UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

October 14, 2011

EPA-CASAC-12-001

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 *Review of the National Ambient Air Quality Standards for Lead: Risk and Exposure Assessment Planning Document*.

Dear Administrator Jackson:

EPA's Clean Air Scientific Advisory Committee (CASAC) Lead Review Panel held a public meeting on July 21, 2011 to conduct a consultation on the Agency's *Review of the National Ambient Air Quality Standards for Lead: Risk and Exposure Assessment Planning Document*.

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.
We thank the Agency for the opportunity to provide advice early in the National Ambient Air Quality Standards review process. The individual CASAC Lead Review Panel members' written comments are provided in Enclosure A.

Sincerely,

/Signed/

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

Enclosure

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.

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

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Enclosure A

Compendium of Individual CASAC Lead Review Panel Comments on EPA's Review of the National Ambient Air Quality Standards for Lead: Risk and Exposure Assessment Planning Document (June 2011)

Comments from Mr. George A. Allen

The health related questions to be addressed in this consultation in the 6/28/11 memo to the DFO) are:

1. The overview of the previous health risk assessment and presentation of results from the last review of the lead NAAQS.
2. The staff evaluation of elements of the risk assessment that were considered in determining the need for an updated quantitative risk assessment (e.g., advances in methods for modeling exposure including the estimation of air-related pathways of exposure, prediction of blood Pb, updated/refined concentration-response functions for IQ loss).
3. The decision to rely on the quantitative health risk assessment from the previous review, interpreted within the context of newly available evidence and information.

General comments:

The document is well written and organized. Questions 1 and 3 have brief answers as follows.
(1) The overview of the previous REA and NAAQS review are adequate and useful.
(3) I agree with the decision to rely on the previous health risk assessment for this review. Published research since the last REA does not change any of the outcomes for the quantitative health risk assessment. New literature is relatively sparse, and some is not directly relevant or useful to this review.

Question 2 (chapter 2) is the core of this consultation for health risk assessment. Overall, the staff evaluation of risk elements are adequate and appropriate. There are many assumptions and uncertainties (both from sparse data and modeling) that make up the overall REA process. This chapter addresses them clearly. The review of recent relevant literature (which is also sparse) is appropriately summarized.

One component of the exposure assessment that is not entirely clear to me is what Pb air sources make up policy relevant background (PRB -- that exposure that can not be controlled by changes in current or future air Pb concentrations. Pb from soil is a factor here. The "air" Pb contribution from re-suspended soil or soil brought into the home is a mix of current and historical deposition of air Pb. In essence, Pb from "old" air deposition can become new Pb in the air if it is re-suspended in an exposure-relevant context. But "old" air Pb is not effected by changes in contemporary air Pb concentrations; does that mean it should be considered as PRB Pb?

Section 2.2.1, Pg. 2-2 defines controllable Pb as:

> "... sources and pathways for which ambient air <u>has</u> played [note tense] a role ("air-related") ... these are exposures with the potential to be affected (over some time frame) by an adjustment to the Pb NAAQS."

and PRB Pb as:

> "... those pathways not associated with Pb <u>originally emitted to the ambient air</u> are considered policy relevant background since an adjustment to the Pb NAAQS is not likely to have an impact on these exposures..." [underlining mine]

This is also discussed in section 2.1.4, pg 2-14, where the terms “recent air” and “past air” are used, and both are considered to be controllable, described as “hav[ing] the potential to respond relatively more quickly to changes in air Pb”.

This brings into focus the importance of the fate of Pb in soils, as discussed in detail in the ISA. Old soil air-Pb can not be controlled, but it also doesn’t quite fit into the framework of PRB Pb. The link between these is the phrase “over some time frame”, presumably meaning that lowering air Pb will “eventually” lower soil Pb. But “over some time frame” is very vague. Old soil lead presumably does get lower over time (ignoring losses from resuspension), but that time is both uncertain and spatially variable for many reasons as noted in the ISA. So yes, old soil Pb can be a part of relevant [controllable] air lead sources, but it’s not clear how much over shorter (a few years?) time frames that are relevant to a NAAQS review. The REA could benefit from a discussion of this component of controllable exposures.

On page 2-15, the REA says:

> “The assessment did not simulate decreases in “past air” exposure pathways (e.g., reductions in outdoor soil Pb levels following reduction in ambient air Pb levels and a subsequent decrease in exposure through incidental soil ingestion and the contribution of outdoor soil to indoor dust). These exposures were held constant across all air quality scenarios.”

Why was “past air” exposure held constant? Elsewhere it is assumed that soil Pb would respond [eventually] to decreases in air Pb.

Pg. 2-6 and 2-8, primary Pb smelter exposures. This case study is from a single (very old) primary smelter in MO. Emissions from that smelter are from combustion and mechanical sources - e.g., both fine and coarse modes. But that smelter has tall stacks (550 ft.); little to none of the stack emissions impact the 1.5 km zone used in the case study. Pb of any size or mode in that zone is essentially all from fugitive emissions. The REA says: “children’s air-related exposures are most impacted by emissions associated with the Pb smelter from which air Pb concentrations were estimated.” For emissions used in the case study, was this taken into account, or were total emissions used? Figure 2-2 lumps them together.

Comments from Dr. Herbert E. Allen

I have reviewed the Ecological Risk Assessment portion of the document and am in agreement with the Key observations and conclusions.

Comments from Dr. Deborah Cory-Slechta

The intent of the document was to summarize the approach to Risk Assessment adopted by the 2006 AQCD on lead including the limitations that were inherent in each of the steps of the process and to evaluate whether data collected since that time could be used to inform any of those limitations and, if so, the extent to which that information would influence the final risk assessment.

The limitations inherent in the previous derivation of both the primary and the secondary NAAQS were thoroughly and completely laid out in this document. For each of those limitations, the document presented the extent of new information related to that limitation that had been reported since the 2006 document. In addition, it addressed the extent to which the information would alter or influence the prior risk assessments.

For both the primary and secondary NAAQS, the conclusion arrived at is that there is insufficient new information that would reduce these specific limitations of the risk assessment and would have thus warranted a new risk assessment. Based on the reading of the Integrated Assessment Documents and the Risk and Exposure Assessment Planning Document, I would concur with those conclusions.

Comments from Dr. Cliff Davidson

Overall comments: generally very well written and organized. I agree with EPA that there is not enough new information to warrant a complete revision of the health and ecological risk and exposure assessment.

I have one overall concern. The REA document does not make it clear that there is a big difference between the importance of the health risk assessment compared with the ecological risk assessment for lead. If we consider IQ deficit in the US, this is of tremendous importance to the national interest; lead exposure is clearly one of the important factors influencing neurocognitive functioning. If we consider the ability of our natural areas to provide ecosystem services, this is also of tremendous importance to the national interest; but lead exposure is not one of the important factors influencing the health of natural ecosystems and the services they provide. Other factors are far more important, such as suburban sprawl and greenfield development, use of chemical fertilizers, herbicides, and pesticides on land, and industrial effluents discharged into waterways. In my opinion, the REA document should make it clear that factors other than lead exposure are far more important in reducing the ability of our natural land to deliver ecosystem services.

There are also some minor comments. At the bottom of page 2-6, footnote 7 uses the term "ratios of relationships in the available air monitoring data between different statistical forms…" I don't understand this phrase. I think too much is being condensed into this sentence.

On page 2-15, center of the page, the text states "On balance, we believe this limitation leads to a slight overestimate of the risks in the "past air" category." I don't understand the rationale behind why this is a slight overestimate.

On page 2-20, within the top center grid square of the table, there is a statement "The draft ISA also notes the cycle of deposition and resuspension for even coarse-phase Pb can be substantial leading to diffusion in the urban context." I suggest revising this so it is clear that the *amplitude* of the cycle is substantial, and the severity of gradients of lead near sources is reduced because of urban diffusion. (I think that is the intended meaning here, but not sure.)

Also on page 2-20, the top right grid square contains the typo "to estimates how many children it may represent". "Estimates" should be "estimate".

On page 2-22, the lower right grid square states "an important limitation of these studies is that they do include measurements of ambient air Pb." Replace "do" with "do not".

On the top of page 3-4, the text refers to Pb exposure affecting ecosystem services. Perhaps this paragraph is where my suggestion about ecosystem services above could be accommodated. While the statements made in this paragraph are true, they give a misimpression, namely that Pb is an important factor influencing ecosystem services, and this could be considered as a reason to decrease ecosystem exposure to Pb. I do not think this is a valid reason to reduce Pb, given that other factors are so much more important in reducing ecosystem services.

Comments from Dr. Philip Goodrum

1. The overview of the previous health risk assessment and presentation of results from the last review of the lead NAAQS.

Overall, the REA is well written and easy to follow. Reliance on the previous health risk assessment is logical given that the All Ages Lead Model has not yet been completed. Once the AALM is available, additional data available for age groups older than 7 years can be incorporated more directly in the assessment.

2. The staff evaluation of elements of the risk assessment that were considered in determining the need for an updated quantitative risk assessment (e.g., advances in methods for modeling exposure including the estimation of air-related pathways of exposure, prediction of blood Pb, updated/refined concentration-response functions for IQ loss).

The presentation of new research findings and information on exposure and alternative risk metrics since 2006 was well organized and clearly described.

3. The decision to rely on the quantitative health risk assessment from the previous review, interpreted within the context of newly available evidence and information.

The decision to substantially rely on scenarios that were previously explored appears well justified. For the GSD term, although NHANES and other recent monitoring data are available from which to explore how the dispersion in the lognormal distribution may have changed since IEUBK was developed with a recommended default of GSD=1.6, the calculations would need to control for variability in exposure conditions. With the GSD term, we are primarily interested in the interindividual variability in PbBs under the condition that a population is exposed to the same environmental concentrations of lead in all exposure media. In the development and implementation of the AALM, the effort required to conduct the appropriate model verification using various community-level monitoring data will be warranted. These statements may be useful to add to the REA.

There is a potential for confusion regarding the intended utility of the mechanistic models for evaluating risks at the national scale. The REA should more clearly emphasize that a series of scenarios are developed to represent a range of plausible exposure conditions at a community level and that, collectively, this provides a perspective on how the distribution of blood lead concentrations may vary among communities that share similar exposure profiles. This is in contrast to developing a single model run, for example, that represents all children in the United States. With this introduction, it will be easier to explain the relevance of various empirical data sets to the overall evaluation. For example, the air-lead / blood-lead relationships reported in the literature generally reflect studies in various communities. Rather than attempting to consolidate the results into a single summary statistic, it's this range of slope factors that is important to understand. Similarly, this explains why the summary statistics of bloodlead distributions reported by NHANES are not used to adjust or otherwise update the default GSD term in the IEUBK model, as discussed above.

Comments from Dr. Sean Hays

Since there has been little data developed since the last NAAQS for lead was established, there are very few aspects of uncertainty associated with the risk assessment used to support the previous NAAQS that can be updated. Having stated this, there are some additional activities I think EPA should consider.

The multi-media nature of lead contamination requires an exposure and risk assessment that encompasses each of the various sources and pathways of exposure for lead. Currently, EPA is developing or has developed risk assessments for lead exposures associated with air exposures (NAAQS), dust (via the dust repair and remediation rule), and water (via a lead in water committee review). Each of these activities is assessing risks to lead via different means. This has the potential to allow important lead exposures or scenarios to 'fall through the cracks' or to result in inconsistent approaches and findings amongst the various lead risk assessments being developed by the agency. This can be improved. Health Canada recently undertook an agency wide risk assessment for lead which involved an assessment of a target blood lead level that the agency believed would be sufficient for protecting public health (1 or 2.5 ug/dL lead in blood). With this decision, the agency then determined which environmental media (soil, dust, air, water, diet, etc.) would be allowed a defined portion of exposure towards achieving the target blood lead level (for instance, lead in soil would be allowed to be at a level that would achieve 25% of the total blood lead target, and respective percentages would be defined for the other media impacted by lead contamination). The percentages prescribed to each media could be decided via a number of different criteria. The same lead toxicokinetic model would be used for each media so as to provide consistent approaches for determining the lead concentration required to achieve the fraction of the target lead concentration. This approach allows for a consistent approach for lead risk assessment across all media sources and assures that exposures to lead from all media will not yield blood lead levels among the population that exceed a pre-determined target. The EPA should seriously consider a similar approach to regulating lead exposures and get away from their current approach which is disconnected and leads to potentially inconsistent approaches within the EPA.

The biggest need for the EPA is data to help reduce uncertainty in their risk assessment. EPA should consider using all resources and money currently allocated to this NAAQS review for lead and spend that money over the next four years to fund research that will fill the identified data gaps in this REA.

Comments from Dr. Philip Hopke

The prior REA is going to be used as the basis of the risk studies of various NAAQS options. However, the primary scenario was the primary lead smelter that will be shutting down in 2013. Thus, the prior work is really not relevant to risk going forward. Given that the ISA suggests that 49% of the anthropogenic lead emissions are from combustion of aviation gasoline and general aviation facilities are much more prevalent than secondary smelters, it seems that this source needs to be specifically addressed. If not in a revised REA, then clearly the policy assessment should address the major remaining controllable sources, general aviation, secondary lead smelters and wheel-weights. The current REA is really not relevant to the next PA and provide little useful basis for making policy decisions based on exposure scenarios that will not exist in the future.

Comments from Dr. Chris E. Johnson

In reviewing the Risk and Exposure Assessment (REA) Planning Document, I focused my efforts on the welfare risk assessment. Others on the CASAC are much better equipped to judge the health risk assessment.

1. Overview of the previous ecological risk assessment and the presentation of results from the last review of the Pb NAAQS.

The REA Planning Document does a good job of describing the design and conduct of the ecological risk assessment done for the 2008 NAAQS process. The REA Planning document does not present any results from that assessment, aside from the limitations and uncertainties that were identified.

2. Evaluation of new evidence and information in light of limitations and uncertainties of the risk assessment from the previous review.

The authors of the REA Planning Document highlighted several areas where recent studies have added to our understanding of key issues related to ecological risk assessment. While there is much new material that supports the previous work, the authors conclude that few of the limitations and uncertainties identified in the previous assessment have been adequately addressed.

3. Staff assessment of the new information and conclusions regarding the use of critical loads modeling.

The REA Planning Document highlights the many difficulties in carrying out a comprehensive critical loads based assessment for Pb in terrestrial and aquatic ecosystems. In addressing new information, the REA Planning Document refers to the ISA, which considered three studies of critical loads in terrestrial systems, and claimed that there is no new significant information regarding critical loads in aquatic systems. It is, in my opinion, a bit narrow-minded to conclude that the state of science in critical loads modeling has not advanced because few have tried it. The many, many studies carried out since 2005 on toxicological effects, Pb fate and transport, and bioaccumulation, cited in the ISA, all contribute to a better knowledge base for critical loads modeling. Are there gaps? Of course. But there is no question that we are better equipped to do it now than we were six years ago.

4. The decision to rely on the quantitative ecological risk assessment from the previous review, placed within the context of newly available evidence and information.

This is, all things considered, probably the right decision. An ecological risk assessment carried out today, and incorporating insights from studies published since 2005, would be better than the one done for the 2008 NAAQS process. But it would probably not result in any substantially different conclusions. The bioconcentration factors for aquatic plants in the current ISA are much lower than the ones in the 2006 AQCD. Using lower BCF values would reduce the hazard quotient at both the organism and ecosystem level.

On a more general note, I fear that the EPA is placing unreasonably high expectations on the data needs for performing a new quantitative ecological risk assessment. For example, in discussing critical loads modeling, the REA Planning Document says, "…however, application of this methodology at a national scale requires localized data across a wide range of ecosystems, which are currently unavailable or inadequate." Frankly, the United States will probably never have localized data on fluxes, soil fractions, groundwaters, floral and faunal bioaccumulation, and toxicity adequate for a proper critical loads assessment, or other ecological risk assessment for that matter. The amount of work required, coupled with the dearth of funding for research on metal biogeochemistry, makes the development of such a database very unlikely indeed. I do think that it is probably too soon to do another ecological risk assessment at this time, but the tone of this document concerns me.

Comments from Dr. Susan Korrick

Health Risk Assessment:

1. The overview of the previous health risk assessment and presentation of results from the last review of the lead NAAQS.

Section 2-1 provides a clear, comprehensive review of the limitations and uncertainties in modeling risk and exposure modeling done for the last NAAQS review. However, some specifics were unclear. Clarification would be useful to addressing the planned approach:

(1) Presumably indoor air inhalation factors into the risk models. However, it is identified as a direct source in Figure 2-1 but not Figure 2-2.

(2) 'High' air Pb exposures (near the standard) in the general urban case study is modeled as comparable to the 'high' air Pb exposures in the smelter case study. Presumably this is because they are assumed to have the same level of compliance? If so, what is the basis for this assumption? Also, the smelter case study was not included in the final risk assessment because of uncertainties in estimates but, as it seems this case models an important component of air Pb emission regulation, it is unfortunate that it could not be included in some form.

(3) More specific information about the dual linear models (e.g., page 2-12) would be useful including, e.g., how the inflection point (where the two lines intersect, not where data are stratified) was determined.

2. The staff evaluation of elements of the risk assessment that were considered in determining the need for an updated quantitative risk assessment (e.g., advances in methods for modeling exposure including estimation of air-related pathways of exposure, prediction of blood Pb, updated/refined concentration-response functions for IQ loss).

Section 2-2 provides a tabular overview of the limitations and uncertainties of the previous risk assessment, new data/techniques relevant to those limitations and uncertainties, and then a summary of whether that new information is sufficient to improve past limitations. Some issues would benefit from clarification:

(1) In several instances, it was unclear why the new information was not considered applicable to a new risk assessment. E.g., (page 2-22): new studies assessing the relation of dust Pb with blood Pb were not considered sufficient to do performance assessment on the models of the relation of ambient air Pb with indoor dust P and blood Pb, in turn. A more explicit description of the criteria used to determine sufficiency of new information would be important here. Even more important, the document does not explicitly address how newly available information will be used to interpret (re-interpret?) the quantitative health risk assessment from the previous review. A better explanation of this latter proposed process is important especially in the context of relevant new information but a proposed continued reliance on the previous health risk assessment. For example, on page 2-31, the text states, *"New studies based on analysis of NHANES and NHEXAS…provide insights on factors related to PbB in children…while information from these studies is not conducive to updating the risk model…the*

information may be useful in further interpreting risk estimates generated in the previous analysis…" How would this further interpretation work? What other new data would be used for "further interpretation"? Similarly, text later on the same page states , *"…while the newer data on PbB variability could be useful in further interpreting risk estimates generated for the previous review, we believe there is little utility in using these updated GSDs to generate new risk estimates."* How would this work to further interpret risk estimates from the previous review?

(2) The IEUBK model does not work above age 7. It seems there are not other acceptable models (besides the IEUBK) that could be used for a new risk assessment; an important goal for the future should include development of models that could be applied to older children and other age groups.

(3) On page 2-28, the summary describes 2 studies showing associations of childhood blood Pb with delayed puberty. It's useful to keep in mind the two cited publications are only one study. The two papers studied Russian boys using the exact same study/study population with the earlier publication reporting a cross sectional analysis and the later one a longitudinal analysis as more data were available.

3. The decision to rely on the quantitative health risk assessment from the previous review, interpreted within the context of newly available evidence and information.

Overall, the decision to rely on the quantitative health risk assessment from the previous review seems carefully considered and justified (see areas for better explanation in item #2-1 above). The REA provides a point-by-point review of the limitations of the previous risk assessment and the reasons new information will not substantially improve upon the previous work. However, providing examples of how new data will be incorporated into interpretation of the former risk assessment would be useful in understanding (and justifying) the proposed approach. Indeed, this reviewer would like to see how the 'reinterpretation' of the past risk assessment will be done.

Comments from Dr. Michael Kosnett

The following represents my summary evaluation of the draft Risk and Exposure Assessment (REA) Document pertaining to the NAAQS for lead.

I agree with the ultimate conclusion of the REA document that new information available in the current NAAQS science review for lead does not support the development of an updated or enhanced risk model that would substantially improve the health risk estimates for lead.

I concur with the document's conclusion that a quantitative assessment of the relationship between lead exposure via air-related pathways and IQ loss in young children should remain the primary component of the risk estimate for lead.

The document's suggestion (page 2-24) that the 2005-2008 NHANES data offer information that could be used to update or revise the GSD parameter applied in the IEUBK model should be reconsidered. The variability in blood lead concentration presented in the NHANES data reflects variation due to different levels of environmental lead exposure; whereas the GSD utilized for the IEUBK model should reflect variability in blood lead among children with the same level of environmental lead exposure.

In commenting on the impact of certain modeling assumptions on risk estimates, the draft REA document states that the risk attributable to air-related exposure pathways is likely to be bounded on the low end by the risk estimated for the "recent air" pathway, and on the upper end by the risk estimated for the combination of "recent air" plus "past air" pathways (page 2-15). EPA should consider that combining the "recent air" plus "past air" pathways may still represent an underestimation of the upper bound risk, because the sum of these two pathways omits the long term contribution of air-related pathways to lead in the diet. Dietary lead represents the largest component of lead exposure to the general US population, such that even a relatively small percentage change in dietary lead might yield a significant impact on overall lead exposure.

A recent study (Miranda et al, Environmental Health Perspectives, 2011; http://dx.doi.org/10.1289/ehp.1003231) suggested that emission of lead associated with aviation fuel may raise the blood lead concentration of children residing close to airports. EPA should consider the feasibility of developing a case study for the NAAQS analysis that examines the impact of a revised standard on children residing near these facilities.

The REA noted that the prior risk assessment did not possess or utilize models to simulate changes in outdoor soil resulting from a decrement in air lead concentration over time. Much of the information available on lead in soil does suggest that it has long term stability, so EPA's assumption that changes in air lead would not result in a change in soil lead may not represent a significant source of variability or uncertainty in the risk assessment. Site specific documents possessed by EPA's Superfund program might contain data on the temporal pattern of soil lead

in the vicinity of certain hazardous waste sites following the cessation of activities associated with airborne lead emissions. This data, if available, might offer an additional method of verifying the assumption that soil levels remain relatively stable.

Comments from Mr. Richard Poirot

As with the health effects sections of the Pb REA Plan, the proposed approach for the welfare risk assessment is logically conceived and very clearly written. I agree with the staff's general conclusions, that newly available information (especially relating to critical loads) should be helpful in interpreting the results from the previous Pb risk assessment, but that developing a new environmental risk assessment does not appear to be warranted in the current Pb NAAQS review.

Comments from Dr. Michael Rabinowitz

Regarding the REA document, I do concur that the most sensitive outcome of interest seems to be IQ decrement. Also, blood lead (PbB) is the best biomarker of exposure, uptake, and the internal dosage to target organ, the brain.

In an effort to explore the relationship between exposures and outcomes, a variety of models have been tried. My sense is it that the natural variations in Pb and IQ are much greater than the differences among the predictions of the various models. I am satisfied with the empirical models. Any uncertainty caused by model selection is smaller than the variation in the data.

I concur that with the overall conclusion that the newly available evidence does not warrant additional quantitative analysis for either health or welfare standard.

Other comments:

1: I suggest displaying nested models to help show the extent to which Pb is an independent risk factor in the epidemiological modeling, where so much variance is shared. We should be able to see the extent or strength of confounding in the various studies, and see the effect size for Pb along with the whole model's predictive power (r-sqr). That Pb can be shown to have a non-zero coefficient in multiple regression models, for example, of children's mental performance, is insufficient. Because of the extent of the confounding, this is different than showing that Pb is an independent risk factor. Pb and these other risk factors share considerable variances, particularly in some of the higher risk populations, where Pb exposure and other risk factors often coexist.

The relative size of this non-zero coefficient, the size of the Pb effect, should be shown in terms of the model r-sqr, or goodness of fit. How good is that model's fit with and without a Pb term in a series of nested models? Does the r-sqr increase significantly (Wilks criteria) when a Pb term is offered? How much do the confounders' strength shift towards the Pb term, with which it shares variance, when Pb term is introduced? This would help a reader see how much is caused by Pb compared to other risk factors, preventable and otherwise.

My concern is that at progressively lower Pb levels, where Pb effects are small, blood Pb can still be measured relative accurately (often to 2 significant figures) but other, stronger variables, such as maternal education or richness of the child's home environment can be more difficult to measure, subject to reporting errors, and are often entered as broadly categorical variables, while lead is a continuous variable. At these lower but measurable, Pb levels, Pb effects get lower, but the confounders relative strength increases. For these reasons, at these low levels, effects attributed to Pb by statistically adjusting for the other covariates, may overstate the case. For that reason showing models with and without the Pb term would be useful. Also, it may help identify the more critical studies in terms of seeing a "clean" Pb effect.

2: Also, I too concur that using concurrent and some lifetime average is the way to go. At these levels, the paradigm of "windows of vulnerability" appears to have been overcome by the tendency for recovery. So, effects from earlier exposures eventually go un-noticed given subsequent, lifetime exposures and other events.

3: Many individual factors modify uptake and susceptability to Pb (genetics, gender, nutrition, age...), and the document reflects that. It is important to identify those at most risk and better understand biokinetics. I would like to emphasize that enviromental Pb levels, not any of the many host factors, is the biggest determinant of excessive PbB.

4: If I may address the topic of long term trends:
Regarding diet as a source of Pb, from my historical perspective, I am reminded that in LA in the 1970's for adults, absorbed dietary Pb was twice the magnitude of our daily inhaled dose. We learned this at the VA Hospital's metabolic balance ward using isotope tracers and clean-air rooms. Roughly, air was 2 ug/cuM, diets were in excess of 300 ug/day, and PbB were about 20ug/dL (or 0.20 ppm). Some of the dietary Pb had been airborne. Lettuce from Salinas CA was a prime example. Canned food, more common then, especially in institutional diets, had microscopic solder splashes.

Now air is so much lower, diets are a few ug/day, and PbB averages closer to 2ug/dL. Typical urban American Pb exposure and PbB have decreased by about an order of magnitude over these nearly 4 decades. Still, today, diet is predicted to be twice as strong as the remaining inhalable Pb, and our understanding of the sources of dietary Pb is imperfect. I sense a retreating horizon, as we make progress in lowering Pb exposure.

Still there is a lower limit. In that natural soil Pb is about 35 ug/g, and if we ingest perhaps 10 mg/day, about 1/3 ug Pb /day would be ingested. This would be 1/4 kg dirt per 70 yr lifetime.
The proverbial "one peck of dirt" we are required to eat in our lifetime, about 9 L, weights about 13 kg, density of 1.5, equates to 500 mg/day. Likely, Swift, Keats, Uncle Remus and their contemporaries did not lead lives of our cleanliness. And their dirt was not as clean as ours. But even if water and food and air contained minimal Pb, inadvertent ingestion of even clean dirt, which has been documents, suggests a limit only about an order of magnitude yet lower.

What has this order of magnitude reduction in Pb over these last 2 generations done to the usual outcome measures? Linking any beneficial results with the long term trend in lower exposures would be make for a more powerful statements. In the case of IQ improvements, any efforts would be made more difficult in part because IQ test are usually z-scored and the so-called Flynn effect predicts trans-generation improvements in test scores, which have been seen in many countries and cultures. The quantitative Raven's CPM Test, which I've used in Taiwan, may offer some insights into changes in population IQ. We are left to wonder what improvements over time can be attributed to this decline in Pb. Has our collective blood pressure dropped? Pressure is a physical measurement, but also multi-causal. Clearly we have many less children severely poisoned by Pb now. What other results, other than increased margins of safety, have followed our nationwide drop in Pb levels? Any such lines of presentation might be useful.

Given our current exposure levels and standards, perhaps the time has come for us to declare some sort of victory and see if less emphasis could be placed on ambient Pb. Can we de-emphasize its statutory attention or its status as a primary pollutant. I am not seeking less attention to Pb poison prevention, childhood or otherwise. That seems more related to widespread residential sources and industrial settings. But perhaps we are reaching a point of diminishing returns in fighting lower ambient Pb levels.

5: I strongly concur that combining health and ecological effects of lead yields a more useful and effective integration of the scientific evidence. We are obligated to protect both realms, and MOAs can be clarified. My only real concern is direct conversion of doses and concentrations among species.

Because humans and animals occupy different environments and have different eating habits, our sensitivities to environmental lead may be more or less than some other animals. We have seen marine animals take up more lead if they live in the sediments versus animals that live in the water column. Furthermore, the fraction of the whole body burden that is in blood most likely varies among animals. Fish, birds, bovine, and human hemoglobin likely bind Pb with somewhat different strengths, which would profoundly affect their biokinetc distributions. That could be very useful. We have cases of Pb poisoned, nectar feeding birds and, and in another setting, meadow grazed horses, each being a sentinel species, their particular sensitivities proving useful for eventual human protection.

Specific Comments:

Page 2-1, line 5 - I suggest replacing “different” with “improved" (although 2-30 addresses this issue well in detail)

Page 2-1, end of 2nd paragraph – suggest adding ... along with a host of other miscellaneous sources such as ceramics, cosmetics, and plastic venetian blinds.

Page 2-3, para 2, line 5 - suggest adding ...processing, such as bearing metals in machinery and solder seals)

Page 2-3, para 4, last line - ...metric, in part because PbB is viewed as more biologically active and more homogenous than bone.

Page 2-5, Fig 2-1 - Please re-label pools of internal deposition so that "blood" appears in the middle box. Then, you can also add two-way arrows from that middle blood pool left and right to the bone and other indicating exchange between those pools via the blood. Otherwise, a useful and concise diagram.

Section 2.1.2 - perhaps have a table listing the 5 studies, sort of a convolution of
Table 2-1.

List by study and provide scenario time and level for each. Not really new information, just another way to show it.

Figure 2-2 - A+ good explanatory power

Page 2-11, note 10 - I do concur that using concurrent and some lifetime average is the way to go. At these levels, the paradigm of "windows of vulnerability" appears to have been overcome by the tendency for recovery. So, the effects of any earlier insults go un-noticed given subsequent lifetime exposures.

Table 2-3 - Maybe repetitive and long, but very useful

Page 2-32 - first full para, I agree

Page 3-1 - just curious, is reduced atmospheric visibility a welfare effect?
Page 3-1, para 3, line 7 - suggest ...other pollutants, such as ozone or NOX, adding these examples might add strength

Page 3-4 - good discussion of critical load, which I needed

Page 3-10, note 19 - good presentation, only afraid it might be lost in footnote.

Comments from Dr. William Stubblefield

The following are my comments/thoughts following my review of the draft Risk and Exposure Assessment Document pertaining to NAAQS for Pb:

- The REA Planning Document is a well-written document that provides a good discussion of the design and conduct of the ecological risk assessment done for the 2008 NAAQS process.

- The authors of the REA Planning Document are to be commended for wanting to take the time and effort to identify and consider "new" information in the assessment process. Care must be taken, however, when evaluating the data to ensure that consistency of endpoints are used across regulatory groups (this is somewhat addressed in Section 5.1.2). Inclusion of biochemical endpoints (as is suggested on page 4-8, lines 4-6) may represent a break in historical Agency approaches where only endpoints that could be associated with population level effects were considered (i.e., survival, growth and reproduction).

- A great deal of new toxicity information is available from non-published sources as a result of the new European REACH regulations and these data should be obtained and considered.

- As was previously noted, there is a great deal of new environmental toxicity data (i.e., effects data) available for Pb and this information will greatly improve our knowledge about the toxicity of Pb, the factors that influence the bioavailability of Pb in the aquatic and terrestrial environments, and will reduce uncertainty in our estimates of acceptable environmental concentrations. Unfortunately, risk assessment involves the evaluation of both effects and exposures. Efforts have been made to identify and consider media based Pb concentrations in soils, sediments and waters. However, there appears to be no way to attribute empirically determined media concentrations to airborne Pb concentrations or to any source. Without this link there seems little hope to estimate "safe" air Pb concentrations. To this end, the Agency should consider funding research to address the question of how one can appropriately estimate depositional Pb concentrations that result in field determined Pb concentrations. This is a critical need if this effort is to be successful.

Comments from Dr. Ian von Lindern

The REA is a well written and thought out document. The authors clearly understand and present the current situation, data gaps and uncertainties in developing a risk assessment for lead in the US. However, it is unfortunate that little new data regarding lead exposure in the US has evolved. Repeating the 2006-7 analysis would be a waste of scarce resources and poor expenditure of these talented scientists time. The EPA should seriously consider filling these data gaps with respect to exposure, or conduct other analyses of biological or blood lead databases to provide some comfort that children in the US are not exposed to excessive lead levels that could be addressed through enforcement of the NAAQS.

Comments from Dr. Gail Wasserman

Section on Health Risk Assessment

Overall, this section reads well and clearly states both its conclusions and the limitations in model-development.

Figure 2-1. The text on page 2-2 indicates that the boxes that are relevant to air-related pathways are indicated in bold, while in my version they are shaded. I believe shading works better, actually. The Figure clearly presents the focus of the earlier document to which it refers.

Figure 2-2. There is a typo in the lower right-hand box.

p.2-12. Although the 4 models are described, some more text about the justification and implications of each would be helpful for connecting these dots. Useful would also be an indication that the cutpoint in the "log-linear with cutpoint" model in the Lanphear meta-analysis was selected because of too few observations below this point to warrant inferences.

Figure 2-3. I think some other designation for either the first or third model in the legend would be helpful. The patterns of dots and dashes is quite similar. The legend on the Y axis should indicate that there are "points" lost.

My comments elsewhere on the metrics of IQ scoring and the clinical significance of small deficits, could be cross-referenced here. In particular the parsing of IQ scores into "points lost" that translates into fractions of a single point will be troublesome for most psychologists without some explanation, that might also include some discussion of the risk/benefit implications of interventions at very low blood lead levels.

Table 2-3 nicely lays out the new evidence and where the important gaps remain.

There appear some inconsistencies between the conclusions made in this table, and those apparent in Table 2-5 (and section 2.5.1) in the ISA. As examples, the ISA underscores the causal connection between exposure and both child behavior and adult cardiovascular concerns, outcomes for which the REA Planning Document notes unclear evidence. If the issue is that the existing data are insufficient to result in quantitative risk assessments (but sufficient to result in decisions about causality), that should be explicitly stated. In other words, more cross-talk between these documents would be helpful.