 4-4 that the model overestimates the actual ambient concentrations at close distances to the facility and underestimates at more distant locations. However, in case of dry deposition, the data in Table 4-5 indicate that the model overestimates deposition as compared to measurements (although text on page 4-8, line 28-29 says something opposite). Yet, the overall conclusion on page 4-9, line 1-2 is that "modeled concentrations and depositions are fairly representative of conditions over the last 5 years based on comparison with the monitors". This statement seems to be overly optimistic.
    b. One of the possible reasons for the underestimation of Pb ambient concentrations by the model could be a severe underestimation of resuspension of deposited lead
    c. The lack of a proper representation of Pb particle size distribution could be a reason for deposition differences between modeling and measurements
    d. The footnote to Table 4-4 on p. 4-9 says that all non-detects were assigned half of the detection limit for averaging. The document does not specify the MDL, but this may elevate artificially the averages.

2. In the case of secondary smelter (section 4-2, page 4-21) the modeled period was from 1997 to 2000. For the primary Pb smelter, this was 2000-2005. Were emission data not available for a more recent period? The footnote on p 4-21 says that in 2003 the quarterly average Pb concentration at one monitor exceeded the current NAAQS, so the 1997 – 2000 period may not represent current conditions.

3. Air modeling performance assessment for secondary smelters (section 4.2.2.4) indicates 3 times lower modeled concentrations than monitored values at the monitor locations. The potential differences between actual meteorological conditions and used in the model are cited as a possible explanation, but this explanation is not convincing. Again, was the resuspension properly accounted for in the model?

4. Incidentally, the modeled soil concentrations are also 3 times lower than measured soil concentrations at similar facility (page 4-29). So, the modeled results were scaled up 3 times (this procedure was called "hybrid concentrations"). But this scale-up was not done for ambient concentrations – this seems to be inconsistent.

5. Houston was selected as a representative site for the "near roadway" scenario. For characterization of "zone of influence" for the near-roadway location the modeling results for diesel PM were used (page 4-33). Since both diesel and gasoline vehicle (main source of lead) PM are very small (below 1 um) this seems to be a reasonable assumption.

Comments on Chapter 2 of the OAQPS Lead Staff Paper: Characterization of Ambient Lead

Chapter 2 is very well written and provides ample information regarding Pb emissions, concentrations and fate. However, it seems to be a little disconnected from the Technical Report. While the Chapter 4 of the Technical Report deals mostly with modeling, Chapter 2 of the Staff Paper states flatly on pages 2-46 to 2-47 that the Gaussian dispersion models generally underestimate ambient concentrations and are not useful for national scale exposure assessments. However, due to the sparse monitoring data, the modeling is a necessary tool to estimate the exposure of general population. What is the reason that the models are not good enough? Are the emission data inadequate? Or are there some other reasons? No explanation is offered.

Answers to specific charge questions:

1. **To what extent are the emissions and air quality characterizations and analyses clearly communicated, appropriately characterized and relevant to the review of the primary and secondary lead NAAQS?**

Chapter 2 relays primary on the EPA National Emission Inventory of 2002 in describing the major emission sources of lead. The Pb Criteria Document expressed the opinion that the inventory data are inadequate. Chapter 2 of SP does not comment on quality of these data. However, the authors mention on page 2-7 that many Pb sources are not reflected in the national emission inventory, including resuspension of the road dust, Pb present in vehicle emissions from lubricating oil and from traces of Pb in gasoline, break wear, etc. These sources are the most relevant as far as general population is concerned. After all, the majority of population resides in cities, not in the vicinity of primary and secondary smelters.

2. Does the information in Chapter 2 provide a sufficient ambient Pb-related basis for the exposure, human health and environmental effects, health risk assessment and environmental assessment presented in later chapters?

The information presented in this chapter emphasizes that in general, ambient levels of Pb are much lower than the current standard. However, due to the lack of information as to what might be a safe or, at least, an acceptable ambient air lead level, the distribution of ambient lead levels across the country should be better characterized. In this regard, I find the histogram presented by Dr. Joel Schwartz in his comments more informative than the box and whisker plots in Chapter 2. Although the STN data used by Dr. Schwartz are for lead concentrations in $PM_{2.5}$, thus they may represent the lower end of exposure, they seem to be relevant to traffic-related sources. This Chapter needs to emphasize the poor state of ambient air data relative to the potential standard level, which necessitate the dependence on modeling and surrogate data.

The TSP Pb measurement method is rather outdated and the data are sparse, thus the effort should be made to replace it with PM_{10}. In this regard more information about correlations between Pb concentrations in different particle sizes would be highly beneficial.

A few minor editorial comments**:**

1. Page 2-12, line 24. A period is missing after "metal".
2. Page 2-21, line 6: Not all analytical techniques for Pb measurements require filter extraction (e.g., XRF).
3. Page 2-23, line 14-16. I'm not sure if the statement that the decline in the number of Pb NAAQS compliance sites was due to the need to fund $PM_{2.5}$ and ozone monitoring sites, is fully justified.
4. Page 2-29, line 15. What is a "composite" form?
5. Page 2-43. line 3: 2.4.2.3
6. Page 2-49, line 26: remove one of the "as well"

NOTICE

This report has been written as part of the activities of the U.S. Environmental Protection Agency's (EPA) Clean Air Scientific Advisory Committee (CASAC), a Federal advisory committee administratively located under the EPA Science Advisory Board (SAB) Staff Office that is chartered to provide extramural scientific information and advice to the Administrator and other officials of the EPA. The CASAC is structured to provide balanced, expert assessment of scientific matters related to issue and problems facing the Agency. This report has not been reviewed for approval by the Agency and, hence, the contents of this report do not necessarily represent the views and policies of the EPA, nor of other agencies in the Executive Branch of the Federal government, nor does mention of trade names or commercial products constitute a recommendation for use. CASAC reports are posted on the SAB Web site at: http://www.epa.gov/sab.