UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

July 26, 2006

EPA-CASAC-CON-06-006

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

Subject: Clean Air Scientific Advisory Committee (CASAC) Lead Review Panel's Consultation on EPA's Draft *Analysis Plan for Human Health and Ecological Risk Assessment for the Review of the Lead National Ambient Air Quality Standards*

Dear Administrator Johnson:

EPA's Clean Air Scientific Advisory Committee (CASAC), supplemented by subject-matter-expert Panelists — collectively referred to as the CASAC Lead Review Panel ("Lead Panel") — met in a public meeting held in Durham, NC, on June 29, 2006, to conduct a consultation with staff from EPA's Office of Air Quality Planning and Standards (OAQPS), within the Office of Air and Radiation (OAR), on EPA's *Analysis Plan for Human Health and Ecological Risk Assessment for the Review of the Lead National Ambient Air Quality Standards* (Draft, May 31, 2006).

The SAB Staff Office has developed the consultation as a mechanism to advise EPA on technical issues that should be considered in the development of regulations, guidelines, or technical guidance before the Agency has taken a position. A consultation is conducted under the normal requirements of the Federal Advisory Committee Act (FACA), as amended (5 U.S.C., App.), which include advance notice of the public meeting in the *Federal Register*.

As is our customary practice, there will be no consensus report from the CASAC as a result of this consultation, nor does the Committee expect any formal response from the Agency. The current CASAC roster is attached as Appendix A of this letter, and the CASAC Lead

Review Panel roster is found in Appendix B. EPA's charge to the Panel is contained in Appendix C to this letter, and Panelists' individual written comments are provided in Appendix D.

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 – Charge to the CASAC Lead Review Panel

Appendix D – Comments from Individual CASAC Lead Review Panelists

Appendix A – Roster of the Clean Air Scientific Advisory Committee

U.S. Environmental Protection Agency
Science Advisory Board (SAB) Staff Office
Clean Air Scientific Advisory Committee (CASAC)

CHAIR

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

MEMBERS

Dr. Ellis Cowling, University Distinguished Professor-at-Large, North Carolina State University, Colleges of Natural Resources and Agriculture and Life Sciences, North Carolina State University, Raleigh, NC

Dr. James D. Crapo, Professor, Department of Medicine, National Jewish Medical and Research Center, Denver, CO

Dr. Philip Hopke, Bayard D. Clarkson Distinguished Professor, Department of Chemical Engineering, Clarkson University, Potsdam, NY

Dr. Frederick J. Miller, Consultant, Cary, NC

Mr. Richard L. Poirot, Environmental Analyst, Air Pollution Control Division, Department of Environmental Conservation, Vermont Agency of Natural Resources, Waterbury, VT

Dr. Frank Speizer, Edward Kass Professor of Medicine, Channing Laboratory, Harvard Medical School, Boston, MA

Dr. Barbara Zielinska, Research Professor, Division of Atmospheric Science, Desert Research Institute, Reno, NV

SCIENCE ADVISORY BOARD STAFF

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Appendix B – Roster of the CASAC Lead Review Panel

U.S. Environmental Protection Agency
Science Advisory Board (SAB) Staff Office
Clean Air Scientific Advisory Committee (CASAC)
CASAC Lead Review Panel

CHAIR

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

MEMBERS

Dr. Joshua Cohen, Faculty, Center for the Evaluation of Value and Risk, Institute for Clinical Research and Health Policy Studies, Tufts New England Medical Center, Boston, MA

Dr. Deborah Cory-Slechta, Director, University of Medicine and Dentistry of New Jersey and Rutgers State University, Piscataway, NJ

Dr. Ellis Cowling*, University Distinguished Professor-at-Large, North Carolina State University, Colleges of Natural Resources and Agriculture and Life Sciences, North Carolina State University, Raleigh, NC

Dr. James D. Crapo [M.D.]*, Professor, Department of Medicine, National Jewish Medical and Research Center, Denver, CO

Dr. Bruce Fowler, Assistant Director for Science, Division of Toxicology and Environmental Medicine, Office of the Director, Agency for Toxic Substances and Disease Registry, U.S. Centers for Disease Control and Prevention (ATSDR/CDC), Chamblee, GA

Dr. Andrew Friedland, Professor and Chair, Environmental Studies Program, Dartmouth College, Hanover, NH

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

Dr. Bruce Lanphear [M.D.], Sloan Professor of Children's Environmental Health, and the Director of the Cincinnati Children's Environmental Health Center at Cincinnati Children's Hospital Medical Center and the University of Cincinnati, Cincinnati, OH

Dr. Samuel Luoma, Senior Research Hydrologist, U.S. Geological Survey (USGS), Menlo Park, CA

Dr. Frederick J. Miller*, Consultant, Cary, NC

Dr. Paul Mushak, Principal, PB Associates, and Visiting Professor, Albert Einstein College of Medicine (New York, NY), Durham, NC

Dr. Michael Newman, Professor of Marine Science, School of Marine Sciences, Virginia Institute of Marine Science, College of William & Mary, Gloucester Point, VA

Mr. Richard L. Poirot*, Environmental Analyst, Air Pollution Control Division, Department of Environmental Conservation, Vermont Agency of Natural Resources, Waterbury, VT

Dr. Michael Rabinowitz, Geochemist, Marine Biological Laboratory, Woods Hole, MA

Dr. Joel Schwartz, Professor, Environmental Health, Harvard University School of Public Health, Boston, MA

Dr. Frank Speizer [M.D.]*, Edward Kass Professor of Medicine, Channing Laboratory, Harvard Medical School, Boston, MA

Dr. Ian von Lindern, Senior Scientist, TerraGraphics Environmental Engineering, Inc., Moscow, ID

Dr. Barbara Zielinska*, Research Professor, Division of Atmospheric Science, Desert Research Institute, Reno, NV

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* Members of the statutory Clean Air Scientific Advisory Committee (CASAC) appointed by the EPA Administrator

Appendix C – Charge to the CASAC Lead Review Panel

Within each of the main sections of EPA's draft *Analysis Plan for Human Health and Ecological Risk Assessment for the Review of the Lead National Ambient Air Quality Standards*, we ask the CASAC Lead Panel members to focus their review on the following questions:

Human Exposure and Health Risk Assessment:

Do the Panel members have any general comments on the approach presented in the analysis plan for completing the human health exposure and risk assessment?

Exposure Assessment (estimating media concentrations)

1. The draft plan describes the use of ambient monitoring data combined with a range of modeling tools to characterize lead concentrations in ambient air across modeled study areas under specific NAAQS scenarios (*e.g.*, current conditions, current NAAQS, alternate NAAQS).

   a. What are the Panel members' views on the general approach of using monitor data when sufficient, augmented by air quality modeling as needed in particular study areas to characterize air quality?

   b. What are the Panel members' views regarding the use of source-apportionment to estimate contribution to ambient concentrations by particular emissions categories (*e.g.*, reentrainment vs. ongoing point-source emissions)?

   c. Do the Panel members have any comments regarding the options presented in the plan for estimating re-emission of historically deposited lead (reentrainment)?

2. The draft plan describes the use of available soil measurements (either site-specific or obtained from a similar surrogate site), combined with a range of approaches, including modeling tools to characterize soil lead concentrations in modeled study areas associated with specific NAAQS scenarios (*e.g.*, current conditions, attaining the current NAAQS, alternate NAAQS).

   a. What are the Panel members' views on the general approach and tools being considered for use in the assessment?

   b. Do the Panel members have any comments regarding the use of a simple soil reservoir model to predict soil concentrations of lead associated with different air quality scenarios?

   c. Furthermore, what are the Panel members' views regarding the alternative use in the full-scale assessment of a more sophisticated dynamic, mass-conserving fate and transport model such as TRIM.FaTE to predict soil concentrations for different air quality scenarios?

   d. Do the Panel members have any comments on the magnitude of the simulation period duration required to identify changes in soil given changes in ambient air concentrations and related deposition?

3. The draft plan describes the use of a similar approach to that used in the IEUBK blood lead model for predicting indoor dust concentrations (*i.e.*, depending on contributions from outdoor soil lead, outdoor ambient air lead, and other sources).

a. Do the Panel members generally agree with using this approach?

b. Furthermore, the staff is planning to consult the literature (*e.g.*, as cited in the Lead AQCD) to obtain updated outdoor *soil-to-indoor dust* and *ambient air-to-indoor dust* factors. Do the Panel members have any comments regarding estimates for the outdoor *soil-to-indoor dust* and *ambient air-to-indoor dust* factors?

4. The draft plan describes various sources of information for characterizing background lead exposures (*e.g.*, diet, drinking water, lead paint). Do the Panel members have any recommendations for additional or preferred sources of information?

Exposure Assessment (blood lead modeling)

1. The draft plan describes the potential use of multiple models for estimating child and adult blood lead levels.

a. What are the Panel members' views regarding the suite of blood lead models being considered for children, and their potential combined use to inform model uncertainty?

b. Do the Panel members generally agree with the preference the plan assigns to the IEUBK model for children's blood level estimates?

c. Similarly, for adults, what are the Panel members' views regarding the suite of models being considered for estimating adult blood lead levels?

d. What are the Panel members' views on the staff's plans for deriving a geometric standard deviation for blood lead in adults and children which reflects the latest information on inter-individual variability in exposure factors and biokinetics?

e. What are the Panel members' views regarding the option of employing Monte Carlo methods to develop exposures for a set of simulated individuals reflecting variability in exposure factors that are then used to generate a distribution of blood lead levels for that simulated population via a blood lead model?

Effects Assessment and Risk Assessment

1. Do the Panel members generally agree with the three main endpoints being considered for quantitative risk assessment: IQ (children) and blood pressure and renal effects (both for adults)?

2. In considering the assessment of risks to children:

a. What are the Panel members' views on the set of studies the staff is considering for use in estimating childhood IQ decrement associated with blood lead levels, and the preference assigned to Lanphear *et al.* (2005)?

b. Furthermore, do Panel members have any comments or advice regarding the types of models (linear, non-linear) to be used to reflect individual study findings on IQ loss for children at exposure levels below 10 µg/dL (concurrent measurement)?

c. Regarding the specific blood lead level metrics to use in modeling child IQ, do the Panel members have any recommendations regarding the metrics (*e.g.*, lifetime-averaged, concurrent) being considered for use with specific studies?

3. In considering the assessment of blood pressure effects in adults:

   a. What are the Panel members' views on the set of studies the staff is considering for use in estimating blood pressure effects in adults, and the preference assigned to Nawrot *et al.* (2002)?

   b. Furthermore, do the Panel members have comments or advice regarding the nature of the model (*e.g.*, linear, log-linear) which should be used for this endpoint?

4. In considering assessment of renal effects in adults, what are the Panel members' views on the set of studies the staff is considering for use in modeling this endpoint?

5. What are the Panel members' views on the use of the same blood level metric (*e.g.,* average blood lead during adult exposure period) for both adult endpoints?

6. Do Panel members generally agree with the various risk measures that staff is considering?

Uncertainty and Variability Assessment

The analysis plan describes the use of an integrated approach for addressing uncertainty and variability associated with exposure and risk estimates generated in this assessment. This integrated approach combines sensitivity analysis techniques (for addressing model and parameter uncertainty) and probabilistic techniques (for addressing exposure-related variability).

The integrated approach will use a modeling options "tree" to represent the different combinations of modeling options under consideration with the maximum, minimum and central-tendency branches being identified and subject to more intensive analysis including consideration for variability (through probabilistic simulation) and parameter uncertainty (through sensitivity analysis techniques).

   a. What are the Panel members' views concerning this approach of combining sensitivity analysis methods (for examining uncertainty) with probabilistic simulation (to consider variability)?

   b. Specifically, what are the Panel members' views regarding the approach of identifying maximum, minimum and central-tendency modeling branches for focused analysis regarding uncertainty and variability?

   c. Do the Panel members have any recommendations regarding specific sources of uncertainty and/or variability that should be included in this examination of uncertainty and variability?

Ecological Risk Assessment:

Do the Panel members have any general comments on the approach presented in the analysis plan for conducting the ecological risk assessment? Do Panel members think that uncertainty and variability are adequately characterized? Are there additional ways to characterize uncertainty and variability that should be considered as part of this assessment?

Data Sources for Determining Media Concentrations

The analysis plan describes the variety of data sources available for determining current levels of lead in air, soil, freshwater, and sediment.

a. Are there other datasets (air, soil, water, sediment) not mentioned that should be considered for the analysis (either national or region in scale)?

b. Does the age of the USGS soil data present a significant concern to the analysis?

c. What are the Panel members' opinions on using regional or national-scale data to estimate media concentrations at study locations without site-specific data?

d. Do the Panel members feel that sediment data as described in the plan are useful for this analysis, given that it is not available for all regions of the country and is not standardized?

Case Study Selection

The draft plan describes the criteria and process by which case study locations will be selected.

a. What do the Panel members feel are the most important ecological criteria for establishing case study locations?

b. What low-level exposure scenarios might be important to consider if additional case study locations are chosen for tier 2?

TRIM.FaTE Model

The draft plan discusses the use of the TRIM.FaTE model for estimating future media concentrations.

a. Do the Panel members feel that either MPE or TRIM.FaTE is more appropriate for determining future media concentrations?

b. In the Panel members' opinions, is ten years an appropriate time step for future media concentrations for the model under the current ambient air scenario?

Ecotoxicity Screening Values

The analysis plan discusses ecotoxicity screening values for soil, freshwater, and sediment and their uses in this assessment.

a. Do the Panel members feel this is an appropriate use for screening values given the larger scale application?

b. Which option regarding bird Eco-SSL values do the Panel members feel is most appropriate?

c. Do Panel members feel that the current AWQC are useful given that they do not currently factor in pH but are based on water hardness?

d. Given the current state of development of the sediment criteria, is it appropriate to use them in this analysis?

Modeling of Intake Rate or Body/Tissue Concentration

1. Do the Panel members have any additional input into modeling intake rates for susceptible receptors?

2. Do the Panel members feel that it is more appropriate to use NOAELs or LOAELs for comparing concentration effects?

Appendix D – Comments from Individual CASAC Lead Review Panelists

This appendix contains the preliminary and/or final written 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 consultation 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. Panelists providing written comments are listed on the next page, and their individual comments follow.

Panelist **Page #**

Dr. Deborah Cory-Slechta

Comments on 5.0 Effects Assessment in Analysis Plan for Human Health and Ecological Risk Assessment for the Review of the Lead National Ambient Air Quality Standards

p. 32, 5.2.1., the '(note, the current approach does not call for conducting adjustments for covariates for individual case study populations)' should include an explanation of why this is the case to lead the reader through the full logic.

Also, there is no mention made of the basis of the choice of the studies for consideration of IQ in children that are included in Table 5-1.

The same situation applies to the choice of endpoints for blood pressure effects in adults as well as for renal effects in adults.

Dr. Ellis Cowling

Please note that pages 1 and 2 of these Individual Comments were prepared on June 25, 2006, prior to the CASAC Peer Review and Consultation on June 28-29, 2006, and that pages 3-6 of these comments were prepared on July 6-11, 2006 in light of the very valuable discussions during the CASAC Peer Review and Consultation on June 28 and 29, 2006.

Individual Comments prepared before the
CASAC Peer Review of the Second External Review Draft Criteria Document on Lead
and the CASAC Consultation on the Analysis Plan for Human Health and Ecological Risk
Assessment for the Review of the Lead National Ambient Air Quality Standards

The most impressive general conclusion in the First and Second External Review Drafts of the Lead Criteria Document is 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 the past century earlier decades were very substantial indeed.

Total lead cumulative deposition of lead in the United States during the 20th Century is estimated to be 1-3 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.

Recognizing this reality, the consensus statement regarding Environmental Effects of Lead prepared by CASAC Members Cowling and Poirot and by CASAC Lead Panelist Friedland, Luoma, and Newman for inclusion in the CASAC Letter to EPA Administrator Johnson regarding Chapter 8 in the First External Review Draft for Lead recommended that:

> "The information in chapter 8 needs to be presented in a way that is more directly relevant to the issue of whether the Administrator of EPA should retain, increase, or decrease the present primary and secondary National Ambient Air Quality Standards for lead. Since secondary standards are often (neglectfully) set equal to primary standards, a key question is whether there are environmental effects that occur at lead concentrations lower than, or for indicators, forms, or averaging times different from, those that effect human health."

In my opinion, this same general comment applies equally well to the revision of Chapter 8 in the Second External Review Draft and also to the "Ecological Risk Assessment" part of the "Analysis Plan … for the Review of the Lead National Ambient Air Quality Standards."

The newly prepared Executive Summary in the Second External Review Draft, and Chapter 8 in both the Second as well as the First External Review Draft of the Criteria Document for Lead, needs to more clearly indicate how any continuing environmental effects of lead might respond to changes in current and future atmospheric lead emissions, concentrations, or deposition. A further revised Chapter 8 would better help USEPA prepare for such changes if it included a more complete and/or balanced analysis of the status of new advances in the science relevant to environmental management of lead. For example, consideration of monitoring needs and the implications of dietary exposure and trophic transfer are needed, as is more balance in considering equilibrium partitioning in sediments. Some improvement in the discussion and suggested uses of the biotic ligand model are evident in the revision of Chapter 8 for the Second External Review Draft. But it also would be useful if this Chapter were to consider how environmental effects of historically deposited lead or future increases in deposition (if current laws are relaxed) might be modified by land-use changes, or soil amendment treatments, or interactions with other pollutants including other metals or acidifying pollutants, or with changes in climate and climate processes."

Members of CASAC were especially pleased to see the relatively thorough discussion at the end of Chapter 8 in the First External Review Draft regarding the alternative concepts of critical loads, critical limits, target loads, and target times that have been developed in European and Canadian scientific literature 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 United States, we believe, together with the authors of the National Research Council/National Academy of Sciences 2004 report on "Air Quality Management in the United States," that these alternatives should be considered very carefully as air quality management tools for use in this country as well.

The further revised discussion of the Critical Loads concept and its strengths and limitations in Chapter 8 in the Second External Review Draft indicates that progress in American thought about these aspects of environmental management are moving in constructive directions.

In summary, 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.'"

In the case of the current Criteria Document and the associated "Analysis Plan" document for lead, it seems clear that it is not just the present ambient concentration of lead that is emitted into the air by contemporary lead emissions sources that is hazardous to the present and future health and productivity of terrestrial and aquatic ecosystems, but rather, in very large part, the fraction of the historically deposited lead that is redistributed. Thus, maintaining a Secondary (public-

welfare based) NAAQS that is equivalent to the Primary (public-health based) NAAQS, and thus aims only to manage current air concentrations of lead by decreasing contemporary emissions of lead instead of processes and procedures that decrease the redistribution of historically deposited will not provide satisfactory protection of terrestrial and aquatic ecosystems from risks of exposure to lead.

Individual Comments prepared on July 6 and 7 in light of valuable discussions during the CASAC Peer Review of the Second External Review Draft Criteria Document on Lead and CASAC Consultation on the Analysis Plan for Human Health and Ecological Risk Assessment for the Lead National Ambient Air Quality Standards on June 28-29, 2006

The most impressive result of the valuable discussions that occurred during the CASAC Peer Review of the Criteria Document and the Consultation on the Staff Paper on lead is the almost complete absence of discussion in both the Second External Review Draft of the Criteria Document (including the Integrative Synthesis Chapter and the Executive Summary) and the Analysis Plan for Human Health and Ecological Risk Assessment for the Lead National Ambient Air Quality Standard about the following four science and policy-relevant issues:

1) The many unique features of lead as a Criteria Pollutant compared to the other gaseous pollutants for which air concentrations and exposures are the primary mode of action in inducing both human health and human welfare effects,
2) How the Identical Primary and Secondary National Ambient Air Quality Standards for lead established very early (in 1977) by the USEPA have remained unchanged since 1977 despite the significant review of the NAAQS for lead that occurred in 1990 and 1991,
3) How different the contemporary standards or targets for lead pollution established by the World Health Organization and some other countries of the world are from those established by the USEPA for use in the United States, and
4) How many members of CASAC are coming to realize that continuing to adopt identical Primary and Secondary National Ambient Air Quality Standards for Criteria Pollutant involves a (usually unstated) assumption regarding policies and procedures appropriate for protection of human health and the environment. In fact, policies and procedures appropriate for protection of human health and human welfare (the latter including both ecological and other welfare effects such as visibility) may not be as effective (or even well-justified scientifically) for protection of the environment (including ecological, visibility, and other human welfare effects.

This last statement often is true either because:

a) There are ecologically important living organisms that are even more sensitive to some criteria pollutants than human beings, and/or
b) The mode of ingestion, mechanism of action, or other features of some pollutants may be sufficiently different from that of other pollutants that a different level (air concentration), indicator, form, or averaging time for a National Ambient Air Quality Standards – or even an entirely different approach (such as critical loads

or levels) may be important and thus should be considered even more thoroughly in the Criteria Documents and policy-focused Staff Papers prepared by the USEPA.

In summary, I hope that these four limitations of the Ecological Effects Chapter (Chapter 8), the Integrative Synthesis Chapter, and the Executive Summary in the Second External Review Draft of the Criteria Document, and the initial Analysis Plan for Human Health and Ecological Risk Assessment for the Lead National Ambient Air Quality Standards will be considered in preparing the final drafts of these important documents – even in the limited time that is available before the court-ordered deadline for completion of these documents – but especially in the future in developing other Criteria Documents and Staff Papers or other scientific assessment and policy assessment documents for other Criteria Pollutants.

With regard to the second and third issues listed above, 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).
>
> <u>The 1990 CASAC Report on the NAAQS</u>
>
> 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.

The 2000 (second edition) WHO-Europe "Air Quality Guidelines for Europe" took an even more quantitative approach, which permitted a single, low air lead guideline to be selected, a guideline value at the lower end of the previous range given in 1987. Elements of the recommendation in the Guidelines update for air lead included 1) derivation of a guideline value based on a Pb-B level of 10 µg/dl in young children; 2) lead ingestion as well as lead inhalation are important for young children; 3) an air lead value of 1.0 $\mu g/m^3$ translates via direct and indirect (dust/soil/diet) pathways to a Pb-B of at least 5 µg/dl; 4) 98% of young children should have a Pb-B that does not exceed 10 µg/dl; 4) this translates to the median Pb-B not exceeding 5.4 µg/dl. All of this, plus factoring in the non-air inputs to children's Pb-B levels, works out to the air lead not exceeding 0.5 $\mu g/m^3$ and this value was the recommended Guideline.

World Health Organization. 2000. Air Quality Guidelines for Europe. Second Edition. Lead. Ch. 6.7. WHO Regional Bureau for Europe, Bilthoven, The Netherlands, pp. 149-153.

If CASAC wishes the relevant sections of these two WHO documents, they presumably are in the EPA docket for the current process. Otherwise, I would be happy to provide them.

Dr. James Crapo

Chapter 3 of the analysis plan for human health and ecological risk assessment provides an appropriate overall general approach to this problem. The case study approach, initially focusing on three cases to represent particular types of ambient lead emissions and exposure scenarios, is excellent. 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.

It is clear that the most extensive, complete data sets available to the EPA for risk assessment purposes are those regarding IQ decrements and neurobehavioral endpoints for blood lead levels in children. These data sets are robust, appropriate for modeling for risk assessment, and most relevant to current lead exposures. Although highly quantitative, the magnitude of change in blood pressure effects is low and its clinical significance questionable. Renal effects in adults also have substantial uncertainties that reduce the value of this endpoint for risk modeling. It is recommended that for pilot analysis, the EPA place its primary focus on modeling IQ loss for children. An additional case study that should be considered is modeling the effects of soil in and around residential dwellings since this can serve as a direct source of oral lead exposure for children.

The modeling approach needs to consider the environmental lead burden both in terms of its historical accumulation and current lead uses that contribute to the lead burden both through air and water emissions. Modeling the effects of changes in one of the sources of lead production into the environment or possible lead removal from the environment needs to be done to determine the feasibility and impact of various approaches to control adverse health effects from lead.

Dr. Bruce Fowler

BAF 7/706

Bruce Fowler Comments on Analysis Plan for Lead Risk Assessment Draft

As discussed at our meeting on the 7/ 28-29 / 06, it seems very clear that the most extensive / complete data sets available to EPA for risk assessment purposes are those regarding IQ decrements and neuro- behavior endpoints for blood lead values most commonly encountered among children in the general U.S. population. These data sets are more robust than those related to alterations in renal function or blood pressure which occur among adults at higher blood lead levels. For these reasons, the documented neurotoxicity of lead among children at low blood lead levels would seem to be the most sensible and reasonable data upon which to conduct risk assessments for lead. The neurotoxic effects of lead are also supported on a mechanistic basis by data from experimental animal model system studies such that mechanism of action (MOA) risk assessments may be possible to at least some degree.

Dr. Andrew Friedland

Andy Friedland, Dartmouth College
6 July 2006

Individual input for letter
Comments on Draft Lead Risk Assessment Plan

I am pleased with the draft text circulated by Ellis Cowling regarding comments to be included in the letter from the panel regarding the 2nd draft Lead AQCD. I still maintain (as I did in my submission in June 2006) that there needs to be more detailed information within Chapter 8 on the present-day sources of Pb to terrestrial ecosystems.

Regarding the OAQPS Analysis Plan for Lead Risk Assessment, Draft 05/31/06, I suggest that the authors rethink the reasoning behind the third paragraph on page 51, which states:

"The USGS [soil] data primarily were collected in the 1970's and therefore were taken before the removal of Pb in gasoline; however, collection sites were far from urban areas and another point sources of Pb…..Given that Pb is very stable in soil, these data are probably still the best available for large areas of the country."

There is a recent body of literature that shows that although Pb is stable in soil, Pb-organic matter complexes are not stable and that their mobilization rates are dependent on the mobilization of organic matter. In the temperate north-central and northeast US, organic matter can have a response time of 25-50 years, which means that Pb may be mobile in the same time frame. A number of papers in Chapter 8 of the Lead AQCD review this topic and should be consulted.

Dr. Robert Goyer

Comments on Analysis Plan for Lead Risk Assessment Draft, revised July 5

Post meeting summary of comments made at meeting regarding Effects Assessment and Risk Assessment

Robert A. Goyer, M.D.

The panel was asked about the three main (health) end-points being considered for risk assessment, IQ (children) and blood pressure and renal effects (both for adults).

There is substantial epidemiological and supportive experimental evidence relating low dose lead exposure in children at the ≥5μg/dl blood lead level and the population at risk is large so this must be a high priority health end-point for the risk assessment.

Their are numerous epidemiological studies regarding the relationship of blood lead levels in adults showing a small effect on blood pressure (for each doubling of blood lead there is ~ 1.0 mmHg increase in systolic pressure). This effect has been found as plausible by animal studies and several possible mechanisms identified. However, because of the small size of the effect, it deserves a lesser priority than lead effects on IQ in children.

Chapter 6 (epidemiological studies) cites 6 studies relating measures of renal function (increase in glomerular filtration rate and/or increase in blood creatinine) to blood levels as low as 5 μg/dl for adults in the general population. These observations do not seem consistent with numerous studies of renal effects of lead in occupationally exposed workers where traditional studies do not show a lead effect below the 40-50 μg/dl level. There has been considerable experimental study regarding mechanisms for lead effects on the kidney. These studies have largely focused on functional and cytotoxic effects on the nephron (glomerulus and tubule) and have produced a number of biochemical biomarkers of renal cell injury that are used clinically to assess renal injury. These indicators of renal cell injury are reviewed in detail in Chapter 5. None of these indicators, (enzymuria, proteinuria) are reported as abnormal in the studies of adults in general population. The most plausible mechanism for the observed effect of lead in adults in the general population is some effect of lead on renal hemodynamics, (hypefiltration), which is mentioned in Chapter 6 but this is speculation. There are studies showing glomerular hyperfiltration in experimental animal with high levels of lead exposure but this has not been studied in adults exposed to lead. Given these uncertainties I do not believe that renal effect in adults observed at low blood lead levels merit significant priority for inclusion in the Risk Assessment Plan when compared to neurotoxicity in children.

Mr. Sean Hays

Comments on: **Analysis Plan for Human Health and Ecological Risk Assessment for the Review of the Lead National Ambient Air Quality Standards**

Provided by: Sean Hays
Date: June 28, 2006

These comments focus on the exposure assessment (blood lead modeling) and human health risk assessment portions of the Plan.

The "Plan" advocates the use of a series of Case Studies for assessing risks to discrete populations of concern. At first I did not see the usefulness of this approach for setting a NAAQS, but I can see the value in conducting "case studies" since the control of lead exposures has been reduced to controlling releases from discrete point sources such as smelters, etc. The challenge will be extrapolating what is learned from the case studies into a generalization of a NAAQS.

Comments on specific sections of the Plan are included below:

Exposure Assessment

Section 4.3: Estimating Blood Lead
The criticism of the O'Flaherty PBPK model is a cop out. The lead PBPK model has been well documented, as thoroughly as the other models (e.g., Leggett and IEUBK). The authors have not utilized a consistent criterion to judge the various models (biokinetic and slope factor models) for their applicability for risk assessment purposes. The agency should use consistent criteria to judge the various models (a table might be helpful). Furthermore, the PBPK model is adequate for modeling adult blood lead levels. Not including it as an option for predicting adult levels is an omission.

Risk Assessment

Section 6.1: Defining Modeled Populations
The Uniform Age cohort approach will be OK if children are the only receptor of interest for the risk assessment. The Distributed Cohort Approach is probably the most defensible approach, but it will be critical to include historical exposures for adults. However, including historical exposures will be impossible to account for accurately using the IEUBK or slope factor models. The PBPK model or Leggett models will be the only models capable of accounting for historical exposures.

Uncertainty in Dose (Blood Lead Levels)

As I commented on Chapter 4 of the AQCD, I believe the authors of the risk assessment need to appreciate the uncertainty involved in the blood lead predictions made by the various biokinetic and slope factor models. Each approach involves inherent uncertainty. The slope factor models contain large variability in blood lead predictions for given exposure media concentrations (see

Table 4-10 in AQCD) and the media concentrations that were quantified only accounted for a portion of the total variability. The slope factor models are usually only applicable for the populations and exposure scenarios (homes, certain types of lead, etc.) for which it was derived. The slope factor models have limited utility for predicting transient changes in blood lead levels associated with changes in exposure scenarios. Furthermore, uncertainty is introduced when using a slope factor model for populations for which they were not parameterized. The biokinetic models are fairly accurate and well validated for describing lead kinetics in humans (adults and children) following known doses of lead (e.g., an i.v., oral, inhalation exposure). Therefore, we have reasonable confidence in the biokinetic models to accurately predict a mean blood lead level in humans given a "dose" (e.g., in terms of µg/kg/day). We have less confidence in the ability of these models to predict an absolute level of lead in blood of humans for a complex exposure scenario (e.g., soil lead concentration, dust loading rates or concentrations of lead in house dust, concentrations of lead in air, water, food and paint chips, etc.) because we have less confidence in understanding and modeling how humans interact with their environment (e.g., contact rates, ingestion rates, inhalation rates, drinking water ingestion rate, etc.). We do have better confidence, though, in using biokinetic model to predict the relative difference in blood lead levels for two scenarios. This could be used to the authors' advantage to develop a relative (differential) risk assessment. This can be done by using observed biomonitoring levels (blood lead levels) in a case study along with measured levels of lead in environmental media to parameterize a biokinetic model (either PBPK or IEUBK) for the population of interest. Then the model can be used to simulate the change in blood lead levels for a scenario where air concentrations (and related soil and dust due to different deposition rates) change to predict the change in blood lead levels. This will raise new challenges if a dose-response model (either linear or log-linear) is used, because it will still require knowing an absolute level of blood lead to know where on the dose-response curve one lies and the resulting predicted IQ loss and confidence intervals on these estimates. To the degree that case studies can be selected that already have blood lead monitoring for the population of interest to dictate what the baseline absolute blood lead levels are, this uncertainty can be overcome.

Uncertainty in Dose-Response Relationship

The whole risk assessment approach is based on the paradigm that there is a linear or log-linear relationship between IQ loss and blood lead levels, with no lower bound on where this relationship holds true. The uncertainty in this dose (blood lead levels) - response (IQ loss) relationship, needs to be characterized, especially at low blood lead levels, for this risk assessment plan to be adequately conducted. For instance, the confidence intervals for the slope of this line needs to be broken out for fairly narrow blood lead ranges (e.g., 0-1, 1-2, 2-3, 3-4, 4-5, 5-7, 7-10, etc. – agency staff can better define what blood lead ranges are required for their risk assessment). If there is no confidence that there is an association between blood lead levels below some certain level (e.g., 5 µg/dL), then the risk assessment approach should perhaps undertake a different approach…one based on the conventional Reference Concentration (RfC) type approach. This would require the EPA staff or CASAC committee to opine on what they think is the most defensible point of departure (blood lead level below which there is no evidence for a decline in IQ). I would recommend the EPA have a statistician analyze the dose-response data of the CASAC's choice (such as Lanphear et al., 2005) to determine both a point of departure and the confidence interval of the dose-response relationship as a function of blood lead levels. This will be required for the risk assessment.

Alternative Approaches

The current risk assessment approach advocated by EPA relies on multiplying a predicted blood lead level with a dose-response curve that relates blood lead levels with IQ loss, to get an estimated IQ loss for a population. If there is not sufficient evidence to indicate blood lead levels below a certain level still cause a decrement in IQ, then it is not appropriate to utilize this "linear"[1] extrapolation method at blood lead levels below this "point of departure"[2]. To the degree that current background blood lead levels or levels associated with the case studies lie below the point of departure, then a different approach should be considered. The EPA should consider deriving a point of departure (POD) and conducting a margin of exposure (MOE) analysis for their case study. The agency will obviously be required to derive a POD for this approach. I challenge the CASAC panel to give the EPA advice on what risk assessment approach is most scientifically justified for them to incorporate in their risk assessment. Does the epidemiology studies support a decrement of IQ at all blood lead levels, even down below 1 µg/dL? If not, then at what blood lead level is there no evidence for a decrement in IQ? Can this be derived easily with the available studies, or does EPA need to conduct a NOAEL/LOAEL analysis of the data that underlie the epidemiology studies of IQ loss in children. I recommend the CASAC ask EPA to undertake this analysis so that the CASAC can provide guidance on the most defensible approach for modeling IQ deficits at low blood lead levels.

Charge Questions

Exposure Assessment

1a&b) The PBPK model should be included. The PBPK model has been developed for all life stages; children, adults, osteoporosis.
1c) The GSD is a reasonable approach. The key will be defining a defensible GSD.
1d) Monte Carlo is a reasonable approach, but it may be no more precise than defaulting to a GSD.

Effects Assessment and Risk Assessment

1. Blood pressure and kidney effects will only be important to characterize if they are more sensitive than IQ loss in children.
2. a) I defer to the epidemiologists on the panel.

b) Consider providing both; linear and non-linear. The bigger question will be are either supported by the data. Is there sufficient evidence to support no threshold for IQ loss as a function of blood lead levels?

c) If IQ loss is reversible, then use concurrent. If IQ loss is not reversible, then use peak blood levels or a surrogate such as lifetime average.

[1] The term "linear" here is used to indicate the approach of multiplying dose (blood lead levels) times a dose-response slope factor (whether it be derived using a linear or log-linear extrapolation model) to yield an IQ loss.
[2] The term "point of departure" here refers to a blood lead level below which a decrement in IQ cannot be supported by the epidemiology literature.

Dr. Bruce Lanphear

Comments on Analysis Plan for Human Health and Ecological Risk Assessment (Lanphear)

Page 2: In addition to roadways and lead smelters, another prevalent example of lead deposition from air to non-air media that should be described is "soil in and around residential dwellings." Soil that was contaminated with airborne lead can serve as a direct source of exposure via mouthing behaviors (e.g., soil ingestion) or by tracking soil into the house.

Page 6: If EPA uses the case study approach, they will quickly find that there are few (if any) communities with all of the relevant variables. To validate the IEUBK models and minimize the number of variables they will need to estimate from other sources (as described on page 13), they should consider using one of the Rochester data sets. These data sets have extensive measures of residential lead exposure, mouthing behaviors, sociodemographic factors and children's blood lead concentration. There is also air lead data available that can be used in modeling.

Page 16, Estimating health effects incidence in children:
There is substantial epidemiological and supportive experimental evidence linking low dose lead exposure in children with blood lead levels of 5 μg/dL and higher. There is consistent, but less definitive evidence of adverse effects for children whose blood lead levels were < 5 μg/dL.

It is worth pointing out that if we use concurrent blood lead concentration (instead of maximal below a select cut-off), there is considerable evidence that blood lead levels < 5 μg/dL are associated with adverse effects. Thus, EPA should consider setting a standard with a margin of safety (e.g., by dividing 5 μg/dL by 2 for a standard of 2.5 μg/dL) to insure that children are adequately protected from lead exposure.

Page 24: The citation on page 24 (and in the reference list) should read: "Jacobs et al. 2002", not "Jacobson et al. 2002".

Page 27: In concur with the decision to rely on two models: the IEUBK and the Lanphear empirical model (1998). Because the biokinetic models make numerous assumptions, it is critical to test and validate the IEUBK model using epidemiologic data.

Some reviewers argue that the slope ratio models cannot be extrapolated on a national scale, but there are examples where epidemiologic data were used to promulgate US EPA lead standards (e.g., residential dust lead standards). To be accurate, both the IEUBK model and the epidemiologic data were used, but the floor dust lead loading standard was first set by the US Department of Housing and Urban Development using epidemiologic data.

In the end, it is essential that both the IEUBK model and empiric data are used. If the results are inconsistent, we should understand the factors that are driving the differences.

Page 28, 4.3.3: I concur with the decision to use concurrent blood lead concentration. This was among the strongest predictor of lead-associated IQ deficits and is the blood lead index that is most widely available from epidemiologic studies.

Dr. Frederick J. Miller

"Analysis Plan for Human Health and Ecological Risk Assessment for the Review of the Lead National Ambient Air Quality Standard" May 06 Draft

The consultation with OAQPS staff was very helpful because the analysis plan was not written in enough detail that one could extract all of the assessment nuisances from it. Before providing the limited number of specific comments that I have, I will present in bullet form, the guidance that I believe will help staff develop the risk assessment (RA) for lead.

- There does not appear to be adequate data to include renal effects in the RA. Priority should be given to completing all tasks involving children as a susceptible group before attention is possibly paid to cardiovascular effects in adults.
- The 1990 assessment and guidance from CASAC included the recommendation that the averaging time for the Pb NAAQS be changed from 90 days to 30 days. This was due to being able to better address short term exposure implications for children's health. Nothing has changed to alter the recommendation that the averaging time of the standard be based up 30 days.
- There is insufficient time to do a full probabilistic RA. Staff has appropriately identified the types of sensitivity analyses that will be useful to provide insights on uncertainty and variability.
- Monte Carlo modeling is being considered by staff, but again the time frame for completing things to meet the court ordered deadline probably prohibits these types of analyses.
- Staff should be aware that the data neither support nor refute that there is a threshold below which adverse effects of Pb are not seen. As such, some policy relevant background is probably going to be needed for the RA.
- Predictions of the biokinetic models for some of the case specific pilot study locations should be compared to predictions from any slope ratio models that have been developed for these areas. This would help to determine the extent to which the biokinetic models can be applied on a national scale because the slope ratio models can definitely not be extrapolated in this manner.
- The ecological RA approach needs to be refined more. There are too many potential pathways that could be explored, such that staff needs to prioritize their efforts.

Specific Comments

p. 3	Do the current soil data support the 6 year constancy assumption?
p. 6	Soil Pb variability – how useful is this because it would seem that this is a highly variable endpoint
p. 7	Housing age – will be more useful if persons have resided there a long time. What do we know about average residence time in older homes/
p. 13	IQ is not a metric – it is a response variable. We talk about dose metrics and response variables, not response metrics
p. 18	Census Tracts and census blocks are discussed here, but some of the wording

	implies census tracts is what the authors intended
p. 18	What data limitations prevent using probabilistic simulations to address uncertainties?
p. 18	Why pursue the lowest risk branch? To what extent have all these exposure models been validated or evaluated?
p. 23	Something doesn't appear right with the IEUBK model default values cited at the bottom of the page.
p. 55	Where does the acute to chronic ratio of 26 come from? If it is a simple time adjustment, then Haber's Law is being invoked. This reviewer has shown that Haber's Law is a special case of the generalized power law family and that it does not hold for most response variables.
	The eco system risk assessment seems to have so many "if" that staff could waste a lot of resources on a "fishing" expedition. Are there not guidelines that could be invoked as to how to proceed?

Dr. Paul Mushak

MEETING COMMENTS ON SECTION 6.0, RISK ASSESSMENT, OF THE OAQPS 5/06 "ANALYSIS PLAN" DRAFT

Reviewer: Paul Mushak, Ph.D.

Section 6 of the titled document deals with the proposed OAQPS risk assessment with reference to structuring a Staff Paper for use by the Administrator.

The overall plan for the OAQPS Pb risk assessment generally adheres implicitly and explicitly to the usual conceptualization of the four elements in a risk assessment: hazard characterization, dose-response relationships, exposure assessment, and risk characterization. The last-named typically also partakes of sensitivity and uncertainty analyses. Figure 3-2, p. 14, of the OAQPS Draft Plan more or less captures these elements. Two of these, the hazard characterization and the specific dose-response functions for developmental neurotoxic insults in young children and both hypertensive and nephrotoxic markers in adults, will inform the risk metrics.

Exposure Assessment Issues

The exposure assessment is typically the element in risk assessment that drives the overall process of risk characterization in those cases such as lead that are pervasive in distribution across environmental media and also highly variable in that distribution.

The range of exposures typically exceeds the range of toxic responses for a given exposure. Two children otherwise similarly characterized socioeconomically and demographically might have a ten-fold range of exposures across scenarios. Two children otherwise comparable and with the same Pb-B level would not usually have a ten-fold range in lead-induced toxic insults.

Case studies using exposure modeling data often end up using sequential modeling. In the flow scheme depicted in Figure 3.2, modeling output is sequentially input to the next model. There is little discussion of this, including in the variability/uncertainty sections.

Sections 4.2 and 4.3 discuss the lead exposure aspects with reference to selected risk populations. However, there is little discussion in Section 4 or Section 6 of what is to be the universe of the populations being modeled. I understand that one can use uniform age cohorts or distributed age cohorts but this breakout is methodological in nature. What fraction of the U.S. population is to be modeled when using four discrete exposure scenarios? This reviewer has a problem linking Sections 3 and 4 (4.2, 4.3) with Section 6.1.

As I noted in pre-meeting comments, the four source/emission/exposure scenarios would be expected to represent quite distinct exposure populations as to their size and extent of impact. Each of these population subsets, however, would follow the same dose-response relationships

refined by the known confounders or risk factors that affect the dose-response. On the other hand, if the exposure assessment is made too unwieldy, the overall process suffers.

Also noted earlier, case studies using measurement (as opposed to modeling) data are constrained and pre-biased by the quality of the available information. What exclusionary or inclusionary criteria will OAQPS use to select communities under each scenario or to then accept or reject blood lead and/or environmental lead data sets at the selected case study communities? Little is offered in the draft Analysis Plan about such questions, yet the Panel is asked to advise on the matter. Much is qualitative in the draft's presentations and this applies as well to the conceptualization graphic in Figure 3-2.

Probabilistic risk assessment (PRA) is largely useful when the size of the database is large and the database quality sufficient to allow meaningful PRA to produce a characterization of some acceptable level of uncertainty and variability. It is not clear in the Analysis Plan what criteria will be employed to deal with the eligibility of exposure data sets (also inputs to the exposure module of the biokinetic models) for PRA in terms of estimating probability distributions.

Finally, there is the exposure assessment aspect to the question of "margin of safety" mandated in the language of the CAA. How is that to be accommodated in technical terms? One logically could not use modeling approaches that computationally attenuate, i.e., underestimate, the actual levels of lead exposures or the levels of associated risk from such exposures for children in the upper tail of any nonlinear distribution of Pb-B measurements.

The use of uniform-age or distributed age-fraction cohorts will depend on which alternative between the two turns up the most protective option for children < 72 mos. of age and the intent of OAQPS to do such runs first and then select the better option appears prudent. It is not obvious which approach is inherently most protective given the operation of four case-study scenarios.

I don't quite understand how modeling of exposures to lead of the U.S. population of young kids, presumably the universe of the intended "exposure population" driving the NAAQS process, is to be done with the models at hand. Risk assessments for lead and other multi-media pollutants done by EPA, such as those done by various Regional staff for waste site evaluations, employ the IEUBK model for modeling lead exposure of a typical child or a typical cluster of same-residence children as the universe of the child population. Furthermore, the child's residence (house plus yard) is typically defined as the exposure unit. In some few cases, there have been extensions to a proximate community lead contributor such as in the human health risk assessment done for the Coeur d'Alene River Basin, ID, lead contamination site by Region X, EPA, and the State of Idaho. Use of the IEUBK model for general community-wide applications needs to be approached with caution.

The proposed use of modeling for adult populations is vague and confusing to the reader. As noted in other, pre-meeting comments, modeling of adult lead exposure for the near future consists of the ad-hoc equation model first presented by Bowers in 1994. Other models may be problematic for use at their current stages of development for adult/all-ages use. The background

Pb-B used in the model is that taken from NHANES data, something which is statistically inadvisable for subsets of the U.S. population. However, if OAQPS is modeling the entire adult U.S. population then this caveat becomes less pressing.

OAQPS proposes "tracking" of adult Pb-Bs across ages of the modeled adult population. This obviously requires a PB-PK model, not a statistical one. That is, the statistical (equation) model for adults at all times of adulthood gives outputs driven by the environmental lead contact, not biokinetic aspects. A 25-year-old adult in contact with soil Pb of 2000 ppm is estimated to have the same Pb-B as a 35- or 45-year-old in contact with a soil lead of 2000 ppm.

Risk Metrics for Children

The risk of adverse effects as the direct risk metric for routine regulatory use is, in a sense, new to EPA in the case of lead. It is certainly new to EPA for purposes of a national air lead standard. It has typically been the case, for children's lead exposures simulated using the IEUBK model, that dosimetry via the geometric mean and percentile cut-off combinations in simulated Pb-B was the principal output of interest. The not-to-exceed Pb-B value in tandem with the percent not-to-exceed the ceiling Pb-B represented in the first instance a sense of the biomedical literature and in the second instance a policy call by EPA's OSWER.

It is not clear to this reviewer what the specific methodological approaches will be in determining distributions of population-wide IQ decrements across the child population. Will specific prospective study data for child populations most closely matching the children in each of the four exposure scenarios be selected for Pb-B stratified data to determine that study's finding of IQ decrement empirically? For example, the Port Pirie smelter town prospective data of McMichael, Baghurst, Vimpani et al. would be considered for the operating smelter scenario described by OAQPS in Section 3.

Will one simply translate Pb-B change distributions into IQ decrement distributions using either -0.6 IQ point/Fg/dl Pb-B for final levels below 10 µg/dl and -0.3 IQ point/µg/dl for final Pb-Bs 10 µg/dl or higher?

I noted in pre-meeting comments on the OAQPS Plan that the draft Plan discussed, p. 37-38, 6.2, Risk Metrics for Children, the intent to use IQ decrements assumed to be expressed across the entire case study populations. This is to be done in lieu of modeling absolute IQ shifts, a difficult undertaking with problematic assumptions. The available data from the longitudinal studies, as I seem to recall but maybe incorrectly, indicate that in at least the Boston studies there was a full spectrum shift in the IQ decrement. That is, the overall associations were not being driven by the lower end of the IQ distribution. This should be checked out.

Risk Metrics for Adults

A number of my comments on risk metrics for kids apply to adults as well. The Plan notes the intent to select some of the parameters from NHANES data. Use of NHANES data is OK for national population modeling but is inadvisable for use of groups smaller than the U.S. population as a whole.

CASAC REVIEW OF THE OAQPS "ANALYSIS PLAN FOR HUMAN HEALTH AND ECOLOGICAL RISK ASSESSMENT FOR THE REVIEW OF THE LEAD AMBIENT AIR QUALITY STANDARDS"

Reviewer: Paul Mushak, Ph.D.

I have a number of general and specific comments about the draft Analysis Plan. Comments are confined to the human health effects portions, to include the risk characterization portion. An earlier, February 2006, OAQPS document was presented to the Panel in its last meeting but that was principally informational.

GENERAL COMMENTS

The OAQPS, based on this reviewer's experience with these and other types of expert consensus public documents, has a considerable task. Distillation of the scientific data from an AQCD or Hazardous Air Pollutants document to a Staff Paper or other regulatory advisory instrument is often more scientifically difficult than doing the original scientific assessments. That sense certainly seems to exist in the present instance. I am sure that I'm not the only Panel member who has a lot of questions about the basic approaches set forth and the amount of information provided to the CASAC Panel for advising on OAQPS blueprints for complicated approaches.

Two main tasks are presented to the CASAC panel in advising OAQPS on the matter of the lead NAAQS. First, there is the assessment of the wealth of new information to consider in the areas of exposure and health risk assessments. Secondly, the Panel will also be evaluating new methodologies for dealing with this wealth of new information that are more complex and involved than was the case with derivation of the 1978 and still current lead NAAQS. The methodology employed for the 1978 promulgation of the current standard was relatively straightforward as to derivation of the number.

Role of the CASAC Panel in Reviewing OAQPS Blueprints

It is my understanding that OAQPS seeks review of the 5/06 Risk Assessment Plan with reference to the validity of the choices of the exposure and health effects data sets that will feed into the overall plan. But I also interpret the charges to the Panel by OAQPS to mean the staffers wish recommendations as to the pros and cons of the methodologies that will draw an overall blueprint for the plan. It is standard practice for EPA to ask CASAC panels to review what's been done. Here is also a case of OAQPS seeking Panel review beforehand of how the Agency is to actually craft the elements of the Staff Paper.

Lead is likely a special case requiring Panel input at the start owing to its status as (*i*) a multi-media pollutant, (*ii*) a multi-media pollutant with a huge historical and persisting impact on

environmental domains within and outside of EPA's regulatory purview, and (*iii*) a multi-media pollutant with well-characterized risk assessment elements, i.e., the typical hazard characterization, dose-responses, exposure assessment, and risk characterization parts of a risk assessment within generally recognized elements of variability and uncertainty.

The CASAC Panel can certainly review the scientific validity of the actual scientific data sets OAQPS plans to use and the disciplinary validity of OAQPS' perceptions and judgments as to how they are intended for use. It is somewhat less clear how thoroughly the Panel can advise on theoretical approaches without more information on the details of what's to actually be done.

OAQPS will come back to the Panel two more times in the future --with pilot and then complete study results respectively. OAQPS needs to understand that concurrence by the Panel in the conceptual and general approach to be used in the risk assessment plan does not in any way assure unqualified agreement with the results obtained for the pilot and full studies. Agreement with the Plan in principle does not assure in any way the Panel's future agreement with results in practice.

The Analysis Plan and Scientific Elements of a NAAQS

The Analysis Plan appears to cover data that inform only two of the four scientific elements of a NAAQS promulgation. The other two elements, the averaging time and the statistical form of the attainment and compliance metric, apparently requires much less input from the Panel. Correct?

Writing Clarity of the Action Plan Document

I found this document to be quite confusing and difficult to read. It reads like an exchange of memos among individuals fully familiar with the regulatory shorthand. Besides language form, the flow across sections is not clear or smooth. I recommend a read through for crispness and clarity.

For example, Section 2 discusses the Case Study approach. However, there are two sets of case study types presented. They are made to look distinct, although they are closely connected. The first bullet on p. 6, "Variety of ambient air sources..." actually subsumes the three bullets on p. 7, even though the latter starts with "We also plan to select case studies...of ambient air emissions..."

Conceptual Air Pb Scenarios and the NAAQS Selection Process

The 5/06 risk assessment plan presents a set of four hypothetical scenarios to capture the range of contamination and exposure settings likely to collectively represent the need for, and direction of, the lead primary and secondary standards for development versus the current Pb NAAQS. In reality, however, the choices are extremely limited in some cases and almost endless in others. Each scenario arguably requires technical assessment and Panel input for the specific locales and contamination or exposure. Given the pressures of time, perhaps some specific communities have already been selected by OAQPS for pilot study.

The operating lead smelter scenario can only be developed for the one operating smelter in the U.S., unless we are to consider historical data for defunct facilities with a lot of historical environmental and blood lead data, e.g., the Bunker Hill (Box)/Silver Valley, ID Superfund site. The operating facility is the smelter complex in Herculaneum, MO. There are multiple sets of environmental lead and blood lead data for lead contamination in and around Herculaneum. However, there is in the regulatory record the fact that the community around this smelter has been the subject of remediation interventions and other interventions affecting blood lead-environmental lead relationships.

Presumably, this site could also be studied, using available data, for exceedences as a function of averaging time, and as to the form. These components are defined in footnote #3, p. 1, in the 5/06 risk assessment document.

We know that the more appropriate averaging time should be no more than 30 days for mobile sources, based on Panel member Dr. Schwartz's temporal analyses of responses in children's Pb-B levels versus air Pb changes, done back in the mid-1980s for OAQPS. OAQPS in its recommendation for the averaging time in its 1989 Staff Paper "Review of the National Ambient Air Quality Standards for Lead: Assessment of Scientific and Technical Information" also recommended a calendar month for point sources (p. IV-7). That document, p. IV-7 to IV-11, also came up with the statistical form of the standard in its recommendations.

A second scenario is to represent a case study for re-entrained dust, specifically along roadways. Numerous locales would qualify but each would differ as to fugitive dust lead levels as a function of such factors as heterogeneity of the "reservoirs", precipitation, vehicular density, wind patterns, etc. What are the criteria for selecting among them? The document is not clear on this. Also, the scenario for fugitive dusts excludes the real problem of leaded dusts generated from contaminated waste sites, e.g., tailing and related waste piles, such as are found in and around the numerous extractive industry sites in the West and Midwest.

Another scenario considers multiple emitters at a locale under the rubric of stationary sources, e.g., incinerators, battery production facilities, etc. The mix of individual emitters could be many and highly variable site to site. Again, the Panel is given little specific information as to how choices among many possibilities would be made or how well this scenario would serve for formulation of Pb NAAQS.

Relevance of the Case Study Approach to the Scientific Basis of National Criteria Pollutants

It's not at all clear to this reviewer how a variety of exposure scenarios within Case Study frameworks will scientifically translate to the scientific basis for Staff Paper recommendations on a single NAAQS, or a range of NAAQS recommendations from which the Administrator is to select one. Panel review of the exposure data within the case studies would arguably be guided by the purposes to which the exposure data are to be put. If a range of NAAQS is recommended for selection, what would be the scientific guidance for the selection? Just out of curiosity, can

the Administrator even pick more than one value for a national standard? If so, how would that be enforced? All of this should be made clear in any presentation by OAQPS to the Panel at its upcoming meeting.

For example, the four source/ emission/ exposure scenarios would be expected to represent quite distinct exposure populations as to their size and extent of impact. Each of these population subsets, however, would follow the same dose-response relationships refined by the known confounders or risk factors that affect the dose-response. In conventional risk assessment methodologies, including those typically used by EPA Regional staffers for Superfund and related sites, it is the exposure assessment that drives the overall risk characterization. If the exposure assessment is made too unwieldy, the overall process suffers.

On the other hand, the range of exposures typically exceeds the range of toxic responses for a given exposure. Two children otherwise similarly characterized might have a ten-fold range of exposures across scenarios. Two children otherwise comparable and with the same Pb-B level would not usually have a ten-fold range in lead-induced toxic insults.

Case studies using measurement (as opposed to modeling) data are constrained and pre-biased by the quality of the available information. What exclusionary or inclusionary criteria will OAQPS use to select communities under each scenario or to then accept or reject blood lead and/ or environmental lead data sets at the selected case study communities? Little is offered in the draft Analysis Plan about such questions, yet the Panel is asked to advise on the matter. Much is qualitative in the draft's presentations.

The use of probabilistic risk assessment (PRA) is largely useful when the size of the database is sufficiently large as to allow meaningful PRA and to produce a characterization of some acceptable level of uncertainty and variability. It is not clear in the Analysis Plan what criteria will be employed to deal with the eligibility of exposure data sets (also inputs to the exposure module of the biokinetic models) for PRA in terms of estimation of probability distributions.

Case studies using modeling data will often end up using sequential modeling data. In the flow scheme depicted in Figure 3.2, modeling output is sequentially input to the next model. There is little discussion of this, including in the variability/uncertainty sections.

Finally, there is the scientific aspect to the question of "margin of safety" mandated in the language of the CAA. How is that to be accommodated in technical terms?

<u>Relevance of Certain Risk Assessment Components for NAAQS Options Presented to the Administrator</u>

The draft Plan, Sec. 7.2, describes OAQPS' plans to use an "integrated approach" for the various model variability and uncertainty generated for the max, min and central tendency branches. This is graphically depicted in Figure 7.2. How exactly are regulators to use this information in any scientifically useful way? This Figure is one of a number that need to be

better defined and plugged into application scenarios for the benefit of the Panel in the upcoming meeting.

SPECIFIC COMMENTS: CHARGES TO THE PANEL

Human Exposure and Health Risk Assessment

Do the Panel members have general comments on the approach proposed?

Yes. See my general comments above.

Exposure Assessment (estimating media concentrations)

1a. What are the Panel members' views on the general approach using monitoring... and modeling data?

The general approach of using measured data where available and modeled data where measured data are not available in theory sounds O.K. However, this either/ or approach of measured versus modeled information needs to be carefully considered by OAQPS for each of the scenarios in the case studies. Measured data can also differ with modeled data, as for example, in the case of blood lead screenings. The discordance can arise for any of three reasons. Measurement was good but the model's lousy; measurement was bad but the model was good; both measurement and modeling were lousy, but each was lousy for different reasons.

Lousy measurement data is certainly not better than good modeled data. There are many reasons why measured data may be badly flawed. Conversely, one has to be circumspect about validation and calibration of models.

It would help if the draft Action Plan contained more details about the use of monitoring or modeling data. For example, will OAQPS confine itself for the "smelter" scenario to operating facilities, of which there is only one, or will it make use of the large archives of data gathered for now-defunct facilities? The Bunker Hill, East Helena, and Omaha operations have extensive archival data from the respective EPA Regions.

OAQPS would certainly agree that various types of exposure scenario sites and communities will have measurement (and/or modeled) data because there was a purpose for doing the measurements in the first place. Typically the reason was regulatory and/or litigatory. The question then arises as to how the purpose of the measurements would result in any actions that then produce consequences for multi-media exposure relationships to adverse endpoints.

Section 2.1 states that, for the smelter scenario, the operating smelter in Herculaneum will provide more measurement data of use to OAQPS than any of the other sources. Presumably, OAQPS would be aware of the various activities over the years in this smelter community related to childhood lead exposures and soil lead abatement. For example, the owners in 2001 entered into a remediation agreement with Region 7 and the MO DNR. The

environmental lead and childhood blood lead data sets need to be carefully evaluated with reference to alterations through remediation activities. That certainly would be required if Pb-B measurement data are to be used to buttress modeling.

Data are also available from this site that address the question of recontamination rates for post-remediation, clean soils. The MO DNR found, as I recollect, that remediated properties were rapidly becoming contaminated by some combination of fugitive dusts and direct deposition from the atmosphere. This finding is consistent with the findings at the Bunker Hill site communities, which Dr. von Lindern can speak to in more detail.

1b. What are the Panel's views...on use of source-apportionment...?

Question 1b should be Question 1c and vice-versa. One cannot respond to apportioning Qs if the components of the apportionment method have not yet been posed for Qs.

Little is said in the draft Plan about how OAQPS plans to do apportionment of air lead levels into reentrained dust lead and new emissions. The discussion is confined to the second and third bullets on p. 21, Sec. 4.1.1. The approach of chemical mass balance seems reasonable. I am unfamiliar with the positive matrix factorization (PMF) methodology. There does not appear to be enough information to answer the question of which approach would be better without details on the methodology.

1c. Do the Panel members have comments...on options presented...for estimating re-emission of ...lead?

The brief discussion in the third bullet, p. 21, notes the dust resuspension model of the USDA-ARS. What's wrong with also using the fugitive dust reentrainment modeling described in the publicly available 1985 EPA document on fugitive dusts? Cowhert and colleagues at Mid West Research Institute authored this report. This document was, I believe, for the EPA-Cincinnati Risk Reduction Laboratory. The 1985 document is especially useful for roadway fugitive dust modeling.

2a. What are the Panel members' views on the general approach and tools...for use in the assessment [for soils]?

All modeling approaches have their limitations. The single reservoir model, such as that in the MPE approach, would assume relative homogeneity as to the deposition history and source of deposited lead stability.

2b. I have no comment.

2c. What are the views regarding use of the TRIM.FaTE?

I am not familiar enough with this model to comment on it.

2d. Do the Panel members have comments...regarding changes in soil given changes in ambient air concentrations and related deposition?

One can readily calculate that it requires a long time for direct deposition of lead from ambient air onto soils to produce lead recontamination at actionable levels, e.g., 400 ppm Pb. This is the opposite of the case for the rapid lead loadings via dust onto hard surfaces. See my comments on the latter topic for the earlier draft reviews of the AQCD chapters, re: Chapter 4 modeling issues.

The equations to do this are contained in such places as the 1996 EPA document on the 3-city soil lead abatement demonstration project:

> 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, p. 2-25, in this document can be used to calculate that for a 1.0 $\mu g/m^3$ air Pb level, and a reasonable deposition rate of 0.2 cm/sec, that one collects lead at the soil surface at the rate of 6 $\mu g/cm^2$-year. Since soil lead from air deposition is known to be concentrated in the top 1 cm or so depth, the annual rate is 6 $\mu g/cc$-year. A soil mass of 2 g/cc gives an annual accumulation rate of lead at this air level of 3 ppm.

This calculation is only for simple newly emitted lead deposition. Re-deposition of reentrained dust lead at huge lead content can lead to much quicker soil recontamination rates. I would note the MO NDR finding about rapid recontamination from a combination of direct and fugitive leaded dust depositions onto remediated soils at Herculaneum, MO. The Bunker Hill site has produced data of this type as well. Panel member Dr. von Lindern can speak to the latter case.

3a. Do the Panel members agree with the approach of using the indicated ratios for relating interior dust lead to air lead and exterior soil?

For default ratio purposes, the value of 0.7 x Pb-soil, ppm = Pb-dust probably is acceptable for those lead exposure scenarios where the bulk of soil lead is historical with little ongoing soil contamination. I am less comfortable with the air Pb relationship to interior dusts, i.e., a ratio of 100/1. An air lead of 1 unit = 100 ppm dust lead is the default assumption for the IEUBK model.

First, the lead-loading rate for interior dusts on hard surfaces shows huge growth in lead loadings over short time periods. I showed this in prior comments and calculations provided to NCEA and CASAC. I am not at all sure that children doing hand-mouth activity with dust lead on hard surfaces would ingest dust lead at a level rate of only 100 ppm for an air lead of 1 $\mu g/m^3$.

3b. Do Panel members have comments regarding estimates for the outdoor soil-to-indoor dust and ambient air-to-indoor-dust factors?

See 3a. The current defaults as described in the 1994 IEUBK model guidance document (pp. 2-40, 2-41) were arrived at from materials in the 1989 OAQPS Staff Paper "Exposure Analysis and Model Validation." There are a number of more current or updated data sets that should permit a range of ratio estimates provided the interior dust levels have been appropriately sampled. The more recent Bunker Hill, ID data sets can be examined. An important point with interior dust sampling is to gather dust samples for which there is some idea of accumulation time stability. One of the virtues of dust lead loading versus interior dust lead concentration as the lead metric is that it permits a lead loading rate per unit surface area per unit time.

As I mentioned in earlier comments, life would be simpler in terms of doing these modelings if dust lead loadings from ambient air Pb could be used instead of dust lead concentrations. Combining dust lead loading rates with child hand-surface and hand-in-mouth contact data to give an idea of ingestion from hand lead loading would close the gap to arrive at daily lead intakes. Check with the staff at CPSC regarding transfer rates. They published material some years back when doing estimates of vinyl blind lead transfer quantification. Also, NCEA has come up with transfer rates for the All Ages Lead Model exposure module.

Exposure Assessment (Blood Lead Modeling)

1a. What are the Panel members' views about the intended use of the suite of models for modeling children's Pb-B levels?

As a practical matter, only the IEUBK model can be used at this time for modeling children's Pb-B levels. The other biokinetic models have limitations that will persist through the short time frame permitted for framing and filling out of the Staff Paper. See my earlier comments for Ch. 4 regarding the pros and cons surrounding the current stages of biokinetic models for childhood exposures.

The interdigitation of the biokinetic or mechanistic models with the slope factor approach in the Lanphear report could be done, but perhaps purely for purposes of comparisons. The text of 4.3.1 on p. 27 of the OAQPS draft Plan points out problems with the ad-hoc statistical model approach by Lanphear et al., 1998. The slope factor approach would be logistically difficult to deal with without working it into a program for doing batch runs.

1b. What are the Panel members' views about the use of the suite of biokinetic models for adult exposures?

The adult models that are in any shape for near-future use by OAQPS are all relatively crude equation models, with a focus on a biokinetic coefficient, soil Pb as the independent variable and a Y intercept in Pb-B that presumably integrates all other lead exposures. The main objection with two of these is that they rely on national NHANES data for the intercept. One

cannot disaggregate NHANES national data for site-specific uses. See my comments on the topic for Chapter 7, the Integrative Synthesis chapter.

It is not very easy to use Leggett, 1993, or any other biokinetic model for adult lead exposures at this time because we would have to have a reasonable idea of the historical or life-long lead intake rates for scenario-specific populations. This would allow us to more accurately or at least more adequately model the full bone lead compartment vis-à-vis ongoing lead inputs to the biokinetic module. This dilemma also plagues the development of EPA's All Ages Lead Model (AALM). Any AALM is arguably more useful at this stage of development for simulations to the out years, i.e., for use in going-forward or future-risk scenarios as consequences of current actions, rather than trying to reconstruct past life-time exposures as a baseline exposure level on which to place further increments, i.e., in "predicting the past."

It is likely that the ad-hoc statistical or equation models are what we are currently limited to.

1c. What are the Panel members' views on...plans for deriving a geometric standard deviation...?

This would be difficult to do in terms of relevance to certain of the scenarios. We would expect that those exposure scenarios with a high potential for heterogeneity to environmental media lead levels, e.g., "hot spots," would be difficult to cover with a "one size fits all" GSD. I am aware of a number of site-specific calculated GSDs, for example, for extractive industry and related sites that markedly exceed the 1.6 value used as the default in the IEUBK model that was originally generated from diverse data. For example, the Leadville, CO and other site Pb-B studies produced back in the 1990s yielded GSDs higher than 1.6.

OAQPS has attempted to deal reasonably with the arcane argument going back and forth among interested parties as to what type of a GSD is to be generated for use in the IEUBK and like models and what does a GSD capture in terms of variability. The GSD issue is not a trivial one, since in any log-normal depiction of a distribution of lead-exposed children's Pb-B levels, the number of children captured in the upper tail of the distribution, i.e., the most exposed children, changes with changes in the GSD.

1d. What are the Panel members' views regarding the option of employing Monte Carlo methods to develop exposures...

The general criterion for use of Monte Carlo or any other probabilistic modeling approach is that there be sufficient measurement data to result in distributions that reflect reduction of uncertainty below that which might exist for simply using point estimates. If the empirical data are not there, or if the amount of data to meet Staffers' criteria for inclusion is insufficient, why waste time and effort? No point in having paralysis through seemingly meaningless analysis.

A second issue is, how does all of this feed into the Administrator having to pick a number?? See my general comment about the usefulness of having OAQPS' presentation at the

upcoming meeting include what and how the Staff Paper will distill and present all of these risk modeling exercises so as to be of use to the Administrator's decision on a NAAQS for Pb.

The National Academy of Sciences, in its 2005 report "Superfund and Mining Megasites. Lessons From..." gave over its chapter 6 to the IEUBK model. Recommendation 3, p. 271, recommended that EPA proceed with implementing a probabilistic stochastic exposure model component for the model. Implicit in this recommendation given its context, however, is that an adequate amount of measurement data would exist for media-specific inputs such as would often occur at Superfund and similar sites. Furthermore, the NAS Committee also made clear that this proposed version must be subjected to verification and validation. It is unclear that a probabilistic input add-on could be verified and validated at this stage of the game given the timelines and deadlines. The Committee also noted that a probabilistic statement of exposure inputs would reduce uncertainty about the GSD selection.

Effects Assessment and Risk Assessment

1. Do the Panel members generally agree with the three endpoints being considered...?

I have no problem with the choices, especially the choice for children. At the present stage of epidemiological and mechanistic toxicological knowledge about toxic effects in adult humans, kidney effects of the type indicated are quite appropriate.

Choice of these age-scaled endpoints also adhere to the CAA requirement for criteria pollutants that their analysis be focused on, and regulated for, the most vulnerable subsets of the population.

2a. What are the Panel members' views on the set of studies the staff is considering for estimating childhood IQ decrements associated with blood lead levels...?

The AQCD document makes a strong case for the validity of the Lanphear et al. pooled analysis for use in staff analysis for the Staff Paper. There is little credible challenge to the validity of the shape of the dose-response curve in the sub-10 µg/dl vis-à-vis 10 or higher segments.

2b. Do Panel members have comments or advice on the types of models regarding the individual study findings on IQ loss for children at exposures below 10 units (concurrent measurement)?

I'm not sure I understand the point of the question. It should be clarified and differentiated from the next question.

2c. Do Panel members have any recommendations regarding the Pb-B metric to be used?

Concurrent measurement, average measurement, and even *in utero* maternal Pb-Bs have now all been linked to IQ decrements. Dr. Dietrich in the previous CASAC meeting, in response to my question on distinguishing concurrent Pb-B as a separate variable from simply the effect of

rank ordering, i.e., "tracking," pointed out that concurrency is a distinct and specific dose metric. That has major implications not only for mechanistic neurotoxicology but also for how OAQPS weaves its magic for the Staff Paper.

OAQPS should, in my opinion, carefully examine the Pb-B metrics in the context of the characteristics of the respective cohorts. Overall, Lanphear et al. (2005), which has the most current analysis of the aggregated database so far for these associations, found that concurrent Pb-Bs were the best predictors of cognitive deficits. Refer to the exchange in EHP (Letters) in the Feb. 2006 issue about this point between Drs. Ernhart and Lanphear.

Ernhart CB. 2006. [Letter]. Effect of lead on IQ in children. Environ. Health Perspect. 114:A85-A86.

Lanphear BP, Hornung R, Khoury J, Yolton K, Dietrich KN. 2006. [Letter]. Lead and IQ in children: Lanphear et al. respond. Environ. Health Perspect. 114:A86-A87.

3a. What are the Panel members' views on the studies selected in modeling blood pressure effects in adults?

The choices appear appropriate, given the options available to the staff. There are a number of other studies which generally buttress this choice of Nawrot et al.

3b. Opinions on linear versus log-linear model selections for this endpoint?

No opinions.

4. Panel members' views on the studies for non-HT renal effects?

The selected studies appear reasonable, among the options in the current literature.

5. Panel members' views on the same metric of exposure, average Pb-B, for both endpoints?

As a practical matter, there is little other choice. Rarely are adult subjects in such studies accompanied by a lifetime history of serial Pb-B measurements. Furthermore, it is difficult at this stage of development to use modeled Pb-Bs from lifelong selected intakes using all-ages biokinetic models, like O'Flaherty or Leggett.

6. Do Panel members generally agree with the various risk measures the staff is considering?

Overall, the choices seem appropriate. I have some added comments not in response to Charge Qs.

The draft Plan discussed, p. 37-38, 6.2, Risk Metrics for Children, the intent to use IQ decrements assumed to be expressed across the entire case study populations. This is to be done in lieu of modeling absolute IQ shifts, a difficult undertaking with problematic assumptions. The

available data from the longitudinal studies, as I seem to recall but maybe incorrectly, indicate that in at least the Boston studies there was a full spectrum shift in the IQ decrement. That is, the lower end of the IQ distribution was not driving the overall associations. This should be checked out.

Sections 7.1 and 7.2, in the portion of the draft dealing with an "Integrated Approach," need to be clarified as to what you are talking about in terms of conventional uses of sensitivity and related analyses. Figures 7-1 and 7-2 should be explained in clear detail during the OAQPS presentation at the upcoming meeting.

POST-MEETING COMMENTS: CASAC PANEL REVIEW OF THE SECOND PB AQCD DRAFT AND CONSULTATION FOR THE OAQPS DRAFT ANALYSIS PLAN

Reviewer: Paul Mushak, Ph.D.

July 3, 2006

Overall Recommendations for Pb AQCD-2.

The second draft of the Pb AQCD, on balance, is of sufficiently good scientific quality that it can go forward in the overall process for review of the NAAQS. Going forward assumes attention to recommendations for changes in draft Chapter 7.

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 time frame, 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.

The 2000 (second edition) WHO-Europe "Air Quality Guidelines for Europe" took an even more quantitative approach, which permitted a single, low air lead guideline to be selected, a guideline value at the lower end of the previous range given in 1987. Elements of the recommendation in the Guidelines update for air lead included 1) derivation of a guideline value based on a Pb-B level of 10 µg/dl in young children; 2) lead ingestion as well as lead inhalation are important for young children; 3) an air lead value of 1.0 $\mu g/m^3$ translates via direct and indirect (dust/soil/diet) pathways to a Pb-B of at least 5 µg/dl; 4) 98% of young children should have a Pb-B that does not exceed 10 µg/dl; 4) this translates to the median Pb-B not exceeding 5.4 µg/dl. All of this, plus factoring in the non-air inputs to children's Pb-B levels, works out to the air lead not exceeding 0.5 $\mu g/m^3$ and this value was the recommended Guideline.

World Health Organization. 2000. Air Quality Guidelines for Europe. Second Edition. Lead. Ch. 6.7. WHO Regional Bureau for Europe, Bilthoven, The Netherlands, pp. 149-153.

If CASAC wishes the relevant sections of these two WHO documents, they presumably are in the EPA docket for the current process. Otherwise, I would be happy to provide them.

Overall Consultation Recommendations on the OAQPS Draft Action Plan

I concur in the recommendations of others regarding elements of the draft OAQPS Action Plan and add several more. Overall, the Action Plan process should go forward only within a number of recommendations for prioritization or limitation:

- The Case Study approach appears acceptable in principle, but it will be the details in the Pilot and Full phases that determine how many devils there are to deal with.

- Concurrence with the blueprint does not translate to acceptance of the results there from, and results review by the Panel in the future will say what they say.

- The Risk evaluation should be focused on IQ and any other neurobehavioral deficits in young children as a first priority of business for risk quantification, with further sensitive population evaluations only proceeding when the first evaluation is finished.

- The most acceptable dose-response data set for assessment of IQ decrement distributions are contained in the international pooled analysis by Lanphear et al., 2005.

- Lead exposure modeling will necessarily entail the IEUBK model, but use will need to be in harmony with risk assessment use by EPA sister offices and EPA Regions.

- Comparisons can be made of biokinetic with statistical, slope-factor modeling approaches as part of the dose-response calculus.

- The development and evaluation/validation of a probabilistic distribution exposure input module for any biokinetic model at this time is simply not feasible and the use of exposure inputs to the biokinetic module and its outputs as point estimates will be necessary.

- I would urge that OAQPS include the full assessment of the impact of even modest air lead concentrations on significant lead exposures, through dust lead loadings onto interior and exterior hard surfaces, of very young children; such modest air lead levels are a combination of both new emissions and reentrained, dust lead movement back to the atmosphere.

- I recommend that OAQPS take special note of the above fact that air lead levels reflect both new lead emissions and reentrained lead from already-contaminated surfaces; this recognition will greatly assist in air lead NAAQS review in that any further direct emissions from point sources will have to be kept to a minimum, and certainly below 1.5 $\mu g/m^3$.

- Finally, I would especially urge OAQPS to keep in mind that the current low levels of lead in air in many areas are absolutely no scientific or biomedical rationale for retention of the current NAAQS of 1.5 air lead units for lead; at the 1.5 current standard, a significant window of permissible pollution would occur should there be abrupt entry of new industrial technologies having potentially significant waste streams that include significant new air lead emissions; re-attainment of typical levels nationally at the 1.5 current standard would be a major source of, and would actually produce, new exposures and associated toxicity in sensitive populations. To illustrate, Table 4-3 in the OAQPS 3/89 Staff Paper showed that at 1.5 $\mu g/m^3$, the fraction of young children with Pb-B > 10 $\mu g/dl$ is two-to-three times higher than is the case for Pb-air at 0.5 $\mu g/m^3$.

<u>Other Comments</u>

Dr. Crapo correctly noted at the meeting that there is a sizeable accumulated lead burden in various environmental compartments with which risk populations come in contact. There was the implication that this accumulated burden would overwhelm new air lead emissions from

point sources. I would like to add further discussion by first noting that, yes, there is an accumulated burden of lead that contributes to children's Pb-B levels, but no, this complication does not trivialize the impact of new lead emission inputs to children's Pb-Bs nor does it render the NAAQS lead regulation approach moot. Past, present and future inputs to air lead all need to have equal billing.

Dr. Crapo cited lead levels normalized over huge expanses. The localized or "hot spot" distributions of anthropogenic lead as a fraction of that overall amount is the metric that is comparatively more at issue for human lead exposures. Areas of soil surfaces receiving anthropogenic lead input in the form of fallout from point or past mobile emissions will exceed areas of natural or background crustal origin by many-fold. Compare a natural crustal abundance of lead at 20-50 ppm versus a 20- to 50-fold higher roadway or point source impact zone soil of 1,000 ppm. There are numerous publications on the topic.

In addition, those areas with the most anthropogenic lead inventories and those most likely to receive more new lead emissions are also those with the most numbers of children, either in terms of children near past mobile source emissions or child populations around lead point sources. Of the U.S. total child population ages 6 months to 6 years of age, the great majority live in and around areas with elevated soil lead levels from anthropogenic activity and these figures are to be found in such sources as the Appendix and summary tables in the 1988 U.S. ATSDR report to Congress on childhood lead poisoning in America.

U.S. ATSDR. 1988. The Nature and Extent of Childhood Lead Poisoning in Children in the United States. Atlanta, GA: Agency for Toxic Substances and Disease Registry, U.S. Centers for Disease Control.

Equally important, natural or geochemical lead still encased in natural soil matrices, e.g., silicates, is quite different from deposited anthropogenic lead, the latter tending to be in smaller and in more chemically and biochemically available, i.e., more bioavailable, particulate forms. This means that transport of lead in soils to children's residential interiors via various mechanisms favors relatively higher inputs to interior dusts from anthropogenic lead in soils than from naturally derived lead in soils and also favors higher uptake rates of anthropogenic lead in terms of bioavailability.

The cumulative inputs to the hot-spot subsets of U.S. surfaces of anthropogenic lead can be estimated in various ways. Our 1988 ATSDR Report to Congress noted the tonnages deposited from leaded gasoline combustion and from lead paint use as being cumulatively about 10 million metric tons (MT). Extractive industry wastes from smelters, milling operations, slags, etc. have been computed by Nriagu and Pacyna, 1988, and are sizable in terms of impact on nearby communities and are contained in the 1993 NAS/NRC report on lead exposure in sensitive populations.

NAS/NRC.1993. Measurement of Lead Exposure in Infants, Children, and Other Sensitive Populations. National Research Council. Washington, DC: National Academy Press., Ch. 3: pp. 99-141.

Nriagu JO, Pacyna JM. 1988. Quantitative assessment of worldwide contamination of air, water and soils by trace metals. Nature 338: 47-49.

One can also calculate U.S. anthropogenic lead dispersals as an upper bound by assuming that cumulative figures for U.S. lead consumption over the decades eventually translates to environmental dispersal. Annual figures for U.S. lead use since the 19th C. to the present, as contained in annual estimates from the U.S. Bureau of Mines or the U.S. Geological Survey, are available. These can be summed to achieve a tally of around 100 million MT. I suggest Staff do this. There have been recent summary Tables produced by USGS, as the successor to the U.S. Bureau of Mines Mineral Yearbook statistics. The Bureau essentially went out of business in 1994-95.

DiFrancesco CA, Smith, GR: U.S. Geological Survey. 2005. Lead Statistics. Last Modification, December 2003.

Smith GR: U.S. Geological Survey. 2002. Lead. Accessed at URL http://minerals.usgs.gov/minerals/pubs/commodity/lead/leadmyb02r.pdf.

U.S. Geological Survey, 1901-1927, Mineral Resources of the United States, 1900- 23.

U.S. Geological Survey, 1997-2004, Mineral Commodity Summaries, 1997-2004.

U.S. Geological Survey, 1997-2003, Minerals Yearbook, vol. 1, 1995-2001.

U.S. Geological Survey and U.S. Bureau of Mines, 1996, Mineral Commodity Summaries, 1996.

U.S. Bureau of Mines, 1927-1934, Mineral Resources of the United States, 1924-1931.

U.S. Bureau of Mines, 1933-1996, Mineral Yearbook, 1932-1994.

U.S. Bureau of Mines, 1978-1995, Mineral Commodity Summaries, 1978-1995.

Whatever the size of the current anthropogenic lead burden in soils and other sources of dust-sized particles that can be reentrained into risk population environments, the fact remains that newly deposited dust lead from new emissions can itself add to risks of children's lead exposures as per EPA's own calculations and from which I was able to generate Tables submitted after the first meeting.

To the extent that total but quite low air lead arises from both new air emissions and reentrained dusts, and that total air lead from both sources even at low concentrations can be potentially toxic via dust lead loadings onto hard surfaces, there is clearly very little margin for permissible newly-emitted air lead. The adherence of air lead emissions to this minimal permissible value obviously requires the continued use of an air lead NAAQS.

Dr. Michael Rabinowitz

Comments on "Analysis Plan for Human Health and Ecological Risk Assessment for the Review of the Lead National Ambient Air Quality Standard" May 06 Draft

by Michael Rabinowitz, PhD June 20, 2006

Overall, I see no particular problems with the general approach taken. Although it may well be that the current uncertainties encountered in the various scenarios may prevent clear-cut, precise decision making, should additional pertinent data become available in the future, the strategy proposed here will prove only more useful.

Thus, I have only a few minor corrections or requests for changes in the text.

Chapter Four-Exposure Assessment

section 4.1 mid page 20 suggest "...surfaces to reduce *hypothetical* air concentrations..."

4.1.1 mid page 21 Should the PM be instead PMF in two spots

4.1.6 Page 25 end of paragraph about dietary exposure Do you want to allow for home gardening or shellfish taking?

4.3 Page 26 First paragraph Can you add for clarity "....developed outside the model. *These (or Such)* "empirical" approaches bypass..." I suggest this for clarity and emphasis because the sentence before and this sentence both describe the same methodology. Or don't they?

Page 26 last line how about for emphasis "....will be reflective *only* of differences in the models..."

4.3.4 I particularly liked the first paragraph for clarity.

4.3.5 May I suggest that you have written down in advance, for your own use, specific templates or blank tables of the summary data from the various models and age that you intend to fill in.

Page 62, a small spelling error in the Reference Bellinger et al- *academic*

Responding to the Charge Questions proposed on May 31:

Exposure Assessment: media concentrations

1.a. It is the best that can be done under the circumstances

1.b. Seems appropriate, but care needs to be taken in not only looking at masses of lead emitted, but also their physical properties. For example, lead emitted from a factory may be very different in particle size, and hence respirability and bioavailability than wind blown reentrainment. So, I suppose the exercise needs to be done for different moieties or species of lead.

1.c. I'm not familiar with them, and, so, I can only hope they are reliable and have been tested.

2. Regarding modeling of soil lead, I suppose either a simple reservoir model or a more dynamic model would give similar predictions over a short time span (less than a decade). Beyond that, I would need to see some side-by-side comparisons of the two approaches to make any judgment.

3. Yes, I do agree, but again with the concern about particle size and form of the indoor dust lead. Typically, we have only looked at bulk dust, and the proposed source each generate somewhat different spectrums of particles sizes.

The outdoor-indoor factors will vary with season, climate, housing stock, and other factors. So, either a very wide range can be offered that accommodates all situations, or the model may have to allow for specifying the many important factors related to the house and the season.

4. No, I do not.

Exposure Assessment: blood lead modeling

1.a. Although I must await the values generated by these various models, I suspect that the uncertainty from each model will be so great that the models would be equivalent in their predications.

b. Same answer. The range we see in adult values for nearly the same exposures is so great that the models would be equally predictive.

c. Good idea. It would capture the mean and variance of the distribution, but what about the tails? The top 10 or 1%. Can we not also deal with the next higher moment of the distribution? Skewness?

d. I'd like to see that, but also don't you want to vary the biokinetic factors. These physiological rates vary significantly among individuals and will also cause a broadening in the predicted values.

Dr. Frank Speizer

Comments on Analysis Plan for Lead Risk Assessment, Draft May 31, 2006

Submitted by Frank Speizer

General comment:

The section of the document that describes the planned analyses for IQ change in children, BP risk in adults, and potential renal function change suggests further that in some way these three, really quite different risks (and risk groups) will be combined in some sort of overall risk estimate. No details are provided on how this will be done. The question could be asked should this be done? In terms of risk estimate for the NAAQS one might argue that the IQ in children should provide the most powerful incentive to set a limit and that limit would without any analysis take into account almost any other health parameter. This certainly would reduce the computational needs, simplify the analysis and provide a rather straightforward assessment of risk.

In addition from the standpoint of dealing with both the availability of data, the complication of not having "time specific" data on prior exposure (the changes in environmental lead levels would suggest that a different background level would be needed for each cohort for about each 10 years of age), and the different pathophysiological interpretation of exposure of brain, cardiovascular and renal effects by age and different background exposures, it would seem impossible to realistically think about a combined assessment. Perhaps this is why no model for such was offered.

Furthermore, by restricting the analyses to truly effects below 10µg/dL it seems it would make no difference if one used linear vs. log-linear analyses. Would it simplify interpretation if a linear model were being used? It certainly would likely lead to a no threshold model and then the policy implication would be to establish what proportion of the population would remain at risk for any given level. Perhaps rather than spending time on trying to combine effects that will be difficult to interpret, it would be more useful to run both linear and log-linear models for the IQ effects and compare these models.

Specific Comments:

Table 5-2 I question the value of including the occupational exposure studies in the Nawrot reference. These will generally be higher exposures and introduces the complication of non-linear effects as well as potential misclassification of exposure attributed to lead when complex other mixtures may be present. In addition issues of background and disease state for a given calculated exposure may be quite different.

Table 5-3 Careful consideration of the Normative Aging Study will have to be made. It is not clear to what degree the three reports are reporting overlapping data and the weighting of each

study for a risk assessment analysis may need to consider the degree of independence of these 3 studies.

Section 6.1 In considering background levels further consideration will need to be explored as to how background has changed over the years. In the last 30 years background has come down substantially, but adults by age have different accumulated burdens of lead and thus the internal dose (in bone) maybe more important than current background (another reason for focusing on neurotox in children).

Section 7. I am not sure I am smart enough to understand how these models will be generated. However, in Figure 7.2 it is not clear in the Monte Carlo-based simulations what these rates are. Further description is needed.

Dr. Barbara Zielinska

Comments on the OAQPS Analysis Plan for Lead Risk Assessment, Draft 05/31/06
Section 3.0 – Overview of Analysis Plan

Submitted by Barbara Zielinska

In my opinion, the general approach presented in the overview of the analysis plan seems to be suitable for estimating human exposure and health risk assessment. The "case study" approach focusing on three cases that represent particular types of ambient Pb emissions and exposure scenario (primary lead smelter, other significant stationary sources and near roadway re-entrainment), is appropriate. I have a few specific comments/questions:

1. The document proposes using ambient monitoring data, when available, and supplements them with modeling data, if necessary. I strongly agree with this approach. My question is if Pb concentrations measured in TSP (as specified by the EPA method) or PM_{10} are going to be used? This could make a difference…. Also, quarterly data or possibly better time-resolved data? 30-day averaging time was recommended for Pb NAAQS in 1990.

2. The "background" is defined as sources or exposures associated with pathways that do not involve ambient air (such as indoor Pb paint, Pb in food or drinking water). My question is if the Pb concentrations resulting from natural sources would be considered, i.e. policy-relevant background conditions. It is not clear what the background lead concentrations in the US are. Chapter 2 cites the significant annual worldwide emissions of lead from natural sources (median 12,000 of metric ton/year) but it does not give the data for the US. I assume this would be a small number in comparison with estimated emissions from anthropogenic sources (Chapter 2 is rather vague regarding this subject), but some specific data are needed.

3. The use of source apportionment to estimate contribution to ambient air concentrations by particular emissions categories seems like a good idea to me. What kind of model is considered? Chemical mass balance (CMB) or positive matrix factorization (PMF) analysis? CMB seems to be adequate, if sufficient source profiles are available.

4. The re-entrainment of historically deposited lead (through resuspension mechanism) seems to be the main exposure pathways in most areas in the US. As pointed out in Chapter 2 of the CD, the stationary and mobile source emissions account only for about 10% of the total lead emissions in the South Coast Air Basin in California; the remaining 90% of emissions are from resuspended soil. Thus, this source of emissions has to be estimated and included in risk analysis.

5. Near roadway re-entrainment exposure scenario - what about San Joaquin Valley in Central California? I believe that the large data base is available.

NOTICE

This letter 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 letter 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 letter and reports are posted on the SAB Web site at: http://www.epa.gov/sab.