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

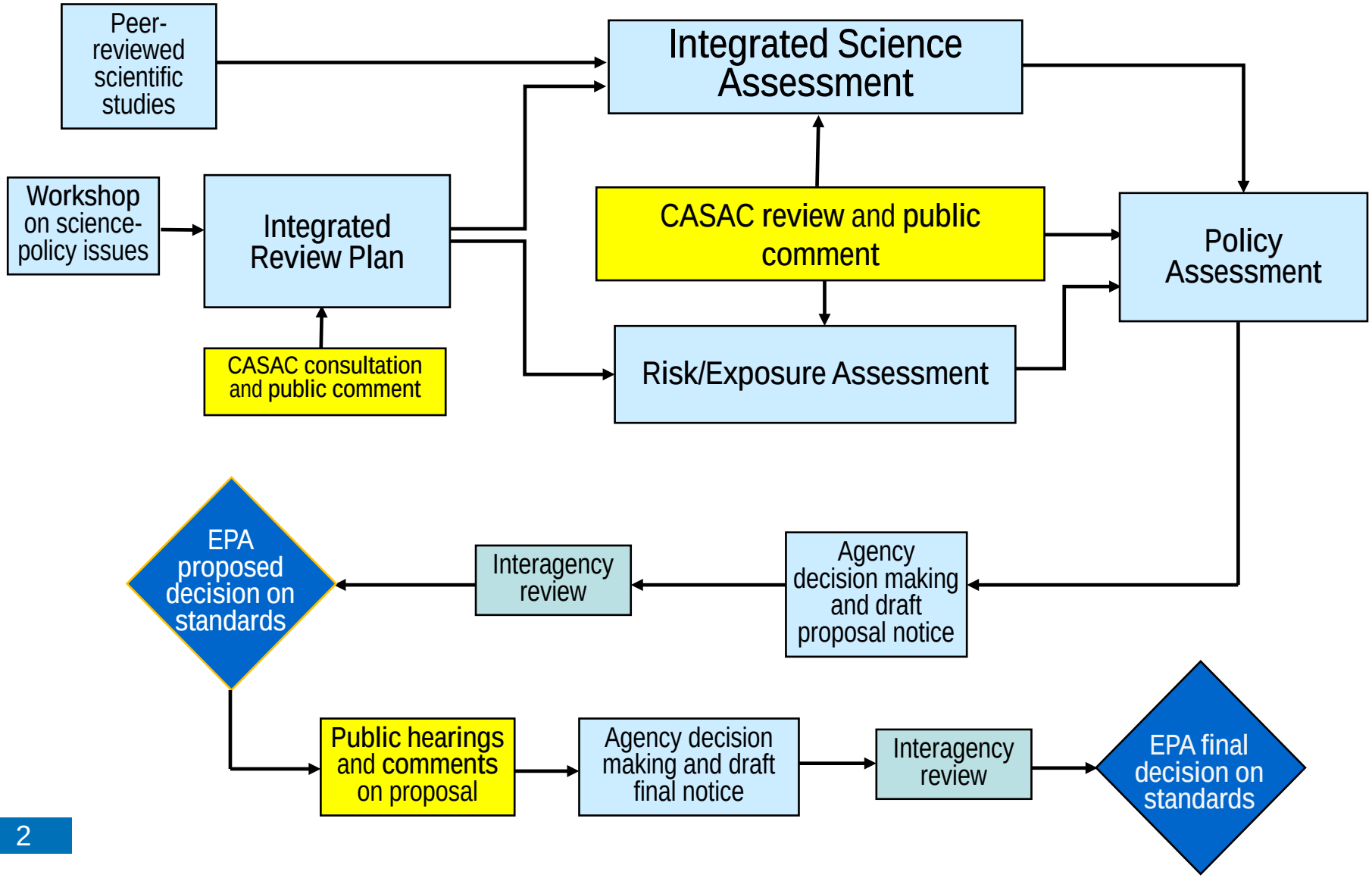
John J. Vandenberg, ORD/NCEA

Ellen Kirrane, ORD/NCEA

Debra B. Walsh, ORD/NCEA

RTP, NC
July 20-21, 2011

NAAQS Review Process



Schedule for the Current Review

Major Milestones		Projected Completion Date	Projected CASAC Review Date
Science and Policy Issue Workshop		May 2010	
Integrated Science Assessment Peer Input Workshop		December 2010	
Integrated Review Plan	Draft Final	March 2011 September 2011	May 5, 2011
Integrated Science Assessment	First Draft Second Draft Final	May 2011 Dec 2011 July 2012	July 20-21, 2011 * Feb/March 2012 *
Risk/Exposure Assessment	Planning Document If warranted, First Draft Second Draft Final	June 2010 Jan 2012 July 2012 Jan 2013	July 21, 2011 * Feb/March 2012 * Sept/Oct 2012
Policy Assessment (PA) Rulemaking	First Draft PA Second Draft PA Final PA Proposed Rulemaking Final Rulemaking	Aug 2012 Feb 2013 June 2013 Jan 2014 Nov 2014	Sept 2012 March 2013

*Indicates that a single CASAC meeting will address both documents

Pb ISA Team

NCEA Team:

Ellen Kirrane, Team Leader
James Brown
Jean-Jacques Dubois
Allen Davis
Tara Greaver
Erin Hines
Dennis Kotchmar
Meredith Lassiter
Qingyu Meng*
Stephen McDow
Elizabeth Owens
Molini Patel
Jennifer Richmond-Bryant
David Svendsgaard
Lindsay Stanek
Lisa Vinikoor-Imler

*ORISE Fellow

NCEA Management:

John Vandenberg, NCEA-RTP Director
Debra Walsh, Deputy Director
Mary Ross, Branch Chief

Production Team:

Connie Meacham
Deborah Wales

External Authors:

Ecology
Anne Fairbrother

Toxicology
Robyn Blain
Rodney Dietert
Jay Gandy
Harvey Gonick
William Mendez
Chandrika Moudgal
Mary Jane Selgrade
John Wise

Atmospheric Science
Margaret Graham
Howard Mielke

Epidemiology
Stephen Rothenberg
Virginia Weaver
Marc Weisskopf
Robert Wright
Rosalind Wright

Toxicokinetics
Gary Diamond

Chapters of the 1st ERD Pb ISA

Chapter 1: Introduction

Chapter 2: Integrative Health and Ecological Effects Overview

Chapter 3: Ambient Pb Source to Concentration

Chapter 4: Exposure, Toxicokinetics, and Biomarkers

Chapter 5: Integrated Health Effects of Pb Exposure

Chapter 6: Susceptible Populations

Chapter 7: Ecological Effects of Pb

General Charge

EPA has attempted to succinctly present and integrate the policy-relevant scientific evidence for the review of the Pb NAAQS.

Previous panels have emphasized the importance of older studies and concluded that if older studies are open to reinterpretation in light of newer data and/or they remain the definitive works available in the literature, they should be discussed in detail to reinforce key concepts and conclusions.

In considering subsequent charge questions and recognizing an overall goal of producing a clear and concise document, **are there topics that should be added or receive additional discussion?**

Similarly, **are there topics that should be shortened or removed?** Does the Panel have opinions on how the document can be shortened without eliminating important and necessary content?

Specific Charge – Chapter 1

The legislative history of Pb NAAQS reviews and the framework for causal determination and judging the overall weight of evidence is presented in Chapter 1.

Selection criteria used to identify studies for inclusion in the ISA are also described in Chapter 1. Please comment on the **consistency and appropriateness of the application of these criteria and the appropriateness of the decision to consider studies within approximately one order of magnitude of current exposure levels** (e.g. was the determination of "informative" occupational studies and their subsequent inclusion in the document appropriate and consistently applied across endpoints?)

Please comment on the application of the Health and Environmental Research Online (HERO) system to support a more transparent assessment process.

Specific Charge – Chapter 2

Chapter 2 presents the integrative summary and conclusions from the Pb ISA with a discussion of evidence presented in detail in subsequent chapters.

Is this a useful and effective summary presentation? Is the framework for causal determination appropriately applied?

Please comment on approaches that may improve the communication of key ISA findings to varied audiences. The health and ecological effects of Pb are mediated through multiple interconnected modes of action and there is substantial overlap between the ecological and health endpoints considered in the causal determinations. Since the mechanism of Pb toxicity is likely conserved from invertebrates to vertebrates to humans in some organ systems, **the scientific evidence was integrated across the disciplines of health and ecology**. Please comment on this approach e.g., **is this a useful and effective integration of the scientific evidence?**

Framework for Causal Determination

Weight of Evidence for Causal Determination

- Causal relationship
- Likely to be a causal relationship
- Suggestive of a causal relationship
- Inadequate to infer a causal relationship
- Not likely to be a causal relationship

United States Environmental Protection Agency

Summary of Causal Determinations for Health and Ecological Effects

Adapted from Table 2-5 1st ERD Pb ISA

Outcome	Human Health Causal Determination	Ecological Receptors Causal Determination
Neurological Effects	**Causal Relationship**	**Causal Relationship:** Invertebrates and Vertebrates
Cardiovascular Effects	**Causal Relationship**	N/A
Renal Effects	**Causal Relationship**	N/A
Immune System Effects	**Causal Relationship**	N/A
Heme Synthesis and RBC Function	**Causal Relationship**	**Causal Relationship**: Invertebrates and Vertebrates
Reproductive Effects and Birth Outcomes	**Causal Relationship**	Causal Relationship: Invertebrates and Vertebrates Inadequate to Infer Causal Relationship: Plants
Cancer	**Likely to be a causal relationship**	N/A
Bioaccumulation	N/A (Although Pb bioaccumulates in all organisms the ecological effect causal determination was made with respect to ecosystem services.)	**Causal Relationship**
Mortality	Note: Evidence of Pb-induced mortality in humans was considered under cardiovascular effects.	**Causal Relationship**: Invertebrates and Vertebrates Inadequate to Infer Causal Relationship: Plants
Growth	N/A	**Causal Relationship**: Plants and Invertebrates Suggestive of a Causal Relationship: Vertebrates
Physiological Stress	Note: Oxidative stress was considered as a upstream event in the Pb MOA .	**Causal Relationship** Note: Oxidative stress is a proxy indicator of overall fitness.
Community and Ecosystem Level Effects	N/A	**Causal Relationship**

Specific Charge – Chapter 3

Chapter 3 provides a wide range of information to inform the exposure and health sections of the ISA.

To what extent are the atmospheric science and air quality analyses presented in Chapter 3 clearly conveyed and appropriately characterized?

Is the information provided regarding **Pb source characteristics, fate and transport of Pb in the environment, Pb monitoring, and spatial and temporal patterns of Pb concentrations in air and non-air media accurate**, **complete, and relevant** to the review of the Pb NAAQS?

Does the ISA adequately characterize the available evidence on the **relationship between ambient air Pb concentrations and concentrations of Pb in other environmental media?**

Specific Charge – Chapter 4

Chapter 4 describes the multimedia nature of Pb exposure, toxicokinetics of Pb in humans, biomarkers of Pb exposure and body burden, as well as models of the relationship between Pb biomarkers and environmental Pb measurements.

4 a (of c)

How well do the choice and emphasis of topics provide a useful context for the evaluation of human health effects of Pb in the ISA? Is the current organization of the chapter clear and logical? Are there ways that information on exposure and toxicokinetics can be more clearly integrated throughout the chapter? **Does the ISA adequately describe and balance air-related and non-air related pathways of Pb exposure?**

Specific Charge – Chapter 4 (continued)

4b (of c)

Biological markers of Pb exposure and body burden are discussed in Section 4.3. **How well does this section reflect the current state of knowledge of Pb biomarkers and their interpretation as it relates to exposure and dose?** Is the focus on blood Pb and bone Pb appropriate, given that the epidemiologic literature largely assesses exposure through these two biomarkers? Is there sufficient and accurate discussion of the relationship between blood Pb and bone Pb? Are relationships between blood Pb and Pb in soft tissues and urine Pb adequately described?

4c (of c)

Section 4.5.1 discusses empirical models of air Pb-blood Pb relationships from new and old studies. This was an important policy issue in the last Pb NAAQS review. **Does this section accurately reflect what is known about air Pb-blood Pb relationships?** Are there particular studies that should receive less or greater emphasis?

Blood Pb-Air Pb Slope
(Adapted from Table 2-7)

- Relationship between blood Pb and air Pb very important to 2008 NAAQS consideration
- The nature of the blood Pb-air Pb slope depends on the model selected by the investigators
- Concentration ranges at which blood Pb-air Pb slopes were derived vary substantially (0.03-1.1 µg/m3 for Hilts and 0.2-10 µg/m3 for Brunekreef)

Reference	Population, Location	Slope (µg/dL per µg/m^3)
Schwartz and Pitcher (1989)	Children 0.5-7 yr, US	9.3
	Children 0-5 yr, Chicago	7.7
Hilts et al. (2003)	Children 0.5-6 yr, Trail, BC	6.5
Brunekreef et al. (1984)	Children, Various Countries	All: 18[A], 6.1[B] <20 µg/dL:13[A], 3.0[B]
Hayes et al. (1994)	Children 0.5-6 yr, Chicago	24[A], 5.7[B]
Tripathi et al. (2001)	Children 6-10 yr, Mumbai, India	3.6
Ranft et al. (2008)	Children 6-11 yr, Duisburg, Germany	3.2[C]
Schnaas et al. (2004)	Children 0.5-10 yr, Mexico City	4.8[A], 1.1[B]

A – Non-linear model, slope calculated for air Pb of 0.15 µg/m^3
B – Non-linear model, slope calculated for air Pb of 1 µg/m^3
C – Adjusted for soil Pb concentration, which may reduce slope

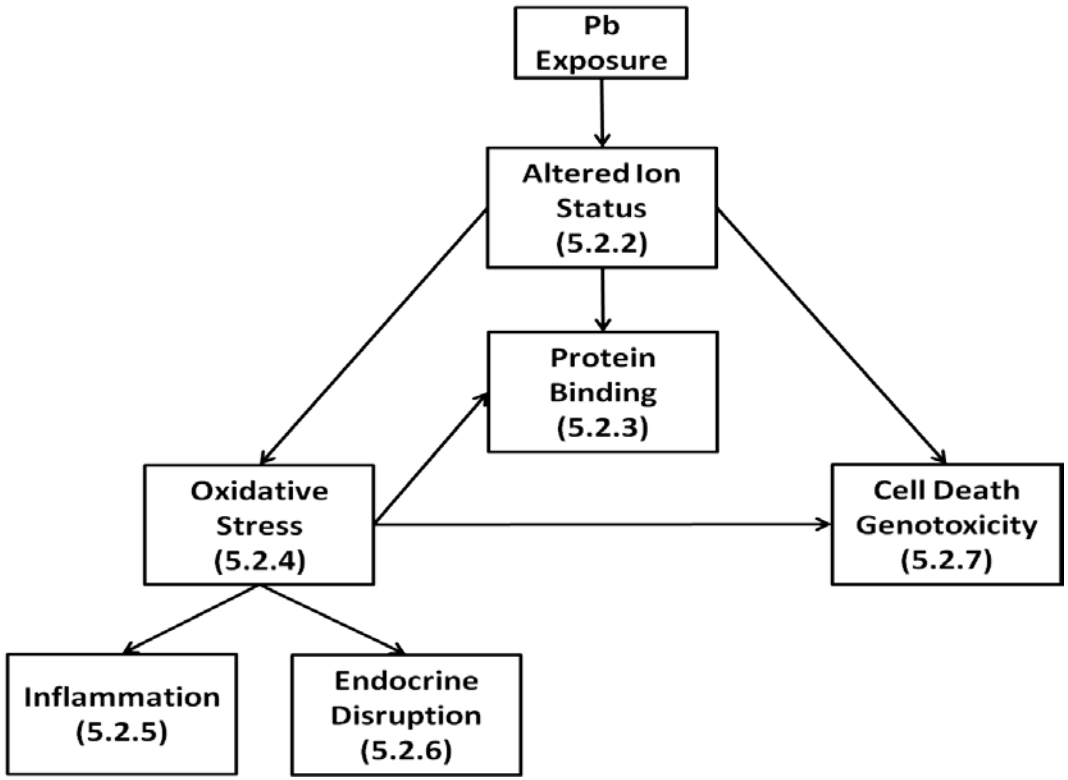
Specific Charge – Chapter 5

Chapter 5 presents assessments of the health effects of Pb, with evidence organized by health effect category, endpoint and scientific discipline.

5a (of e)

To what extent are the discussion and integration of the potential modes of action underlying the health effects of Pb exposure presented accurately and in sufficient detail? Are there additional modes of action that should be included in order to characterize fully the underlying mechanisms of Pb?

Modes of Action



- Pb-induced events upstream from observed health effects
- Multiple modes of action responsible for health effects
- Altered ion status is the major, unifying MOA

Specific Charge – Chapter 5 (continued)

5b (of e)

Does the ISA adequately describe the evidence with regard to the range of exposure concentrations (and/or blood or bone levels) associated with the identified endpoints?

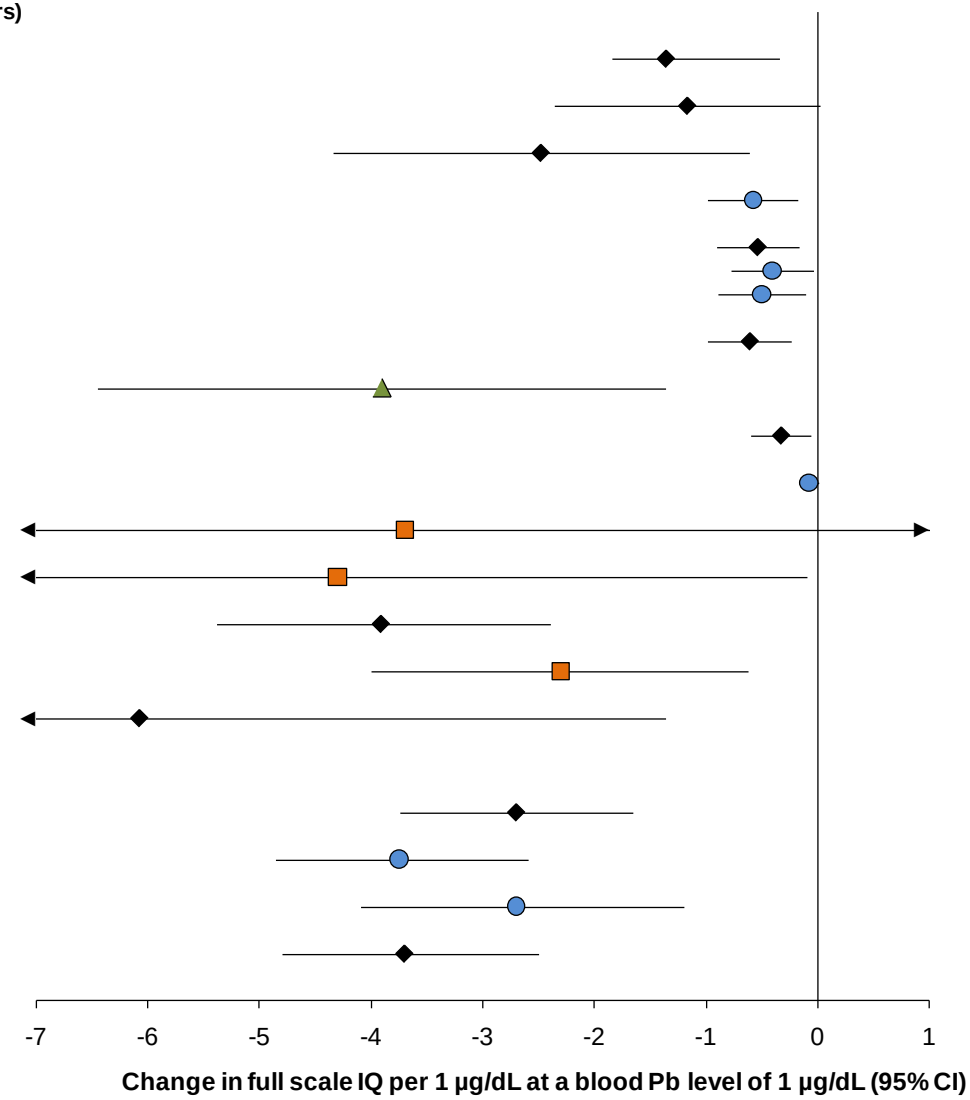
What are the views of the panel regarding the clarity and effectiveness of figures and tables in conveying information about the consistency of evidence for a given health endpoint, lifestage of exposure, or biomarker of exposure (e.g., blood versus bone Pb levels)?

Lowest blood Pb levels at which effects are substantiated: Children (adapted from Table 2-2)

Observed Effect Blood Pb Level	Neurological Effects	Renal Effects	Immune Effects	Reproductive Effects	Effects on Heme and Red Blood Cell Function
20 µg/dL			Macrophage hyper-inflammation		
15 µg/dL			Lymphocyte activation		↑Zn protoporphyrin Lipid peroxidation
10 µg/dL	Delinquent behavior ↑hearing threshold				Altered antioxidant enzyme activities ↓aminolevulinic acid dehydratase activity ↓hemoglobin
5 µg/dL 1 µg/dL	Inattention Decrements in full-scale IQ Decrements in specific neurocognitive domains Poorer school performance ADHD	Decreased estimated glomerular filtration rate	Increased B cell abundance Increased IgE Increased risk of infection Decreased T cell abundance Allergic sensitization	Delayed puberty	Decreased Ca-Mg ATPase activity (cell signaling)

Studies of blood Pb level and Full-Scale IQ in children

Study	Blood Pb Timing	Mean (SD) blood Pb (µg/dL)	FSIQ age (yrs)
Kim et al. (2009)	Concurrent	1.73 (0.80)	8-11
Walkowiak et al. (1998)	Concurrent	4.7 (2.3)	6
Chiodo et al. (2004)	Concurrent	5.4 (3.3)	7.5
Bellinger et al. (1992)	2 yrs	6.5 (4.9)	10
Min et al. (2009)	Concurrent	7.0	11
	4 yrs		9
	4 yrs		4
Canfield et al. (2003)	Concurrent	5.8 (4.1)	5
Schnaas et al. (2006)	Prenatal	7.8	6-10
Dietrich et al. (1993)	Concurrent	NR	6.5
Ris et al. (2004)	6.5 yrs	NR	15-17
Baghurst et al. (1992)	0 to 8 yr avg	21.7 (25th-75th mean)	7-8
Tong et al. (1996)	0 to 11-13 yr avg	17.8 (5.8)	11-3
Factor-Litvak et al. (1999)	Concurrent	30 and 8	7
Wasserman et al. (2003)	0 to 11-12 yr avg	31.6 and 6.3	10-12
Surkan et al. (2007)	Concurrent	5-10 vs 1-2	6-10
POOLED/META-ANALYSES			
Lanphear et al. (2005)	Concurrent	6.9 (1.2)	4.8-10
Schwartz et al. (1994)	2 to 5 yrs	6.5-23	Schoolage
Pocock et al. (1994)	~ 2 yrs	6.8-21.2	5-14
	Concurrent	7.4-23.7	5-14



Legend: ◆ Estimates for concurrent blood Pb; ● Estimates for blood Pb measured earlier in childhood;
■ Estimates for blood Pb levels averaged over multiple years; ▲ Estimates for prenatal blood Pb levels.

Specific Charge – Chapter 5 (continued)

5c (of e)

Should discussion of specific endpoints be expanded to provide a more comprehensive assessment of health effects associated with current Pb exposures in the U.S. population overall or in susceptible groups?

5 d (of e)

What are the views of the panel on the **integration of epidemiologic and toxicological evidence**, in particular, on the balance of emphasis placed on each discipline and the accuracy with which the evidence is presented? Considering the Pb exposure concentrations and durations in toxicological studies and the potential for confounding in epidemiological studies, please comment on the conclusions drawn about the coherence of the evidence and biological plausibility.

Neurological Effects (Causal)

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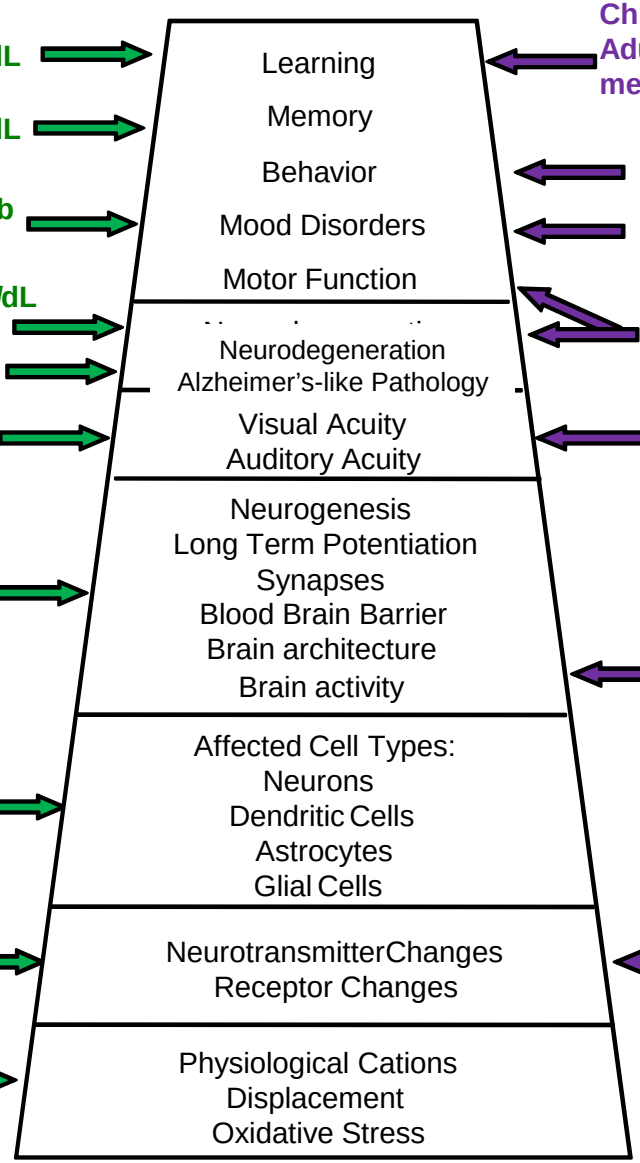
Toxicological

Epidemiologic



Rodents: blood Pb ≥ 11.6 µg/dL

Rodents: blood Pb ≥ 12.6 µg/dL

Rodents: residual blood Pb 5-7 µg/dL

Monkeys: bloodPb 19-26 µg/dL
Mice: blood Pb 46 µg/dL

Rodents: 0.78 µg/gPb

Male rodents: bloodPb 8-12 µg/dL

Rodents: bloodPb 12 µg/dL

Rodents: bloodPb 12-20 µg/dL

Rodents: blood Pb 11 µg/dL

Rodents andMonkeys: 20-200 pM Pb

Children: blood Pb quantiles ≥ 2 µg/dL
Adults: mean blood Pb ≥ 3 µg/dL; mean tibia Pb ≥ 11 µg/g

Children: blood Pb quantiles <1-8 µg/dL
Adults: mean blood Pb 8 µg/dL

Adults: blood Pb quantiles ≥ 3 µg/dL; mean tibia Pb 22 µg/g

Adults: mean blood Pb ≥ 2.7 µg/dL; tibia Pb quantile 19 µg/g

Children: mean blood Pb ≥ 7 µg/dL
Adults: mean patella Pb 32 µg/g

Adults: mean blood Pb 8 µg/dL

Boys: greater cognitive deficits with lower affinity dopamine receptor (mean blood Pb 6.1 µg/dL)

Specific Charge - Chapter 5 (continued)

5 e (of e)

The 2006 AQCD described a nonlinear dose-response relationship between blood Pb levels and cognitive function in children.

The ISA presents evidence from epidemiologic and toxicological studies to further evaluate potential explanations for the nonlinear shape (e.g., differential proportions of susceptible populations in different segments of the blood Pb level distribution, differential activation of mechanisms).

Please comment on the extent to which the expanded discussion is informative and consistent with the available evidence.

Specific Charge – Chapter 6

Chapter 6 is a discussion of potential susceptibility factors.

Are the characteristics included within the broad susceptibility categories appropriate and consistent with the definitions used?

Are there any key susceptibility factors that were not included and need to be added?

Is it appropriate to include material on susceptibility factors related to Pb exposure and dose, or should the chapter focus solely on susceptibility factors as they influence Pb-induced health effects?

Definition of Susceptibility

Individual- and population-level characteristics that increase the risk of Pb-related health effects in a population including, but not limited to: genetic background, birth outcomes (e.g., low birth weight, birth defects), race, sex, lifestage, lifestyle (e.g., smoking status, nutrition), preexisting disease, SES (e.g., educational attainment, reduced access to health care), and characteristics that may modify exposure to Pb (e.g., time spent outdoors).

Susceptible Populations and Lifestages

Factor Evaluated	Susceptible Population
Lifestage	Children
Sex	Outcome-dependent
Genetics	ALAD, VDR, DRD4, GSTM1, TNF-alpha, eNOS, APOE, HFE
Pre-existing Disease	Hypertension
Smoking	Smokers
Race/Ethnicity	Non-Hispanic Blacks, Hispanics
Socioeconomic Status	Low SES
Nutrition	Iron Deficiency
Stress	High Stress
Cognitive Reserve	Low cognitive reserve
Other Metals	Co-exposure to Cd, As, Mn

Specific Charge – Susceptibility (continued)

Susceptibility to Pb associated effects is also discussed in sections of the ISA other than Chapter 6.

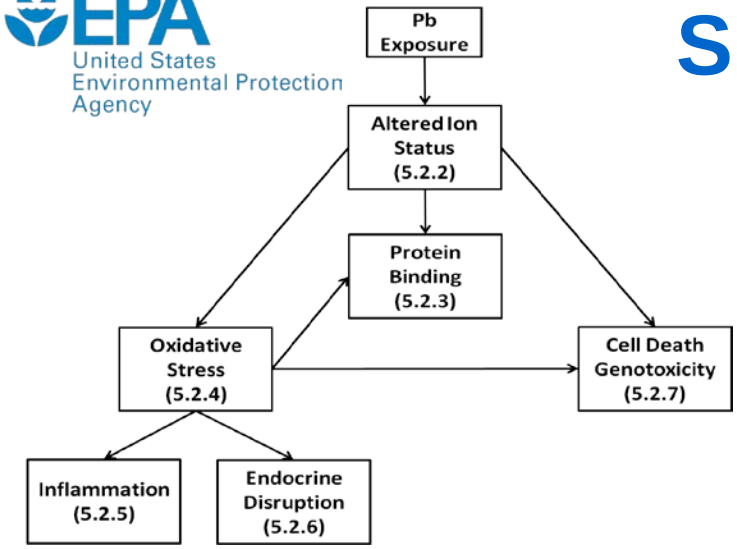
Does the ISA adequately cover and appropriately distinguish lifestage-dependent differences (e.g. differences between children and adults) as they relate to the modes of action of Pb, potential exposures to Pb, toxicokinetics and Pb biomarkers, health effects of Pb and susceptibility to Pb induced effects?

Specific Charge – Chapter 7

Chapter 7 is a discussion of the ecological effects of Pb. Effects on terrestrial and aquatic ecosystems are first considered separately. They are then integrated by classes of endpoints (bioaccumulation, growth, mortality, hematological effects, development and reproduction, neurobehavior, community and ecosystem effects).

Does the panel consider this approach appropriate?
Is it appropriate to derive a causal determination for bioaccumulation as it affects ecosystem services?

Shared Modes of Action



The same Pb-induced upstream events that lead to observed human health effects are found in ecosystems

Mode of Action	Evidence in Biota
Altered Ion Status	Plants, Invertebrates, Vertebrates
Protein Binding	Plants, Invertebrates, Vertebrates
Oxidative Stress	Plants, Invertebrates, Vertebrates
Endocrine Disruption	Invertebrates, Vertebrates
Cell Death/Genotoxicity	Plants, Invertebrates, Vertebrates

Effects Common to Human Health and Ecosystems

Outcome	Causality Determination
Neurobehavior: Invertebrates and Vertebrates	Causal
Developmental and Reproductive Effects: Invertebrates and Vertebrates	Causal
Hematological Effects: Invertebrates and Vertebrates	Causal
Physiological Stress: All Organisms	Causal

Ecosystem Effects

Outcome	Causality Determination
Bioaccumulation as it Affects Ecosystem Services: All Organisms	Causal
Mortality: Invertebrates and Vertebrates	Causal
Growth: Plants and Invertebrates	Causal
Growth: Vertebrates	Suggestive
Community and Ecosystem Level Effects	Causal

Specific Charge – Chapter 7 (continued)

Has the ISA adequately characterized the available information on the relationship between Pb exposure and effects on individual organisms and ecosystems, as well the range of exposure concentrations for the specific endpoints?

Are there subject areas that should be added, expanded upon, shortened or removed? If the ISA was expanded to consider dose-response in terrestrial systems, should we limit data to field soils? If the ISA were expanded to consider dose-response in aquatic systems, how might we most efficiently present toxicity data that varies greatly by organism, and environmental parameters that influence bioavailability (pH, dissolved organic carbon etc.)?

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The database includes more than 300,000 scientific articles from the peer-reviewed literature used by EPA to develop its Integrated Science Assessments (ISA) that feed into the NAAQS review. It also includes references and data from the Integrated Risk Information System (IRIS), a database that supports critical agency policymaking for chemical regulation. Risk assessments characterize the nature and magnitude of health risks to humans and the ecosystem from pollutants and chemicals in the environment.

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Read about how EPA scientists use HERO to find and evaluate key studies.

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Integrated Science Assessments (ISAs) for Criteria Air Pollutants

Lead | Ozone | Carbon Monoxide | Particulate Matter | NOx-SOx Eco

View the studies cited in the 2011 Lead Integrated Science Assessment First External Review Draft
View the studies considered for the 2011 Lead Integrated Science Assessment First External Review Draft

5/6/2011: EPA announced that the First External Review Draft of the Integrated Science Assessment (ISA) for Lead (Pb) has been made available for independent peer review and public review. This draft ISA represents a concise synthesis and evaluation of the most policy-relevant science and will ultimately provide the scientific bases for EPA's decision regarding whether the current standards for Pb sufficiently protect public health and the environment. Read the assessment.

Integrated Risk Information System (IRIS) Chemical Assessments

Chloroprene | 1,4 Dioxane | EGBE | Acrylamide | Methanol

View the studies used in the Toxicological Review of Chloroprene

Chloroprene (C4H5Cl) is a volatile, flammable liquid used primarily in the manufacture of polychloroprene or neoprene rubber. The latter is used to make diverse products, such as tires, wire coatings, and tubing. Human health risk concerns for chloroprene are primarily related to exposures via effluent and emissions from facilities that use chloroprene to produce polychloroprene elastomers or transport the product, although these exposure concerns have not been well characterized. Read the assessment.

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HERONet Home

What is HERO?

The Health and Environmental Research Online is a database of scientific studies and other references used to develop EPA's risk assessments aimed at understanding the health and environmental effects of pollutants and chemicals. It is developed and managed in EPA's Office of Research and Development (ORD) by the National Center for Environmental Assessment (NCEA).

What data does HERO provide?

For each reference, HERO contains:

- Reference type
- Citation elements: authors, title, year of publication, source. Depending on the type of reference, the citation will also include volume, page numbers, URL, etc.
- Abstract or brief description
- Topic areas that describe the reference (e.g., carbon monoxide, asthma)
- Assessment(s) in which the reference was used, if relevant. Note that HERO contains references *considered for use* in assessment development, not just those references actually used.
- For "key" studies: objective, quantitative extracted study data [future enhancement, planned for summer, 2010]

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Details

HERO ID	156743
Author(s)	Mauad T; Rivero DH; de Oliveira RC; Lichtenfels AJ; Guimaraes ET; de Andre PA; Kasahara DI; Bueno HM; Saldiva PH
Year	2008
Title	Chronic exposure to ambient levels of urban particles affects mouse lung development
Reference Type	Journal Article
Journal	American Journal of Respiratory and Critical Care Medicine
Volume	178
Page(s)	721-728
Abstract	RATIONALE: Chronic exposure to air pollution has been associated with adverse effects on children's lung growth. OBJECTIVES: We analyzed the effects of chronic exposure to urban levels of particulate matter (PM) on selected phases of mouse lung development. METHODS: The exposure occurred in two open-top chambers (filtered and nonfiltered) placed 20 m from a street with heavy traffic in São Paulo, 24 hours/day for 8 months. There was a significant reduction of the levels of PM(2.5) inside the filtered chamber (filtered = 2.9 +/- 3.0 microg/m(3), nonfiltered = 16.8 +/- 8.3 microg/m(3); P = 0.001). At this exposure site, vehicular sources are the major components of PM(2.5) (PM
Cited In	PM 2009

Lead Landing Page

You are here: EPA Home » Research » NCEA » HERO » Lead Integrated Science Assessment (ISA) – References

Lead Integrated Science Assessment (ISA) – References

May 6, 2011: EPA released the ISA for Lead (first external review draft). Evidence from recent studies of the health and ecological effects of lead was reviewed, evaluated, and integrated with the evidence that was included in the 2006 Lead Air Quality Criteria Document. Literature searches were conducted to identify studies published since the 2006 review.

This page includes references cited in the ISA, as well as those considered, but not cited. You may refine by subject, health effect, and ecological effect. Click on the title of the reference to see more details.

References "considered for inclusion" were not added systematically. We are working toward a more systematic method for future ISAs.

Considered for inclusion (sorted by author)

Page: 1 2 3 4 5 Next Last (5490 references)

Comparison of methods for measuring atmospheric deposition of arsenic, cadmium, nicke and lead
Authors: Aas, W.; Alleman, L. Y.; Bieber, E.; Gladtke, D.; Houdret, J. L.; Karlsson, V.; Monies, C.
Source: Journal of Environmental Monitoring, 11:(6)1276–1283.
Year: 2009
Tags: Health Effects

Lead poisoning as possible cause of deaths at the Swedish House at Kapp Thordsen, Spitsbergen, winter 1872–3
Authors: Aasebo, U.; Kjaer, K. G.
Source: BMJ, 339:b5038.
Year: 2009
Tags: Health Effects

Guidance for risk assessment of exposure to lead: A site-specific, multi-media approach
Authors: Abadin, H. G.; Wheeler, J. S.
Source: In Hazardous waste and public health: International congress on the health effects hazardous waste, 477–485. Princeton, NJ: Princeton Scientific Publishing. .
Year: 1997
Tags: Toxicokinetics

Reversal of ionoregulatory disruptions in occupational lead exposure by vitamin C
Authors: Abam, E.; Okediran, B. S.; Odukoya, O. O.; Adamson, I.; Ademuyiwa, O.
Source: Environmental Toxicology and Pharmacology, 26:(3)297–304.
Year: 2008
Tags: Cardiovasc/Mortality, Health Effects, Hematological

Effect of lead toxicity on coenzyme Q levels in rat tissues
Authors: Abdallah, G. M.; El-Sayed, E. S.; Abo-Salem, O. M.
Source: Food and Chemical Toxicology, 48:(6)1753–1756.
Year: 2010
Tags: Health Effects, Renal

Refine By:
- (•) Considered References
- ( ) Cited References

Subjects
- [x] All Subjects
- [ ] Atmospheric Science
- [ ] Exposure
- [ ] Toxicokinetics
- [ ] Health Effects
- [ ] Ecological Effects

Ecological Effects
- [x] All Ecological Effects
- [ ] Aquatic Effects
- [ ] Terrestrial Effects

Health Effects
- [x] All Health Effects
- [ ] Neurological
- [ ] Cardiovasc/Mortality
- [ ] Renal
- [ ] Immune
- [ ] Repro/Develop
- [ ] Cancer/Genotox
- [ ] Hematological
- [ ] Other Organs

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LitReported (NOx Data)

NOx studies

Back to List of Reports

Study	Age	Country	Pollutant	Exposure Duration	Endpoint Category	Endpoint	Outcome	Clinical Measurement	Multicity?	Copollutant?	Susceptibility?	Age Category	Age Start	Age End	Findings
Adamkiewicz et al. (2004)		US	03, NOx, O3, PM2.5, S02	ST	Resp	AHR, Symptoms									Participant characteristics and their relationship with the fraction of exhaled NO (FENO) can be found in Table 1, pg 206; Ambient pollution and meteorological variables during breath sampling can be found in Table 2 on page 206; Association of FENO with PM2.5 at different lags between exposure and effect can be found in Figure 1 on page 207; Exposure effect estimates for single pollutant models can be found in Table 3, page 207; Multipollutant models for PM2.5 and NO can be found in Table 4 on page 207.
Arbex et al. (2009)		Brazil	CO, NO2, O3, PM10, SO2	ST	Resp		ED Visits				Yes				Descriptive analyses of COPD ED visits can be found in Table 1 on page 5; A summary of pollutant and meteorological variables can be found in Table 2 on page 6; Percentage increases and 95% Cis for COPD-related ED visits due to interquartile increases in PM10, SO2, and CO by gender and age can be found in Table 3 on page 7.
Barraza-Villarreal et al. (2008)		Mexico	NO2, O3, PM2.5	ST	Resp			AHR, Pulmonary Function				Age Range	7	11	Basic characteristics and main outcomes of the study population can be found in Table 1 on page 834; Eight-hour moving average concentrations of PM2.5 during the study period can be found in Figure 2, page 834; Air pollutants and

Meeting Agenda
Wednesday July 20, 2011

9:00 a.m.	Convene Meeting
9:05 a.m.	Welcome
9:10 a.m.	Introduction of Panel Members, Purpose of Meeting and Review of Agenda
9:20 a.m.	EPA Presentation on the Lead Integrated Science Assessment (ISA)
10:00 a.m	Public Comments
10:15 a.m.	Break
10:30 a.m.	Discussion of Charge Question 1 – Study Selection
11:15 a.m.	Discussion of Charge Question 3 – Ambient Lead
12:00 p.m.	Lunch
1:00 p.m.	Discussion of Charge Question 4 – Exposure, Toxicokinetics, Biomarkers
1:40 p.m.	Discussion of Charge Question 5 – Integrated Health Effects of Lead Exposure
2:45 p.m.	Break
3:00 p.m.	Discussion of Charge Question 7 – Ecological Effects of Pb
4:00 p.m.	Discussion of Charge Question 6 – Susceptible Populations and Life Stages
4:30 p.m.	Discussion of Charge Question 2 – Integrative Health and Ecological Effects Overview
5:15 p.m.	Summary of Action Items
5:30 p.m.	Adjourn