UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

May 25, 2011

EPA-CASAC-11-007

The Honorable Lisa P. Jackson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, D.C. 20460

Subject: Consultation on EPA's *Draft Integrated Review Plan for the National Ambient Air Quality Standards for Lead*

Dear Administrator Jackson:

EPA's Clean Air Scientific Advisory Committee (CASAC) Lead Review Panel held a public advisory teleconference meeting on May 5, 2011. The purpose of the teleconference was to conduct a consultation with staff from the Agency's National Center for Environmental Assessment (NCEA) and EPA's Office of Air Quality Planning and Standards (OAQPS) on the Agency's *Integrated Review Plan for the National Ambient Air Quality Standards for Lead* (External Review Draft, March 2011). The Panel generally found the *Integrated Review Plan* to be a useful roadmap for the upcoming Lead National Ambient Air Quality Standards (NAAQS) review.

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 CASAC Lead Review Panel roster is in Enclosure A of this letter and individual CASAC Lead Review Panel members' written comments are provided in Enclosure B.

Sincerely,

/signed/

Dr. H. Christopher Frey, Chair
CASAC Lead Review Panel

Enclosures

NOTICE

This report has been written as part of the activities of the EPA's Clean Air Scientific Advisory Committee (CASAC), a federal advisory committee independently chartered to provide extramural scientific information and advice to the Administrator and other officials of the EPA. CASAC provides balanced, expert assessment of scientific matters related to issues and problems facing the Agency. This report has not been reviewed for approval by the Agency and, hence, the contents of this report do not necessarily represent the views and policies of the EPA, nor of other agencies within the Executive Branch of the federal government. In addition, any mention of trade names or commercial products does not constitute a recommendation for use. CASAC reports are posted on the EPA Web site at: http://www.epa.gov/casac.

Enclosure A - Roster

U.S. Environmental Protection Agency
Clean Air Scientific Advisory Committee
CASAC Lead Review Panel (2010-2013)

CHAIR

Dr. H. Christopher Frey, Professor, Department of Civil, Construction and Environmental Engineering, College of Engineering, North Carolina State University, Raleigh, NC

MEMBERS

Mr. George A. Allen, Senior Scientist, Northeast States for Coordinated Air Use Management (NESCAUM), Boston, MA

Dr. Herbert Allen, Professor Emeritus, Department of Civil and Environmental Engineering, University of Delaware, Newark, DE

Dr. Richard Canfield, Senior Research Associate, Division of Nutritional Sciences, Cornell University, Ithaca, NY

Dr. Deborah Cory-Slechta, Professor, Department of Environmental Medicine, School of Medicine and Dentistry, University of Rochester, Rochester, NY

Dr. Cliff Davidson, Professor, Civil and Environmental Engineering, Syracuse University, Syracuse, NY

Dr. Philip E. Goodrum, Senior Project Manager, Environmental Resources Management (ERM), Dewitt, NY

Dr. Sean Hays*, President, Summit Toxicology, Allenspark, CO

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

Dr. Chris Johnson, Professor, Department of Civil and Environmental Engineering, Syracuse University, Syracuse, NY

Dr. Susan Korrick, Assistant Professor of Medicine , Department of Medicine, Brigham and Women's Hospital, Channing Laboratory, Harvard Medical School, Boston, MA

Dr. Michael Kosnett, Associate Clinical Professor, Division of Clinical Pharmacology and Toxicology, Department of Medicine, University of Colorado Health Sciences Center, Denver, CO

Dr. Roman Lanno, Associate Professor and Associate Chair, Department of Evolution, Ecology, and Organismal Biology, Ohio State University, Columbus, OH

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

Dr. Joel Pounds, Scientist, Cell Biology & Biochemistry, Biological Sciences Division, Battelle - Pacific Northwest National Laboratory, Richland, WA

Dr. Michael Rabinowitz*, Geochemist, Clinical Instructor in Neurology, Harvard University, Newport, RI

Dr. William Stubblefield, Senior Research Professor, Department of Molecular and Environmental Toxicology, Oregon State University, Corvallis, OR

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

Dr. Gail Wasserman, Professor of Clinical Psychology in Child Psychiatry, Division of Child and Adolescent Psychiatry, College of Physicians and Surgeons, Columbia University, New York, NY

Dr. Michael Weitzman, Professor, Pediatrics; Psychiatry, New York University School of Medicine, New York, NY

*Did not participate in this review.

SCIENCE ADVISORY BOARD STAFF

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Enclosure B

Compendium of Individual Comments

CASAC Lead Review Panel

Integrated Review Plan for the National Ambient Air Quality Standards for Lead

(External Review Draft, March 2011)

Comments from Mr. George A. Allen

These comments focus on chapters 5 (risk and exposure) and 6 (monitoring).

Chapter 5: Quantitative risk and exposure assessments

As noted in this chapter, it is very difficult to link typical (near or below the current Lead NAAQS) ambient air lead (Pb) concentrations to the bio-indicator of dose (blood Pb levels), since inhalation is not a primary exposure pathway. Thus ambient air concentrations are a poor indicator of potential dose (we can't even talk about exposure in this framework). Pb is a unique primary NAAQS in this respect. These issues are explained well, and make clear the need to better understand and quantify this linkage to the extent that it is relevant in the Pb exposure framework. The disconnect between air Pb and dose is somewhat similar to the NOx-SOx secondary NAAQS that is in the final stages of the review process. In both cases the NAAQS framework is constrained to measurements of the respective pollutants in ambient air, but the endpoints (aquatic acidification index for the Oxides of Nitrogen (NOx) and Sulfur Oxides (SOx) secondary NAAQS) are driven by other mechanisms. For Pb, it is primarily Pb in dirt, and for NOx-SOx it is deposition (primarily wet). Neither NAAQS can directly measure what is most important in terms of health or welfare effects, making a rational health or welfare assessment very difficult.

There are very large spatial variations in elevated air Pb concentrations for all particle size ranges. Elevated levels of concern are only found near sources. Fine mode lead (from combustion sources such as smelters) disperses rapidly, with minimal local deposition and a limited area of elevated concentrations (driven in part by low background Pb air levels). Coarse (or larger) mode lead particles (from mechanical sources) deposit rapidly, near the source. A complicating factor in exposure assessment is existing (historical) Pb in soils, usually from lead paint from before the late 1970s. Soil Pb becomes airborne usually by wind or vehicle turbulence. However the temporal and spatial patterns of Pb in air from soil are very complex and difficult to measure. Modeling could be done if sufficiently detailed mapping of soil Pb were available, but it is generally not (other than from special studies).

The linkage between ambient air Pb levels and indoor dust Pb from outdoor Pb air concentrations is especially weak, but indoor dust is one of the major (ingestion) exposure pathways for children. For homes with lead paint, this linkage becomes almost impossible to assess and is probably irrelevant. Because of the local soil's Pb "history", this linkage gets even weaker if you consider how the ambient air component of indoor dust Pb levels might decline with a decline in ambient air Pb levels.

I am not aware of significant progress in improving estimates of air-related Pb concentrations that are relevant to human health. Lack of sufficient measurement data that addresses this issue is the limiting factor, and that has not changed much since the last review.

Chapter 6: Ambient Air Monitoring Considerations

Monitoring methods:
http://replay.web.archive.org/20010619094708/http://rpco.com/cartoons/c199801.htm
http://replay.web.archive.org/20010527041057/http://rpco.com/cartoons/c199904.htm
http://replay.web.archive.org/20010513124050/http://rpco.com/cartoons/c199811.htm
http://replay.web.archive.org/19990830175531/http://rpco.com/cartoons/c199806.htm

The Hi-Volume (HiVol) Federal Reference Method (FRM) sampler has long outlived its design lifetime; it was first used in the mid 1940's to sample PM levels from welding fumes. Its fundamental design has not changed. If the HiVol instrument were proposed today as an FRM, it would not get any consideration as a plausible method. Others have pointed out its many shortcomings; I will not repeat them here. I will state clearly that its continued use as an FRM sampler for Pb in the 21st century is indefensible. In addition to its poor performance, the HiVol is loud and resource intensive (when only 1 or 2 are run by an air agency); equipment and methods for flow calibrations and audits are unique to this sampler.

There is only limited evidence to suggest that it is important to sample particles larger than 10 um to characterize exposure to Pb from air. HiVol Pb can sometimes be moderately higher than PM10, as presented in previous CASAC and Pb NAAQS reviews. However, there is no value to sampling particles larger than 20 um; larger particles deposit rapidly, making the spatial scale of representation too small to be meaningful. Height of sampler inlet above ground becomes a critical sampling parameter above 10 um. Current EPA monitoring requirements allow this height to be 2 to 7 meters for “micro-scale” sites, and 2 to15 meters for all other sites. This is a huge range for particles larger than 10 um; you substantially change the spatial scale of the monitor by changing the inlet height. EPA should consider a much smaller range of inlet height in this round of NAAQS evaluation; 3 to 5 meters (regardless of micro-scale siting or not) might be more appropriate for consistency in network data.

If there is sufficient concern about capturing Pb aerosol greater than 10 um, it is practical to design a new sampler with well characterized performance at reasonable wind speeds and a D50 cutpoint of ~ 18-20 um. Unlike PM2.5 or PM10 FRM samplers, the cutpoint for this method does not have to be sharp -- just well characterized and stable with wind speed and direction.

The existing FRM louvered inlet has been characterized over the size range of interest, but only at low wind speeds (1 m/s or 2.2 mph); the aspiration efficiency at 1 m/s = 1.00 at 15 um, 0.68 at 24um, and 0.35 at 45um. Values are approximate, from Figure 7 in “Aspiration and sampling efficiencies of the TSP and louvered particulate matter inlets.” Kenny et al., J. Environ. Monit., 2005,7,481-487.

EPA’s Office of Research and Development (ORD) needs to evaluate this inlet further by wind-tunnel testing it as required for PM10 inlets:
http://ecfr.gpoaccess.gov/cgi/t/text/text-idx?c=ecfr&rgn=div6&view=text&node=40:5.0.1.1.1.4&idno=40

Texas A&M is working to once again have a functioning facility for this purpose (EPA does not have a working wind tunnel suitable for these tests). Initial tests should first be done at the highest wind speed of concern (24 km/h) -- which will have the lowest D50. At lower wind speeds, the inlet aspiration efficiency should approach the data reported by Kenny.

To be of use in this round of the Pb NAAQS review process, ORD needs to start work on inlet testing now. This effort would include working with Texas A&M to validate their re-built wind tunnel. There should be sufficient resources for this effort within ORD's existing budget, but unless this task is made a high priority for funding, it may not happen.

If deemed essential (and only if), there is an upcoming opportunity to gather additional data on a relative large scale (25 sites nationally) for Pb in fine, coarse, and "TSP" fractions using the upcoming EPA PM-coarse speciation dichot network. If HiVol samplers were added to some or all of these sites for a year, a robust data set of Pb in these 3 size modes could be generated to assess the extent of "uber-coarse" particles as measured by the HiVol.

Network Design

The existing network design, including the modifications in the Dec. 27, 2010 FR, is sufficient to provide oversight to stationary sources that could contribute to elevated air Pb concentrations, and to track long-term urban lead trends (urban NCore sites). This new network design is not sufficient to allow estimation of spatial gradients; it never will be given resource constraints and the large spatial variability of Pb in air.

Although I support EPA's efforts to remove Pb from aviation gasoline (AvGas) as a long term goal, I do not agree with EPA that general aviation airports are a substantial source of elevated Pb in "ambient" air near airports. However, EPA's Office of General Counsel has defined ambient air to include the air on the tarmac at the location of maximum concentration. With this siting criteria, it is possible that elevated Pb levels will be monitored, and these HiVol monitors would become "permanent".

Ambient air Pb exposures to populations living near or adjacent to airports has been shown to be similar or slightly elevated compared to relevant background levels, and not a substantial health issue. If the EPA airport sampler siting does show exceedances of the "never to be exceeded" 3-month Pb NAAQS, there is very little a local air agency could do to reduce monitored Pb concentrations. Any effort to do so would have minimal to no benefit to human health, given that no one stays near the (hot spot) location of the sampler for 3 months (e.g., the sampler does not reflect a NAAQS-relevant Pb exposure to anyone).

The Dec. 27, 2010 EPA regulation requires Pb monitoring at all airports with estimated annual emissions of 1 ton or more, a reasonable approach. There are only a handful of such airports in this country. The same regulation requires a 1-year pilot study at 15 additional specific airports with estimated Pb emissions between 2 and 1 ton per year; HiVols (with break-away stands) are required for those sites. Especially in these difficult economic times, large-scale general aviation (GA) airport monitoring is not an appropriate use of very limited local air agency resources; there is more important (non-Pb) monitoring to do that is much more health-relevant.

Comments from Dr. Herbert Allen

Overall, the document is well-written and considers most major points. There should be additional consideration to the following points.

Should biomarkers of exposure/effects be considered? For example, there is a large and expanding literature on reactive oxygen species (ROS). Biological effects are well-documented. A quick look reveals hundreds, if not thousands, of citations. If, for example, stress proteins are considered, should multiple metal (and other toxicant) exposures also be considered? This is an important consideration because many effects result not only from exposure to lead, but also from numerous other metals and also other contaminants. If effects are a result of exposure to multiple contaminants, then setting a standard based on a single contaminant will result in underestimation of risk. Other approaches are available and have been applied, for instance explicit modeling of additivity of toxicants or approaches such as expression in terms of dioxin equivalents.

The document has not considered EPA 120/R-07/001 Framework for Metals Risk Assessment at all. This document indicates that the chemistry of metals is of paramount importance. This is well-known in aquatic toxicology and in soil contamination. To reduce bioavailability of lead in contaminated soil, phosphate is added to form pyromorphite, a lead-containing mineral with a reduced solubility in ingested soil. All chemical species containing lead do not have equal bioaccessibility. This is ignored in assessment and analysis where the only consideration to differentiation of lead forms is by size of particles considered. Is the bioavailability of lead in dust at an area contaminated from mining, such as at the Couer d'Alene Superfund Site, the same as that in an old house which has lead-based paint? If the bioaccessibility and bioavailability are different for the same size particulate matter, is a single value of a criterion or standard appropriate? The level of protection or risk for the two types of material will not be the same. This will result in either over or under protection.

For some routes of exposure there are multiple Pb sources, some of which may have originated in air whereas others are not airborne. Effects in organisms depend on exposure not the material source. Lead in vegetables grown in urban gardens may have originated from deposition of airborne lead onto the soil. If a person eating those vegetables is also exposed to lead in drinking water resulting from corrosion of lead pipe, there are two sources that must be considered in the risk assessment. A lead standard that considers the atmospheric route only will under protect the person having other, non-negligible, exposure to lead. In this case the lead from the plumbing cannot be ignored. In aquatic toxicology both water and food as sources of metals is being considered more frequently. For another example, a child may be exposed to lead from paint and from dust.

The Primary and Secondary Standards are on a "not-to-be-exceeded" basis. Such a basis is not sound. Concentrations of all substances in the environment vary. There is a finite probability that any value will be exceeded. The revised means of averaging will lower the probability, but will not reduce it to zero. The question that should be addressed is what remedial action should be triggered by an exceedance? In light of the nuclear reactor failures in Japan, we should

become more aware of low probability, but high consequence events. Failure of the air pollution control system at a secondary lead refinery or explosion at airport leading to dissipation of stored gasoline are two scenarios that could result in very highly elevated lead exposure and exceedance of the standards.

Comments from Dr. Richard Canfield

I wish to commend the EPA for producing a very strong draft integrated review plan for evaluating the National Ambient Air Quality Standards for Lead. After a close review of the Integrated Science Assessment (ISA) section I found it to be well organized, clearly written, and quite comprehensive. I have a few comments, most of a fairly specific nature.

p. 4-5 line 1 - It might add clarity and focus to point 4 by including the criterion of "validity" to the criteria "meaningful and reliable." Validity is a more specific term than meaningful in that it refers to whether a given health effect measurement measures what it is intended or claimed to measure (internal validity).

p. 4-5 lines 28-33 - The text suggests but does not explicitly state that studies conducted on U.S. populations will, given acceptable quality, be considered as among the most informative. Also, the issue of how to incorporate results from studies conducted on non U.S. populations can require subtle judgments that depend on how one construes the phrase "the corresponding U.S. population." If a general population from a study in a non U.S. sample has a higher mean blood Pb than the general U.S. population then it could still be that the results of the study are relevant to a well-defined subpopulation in the U.S. that experiences higher than average exposures. It would be helpful to provide a little more detail on how such judgments will be made. Precisely this sort of detail is provided when discussing research with laboratory animals (p. 4-6 lines 4-7).

p. 4-6, 4-7 section 4.2.4 Quality Assurance - The document refers to an "Agency-wide Quality Management System" but does not provide adequate information about how it functions. Some more detail in the text would be helpful and a reference to a document describing how this system is implemented and monitored at EPA should be added.

p. 4-8 line 27 (and throughout document) - In some places it is unclear whether "ambient Pb" is meant to refer only to "ambient air Pb."

p. 4-9 lines 30-35 (point 6) - I found this paragraph difficult to parse. Can it be clarified what "this relationship" refers to (line32)? The word "relationship" is used three times and in combination with "variation" and "changes" and so I'm getting lost trying to think about variations in relationships of relationships.

p. 4-11 lines 18-20 - The final question posed under point 5 seems sufficiently distinct to stand alone.

p. 4-11 line 40 through 4-12 line 2 (point 2.) - This question mentions uncertainties about lead effects estimates due to confounding factors. The list includes genetic susceptibility, which would be a true confounder if it is associated with increased blood lead level and causes differences in the health endpoint through a non-Pb mechanism. This should be distinguished from genetic factors that act as an effect modifier such that the Pb effects estimates differ across genetic subgroups with the same blood Pb level. Genetic susceptibility as an effect modifier is covered on page 4-12 under susceptible populations and life stages. If it is plausible that genetic

factors act as a true confounder then it should be clearly noted in the question. If it is only believed to be an effect modifier then it should not appear on the list under point number 2.

Similarly, how does occupational exposure (to Pb, I assume) function as a confounder? It might be helpful to organize this part of the ISA by considering confounders, effect modifiers, and precision of estimate variables in clearly identified sections. Of course there will be some overlap but this would make the conceptual issues regarding the status of any particular "third variable" more evident to the reader.

p. 4-12 lines 28-40 - There is inconsistency in that on line 29-30 susceptibility refers to likelihood of experiencing health effects of exposure whereas in other places (line 37) susceptibility refers to likelihood of exposure to Pb per se. On line 40 it is unclear whether susceptibility is meant to refer to exposure, health effects, or both. This should be clarified. Also, from a developmental science point of view, "childhood" is a not a well-defined life stage. Infancy and adolescence are associated with the sorts of dramatic changes in brain and behavior that often produce periods of special vulnerability to environmental insults.

p. 4-13 - Again, clarify the use of "susceptible" throughout.

p. 4-13 lines 22-27 - Will the economic burden of the health effects be considered in the discussion of public health implications/significance?

Comments from Dr. Deborah Cory-Slechta

Chapter 3 - Key Policy-Relevant Issues

The EPA staff is to be commended for its generally thorough coverage of the Review Plan presented in the various chapters of the document as well as the strategy to leverage the new review on both the past experience as well as updated information since that time.

Key to the policy-relevant issues is the assessment of the adverse consequences of lead in a multimedia, multipathway context. The derivation of a NAQQS for lead in air in the absence of this recognition would never achieve the stated purpose of deriving such values. The structuring of EPA offices largely based on legislative requirements can to some extent lead such artificial distinctions.

Although explicitly stated in the issues related to the Secondary NAAQS, the issue of recognition of multimedia, multipathway is not stated for the primary NAAQS policy relevant issues.

In accord with new and concerted efforts within the Agency, it would seem that cumulative risks as related to lead exposure should clearly be a policy-relevant consideration. While cumulative risk can be considered in different contexts, in the Primary NAAQS, this would include the potential for either enhanced effects of lead, or for lead effects to be present at lower levels of exposure, when such lead exposures occur in the presence of other environmental/occupational exposures, and/or in combination with other risk factors for human diseases and disorders (underlying co-morbidities, stress, nutritional insufficiencies or deficiencies, etc.), where such data are available. Such scenarios are far more consistent with human environmental exposures than is a situation where lead would be the only environmental exposure or risk factor present.

Comments from Dr. Cliff Davidson

I only have a few comments on the Science Assessment section, Chapter 4. On page 4-4, it is not clear to me who does the reviews and makes the decisions on which studies to include (lines 20-21).

I am not sure why the Criteria for Study Selection (section 4.2.3) only address health and welfare effects. Should any of the topics in Section 4.3 Content and Organization of the ISA have criteria listed for studies to be included in the ISA?

Lastly, there will not be a huge number of studies since 2006, and it takes a long time to get studies published. Is there a mechanism for including studies that may be relevant but have not yet been published? (e.g., are in the middle of peer review)

Comments from Dr. Philip E. Goodrum

Chapter 5 - Quantitative Risk and Exposure Assessment

In general, the draft Integrated Review Plan is well conceived and clearly presented. The focus of the updated ISA and Risk and Exposure Assessment (REA) is to determine if more recent empirical data and modeling approaches provide sufficient evidence to warrant changes to the existing NAAQS. The multi-media, multi-pathway exposure component of a lead risk assessment presents challenges, particularly as the emphasis increasingly shifts to quantifying the contribution of air-borne lead to the dose-response relationship at lower cumulative doses.

A logical starting point for the re-assessment is to build from the uncertainty analysis that was conducted in the previous review. I would encourage EPA staff to carefully evaluate the previous work and subsequent literature to consider whether sufficient evidence is available to distinguish between outcomes of the uncertainty analysis in low-dose versus high-dose regions of the dose-response relationship. Uncertainty in estimates of exposures via inhalation, diet and water ingestion, for example, may be more critical at the low dose region, especially if the risk metric is defined by an "absolute" blood lead (PbB) concentration threshold (e.g., probability of exceeding 5 or 10 µg/dL). At some point, it may become impractical to expect to isolate the relative contribution of one exposure medium (e.g., air) when the distribution of blood leads at low doses is heavily dependent on a suite of factors.

EPA intends to continue evaluating variability and uncertainty using a probabilistic approach (Monte Carlo analysis). This is particularly useful for understanding relative contributions of various exposure factors to the estimate of an average daily dose (uptake). Previously, the limitation of PRA for lead risk assessment has been the limited information available to incorporate probabilistic methods in the biokinetic component of the model; therefore, the distribution of blood lead concentrations was interpreted as underestimating the likely variance in an exposed population. To advance the methods used to inform the previous risk assessment, literature reviews should focus on understanding the likely magnitude of variance in blood lead that can be attributed to biokinetics.

The list of limitation, assumptions, and uncertainties given on pages 5-3 and 5-4 captures the key factors well, and should help to focus the reassessment effort. Care should be taken to try to distinguish between estimates of variability and estimates of uncertainty.

For the ecological risk assessment, in addition to focusing on specific case studies, databases developed from site assessments should be considered – particularly to understand how conditions at sites within the same watershed may vary.

Pages 5-9 and 5-10. EPA staff should consider the evaluations of the National Health and Nutritional Examination Survey (NHANES) data as an additional resource for matched PbB/ dietary lead levels.

p. 5-10. There is discussion of the use of empirical data to estimate the Geometric Standard Deviation (GSD) parameter. EPA staff should be careful to control for variability in media concentrations if these new data are to be used to derive a plausible range of GSD for modeling purposes.

Comments from Dr. Philip Hopke

There are several aspects of the ambient monitoring and related choice of indicators that needs to be addressed to the extent it can. We need to have as much understanding as possible of the relationship between Pb in TSP and Pb in PM10 and the variation of these values as a function of location across the U.S. If lead is primarily from aviation gas, then the lead in PM10 and TSP will be essentially identical whereas sources like secondary lead smelters my produce significant quantities of coarse particle lead. In that case, the variation is size distributions may make it difficult to ensure that a PM10 standard provides the same level of protection as a Pb in TSP standard.

There is also a significant need to move away from low precision high volume samplers. It is quite possible to design high volume samplers with adequate precision in their flow control to match that of low volume samplers. It is also possible to provide TSP heads for low volume samplers (several are commercially available, but have not been well characterized). It would not take an enormous amount of time or resources to substantially improve the options for ambient monitoring and the ISA that is to be prepared needs to adequately address these issues.

Comments from Dr. Chris Johnson

I agree with the other committee members that the draft IRP was well done. Having participated in the workshop on science/policy issues last year, I can affirm that the draft IRP incorporated the essential elements of those discussions.

The comments below are revisions of the preliminary comments that I submitted prior to the teleconference on 5/5/11.

Page 4-5, lines 8-20 - In selecting studies to consider, it is probably best to cast a wide net. Setting a quantitative example for the "low end of the exposure distribution" in item #1 (< 10 ug/dL blood) here may result in the omission of valuable studies measuring effects at slightly higher levels.

Page 4-5, line 12, and throughout the document - Has the EPA determined an agreed-upon list of "susceptible populations?" If so, it should be clearly stated here and in the ISA. If not, then one of the goals of the ISA should be to identify those populations that are judged to be susceptible.

Page 4-7, lines 8-9 - ~~How does EPA propose to assess the quality of data found in the peer-reviewed literature?~~ After the teleconference, I am satisfied with the EPA's approach. This is no longer an issue for me.

Page 4-13, lines 1-3 - Item 1a: How realistic is it to use animal studies of susceptibility factors to identify susceptible human populations?

Page 4-14, lines13-14 - To the best of my knowledge, there is no general agreement on which terrestrial receptors are particularly valuable as indicators of risk. Thus, the identification of key terrestrial ecosystem receptors useful for risk assessment would be a valuable contribution in this ISA process, even if there are few data on specific effects.

Page 4-14, lines 7-35 - Critical loads are generally calculated on the basis of chemical flux rather than air concentration. Could a secondary standard be developed on the basis of, say, atmospheric deposition of Pb rather than air concentration? If not, are relationships between air Pb concentrations and atmospheric Pb deposition sufficiently strong to "translate" a flux-based critical load into an air Pb concentration standard?

Page 6-3, lines 7-16, Figure 6-1 - It is apparent that the national monitoring network is problematic at best and insufficient at worst. It appears that most of the sites in the network were not in service until very recently, and even fully implemented it is a rather sparse spatial density. The ISA should address the likely effectiveness of this network to monitor Pb standards attainment.

Comments from Dr. Susan Korrick

Overall the draft IRP is well-written and provides a thoughtful and thorough overview of the key scientific questions and policy-relevant issues that will be considered in the current review of the NAAQS for Pb. I have some general comments:

(1) Some of the plan appears to be predicated on the implicit assumption that young children and IQ represent, respectively, the key population and health effect of concern whereas elsewhere it is acknowledged that other populations (e.g., other age groups) and health outcomes may be important. The plan needs to incorporate breadth and flexibility to comprehensively and consistently assess the latter possibility.

(2) As an extension of comment #1, it would be useful at this planning phase to be more specific about other populations and/or health outcomes of potential interest. E.g., childhood neuropsychological outcomes other than IQ (e.g., as described by Dr. Wasserman, executive function development during adolescence) may be even more sensitive to Pb exposure than global cognition measures such as IQ. The potential role of Pb as a risk factor for adverse childhood growth and pubertal development (as described by Dr. Kosnett) and even diabetes risk are also areas of public health importance not explicitly acknowledged here.

(3) It is not clear how the review might address the potential for temporal issues (differences in the timing and duration of exposure) to affect health risk. E.g., health risks may vary by when exposures occur (prenatal vs. childhood vs. adult), exposure rate (episodic high-level vs. chronic low-level), exposure duration (short- vs. long-term), and exposure latency (recent vs. past). These issues may be increasingly important as overall background Pb levels decline. E.g., long term exposures to even very low-level Pb may have a more deleterious effect than relatively short-term exposure to the same Pb level. Accounting for such issues is challenging but important as overall Pb levels decline.

(4) Because a relatively short time has elapsed since the last review, the scientific literature available at the last review (pre-2006) will likely still be relatively central to this new review.

(5) Because a short time has elapsed since the last substantial revision of the NAAQS for Pb (from 1.5 $\mu g/m^3$ since 1978 to 0.15 $\mu g/m^3$ as of 2008), changes related to the new Pb NAAQS have not yet been implemented (except perhaps in a few isolated cases in which point sources have changed or will shut down operations in the near future as discussed on May 3). It is unfortunate that it is premature to collect data to assess the efficacy of the current standard. E.g., given uncertainties in exposure modeling and characterization, such data would otherwise constitute an important context for assessing the scientific basis for any revisions to the standard.

Comments from Dr. Michael Kosnett

Overall, the IRP presents an approach to developing the Integrated Scientific Assessment and the Risk/Exposure Assessment that is quite comprehensive. There is appropriate attention to key issues of uncertainty and variability identified in the last reviews conducted for the lead NAAQS.

I. Comments related to Chapter 5 (Quantitative Risk and Exposure Assessments)

1. The narrative appropriately notes the value of reviewing datasets that have examined the impact of housing age on interior dust lead, and the concurrent contribution of housing age (as a surrogate for lead paint) and interior lead dust on blood lead in children. The relatively recent availability of a large dataset examining the inter-relationship of housing age, interior lead dust, and childhood blood lead in a subset of the NHANES 1999 – 2004 investigations (Gaitens et al, 2009; Dixon et al, 2009) may be informative in this regard.

2. In like manner, the recent NHANES studies incorporating data on house dust lead and childhood blood lead (Dixon et al, 2009), and data from other investigations such as the HUD National Risk Assessment Study (Wilson et al, 2007) and low blood lead subsets extracted from the pooled dust lead analysis by Lanphear et al (Lanphear et al, 1998), might offer a means of examining empiric relationships between low levels of interior lead dust, soil lead (available in some of the studies), and blood lead. The findings from this empiric data could be productively compared to the results yielded by the Integrated Exposure Uptake Biokinetic (IEUBK) model.

3. Certain studies conducted at Superfund sites in the past decade are likely to offer information on the inter-relationships between lead in various media (i.e. exterior soil, interior dust, ambient air) that could be useful in the upcoming lead NAAQS review. For example, the Baseline Risk Assessment for the Vasquez Boulevard/Interstate 70 Superfund site in Denver, CO contained empiric data on the relationship between outdoor soil lead and indoor lead dust in an urban residential community unaffected by recent lead emissions from a point source (EPA, 2001). Datasets such as this might assist EPA in refining the hybrid indoor dust lead model it utilized in the last NAAQS review. In like manner, it may be valuable for EPA to examine recent empiric datasets in evaluating the fs parameter (fraction of total soil/dust mass ingested daily by a child that comes from soil), a term that has substantial impact on the output of IEUBK modeling.

4. A few recently published studies (e.g. Manton et al, 2005; Gulson et al, 2006) have utilized measurement of stable lead isotopes to examine the inter-relationships of lead in various media (air, dust, diet). These studies might contribute useful information to the lead exposure pathway analyses that will be examined by EPA in the ISA and REA documents. A recent article using isotopic analysis reported that lead in the soluble fraction of PM2.5, but not the insoluble fraction, contributes to blood lead in urban residents (Chen et al, 2009).

5. In preparing its analysis of the impact of lead derived from air emissions on blood lead concentrations, EPA is encouraged to consider an “incremental” risk assessment approach, in which modeling is used to identify a level of lead from air emissions that would increase blood lead concentration of the population by a designated target increment, e.g. 1 µg/dL. For example,

applying the IEUBK model, a level of lead in "air-related" pathways could then be identified that would result in a designated percentile of the blood lead distribution (e.g. the 90th percentile) reaching a "benchmark" increment in blood lead of 1 µg/dL. Such an approach has been adopted by the Office of Environmental Health Hazard Assessment of the California Environmental Protection Agency, which, using certain health protective assumptions, associated a 1 µg/dL increment in blood lead with a 1 point decrement in childhood IQ (Carlisle and Dowling, 2007; Carlisle, 2009). Alternatively, using an empirical dataset, standardized coefficients or partial regression plots examining the slope of the relationship between "air-related lead" to blood lead at low levels could be used to identify a level of "air-related lead" that would increase blood lead by 1 µg/dL. It may be noted that such an approach has recently been recommended by the EPA Science Advisory Board review panel for lead dust hazard standards.

6. The effect that the aerodynamic size of airborne lead particulate may exert on blood lead concentration in exposed individuals has been studied in occupational settings by Froines and colleagues. Although the exposures in question were considerably higher than what would be expected from lead in ambient air, these studies may nonetheless be of interest to EPA in assessing the toxicokinetics of inhaled lead in humans (Froines et al, 1986; Froines et al, 1995). This may be of importance in assessing the risk posed by airborne lead from sources that have significantly different particle size (e.g. stack emissions versus re-entrained lead contaminated soil).

II. Comments related to chapter 4 (Science Assessment)

1. The narrative describes EPA's intent to review the recent literature pertaining to the impact of lead of multiple health endpoints. In the prior NAAQS review documents, the impact of lead on childhood IQ emerged as a primary endpoint of concern, and that is likely to remain the case in the current review. However, it is possible that the current NAAQS may be able to include an expanded discussion of the impact of lead on the risk of cardiovascular morbidity and mortality, particularly in light of recent studies (e.g. Menke et al, 2006; Weisskopf et al, 2009). In addition, the potential contribution of ambient lead particulate in different size fractions to the oxidant stress exerted by ultrafine and fine ambient air pollution may be of interest, given the considerable public health concern that exists regarding the effect of such pollution on cardiovascular disease (Brook et al, 2010). Certain studies suggest that lead may contribute to inflammatory effects in the lung of experimental animals exposed to concentrated ambient air particulate (e.g. Saldiva et al, 2002; Seagrave et al, 2006).

2. Studies published since the last NAAQS review may now permit an expanded examination of the impact of low levels of prenatal and postnatal lead exposure on childhood growth and stature, and certain endocrinological endpoints (e.g. Selevan et al, 2003; Min et al, 2009).

3. With respect to "ecological and other welfare effects", the IRP indicated that it will not consider the extent to which lead ammunition contributes to the food chain and lead body burden of wildlife (page 4-13), noting that this is not a source "relevant to consideration of air-related lead." Nonetheless, it may be still be helpful for the ISA to briefly discuss the extraordinarily high lead burden experienced by a significant proportion of certain predatory birds (such as bald eagles and golden eagles) that is attributable to lead ammunition, in order for the cumulative

impact of lead from all sources (including air-related pathways) to be considered. For example, a recent study of bald eagles and golden eagles in the Yellowstone ecosystem (n = 63) reported a median blood lead concentration of 41 µg/dL, with 74.9% of the birds having blood lead concentrations greater than 20 µg/dL (Bedrosian and Craighead, 2009).

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Comments from Dr. Roman Lanno

Here are my comments on the IRP document from a terrestrial ecotoxicology perspective on Chapter 4. In the context of a planning document, this document was well written and organized, and cast a broad net of questions to assess. My specific comments are provided below.

Page 4-8: Line 6 – What is specifically meant here by the "organisms' biochemical characteristics"?

Bioavailability is affected the physical, chemical, and biological nature of the interaction between the organism, Pb, and components of the environmental medium in which it resides. So, in this context, the bioavailability of Pb cannot be defined by the organisms' biochemical characteristics alone, but the entire conceptual model needs to be considered. Additionally, there is a wide diversity of biochemical responses to Pb exposure by different taxa of invertebrates and plants. The biochemical endpoints to be considered must be clearly defined. The effects of aging, i.e., soil-Pb contact time, also need to be considered.

Page 4-12: It's unclear to me why both < 10 μg/dL or < 5 μg/dL are stated – seems redundant; perhaps a two-fold difference is meaningful here

Page 4-14: Lines 13-14 – This probably should explicitly state "specific exposure levels related to aerial Pb". The focus of this review is on either dry or wet aerial deposition of Pb, which should be distinct from other Pb sources. However, the distinct fate of aerial Pb from other sources is unclear at best. We do have information on the toxicity and fate of Pb in terrestrial environments in a couple of excellent syntheses of Pb toxicity and fate in the terrestrial environment given in the EPA 120/R-07/001 Framework for Metals Risk Assessment and the US EPA Ecological Soil Screening Levels for Lead (OSWER Directive 9285.7-70, 2005) that need to be considered.

Also under point 3: What are the effects of aging on Pb in soils that results from aerial deposition?

Comments from Mr. Richard L. Poirot

Overall, the Draft Pb Review Plan looks quite good. The Plan exhibits a very clear and in-depth familiarity with the details of the 2008 Pb NAAQS review process, and provides a sharp focus on a number of policy-relevant issues, which represented key decision points in the last review and which were also characterized by relatively high levels of uncertainty. The plan aims to reduce these areas of uncertainty by posing a series of specific questions which might be clarified by newly available information. This is a logical framework in which key uncertainties in the last review provide the basis for a focused literature review in the current cycle.

However, while there is a long list of good questions that begin with "Does the newly available information indicate…" or "To what extent is there new information to support", there are no examples provided that there is any such new information that will illuminate these questions. It feels like a very short time since the 2008 NAAQS review – or conversely that the pace of new research for air lead causes and effects may proceed more slowly than for pollutants like PM or ozone. I question the extent that there will really be a lot of new information to address all of these questions. This leads to a suggestion that it might be useful to provide a few examples of recent publications (or potential modeling or data analysis activities) which could help reduce uncertainties for some of these key issues. Another possibility would be to start with your recent literature review – presumably not complete but surely in progress – and consider what kinds of new insights the new information you've seen so far can provide. Combining a "questions first" approach with a "literature first" perspective might lead to a more efficient focus on key questions that can be answered (and the ones for which new research, measurements, modeling or data analyses is most needed).

Ambient Air Monitoring Considerations

Among the problems associated with the 30-year failure to revise the Pb NAAQS between 1978 and 2008 is the quaint historical persistence of antique high-volume TSP sampling technology as the basis for the Pb FRM (and for some 24 FEMs). Following the 2008 review, an option was provided to utilize the more modern and precise low volume PM_{10} sampler in situations where expected 3-month mean concentrations are below 0.1 $\mu g/m^3$ (2/3 of standard) and where a substantial majority of Pb is in the PM_{10} size fraction.

Section 6.1 (sampling and analysis) of the Plan poses the question: "Are new TSP samplers available and adequately characterized for use in PB-TSP sampling?" Presumably such new samplers would not exhibit the sampling biases with wind speed and sampler orientation that characterized the old hi-vol TSP samplers – and there have been indications that EPA is developing a medium volume "TSP" sampler that would be superior in all respects to the old hi-vols. While this should certainly be given a high priority, it's not entirely clear what "TSP" means in this case, or more importantly: what should be the particle cut size characteristics of a sampler (or samplers) used to determine compliance with the Pb NAAQS?

Some prefacing questions might be:

- What are the averages and ranges of absolute concentrations (and percentages) of current Blood Lead which originates as current air lead? Let's call this BL(a).
- What are averages and ranges of ratios between current BL(a) and current Air Lead (AL)?
- (How) do these BL(a):AL ratios vary for Air Lead in different particle size ranges (<2.5, 2.5-10, 10-20, >20μm, etc.)?
- What fractions of BL(a) result from direct inhalation vs. aural ingestion of deposited AL?

I'm not sure these kinds of questions have clear answers in the literature, but perhaps could be addressed in some sort of dosimetry modeling. The point being that it would be useful to have some apriori indication of the ideal size distribution & cut point characteristics of Pb samplers that would best reflect human exposures. It would also be useful to consider:

- What are the particle size characteristics of Pb emissions from major Pb sources?
- What spatial scales of population exposure near Pb sources, and how would these be represented by Pb samplers with alternative cut sizes?
- (What) is (there) an upper particle cut size (which may contribute to deposited and ingested Pb but) shouldn't be considered as airborne Pb that can be effectively controlled by NAAQS?
- What would be potential applications for a new "TSP" sampler other than Pb NAAQS?
- For sources exceeding standards would adding something like a dichotomous sampler provide additional useful information on emissions characteristics and population exposures?

Given the inherent physical difficulties of achieving sharp cut points in the $\geq$10 μm size range, it seems likely that a compromise will be required between what's desirable and what's possible. Still, it should be relatively easy to improve considerably on and replace the hi-vol sampler, which would stand no chance of being designated FRM if it were being considered anew today.

Section 6.2 (monitoring network requirements) raises the question of whether current source-oriented monitoring thresholds (0.5 tons/yr for industrial sources and 1 ton/yr for airports are appropriate for determining compliance with current or alternative NAAQS. Given inconsistencies (or changes from one draft to the next) in the identification and ranking of major source categories encountered during the last Pb NAAQS review cycle, it seems important to evaluate (from new monitor data or revised emissions estimates) how reasonable these source-related monitoring requirements are. Useful results along these lines should also be coming in from a "pilot network" of about 15 smaller (0.5 to 1.0 ton) airport sites. Since airport emissions are fuel-combustion related (fine particle mode), and since there would obviously be strong directional influences and typically relatively high wind speeds at airports, the requirement to use antique, directionally and wind speed-biased hi-vols for such sampling seems questionable.

Given that there are likely to be fairly extreme spatial gradients in concentrations and population exposures around major Pb emission sources (especially for those with significant coarse-mode emissions), it would seem important to pay more attention to the details of siting characteristics for source oriented monitoring, and to better evaluate how these relate to exposures of populations at risk.

Comments from Dr. Joel Pounds

The scope and organization of the science assessment is logical and quite thorough. The integrated review plan was clear without significant ambiguities and was well written and organized. This plan gives confidence that preparation of the NAAQS will be efficient and the resulting analysis very appropriate and useful. Two suggestions for criteria revisions are suggested.

Page 4-4. The "Criteria for Study Selection" does not explicitly include evaluation of the adequacy of the analytical methods to support the conclusions. This evaluation is particularly important for evaluation of bone lead measurements by X-ray fluorescence. Of course, the appropriateness of analytical methods should be evaluated in any case. I suggest this section 4.2.3. include a new bulleted items such as,
"Do the analytical methods provide adequate sensitivity and precision to support meaningful and reliable conclusions?"

Page 4-10, Item 7 addresses the new scientific understanding of lead exposure and dosimetry. However, many systems-based approaches and technologies are beginning to be employed for biomarker discovery, toxicity pathway identification, gene-environment interactions, etc. Consider explicit criteria to evaluate new literature and studies related to biological response to lead exposure. Perhaps,

#8. To what extent do new scientific tools (transcriptomics, proteomics, metabolomics, SNPs, etc.) increase our understanding of modes of action, biological markers of lead exposure and response, or exposure-dose-response relationships?

Comments from Dr. William Stubblefield

These comments focus primarily on Chapter 5 (Quantitative Risk and Exposure Assessments) and address issues associated with ecological risk assessment.

In general, the draft PB review plan is very clear, concise, and well-organized. The planned approach seems appropriate and should provide a means to achieve the overall programmatic goals.

The agency should be commended for acknowledging the need to identify and consider the most recent toxicological information in developing the Pb NAAQS. A substantial body of information is now available which is not reflected in the US EPA's current criteria documents (as is indicated on page 5-6). Much of the new information and toxicological data have been developed as a result of industry's efforts to comply with the European Chemical Agency's REACH regulations and the EPA is encouraged to consider this information as efforts move forward on the Pb NAAQS. A greater understanding of the importance of Pb "bioavailability" and its importance in assessing toxicity to non-human receptors in a variety of matrices (water, sediment, soil) is now available and should be considered in the next review of the Pb air-quality standards.

It is noted that the EPA recognizes the difficulty in identifying Pb exposures resulting from air-deposition versus other potential sources of lead, and the importance of being able to identify possible effects to receptor species resulting from the various exposure routes. This will not be easy, but is very important in making sure that the Pb NAAQS is appropriate.

Finally, a great deal of technical advancement has occurred in the last several years that improve our ability to evaluate the potential toxicity of metals in the environment. Methods are now available for incorporating the new information and approaches into the procedures used in developing the Pb NAAQS. The US EPA's Metal Risk Assessment Framework document (2005) and the European Metals Environmental Risk Assessment Guidance (MERAG; 2007) both provide an excellent discussion of state-of-the-science approaches for addressing metals risk assessment and these approaches should be considered in the development of the Pb NAAQS.

Comments from Dr. Ian von Lindern

Overview

The draft Integrated Review Plan (IRP) is generally well organized, comprehensive, and addresses the main issues to be considered in the National Ambient Air Quality Standards (NAAQS) review. The document provides a concise summary of the previous NAAQS review effort and the uncertainties and difficulties encountered in developing the current Air Quality Criteria Document (AQCD) and subsequent NAAQS. The underlying strategy seems to focus on new information since the last review that will determine whether any addenda are required. The document implies that additional action regarding the NAAQS may be curtailed based on the determination of the relevancy of the new material.

Taking advantage of the lessons learned from the 2006-7 revision should facilitate the Agency in responsibly meeting the 5 year review requirements. This is in contrast to the last review, when decades of information was allowed to accumulate across orders of magnitude changes in ambient concentrations, absorption, blood lead levels, internal exposures and identified health effects; in addition to monitoring and analytical capabilities, control technologies, and market applications for lead.

It is appropriate for the Agency to concentrate on new additions to the knowledge base and evaluate the degree to which the new information informs the analyses completed in 2007. This should streamline the process. However, the EPA should not confine the review and revision of the AQCD to this narrow period of lead regulatory history. There were challenges encountered in the previous review that indicated significant gaps in the knowledge base. These information deficiencies introduced uncertainties into the process that should be revisited. The EPA should learn from and evaluate the effectiveness of the NAAQS in the context of the longer history of lead regulation.

Historic Perspective

It was clear long before the 2006-7 review that the 1970's standard had become largely irrelevant throughout most of the country. The Agency was remiss in failing to update the NAAQS, as recommended in 1991, and it effectively stopped monitoring concurrent ambient air lead emissions and related absorption metrics for nearly fifteen years. In finally reassessing the NAAQS in 2007, the EPA struggled with whether to develop a relevant standard, or eliminate lead as a criteria pollutant. In deference to its responsibility to protect the public health, the EPA retained lead as a criteria pollutant and adopted a standard reduced by an order of magnitude. The last AQCD showed that EPA had, in maintaining the irrelevant standard for so long, "lost track" of key parameters necessary to effectively assess the health and ecological risks of airborne lead in the U.S. Relatively few data were available in several key areas for performing a responsible risk assessment.

Uncertainties in the Current NAAQS

Development of effective exposure estimates was the weakest point in the Agency's analyses that supported the current NAAQS. This was due to the lack of monitoring data available to assess contemporary exposures in the U.S., or to support the modeling analyses relied upon in subsequent development of the NAAQS. As the current AQCD points out, previously undetected deleterious health effects were occurring at lower blood lead levels, and potentially at the environmental concentrations the Agency had failed to monitor.

As a result, in the last review the EPA was challenged in effectively estimating the extent of potential damage in the general population; the relationship between air lead levels, emissions and absorption; safe air lead concentrations; or the number of citizens exposed to potentially dangerous levels. There were insufficient data to characterize active emissions and emission rates, ambient concentrations and the degree, extent and severity of ongoing redistribution of residual lead in the nation's environment. As a result, EPA relied on modeling and decades-old empirical relationships to quantify exposures.

This weakness was identified in the AQCD; and subsequently the Office of Air Quality Planning and Standards (OAQPS) made the best of a poorly characterized situation in developing the NAAQS. This resulted in considerable uncertainty inherent in the overall process.

It is important in this revision that the AQCD address these uncertainties. Frank discussions of the unknowns and lack of certainty in the existing analysis are found on pages 5-3, 5-9 and 5-10 of the IRP regarding the modeling approach. The subsequent policy analyses should examine whether relevant databases are being developed, both internal and external to the EPA. A determination should be made as to whether it is possible to move from the near total reliance on modeling to observational and empirical analysis of contemporaneous data.

Several questions should be answered with respect to monitoring and surveillance. What data base is accumulating with respect to compliance with the new standard? Is a network established, is it adequate and effective, and are there a detectable blood lead levels associated with any gradient in exposure? Have there been excursions? Certain U.S. sub-populations (e.g., immigrants and inner city children) are at substantially greater risk of exposure due to co-factors associated with different cultures, climates, dietary and nutritional regimes, as are being encountered in the global lead poisoning epidemic. Is there monitoring in these venues?

If indeed these types of data are becoming available, then it seems incumbent upon the Agency to undertake the necessary analyses to reduce the level of uncertainty inherent in the current regulatory scheme and respond accordingly. If no such data are being accumulated, the EPA should explain why.

Data Sources

A major concern in the last review was that EPA limited the information search to *"… where information is available in the peer-reviewed literature."* Unfortunately, the best sources of production data, emission information, industry transition and economic indicators are more

likely found in the trade literature and government agency records. Much of the practical knowledge that has been developed in applying scientific findings and methods to remedial and regulatory activities is generated and housed in programmatic activities within EPA and the States. It would not be prudent to limit the combined analyses to the relatively obscure and less-representative studies that have reached the peer-reviewed journals.

The Data Quality Objectives (DQOs) have apparently been expanded somewhat to *"peer-reviewed journal articles, books, and government reports"*. This expansion is more inclusive of the recognition that much of the information to develop a nation-wide assessment of lead exposures is unlikely to be found in peer-reviewed journals. The CASAC urged the Agency to mine these data sources in the last review, but was told that there was insufficient time to effectively exploit these resources in the context of the ongoing litigation and budgetary constraints. Perhaps in the current effort, sufficient resources could be allocated to better characterize sources, emissions, ambient concentrations, transport and transformation, and effective control technologies through broader examination of programmatic successes and failures. In the event that EPA is restricted from utilizing this extensive experience in crafting effective regulations, the scarcity and short-comings of information available from journal articles should be noted.

Policy Implications

The demand for and consumption of lead in the U.S. has increased markedly in this century, accompanied by substantial price increases in the domestic and world market (see Figure 1). The IRP seems heavily weighted toward assessing and characterizing new information regarding health risk assessments and health effects. As noted above, other general exposure considerations related to market and use factors (i.e., emission sources, commercial applications, waste, recovery, recycling, and disposition and fate of lead) in the U.S. today are poorly understood, nor have exposure parameters been quantified. Several other issues not mentioned include advancements in pollution control capacity, best available technologies and best practices for source control, monitoring protocols, detection limits, and analytical methods; and assessment methodologies particularly with regard to health and ecological effects.

Although demand has increased, domestic production and recycling and recovery of many discarded lead products have been diverted to developing countries. Much of this diversion results from EPA policies.

Price increases noted during the early and mid-1970s indirectly accounted for the lead poisoning epidemics noted around production facilities that ultimately played a significant role in the development of the NAAQS in 1977-80. Excessive and irresponsible emissions from smelters at Bunker Hill in Idaho, Broken Hill in Zambia and Australia, and Kosovo, (former Yugoslavia) resulted in severe poisoning, disease and some death during the run up in prices. These problems were documented and aired in the earlier AQCD, which played no small role in bringing about substantial reductions in both emissions and irresponsible operations.

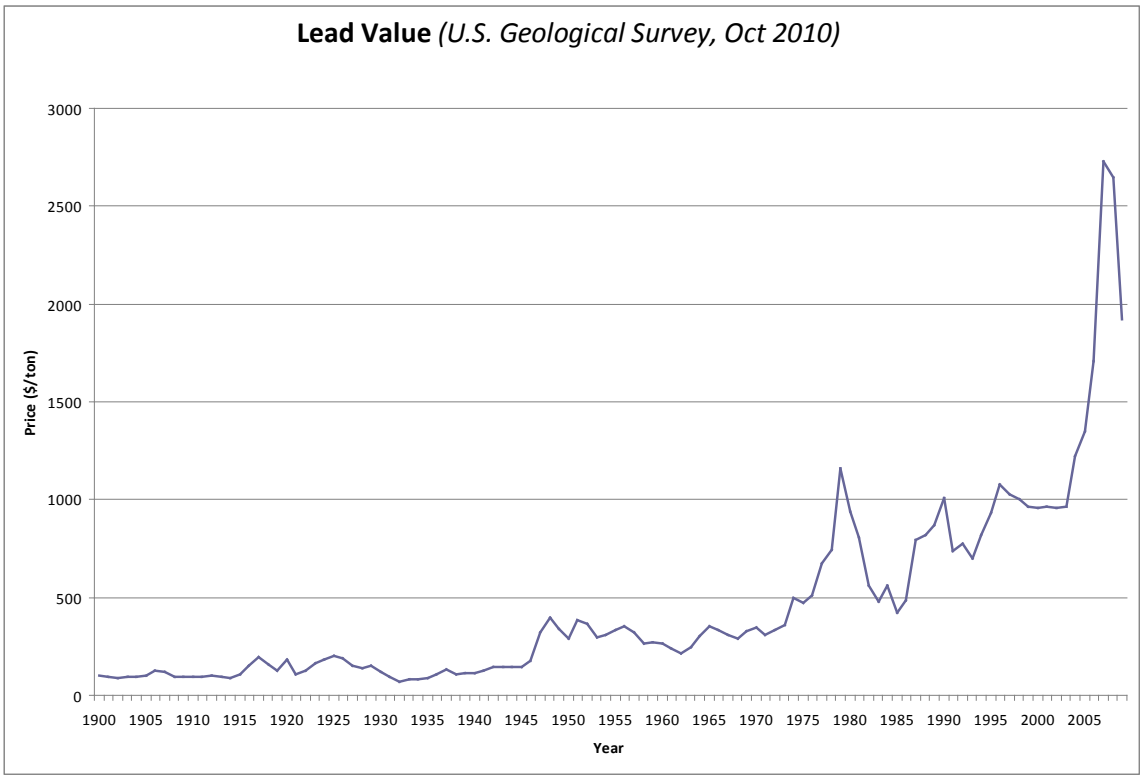
On the international scene, the increased price and demand observed in the last five years (see Figure 1) has had devastating effects, substantially more severe than those observed in the 1970s.

Environmental exposures and lead poisoning are increasing with several incidences of severe morbidity and substantial mortality associated with the increased demand and high price of metals. Hundreds of children have died at some sites and thousands suffer irreversible health effects that will leave them a burden on their families and communities for decades.

Although these factors may have little direct impact on air lead levels in the U.S., the implications for regulation of lead releases and the impact of U.S. policies in the global environment and human health can be substantial. The Agency also has an obligation to export the scientific knowledge base, consequences of irresponsible practices, and information regarding mitigation of adverse effects. Regardless of the statutory requirements, the 1977, 1986 and 2007 AQCD revisions and addenda have served as seminal documents. The AQCD are utilized, referenced, critiqued and practically applied on an almost daily basis throughout the world and provide an invaluable framework for society, industry and the scientific community to develop and implement strategies to meet public health and environmental needs. The subsequent regulatory policies when implemented have ramifications, not only beyond ambient air lead levels, but throughout the world. These issues should be developed in the IRP.

Finally, there are two major lessons from the last 40 years of implementing lead control policies that should be applied. First, lead remains and must always be evaluated in a multimedia context. The need to disentangle the air component from global exposure is more a function of fractured media-based regulatory policy than of science or public health protection. The EPA should continue to apply, refine, develop, improve and learn from multi-media integrated exposure models. Second, the implementation of lead reduction rules has lowered ambient levels, and subsequently absorption and blood lead levels. This provided a population base for further research that allowed detection, and ultimately mitigation of adverse health effects previously unknown or unproven. In the interest of advancement of science, health effects reviews should consider assessing co-exposures with other metals and toxins; and to the extent possible this approach should be extended to the ecological risk assessments.

Figure 1.


Lead Value (U.S. Geological Survey, Oct 2010)
Price ($/ton)
3000
2500
2000
1500
1000
500
0
1900 1905 1910 1915 1920 1925 1930 1935 1940 1945 1950 1955 1960 1965 1970 1975 1980 1985 1990 1995 2000 2005
Year

Comments from Dr. Gail Wasserman

Chapter 4 (Science Assessment)

1. The planned review

- EPA staff are preparing an updated review, summarizing evidence on consequences of exposure. This review sets the ground for selection of articles to review, including newer (since 2006) studies, and older studies (if of continued importance).

- To do so, they have requested input from the public, and will review other EPA reports and databases.

- Chapter 4 specifies criteria for inclusion. These are generally excellent.

Question/comment: it is unclear if there is a formal weighting that will help evaluate the adequacy of the evidence base.

2. Content of the planned review

- In the planned review, there is particular interest in certain questions, including vulnerable populations, confounding, the timing and duration of exposure.

- There will be particular concern for studies at levels relevant to US population.

    - ***Question/comment***: Is this misguided? Because current US levels are very low, this would restrict review to studies of very low exposures. On the other hand, study of a wider range might provide more information about mechanisms/consequences.

- EPA staff will review information on exposure sources (with concern for separating out air-, water- and soil-pathways), and on toxicokinetics.

- They plan to review information on health outcomes, integrating findings to evaluate strength and consistency as well as biological plausibility, with focus on lower exposures.

    - ***Question/comment***: Again, is this misguided, for the same reasons noted above?

    - ***Question/comment***: I believe that in most places, we should consider changing NEUROLOGICAL to NEUROPSYCHOLOGICAL. At levels at which traditional neurological examination would be negative, we would anticipate neuropsychological deficits (e.g., IQ).

- ***Question/comment***: While as written, the neuropsychological content is restricted to IQ, although there is interesting work on other cognitive functions, such as Executive Function, which is of interest.

- It is anticipated that this review will consider the roles of co-exposures, confounding, and mechanisms of action, which will add to its thoroughness.

- The planned review will consider developmental periods of susceptibility.

    - ***Question/comment***: So much of the neuropsychological work is based on very young children, in keeping with the high level of brain development at this time period. On the other hand, we increasingly understand that there are other periods of rapid brain growth (i.e. around adolescence). Particularly because it appears that certain neuropsychological functions (such as Executive Function) continue maturing into adolescence (paralleling growth in frontal/prefrontal structures), so that examining these only early on might miss deficits. Perhaps highlight the importance of other developmental periods?

Comments from Dr. Michael Weitzman

This is an excellent document, well written and coherent.

1. It should be clarified as to how the literature search will be conducted, what criteria will be used for selecting articles to review, and how the quality of articles will be assessed, especially given the fact that articles are likely to have varying populations, methods for sample selection and analytic approaches.

2. It would be useful to search for and analyze separately literature that looks at (a) acute and chronic exposure and (b) lead exposure alone vs. lead exposure concurrently with other potentially synergistic exposures

3. It is not clear to me what “susceptible populations” refers to, i.e. susceptible for exposure and/or susceptible as relates to the effects of exposure.