



Policy Assessment for the Review of the Lead National Ambient Air Quality Standards

External Review Draft

This page is intentionally blank.

EPA-452/P-13-001
January 2013

**Policy Assessment
for the Review of the Lead
National Ambient Air Quality Standards**

External Review Draft

U.S. Environmental Protection Agency
Office of Air Quality Planning and Standards
Health and Environmental Impacts Division
Research Triangle Park, North Carolina

DISCLAIMER

This draft document has been reviewed by the Office of Air Quality Planning and Standards (OAQPS), U.S. Environmental Protection Agency (EPA), and approved for publication. This OAQPS Policy Assessment contains preliminary conclusions of the staff of the OAQPS and does not necessarily reflect the views of the Agency. Mention of trade names or commercial products is not intended to constitute endorsement or recommendation for use.

ACKNOWLEDGMENTS

This draft Policy Assessment is the product of the Office of Air Quality Planning and Standards. It has been developed as part of the Environmental Protection Agency's (EPA) ongoing review of the national ambient air quality standards (NAAQS) for lead (Pb). The Pb NAAQS review team has been led by Dr. Deirdre Murphy. Dr. Karen Martin has managed the project. For the chapter on the health effects evidence and exposure/risk information, the principal authors include Dr. Deirdre Murphy and Dr. Zach Pekar, and Dr. Murphy served as the principal author for the chapter on the primary Pb standard. For the chapters on welfare effects evidence and exposure/risk information and the secondary Pb standard, the principal author is Ms. Ginger Tennant. The principal author on the section discussing ambient air monitoring is Mr. Kevin Cavender. Contributors of emissions information and air quality analyses are Mr. Marc Houyoux, Mr. Josh Drukenbrod, Dr. Halil Cakir and Mr. Mark Schmidt. Staff from other EPA offices, including the Office of Research and Development and the Office of General Counsel, also provided valuable comments and contributions.

This page is intentionally blank

TABLE OF CONTENTS

List of Figures	iv
List of Tables	v
EXECUTIVE SUMMARY	ES-1
1 INTRODUCTION	1-1
1.1 PURPOSE.....	1-1
1.2 BACKGROUND	1-3
1.2.1 Legislative Requirements.....	1-3
1.2.2 History of Lead NAAQS Reviews.....	1-5
1.2.3 Current Lead NAAQS Review.....	1-7
1.3 SCOPE OF CURRENT REVIEW: FATE AND MULTIMEDIA PATHWAYS OF AMBIENT AIR LEAD.....	1-9
1.3.1 Environmental Distribution and Exposure Pathways.....	1-10
1.3.1.1 Human Exposure Pathways.....	1-11
1.3.1.2 Ecosystem Exposure Pathways	1-12
1.3.2 Considerations Related to Historically Emitted Lead.....	1-13
1.4 GENERAL ORGANIZATION OF THE DOCUMENT	1-15
1.5 REFERENCES	1-16
2 AMBIENT AIR LEAD	2-1
2.1 SOURCES AND EMISSIONS TO AMBIENT AIR	2-1
2.1.1 Temporal Trends on a National Scale.....	2-2
2.1.2 Sources and Emissions on National Scale - 2008	2-3
2.1.2.1 Stationary Sources.....	2-5
2.1.2.2 Mobile Sources.....	2-6
2.1.2.3 Natural Sources and Long-range Transport	2-7
2.1.2.4 Previously Deposited or Released Lead.....	2-7
2.1.3 Sources and Emissions on Local Scale	2-9
2.2 AMBIENT AIR QUALITY.....	2-12
2.2.1 Air Monitoring	2-12
2.2.1.1 Lead NAAQS Surveillance Network.....	2-12
2.2.1.2 Other Lead Monitoring Networks.....	2-16
2.2.1.3 NAAQS Surveillance Monitoring Considerations.....	2-19
2.2.1.3.1 Sampling Considerations	2-19
2.2.1.3.2 Analysis Considerations	2-20
2.2.1.3.3 Network Design Considerations	2-21
2.2.2 Ambient Concentrations.....	2-24
2.2.2.1 Temporal Trends	2-24
2.2.2.2 Current Concentrations	2-26
2.3 AMBIENT AIR LEAD IN OTHER MEDIA	2-32

2.3.1	Atmospheric Deposition.....	2-32
2.3.2	Terrestrial Media	2-33
2.3.2.1	Indoor Household Dust	2-34
2.3.2.2	Outdoor Dust in Areas of Human Activity	2-34
2.3.2.3	Soil	2-36
2.3.2.4	Biota	2-38
2.3.3	Aquatic Media.....	2-38
2.3.3.1	Surface Waters	2-38
2.3.3.2	Sediments.....	2-39
2.4	REFERENCES	2-40
3	HEALTH EFFECTS AND EXPOSURE/RISK INFORMATION	3-1
3.1	INTERNAL DISPOSITION AND BIOMARKERS OF EXPOSURE AND DOSE ..	3-1
3.2	NATURE OF EFFECTS.....	3-14
3.3	PUBLIC HEALTH IMPLICATIONS AND AT-RISK POPULATIONS	3-27
3.4	EXPOSURE AND RISK	3-36
3.4.1	Conceptual Model for Air-Related Lead Exposure and Risk	3-37
3.4.2	Case Studies	3-39
3.4.3	Analysis Approach	3-41
3.4.3.1	Estimating Exposure	3-43
3.4.3.2	Air Quality Scenarios Included in 2007 Assessment.....	3-46
3.4.3.3	Methods for Deriving Risk Estimates	3-48
3.4.3.3.1	Full Risk Model in 2007 REA	3-49
3.4.3.3.2	Air Quality Scenarios Reflecting the Current Standard.....	3-51
3.4.4	Challenges in Characterizing Air-related Exposure and Risk.....	3-53
3.4.5	Risk Estimates	3-55
3.4.6	Treatment of Variability.....	3-60
3.4.7	Characterizing Uncertainty	3-61
3.4.8	Updated Interpretation of Risk Estimates	3-64
3.5	REFERENCES	3-66
4	REVIEW OF THE PRIMARY STANDARD FOR LEAD.....	4-1
4.1	APPROACH	4-1
4.1.1	Approach Used in the Last Review.....	4-2
4.1.1.1	Conclusion Regarding the Need for Revision.....	4-3
4.1.1.2	Conclusions on Elements of Revised Standard.....	4-5
4.1.2	Approach for the Current Review	4-10
4.2	ADEQUACY OF THE CURRENT STANDARD.....	4-13
4.2.1	Evidence-based Considerations.....	4-13
4.2.2	Exposure/Risk-based Considerations	4-21
4.3	PRELIMINARY STAFF CONCLUSIONS ON THE PRIMARY STANDARD.....	4-25
4.4	KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH AND DATA COLLECTION	4-33
4.5	REFERENCES	4-36

5	WELFARE EFFECTS AND RISK/RISK INFORMATION	5-1
5.1	WELFARE EFFECTS INFORMATION	5-1
5.2	EXPOSURE AND RISK INFORMATION	5-13
5.2.1	Screening Assessment from Last Review	5-14
5.2.2	Screening Assessment Results and Interpretation.....	5-17
5.3	REFERENCES	5-22
6	REVIEW OF THE SECONDARY STANDARD FOR LEAD.....	6-1
6.1	APPROACH	6-1
6.1.1	Approach Used in the Last Review.....	6-2
6.1.2	Approach for the Current Review	6-3
6.2	ADEQUACY OF THE CURRENT STANDARD.....	6-5
6.2.1	Evidence-based Considerations.....	6-5
6.2.2	Exposure/Risk-based Considerations.....	6-9
6.3	PRELIMINARY STAFF CONCLUSIONS ON THE SECONDARY STANDARD.....	6-10
6.4	KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH AND DATA COLLECTION	6-12
6.5	REFERENCES	6-14
 CHAPTER APPENDICES		
	Appendix 2A. The 2008 NEI: Data Sources, Limitations and Confidence	2A-1
	Appendix 2B. Recent Regulatory Actions on Stationary Sources of Lead	2B-1
	Appendix 2C. Criteria for Air Quality Data Analysis	2C-1
	Appendix 2D. Air Quality Data Analysis Summary	2D-1
	Appendix 3A. Interpolated Risk Estimates for the Generalized (Local) Urban Case Study ..	3A-1
	Appendix 5A. Additional Detail on 2006 Ecological Screening Assessment	5A-1

LIST OF FIGURES

Figure 1-1. Pathways of human and ecosystem exposure to lead from ambient air.	1-11
Figure 2-1. Temporal trend in air emissions: 1970-2010.	2-3
Figure 2-2. Geographic distribution of facilities estimated to emit at least 0.50 tpy in 2008.	2-11
Figure 2-3. Map of Pb-TSP Monitoring Sites in Current Pb NAAQS Monitoring Network.	2-15
Figure 2-4. Map of sites near airports for which one year of monitoring is required.	2-16
Figure 2-5. Pb-PM ₁₀ monitoring sites.	2-17
Figure 2-6. Pb-PM _{2.5} monitoring sites in CSN and IMPROVE networks (2012).	2-18
Figure 2-7. Temporal trend in Pb -TSP concentrations: 1980-2010 (31 sites).	2-24
Figure 2-8. Temporal trend in Pb-TSP concentrations: 2000-2010 (92 sites).	2-24
Figure 2-9. Airborne Pb -TSP concentrations (3-month average) at five sites near roadways: 1979-2010.	2-25
Figure 2-10. Pb-TSP maximum 3-month means (215 sites), 2009-2011.	2-27
Figure 2-11. Distribution of maximum 3-month mean concentrations of Pb-TSP, Pb-PM ₁₀ and Pb-PM _{2.5} at different site types, 2009-2011.	2-29
Figure 2-12. Distribution of annual mean concentrations of Pb-TSP, Pb-PM ₁₀ and Pb-PM _{2.5} at different site types, 2009-2011.	2-30
Figure 2-13. Distribution of maximum monthly mean concentrations of Pb-TSP, Pb-PM ₁₀ and Pb-PM _{2.5} at different site types, 2009-2011.	2-31
Figure 2-14. Temporal trend in sediment concentration from core samples in 12 lakes at eight National Parks or Preserves.	2-41
Figure 3-1. Temporal trend in mean blood Pb levels for NHANES cohorts.	3-4
Figure 3-2. Human exposure pathways for air-related lead.	3-38
Figure 3-3. Overview of analysis approach.	3-42
Figure 3-4. Comparison of four concentration-response functions used in risk assessment.	3-50
Figure 3-5. Parsing of air-related risk estimates.	3-53
Figure 4-1. Overview of approach for review of current primary standard.	4-12
Figure 5-1. Analytical approach for screening-level assessment in the last review (2006 REA, Exhibit 2-6).	5-16
Figure 6-1. Overview of approach for review of current secondary standard.	6-4

LIST OF TABLES

Table 2-1. U.S. lead emissions by source categories estimated to emit at least 4 tpy.	2-4
Table 2-2. Facilities estimated to emit at least 0.50 tpy in 2008.	2-10
Table 2-3. Dry deposition of lead in large metropolitan areas and in a smelter town.	2-36
Table 3-1. Empirically derived air-to-blood ratios for populations inclusive of children.	3-12
Table 3-2. Associations with neurocognitive function measures in analyses with child study group blood lead levels <5 µg/dL.	3-21
Table 3-3. Summary of quantitative relationships of IQ and blood Pb for analyses with blood Pb levels closest to those of young children in the U.S. today.	3-26
Table 3-4. Number of children aged 5 and under in areas of elevated ambient Pb concentrations relative to the NAAQS.	3-34
Table 3-5. Population size near larger sources of lead emissions.	3-35
Table 3-6. Types of population exposures assessed.	3-40
Table 3-7. Summary of approaches used to estimate case study media concentrations.	3-44
Table 3-8. Air quality scenarios assessed.	3-48
Table 3-9. Comparison of total and incremental IQ loss estimates for blood Pb below 10 µg/dL based on the four concentration-response functions.	3-50
Table 3-10. Estimates of air-related risk from 2007 risk assessment.	3-57
Table 3-11. Estimates of air-related risk for the generalized (local) urban case study, including interpolated estimates for current standard.	3-58

This page is intentionally blank

EXECUTIVE SUMMARY

This draft Policy Assessment (PA) has been prepared by staff in the Environmental Protection Agency's (EPA) Office of Air Quality Planning and Standards (OAQPS) as part of the Agency's ongoing review of the primary (health-based) and secondary (welfare-based) national ambient air quality standards (NAAQS) for lead (Pb). It presents analyses and preliminary staff conclusions regarding the policy implications of the key scientific and technical information that informs this review. The final PA is intended to "bridge the gap" between the relevant scientific evidence and technical information and the judgments required of the EPA Administrator in determining whether to retain or revise the current standards. Development of the PA is also intended to facilitate advice and recommendations on the standards to the Administrator from an independent scientific review committee, the Clean Air Scientific Advisory Committee (CASAC), as provided for in the Clean Air Act (CAA).

Staff analyses in this draft PA are based on the scientific assessment presented in the third draft *Integrated Science Assessment for Pb* (ISA) prepared for this review by the EPA's Office of Research and Development (ORD) as well as scientific and technical assessments from prior Pb NAAQS reviews. Such assessments include quantitative human health and ecological risk and exposure assessments (REAs) developed in the last review as new health and ecological REAs were not warranted based on staff's and CASAC's consideration of the evidence newly available in this review with regard to risk and exposure assessment. In considering the scientific evidence and other technical information available in this review, emphasis is given to consideration of the extent to which the evidence newly available since the last review alters conclusions drawn in the last review with regard to health and welfare effects of Pb, the exposure levels at which they occur and the associated at-risk populations and ecological receptors or ecosystems.

The overarching questions in this review, as in all NAAQS reviews, regard the support provided by the currently available scientific evidence and exposure/risk-based information for the adequacy of the current standards and the extent to which the scientific evidence and technical information provides support for concluding that consideration of alternative standards may be appropriate. The analyses presented in this draft PA to address such questions lead to preliminary staff conclusions that it is appropriate to consider retaining the current primary and secondary standards without revision; accordingly, no potential alternative standards have been identified by staff for consideration in this review. Comments and recommendations from CASAC, and public comments, based on review of this draft PA, will inform final staff conclusions and the presentation of information in the final PA.

1 Current Lead NAAQS and Scope of Review

2 The NAAQS for Pb was initially set in 1978. Review of the 1978 NAAQS for Pb,
3 completed in October 2008, resulted in substantial revision based on the large body of evidence
4 accumulated over the intervening three decades. In terms of the basic elements of the NAAQS,
5 the *level* of the primary standard was lowered by an order of magnitude from 1.5 $\mu\text{g}/\text{m}^3$ to 0.15
6 $\mu\text{g}/\text{m}^3$ and the *averaging time* was revised to a rolling three-month period (from a period based
7 on calendar quarters) with a maximum (not-to-be exceeded) *form*, evaluated over a 3-year
8 period. The *indicator* of Pb in total suspended particles (Pb-TSP) was retained, reflecting the
9 evidence that Pb particles of all sizes pose health risks. The secondary standard was revised to
10 be identical in all respects to the revised primary standard.

11 The multimedia and persistent nature of Pb contributes complexities to the review of the
12 Pb NAAQS unlike issues addressed in other NAAQS reviews. Air-related Pb distributes from
13 air to other media, including indoor and outdoor dusts, soil, food, drinking water, as well as
14 surface water and sediments. As a result, review of the Pb NAAQS considers the protection
15 provided against the health and environmental effects of air-related Pb associated both with
16 exposures to Pb in ambient air and with exposures to Pb that makes its way from ambient air into
17 other media. Additional complexity derives from the recognition that exposure to Pb also results
18 from nonair sources, including Pb in paint, tap water affected by plumbing containing Pb, lead-
19 tainted products, as well as surface water discharges and runoff from industrial sites. Such
20 nonair sources contribute to the total burden of Pb in the human body and in the environment,
21 making it much more difficult to assess independently the health and welfare effects attributable
22 to air-related Pb that are the focus of the NAAQS. Further, the persistence of Pb in the human
23 body and the environment is another important consideration in assessing the adequacy of the
24 current Pb standards. In so doing, staff is mindful of the history of the greater and more
25 widespread atmospheric emissions that occurred in previous years (e.g., under the previous Pb
26 standard, and prior to establishment of any Pb NAAQS) and that contributed to the Pb that exists
27 in human populations and ecosystems today. Likewise, staff also recognizes the role of nonair
28 sources of Pb, now and in the past, that also contribute to the Pb that exists in human populations
29 and ecosystems today. As in the last Pb NAAQS review, this backdrop of environmental Pb
30 exposure, and its impact on the populations and ecosystems which may be the subjects of the
31 currently available scientific evidence, complicates our consideration of the health and welfare
32 protection afforded by the current NAAQS.

1 **Characterization of Ambient Air Lead**

2 Emissions to ambient air and associated air Pb concentrations have declined substantially
3 over the past several decades. The most dramatic reductions in Pb emissions occurred prior to
4 1990 in the transportation sector due to the removal of Pb from gasoline. Lead emissions were
5 further reduced substantially between 1990 and 2008, with significant reductions occurring in the
6 metals industries at least in part as a result of national emissions standards for hazardous air
7 pollutants. Additional reductions in stationary source emissions are also anticipated from
8 regulations which have been promulgated since 2008 under Section 112 of the CAA.

9 As at the time of the last review, the majority of Pb emissions nationally is associated
10 with combustion of leaded aviation gasoline by piston-driven aircraft. The largest sources on a
11 local scale are generally associated with metals industries. As a result of revisions to monitoring
12 regulations stemming from the last Pb NAAQS review, Pb NAAQS monitors are required near
13 the largest Pb emissions sources, as well as in sites distant from such sources in large population
14 areas. Ambient air Pb monitoring data available thus far from this expanded network continue to
15 illustrate the source-related aspect of airborne Pb, with highest concentrations near large sources
16 and lowest in areas removed from sources. In addition, Pb monitoring data are also being
17 collected over at least a one-year period near a set of airports identified as most likely to have
18 elevated Pb concentrations due to leaded aviation gasoline usage. These data will inform future
19 airport monitoring activities, as well as an ongoing investigation into Pb emissions from piston-
20 engine aircraft under Section 231 of the Clean Air Act, separate from this Pb NAAQS review.

21 Lead occurs in ambient air in particulate form and, with characteristics and spatial
22 patterns influenced by a number of factors, deposits from air to surfaces in natural and human-
23 made environments. By this deposition process and subsequent transfer processes, ambient air
24 Pb is distributed into multiple human exposure pathways and environmental media in aquatic
25 and terrestrial ecosystems. In areas removed from large air emissions sources, currently
26 available information on Pb concentrations in nonair media includes numerous examples of
27 declines in surface concentrations reflective of the reductions in deposition over the past several
28 decades. In areas near large air sources where emissions reductions have occurred, only very
29 limited information is available, such as for reductions in air and surface dust concentrations,
30 with even less information available on trends for other media such as surface soils.

31 **Health Effects and Review of the Primary Standard**

32 Lead has long been recognized to exert a broad array of deleterious effects on multiple
33 organ systems as described in the ISA for this review and consistent with conclusions of prior
34 scientific assessments. Over the three decades from the time the standard was initially set in
35 1978 through its revision with the NAAQS review completed in 2008, the evidence base

1 expanded considerably in a number of areas, including with regard to effects on neurocognitive
2 function in young children at increasingly lower blood Pb levels. These effects formed the
3 primary basis for the 2008 revisions to the primary standard. The current standard was set most
4 specifically to provide appropriate public health protection from the effects of air-related Pb on
5 cognitive function (e.g., IQ loss) in young children. In so doing, the standard was judged to
6 provide the requisite public health protection from the full array of health effects of Pb,
7 consistent with the CAA requirement that the primary standard, in the judgment of the
8 Administrator, based on the latest scientific knowledge, is requisite to protect public health with
9 an adequate margin of safety.

10 The health effects evidence newly available in this review, as critically assessed in the
11 ISA in conjunction with the full body of evidence, reaffirms conclusions on the broad array of
12 effects recognized for Pb in the last review. Further, staff observes the general consistency of the
13 current evidence with the evidence available in the last review, particularly with regard to key
14 aspects of the evidence on which the current standard is based. These key aspects include those
15 regarding the relationships between air Pb concentrations and the associated Pb in the blood of
16 young children (i.e., air-to-blood ratios) as well as between total blood Pb levels and effects on
17 neurocognitive function (i.e., concentration-response (C-R) functions for IQ loss). Factors
18 characterizing these two relationships are the key inputs to the framework developed in the last
19 review to translate the available evidence into a basis for considering a primary Pb standard that
20 would be requisite to protect against this and other Pb-related health endpoints. This framework
21 is again considered in light of the current available evidence. This Pb NAAQS review, like any
22 NAAQS review, requires public health policy judgments. The public health policy judgments
23 for this review include the public health significance of a given magnitude of IQ loss in a small
24 subset of highly exposed children (i.e., those likely to experience air-related Pb exposures at the
25 level of the standard), as well as how to consider the nature and magnitude of the array of
26 uncertainties that are inherent in the evidence and in the application of this specific framework.

27 In also considering the quantitative risk estimates associated with the current standard,
28 based on the risk assessment conducted in the last review, staff observes that these estimates
29 indicate a level of risk that is roughly consistent with and generally supportive of conclusions
30 drawn from the evidence using the evidence-based air-related IQ loss framework. Staff
31 additionally recognizes the complexity of the modeling done as part of that assessment and the
32 substantial limitations and uncertainties in the resulting risk estimates.

33 Based on the above considerations, staff preliminarily concludes that the currently
34 available information supports a primary standard as protective as the current standard and that it
35 is appropriate to consider retaining the current standard without revision. In so doing, staff
36 additionally notes that the final decision on the adequacy of the current standard is largely a

1 public health policy judgment to be made by the Administrator, drawing upon the scientific
2 information as well as judgments about how to consider the range and magnitude of uncertainties
3 that are inherent in the scientific evidence and technical analyses. In this context, staff
4 recognizes that the uncertainties and limitations associated with the many aspects of the
5 relationship between air Pb concentrations and blood Pb levels and associated health effects are
6 amplified with consideration of increasingly lower air concentrations. In staff's view, based on
7 the current evidence there is appreciable uncertainty associated with drawing conclusions
8 regarding whether there would be reductions in risk to public health from alternative lower levels
9 as compared to the level of the current standard. Thus, staff concludes that the basis for any
10 consideration of alternative lower standard levels would reflect different public health policy
11 judgments as to the appropriate approach for weighing uncertainties in the evidence and for
12 providing requisite protection of public health with an adequate margin of safety. Accordingly,
13 and in light of the preliminary staff conclusion that it is appropriate to consider the current
14 standard to be adequate, this document does not identify potential alternative standards for
15 consideration in this review.

16 **Welfare Effects and Review of the Secondary Standard**

17 Consideration of the welfare effects evidence and screening-level risk information in the
18 last review (completed in 2008) led to the conclusion that there was a potential for adverse
19 welfare effects occurring under the then-current Pb standard (set in 1978), although there were
20 insufficient data to provide a quantitative basis for setting a secondary standard different from
21 the primary standard. Accordingly, the secondary standard was substantially revised to be
22 identical in all respects to the newly revised primary standard.

23 In assessing the currently available scientific evidence and the exposure/risk information
24 with regard to support for the adequacy of the protection afforded by the current standard, staff
25 observes the general consistency of the current evidence with that available in the last review,
26 including the substantial limitations in the current evidence that complicate conclusions
27 regarding the potential for Pb emissions under the current, much lower standard to contribute to
28 welfare effects. Thus, based on the staff analysis, framed by key policy-relevant questions for
29 the review, staff preliminarily concludes that consideration should be given to retaining the
30 current standard, without revision, and this document does not identify potential alternative
31 standards for consideration in this review.

32

1 INTRODUCTION

1.1 PURPOSE

The U.S. Environmental Protection Agency (EPA) is presently conducting a review of the primary (health-based) and secondary (welfare-based) national ambient air quality standards (NAAQS) for lead (Pb). The overall plan and schedule for this review were presented in the *Integrated Review Plan for the National Ambient Air Quality Standards for Lead* (IRP; USEPA, 2011a). The IRP also identified key policy-relevant issues to be addressed in this review and discussed the key documents that generally inform NAAQS reviews, including an Integrated Science Assessment (ISA), Risk and Exposure Assessments (REAs), and a Policy Assessment (PA). The PA presents a staff evaluation of the policy implications of the key scientific and technical information in the ISA and REAs for EPA's consideration.¹ The PA generally provides a transparent evaluation and staff conclusions regarding policy considerations related to reaching judgments about the adequacy of the current standards, and, if revision is considered, what revisions may be appropriate to consider.

When final, the PA is intended to help "bridge the gap" between the Agency's scientific assessments presented in the ISA and REAs, and the judgments required of the EPA Administrator in determining whether it is appropriate to retain or revise the NAAQS. In evaluating the adequacy of the current standard and whether it is appropriate to consider alternative standards, the PA focuses on information that is most pertinent to evaluating the basic elements of the NAAQS: indicator,² averaging time, form,³ and level. These elements, which together serve to define each standard, must be considered collectively in evaluating the health and welfare protection afforded by the Pb standards. The PA integrates and interprets the information from the ISA and REAs to frame policy options for consideration by the Administrator. In so doing, the PA recognizes that the selection of a specific approach to reaching final decisions on primary and secondary NAAQS will reflect the judgments of the Administrator.

¹ The terms "staff" and "we" throughout this document refer to staff in the EPA's Office of Air Quality Planning and Standards (OAQPS). In past NAAQS reviews, this document was referred to as the OAQPS Staff Paper.

² The "indicator" of a standard defines the chemical species or mixture that is to be measured in determining whether an area attains the standard. The indicator for the Pb NAAQS is lead in total suspended particles.

³ The "form" of a standard defines the air quality statistic that is to be compared to the level of the standard in determining whether an area attains the standard. For example, the form of the annual PM_{2.5} NAAQS is the 3-year average of the weighted annual mean PM_{2.5} concentrations, while the form of the 8-hour CO NAAQS is the second-highest 8-hour average in a year.

1 The development of the PA is also intended to facilitate advice to the Agency and
2 recommendations to the Administrator from an independent scientific review committee, the
3 Clean Air Scientific Advisory Committee (CASAC), as provided for in the Clean Air Act. As
4 discussed below in section 1.2.1, the CASAC is to advise not only on the Agency's assessment
5 of the relevant scientific information, but also on the adequacy of the existing standards, and to
6 make recommendations as to any revisions of the standards that may be appropriate. The EPA
7 facilitates CASAC advice and recommendations, as well as public input and comment, by
8 requesting CASAC review and public comment on one or more drafts of the PA.

9 The decision whether to prepare one or more drafts of the PA is influenced by
10 preliminary staff conclusions and associated CASAC advice and public comment, among other
11 factors. Typically, a second draft PA has been prepared in cases where the available information
12 calls into question the adequacy of the current standard and analyses of potential alternative
13 standards are developed taking into consideration CASAC advice and public comment. In such
14 cases, a second draft PA includes preliminary staff conclusions regarding potential alternative
15 standards and undergoes CASAC review and public comment prior to preparation of the final
16 PA. When such analyses are not undertaken, a second draft PA may not be warranted.

17 In this draft PA for this review of the Pb NAAQS, we consider the scientific and
18 technical information available in this review as assessed in the third draft *Integrated Science*
19 *Assessment for Lead* (henceforth referred to as the ISA [USEPA, 2012a]), prepared by EPA's
20 National Center for Environmental Assessment (NCEA), and the quantitative human exposure
21 and health risk and screening-level ecological risk assessments performed in the last review. As
22 discussed below in section 1.2.3, upon consideration of the evidence newly available in this
23 review with regard to risk and exposure assessment, staff concluded that new health and welfare
24 REAs were not warranted. Accordingly, the quantitative risk information considered in this PA
25 is drawn from the quantitative human exposure and health risk and screening-level ecological
26 risk assessments performed in the last review (the 2007 Health Risk Assessment Report or 2007
27 REA [USEPA, 2007a] and the 2006 screening-level Ecological Risk Assessment or 2006 REA
28 [ICF, 2006]), and is interpreted in the context of newly available evidence in this review.

29 The evaluation and preliminary staff conclusions presented in this draft PA for the Pb
30 NAAQS have been informed by comments and advice received from CASAC in their reviews of
31 the other draft Agency documents prepared thus far in this NAAQS review. Review and
32 comments from CASAC, and public comment, on this draft PA will inform the final evaluation
33 and staff conclusions in the final PA.

34 Beyond informing the EPA Administrator and facilitating the advice and
35 recommendations of CASAC and the public, the PA is also intended to be a useful reference to
36 all parties interested in the Pb NAAQS review. In these roles, it is intended to serve as a single

1 source of the most policy-relevant information that informs the Agency’s review of the lead
2 NAAQS, and it is written to be understandable to a broad audience.

3 **1.2 BACKGROUND**

4 **1.2.1 Legislative Requirements**

5 Two sections of the Clean Air Act (CAA or the Act) govern the establishment and
6 revision of the NAAQS. Section 108 (42 U.S.C. section 7408) directs the Administrator to
7 identify and list certain air pollutants and then to issue air quality criteria for those pollutants.
8 The Administrator is to list those air pollutants that in her “judgment, cause or contribute to air
9 pollution which may reasonably be anticipated to endanger public health or welfare;” “the
10 presence of which in the ambient air results from numerous or diverse mobile or stationary
11 sources;” and “for which . . . [the Administrator] plans to issue air quality criteria...” Air quality
12 criteria are intended to “accurately reflect the latest scientific knowledge useful in indicating the
13 kind and extent of all identifiable effects on public health or welfare which may be expected
14 from the presence of [a] pollutant in the ambient air . . .” 42 U.S.C. § 7408(b). Section 109 (42
15 U.S.C. 7409) directs the Administrator to propose and promulgate “primary” and “secondary”
16 NAAQS for pollutants for which air quality criteria are issued. Section 109(b)(1) defines a
17 primary standard as one “the attainment and maintenance of which in the judgment of the
18 Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to
19 protect the public health.”⁴ A secondary standard, as defined in section 109(b)(2), must “specify
20 a level of air quality the attainment and maintenance of which, in the judgment of the
21 Administrator, based on such criteria, is requisite to protect the public welfare from any known
22 or anticipated adverse effects associated with the presence of [the] pollutant in the ambient air.”⁵

23 The requirement that primary standards provide an adequate margin of safety was
24 intended to address uncertainties associated with inconclusive scientific and technical
25 information available at the time of standard setting. It was also intended to provide a reasonable
26 degree of protection against hazards that research has not yet identified. See *Lead Industries*
27 *Association v. EPA*, 647 F.2d 1130, 1154 (D.C. Cir 1980), *cert. denied*, 449 U.S. 1042 (1980);
28 *American Petroleum Institute v. Costle*, 665 F.2d 1176, 1186 (D.C. Cir. 1981), *cert. denied*, 455

⁴ The legislative history of section 109 indicates that a primary standard is to be set at “the maximum permissible ambient air level . . . which will protect the health of any [sensitive] group of the population,” and that for this purpose “reference should be made to a representative sample of persons comprising the sensitive group rather than to a single person in such a group” S. Rep. No. 91-1196, 91st Cong., 2d Sess. 10 (1970).

⁵ Welfare effects as defined in section 302(h) (42 U.S.C. § 7602(h)) include, but are not limited to, “effects on soils, water, crops, vegetation, man-made materials, animals, wildlife, weather, visibility and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being.”

1 U.S. 1034 (1982); *American Farm Bureau Federation v. EPA*, 559 F. 3d 512, 533 (D.C. Cir.
2 2009); *Association of Battery Recyclers v. EPA*, 604 F. 3d 613, 617-18 (D.C. Cir. 2010). Both
3 kinds of uncertainties are components of the risk associated with pollution at levels below those
4 at which human health effects can be said to occur with reasonable scientific certainty. Thus, in
5 selecting primary standards that provide an adequate margin of safety, the Administrator is
6 seeking not only to prevent pollution levels that have been demonstrated to be harmful but also
7 to prevent lower pollutant levels that may pose an unacceptable risk of harm, even if the risk is
8 not precisely identified as to nature or degree. The CAA does not require the Administrator to
9 establish a primary NAAQS at a zero-risk level or at background concentration levels, see *Lead*
10 *Industries v. EPA*, 647 F.2d at 1156 n.51, but rather at a level that reduces risk sufficiently so as
11 to protect public health with an adequate margin of safety.

12 In addressing the requirement for an adequate margin of safety, the EPA considers such
13 factors as the nature and severity of the health effects involved, the size of sensitive population(s)
14 at risk, and the kind and degree of the uncertainties that must be addressed. The selection of any
15 particular approach to providing an adequate margin of safety is a policy choice left specifically
16 to the Administrator’s judgment. See *Lead Industries Association v. EPA*, 647 F.2d at 1161-62;
17 *Whitman v. American Trucking Associations*, 531 U.S. 457, 495 (2001).

18 In setting primary and secondary standards that are “requisite” to protect public health
19 and welfare, respectively, as provided in section 109(b), EPA’s task is to establish standards that
20 are neither more nor less stringent than necessary for these purposes. In so doing, the EPA may
21 not consider the costs of implementing the standards. See generally, *Whitman v. American*
22 *Trucking Associations*, 531 U.S. 457, 465-472, 475-76 (2001). Likewise, “[a]ttainability and
23 technological feasibility are not relevant considerations in the promulgation of national ambient
24 air quality standards.” *American Petroleum Institute v. Costle*, 665 F. 2d at 1185.

25 Section 109(d)(1) requires that “not later than December 31, 1980, and at 5-year
26 intervals thereafter, the Administrator shall complete a thorough review of the criteria
27 published under section 108 and the national ambient air quality standards . . . and shall make
28 such revisions in such criteria and standards and promulgate such new standards as may be
29 appropriate” Section 109(d)(2) requires that an independent scientific review committee
30 “shall complete a review of the criteria . . . and the national primary and secondary ambient air
31 quality standards . . . and shall recommend to the Administrator any new . . . standards and
32 revisions of existing criteria and standards as may be appropriate” Since the early 1980’s,

1 this independent review function has been performed by the Clean Air Scientific Advisory
2 Committee (CASAC).⁶

3 **1.2.2 History of Lead NAAQS Reviews**

4 Unlike pollutants such as particulate matter and carbon monoxide, air quality criteria had
5 not been issued for Pb as of the enactment of the Clean Air Act of 1970, which first set forth the
6 requirement to set national ambient air quality standards based on air quality criteria. In the
7 years just after enactment of the CAA, the EPA did not intend to issue air quality criteria for Pb,
8 and accordingly had not listed Pb under Section 108 of the Act. The EPA had determined to
9 control Pb air pollution through regulations to phase-out use of Pb additives in gasoline and the
10 EPA viewed those controls, and possibly additional federal controls, as the best approach to
11 controlling Pb emissions (See 41 FR 14921 (April 8, 1976). However, the decision not to list Pb
12 under Section 108 was challenged by environmental and public health groups and the U.S.
13 District Court for the Southern District of New York concluded that the EPA was required to list
14 Pb under Section 108. (Natural Resources Defense Council v. EPA, 411 F. Supp. 864 21 [S.D.
15 N.Y. 1976], *aff'd*, 545 F.2d 320 [2d Cir. 1978]).

16 Accordingly, on April 8, 1976, the EPA published a notice in the *Federal Register* that
17 Pb had been listed under Section 108 as a criteria pollutant (41 FR 14921) and on October 5,
18 1978, the EPA promulgated primary and secondary NAAQS for Pb under Section 109 of the Act
19 (43 FR 46246). Both primary and secondary standards were set at a level of 1.5 micrograms per
20 cubic meter ($\mu\text{g}/\text{m}^3$), measured as Pb in total suspended particles (Pb-TSP), not to be exceeded
21 by the maximum arithmetic mean concentration averaged over a calendar quarter. These
22 standards were based on the 1977 Air Quality Criteria for Lead (USEPA, 1977).

23 The first review of the Pb standards was initiated in the mid-1980s. The scientific
24 assessment for that review is described in the 1986 Air Quality Criteria for Lead (USEPA,
25 1986a), the associated Addendum (USEPA, 1986b) and the 1990 Supplement (USEPA, 1990a).
26 As part of the review, the Agency designed and performed human exposure and health risk
27 analyses (USEPA, 1989), the results of which were presented in a 1990 Staff Paper (USEPA,
28 1990b). Based on the scientific assessment and the human exposure and health risk analyses, the
29 1990 Staff Paper presented recommendations for consideration by the Administrator (USEPA,
30 1990b). After consideration of the documents developed during the review and the significantly
31 changed circumstances since Pb was listed in 1976, the Agency did not propose any revisions to
32 the 1978 Pb NAAQS. In a parallel effort, the Agency developed the broad, multi-program,
33 multimedia, integrated *U.S. Strategy for Reducing Lead Exposure* (USEPA, 1991). As part of

⁶ Lists of CASAC members and of members of the CASAC Pb Review Panel are available at:
<http://yosemite.epa.gov/sab/sabproduct.nsf/WebCASAC/CommitteesandMembership?OpenDocument>.

1 implementing this strategy, the Agency focused efforts primarily on regulatory and remedial
2 clean-up actions aimed at reducing Pb exposures from a variety of nonair sources judged to pose
3 more extensive public health risks to U.S. populations, as well as on actions to reduce Pb
4 emissions to air, such as bringing more areas into compliance with the existing Pb NAAQS
5 (USEPA, 1991).

6 The most recent review of the Pb air quality criteria and standards was initiated in
7 November 2004 (69 FR 64926) and the Agency's plans for preparation of the Air Quality
8 Criteria Document and conduct of the NAAQS review were presented in documents completed
9 in 2005 and early 2006 (USEPA, 2005; USEPA 2006a).⁷ The schedule for completion of the
10 review was governed by a judicial order in *Missouri Coalition for the Environment v. EPA* (No.
11 4:04CV00660 ERW, Sept. 14, 2005; and amended on April 29, 2008 and July 1, 2008), which
12 specified a schedule for the review of duration substantially shorter than five years.

13 The scientific assessment for the review is described in the 2006 *Air Quality Criteria for*
14 *Lead* (USEPA, 2006b; henceforth referred to as the 2006 CD), multiple drafts of which received
15 review by CASAC and the public. The EPA also conducted human exposure and health risk
16 assessments and a pilot ecological risk assessment for the review, after consultation with
17 CASAC and receiving public comment on a draft analysis plan (USEPA, 2006c). Drafts of these
18 quantitative assessments were reviewed by CASAC and the public. The pilot ecological risk
19 assessment was released in December 2006 (ICF, 2006) and the final health risk assessment
20 report was released in November 2007 (USEPA, 2007a). The policy assessment based on both
21 of these assessments, air quality analyses and key evidence from the AQCD was presented in the
22 Staff Paper (USEPA, 2007b), a draft of which also received CASAC and public review. The
23 final Staff Paper presented OAQPS staff's evaluation of the public health and welfare policy
24 implications of the key studies and scientific information contained in the Criteria Document and
25 presented and interpreted results from the quantitative risk/exposure analyses conducted for this
26 review. Based on this evaluation, the Staff Paper presented OAQPS staff recommendations that
27 the Administrator give consideration to substantially revising the primary and secondary
28 standards to a range of levels at or below 0.2 µg/m³.

29 Immediately subsequent to completion of the Staff Paper, the EPA issued an advance
30 notice of proposed rulemaking (ANPR) that was signed by the Administrator on December 5,
31 2007 (72 FR 71488).⁸ CASAC provided advice and recommendations to the Administrator with
32 regard to the Pb NAAQS based on its review of the ANPR and the previously released final Staff

⁷ In the current review, these two documents have been combined in the IRP.

⁸ The ANPR was one of the features of the revised NAAQS review process that EPA instituted in 2006. In 2009 (Jackson, 2009), this component of the process was replaced by reinstatement of policy assessment prepared by OAQPS staff (previously termed the OAQPS Staff Paper).

1 Paper and risk assessment reports. The proposed decision on revisions to the Pb NAAQS was
2 signed on May 1, 2008 and published in the Federal Register on May 20, 2008 (73 FR 29184).
3 Members of the public provided both written and, at two public hearings, oral comments and the
4 CASAC Pb Panel also provided advice and recommendations to the Administrator based on its
5 review of the proposal notice. The final decision on revisions to the Pb NAAQS was signed on
6 October 15, 2008 and published in the Federal Register on November 12, 2008 (73 FR 66964).

7 The November 2008 notice described EPA's decision to revise the primary and
8 secondary NAAQS for Pb, as discussed more fully in section 4.1.1 below. In consideration of
9 the much-expanded health effects evidence on neurocognitive effects of Pb in children, the EPA
10 substantially revised the primary standard from a level of 1.5 $\mu\text{g}/\text{m}^3$ to a level of 0.15 $\mu\text{g}/\text{m}^3$.
11 The averaging time was revised to a rolling three-month period with a maximum (not-to-be-
12 exceeded) form, evaluated over a three-year period. The indicator of Pb-TSP was retained,
13 reflecting the evidence that Pb particles of all sizes pose health risks. The secondary standard
14 was revised to be identical in all respects to the revised primary standards.⁹ Revisions to the
15 NAAQS were accompanied by revisions to the data handling procedures, the treatment of
16 exceptional events and the ambient air monitoring and reporting requirements, as well as
17 emissions inventory reporting requirements.¹⁰ One aspect of the new data handling requirements
18 is the allowance for the use of Pb-PM₁₀ monitoring for Pb NAAQS attainment purposes in
19 certain limited circumstances at non-source-oriented sites. Subsequent to the 2008 rulemaking,
20 additional revisions were made to the monitoring network requirements as described in chapter 2
21 below.

22 **1.2.3 Current Lead NAAQS Review**

23 On February 26, 2010, the EPA formally initiated its current review of the air quality
24 criteria and standards for Pb, requesting the submission of recent scientific information on
25 specified topics (75 FR 8934). Soon after this, the EPA held a science policy workshop to
26 discuss the policy-relevant science, which informed identification of key policy issues and
27 questions to frame the review of the Pb NAAQS (75 FR 20843). Drawing from the workshop
28 discussions, the EPA developed the draft IRP (USEPA, 2011c). The draft IRP was made
29 available in late March 2011 for consultation with the CASAC Pb Review Panel and for public
30 comment (76 FR 20347). This document was discussed by the Panel via a publicly accessible

⁹ The current NAAQS for Pb are specified at 40 CFR 50.16.

¹⁰ The current federal regulatory measurement methods for Pb are specified in 40 CFR 50, Appendix G and 40 CFR part 53. Consideration of ambient air measurements with regard to judging attainment of the standards is specified in 40 CFR 50, Appendix R. The Pb monitoring network requirements are specified in 40 CFR 58, Appendix D, section 4.5. Guidance on the approach for implementation of the new standards was described in the Federal Register notices for the proposed and final rules (73 FR 29184; 73 FR 66964).

1 teleconference consultation on May 5, 2011 (76 FR 21346; Frey, 2011a). The final IRP,
2 developed in consideration of CASAC advice and public comment, was released in November,
3 2011 (USEPA, 2011a; 76 FR 76972).

4 In developing the ISA for this review, the EPA held a workshop in December 2010 to
5 discuss with invited scientific experts preliminary draft materials and released the first external
6 review draft of the document for CASAC review and public comment on May 6, 2011 (USEPA,
7 2011d; 76 FR 26284; 76 FR 36120). The CASAC Pb Review Panel met at a public meeting on
8 July 20, 2011 to review the draft ISA (76 FR 36120). The CASAC provided comments in a
9 December 9, 2011 letter to the EPA Administrator (Frey and Samet, 2011). The second external
10 review draft ISA was released for CASAC review and public comment in February 2012
11 (USEPA, 2012b, 77 FR 5247) and was the subject of a public meeting on April 10-11, 2012 (77
12 FR 14783). The CASAC provided comments in a July 20, 2012 letter (Samet and Frey, 2012).
13 The third external review draft was released for CASAC review and public comment in
14 November 2012 (USEPA, 2012a, 77 FR 70776). That document, together with this draft Policy
15 Assessment, will be the subject of a public meeting on February 5-6, 2013 (78 FR 938). The
16 final ISA is targeted for release in late spring 2013.

17 As described in the IRP, the EPA developed and released for CASAC review and public
18 comment in June 2011 an REA Planning Document (USEPA, 2011b; 76 FR 58509). This
19 document presented a critical evaluation of the information related to Pb human and ecological
20 exposure and risk (e.g., data, modeling approaches) newly available in this review, with a focus
21 on consideration of the extent to which new or substantially revised REAs for health and
22 ecological risk are warranted by the newly available evidence. Evaluation of the newly available
23 information with regard to designing and implementing health and ecological REAs for this
24 review led us to conclude that the currently available information did not provide a basis for
25 developing new quantitative risk and exposure assessments that would have substantially
26 improved utility for informing the Agency's consideration of health and welfare effects and
27 evaluation of the adequacy of the current primary and secondary standards, respectively (REA
28 Planning Document, sections 2.3 and 3.3, respectively). The CASAC Pb Panel provided
29 consultative advice on that document and its conclusions at a public meeting on July 21, 2011
30 (76 FR 36120; Frey, 2011b). Based on their consideration of the REA Planning Document
31 analysis, the CASAC Pb Review Panel generally concurred with the conclusion that a new REA
32 was not warranted in this review (Frey, 2011b). In consideration of the conclusions reached in
33 the REA Planning Document and CASAC's consultative advice, the EPA has not developed
34 REAs for health and ecological risk for this review. Accordingly, this Policy Assessment
35 considers the risk assessment findings from the last review with regard to any appropriate further
36 interpretation in light of the evidence newly available in this review.

1 This draft Policy Assessment is being released for public comment and review by
2 CASAC. Advice from CASAC and public comments received on this draft will be considered in
3 preparing the final PA.

4 **1.3 SCOPE OF CURRENT REVIEW: FATE AND MULTIMEDIA PATHWAYS OF** 5 **AMBIENT AIR LEAD**

6 The multimedia and persistent nature of Pb contributes complexities to the review of the
7 Pb NAAQS unlike issues addressed in other NAAQS reviews.¹¹ As described in section 1.1,
8 NAAQS are established to protect public health with an adequate margin of safety, and public
9 welfare from known or anticipated adverse effects, from air pollutants (substances emitted to
10 ambient air). Since Pb distributes from air to other media and is persistent, our review of the
11 NAAQS for Pb considers the protection provided against such effects associated both with
12 exposures to Pb in ambient air and with exposures to Pb that makes its way into other media
13 from ambient air. Additionally, in assessing the adequacy of protection afforded by the current
14 NAAQS, we are mindful of the history of the greater and more widespread atmospheric
15 emissions that occurred in previous years (e.g., under 1978 NAAQS, and prior to establishment
16 of any Pb NAAQS) and that contributed to the Pb that exists in human populations and
17 ecosystems today. Likewise, we also recognize the role of other, nonair sources of Pb now and
18 in the past that also contribute to the Pb that exists in human populations and ecosystems today.
19 As in the last Pb NAAQS review, this backdrop of environmental Pb exposure, and its impact on
20 the populations and ecosystems which may be the subjects of the currently available scientific
21 evidence, complicates our consideration of the health and welfare protection afforded by the
22 current NAAQS. In the first section below, we summarize the environmental pathways of
23 human and ecosystem exposures to Pb emitted to ambient air and associated complexities. The
24 subsequent section briefly discusses the role of historically emitted Pb in our consideration of the
25 adequacy of the current NAAQS for Pb.

26 **1.3.1 Environmental Distribution and Exposure Pathways**

27 Lead emitted to ambient air is transported through the air and is also distributed to other
28 media through the process of deposition, which may occur in dry conditions or in association
29 with precipitation, as summarized further in section 2.3 below (ISA, section 3.7.2). Once
30 deposited, the fate of Pb is influenced by the type of surface onto which the particles deposit and
31 by the type and activity level of transport processes in that location. Precipitation and other

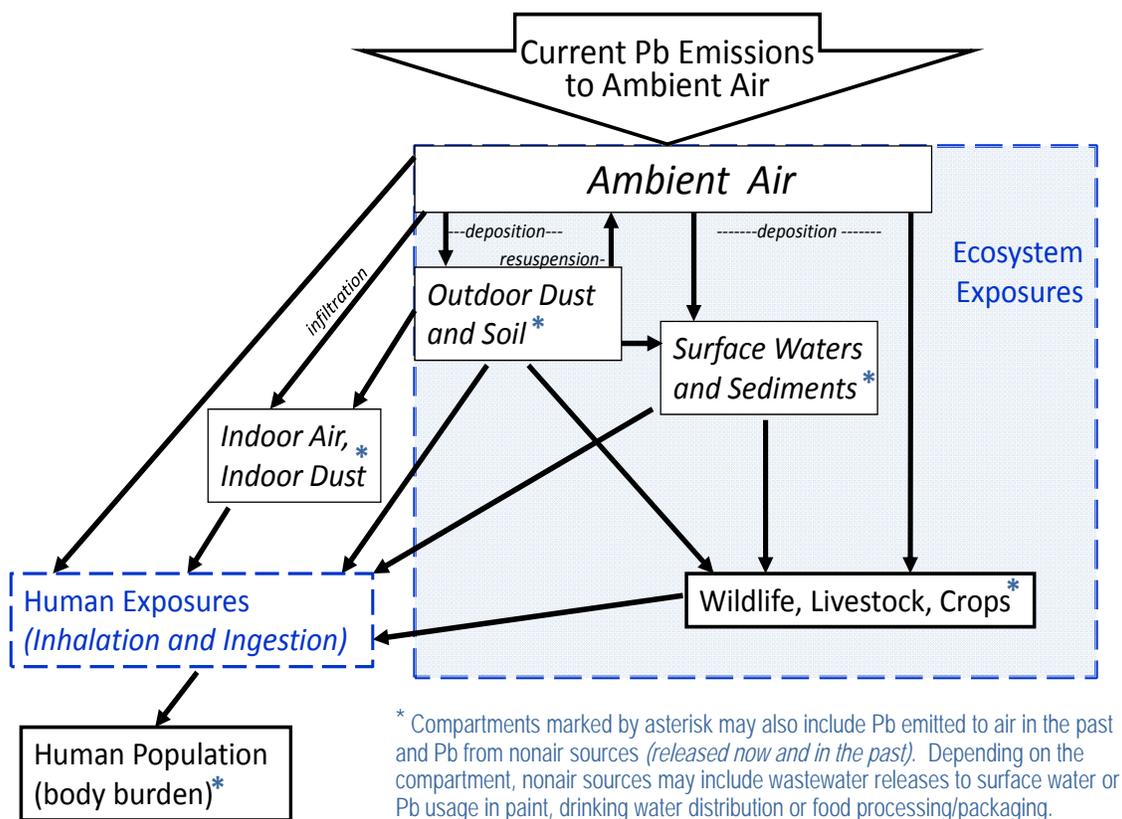
¹¹ Some aspects of the review of the secondary standard for oxides of nitrogen and sulfur, which involved consideration of pollutant transport and fate in nonair media with a focus on impacts to aquatic ecosystems, have some similarity to considerations for Pb, while the Pb review also differs in other important aspects.

1 natural, as well as human-influenced, processes contribute to the fate of such particles, which
2 affects the likelihood of subsequent human and ecological exposures, e.g., tracking into nearby
3 houses or transport with surface runoff into nearby water bodies (ISA, sections 3.3 and 4.1). For
4 example, Pb particles deposited onto impervious surfaces, such as roadway, sidewalk or other
5 urban surfaces, may be more available for human contact while they remain on such surfaces or
6 are transferred to other human environments, such as on clothing or through resuspension and
7 infiltration (ISA, section 4.1.1.1). Deposited Pb can also be transported (by direct deposition or
8 stormwater runoff) to water bodies and into associated sediments, which may provide a storage
9 function for Pb in aquatic ecosystems (ISA, sections 3.3.2 and 7.2.1). Lead deposited in
10 terrestrial ecosystems can also be incorporated into soil matrices (ISA, sections 3.3.3 and 7.2.1).

11 Figure 1-1 illustrates, in summary fashion, the pathways by which Pb emitted into
12 ambient air can be distributed in the environment and contribute to human and ecosystem
13 exposures. As shown in this figure, the multimedia distribution of Pb emitted into ambient air
14 (air-related Pb) contributes to multiple air-related pathways of human and ecosystem exposure
15 (ISA, sections 4.1.1 and 4.7.1).¹² As illustrated in Figure 1-1, air-related pathways may involve
16 media other than air. Additionally, as recognized by the figure and discussed more completely in
17 the subsections below, Pb that has not passed through ambient air (nonair Pb) may complicate
18 our consideration of ambient air Pb exposures. Further, the persistence of Pb poses an additional
19 complication, also discussed below, with regard to consideration of exposures associated with
20 current Pb emissions.

21

¹² The exposure assessment for children performed for the review completed in 2008 employed available data and methods to develop estimates intended to inform a characterization of these pathways, as described in the rulemaking notices for that review (73 FR 29184; 73 FR 66964) and the final Risk Assessment Report (USEPA, 2007).



Note: Arrows indicate general pathways by which Pb distributes in environment and human populations. Individual pathway significance varies with location- and receptor-specific factors.

Figure 1-1. Pathways of human and ecosystem exposure to lead from ambient air.

1.3.1.1 Human Exposure Pathways

Air-related Pb exposure pathways for humans include inhalation of ambient air or ingestion of food, water or other materials, including dust and soil, that have been contaminated through a pathway involving Pb deposition from ambient air (ISA, section 4.1.1.1). Ambient air inhalation pathways include both inhalation of air outdoors and inhalation of ambient air that has infiltrated into indoor environments. The air-related ingestion pathways occur as a result of Pb passing through the ambient air, being distributed to other environmental media and contributing to human exposures via contact with and ingestion of indoor and outdoor dusts, outdoor soil, food and drinking water.

Lead currently occurring in nonair media may also derive from sources other than ambient air (nonair Pb sources), as summarized further in section 2.3 below (ISA, section 4.7.1). For example, Pb in dust inside some houses or outdoors in some urban areas may derive from the

1 common past usage of leaded paint, while Pb in drinking water may derive from the use of
2 leaded pipe or solder in drinking water distribution systems (ISA, section 4.1.3.3). We also
3 recognize the history of much greater air emissions of Pb in the past, such as that associated with
4 leaded gasoline usage and higher industrial emissions (as summarized in section 2.1.1 below)
5 which have also contributed to Pb currently occurring in other (nonair) media.

6 The relative importance of different pathways of human exposure to Pb, as well as the
7 relative contributions from Pb resulting from recent and historic air emissions and from nonair
8 sources, vary across the U.S. population as a result of both extrinsic factors, such as a home's
9 proximity to industrial Pb sources or its history of leaded paint usage, and intrinsic factors, such
10 as a person's age and nutritional status (ISA, sections 6.1, 6.2, 6.2.1, 6.2.5 and 6.2.6). For
11 example, a predominant Pb exposure pathway for very young children is the incidental ingestion
12 of indoor dust by hand-to-mouth activity (ISA, section 4.1.1.1). For adults, diet may be the
13 primary Pb exposure pathway (2006 CD, section 3.4). Similarly, the relative importance of air-
14 related and nonair-related Pb also varies with the relative magnitudes of exposure by those
15 pathways, which may vary with different circumstances. For example, relative contributions to a
16 child's total Pb exposure from air-related exposure pathways compared to other (nonair) Pb
17 exposures depend on many factors, including ambient air concentrations and air deposition in the
18 area where the child resides (as well as in the area from which the child's food derives), as well
19 as access to other sources of Pb exposure such as Pb paint, tap water affected by plumbing
20 containing Pb, and lead-tainted products. Studies indicate that in the absence of paint-related
21 exposures, Pb from other sources such as nearby stationary sources of Pb emissions may
22 dominate a child's Pb exposures (ISA, sections 4.1 and 4.1.3.2; 2006 CD, section 3.2.3). In
23 other cases, such as children living in older housing with peeling paint or where renovations have
24 occurred, the dominant source of Pb exposure may be dust from lead paint used in the house in
25 the past. Depending on Pb levels in a home's tap water, drinking water can sometimes be a
26 significant source. Lead exposure may also be the result of a mixture of contributions from
27 multiple sources, with no one source dominating. Our understanding of the relative contribution
28 of air-related Pb to ingestion exposure pathways is limited by the paucity of studies that parse
29 ingestion exposure pathways with regard to air-related and nonair Pb. Our understanding of the
30 relative contribution of air-related Pb associated with historical emissions and that from recent
31 emissions is similarly limited.

32 **1.3.1.2 Ecosystem Exposure Pathways**

33 The distribution of Pb from ambient air to other environmental media also influences the
34 exposure pathways in terrestrial and aquatic ecosystems. Exposure of terrestrial animals and
35 vegetation to air-related Pb can occur by contact with ambient air or by contact with soil, water

1 or food items that have been contaminated by Pb from ambient air (ISA, section 7.2). Transport
2 of Pb into aquatic systems similarly provides for exposure of biota in those systems, and
3 exposures may vary among systems as a result of differences in sources and levels of
4 contamination, as well as characteristics of the systems themselves. In addition to Pb contributed
5 by current atmospheric deposition, Pb may occur in aquatic systems as a result of nonair sources
6 such as industrial discharges or mine-related drainage, of historical air Pb emissions (e.g.,
7 contributing to deposition to a water body or via runoff from soils near historical air sources) or
8 combinations of different types of sources (e.g., resuspension of sediments contaminated by
9 urban runoff and surface water discharges).

10 The persistence of Pb contributes an important temporal aspect to lead's environmental
11 pathways, and the time (or lag) associated with realization of the impact of air Pb concentrations
12 on concentrations in other media can vary with the media (e.g., ISA, section 7.2.2). For example,
13 human exposure pathways most directly involving Pb in ambient air and exchanges of ambient
14 air with indoor air can respond more quickly while pathways involving exposure to Pb deposited
15 from ambient air into the environment (e.g., diet) generally respond more slowly. An additional
16 influence on the response time for nonair media is the environmental presence of Pb associated
17 with past, generally higher, air concentrations. For example, after a reduction in air Pb
18 concentrations, the time needed for sediment or surface soil concentrations to indicate a response
19 to reduced air Pb concentrations might be expected to be longer in areas of more substantial past
20 contamination than in areas with lesser past contamination. Thus, considering the Pb
21 concentrations occurring in nonair media as a result of air quality conditions that meet the
22 current NAAQS is a complexity of this review, as it also was, although to a lesser degree, with
23 regard to the prior standard in the last review.

24 **1.3.2 Considerations Related to Historically Emitted Lead**

25 In reviews of NAAQS, the overarching consideration of each review is first focused on
26 the general question as to whether the currently available information supports or calls into
27 question the adequacy of the current standard(s). In addressing that consideration for the
28 NAAQS for Pb, our focus is on Pb emitted to ambient air under conditions meeting the current
29 standard and its potential to cause health or welfare effects as a result of exposures to Pb in air or
30 in other media. We frame the focus in this way, which differs from that for NAAQS reviews
31 involving other pollutants, in consideration of the persistence of Pb in the environment over time,
32 a characteristic which does not affect reviews of other NAAQS. In considering the case for Pb,
33 however, we recognize that, because of its persistence, both recent and past Pb emissions to
34 ambient air contribute to current Pb exposures (via multiple exposure pathways, as summarized
35 in section 1.4.1 above). And we recognize that past Pb emissions in many situations were well in

1 excess of the current Pb standard. Yet our task in this and every NAAQS review is focused on
2 assessing the adequacy of the current standard.

3 Lead emissions, and air concentrations, were higher in the past, and most substantially so
4 during the time of leaded gasoline usage, as summarized in sections 2.1 and 2.2 below. Nonair
5 sources of Pb to human exposure and the environment, an additional complicating feature of Pb
6 NAAQS reviews, were also much higher in the past. These air and nonair sources contributed
7 to historical human and environmental exposures. Because of the persistence of Pb, historical
8 exposures associated both with air-related and nonair-related sources, have contributed Pb that is
9 now stored within older humans and ecosystems. For example, concentrations of Pb in the bone
10 and blood of older members of the U.S. population, who lived during the time of widespread air
11 emissions associated with leaded gasoline usage (as well as higher industrial emissions) under
12 the previous Pb NAAQS or prior to establishment of any Pb NAAQS, are greater than what
13 would result from air quality conditions allowed by the current, more restrictive NAAQS.
14 Epidemiological studies of these populations, in which this exposure history is represented by
15 current bone or blood Pb concentrations, contribute to the overall evidence base regarding lead-
16 related health effects (as discussed in chapter 3 below). Such studies of these historically
17 exposed populations, however, are generally less informative in judging the adequacy of the
18 current primary standard (as discussed in chapter 3 below). This is in contrast to epidemiological
19 studies of very young populations with much shorter and more recent exposure histories (also
20 discussed in chapter 3).

21 The current distribution of Pb in U.S. ecosystems also reflects the widespread and greater
22 air emissions that occurred in the past, under the prior Pb NAAQS and prior to establishment of
23 any Pb NAAQS.¹³ We lack information on whether adverse effects could be anticipated from
24 the Pb in terrestrial and aquatic ecosystems that results from air quality conditions allowed by the
25 current, more restrictive NAAQS. As is discussed in section 2.3 below, media in ecosystems
26 across the U.S. are still recovering from the past period of higher atmospheric emissions, and
27 their responses (e.g., in terms of temporal change in media concentrations) differ with the extent
28 of contamination in individual systems as well as with ecosystem-specific characteristics. Thus,
29 the time required for these ecosystem media to “equilibrate” or come to a “steady-state” that
30 reflects the influence of the current NAAQS (established in 2008) will also vary, and the
31 resulting “steady-state” media concentrations for the range of U.S. ecosystems are unknown.
32 Evidence of effects pertaining to the concentrations associated with the past, higher emissions (as
33 well as from nonair sources), while informative to our understanding of welfare effects
34 associated with environmental Pb generally, does not directly inform our consideration of

¹³ The current distribution of Pb in U.S. ecosystems also reflects historical nonair releases.

1 welfare effects that might be anticipated under the current secondary standard and thus may be
2 generally less informative in judging the adequacy of the current standard.

3 **1.4 GENERAL ORGANIZATION OF THE DOCUMENT**

4 Following this introductory chapter, this document is organized into three main parts: the
5 characterization of ambient Pb; lead-related health effects and the primary Pb NAAQS; and lead-
6 related welfare effects and the secondary Pb NAAQS. The characterization of ambient Pb is
7 presented in Chapter 2 and includes information on Pb properties in ambient air, current Pb
8 emissions and air quality patterns, historic trends, and background levels. Chapter 2 also
9 describes the Pb NAAQS surveillance and other Pb monitoring networks. In recognition of the
10 multimedia nature of Pb and the distribution into other media of Pb emitted into the air, Chapter
11 2 also includes information on Pb in media other than air including outdoor dust, soil, surface
12 water and sediment. This chapter provides a frame of reference for exposure and risk-related
13 considerations and subsequent discussion of the Pb NAAQS.

14 Chapters 3 and 4 comprise the second main part of this document, dealing with human
15 health and the primary standard. These chapters are organized around a series of questions,
16 drawn from the IRP, that address the key policy-relevant issues related to the primary standard.
17 Chapter 3 presents an overview of key policy-relevant health effects evidence and major health-
18 related conclusions from the ISA; an examination of issues related to the quantitative assessment
19 of health risks; key results from quantitative assessments together with a discussion of
20 uncertainty and variability in the results; and discussion of the public health implications of the
21 evidence and exposure/risk information. In this draft PA, chapter 4 includes staff's preliminary
22 consideration of the scientific evidence and exposure/risk information related to the primary
23 standard and associated preliminary conclusions on the adequacy of the current primary
24 standard.

25 Chapters 5 and 6 comprise the third main part of this document. These chapters are
26 similarly organized around a series of questions, drawn from the IRP, that address the key
27 policy-relevant issues related to the secondary standard. Chapter 5 presents an overview of
28 welfare effects evidence related to these key policy-relevant issues and major welfare effects
29 related conclusions from the ISA; an examination of issues related to the screening level
30 ecological risk assessment; and key results from the risk assessment together with a discussion of
31 uncertainty and variability in the results; and discussion of the public welfare implications of the
32 quantitative assessment with regard to the current standard. The final chapter, chapter 6,
33 includes staff's preliminary conclusions related to the adequacy of the current secondary
34 standard.

1 1.5 REFERENCES

- 2 Frey, H.C. (2011a) Letter from Dr. H. Christopher Frey, Chair, Clean Air Scientific Advisory Committee Lead
3 Review Panel, to Administrator Lisa P. Jackson. Re: Consultation on EPA's Draft Integrated Review Plan
4 for the National Ambient Air Quality Standards for Lead. May 25, 2011.
- 5 Frey, H.C. (2011b) Letter from Dr. H. Christopher Frey, Chair, Clean Air Scientific Advisory Committee Lead
6 Review Panel, to Administrator Lisa P. Jackson. Re: Consultation on EPA's Review of the National
7 Ambient Air Quality Standards for Lead: Risk and Exposure Assessment Planning Document. October 14,
8 2011.
- 9 Frey, H.C. and Samet, J.M. (2011) Letter from Drs. H. Christopher Frey, Chair, Clean Air Scientific Advisory
10 Committee Lead Review Panel, and Jonathan M. Samet, Chair, Clean Air Scientific Advisory Committee,
11 to Administrator Lisa P. Jackson. Re: CASAC Review of the EPA's Integrated Science Assessment for
12 Lead (First External Review Draft – May 2011). December 9, 2011.
- 13 ICF International. (2006) Lead Human Exposure and Health Risk Assessments and Ecological Risk Assessment for
14 Selected Areas. Pilot Phase. Draft Technical Report. Prepared for the U.S. EPA's Office of Air Quality
15 Planning and Standards, Research Triangle Park, NC. December.
- 16 Jackson, L. (2009) Memorandum from Administrator Lisa Jackson, Subject: Development of regulations and
17 policies. September 11, 2009. Available at:
18 <http://www.epa.gov/ttn/naaqs/pdfs/NAAQSReviewProcessMemo52109.pdf>
- 19 Samet, J.M. and Frey, H.C. (2012) Letter from Drs. Jonathan M. Samet, Chair, Clean Air Scientific Advisory
20 Committee and H. Christopher Frey, Chair, Clean Air Scientific Advisory Committee Lead Review Panel,
21 to Administrator Lisa P. Jackson. Re: CASAC Review of the EPA's Integrated Science Assessment for
22 Lead (Second External Review Draft – February 2012). July 20, 2012.
- 23 U.S. Environmental Protection Agency. (1977) Air quality criteria for lead. Research Triangle Park, NC: Health
24 Effects Research Laboratory, Criteria and Special Studies Office; EPA report no. EPA-600/8-77-017.
25 Available from: NTIS, Springfield, VA; PB-280411.
- 26 U.S. Environmental Protection Agency. (1986a) Air quality criteria for lead. Research Triangle Park, NC: Office of
27 Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no.
28 EPA-600/8-83/028aF-dF. 4v. Available from: NTIS, Springfield, VA; PB87-142378.
- 29 U.S. Environmental Protection Agency. (1986b) Lead effects on cardiovascular function, early development, and
30 stature: an addendum to U.S. EPA Air Quality Criteria for Lead (1986). In: Air quality criteria for lead, v.
31 1. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria
32 and Assessment Office; pp. A1-A67; EPA report no. EPA-600/8-83/028aF. Available from: NTIS,
33 Springfield, VA; PB87-142378.
- 34 U.S. Environmental Protection Agency. (1989) Review of the national ambient air quality standards for lead:
35 Exposure analysis methodology and validation: OAQPS staff report. Research Triangle Park, NC: Office of
36 Air Quality Planning and Standards; report no. EPA-450/2-89/011. Available on the web:
37 http://www.epa.gov/ttn/naaqs/standards/pb/data/rnaaqs_l_eamv.pdf
- 38 U.S. Environmental Protection Agency. (1990a) Air quality criteria for lead: supplement to the 1986 addendum.
39 Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and
40 Assessment Office; report no. EPA/600/8-89/049F. Available from: NTIS, Springfield, VA; PB91-138420.
- 41 U.S. Environmental Protection Agency. (1990b) Review of the national ambient air quality standards for lead:
42 assessment of scientific and technical information: OAQPS staff paper. Research Triangle Park, NC: Office

- 1 of Air Quality Planning and Standards; report no. EPA-450/2-89/022. Available from: NTIS, Springfield,
2 VA; PB91-206185. Available on the web: http://www.epa.gov/ttn/naaqs/standards/pb/data/rnaaqsl_asti.pdf
- 3 U.S. Environmental Protection Agency. (1991) U.S. EPA Strategy for Reducing Lead Exposure. Available from
4 U.S. EPA Headquarters Library/Washington, D.C. (Library Code EJBD; Item Call Number: EAP
5 100/1991.6; OCLC Number 2346675). http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_pr.html
- 6 U.S. Environmental Protection Agency. (2005) Project Work Plan for Revised Air Quality Criteria for Lead.
7 CASAC Review Draft. National Center for Environmental Assessment, Research Triangle Park, NC.
8 NCEA-R-1465. Available at: http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_cr_pd.html
- 9 U.S. Environmental Protection Agency. (2006a) Plan for Review of the National Ambient Air Quality Standards for
10 Lead. Office of Air Quality Planning and Standards, Research Triangle Park, NC. Available at:
11 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_cr_pd.html
- 12 U.S. Environmental Protection Agency. (2006b) Air Quality Criteria for Lead. Washington, DC, EPA/600/R-
13 5/144aF. Available online at: http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_cr.html
- 14 U.S. Environmental Protection Agency. (2006c) Analysis Plan for Human Health and Ecological Risk Assessment
15 for the Review of the Lead National Ambient Air Quality Standards. Office of Air Quality Planning and
16 Standards, Research Triangle Park, NC. Available at:
17 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_cr_pd.html
- 18 U.S. Environmental Protection Agency. (2007a) Lead: Human Exposure and Health Risk Assessments for Selected
19 Case Studies, Volume I. Human Exposure and Health Risk Assessments – Full-Scale and Volume II.
20 Appendices. Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-452/R-07-
21 014a and EPA-452/R-07-014b. http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_cr_td.html
- 22 U.S. Environmental Protection Agency. (2007b) Review of the National Ambient Air Quality Standards for Lead:
23 Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper. Office of Air Quality
24 Planning and Standards, Research Triangle Park, NC. EPA-452/R-07-013. Available at:
25 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_cr_sp.html
- 26 U.S. Environmental Protection Agency. (2011a) Integrated Review Plan for the National Ambient Air Quality
27 Standards for Lead. Research Triangle, NC. EPA-452/R-11-008. Available online at:
28 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_index.html
- 29 U.S. Environmental Protection Agency. (2011b) Review of the National Ambient Air Quality Standards for Lead:
30 Risk and Exposure Assessment Planning Document. Office of Air Quality Planning and Standards,
31 Research Triangle Park, NC. EPA/452/P-11-003. Available at:
32 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_2010_pd.html
- 33 U.S. Environmental Protection Agency. (2011c) Integrated Review Plan for the National Ambient Air Quality
34 Standards for Lead. External Review Draft. Research Triangle, NC. EPA-452/D-11-001. Available online
35 at: http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_index.html
- 36 U.S. Environmental Protection Agency. (2011d) Integrated Science Assessment for Lead (First External Review
37 Draft). Washington, DC, EPA/600/R-10/075A. Available online at:
38 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_index.html
- 39 U.S. Environmental Protection Agency. (2012a) Integrated Science Assessment for Lead (Third External Review
40 Draft). Washington, DC, EPA/600/R-10/075C. Available online at:
41 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_2010_isa.html

1 U.S. Environmental Protection Agency. (2012b) Integrated Science Assessment for Lead (Second External Review
2 Draft). Washington, DC, EPA/600/R-10/075B. Available online at:
3 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_index.html

2 AMBIENT AIR LEAD

The focus for this Pb NAAQS review is on Pb derived from those sources emitting Pb to ambient air. As noted in section 1.3, air emissions contribute to concentrations in multiple environmental media and the role of the nonair media is enhanced by the persistent nature of Pb. Consequently this chapter discusses our current understanding of Pb in ambient air and of ambient air-related Pb in other media.

Lead emitted to the air is predominantly in particulate form, with the particles occurring in various sizes (ISA, section 3.3.1). While in some limited circumstances associated with incomplete combustion of leaded gasoline, Pb may be emitted in gaseous form, atmospheric conditions readily contribute to condensation into particles (ISA, section 3.2). Once emitted, Pb can be transported long or short distances depending on their size, which influences the amount of time spent in the aerosol phase. Consistent with previous evidence, recent research on particulate matter with mass median diameter of 2.5 and of 10 micrometers (PM_{2.5} and PM₁₀) confirms the transport of airborne Pb appreciable distances from its sources. For example, samples collected at altitude over the Pacific Ocean, as well as the seasonal pattern of Pb-PM_{2.5} at rural sites in the western U.S. indicate transport of Pb from sources in Asia, although such sources have been estimated to contribute less than 1 ng/m³ to western U.S. Pb concentrations (ISA, section 3.3.1; Murphy et al., 2007). As a generality, larger particles tend to deposit more quickly, within shorter distances from emissions points, while smaller particles remain in aerosol phase and travel longer distances before depositing (ISA, section 2.2.1). As summarized in section 2.2.2 below, airborne concentrations of Pb near sources are much higher, and the representation of larger particles generally greater, than at sites not directly influenced by sources.

In this chapter, we discuss the current information on ambient Pb regarding sources and emissions (section 2.1), current ambient air monitoring methods and networks and associated measurements (section 2.2) and the contribution of ambient air Pb to Pb in other media (section 2.3).

2.1 SOURCES AND EMISSIONS TO AMBIENT AIR

In this section we describe the most recently available information on sources and emissions of Pb into the ambient air. The section does not provide a comprehensive list of all sources of Pb, nor does it provide estimates of emission rates or emission factors for all source categories. Rather, the discussion here is intended to identify the larger source categories, either on a national or local scale, and generally describe their emissions and distribution within the U.S.

1 The primary data source for this discussion is the National Emissions Inventory (NEI) for
2 2008¹. The NEI is a comprehensive and detailed estimate of air emissions of both criteria and
3 hazardous air pollutants from air emissions sources. The NEI is generally prepared every three
4 years by the EPA based primarily upon emission estimates and emission model inputs provided
5 by state, local, and tribal air agencies for sources in their jurisdictions, and supplemented by data
6 developed by the EPA. Some of these estimates are required by regulation while some are
7 voluntarily reported. For example, states are required to report Pb emissions from facilities
8 emitting more than 5 tons of Pb per year (tpy) and from facilities emitting greater than threshold
9 amounts for other criteria pollutants (e.g., 100 tpy of particulate matter or volatile organic
10 compounds; CFR 51, subpart A). Estimates of Pb emissions presented in this document (and in
11 the ISA) are drawn from the 2008 NEI version 3.² As a result of various Clean Air Act
12 requirements, emissions standards implemented since 2008 for a number of source categories
13 represented in the NEI are projected to result in considerably lower emissions at the current time
14 or in the near future.

15 The following sections present information relative to 2008 Pb emissions on a national
16 and local scale. Lead is emitted from a wide variety of source types, some of which are small
17 individually but for which the cumulative emissions are large, and some for which the opposite is
18 true. For example, a source category may be composed of many small (i.e., low-emitting)
19 sources or of just a few very large (high-emitting) sources. Temporal trends in the national totals
20 of Pb emissions are presented in Section 2.1.1. Information about the emissions source types or
21 categories that are large on a national scale as of 2008 is presented in Section 2.1.2, while
22 information on the sources that are large at the local scale is presented in Section 2.1.3.
23 Additional information on data sources for, limitations of and our confidence in the information
24 summarized here is described in Appendix 2A.

25 **2.1.1 Temporal Trends on a National Scale**

26 Figure 2-1 shows the substantial downward trend in Pb emissions that has occurred over
27 the past several decades. The most dramatic reductions in Pb emissions occurred prior to 1990
28 in the transportation sector due to the removal of Pb from gasoline. Lead emissions were further
29 reduced substantially between 1990 and 2008, with significant reductions occurring in the metals
30 industries at least in part as a result of national emissions standards for hazardous air pollutants.

31

¹ <http://www.epa.gov/ttn/chief/net/2008inventory.html>

² With regard to Pb emissions, the 2008 NEI, version 3 (January 2013) has been augmented with sources not included in the 2008 NEI, version 2.

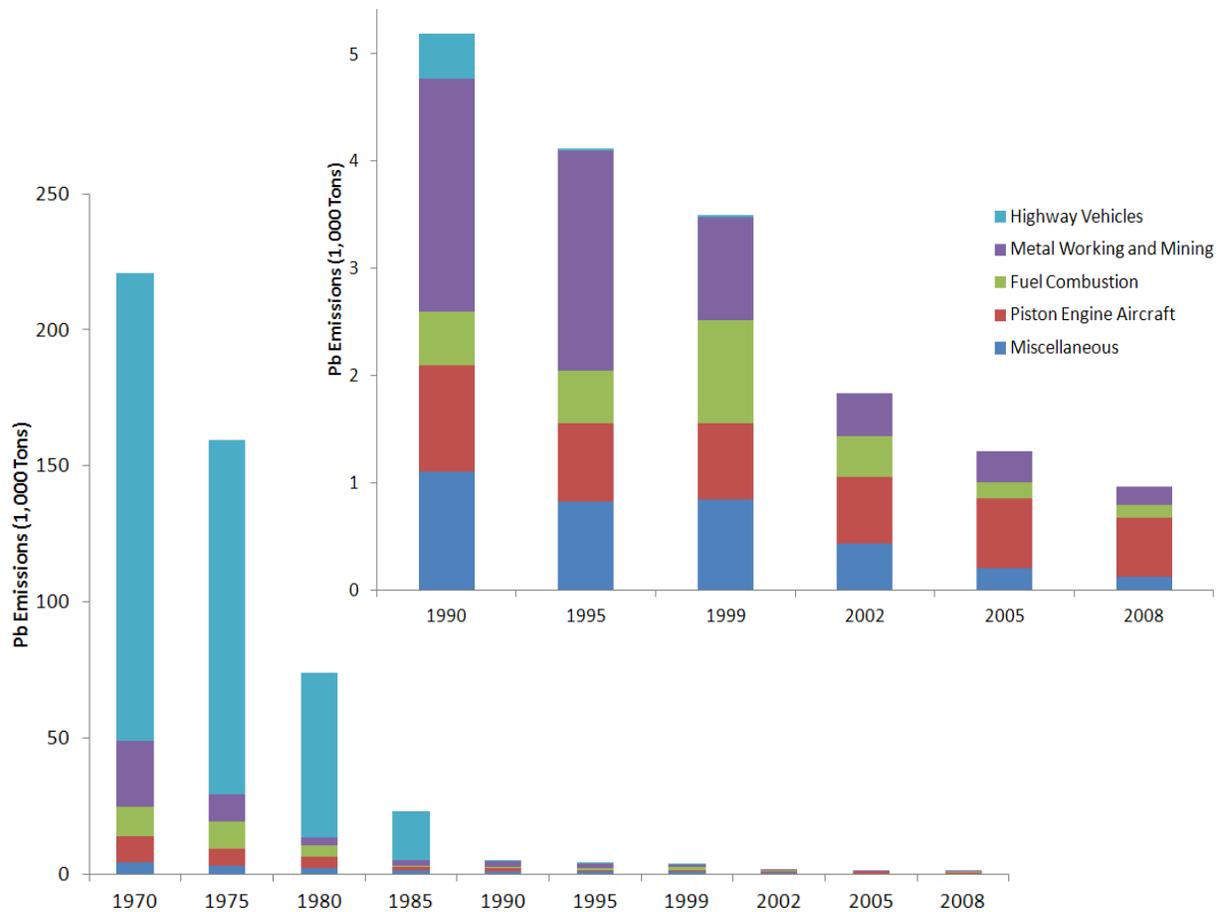


Figure 2-1. Temporal trend in air emissions: 1970-2010.

2.1.2 Sources and Emissions on National Scale - 2008

As indicated in Figure 2-1, the largest source sector emitting Pb into the atmosphere on a national scale is aviation gasoline usage by piston engine driven aircraft. The next largest nationally is metal working and mining. Considering the national estimates at a more detailed scale, the largest source categories emitting Pb into the atmosphere on a national scale, after emissions from aircraft operating on leaded fuel, are boilers and process heaters (fuel combustion) which, while individually are generally small sources, are large when aggregated nationally (Table 2-1). The next largest categories are various metals industries, including lead-specific industries (Table 2-1). Together these and other sources were estimated to emit just under a thousand tpy of Pb in the U.S. in 2008.

1 **Table 2-1. U.S. lead emissions by source categories estimated to emit at least 4 tpy.**

Source Category Description	2008 Emissions (tons)
ALL CATEGORIES ^A	950
Aircraft operating on leaded fuel ^B	550
Industrial/Commercial/Institutional Boilers & Process Heaters	64
Utility Boilers	51
Iron and Steel Foundries	30
Integrated Iron and Steel Manufacturing	27
Steel Manufacturing: Electric Arc Furnaces	22
Secondary Lead Smelting	20
Primary Lead Smelting	19 ^C
Primary Copper Smelting	17
Mining	15
Military Base	13
Cement Production	8
Glass Manufacturing	8
Battery Manufacturing	7
Secondary Non-ferrous Metals (other)	7
Primary Non-ferrous Metals (other)	7
Carbon Black	6
Pulp and Paper Production	6
Secondary Copper Smelting	5
Fabricated Metal Products Manufacturing	5
Residential Heating	5
Municipal Waste Incineration	5
Commercial Marine Vessels	5
Sewage Sludge Incineration	4
Mineral Products Manufacturing	4

A - Emissions estimate totals from 2008 National Emissions Inventory, version 3 (January 2013) for point sources and 2008 NEI version 2 for nonpoint sources (residential heating).

B - This category includes Pb emitted at or near airports as well as Pb emitted in-flight. Lead emissions at or near airports comprise 46% of the total aircraft lead emissions inventory. Emissions value based on EPA estimates.

C - There is some uncertainty regarding the total emissions estimate for this source category in which there is one operational smelter, which is planning to cease the existing smelter operations at this site by April 2014 (DRRC, 2010).

Explanation of aggregation approaches used for Tables 2-1 and 2-2:

Facilities have numerous processes that can fall into different source categories and the NEI includes process-specific emissions estimates. Source categories are groups of facilities that can be considered as the same type of emissions source. In order to present the emissions for source categories (e.g., secondary copper smelting) rather than for processes (e.g., Secondary Metal Production, Copper or Rotary Furnace) in Table 2-1, we aggregated processes for each facility and then present national estimates for source categories. The source categories used were assigned using a three-tiered approach. First, processes known to be affected by sector-specific rules were set to the source categories. This was done for Utility Boilers, Portland Cement plants, Electric Arc Furnaces, Municipal Waste Combustors, and Taconite Ore facilities (mapped to Integrated Iron and Steel Manufacturing). Other source categories did not use this first tier because the processes in the inventory have not yet been mapped to other rules. Second, for processes that clearly map to source categories, the inventory process descriptions (Source Classification Codes) were used to assign the source category. A good example of this is for Industrial, Commercial, and Institutional Boilers and Process Heaters. For all remaining processes, the Facility Type inventory field was mapped to a source category. Facility Types are the basis for aggregation used in Table 2-2. Facility Types in the NEI were set manually by EPA staff for facilities greater than 0.5 tons of Pb and using the North American Industrial Classification System (NAICS) codes for smaller facilities. This (setting of the facility type) was done with consideration of the primary activity identified for the facility, which usually confirmed the NAICS code.

A facility only has a single Facility Type but can have multiple processes and source categories. For example, some facilities are secondary metal processing plants for copper, aluminum, and non-ferrous metals, which divides their emissions into Secondary Copper and Secondary Non-Ferrous metal source categories. In these cases, the facility website was reviewed to try to assess the predominant activity and the NAICS code was considered as well, and a Facility Type was set using the best judgement of EPA staff. However, the emissions for these facilities are split across multiple processes as summed in Table 2-1. To prevent double counting of facility and state counts in Table 2-2, the Facility Type was used so that each facility shows up only once in this table.

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15

2.1.2.1 Stationary Sources

Since the last review of the Pb NAAQS, the EPA has completed a number of regulations which will result in reduced Pb emissions from stationary sources regulated under the Clean Air Act sections 112 and 129. For example, in January 2012, the EPA updated the National Emission Standards for Hazardous Air Pollutants (NESHAP) for Secondary Lead Smelting (77 FR 555). These amendments to the original maximum achievable control technology standards apply to facilities nationwide that use furnaces to recover lead from lead-bearing scrap, mainly from automobile batteries (15 existing facilities, one under construction). By the effective date in 2014, this action is estimated to result in a lead emissions reduction of 13.6 tpy across the category (a 68% reduction). Also, the NESHAP for Primary Lead Smelting was revised in 2011 (76 FR 70834) and more than a dozen additional EPA actions taken in the past 5 years, which would not be reflected in the 2008 NEI estimates, will result in Pb emissions reductions (Appendix 2B).

2.1.2.2 Mobile Sources

Forty years ago, combustion of leaded gasoline was the main contributor of Pb to the air. In the early 1970s, the EPA set national regulations to gradually reduce the Pb content in gasoline. In 1975, unleaded gasoline was introduced for motor vehicles equipped with catalytic converters. The EPA banned the use of leaded gasoline in highway vehicles after December 1995.

Lead emissions from piston-engine aircraft operating on leaded fuel are currently the largest source of Pb air emissions on a national scale. Lead is added to aviation gasoline (commonly referred to as “avgas”) used in most piston-engine aircraft in order to boost octane and prevent engine knock.³ The most commonly used avgas, 100 Octane Low Lead, contains up to 2.12 grams Pb per gallon (ASTM D 910). The Federal Aviation Administration estimates that in 2008, 248 million gallons of avgas were consumed in the U.S.⁴ contributing an estimated 550 tons of Pb to the air that comprise 57% of the national Pb inventory.⁵ Leaded avgas is used at approximately 20,000 airport facilities in the U.S.

The EPA is currently collecting and evaluating information regarding emissions and air concentrations of lead resulting from avgas combustion by piston-engine aircraft. This is part of an ongoing investigation under section 231 of the Clean Air Act into the potential for these emissions to cause or contribute to air pollution that may reasonably be anticipated to endanger public health or welfare. This evaluation by the EPA is occurring separate from the NAAQS review. The EPA’s investigation includes substantial analytical work. The timeline for completion of this investigation and possible issuance of a final endangerment determination includes completion of necessary modeling and monitoring information and other data, development of a proposal which will be published for public comment, review and analysis of comments received and issuance of the final determination. If the EPA issues a positive determination that Pb emissions from aircraft engines cause or contribute to air pollution that may reasonably be anticipated to endanger public health or welfare, the EPA would then be required to propose and promulgate emissions standards to control aircraft engine Pb emissions, and the Federal Aviation Administration would be required to promulgate regulations addressing

³ Lead is not added to jet fuel used in commercial aircraft, military aircraft, or other turbine engine aircraft.

⁴ U.S. Department of Transportation Federal Aviation Administration Aviation Policy and Plans. FAA Aerospace Forecast Fiscal Years 2010-2030. p.99. Available at: http://www.faa.gov/about/office_org/headquarters_offices/apl/aviation_forecasts/aerospace_forecasts/2010-2030/media/2010%20Forecast%20Doc.pdf This document provides historical data for 2000-2008 as well as forecast data.

⁵ EPA (2010) Calculating Piston-Engine Aircraft Airport Inventories for Lead for the 2008 National Emissions Inventory. EPA-420-B-10-044. Available at: <http://www.epa.gov/otaq/regs/nonroad/aviation/420b10044.pdf>

1 the fuel used by those aircraft. More information about EPA's actions is available at
2 www.epa.gov/otaq/aviation.htm.

3 Vehicles used in racing are not regulated by the EPA under the Clean Air Act and can
4 therefore use alkyl-Pb additives to boost octane. The National Association for Stock Car Auto
5 Racing (NASCAR) formed a voluntary partnership with the EPA with the goal of permanently
6 removing alkyl-Pb from racing fuels used in the racing series now known as the Sprint Cup, the
7 Nationwide Series and the Camping World Truck Series. The major NASCAR race series now
8 use unleaded fuels.

9 Due to the presence of Pb as a trace contaminant in gasoline, diesel fuel and lubricating
10 oil, cars, trucks, and engines operating in nonroad equipment, marine engines and jet aircraft
11 emit small amounts of Pb (ISA, Section 3.2.2.6). Additional mobile sources of Pb include brake
12 wear, tire wear, and loss of Pb wheel weights (ISA, Section 3.2.2.6).

13 **2.1.2.3 Natural Sources and Long-range Transport**

14 Some amount of Pb in the air derives from natural sources, such as volcanoes, sea salt,
15 and windborne soil particles from areas free of anthropogenic activity and some may also derive
16 from anthropogenic sources of airborne Pb located outside of the U.S. (ISA, section 3.5.5).
17 Emissions estimates for these sources, as well as wild forest fires and biogenic sources have not
18 been developed for the NEI and quantitative estimates for these processes remain an area of
19 significant uncertainty. Based on several different approaches, the ISA identifies several
20 estimates of the concentration of airborne Pb derived from natural sources. All of the estimates
21 are no higher than 1 nanogram per cubic meter (ng/m^3), and range down as low as $0.02 \text{ ng}/\text{m}^3$
22 (ISA, section 3.5.5). The data available to derive such an estimate are limited and such a value
23 might be expected to vary geographically with the natural distribution of Pb.

24 Another contribution to U.S. airborne Pb concentrations is long-range transport such as
25 that associated with air masses carrying Pb from sources in Asia, where controls on Pb emissions
26 have lagged those in the U.S. and Canada (ISA, section 3.5.5; Osterberg et al., 2008). The most
27 recent estimates of contributions from Asia, however, conclude that the Asian contribution to
28 U.S. airborne Pb concentrations is generally less than $1 \text{ ng}/\text{m}^3$ (ISA, section 3.5.5; Murphy,
29 2007; Ewing et al., 2010).

30 **2.1.2.4 Previously Deposited or Released Lead**

31 Lead-bearing particles that occur on surface soils or built surfaces as a result of previous
32 or historic deposition can be a source of airborne Pb as a result of particle resuspension and may
33 be a significant source of airborne Pb in areas near major sources of Pb emissions. Outdoor dust
34 may be resuspended into the air by wind or human-induced mechanical forces, such that the
35 main drivers of particle resuspension are typically mechanical stressors such as vehicular traffic,

1 construction and agricultural operations, and, generally to a lesser extent, the wind (ISA, section
2 3.3.1.3; 2006 CD, section 2.3.3). Wind resuspension is often defined in terms of a resuspension
3 rate, which is the fraction of a surface contaminant released per unit time. Resuspension rates
4 are dependent on many factors, including wind speed, soil/surface moisture, particle sizes,
5 presence of saltating particles and presence of vegetation; typical values range over several
6 orders of magnitude (ISA, section 3.3.1.3; 2006 CD, section 2.3.3). Vehicular resuspension
7 results from shearing stress of tires or turbulence generated by a passing vehicle and can be
8 affected by a number of factors including vehicle size, vehicle speed, moisture and particle size
9 (ISA, section 3.3.1.3; 2006 CD, section 2.3.3). Additionally, the amount of material available
10 for resuspension can be influenced by removal processes such as surface runoff associated with
11 rainfall (ISA, sections 2.2.1, 3.3.2 and 3.3.2.4). Rather than a continuous process, resuspension
12 may occur as a series of events. Short episodes of conditions or factors conducive to
13 resuspension may dominate annual averages of upward flux (2006 CD, p. 2-65). Variability and
14 uncertainty in these factors, and with regard to surface soil/dust composition, affect quantitative
15 emissions estimates for these processes (2006 CD, section 2.3.3).⁶

16 Consideration of Pb concentrations in air and surface soil/dust in different types of
17 locations also informs our understanding of the relative importance of resuspension of previously
18 deposited dust particles as an influence on airborne Pb concentrations. The relative importance
19 would be expected to vary with site-specific circumstances, such as the magnitude of Pb
20 concentrations in the surface dust and air Pb contributions from nearby sources of new Pb
21 emissions, as well as with variation in the forces that influence particle resuspension. For
22 example, the contribution of resuspension to airborne concentrations appears to be greatest in
23 areas of highly contaminated surface dust such as at historically active industrial sites, which
24 may or may not be currently active (ISA, sections 3.3.1.3 and 3.5.3).

25 Emissions from currently active metals industries include those related to current
26 industrial activity and to resuspension of previously deposited material (often a component of
27 “fugitive” emissions estimates⁷). Accordingly, the relative magnitude of air Pb concentrations
28 associated with no longer active industries would be expected to generally be lower than that for

⁶ Quantitative estimates of resuspension-related emissions associated with many active industrial sources (particularly metals sources) are included within the NEI, although such emissions associated with previously and no longer active Pb sources are not as generally included in the NEI.

⁷ For example, emissions factors have been established to estimate fugitive emissions from resuspension of previously deposited material as a result of vehicular traffic on facility roadways (USEPA, 1996-2011). Where used, these estimates are combined with estimates for "process fugitives" (emissions that escape capture by control devices) to estimate total fugitive emissions from a facility. Accordingly, control of resuspension resulting from facility roadways, buildings or other property may be part of a strategy to meet regulatory emissions requirements (e.g., national emissions standards for hazardous air pollutants for secondary lead smelting, 77 FR 556).

1 active industries yet greater than that in locations somewhat removed from industrial sources.
2 The limited available data for comparison generally confirms this (section 2.2.2.2 below).

3 Lead-bearing particulate matter in other, non-industrial, locations of appreciable historic
4 lead contamination, such as in soils or on surfaces in older urban areas or near older
5 transportation corridors may, if disturbed, also become suspended into the air and contribute to
6 air Pb concentrations. In older transportation corridors or other locations not influenced by
7 active industries, the significance of resuspension (e.g., in terms of resultant air Pb
8 concentrations) appears to be much less than that associated with active industries or now closed
9 industries with substantial emissions in the past.⁸ For example, the available data indicate that
10 current Pb concentrations near roadways are substantially lower than those near large, currently
11 active industrial sources (see ISA, section 3.3.1.3 and Figures 2-9 and 2-11 below). In general,
12 air Pb concentrations at sites described as not influenced by an active industry are much lower
13 than those near active sources (see ISA, section 3.5.1.2 and Figure 2-11 below).

14 **2.1.3 Sources and Emissions on Local Scale**

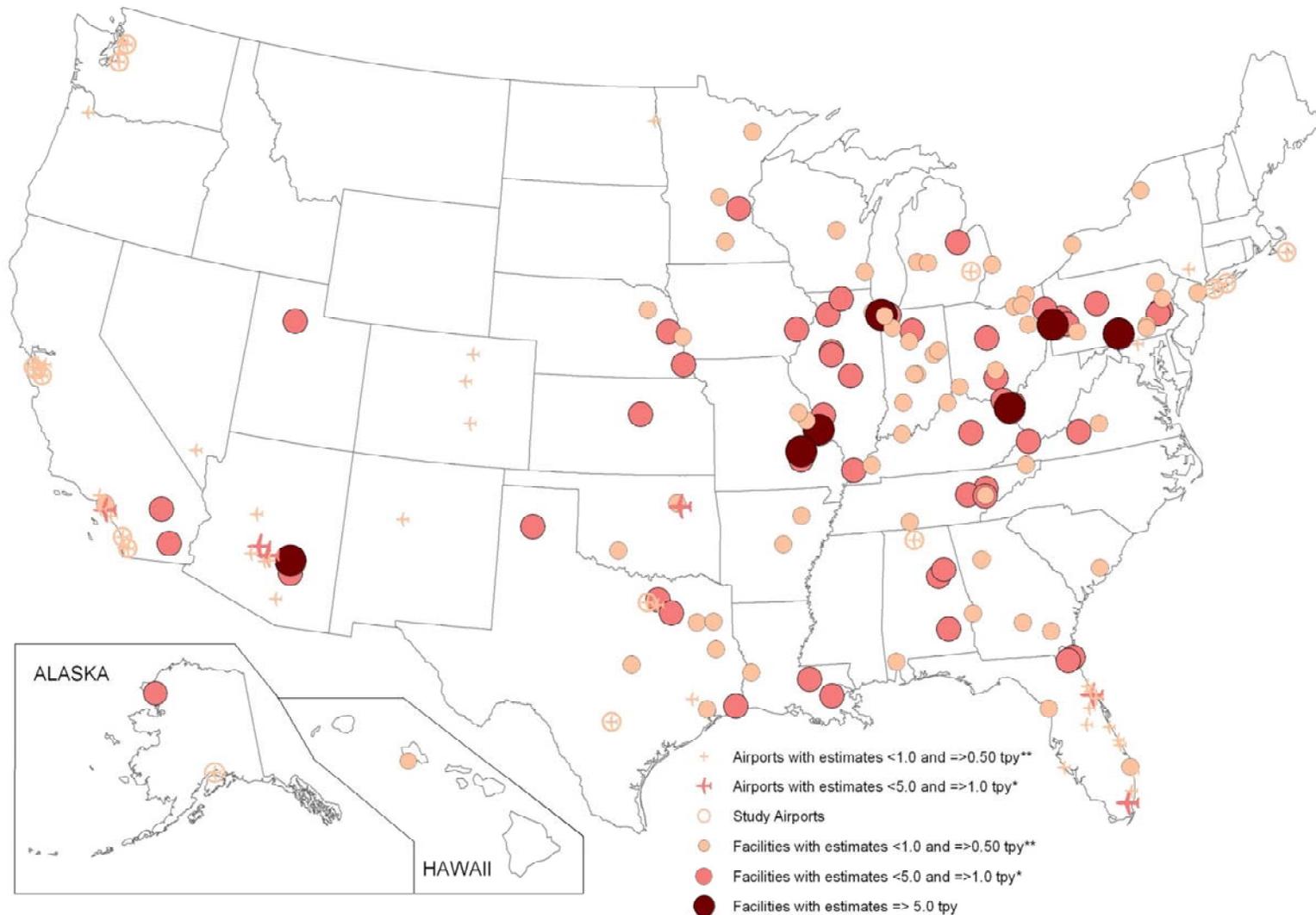
15 Based on the 2008 estimates, the highest emissions in specific (local) situations are
16 different types of metals industries, 23 of which had 2008 estimates greater than or equal to 1.0
17 tpy Pb (Table 2-2). The geographic distribution of the facilities summarized in Table 2-2 is
18 presented in Figure 2-2.
19

⁸ Mass-balance analyses of emissions in southern California newly available in the last review suggested that Pb in resuspended road dust may represent between 40% and 90% of Pb emissions in some areas (2006 CD, p. 2-65; ISA, section 3.2.2.7). Air Pb monitoring data near roadways, however, including those in California, indicate Pb concentrations well below those near significant industrial sources and below the current Pb NAAQS (e.g., ISA, section 3.5.1.2).

1 **Table 2-2. Facilities estimated to emit at least 0.50 tpy in 2008.**

Facility Type	Facilities emitting ≥ 1.0 tpy ^{A,B}			Facilities emitting < 1.0 and ≥ 0.50 ^{A,C}	
	No. Facilities	No. States	Facility Emissions	No. Facilities	No. States
Primary Lead Smelting Plant	1	1	19.2 ^D		
Steel Mill	14	8	1.0 - 9.8	10	9
Primary Copper Smelting/Refining Plant	3	2	3.1 - 8.8		
Military Base	4	3	1.4 - 7.2	3	3
Secondary Lead Smelting Plant	5	5	1.4 - 7.1	3	2
Carbon Black Plant	1	1	6.3		
Primary Non-ferrous Metal Smelting/Refining Plant (not Lead, Aluminum, or Copper)	1	1	5.4		
Mine or Quarry	5	2	1.6 - 3.8		
Ethanol Biorefinery	2	2	1.5 - 3.8		
Portland Cement Plant	1	1	3.3		
Pulp and Paper Plant	2	2	2.5 - 3.0	2	2
Calcined Pet Coke Plant	1	1	2.5		
Battery Plant	3	3	1.5 - 2.5	1	1
Wood Products Plant	1	1	2.3		
Foundry, Iron and Steel	3	3	1.1 - 2.3	10	8
Secondary Copper Smelting/Refining Plant	1	1	2.3		
Foundry, Non-ferrous	3	2	1.2 - 2.0	1	1
Secondary Non-ferrous Metal Smelting/Refining Plant (not Lead, Aluminum, or Copper)	2	2	1.2 - 2.0	1	1
Coke Battery	2	2	1.0 - 1.8		
Electricity Generation via Combustion	2	2	1.0 - 1.7	7	5
Chemical Plant	1	1	1.7	1	1
Airport	6	4	1.0 - 1.3	52	17
Automobile/Truck or Parts Plant	1	1	1.2	3	2
Wastewater Treatment Facility	1	1	1.0	2	2
Munition or Explosives Plant	1	1	1.0	2	2
Fabricated Metal Products Plant				6	5
Municipal Waste Combustor				3	3
Glass Plant				2	2
Lumber/Sawmill				2	1
Plastic, Resin, or Rubber Products Plant				1	1
Taconite Processing Plant				1	1
Petrochemical Plant				1	1
Bulk Terminal or Bulk Station				1	1

A - Emissions totals from 2008 National Emissions Inventory, version 3 (January 2013), except in the case of airports for which EPA specific estimates were used.
B - This category includes facilities with total emissions estimates greater than or equal to 0.95 tpy.
C - This category includes facilities with total emissions estimates greater than or equal to 0.495 and less than 0.95 tpy.
D - There is some uncertainty regarding this total emissions estimate for this facility, the only remaining operational primary Pb smelter in the U.S., which is planning to cease the existing smelter operations at this site by April, 2014 (DRRC, 2010).



* Includes facilities with emissions greater than or equal to 1.0 tpy (after rounding to 1 decimal place).

** Includes facilities with emissions greater than or equal to 0.50 tpy (after rounding to 2 decimal places) and less than 1.0 (after rounding to 1 decimal place).

Study airports are described in section 2.2 below.

1
2 **Figure 2-2. Geographic distribution of facilities estimated to emit at least 0.50 tpy in 2008.**

1 **2.2 AMBIENT AIR QUALITY**

2 The EPA and state and local agencies have been measuring Pb in the atmosphere since
3 the 1970s. In response to reduced emissions (see section 2.1), Pb concentrations have decreased
4 dramatically over that period. Currently, the highest concentrations occur near some metals
5 industries where some individual locations have concentrations that exceed the NAAQS. This
6 section describes the ambient Pb measurement methods, the sites and networks where these
7 measurements are made, and how the ambient Pb concentrations vary geographically and
8 temporally.

9 **2.2.1 Air Monitoring**

10 Ambient air Pb concentrations are measured by five national monitoring networks. The
11 networks include the State and Local Air Monitoring Sites (SLAMS) intended for Pb NAAQS
12 surveillance, the PM_{2.5} Chemical Speciation Network (CSN), the Interagency Monitoring of
13 Protected Visual Environments (IMPROVE) network, the National Air Toxics Trends Stations
14 (NATTS) network, and the Urban Air Toxics Monitoring program. All of the data from these
15 networks are accessible via EPA's Air Quality System (AQS):

16 <http://www.epa.gov/ttn/airs/airsaqs/>. In addition to these networks, various environmental
17 organizations have operated other sampling sites yielding data (which may or may not be
18 accessible via AQS) on ambient air concentrations of Pb, often for limited periods and/or for
19 primary purposes other than quantification of Pb itself. The subsections below describe each
20 network and the Pb measurements made at these sites.

21 **2.2.1.1 Lead NAAQS Surveillance Network**

22 This section describes sample collection, analysis and network aspects for the Pb SLAMS
23 network, the main purpose of which is surveillance of the Pb NAAQS. As such, the EPA
24 regulates how this monitoring is conducted in order to ensure accurate and comparable data for
25 determining compliance with the NAAQS. The code of federal regulations (CFR) at parts 50, 53
26 and 58 specifies required aspects of the ambient monitoring program for NAAQS pollutants.⁹ In
27 order to be used in NAAQS attainment designations, ambient Pb concentration data must be
28 obtained using either the federal reference method (FRM) or a federal equivalent method (FEM).
29 The indicator for the current Pb NAAQS is Pb-TSP. However, in some situations,¹⁰ ambient Pb-

⁹ The federal reference methods (FRMs) for sample collection and analysis are specified in 40 CFR part 50, the procedures for approval of FRMs and federal equivalent methods are specified in 40 CFR part 53 and the rules specifying requirements for the planning and operations of the ambient monitoring network are specified in 40 CFR part 58.

¹⁰ The Pb-PM₁₀ measurements may be used for NAAQS monitoring as an alternative to Pb-TSP measurements in certain conditions defined in 40 CFR part 58 Appendix D paragraph 2.10.1.2. These conditions

1 PM₁₀ concentrations may be used in judging nonattainment. Accordingly, FRMs have been
2 established for Pb-TSP and for Pb-PM₁₀. The current FRM for the measurement of Pb-TSP is
3 provided in 40 CFR part 50 Appendix G. This FRM includes sampling using a high-volume
4 TSP sampler that meets the design criteria identified in 40 CFR part 50 Appendix B and sample
5 analysis for Pb content using flame atomic absorption. There are 24 FEMs currently approved
6 for Pb-TSP.¹¹ All 24 FEMs are based on the use of high-volume TSP samplers and a variety of
7 approved equivalent analysis methods.

8 A new FRM for Pb-PM₁₀ was promulgated as part of the 2008 review. This FRM is
9 based on the PM₁₀ sampler defined in 40 CFR part 50 Appendix J coupled with x-ray
10 fluorescence (XRF) analysis. In addition, one FEM for Pb-PM₁₀ has been finalized for the
11 analysis of Pb-PM₁₀ based on inductively coupled plasma mass spectroscopy (ICP-MS).

12 The current Pb monitoring network design requirements for NAAQS compliance
13 purposes (40 CFR part 58, Appendix D, paragraph 4.5) include two types of monitoring sites –
14 source-oriented monitoring sites and non-source-oriented monitoring sites. Source-oriented
15 monitoring sites are required near sources of air Pb emissions which are expected to or have been
16 shown to contribute to ambient air Pb concentrations in excess of the NAAQS. At a minimum,
17 there must be one source-oriented site located to measure the maximum Pb concentration in
18 ambient air resulting from each non-airport Pb source estimated to emit 0.50 or more tons of Pb
19 per year and from each airport estimated to emit 1.0 or more tons of Pb per year.¹² The EPA
20 Regional Administrators may require additional monitoring beyond the minimum requirements
21 where the likelihood of Pb air quality violations is significant. Such locations may include those
22 near additional industrial Pb sources, recently closed industrial sources and other sources of
23 resuspended Pb dust, as well as airports where piston-engine aircraft emit Pb (40 CFR, part 58,
24 Appendix D, section 4.5(c)).

25 Monitoring agencies are also required, under 40 CFR, part 58, Appendix D, to conduct
26 non-source-oriented Pb monitoring at the NCore sites required in Core metropolitan with a
27 population of 500,000 or more.¹³ NCore is a new network of multipollutant monitoring stations

include where Pb concentrations are not expected to equal or exceed 0.10 micrograms per cubic meter as an arithmetic three-month mean and where the source of Pb emissions is expected to emit a substantial majority of its Pb in the size fraction captured by PM₁₀ monitors.

¹¹ A complete list of FEM can be found at the following webpage -
<http://www.epa.gov/ttn/amtic/files/ambient/criteria/reference-equivalent-methods-list.pdf>

¹² The Regional Administrator may waive the requirement in paragraph 4.5(a) for monitoring near Pb sources if the State or, where appropriate, local agency can demonstrate the Pb source will not contribute to a maximum three-month average Pb concentration in ambient air in excess of 50 percent of the NAAQS level based on historical monitoring data, modeling, or other means (40 CFR, part 58, Appendix D, section 4.5(a)(ii)).

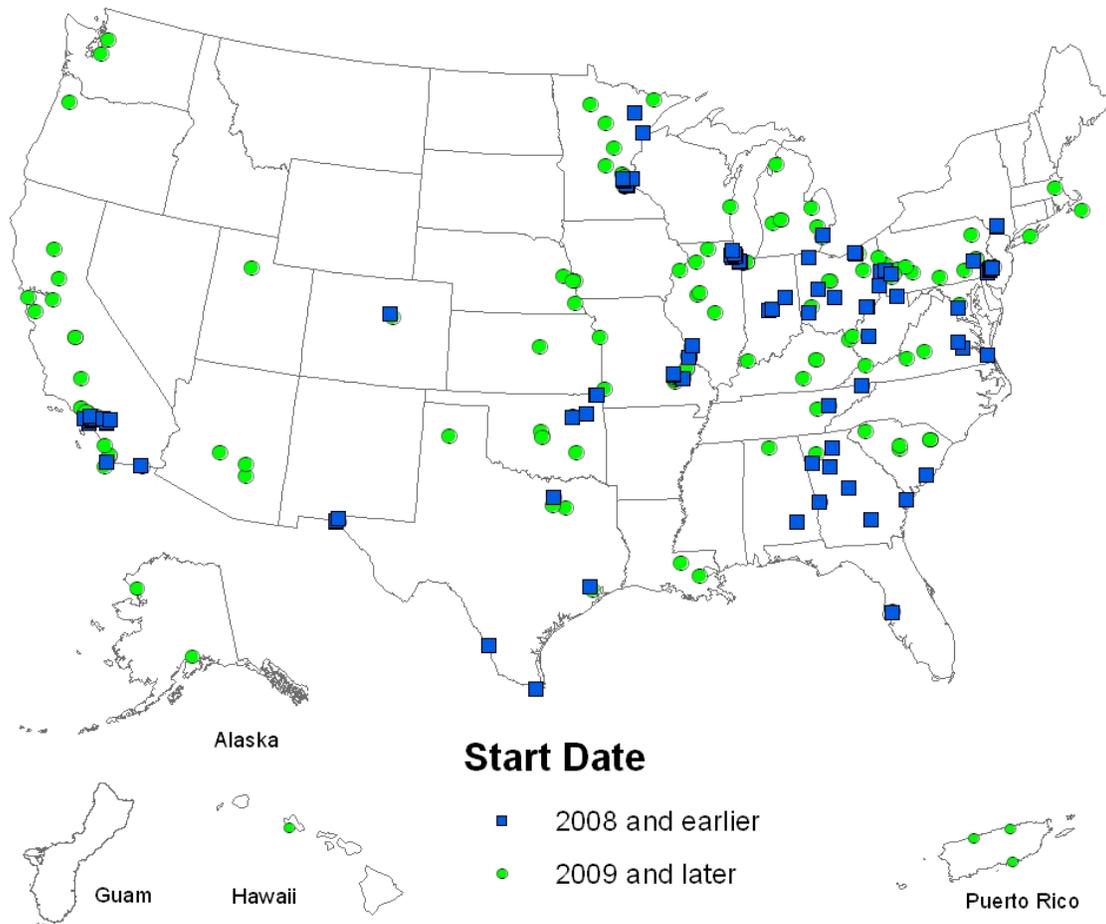
¹³ Defined by the US Census Bureau - <http://www.census.gov/population/www/metroareas/metroarea.html>

1 intended to meet multiple monitoring objectives. The NCore stations are a subset of the state and
2 local air monitoring stations network and are intended to support long-term trends analysis,
3 model evaluation, health and ecosystem studies, as well as NAAQS compliance. The complete
4 NCore network consists of approximately 60 urban and 20 rural stations, including some existing
5 SLAMS sites that have been modified for additional measurements. Each state will contain at
6 least one NCore station, and 46 of the states plus Washington, DC, will have at least one urban
7 station.

8 Either Pb-TSP or Pb-PM₁₀ monitoring may be performed at these sites. Of 24 NCore
9 sites measuring Pb concentrations, 17 are measuring Pb in TSP and 7 are measuring Pb in PM₁₀.
10 While non-source-oriented monitoring data can be used for purposes of NAAQS attainment
11 designations, a main objective for non-source-oriented monitoring is to gather information on
12 neighborhood-scale lead concentrations that are typical in urban areas in order to better
13 understand ambient air-related Pb exposures for populations in these areas.

14 Source-oriented monitors near sources estimated to emit 1.0 tpy Pb were required to be
15 operational by January 1, 2010, and the remainder of the newly required source-oriented
16 monitors were required to be operational by December 27, 2011 (75 FR 81126). Currently,
17 approximately 260 Pb-TSP monitors are in operation; these are a mixture of source- and non-
18 source-oriented monitors. Figure 2-3 shows the geographic distribution of these monitors (in
19 addition to the airport study monitors described below) in the current¹⁴ Pb NAAQS surveillance
20 network, with the Pb-TSP monitors existing at the time of the 2008 rulemaking indicated
21 separately from the newly sited Pb-TSP monitors.

¹⁴ This figure reflects Pb-TSP monitors in AQS as of September 2012.

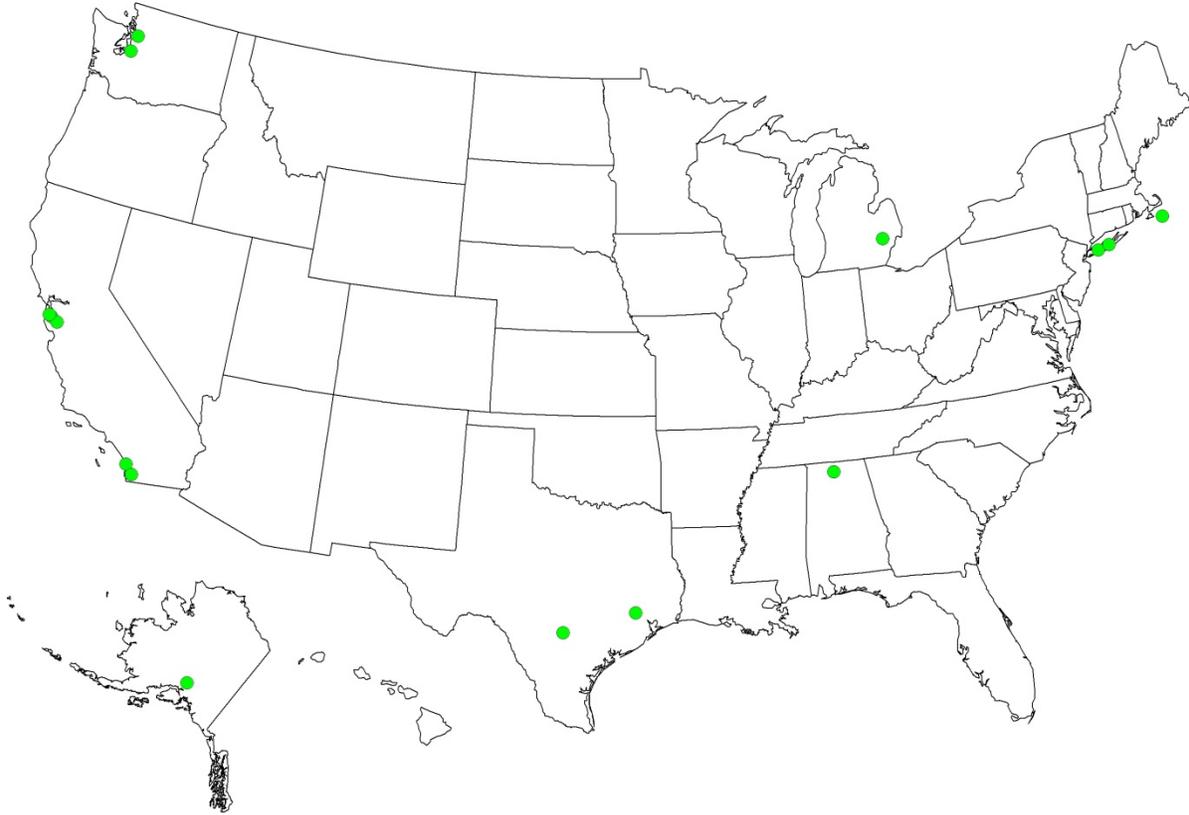


1
 2 **Figure 2-3. Map of Pb-TSP Monitoring Sites in Current Pb NAAQS Monitoring**
 3 **Network.¹⁵**

4 The current regulations also required one year of Pb-TSP monitoring (using FRM or
 5 FEM methods) near 15 specific airports in order to gather additional information on the
 6 likelihood of NAAQS exceedances near airports due to the combustion of leaded aviation
 7 gasoline (75 FR 81126). These airports were selected based on three criteria: annual Pb
 8 inventory between 0.5 ton/year and 1.0 ton/year, ambient air within 150 meters of the location of
 9 maximum emissions (e.g., the end of the runway or run-up location), and airport configuration
 10 and meteorological scenario that leads to a greater frequency of operations from one runway.
 11 These characteristics were selected because they are expected, collectively, to identify airports
 12 with the highest potential to have ambient Pb concentrations approaching or exceeding the Pb
 13 NAAQS. Data from this monitoring study will be used to assess the need for additional Pb

¹⁵ Estimates for source-oriented monitors are based on Pb emissions estimates in the 2008 National Emissions Inventory.

1 monitoring at airports. These 15 sites (Figure 2-4) were intended to be operational no later than
2 December 27, 2011, although delays in monitor siting extended the installation of some of these
3 monitors into late 2012.



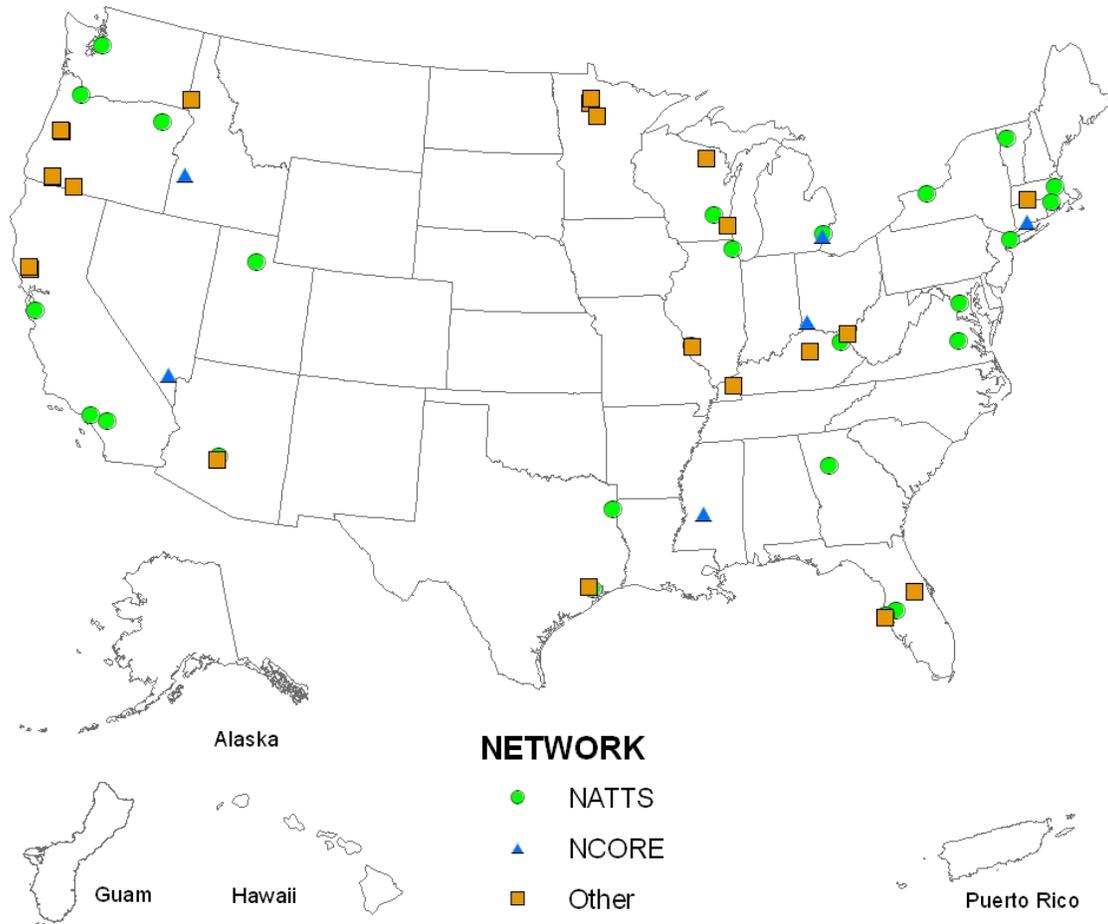
4
5 **Figure 2-4. Map of sites near airports for which one year of monitoring is required.**¹⁶

6
7 **2.2.1.2 Other Lead Monitoring Networks**

8 The NATTS network, designed to monitor concentrations of hazardous air pollutants
9 including lead compounds, included 25 sites measuring Pb-PM₁₀ as of June 2012. Most of these
10 sites (20) are in urban areas (Figure 2-5). In addition to the NATTS network, states collect Pb-
11 PM₁₀ at an additional 28 sites (most as part of the Urban Air Toxics Monitoring program). All
12 collect particulate matter as PM₁₀ for toxic metals analysis using either a high-volume PM₁₀
13 sampler or a low volume PM₁₀ sampler, typically on a 1 in 6 day sampling schedule. Most of
14 these monitoring locations are not measuring using FRM/FEM methods at this time. Lead in the

¹⁶ The 15 Airports are: Merrill Field (Anchorage, AK), Pryor Field Regional (Limestone, AL), Palo Alto Airport of Santa Clara County and Reid-Hillview (both in Santa Clara, CA), McClellan-Palomar and Gillespie Field (both in San Diego, CA), San Carlos (San Mateo, CA), Nantucket Memorial (Nantucket, MA), Oakland County International (Oakland, MI), Republic and Brookhaven (both in Suffolk, NY), Stinson Municipal (Bexar, TX), Northwest Regional (Denton, TX), Harvey Field (Snohomish, WA), and Auburn Municipal (King, WA).

1 collected sample is generally quantified via an ICP/MS method. The standard operating
2 procedure for metals by ICP/MS is available at: <http://www.epa.gov/ttn/amtic/airtox.html>.

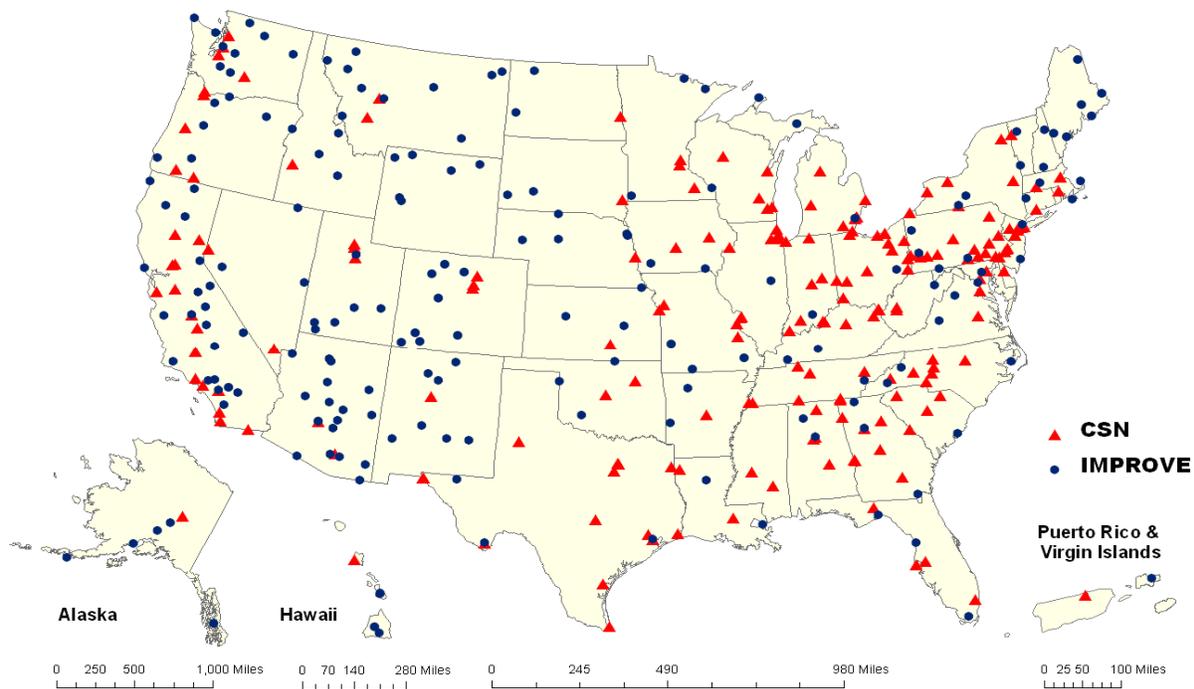


3
4 **Figure 2-5. Pb-PM₁₀ monitoring sites.**

5 Two networks measure Pb in PM_{2.5}, the EPA CSN and the IMPROVE network. The
6 CSN consists of 53 long-term trends sites (commonly referred to as the Speciation Trends
7 Network or STN sites) and approximately 150 supplemental sites, all operated by state and local
8 monitoring agencies. Most STN sites operate on a 1 in 3 day sampling schedule, while most
9 supplemental sites operate on a 1 in 6 day sampling schedule. All sites in the CSN network
10 determine Pb concentrations in PM_{2.5} samples. Lead is quantified via the XRF method.¹⁷ Data
11 are accessible through AQS. The locations of the CSN are shown in Figure 2-6. Nearly all of the
12 CSN sites are in urban areas, often at the location of highest known PM_{2.5} concentrations. The
13 first CSN sites began operation around 2000.

¹⁷ The standard operating procedure for metals by XRF is available at:
<http://www.epa.gov/ttnamti1/files/ambient/pm25/spec/xrfsop.pdf>.

1 The IMPROVE network is administered by the National Park Service, largely with
2 funding by the EPA, on behalf of federal land management agencies and state air agencies that
3 use the data to track trends in rural visibility. Lead in $PM_{2.5}$ is quantified via the XRF method, as
4 in the CSN. Data are managed and made accessible mainly through the VIEWS website
5 (<http://vista.cira.colostate.edu/views/>) but are also available via AQS. Samplers are operated by
6 several different federal, state, and tribal host agencies on the same 1 in 3 day schedule as the
7 STN. In the IMPROVE network, $PM_{2.5}$ monitors are placed in “Class I” areas (including
8 National Parks and wilderness areas) and are mostly in rural locations (Figure 2-6). The oldest
9 of these sites began operation in 1988, while many others began in the mid 1990s. There are 110
10 formally designated IMPROVE sites, which are located in or near national parks and other Class
11 I visibility areas, virtually all of these being rural. Approximately 80 additional sites at various
12 urban and rural locations, requested and funded by various parties, are also informally treated as
13 part of the network.



14

15 **Figure 2-6. Pb- $PM_{2.5}$ monitoring sites in CSN and IMPROVE networks (2012).**

16

2.2.1.3 NAAQS Surveillance Monitoring Considerations

In this section aspects of the methods for sampling and analysis of the Pb are reviewed, and the current NAAQS surveillance network of monitoring locations is considered. The methods for sampling and analysis are considered in light of the indicator for the Pb NAAQS, and conclusions regarding the current NAAQS and associated indicator appear in chapter 4. Consideration of the ambient air monitoring network generally informs the interpretation of current data on ambient air concentrations and helps identify whether the monitoring network is adequate to determine compliance with the NAAQS. This section discusses considerations related to these aspects of the ambient air monitoring program for Pb.¹⁸

2.2.1.3.1 Sampling Considerations

As described in section 2.2.1.1 above, consistent with the Pb NAAQS indicator being Pb-TSP, the FRM for Pb, which is required at all source-oriented sites, is measurement of Pb-TSP using a high-volume TSP sampler meeting the design criteria specified in 40 CFR part 50 Appendix B. During the review of the Pb NAAQS completed in 2008, CASAC noted the variability in high-volume TSP sample measurements associated with the effects of wind speed and wind direction on collection efficiency in their comments regarding the indicator. However, at the time of the 2008 review, no alternative TSP sampler designs were identified that had an adequate characterization of their collection efficiency over a wide range of particle sizes. The existing high-volume sampler was retained as the sampling approach for the Pb-TSP FRM and FEMs.

Since promulgation of the 2008 Pb NAAQS, the EPA has initiated an effort to review alternative sampler designs in an effort to develop a new sampler to replace the current high-volume Pb-TSP sampler. Efficient collection of particles much larger than 10 μm is considerably more challenging because the greater inertia and higher settling velocities of Pb particles hinder their efficient intake by samplers. The sampling difficulties and the long history of research to develop adequate sampling technology for large particles have been thoroughly reviewed (Garland and Nicholson, 1991). Some existing commercially available sampler inlets are designed to collect particles larger than 10 μm with greater than 50% efficiency (Kenny et al., 2005), and these inlets can be tested as potential replacements for TSP sampling. However, no alternatives to the FRM TSP sampler have been identified that have been adequately characterized.

¹⁸ The code of federal regulations (CFR) at parts 50, 53 and 58 specifies required aspects of the ambient monitoring program for NAAQS pollutants. The federal reference methods (FRMs) for sample collection and analysis are specified in 40 CFR part 50, the procedures for approval of FRMs and federal equivalent methods (FEMs) are specified in 40 CFR part 53 and the rules specifying requirements for the planning and operations of the ambient monitoring network are specified in 40 CFR part 58.

1 The EPA has initiated efforts to characterize the collection efficiency of alternative
2 sampler designs through wind tunnel testing as a necessary step towards the development of a
3 new sampler capable of sampling particles larger than PM₁₀ without the noted wind speed and
4 wind direction biases. In addition to the difficulties in collection of particles much larger than 10
5 μm, physical limitations in the ability to generate and transport ultra-coarse particles limit the
6 ability to adequately characterize the new sampler. These limitations ultimately limit the upper
7 cut-point of potential samplers to the range of 18-20 micrometers. Following characterization in
8 a wind tunnel, field testing of promising candidates would be required to evaluate performance
9 and to make comparisons to the existing Pb-TSP samplers. This effort is expected to take
10 several years to complete, and, as such, it is unlikely that this new sampler will be available in
11 time for consideration during this NAAQS review due to the activities which will need to be
12 completed to adequately characterize its performance both in the laboratory and the field. We
13 expect the new sampler to be available for consideration in a future review and consequently do
14 not expect to consider new alternatives for sampling methods for Pb-TSP as part of this review.

15 In addition to the FRM for Pb-TSP, there is also, as noted in section 2.2.1.1 above, a
16 FRM for Pb-PM₁₀, based on the PM₁₀ sampler defined in 40 CFR part 50 Appendix J coupled
17 with XRF analysis. The Pb-PM₁₀ measurements may be used as an alternative to Pb-TSP
18 measurements in certain conditions defined in 40 CFR part 58 Appendix D paragraph 2.10.1.2.
19 These conditions include where Pb concentrations are not expected to equal or exceed 0.10
20 micrograms per cubic meter on an arithmetic 3-month mean and where the source of Pb
21 emissions is expected to emit a substantial majority of its Pb in the PM₁₀ size fraction. At this
22 time, we believe the low-volume FRM sampler for Pb-PM₁₀ to be adequate for this application.
23 Hence, we do not expect to consider new sampling methods for Pb-PM₁₀ as part of this review.

24 **2.2.1.3.2 Analysis Considerations**

25 Due to reduced availability of laboratories capable of performing flame atomic
26 adsorption analyses and general advances in analysis methods, the EPA has initiated an effort to
27 expand FRM analysis methods beyond atomic adsorption to include the more modern analysis
28 method, ICP-MS. A consultation with the CASAC Ambient Air Monitoring and Methods
29 Subcommittee was held on September 15, 2010 (Russell and Samet, 2010), and the EPA plans to
30 propose a new FRM for Pb-TSP based on this more modern analysis method in 2013. In
31 addition, the EPA has approved several new FEMs (for ICP-MS and other analysis methods)
32 since the last Pb NAAQS review was completed in 2008.

33 With regard to Pb-PM₁₀ samples, in addition to the FRM analysis method (XRF), two
34 FEMs have been accepted for Pb-PM₁₀ analysis since the 2008 Pb NAAQS rulemaking. These
35 methods are based on ICP-MS and are consistent with analysis methods used for the NATTS

1 network. The EPA will continue to consider new FEMs for analysis of Pb-PM₁₀ and Pb-TSP as
2 applications are received, although no new FRMs (beyond the Pb-TSP FRM for ICP-MS
3 discussed above) are expected during this NAAQS review.

4 **2.2.1.3.3 Network Design Considerations**

5 Significant revisions to the Pb network design requirements (40 CFR part 58, Appendix
6 D) were made as part of the 2008 Pb NAAQS review and an associated revision to the
7 requirements in 2010. As summarized in section 2.1.1.1 above, the current Pb monitoring
8 network design requirements (40 CFR part 58, Appendix D, paragraph 4.5) include two types of
9 monitoring sites – source-oriented monitoring sites and non-source-oriented monitoring sites - as
10 well as the collection of a year of Pb-TSP measurements at 15 specific airports. This section
11 describes the design considerations for the Pb NAAQS surveillance network.

12 **Source-Oriented Monitoring.** Since the phase out of Pb in on-road gasoline, Pb is
13 widely recognized as a source-oriented air pollutant. As summarized in the ISA, variability in
14 air Pb concentrations is highest in areas including a Pb source, “with high concentrations
15 downwind of the sources and low concentration at areas far from sources” (ISA, p. 3-88).
16 Recent data summarized in section 2.2.2.2 below indicates that the highest ambient Pb
17 concentrations are found near large Pb sources, usually metals industries (see, for example,
18 Figures 2-11 through 2-13 below). Analysis of the monitoring network during the last Pb
19 NAAQS review with regard to adequacy of monitoring near such sources found that monitors
20 were lacking near many of the larger Pb emissions sources, leading the EPA to conclude that the
21 monitoring network existing at that time was inadequate to determine compliance with the
22 revised Pb NAAQS (73 FR 29262). Findings and conclusions of that analysis led to revisions of
23 network design requirements for source-oriented monitoring, begun with the 2008 Pb NAAQS
24 rulemaking (73 FR 66964). Additional revisions were completed as part of a reconsideration of
25 the monitoring requirements in December 2010 (75 FR 81126).

26 The current requirements for source-oriented monitoring include placement of monitor
27 sites near sources of air Pb emissions which are expected to or have been shown to contribute to
28 ambient air Pb concentrations in excess of the NAAQS. At a minimum, there must be one
29 source-oriented site located to measure the maximum Pb concentration in ambient air resulting
30 from each non-airport Pb source which emits 0.50 or more tons of Pb per year and from each
31 airport which emits 1.0 or more tons of Pb per year.¹⁹ The expansion of the network, including
32 these source-oriented sites, is shown in Figure 2-3. Comparison of Figure 2-3 (monitors) with

¹⁹ The Regional Administrator may waive the requirement in paragraph 4.5(a) for monitoring near Pb sources if the State or, where appropriate, local agency can demonstrate the Pb source will not contribute to a maximum Pb concentration in ambient air in excess of 50 percent of the NAAQS (based on historical monitoring data, modeling, or other means).

1 Figure 2-2 (sources) illustrates the coverage which the current monitoring network now provides
2 of large Pb emissions sources.

3 The emissions threshold for source-oriented monitoring sites, 0.50 tpy, was developed
4 based on an analysis intended to estimate the lowest emission rate that under reasonable worst-
5 case conditions (e.g., meteorological and emission release conditions that lead to poor dispersion
6 and associated elevations in Pb concentrations) could lead to Pb concentrations exceeding the Pb
7 NAAQS (Cavender, 2008). This analysis included three approaches. The first two of the three
8 approaches included a simple scaling of the historic 5 tpy emission threshold applied to the old
9 1.5 $\mu\text{g}/\text{m}^3$ Pb NAAQS, and a simplified modeling effort using a screening model. The third
10 approach relied on design values based on Pb monitoring data surrounding large sources (1 tpy
11 or greater) of Pb. At the time of the 2008 review, complete 3-year design values were only
12 available for 7 source-monitor pairs

13 As more recent data become available, analysis of the updated and expanded dataset will
14 inform evaluation of the appropriateness of the current threshold. Since the analysis performed
15 during the 2008 review, over 150 additional source-oriented monitors have been installed that
16 may be useful in such an evaluation. At this time, the new monitors have not collected a
17 complete 3-year dataset, which is needed for the development of complete design values for the
18 new source-oriented monitors. For monitors installed in response to the 2008 revisions (near
19 sources estimated to emit 1.0 tpy or more of Pb), 3-years of certified data will be available in
20 spring 2013. Three-years of certified data for the monitors required in the 2010 reconsideration
21 will be available in spring 2015. Accordingly, analysis of the emissions threshold for source-
22 oriented monitors may be an appropriate issue for consideration in future reviews.

23 **One-year Airport Monitoring.** In addition to the above source-oriented monitoring
24 requirement, one year of monitoring was required near 15 specific airports in order to gather
25 additional information on the likelihood of NAAQS exceedances near airports due to the
26 combustion of leaded aviation gasoline (75 FR 81126). This airport monitoring study may
27 provide useful information to assess the need for additional Pb monitoring at airports, and
28 appropriate requirements, such as consideration of the 1.0 tpy emissions threshold and regional
29 administrator authority for siting of source-oriented monitors at airports (75 FR 81131-81132).
30 This airport monitoring study is currently underway. Given delays in monitor siting, monitor
31 installation at some of these sites extended into late 2012. Accordingly, the timing for
32 completion of the year of monitoring will vary across the 15 locations, with all of them projected
33 to finish by late 2013. The completed datasets, when available and certified, will inform EPA's
34 considerations on next steps with regard to monitoring at airports using leaded aviation gasoline.
35 Also, as described further in section 2.1.2.2 above, the airport monitoring data will also inform
36 EPA's ongoing investigation into the potential for lead emissions from piston-engine aircraft to

1 cause or contribute to air pollution that may reasonably be anticipated to endanger public health
2 or welfare. This investigation is occurring under Section 231 of the Clean Air Act (CAA),
3 separate from the lead NAAQS review.

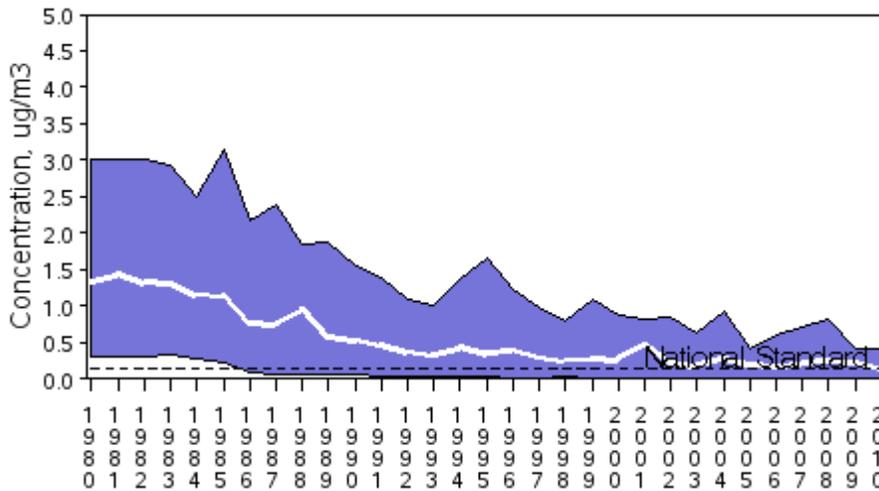
4 **Non-Source-Oriented Monitoring.** Monitoring agencies are also required to conduct
5 non-source-oriented monitoring at NCore sites with a population of 500,000 or more, as noted
6 above.²⁰ Currently, there are 24 NCore sites measuring Pb concentrations, with 17 measuring Pb
7 in TSP and 7 measuring Pb in PM₁₀. While non-source-oriented monitoring data can be used for
8 designation purposes, an alternative objective stated for these sites is the collection of data on
9 neighborhood-scale Pb concentrations that are typical in urban areas to inform our understanding
10 of ambient air-related Pb exposures for the general population. At this still early stage in data
11 collection at these sites, we have not identified specific gaps or inadequacies. Thus, at this stage,
12 the current requirement for non-source-oriented Pb monitoring appears to be adequate for these
13 purposes.
14

²⁰ Defined by the US Census Bureau - <http://www.census.gov/population/www/metroareas/metroarea.html>

1 **2.2.2 Ambient Concentrations**

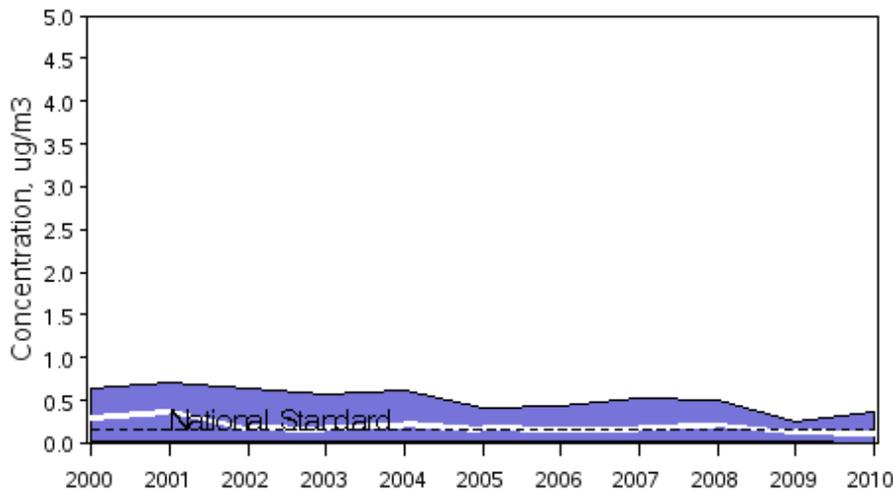
2 **2.2.2.1 Temporal Trends**

3 Ambient air concentrations of Pb in the U.S. have declined substantially over the past 30
4 years. Figure 2-7 illustrates this decline in terms of site-specific maximum 3-month average
5 concentrations at the set of 31 monitoring sites that have been operating across this period. The
6 median of this dataset has declined by more than 90% over the 30-year period, and the average
7 by 89%. Over the past 10 years, a larger dataset of 92 sites operating across that period also
8 indicates a decline, which is on the order of 60% for the average of that dataset (Figure 2-8).²¹



9
10 **Note:** Based on annual maximum 3-month average at 31 sites.

11 **Figure 2-7. Temporal trend in Pb -TSP concentrations: 1980-2010 (31 sites).**

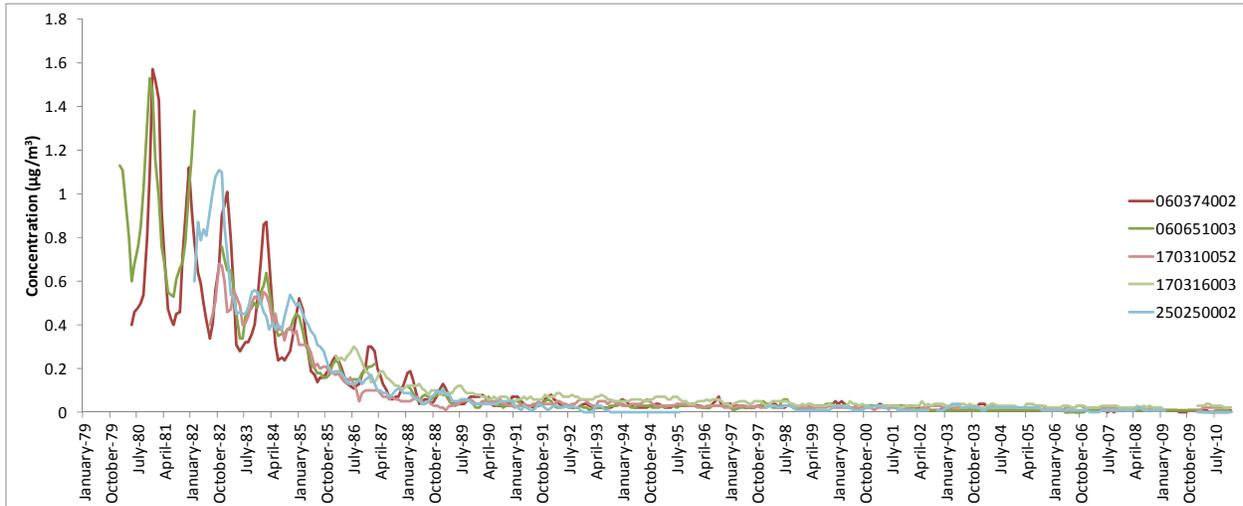


12
13 **Note:** Based on annual maximum 3-month average at 92 sites.

14 **Figure 2-8. Temporal trend in Pb-TSP concentrations: 2000-2010 (92 sites).**

²¹ <http://www.epa.gov/air/airtrends/lead.html>

1 The role of the phase-out of leaded gasoline on declining concentrations is evident from
 2 temporal trends in air Pb concentrations near roadways, as illustrated in Figure 2-9 which
 3 presents data for five monitors sited near roadways.²² These sites additionally indicate the
 4 concentrations currently common at such sites, with the maximum 3-month average
 5 concentrations at all five sites falling below 0.03 $\mu\text{g}/\text{m}^3$ in the most recent years.



6
 7 Monitor locations: Los Angeles, CA (06-037-4002), Riverside, CA (06-065-1003), Cook, IL (17-031-0052, 17-031-6003), Suffolk, MA (25-025-0002).

8 **Figure 2-9. Airborne Pb -TSP concentrations (3-month average) at five sites near**
 9 **roadways: 1979-2010.**

10

²² In selecting these sites, the objective was to identify sites near roadways that do not appear to be near other (stationary) sources of Pb emissions. In addition to consideration of information in national emissions inventories, the areas around the sites were examined using satellite pictures (in Google Maps) for signs of current or historical industrial activity.

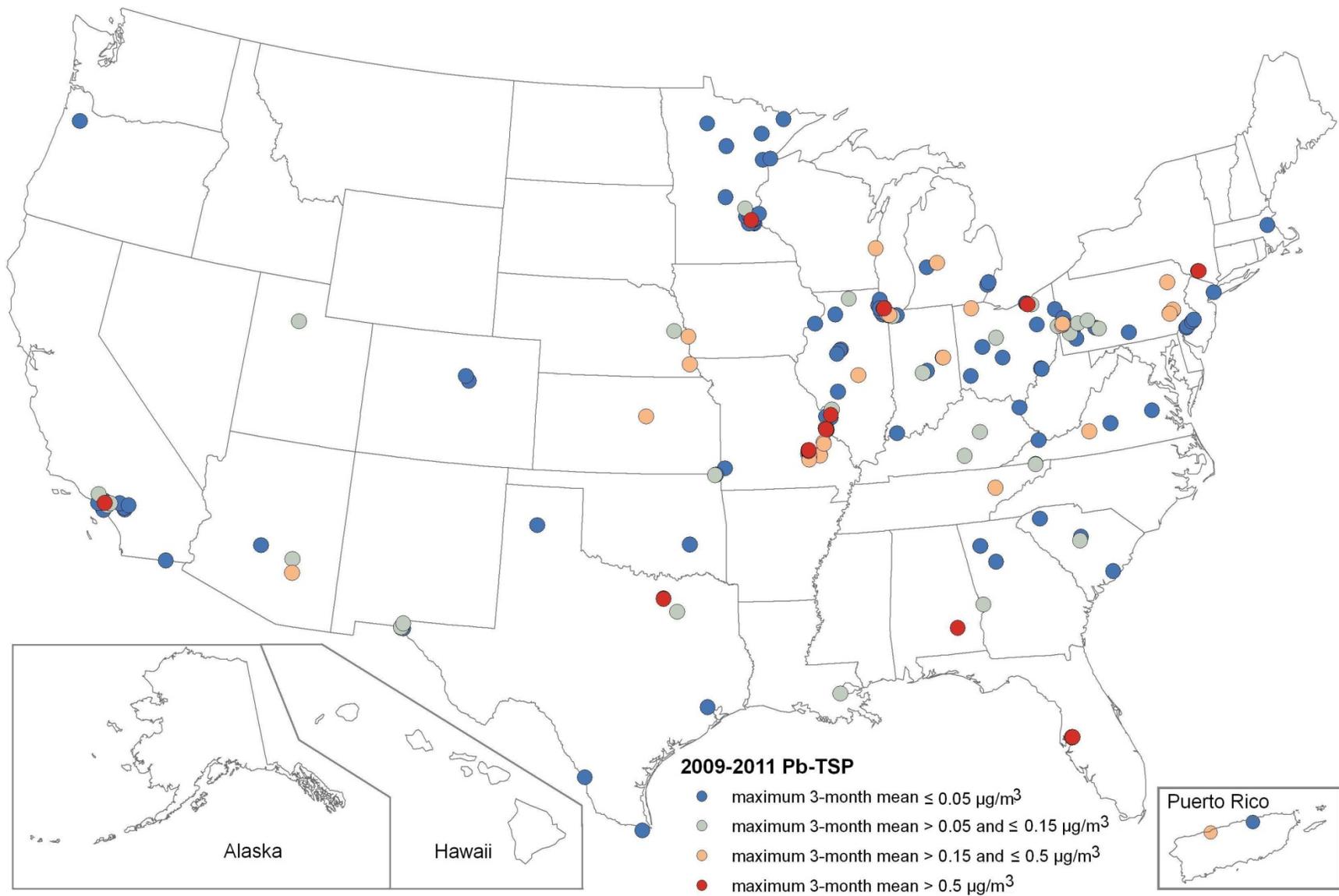
1 **2.2.2.2 Current Concentrations**

2 As a result of revisions to the Pb NAAQS surveillance monitoring requirements
3 (described in section 2.2.1 above), Pb monitoring sites have been in transition over the last few
4 years as indicated by Figure 2-3. For presentation in this document, we have focused on the
5 most recent period for which adequately complete data are available, 2009-2011, recognizing
6 that the dataset developed for this period includes many but not all of the monitors newly
7 required by the December 2010 regulations.

8 Lead concentrations, in terms of maximum 3-month average Pb-TSP concentration, at
9 monitoring sites active across the U.S. during the period 2009-2011, and for which sufficient
10 data are available to meet completeness criteria described in Appendix 2C, are presented in
11 Figure 2-10.²³ Highest concentrations occur in the vicinity of large metals industries, as
12 discussed in section 2.1.2.1.²⁴ As can be seen through comparison of Figure 2-10 with Figure 2-
13 3, only limited data (not meeting completeness criteria for the data analysis) are available to date
14 for some of the monitors newly required by the Pb NAAQS monitoring network revisions
15 completed in 2010 (see section 2.2.1.1).

²³ Criteria for development of the 2009-2011 air Pb-TSP, Pb-PM₁₀ and Pb-PM_{2.5} datasets discussed in this section are described in Appendix 2C. Data summaries are included in Appendix 2D.

²⁴ Information regarding areas of U.S. designated nonattainment with the Pb NAAQS is available at: <http://www.epa.gov/air/oaqps/greenbk/mindex.html>.

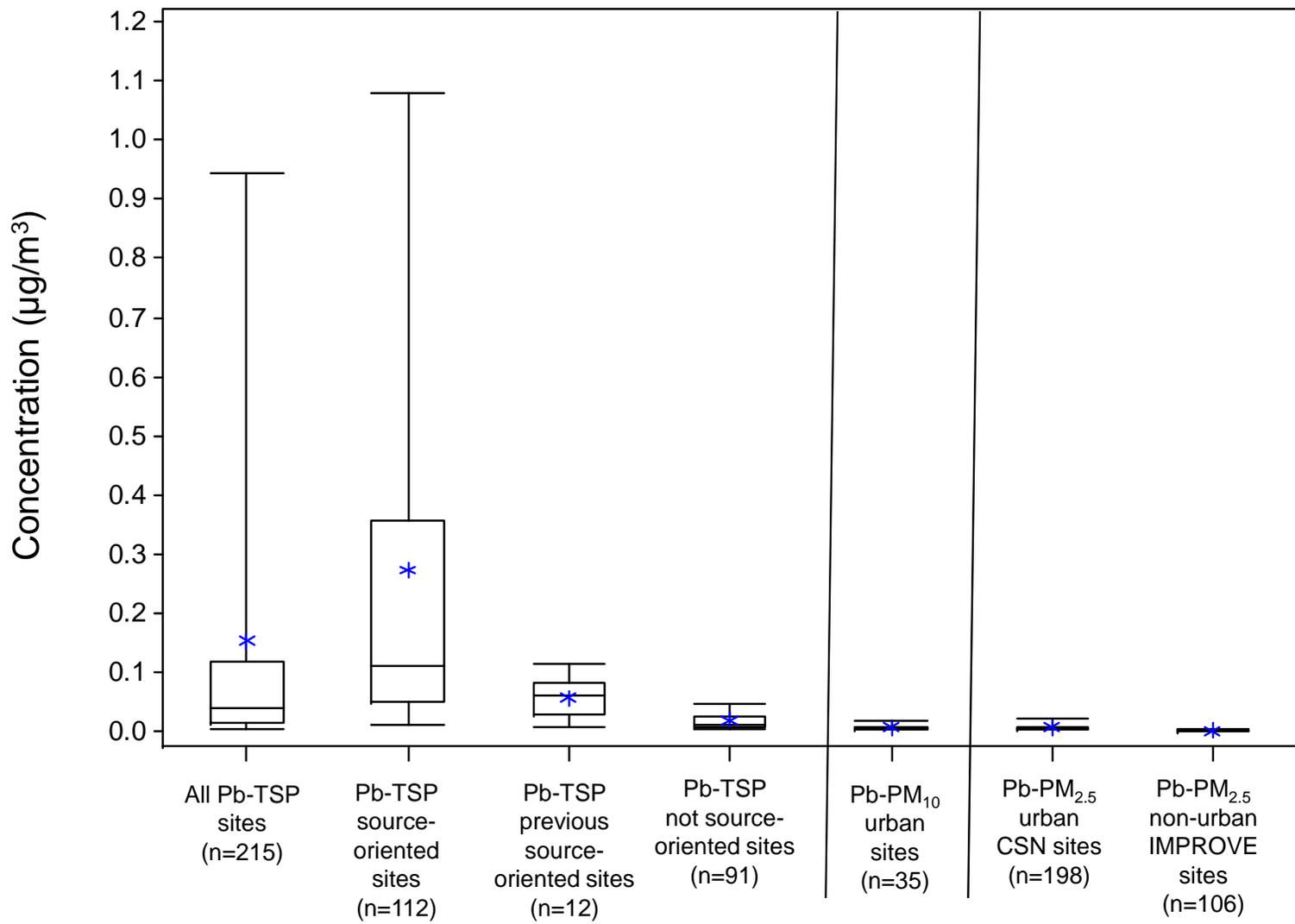


1

2 **Figure 2-10. Pb-TSP maximum 3-month means (215 sites), 2009-2011.**

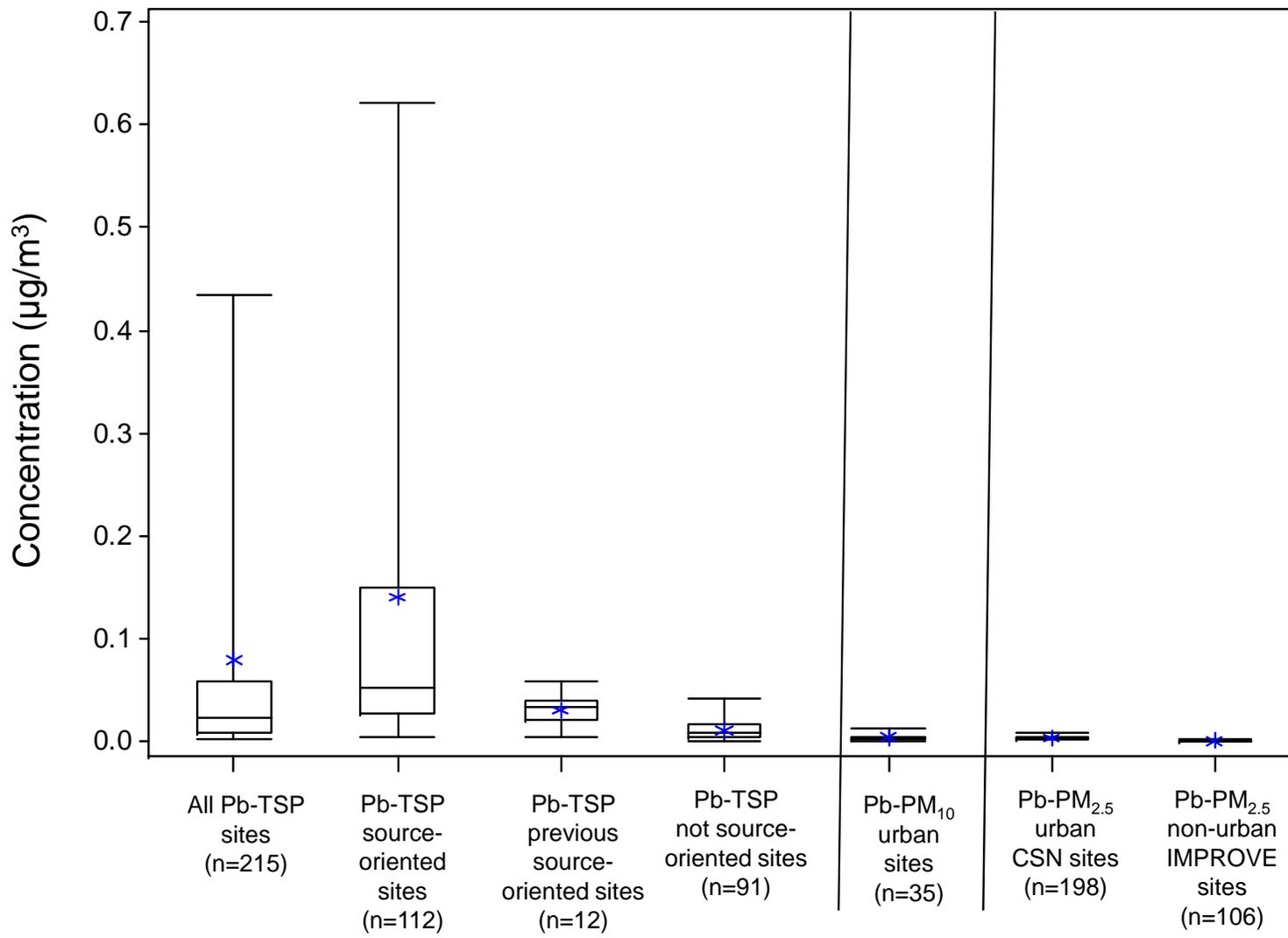
1 Figures 2-11 through 2-13 illustrate differences in Pb-TSP concentrations, in terms of
2 three different metrics (maximum 3-month mean, annual mean, and maximum monthly mean),
3 among sites near and more distant from emissions sources. The Pb-TSP sites indicate the much
4 greater site and temporal variability in concentrations at source-oriented and previous source-
5 oriented sites as compared to non-source-oriented sites. Across the Pb-TSP sites, as would be
6 expected, highest concentrations are observed at the source-oriented sites, followed by the
7 previous source-oriented sites. This is the case for all three metrics analyzed (maximum 3-
8 month mean, maximum monthly mean and annual mean).

9 Figures 2-11 through 2-13 additional indicate distributions of Pb-PM₁₀ and Pb-PM_{2.5}
10 concentrations in urban and rural locations where they are monitored. The Pb-PM₁₀ and Pb-
11 PM_{2.5} networks are described in section 2.2.1.2 and shown in Figures 2-5 and 2-6, respectively. It
12 is important to note that there are few sites in these recent datasets with colocated monitors for
13 the different size fractions. As described in the ISA, at 18 urban sites (without specification as to
14 proximity to Pb sources) with at least 30 co-located Pb-TSP and Pb-PM₁₀ samples collected
15 during various time periods (nearly all between 1990 and 2000), approximately 80% of the Pb
16 mass, on average, is captured by the Pb-PM₁₀ measurements (ISA, sections 3.5.3.1 and 3.8.6).
17 The data distributions in Figures 2-11 through 2-13 indicate reduced variability in concentration
18 for non-source-oriented sites and for particulate Pb of smaller size fractions.



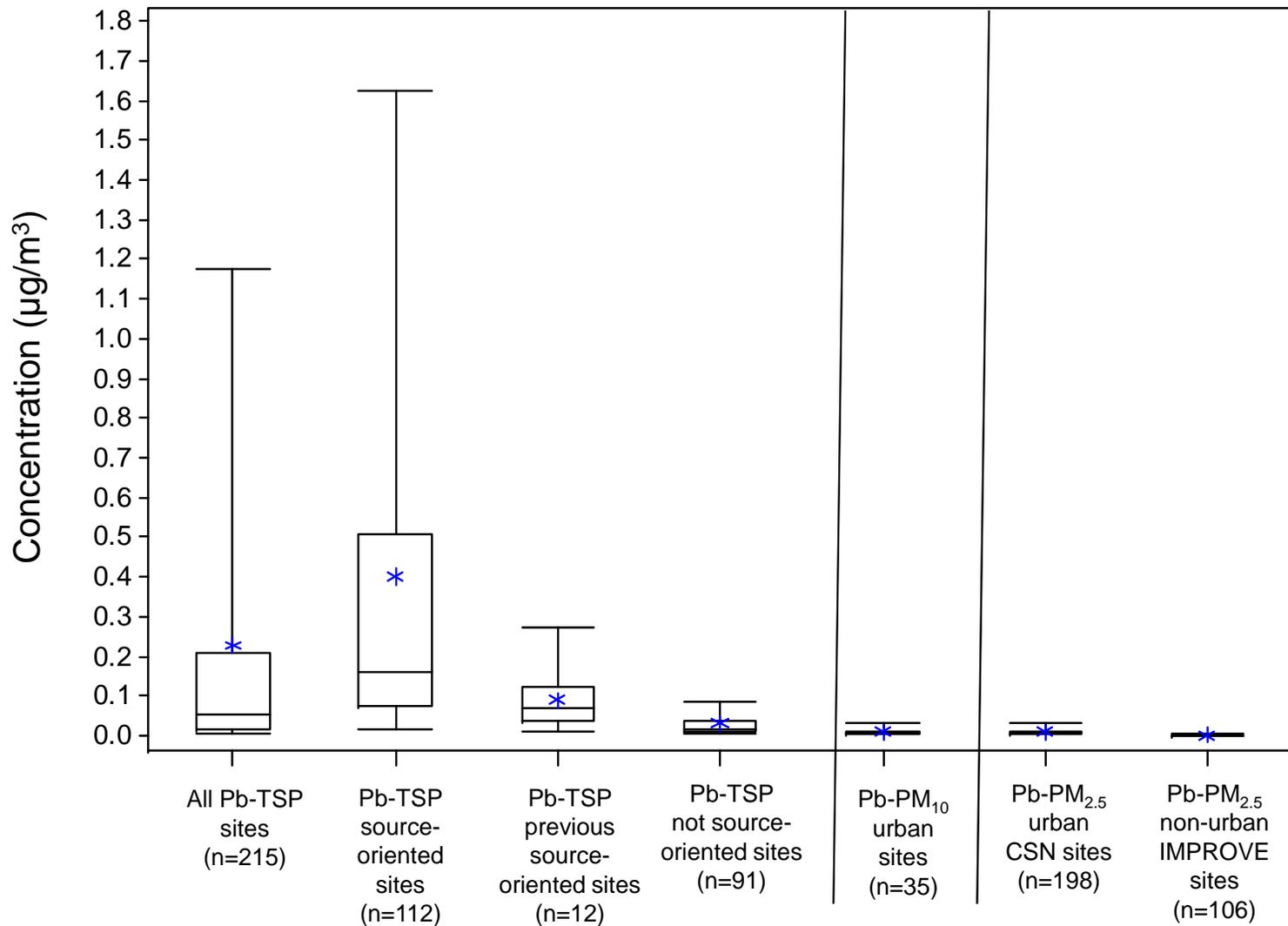
1

2 **Figure 2-11. Distribution of maximum 3-month mean concentrations of Pb-TSP, Pb-PM₁₀ and Pb-PM_{2.5} at different site types,**
 3 **2009-2011.**



1

2 **Figure 2-12. Distribution of annual mean concentrations of Pb-TSP, Pb-PM₁₀ and Pb-PM_{2.5} at different site types, 2009-2011.**



1

2 **Figure 2-13. Distribution of maximum monthly mean concentrations of Pb-TSP, Pb-PM₁₀ and Pb-PM_{2.5} at different site types,**
 3 **2009-2011.**

1 **2.3 AMBIENT AIR LEAD IN OTHER MEDIA**

2 Lead emitted into the ambient air, depending on its chemical and physical characteristics,
3 deposits out of air onto surfaces in the environment. The environmental fate of atmospherically
4 emitted Pb once deposited, is influenced by the type of surface onto which the particles deposit
5 and by the type and activity level of transport forces in that location. Deposited Pb can be
6 incorporated into soil matrices in terrestrial ecosystems or transported (by direct deposition or
7 stormwater runoff) to water bodies and into aquatic sediments, which may serve as aquatic
8 ecosystem sinks. Particles deposited onto impervious surfaces, such as roadway or urban
9 surfaces, are more available for human contact while they remain on such surfaces. For
10 example, they may be available for adherence onto skin or they may be resuspended into the air
11 and inhaled. Precipitation and other natural, as well as human-influenced, processes contribute
12 to the fate of such particles and their potential transport to areas or environments of lesser or
13 greater likelihood for human contact, e.g., transport with surface runoff into nearby water bodies
14 or tracking into nearby houses (ISA, section 3.3).

15 Lead from current and historical air emissions sources, in addition to a range of nonair
16 sources (e.g., land disposal of wastes as well as surface runoff and releases to surface waters),
17 contribute to Pb in outdoor dust, soil and aquatic systems. Because of its persistence, Pb from all
18 of these sources can contribute to media concentrations into the future. The pattern of resulting
19 Pb concentrations changes over time in response to changes in new Pb contributions to the
20 systems (e.g., in types and rates of addition), as well as environmental processes (chemical,
21 biological and physical) particular to each type of media and ecosystem. Accordingly, media
22 and ecosystems differ with regard to Pb concentrations and changes in those concentrations in
23 response to airborne Pb. These differences relate to ecosystem processes as well as Pb source or
24 emissions characteristics and proximity.

25 The initial section below (section 2.3.1) summarizes salient information regarding
26 atmospheric deposition and how it is monitored. Subsequent sections describe the current
27 information related to the presence of ambient air Pb in nonair media and what it indicates
28 regarding impacts of current patterns of Pb in and being emitted to ambient air, including
29 relationships to impacts in the past and to other Pb sources. Section 2.3.2 focuses on terrestrial
30 media, while section 2.3.3 discusses aquatic media.

31 **2.3.1 Atmospheric Deposition**

32 Deposition is the pathway by which Pb particles are removed from the atmosphere and
33 transferred to other environmental media. There are several approaches by which atmospheric
34 deposition, or the transfer of Pb from the atmosphere to soil or water bodies, can be assessed.
35 These include measurements of Pb in rainfall (wet deposition) and on collection surfaces during

1 dry periods (dry deposition); dry deposition is also estimated from measurements of airborne Pb
2 particles coupled with estimates of deposition velocity (see 2006 CD, Section 2.3.2). Less direct
3 approaches include monitoring changes in Pb concentration in various biological or physical
4 media such as lichen or mosses, snowpack, soil and aquatic sediments (ISA, sections 3.6.2, 3.6.4
5 and 3.6.6). Approaches for the latter three media may involve repeated measurements of surface
6 samples over time or core samples coupled with isotope dating. To gain information on
7 atmospheric deposition, unaffected by contributions from direct nonair environmental releases,
8 such studies are generally conducted in somewhat remote areas. As there are currently no
9 nationwide Pb atmospheric deposition monitoring programs, the discussion of atmospheric
10 deposition in this and subsequent sections is drawn from this range of approaches (2006 CD,
11 sections 2.3, 8.2.2 and AX7.1.2.3; ISA, sections 3.3.1.2 and 3.6). Geographic differences in
12 deposition generally relate to differences in the amount and size distribution of airborne Pb
13 particles in that location, as well as meteorology and other site-specific factors. For example, the
14 size of particles, as well as solubility in rainwater, can also influence wet deposition rates (ISA,
15 section 3.3.1.2; 2006 CD, p. 2-59). Factors that particularly influence dry deposition are the
16 level of atmospheric turbulence, especially in the layer nearest the ground, particle size
17 distributions and density, and the nature of the surface itself, such as smooth or rough (2006 CD,
18 pp. 2-55 to 2-57).

19 Evidence for the temporal pattern of U.S. lead deposition generally indicates a peak
20 during the middle twentieth century (e.g., 1940s through 1970s) followed by substantial declines
21 in the latter part of the century (ISA, section 3.3.1; 2006 CD section 2.3.1; Jackson et al., 2004).
22 This pattern reflects the increased use of Pb in the industrial age (including its use in gasoline in
23 the twentieth century) and declines in response to environmental controls on gasoline and metals
24 industries in the latter part of the twentieth century. Studies continue to document declines in
25 atmospheric deposition since the 1980s in areas remote from industrial areas (e.g., Watmough
26 and Dillon, 2007). As would be expected, stationary sources of Pb continue to contribute to
27 relatively higher deposition rates in industrial areas as compared to other areas, although rates in
28 those areas appear to have also declined since the 1970s-80s (ISA, section 3.3.1.2; Sabin and
29 Schiff, 2008).

30 **2.3.2 Terrestrial Media**

31 Lead in the terrestrial media discussed here may originate from current or historical air or
32 other sources. Other sources to these media include historical use of indoor and outdoor leaded
33 paint, more prevalent in areas with older buildings, and the processing of lead-containing
34 materials. Further, the concentrations of Pb in each media, as well as the relative contributions
35 from various types of sources to those concentrations, vary among media and among locations

1 for any one media. Differences among locations in air-related Pb are generally related to the
2 current and historical magnitude of Pb emissions and deposition at that location, as well as
3 location- and media-specific removal factors.

4 **2.3.2.1 Indoor Household Dust**

5 Household dust arises from particulate matter generated indoors and outdoors and Pb in
6 household dust can reflect a combination of these sources. Airborne particles can be transported
7 indoors with ambient air, while particles deposited outdoors from ambient air may be carried in
8 on humans and their clothing or other items transported indoors. Depending on factors such as
9 the proximity of the residence to current or historical metals industries, leaded paint usage, and
10 age of the residence, Pb in indoor residential dust can reflect current or historical atmospheric Pb
11 or nonair-related sources, such as leaded paint. Another indoor source is tobacco smoking (ISA,
12 section 4.1.3.2; Gaitens et al., 2009; 2006 CD, p. 3-15; Mannino et al., 2003). The age of the
13 residence may be an indicator of the potential for the presence of leaded paint and, in areas of
14 significant levels of airborne Pb in the past, such as near mining or smelting industries, may also
15 indicate the potential for the residual presence of historically emitted Pb (ISA, sections 4.1.1 and
16 4.1.3.2; 2006 CD section 3.2.3). A study of households in the Baltimore metropolitan area (in
17 which there are no areas designated non-attainment for the current Pb NAAQS)²⁵ indicated
18 indoor air Pb concentrations to be significantly associated with outdoor air Pb concentrations but
19 did not show a statistically significant relationship of outdoor air Pb with indoor dust Pb, perhaps
20 indicating the greater role of sources other than current airborne concentrations in those
21 households (ISA, 4.1.3.2; Egeghy et al., 2005).

22 As discussed in chapter 4 below, indoor dust is a major pathway for air-related Pb
23 exposure for pre-school children, largely related to prevalent hand-to-mouth behavior at that age.

24 **2.3.2.2 Outdoor Dust in Areas of Human Activity**

25 Lead in particulate matter occurring on outdoor surfaces (outdoor dust) may reflect
26 current or historical Pb emissions, as well as the historical uses of lead in products on buildings
27 and infrastructure in surrounding area. The concentrations of air-related Pb in outdoor dust and
28 the relative contribution of air-related Pb to the total Pb concentrations in outdoor dust varies
29 depending on the location-specific characteristics of air-related (and other) Pb sources. The role
30 of air-related sources in outdoor dust Pb has been documented in areas near industrial sources,
31 such as smelters, where Pb concentrations in outdoor dust have been documented to decline in
32 response to reduced emissions. For example, outdoor dust Pb concentrations in a long-time
33 Canadian smelter town were found to track smelter Pb emissions, as a new smelting technology

²⁵ <http://www.epa.gov/air/oaqps/greenbk/mnc.html>

1 which reduced airborne Pb concentrations by 75% resulted in a 50% reduction in outdoor dust
2 Pb loading rate and concentrations (2006 CD, p. 3-23; Hiltz, 2003).

3 Rates of dry deposition of Pb in large metropolitan areas during the past decade are much
4 lower than those reported for the 1970s (ISA, sections 3.3.1 and 3.6.1; Table 2-4, below). For
5 example, dry deposition of Pb into Los Angeles harbor during 2002-2006 was more than an
6 order of magnitude lower than rates reported for the same location in 1975 (ISA section 3.3.1.2;
7 Sabin and Schiff, 2008; Lim et al., 2006). Across the Los Angeles metropolitan area, average
8 rates generally ranged from 10 to 32 micrograms per square meter per day ($\mu\text{g}/\text{m}^2/\text{day}$) and down
9 to $0.3 \mu\text{g}/\text{m}^2/\text{day}$ for the non-urban site of Malibu during 2002-2003 (Lim et al., 2006). Rates
10 within this range are reported from studies in Manhattan and a non-industrial area of New Jersey,
11 with a somewhat higher rate reported for a major industrial location within the New York City
12 metropolitan area. Although rates have not been reported recently for smelter locations in the
13 U.S., the outdoor dustfall deposition rate reported in a Canadian smelter town after the
14 installation of new technology that reduced average airborne Pb-TSP concentrations to a level
15 still at least twice the current U.S. NAAQS ($0.3 \mu\text{g}/\text{m}^3$ from prior average of $1.1 \mu\text{g}/\text{m}^3$) was
16 several orders of magnitude higher than those reported for New York, Los Angeles and Chicago
17 urban areas (Table 2-4).

18 From the few studies reporting levels of Pb loading or concentration in outdoor dust
19 within the past decade or so, loading to surfaces of pedestrian traffic signals in New York City
20 was on average approximately 15% the loading reported on sidewalks in older, residential areas
21 of downtown Baltimore (e.g., approximately $250 \text{ ug}/\text{ft}^2$ as compared to $1500 \text{ ug}/\text{ft}^2$), an order of
22 magnitude or more below that reported in the long-time Canadian smelter town (Caravanos et al.,
23 2006b; Farfel et al 2005; Hiltz, 2003).

1 **Table 2-3. Dry deposition of lead in large metropolitan areas and in a smelter town.**

Description	Dry Deposition Rate ($\mu\text{g Pb}/\text{m}^2/\text{day}$)	Study
Los Angeles Harbor, June-Nov 2006	14 ^A	Sabin and Schiff, 2008
Los Angeles Harbor, 2002-2003 ^b	15	Lim et al., 2006
Los Angeles Harbor, 1975	300	Sabin and Schiff, 2008
Los Angeles, metropolitan area urban sites, 2002-2003 ^B	10-32	Lim et al., 2006
Los Angeles, metropolitan area, non-urban site Malibu, 2002- 2003 ^B	0.3	"
Los Angeles, I-405, near Westwood (downwind side), spring 2003 ^C	24	Sabin et al., 2006
Los Angeles, I-405, near Westwood (upwind side), spring 2003 ^C	7.3	"
Jersey City, NJ, 2001- 2002	50	Yi et al., 2006
New Brunswick, NJ, 2001- 2002	9	"
Manhattan, NYC, 2 nd story rooftop (unprotected), 2003-2005	22	Caravanos et al., 2006a
Chicago, 1993-95 (most recent available)	38-71	Yi et al 2001; Paode et al., 1998
Trail, British Columbia, Canada, historical smelter town, 1999 ^D	31000	Hilts, 2003
A - Rates for rest of off-shore transect from Santa Barbara to San Diego Bay ranged from 0.52 (Oxnard) – 3.3 (San Diego Bay) $\mu\text{g}/\text{m}^3$. B - Average Pb-TSP range= 0.0056-0.017 $\mu\text{g}/\text{m}^3$ for urban sites; 0.0022 at Malibu site. C - Average Pb-TSP at downwind site over sampling period ~0.02 $\mu\text{g}/\text{m}^3$. D - Average Pb-TSP = 0.3 $\mu\text{g}/\text{m}^3$, this reflects new technology (previously 1.1 $\mu\text{g}/\text{m}^3$) for smelter that existed in this town for past 95 years.		

2

3 **2.3.2.3 Soil**

4 As is the case for Pb in outdoor dust, occurring in surface soils can be derived from
 5 current or historical Pb emissions, as well as leaded paint usage in older residential areas. The
 6 relative role of these sources may vary with the type of environment and proximity to industry
 7 and population centers.

8 In forested areas away from old urban areas, the role of leaded gasoline in soil
 9 contamination is illustrated by the documented reductions in Pb in surface soils subsequent to the
 10 leaded gasoline phasout. For example, forest surface soil (litter) concentrations in a transect
 11 from Vermont, through Maine and up to Gaspe in Quebec, which in 1979 exhibited a significant
 12 spatial trend, ranging from 200 milligrams per kilogram, dry weight (mg/kg dw) at its
 13 southernmost point down to 60 mg/kg dw at its northernmost and most remote point in Quebec,
 14 declined to 32-66 mg/kg dw (with no spatial trend) in 1996 (ISA, section 3.6.1; Evans, 2005). In
 15 forests from the mid-Atlantic to southern New England, a reduction in litter Pb concentration
 16 was observed between 1978 and 2004-05 (ISA, section 3.6.1; Johnson and Richter, 2010). In the
 17 latter study, the authors observed less change in concentration in the more northern sampling
 18 sites, which they attribute to reduced rates of organic matter decomposition in those colder
 19 temperature areas. Similarly, an additional New England study by Kaste et al (2006) also

1 documented a pattern in temporal reductions in soil Pb (o-horizon) related to decomposition
2 activity. These recent findings are generally consistent with findings reported in the last review
3 which indicated the gradual migration of deposited Pb into mineral soils (e.g., Miller and
4 Friedland, 1994; Kaste et al., 2003; Wang and Benoit, 1997; Johnson et al., 1995; Zhang, 2003).

5 Few studies have investigated temporal trends of surface soil Pb concentrations in more
6 populated areas in relation to reductions in usage of leaded gasoline and paint. Current
7 concentrations in areas of past heavy traffic powered by leaded gasoline are generally elevated
8 above areas more distant from these areas (ISA, section 3.6.1). Current roadway-related sources
9 of Pb, while substantially less significant than leaded gasoline, continue to provide some
10 contribution to surface dust/soil in these areas (ISA, section 3.2.2.6). Surface soil and dust
11 concentrations are much higher in such areas in large, older cities than surrounding suburban
12 areas (ISA, section 3.6.1). In older residential areas, the use of leaded house paint is another
13 contributor to surface soil concentrations (ISA, sections 3.6.1 and 3.7.7.2; Yesilonis et al., 2008;
14 Brown et al., 2008; Clark et al., 2006).

15 Areas of long-term Pb emissions from point sources appear to be the areas of highest
16 surface soil Pb concentrations (ISA, sections 3.6 and 3.6.1). For example, Pb surface soil
17 concentrations within approximately 100-250 meters of long-established lead smelters have been
18 five to ten times higher than those at 3-5 km distance (ISA, section 3.6.1; 2006 CD, Table 3-4).
19 Surface soil concentrations near U.S. mines that are no longer active have also been found to be
20 elevated above more distant areas (2006 CD, Table 3-6). Information described in the 2006 CD
21 for areas surrounding smelters after implementation of pollution controls, although showing
22 declines in Pb concentrations in outdoor dustfall, street dust and indoor dustfall, has not indicated
23 a noticeable decline in soil Pb concentrations (2006 CD, pp. 3-23 to 3-24). The continued Pb
24 emissions in such industrial areas likely influence the dynamics of Pb concentrations in the soil,
25 affecting any response to emissions reductions. Estimates of associated steady-state surface soil
26 Pb concentrations or the expected longer-term temporal pattern for this situation have not been
27 made.

28 In summary, findings to date indicate that many of those systems less influenced by
29 current point sources have and may still be responding to reduced Pb deposition rates associated
30 with reduced atmospheric emissions of Pb, including those associated with the phase-out of
31 leaded gasoline, while potential responses of soils near point sources and those involving
32 historically deposited Pb near roadways are less well characterized.

33 **2.3.2.4 Biota**

34 Measurements of Pb in some biota indicate reductions in biologically available Pb over
35 the past 30 years in some remote locations. For example, measurements of Pb in lichen from

1 Golden Lake in Mount Ranier National Park (2005) and Emerald Lake basin in Sequoia/Kings
2 Canyon National Parks (2004) indicate significant reductions (approximately 3-5-fold) since
3 samples were previously collected in those locations in 1984 (ISA, section 3.6.6; Landers et al.,
4 2008). Further, Pb in teeth of juvenile and adult moose in Isle Royale National Park in northern
5 Michigan have also declined substantially (ISA, section 3.6.8; Vucetich et al., 2009).

6 Biota measurements have also documented sources of Pb deposition in mining areas
7 including the transportation of materials along haul roads. Lead deposition from these mining-
8 related activities has been documented in remote wilderness areas in Alaska. For example, Pb
9 concentration in moss in Cape Krusenstern National Monument declines with distance from the
10 road on which product from Red Dog mine²⁶ in northwest Alaska is transported (ISA, section
11 3.3.1.2; Hasselbach et al., 2005). In summer 2001, the median Pb concentration measured within
12 10 meters of the haul road was more than 20 times higher than sites at greater distance from the
13 road and nearly three orders of magnitude greater than those reported for this moss in other
14 Alaska locations in 1990-1992. Concentrations in moss within 10 meters of the haul road ranged
15 from 271-912 mg/kg dw (Hasselbach et al., 2005).

16 **2.3.3 Aquatic Media**

17 **2.3.3.1 Surface Waters**

18 In addition to delivery by atmospheric deposition, Pb is also carried into surface waters
19 via wastewater effluent from municipalities and industry, stormwater runoff, erosion, and
20 accidental discharges (2006 CD, p. AX7-142; ISA section 3.3.2). As a result of the phasing out
21 of leaded gasoline, reductions in Pb concentrations have been documented in surface waters of
22 the North Atlantic Ocean, as well as the relatively less remote areas of the Great Lakes (2006
23 CD, p. 7-23). The availability of studies investigating historical trends in surface waters is
24 limited, in part due to analytical issues that challenged many monitoring programs in the past
25 (2006 CD, AX7.2.2.2). Thus, temporal trends reported in many aquatic systems are based on
26 sediment analyses (see section 2.3.3.2 below).

27 Most Pb occurring in aquatic systems is associated with particles, with the distribution
28 between particle-bound and dissolved form being influenced by water chemistry as well as
29 suspended sediment levels (ISA, 3.3.2; 2006 CD, pp. AX7-117 to AX7-118, Section AX7.2.2).
30 Water columns have been described as “transient reservoirs” for pollutants (ISA, section 3.7.2;
31 2006 CD, p. 2-75). Once deposited to sediments, whether Pb is available for resuspension back
32 into the water column with potential transport further down a watershed versus being buried into
33 deeper sediments depends on the aquatic system. In open ocean waters (generally characterized

²⁶ Red Dog mine is a zinc and Pb mine initiated in 1987. The materials are transported from the mine to a port on the Chukchi Sea via a 52-mile long haul road.

1 by depth and distance from continental sources), resuspension to surface waters is unlikely. In
2 more shallow systems, and additionally those influenced by land sources (e.g., stormwater runoff
3 as well as point sources), resuspension may play a role in water column concentrations. For
4 example, studies in San Francisco Bay, the southern arm of which has an average depth of 2 m,
5 have indicated that Pb particles may be remobilized from surface sediments into the water
6 column (2006 CD, AX7-141).

7 The distribution of Pb dissolved in many U.S. surface waters has been reported by the
8 United States Geological Survey (USGS) National Water-Quality Assessment (NAWQA)
9 program. The NAWQA data set encompasses data, collected from 1991-2003 on Pb
10 concentrations in flowing surface waters for more than 50 river basins and aquifers throughout
11 the country (2006 CD, Section AX7.2.2.3). These data indicate a mean dissolved Pb
12 concentration in U.S. surface waters of 0.66 µg/L (range 0.04 to 30 µg/L) in waters affected by a
13 combined contribution of natural and anthropogenic sources, as compared to a mean of 0.52
14 µg/L (range 0.04 to 8.4 µg/L) for waters in “forest”, “rangeland”, and “reference” sites (2006
15 CD, Section AX7.2.2.3). The highest surface water Pb concentrations were observed in sites
16 impacted by land uses such as mining (2006 CD, p. AX7-131). The role of surface runoff in
17 delivering contamination to waters near metals industries presents a challenge to the
18 disentangling of atmospheric deposition contributions from those associated with surface runoff.

19 **2.3.3.2 Sediments**

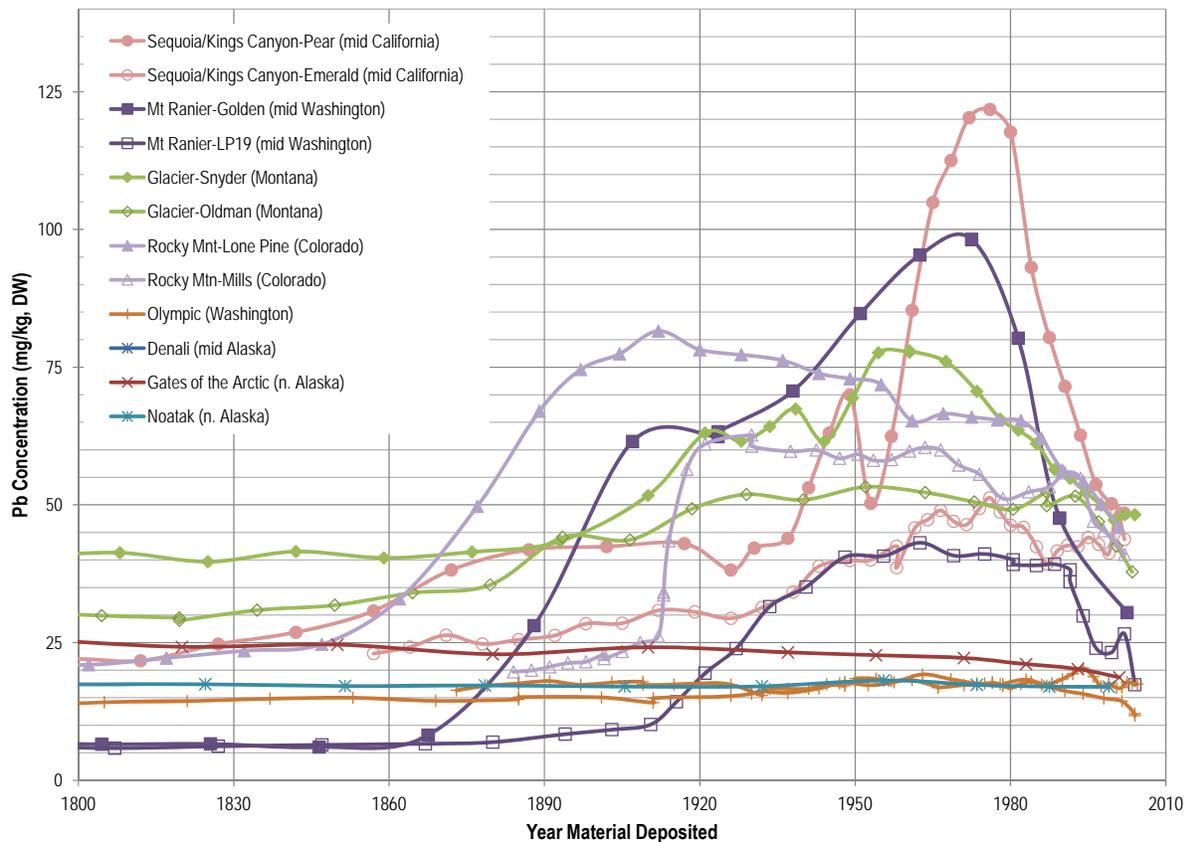
20 Many studies have investigated temporal trends in sediment Pb concentrations, using
21 sediment cores or surface sediment Pb concentration, with declines documented in many systems
22 and usually attributed to the phasing out of leaded gasoline (ISA, section 3.6.2; 2006 CD, section
23 AX7.2.2). Several studies documenting the increased Pb deposition of the industrial age,
24 including specifically leaded gasoline usage, and the subsequent declines associated with leaded
25 gasoline phase-out were reported in the 2006 CD. They include investigations involving
26 sediment cores from the Okefenokee Swamp in Georgia as well as from 35 reservoirs and lakes
27 in urban and reference locations (ISA, section 3.6.2; 2006 CD, p. AX7-141). In the latter, the
28 median reduction in Pb mass accumulation rate in the cores, adjusted for background
29 concentrations, was 246%, with the largest decreases in lakes located in dense urban watersheds
30 (ISA, section 3.6.2; 2006 CD, p. AX7-141; Mahler et al, 2006). A third study of sediment cores
31 in 12 lakes in the Great Lakes area also documented a peak in Pb concentrations consistent with
32 peak use of leaded gasoline in the U.S. in the mid 1970’s and declining concentrations in most
33 lake sediments through the mid 1990’s (2006 CD, p. 2-55; Yohn et al., 2004). Sediment surveys
34 by the USGS NAWQA have reported the highest Pb concentrations in Idaho, Utah, and

1 Colorado, with seven of the highest concentrations at sites classified as mining land use (2006
2 CD, p. AX7-133).

3 Among the more recent investigation of temporal trends in aquatic sediments described in
4 the ISA is that associated with the Western Airborne Contaminants Assessment Project which
5 documented Pb in sediment cores from 14 lakes in eight U.S. national parks in western states,
6 including three in Alaska (ISA, section 3.6.2; Landers et al., 2010). Among the Alaska cores, in
7 which concentrations were generally on the order of 20 $\mu\text{g}/\text{kg}$, dw, there was little variation in Pb
8 concentration, flux or enrichment factor. The other park cores, with few exceptions, generally
9 exhibited an increase in concentration commencing in the mid-nineteenth century, which
10 transitioned to declining trends in the past few decades, with lower concentrations in more
11 recent, surface material. The highest concentration was recorded at core depth corresponding to
12 the mid-1970s, in one of the Sequoia/Kings Canyon lake cores (Figure 2-16; ISA, section 3.6.2;
13 Landers et al., 2010²⁷).

14

²⁷ Figure created from database for Landers et al (1980);
http://www.nature.nps.gov/air/Studies/air_toxics/wacap.cfm.



1
 2 **Figure 2-14. Temporal trend in sediment concentration from core samples in 12 lakes at**
 3 **eight National Parks or Preserves.**

4 Analyses of cores taken in several lakes and reservoirs along the Apalachicola,
 5 Chattahoochee, and Flint River Basin from north of the Atlanta, GA metropolitan area to the
 6 Gulf of Mexico indicate changes in sediment concentration both with time and in relation to
 7 influence of the large metropolitan area of Atlanta (ISA, section 3.6.2; Callender and Rice,
 8 2000). Highest concentrations were documented just downstream from Atlanta at the core depth
 9 corresponding to the 1975-1980 time period; the corresponding concentration for the most
 10 recent time period (1990-1995) at that location was approximately 50% lower (ISA, section
 11 3.6.2; Callender and Rice, 2000). These data may reflect changes in surface water discharges of
 12 Pb in the Atlanta area, during this time period, as well as changes in air deposition. The smaller
 13 reduction (on the order of 20%) observed across the 20 year time period at the upstream site is
 14 likely more generally reflective of changes in air deposition.

1 2.4 REFERENCES

- 2 Brown, RW; Gonzales, C; Hooper, MJ; Bayat, AC; Fornerette, AM; McBride, TJ; Longoria, T; Mielke, HW.
3 (2008). Soil lead (Pb) in residential transects through Lubbock, Texas: A preliminary assessment. Environ
4 Geochem Health 30: 541-547. <http://dx.doi.org/10.1007/s10653-008-9180-y>.
- 5 Callender, E; Rice, KC. (2000). The urban environmental gradient: Anthropogenic influences on the spatial and
6 temporal distributions of lead and zinc in sediments. Environ Sci Technol 34: 232-238.
7 <http://dx.doi.org/10.1021/es990380s>.
- 8 Caravanos, J.; Weiss, A.L.; Jaeger, R.J. (2006a) An exterior and interior leaded dust deposition survey in New York
9 City: results of a 2-year study. Environ. Res. 100: 159-164.
- 10 Caravanos, J; Weiss, AL; Blaise, MJ; Jaeger, RJ. (2006b). A survey of spatially distributed exterior dust lead
11 loadings in New York City. Environ Res 100: 165-172. <http://dx.doi.org/10.1016/j.envres.2005.05.001>.
- 12 Cavender, K. (2008). Update of Analysis of Proposed Source-Oriented Monitoring Emission Threshold.
13 Memorandum to Lead NAAQS Review Docket (OAQ-2006-0735)
14 <http://www.epa.gov/ttnaaqs/standards/pb/data/20081015Cavender.pdf>
- 15 Clark, HF; Brabander, DJ; Erdil, RM. (2006). Sources, sinks, and exposure pathways of lead in urban garden soil. J
16 Environ Qual 35: 2066-2074. <http://dx.doi.org/10.2134/jeq2005.0464>.
- 17 DRRC. (Doe Run Company). (2010). DRRC multi-media Consent Decree. Herculaneum, MO: Doe Run Resources
18 Corporation.
- 19 Egeghy, P. P.; Quackenboss, J. J.; Catlin, S.; Ryan, P. B. (2005) Determinants of temporal variability in
20 NHEXAS Maryland environmental concentrations, exposures, and biomarkers. J. Exposure Anal. Environ.
21 Epidemiol. 15: 388-397.
- 22 Evans, GC; Norton, SA; Fernandez, IJ; Kahl, JS; Hanson, D. (2005). Changes in concentrations of major elements
23 and trace metals in northeastern US-Canadian sub-alpine forest floors. Water Air Soil Pollut 163: 245-267.
- 24 Farfel, MR; Orlova, AO; Lees, PSJ; Rohde, C; Ashley, PJ; Chisolm, JJ, Jr. (2005). A study of urban housing
25 demolition as a source of lead in ambient dust on sidewalks, streets, and alleys. Environ Res 99: 204-213.
26 <http://dx.doi.org/10.1016/j.envres.2004.10.005>.
- 27 Gaitens, JM; Dixon, SL; Jacobs, DE; Nagaraja, J; Strauss, W; Wilson, JW; Ashley, PJ. (2009). Exposure of US
28 children to residential dust lead, 1999-2004: I. Housing and demographic factors. Environ Health Perspect
29 117: 461-467. <http://dx.doi.org/10.1289/ehp.11917>.
- 30 Garland, JA, Nicholson, KW 1991. A review of methods for sampling large airborne particles and associated
31 radioactivity. J. Aerosol Sci. Vol.22 (4):479-499.
- 32 Hasselbach, L; Ver Hoef, JM; Ford, J; Neitlich, P; Crecelius, E; Berryman, S; Wolk, B; Bohle, T. (2005). Spatial
33 patterns of cadmium and lead deposition on and adjacent to National Park Service lands in the vicinity of
34 Red Dog Mine, Alaska. Sci Total Environ 348: 211-230. <http://dx.doi.org/10.1016/j.scitotenv.2004.12.084>.
- 35 Hilts, S. R. (2003) Effect of smelter emission reductions on children's blood lead levels. Sci. Total Environ. 303: 51-
36 58.
- 37 Jackson, B.P., P.V. Winger, P.J. Lasier (2004) Atmospheric lead deposition to Okefenokee Swamp, Georgia, USA.
38 Environ Poll. 130: 445-451.

- 1 Johnson, C. E.; Siccama, T. G.; Driscoll, C. T.; Likens, G. E.; Moeller, R. E. (1995) Changes in lead
2 biogeochemistry in response to decreasing atmospheric inputs. *Ecol. Appl.* 5: 813-822.
- 3 Johnson, AH; Richter, SL. (2010). Organic-horizon lead, copper, and zinc contents of Mid-Atlantic forest soils,
4 1978-2004. *Soil Sci Soc Am J* 74: 1001-1009. <http://dx.doi.org/10.2136/sssaj2008.0337>.
- 5 Kaste, JM; Bostick, BC; Friedland, AJ; Schroth, AW; Siccama, TG. (2006). Fate and speciation of gasoline-derived
6 lead in organic horizons of the northeastern USA. *Soil Sci Soc Am J* 70: 1688-1698.
7 <http://dx.doi.org/10.2136/sssaj2005.0321>.
- 8 Kaste, J.; Friedland, A.; Stürup, S. (2003) Using stable and radioactive isotopes to trace atmospherically deposited
9 Pb in montane forest soils. *Environ. Sci. Technol.* 37: 3560-3567.
- 10 Kenny L; Beaumont G, Gudmundsson A, Thorpe A, Koch W. (2005) Aspiration and sampling efficiencies of the
11 TSP and louvered particulate matter inlets. *J Environ Monit.* May 2005. 7 481-487.
- 12 Landers, DH; Simonich, SL; Jaffe, DA; Geiser, LH; Campbell, DH; Schwindt, AR; Schreck, CB; Kent, ML; Hafner,
13 WD; Taylor, HE; Hageman, KJ; Usenko, S; Ackerman, LK; Schrlau, JE; Rose, NL; Blett, TF; Erway, MM.
14 (2008). The fate, transport, and ecological impacts of airborne contaminants in western national parks
15 (USA). (EPA/600/R-07/138). Corvallis, Oregon: U.S. Environmental Protection Agency, NHEERL,
16 Western Ecology Division. http://www.nature.nps.gov/air/studies/air_toxics/WACAPreport.cfm.
- 17 Landers, DH; Simonich, SM; Jaffe, D; Geiser, L; Campbell, DH; Schwindt, A; Schreck, C; Kent, M; Hafner, W;
18 Taylor, HE; Hageman, K; Usenko, S; Ackerman, L; Schrlau, J; Rose, N; Blett, T; Erway, MM. (2010). The
19 Western Airborne Contaminant Assessment Project (WACAP): An interdisciplinary evaluation of the
20 impacts of airborne contaminants in western U.S. National Parks. *Environ Sci Technol* 44: 855-859.
21 <http://dx.doi.org/10.1021/es901866e>.
- 22 Lim, JH; Sabin, LD; Schiff, KC; Stolzenbach, KD. (2006). Concentration, size distribution, and dry deposition rate
23 of particle-associated metals in the Los Angeles region. *Atmos Environ* 40: 7810-7823.
24 <http://dx.doi.org/10.1016/j.atmosenv.2006.07.025>.
- 25 Mahler, B. J.; Van Metre, P. C.; Callender, E. (2006) Trends in metals in urban and reference lake sediments across
26 the United States, 1970 to 2001. *Environ. Toxicol. Chem.* 25: 1698-1709.
- 27 Mannino, D.M.; Albalak, R.; Grosse, S.; Repace, J. (2003) Second-hand smoke exposure and blood lead levels in
28 U.S. children. *Epidemiology* 14(6): 719-727.
- 29 Miller, E. K.; Friedland, A. J. (1994) Lead migration in forest soils: response to changing atmospheric inputs.
30 *Environ. Sci. Technol.* 28: 662-669.
- 31 Osterberg, E; Mayewski, P; Kreutz, K; Fisher, D; Handley, M; Sneed, S; Zdanowicz, C; Zheng, J; Demuth, M;
32 Waskiewicz, M; Bourgeois, J. (2008). Ice core record of rising lead pollution in the North Pacific
33 atmosphere. *Geophys Res Lett* 35: L05810. <http://dx.doi.org/10.1029/2007gl032680>
- 34 Paode, RD; Sofuoglu, SC; Sivadechathep, J; Noll, KE; Holsen, TM; Keeler, GJ. (1998). Dry deposition fluxes and
35 mass size distributions of Pb, Cu, and Zn measured in southern Lake Michigan during AEOLOS. *Environ*
36 *Sci Technol* 32: 16291635. <http://dx.doi.org/10.1021/es970892b>.
- 37 Russell A. and Samet, J.M. (2010) Letter from Dr. A. Russell, Chair, Clean Air Scientific Advisory Committee,
38 Ambient Air Monitoring Methods Subcommittee and J.M. Samet, Chair, Clean Air Scientific Advisory
39 Committee, to Administrator Lisa P. Jackson. Re: CASAC Review of EPA's White Paper Approach for
40 the Development of a new Federal Reference Method (FROM) for Lead in Total Suspended Particulates
41 (Pb-TSP). November 30, 2010.

1 Sabin, LD; Schiff, KC. (2008). Dry atmospheric deposition rates of metals along a coastal transect in southern
2 California. *Atmos Environ* 42: 6606-6613. <http://dx.doi.org/10.1016/j.atmosenv.2008.04.042>

3 U.S. Environmental Protection Agency. (1996-2011) . AP-42, Compilation of Air Pollutant Emission Factors, 5th
4 Edition. Volume 1: Stationary Point and Area Sources, Chapter 13: Miscellaneous Sources. Available at:
5 <http://www.epa.gov/ttn/chief/ap42/ch13/index.html>

6 U.S. Environmental Protection Agency. (2012) National Emissions Inventory for 2008, version 3. Office of Air
7 Quality Planning and Standards, Research Triangle Park, NC. November 2012.
8 <http://www.epa.gov/ttn/chief/net/2008inventory.html>

9 Vucetich, JA; Outridge, PM; Peterson, RO; Eide, R; Isrenn, R. (2009). Mercury, lead and lead isotope ratios in the
10 teeth of moose (*Alces alces*) from Isle Royale, U.S. Upper Midwest, from 1952 to 2002. *J Environ Monit.*

11 Wang, E. X.; Benoit, G. (1997) Fate and transport of contaminant lead in spodosols: a simple box model analysis.
12 *Water Air Soil Pollut.* 95: 381-397.

13 Watmough, SA; Dillon, PJ. (2007). Lead biogeochemistry in a central Ontario forested watershed. *Biogeochemistry*
14 84: 143-159. <http://dx.doi.org/10.1007/s10533-007-9110-6>

15 Yesilonis, ID; Pouyat, RV; Neerchal, NK. (2008). Spatial distribution of metals in soils in Baltimore, Maryland:
16 Role of native parent material, proximity to major roads, housing age and screening guidelines. *Environ*
17 *Pollut* 156: 723-731. <http://dx.doi.org/10.1016/j.envpol.2008.06.010>.

18 Yi, SM; Totten, LA; Thota, S; Yan, S; Offenberg, JH; Eisenreich, SJ; Graney, J; Holsen, TM. (2006). Atmospheric
19 dry deposition of trace elements measured around the urban and industrially impacted NY-NJ harbor.
20 *Atmos Environ* 40: 6626-6637. <http://dx.doi.org/10.1016/j.atmosenv.2006.05.062>.

21 Yohn, S., Long, D., Fett, J., Patino, L. (2004) Regional versus local influences on lead and cadmium loading to the
22 Great Lakes region. *Appl. Geochem.* 19: 1157-1175.

23 Zhang, Y.-H. (2003) 100 years of Pb deposition and transport in soils in Champaign, Illinois, U.S.A. *Water Air Soil*
24 *Pollut.* 146: 197-210.

3 HEALTH EFFECTS AND EXPOSURE/RISK INFORMATION

This chapter presents key aspects of the current evidence of lead-related health effects and presents exposure/risk information from the quantitative assessment performed in the last review in the context of the currently available evidence. Staff has drawn from EPA's synthesis of the scientific evidence presented in the draft *Integrated Science Assessment for Lead* (USEPA, 2012; henceforth referred to as the ISA) and 2006 *Air Quality Criteria Document for Lead* (USEPA, 2006; henceforth referred to as the 2006 CD), and from the documentation of the 2007 human exposure and health risk assessment (documented in USEPA, 2007a; henceforth referred to as the 2007 REA). The chapter is organized into sections considering the information on blood Pb as a biomarker (section 3.1), the nature of Pb effects on health (section 3.2), public health implications and at-risk populations (section 3.3), and exposure and risk information (section 3.4). Presentation within these sections is organized to address key policy-relevant questions for this review concerning the evidence and exposure/risk information, building upon the questions included in the IRP (IRP, section 3.1).

3.1 INTERNAL DISPOSITION AND BIOMARKERS OF EXPOSURE AND DOSE

The health effects of Pb, discussed in detail in the ISA and summarized in Section 3.2 below, are remote from the portals of entry to the body (i.e., the respiratory system and gastrointestinal tract). Consequently, the internal disposition and distribution of Pb is an integral aspect of the relationship between exposure and effect. Inhaled lead-bearing particles, depending on size and solubility, may be absorbed into the systemic circulation or transported to the gastrointestinal tract (ISA, section 4.2.1.1). The absorption efficiency of Pb from the gastrointestinal (GI) tract varies with characteristics associated with the ingested Pb (e.g., particle size and chemical form or matrix), as well as with an individual's physiology (e.g., maturity of the GI tract), nutritional status (e.g., iron, calcium, and zinc deficiencies increase absorption), and the presence of food in the GI tract (ISA, section 4.2.1.2). Once in the blood stream, where approximately 99% of the Pb is associated with red blood cells (mostly bound to aminolevulinic acid dehydratase, the predominant ligand), Pb is quickly distributed throughout the body (e.g., within days) and is available for exchange with the soft and skeletal tissues, conceptually viewed as the fast and slow turnover pools, respectively (ISA, section 4.2.2). Skeletal tissue serves as the largest storage compartment, with much less Pb stored in soft tissues (e.g., kidney, liver, brain, etc) (ISA, section 4.2.2.2).

The role of the bone as the main storage compartment is related to the ability of Pb to form stable complexes with phosphate and replace calcium in the salt comprising the primary crystalline matrix of bone (ISA, section 4.2.2.2). In infants less than a year old, the bone is

1 estimated to contain approximately 60% of the total body burden of Pb (ISA, section 4.2.2.2;
2 Barry, 1975). Circulating Pb is taken up into the bone regions of active calcification.
3 Accordingly, during early childhood there is rapid uptake of Pb into mineralizing bone, with
4 somewhat more than 70% of total body burden Pb estimated to reside in bone of children aged 2
5 to 16, increasing up to more than 90% by adulthood (ISA, section 4.2.2.2; Barry, 1975). The net
6 accumulation of Pb in bone over a person's lifetime results in bone lead concentrations generally
7 increasing with age (ISA, section 4.2.2.2).

8 The distribution of Pb in the body is dynamic. Throughout life, Pb in the body is
9 exchanged between blood and bone, and between blood and soft tissues (ISA, sections 4.3.5 and
10 4.2.2; 2006 CD, section 4.3.2). The rates of these exchanges vary with age, exposure and
11 various physiological variables. For example, resorption of bone, which results in the
12 mobilization of Pb from bone into the blood, is a gradual process in later adulthood. Resorption
13 rate is appreciably increased in pregnant or nursing women, and in association with osteoporosis
14 in postmenopausal women or, to a lesser magnitude, in older men (ISA, sections 4.3.5.2).
15 Changes in Pb exposure circumstances can also influence these exchanges, e.g., substantial
16 reductions in exposure levels contribute to increased release of Pb from the bone into the blood
17 (ISA, section 4.3.5). The studies that address the relative contributions of bone Pb and current
18 Pb exposure to blood Pb are limited to a few human studies during the 1980s and early 1990s,
19 when leaded gasoline usage was common, and a primate study. These studies indicate an
20 appreciable contribution from bone Pb stores to Pb in blood, on the order of 40-70% of blood Pb
21 contributed from bone, under circumstances with higher concurrent exposure than exposures
22 common today (ISA, section 4.3.5; Smith et al., 1996; Gulson et al., 1995; Manton, 1985;
23 Franklin et al., 1997).

24 During bone resorption that occurs in pregnancy, Pb is released from bone, increasing the
25 bone contribution to maternal blood Pb levels, and maternal Pb circulates across the placenta,
26 posing risk to the developing fetus and providing the fetal body burden (ISA, sections 4.2.2.4,
27 4.3.5, 4.7.2 and 4.7.3; 2006 CD, 6.6.2; Chuang et al., 2001). The relative size of contributions of
28 maternal bone Pb and current maternal exposure to maternal blood Pb and fetal body burden, are
29 influenced by the relative magnitude of current and historical exposures. In various study
30 populations with mean maternal blood Pb levels ranging from 1.7 to 8.6 µg/dL, average blood Pb
31 concentration in the umbilical cord (as representative of newborn blood) has been reported to
32 range from 70% to 100% of average maternal blood Pb (ISA, section 4.2.2.4); the range is
33 similar for four study populations with mean maternal blood Pb level below 4 µg/dL (Amaral et
34 al., 2010; Patel and Prabhu, 2009; Lagerkvist et al., 1996; Jedrychowski et al., 2011). The
35 relationship for individual mother-child pairs is more variable. In a low income population with
36 mean maternal blood Pb of 1.9 µg/dL, factors associated with occurrences of relatively higher

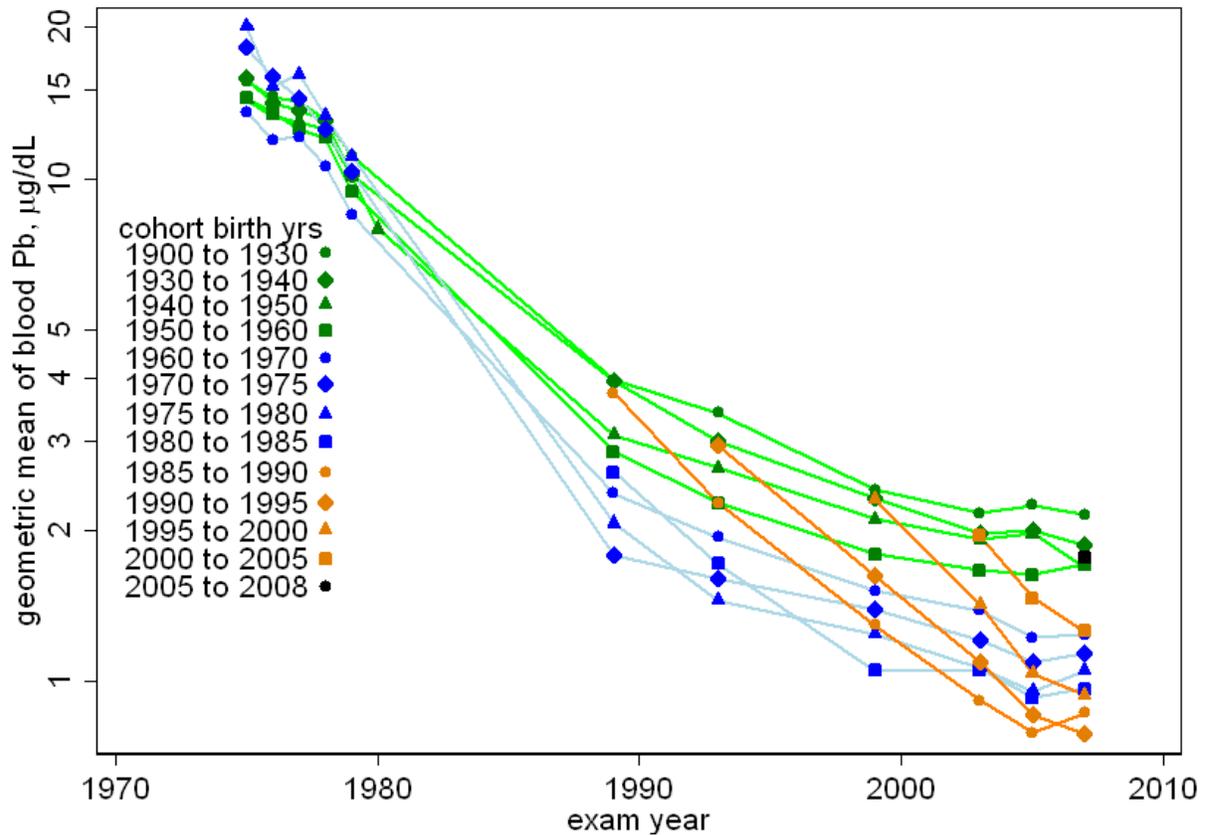
1 cord blood Pb level to maternal blood Pb level pairs included maternal blood pressure and
2 alcohol consumption, while factors associated with relatively lower ratios of cord blood Pb to
3 maternal blood Pb included maternal hemoglobin and sickle cell trait (Harville et al., 2005). As
4 a result of the contributions of maternal bone Pb to maternal blood Pb during pregnancy, the
5 contribution to fetal body burden from maternal bone Pb relative to maternal concurrent
6 exposure is appreciable. This maternal bone Pb contribution to fetal body burden is likely to
7 vary, however, in response to differences between maternal historical exposures and those during
8 pregnancy, among other factors (2006 CD, sections 4.3.1.5 and 4.3.2.5; ISA, section 6.1; Chuang
9 et al., 2001; Gulson et al., 1999; Gulson et al., 2003; Gulson et al., 2004a; Rothenberg et al.,
10 2000).

11 Limited data indicate that blood Pb levels in some newborns may decline during the
12 period extending through the first few months of life; the extent to which this may occur may be
13 influenced by the magnitude of blood Pb level at birth and of subsequent exposure during early
14 infancy (ISA, section 4.2.2.1; Carbone et al., 1998; Simon et al., 2007; Gulson et al., 1999,
15 2001). As infants become more mobile and engage in hand-to-mouth behavior, blood Pb levels
16 then commonly increase to a peak around two years of age (ISA, sections 4.4.1 and 6.2.1.1).

- 17 • **Does the current evidence continue to support blood Pb level as a useful indicator**
18 **of Pb exposure and dose for purposes of characterizing Pb health effects, with**
19 **well recognized strengths and limitations? To what extent does the evidence**
20 **suggest alternatives?**

21 As discussed in past CDs and in the ISA, blood Pb is the most commonly used indicator
22 of human Pb exposure. For example, given the association with exposure and the relative ease of
23 collection, blood Pb levels are extensively used as an index or biomarker of exposure by national
24 and international health agencies, as well as in epidemiological and toxicological studies of Pb
25 health effects and dose-response relationships (ISA, sections 4.3.2, 4.4.1, 5.3, 5.4, 5.5, 5.6, 5.7,
26 and 5.8). Since 1976, the U.S. Centers for Disease Control and Prevention (CDC) has been
27 monitoring blood Pb levels nationally through the National Health and Nutrition Examination
28 Survey (NHANES). This survey has documented the dramatic decline in mean blood Pb levels
29 in all ages of the U.S. population that has occurred since the 1970s, as shown in Figure 3-1, and
30 that coincides with actions on leaded fuels, leaded paint, lead in food packaging, and lead-
31 containing plumbing materials that have reduced Pb exposure in the U.S. (ISA, section 4.4.1;
32 Pirkle et al., 1994; Schwemberger et al., 2005). This decline has continued over the more recent
33 past. For example, the 2009-2010 geometric mean blood Pb level in U.S. children aged 1-5
34 years is 1.17 µg/dL, as compared to 1.51 µg/dL in 2007-2008 (ISA, section 4.4.1) and 1.8 µg/dL
35 in 2003-2004, the most recent data available at the time of the last review (73 FR 67002);

1 somewhat less dramatic declines have also been reported in the upper tails of the distribution and
2 in different groups with higher levels than the general child population (ISA, Figure 4-19).



3
4 Source: Adapted from data from the NHANES (NCHS, 2010)
5 Note: The means of logged blood Pb were weighted to represent national averages. Data were from the publically NHANES II, NHANES III for
6 1988 1991 and 1992 1994, and the continuous NHANES in 1999 2000, 2003 2004, 2005 2006, 2007 2008. Continuous NHANES data from
7 2001 2002 and 2009 2010 are not included because there were only 551 blood Pb samples in each of those data sets. The year plotted for
8 exam year was the reported exam year for NHANES II, the middle year of each of the phases of NHANES III, and the second year of each of
9 the continuous NHANES.

10 **Figure 3-1. Temporal trend in mean blood Pb levels for NHANES cohorts.**

11 The CDC, and its predecessor agencies, have for many years used blood Pb level as a
12 metric for identifying highly exposed children at risk of adverse health effects for whom
13 recommendations might be made for their protection (e.g., CDC, 1991; CDC, 2005). In 1978,
14 when the current Pb NAAQS was established, the CDC recognized a blood Pb level of 30 µg/dL
15 as a level warranting individual intervention (CDC, 1991). In 1985, the CDC recognized a level
16 of 25 µg/dL for individual child intervention, and in 1991, they recognized a level of 15 µg/dL at
17 which individual intervention actions were recommended, and a level of 10 µg/dL at which more
18 general community-wide prevention activities were to be implemented (CDC, 1991; CDC,
19 2005). In 2005, with consideration of a review of the evidence by their advisory committee,

1 CDC revised their statement on *Preventing Lead Poisoning in Young Children*, specifically
2 recognizing the evidence of adverse health effects in children with blood Pb levels below 10
3 $\mu\text{g}/\text{dL}$ and emphasizing the importance of preventive measures (CDC, 2005).¹ Consistent with
4 this statement, the CDC revised their approach in 2012 to one that relies on the 97.5th percentile
5 of blood Pb concentrations in U.S. children (currently 5 $\mu\text{g}/\text{dL}$)² as a reference level for
6 identifying children for whom they recommend particular follow-up actions (CDC, 2012).

7 Multiple studies of the relationship between Pb exposure and blood Pb in children (e.g.,
8 Lanphear and Roghmann 1997; Lanphear et al., 1998), have shown blood Pb levels to reflect Pb
9 exposures, including exposures to Pb in surface dust. These and studies of child populations
10 near active sources of air emissions (e.g., metal smelters), further demonstrate the effect of
11 airborne Pb on interior dust and on blood Pb (ISA, sections 4.4.1, 4.5.1 and 4.5.3; Hilts, 2003;
12 Gulson et al., 2004b). Accordingly, blood Pb level was the index of exposure in the assessment
13 of young children's risk performed in the last Pb NAAQS review (described in section 3.4
14 below).

15 A well recognized strength of using children's blood Pb to investigate relationships
16 between Pb exposure and health effects is the relatively lesser uncertainty in the causality
17 aspects of such relationships than might be associated with such relationships based, for
18 example, on media concentration. Blood Pb is an integrated marker of aggregate exposure
19 across all pathways. For example, the blood Pb-response relationships described in
20 epidemiological studies of lead-exposed populations do not distinguish among different sources
21 of Pb or pathways of Pb exposure (e.g., inhalation, ingestion of indoor dust, ingestion of dust
22 containing leaded paint). Given our focus on ambient air contributions to Pb exposures,
23 discussion in response to the question below considers the available information regarding
24 relationships between Pb in ambient air and the associated Pb in blood. Additionally, the
25 exposure assessment performed for the last review of the Pb NAAQS (and described in section
26 3.4 below), employed biokinetic modeling to estimate blood Pb levels associated with aggregate
27 Pb exposure to inform our estimates of contributions to blood Pb arising from ambient air related
28 Pb.

29 The response of adult blood Pb levels to appreciable changes in exposure circumstances
30 is generally slower than that of blood Pb levels in young children. For example, simulations
31 using biokinetic models indicate that blood Pb levels in adults achieve a new quasi-steady state

¹ With the 2005 statement, CDC identified a variety of reasons, reflecting both scientific and practical considerations, for not lowering the 1991 level of concern, including a lack of effective clinical or public health interventions to reliably and consistently reduce blood Pb levels that are already below 10 $\mu\text{g}/\text{dL}$, the lack of a demonstrated threshold for adverse effects, and concerns for deflecting resources from children with higher blood Pb levels (CDC, 2005).

² CDC intends to update the value every 4 years using the two most recent NHANES surveys (CDC, 2012).

1 within 75-100 days (approximately 3-4 times the blood elimination half-life) subsequent to
2 abrupt increases in Pb intake (ISA, section 4.3.5.2); similar models indicate a much quicker
3 response of blood Pb levels in children both with regard to abrupt increases and reductions in Pb
4 exposure (ISA, section 4.3.5.1). The response in young children may reflect their much more
5 labile bone pool associated with the rapid turnover of bone mineral in response to their rapid
6 growth rates (ISA, section 4.3.5). As a result of these physiological processes in young children,
7 their blood Pb levels tend to more quickly reflect changes in their total body burden (associated
8 with their shorter exposure history), and can also reflect changes in recent exposures (ISA,
9 section 4.3.5).

10 The extent to which blood Pb measurements represent current exposure circumstances
11 may be uncertain, particularly where histories include exposures largely different from those
12 occurring more recently (ISA, section 4.3.2). This uncertainty may be greater for blood Pb
13 measurements taken in older children³ and adults than those for young children as a result of their
14 longer exposure histories. For example, the bone of adults has accumulated decades of Pb
15 exposures (with past exposures often greater than current ones for current U.S. populations) such
16 that the bone may be a significant source of Pb in blood in later years, after exposure has ended.
17 Thus, in populations with past exposures that were appreciably elevated in comparison to more
18 recent exposures, current blood Pb levels may predominantly reflect their exposure history rather
19 than current exposures (ISA, sections 4.3.5.2 and 4.4.1). Accordingly, the extent to which
20 studies using cross-sectional blood Pb measurements as the exposure biomarker inform our
21 understanding of relationships between recent Pb exposures and various health effects, can differ
22 across age groups, and is greatest for very young children, as discussed in section 3.3 below.

23 Alternative biomarkers of Pb exposure include bone Pb, teeth Pb, and hair Pb (ISA,
24 section 4.3.1, 4.3.3 and 4.3.4). Given the role of bone as the repository of more than 90% of an
25 adult body's Pb burden, bone Pb levels are recognized as indicators of body burden and
26 cumulative exposure history (ISA, sections 4.2.2.2 and 4.3). The currently available evidence
27 does not, however, indicate these or any other alternatives to be superior to blood Pb in young
28 children or as commonly used for the purposes of tracking recent Pb exposures and for assessing
29 potential health risk for this age group (ISA, section 4.7.3). In summary, the current evidence
30 continues to support our conclusions from the last review regarding the use of blood Pb levels as
31 an internal biomarker of Pb exposure and dose informative to characterizing Pb health effects in
32 young children.

³ There is a paucity of experimental measurements of Pb biomarkers during adolescence to inform our characterization of Pb biokinetics (and relative roles of recent vs historic exposure on blood Pb levels) during this lifestage, in which individuals undergo rapid changes in sexual development, growth, food and water intake, bone growth and turnover, behavior, etc. (ISA, section 4.3).

1 • **To what extent has new information altered scientific conclusions regarding the**
2 **relationships between Pb in ambient air and Pb in children’s blood?**

3 As described above, blood Pb is an integrated marker of aggregate exposure across all
4 pathways and a reflection of exposure history. Thus, our interpretation of the health effects
5 evidence for purposes of this review necessitates characterization of the relationships between Pb
6 from those sources and pathways of interest in this review (i.e., those related to Pb emitted into
7 the air) and blood Pb. The evidence in this regard derives from analyses of datasets for
8 populations residing in areas with differing air Pb concentrations, including datasets for
9 circumstances in which blood Pb levels have changed in response to changes in air Pb. The
10 control for variables other than air Pb that can affect blood Pb varies across these analyses.

11 Lead in ambient air contributes to Pb in blood by multiple exposure pathways by both
12 inhalation and ingestion exposure routes (ISA, section 4.1.1). The quantitative relationship
13 between ambient air Pb and blood Pb, which is often termed a slope or ratio, describes the
14 increase in blood Pb (in $\mu\text{g}/\text{dL}$) estimated to be associated with each unit increase of air Pb (in
15 $\mu\text{g}/\text{m}^3$). Ratios are presented in the form of 1:x, with the 1 representing air Pb (in $\mu\text{g}/\text{m}^3$) and x
16 representing blood Pb (in $\mu\text{g}/\text{dL}$). Description of ratios as higher or lower refers to the values for
17 x (i.e., the change in blood Pb per unit of air Pb). Slopes are presented as simply the value of x.

18 At the conclusion of the last review in 2008, the EPA interpreted the evidence as
19 providing support for use (in informing the Administrator’s decision on standard level) of a
20 range “inclusive at upper end of estimates on the order of 1:10 and at the lower end on the order
21 of 1:5” (73 FR 67002). This conclusion reflected consideration of the air-to-blood ratios
22 presented in the 1986 CD⁴ and associated observations regarding factors contributing to variation
23 in such ratios, ratios reported subsequently and ratios estimated based on modeling performed in
24 the REA, as well as advice from CASAC (73 FR 66973-66975, 67001-67002). The information
25 available in this review, which is assessed in the ISA and is largely, although not completely,
26 comprised of studies that were available in the last review, does not alter the primary scientific
27 conclusions drawn in the last review regarding the relationships between Pb in ambient air and
28 Pb in children’s blood. The ratios summarized in the ISA in this review span a range generally
29 consistent with the range concluded in 2008 (ISA, section 4.5.1).

30 The air-to-blood ratios for children, or for populations inclusive of children, discussed in
31 the ISA for this review are summarized in Table 3-1. The evidence pertaining to this
32 quantitative relationship between air Pb and children’s blood Pb is now, as in the past, limited by
33 the circumstances in which the data are collected. These estimates are generally developed from
34 studies of populations in a variety of Pb exposure circumstances. Accordingly, there is

⁴ The 2006 CD did not include an assessment of then-current evidence on air-to-blood ratios.

1 significant variability in air-to-blood ratios among the different study populations exposed to Pb
2 through different air-related exposure pathways and at different exposure levels. This variability
3 in air-to-blood estimates can relate to the representation of air-related pathways and study
4 populations, including, for example, relatively narrow age ranges for the population, to reduce
5 age-related variability in blood Pb or including populations with narrowly specified dietary
6 sources. It can also relate to the precision of air and blood measurements and of the study
7 circumstances, such as with regard to spatial and temporal aspects. Additionally, in situations
8 where exposure to nonair sources covaries with air-related exposures that are not accounted for
9 in deriving ratio estimates, uncertainties may relate to the potential for confounding by nonair
10 exposure covariance (ISA, section 4.5).

11 As was noted in the last review, population age is an important influence on the
12 magnitude of air-to-blood ratio estimates derived. Ratios for children are generally higher than
13 those for adults, and higher for young children than older children, perhaps due to behavioral
14 differences between the age groups, as well as the more rapid response of children's blood Pb to
15 exposure and body burden changes (as discussed above) and shorter exposure history. Similarly,
16 given the common pattern of higher blood Pb levels in pre-school aged children than during the
17 rest of childhood, related to behaviors that increase environmental exposures, ratios would be
18 expected to be highest in earlier childhood. Additionally, estimates of air-to-blood ratios that
19 include air-related ingestion pathways in addition to the inhalation pathway are "necessarily
20 higher", in terms of blood Pb response, than those estimates based on inhalation alone (USEPA
21 1986, p. 11-106). Thus, the extent to which studies account for the full set of air-related
22 inhalation and ingestion exposure pathways affects the magnitude of the resultant air-to-blood
23 estimates, such that including fewer pathways as "air-related" yields lower ratios. Estimates of
24 air-to-blood ratios can also be influenced by population characteristics that may influence blood
25 Pb; accordingly, some analyses include adjustments.

26 Given the recognition of young children as a key at-risk population in section 3.3 below,
27 as well as the influence of age on blood Pb levels, the studies presented in Table 3-1 are grouped
28 with regard to the extent of their inclusion of young children (e.g., children barely school age).
29 Among the first group of studies, focused exclusively on young children, is the only study that
30 dates from the end of or after the phase-out of leaded gasoline usage (Hilts, 2003). This study
31 reports changes in children's blood Pb levels associated with reduced Pb emissions and
32 associated air concentrations near a Pb smelter in Canada (for children through age five). Given
33 the timing of this study, after the leaded gasoline phase-out, and its setting near a smelter, the
34 ambient air Pb in this study may be somewhat more comparable to that near sources in the U.S.

1 today than other studies discussed here. The study authors report an air-to-blood ratio of 1:6.⁵
2 An EPA analysis of the air and blood data reported for 1996, 1999 and 2001 results in a ratio of
3 1:6.5, and the analysis focused only on the 1996 and 1999 data (pre- and post- the new
4 technology) yields a ratio of 1:7 (ISA, section 4.5.1; Hiltz, 2003).⁶ The two other studies that
5 focused on children of age 5 or younger analyze variations in air Pb as a result of variations in
6 leaded gasoline usage in Chicago, Illinois. The study by Hayes et al. (1994) compared patterns
7 of ambient air Pb reductions and blood Pb reductions for large numbers of children, aged 6
8 months - 5 years, in Chicago between 1974 and 1988, a period when significant reductions
9 occurred in both measures. The study reports a better fit for the log-log model which describes a
10 pattern of higher ratios with lower ambient air Pb and blood Pb levels (Hayes et al., 1994).
11 Based on the log-log model, the ratio derived for the relationship of quarterly mean air
12 concentration with blood Pb during the period is 1:8 (Table 3-1). Another analysis for a Chicago
13 dataset, performed by Schwartz and Pitcher (1989) focused on the association of blood Pb in
14 black children (aged ≤ 5 years) with the use of leaded gasoline from 1978 through 1980. Given
15 that leaded gasoline exposure occurs by air-related pathways, additional analyses have related
16 the leaded gasoline usage for this period to air concentrations. Using the resulting relationship of
17 blood Pb with air Pb (adjusted for age and a number of other covariates) yields a ratio of 1:8.6
18 (ISA, table 4-12, section 4.5.2). The blood Pb concentrations in the two leaded gasoline studies
19 are appreciably higher (a factor of two or more) than those in the study near the smelter (Hiltz,
20 2003).

21 The second group of studies in Table 3-1 (comprised of studies including but not limited
22 to children less than or equal to five years of age) includes a complex statistical analysis and
23 associated dataset for a cohort of children born in Mexico City from 1987 through 1992 (Schnaas
24 et al., 2004). This study, which was not assessed in the last review, encompasses the period of

⁵ Sources of uncertainty include the role of factors other than ambient air Pb reduction in influencing decreases in blood Pb (ISA, section 4.5.1). The author cited remedial programs (e.g., community and home-based dust control and education) as potentially responsible for some of the blood Pb reduction seen during the study period (1997 to 2001), although he notes that these programs were in place in 1992, suggesting they are unlikely to have contributed to the sudden drop in blood Pb levels occurring after 1997 (Hiltz, 2003). Other aspects with potential implications for ratios include the potential for children with lower blood Pb levels not to return for subsequent testing, and the age range of 6 to 36 months in the 2001 blood screening compared to ages up to 60 months in previous years (Hiltz, 2003).

⁶ This study considered changes in ambient air Pb levels and associated blood Pb levels over a five-year period which included closure of an older Pb smelter and subsequent opening of a newer facility in 1997 and a temporary (3 month) shutdown of all smelting activity in the summer of 2001. The author observed that the air-to-blood ratio for children in the area over the full period was approximately 1:6. The author noted limitations in the dataset associated with exposures in the second time period, after the temporary shutdown of the facility in 2001, including sampling of a different age group at that time and a shorter time period (3 months) at these lower ambient air Pb levels prior to collection of blood Pb levels. Consequently, EPA calculated an alternate air-to-blood Pb ratio based on ambient air Pb and blood Pb reductions in the first time period, after opening of the new facility in 1997 (ISA, section 4.5.1).

1 leded gasoline usage and further informs our understanding of factors influencing the
2 quantitative relationship between air Pb and children’s blood Pb. Air-to-blood ratios developed
3 from this study are influenced by a number of factors and appear to range from roughly 2 to 6, in
4 addition to an estimate of 9 (ISA, section 4.5.1), although this is derived from data set restricted
5 to the latter years of the study when little change in air Pb concentration occurred, such that the
6 role of air Pb may be more uncertain. Estimates associated with the developmental period of
7 highest exposure – e.g., age 2 years - range up to approximately 6, illustrating the influence of
8 age on the ratio (ISA, section 4.5.1). Also in the Table 3-1 second group are two much older
9 studies of populations comprising age ranges extending well beyond 6 years. The first is the
10 review and meta-analysis by Brunekreef (1984) using datasets available at the time for variously
11 aged children as old as 18 years with identified air monitoring methods and reliable blood Pb
12 data for 18 locations in the U.S. and internationally.⁷ The author discusses potential confounders
13 of the relationship between air Pb and blood Pb, recognizing the desirability of taking them into
14 account when deriving an air-to-blood relationship from a community study, but noting that was
15 not feasible in such an analysis. Two models were produced, one based on the full pooled dataset
16 and a second limited to blood Pb-air Pb data pairs with blood Pb levels below 20 µg/dL (r^2
17 values were 0.692 and 0.331, respectively). From these two \log_n - \log_n models, air-to-blood ratio
18 estimates were derived for air concentrations corresponding to the geometric means of the two
19 sets of data pairs. At those concentrations (1.5 and 0.54 µg/m³, respectively), the resultant ratios
20 both round to 5. The study by Schwartz and Pitcher (1989) described above also analyzed the
21 relationship between U.S. NHANES II blood Pb levels for white subjects, aged ≤74 years, and
22 national usage of leaded gasoline. A separate, less specifically described, air Pb dataset was used
23 to convert the relationship of blood Pb with gas Pb to one for blood Pb with air Pb, with a
24 resultant ratio on the order of 9, adjusted for age and other covariates (Henderson, 2007, pp. D-2
25 to D-3; ISA, Table 4-12).

26 The last two studies included in Table 3-1 are focused on older children (ages 6-11). The
27 methods for characterization of air Pb concentrations (and soil Pb for latter study) also differ
28 from other studies in Table 3-1. The first study regressed average blood Pb concentrations for
29 multiple locations around Mumbai, India on average air Pb concentrations at those locations
30 (Tripathi et al., 2001). The values in the linear regression were 13 pairs of location-specific
31 geometric means of all the data collected over the 13-year period from 1984 to 1996; the
32 reported slope was 3.6 (Tripathi et al., 2001). The location-specific geometric mean blood Pb

⁷ In the dataset reviewed by Brunekreef (1984), air-to-blood ratios from the subset of those studies that used quality control protocols and presented adjusted slopes include values of 3.6 (Zielhuis et al., 1979), 5.2 (Billick et al., 1979, 1980); 2.9 (Billick et al., 1983), and 8.5 (Brunekreef et al., 1983). The studies cited here adjusted for parental education (Zielhuis et al., 1979), age and race (Billick et al., 1979, 1980) and air Pb monitor height (Billick et al., 1983); Brunekreef et al. (1984) used multiple regression to control for several confounders (73 FR 66974).

1 levels in this study (8.6-14.4 $\mu\text{g}/\text{dL}$) indicate blood Pb distributions in this age group much
2 higher than those pertinent to similarly aged children in the U.S. today. The second study
3 analyzed air, soil and children's blood Pb concentrations in Duisburg, Germany during the
4 leaded gasoline phase-out (Ranft et al., 2008). Average blood Pb levels declined over the nearly
5 20-year study period from 9 $\mu\text{g}/\text{dL}$ in 1983 (345 children average age of 9 years) to 3 $\mu\text{g}/\text{dL}$ in
6 2000 (162 children average of 6 years).⁸ Average air Pb concentration declined from 0.45 $\mu\text{g}/\text{m}^3$
7 to 0.06 $\mu\text{g}/\text{m}^3$ over the same period, with the largest reduction occurring between the first study
8 year (derived from two monitoring sites for full study area) and the second study year, 1991, for
9 which air concentrations were derived from a combination of dispersion modeling and the two
10 monitoring sites.⁹ For a mean air Pb concentration of 0.1 $\mu\text{g}/\text{m}^3$, the study's multivariate log-
11 linear regression model predicted air to blood ratios of 3.2 and 6.4 for "background" blood Pb
12 concentrations of 1.5 and 3 $\mu\text{g}/\text{dL}$, respectively. In this study, background referred to Pb in
13 blood from other sources; the blood Pb distribution over the study period, including levels when
14 air Pb concentrations are lowest, indicates 3 $\mu\text{g}/\text{dL}$ may be the better estimate of background for
15 this study population. Inclusion of soil Pb as a variable in the model may have contributed to an
16 underestimation of the these blood Pb-air Pb ratios for this study because some of the Pb in soil
17 likely originated in air and the blood Pb-air Pb slope does not include the portion of the soil/dust
18 Pb ingestion pathway that derives from air Pb. The ISA also presents somewhat higher air-to-
19 blood ratio estimates derived using univariate linear, log-log and log-linear models on the
20 median air and blood Pb concentrations reported for the five years included in this study (ISA,
21 section 4.5.1; Ranft et al., 2008, Table 2). Uncertainties related to this study's estimates include
22 those related to the bulk of air concentration reduction occurring between the first two time
23 points (1983 and 1991) and the difference among the year's air datasets (e.g., two data sources
24 [air monitors] in 1983 and multiple geographical points from a combination of the monitors and
25 modeling in subsequent years).

⁸ Blood Pb measurements were available on a total of 843 children across five time periods, in the first of which the average child age was 9 years while it was approximately 6 years in each of the latter years: 1983 (n=356), 1991 (n=147), 1994 (n=122), 1997 (n=56), and 2000 (n=162) (Ranft et al., 2008).

⁹ The 1983 air Pb concentrations were based on two monitoring stations, while a combination of dispersion modeling and monitoring data was used in the later years. Surface soil Pb measurements were from 2000-2001, but geo-matched to blood Pb measurements across full study period (Ranft et al., 2008).

1 **Table 3-1. Empirically derived air-to-blood ratios for populations inclusive of children.**

Study Information	Quantitative Analysis	Air-to-Blood Ratio ^A
Focused on children ≤ 5 years old		
Children, 0.5-5 yr (n = 9,604), average age 2.5 yr Chicago, IL, 1974-1988 Urban area with lead-emitting industries, leaded gasoline usage Hayes et al. (1994)	<u>log_n-log_n</u> regression: quarterly mean PbB and PbA [unadjusted] PbB: 10-28 µg/dL (quarterly mean) PbA: 0.05-1.2 µg/m ³ (quarterly mean)	8.2 (@ 0.62) ^B
Children, 0.5-5 yr (1996-1999), 0.5-3 yr (2001) (n = 200-500) Trail, BC, 1996-2001; Small town before/after cleaner technology on large metals smelter (at end of/after leaded gasoline phase-out) Hilts (2003)	<u>linear</u> regression: annual GM PbB and AM PbA [unadjusted] PbB: 4.7-11.5 µg/dL (annual GM) PbA: 0.03-1.1 µg/m ³ (annual AM)	6- 7.0 ^C
Black children, <5 yr (n = 5,476) Chicago, IL, Feb 1976- Feb 1980 Area with lead-emitting industries, leaded gasoline usage Schwartz and Pitcher (1989), U.S. EPA (1986a)	<u>linear</u> regression: quarterly mean PbB with gasoline Pb (usage) [adjusted for demographic covariates] combined with gasoline Pb – air Pb relationship. PbB: 18-27 µg/dL (quarterly mean, adjusted) PbA – gas Pb relationship based on annual U.S. means of per-site maximum quarterly means (0.36-1.22 µg/m ³)	8.6 ^D
Larger age range, inclusive of children ≤ 5 years old		
Children, 96 groups of various age ranges. (n>190,000) Various countries (18 locations), 1974-1983 urban or near lead-emitting industries, leaded gasoline usage Brunekreef et al. (1984)	Meta-analyses (<u>log_n-log_n</u> regression of group means): (1) all children, (2) children <20 µg/dL [unadjusted] PbB: 5-76 µg/dL (study group means) PbA: 0.1-24 µg/m ³ (location means)	Full dataset: 4.6 (@1.5) ^E <20µg/dL: 4.8 (@ 0.54) ^F
Children, born 1987-92 (n = 321); Mexico City, 1987-2002 Ave age increased over study period: <3yrs (1987-1992), and increased by a year each year after that Urban area during/after leaded gasoline usage Schnaas et al. (2004)	<u>Linear</u> , <u>log_n-log_n</u> regressions: annual mean PbB and PbA [unadjusted] PbB: 5-12 µg/dL (annual GM), aged 0.5-10 yr PbA: 0.07-2.8 µg/m ³ (AM); 0.1-0.4 over last 6 years	Linear: 9.0 (0.1-0.4) 2.5 (full range) Log-log: 4.5 (@ 0.4) ^C
U.S. NHANES II white subjects, 0.5-74 yr, Feb 1976-1980 National survey during time of leaded gasoline usage Schwartz and Pitcher (1989), U.S. EPA (1986a)	<u>Linear</u> regression: PbB with mass Pb in gasoline as described above. [adjusted for demographic covariates] PbB: 11-18 µg/dL (mean per gas Pb, adjusted) PbA – gas Pb relationship based on annual U.S. means of per-site maximum quarterly means (0.36-1.22 µg/m ³)	9.3 ^D
Focused on children ≥ 6 years old		
Children, 6-10 yr (n = 544) Mumbai, India, 1984-1996 Large urban area, leaded gasoline usage Tripathi et al. (2001)	<u>Linear</u> regression: 13-year location-specific GM PbB and PbA [unadjusted] PbB: 8.6-14.4 µg/dL (location GM) PbA: 0.11-1.18 µg/m ³ (location GM); 0.45 (overall GM)	3.6
Children, 6-11 yr (n=843); ave~9.5 yr (1983), ~6.5 (others) Duisburg, Germany–5 areas: 1983, 1991, 1994, 1997, 2000 Industrial urban area during/after leaded gasoline usage Ranft et al. (2008)	<u>Linear</u> , <u>log_n-log_n</u> , <u>log-linear</u> regressions: annual mean PbB and air Pb [unadjusted] PbB: 3.33-9.13 µg/dL (AMs of 5 study years) PbA: 0.06-0.45 µg/m ³ (5 AMs), 0.10 (overall median)	Log-linear ^G 6.4, 3.2
<p>A - Predicted change in blood Pb (µg/dL per µg/m³) over range ± 0.01 µg/m³ from study's central air Pb, which is provided in µg/m³ in parentheses. For linear models, this is simply the air Pb coefficient.</p> <p>B - $\ln(\text{PbB}) = \ln(\text{PbA}) \times 0.24 + 3.17$ (ISA, Table4-12; Hayes et al., 1994)</p> <p>C - See discussion in text and ISA, section 4.5.1.</p> <p>D - Based on data for U.S. (1986 Pb CD). See ISA, section 4.5.1. Log-lin = log-linear model.</p> <p>E - $\ln(\text{PbB}) = \ln(\text{PbA}) \times 0.3485 + 2.853$</p> <p>F - $\ln(\text{PbB}) = \ln(\text{PbA}) \times 0.2159 + 2.620$</p> <p>G -Derived from regressions with separate soil Pb variable, contributing to underestimation of contribution from air Pb (see text and ISA, section 4.5.1).</p> <p>GM, geometric mean; AM, arithmetic mean; GSD, geometric standard deviation; PbB, blood Pb concentration, µg/dL; PbA, air Pb concentration, µg/m³</p>		

2

1 In the 2008 review, in addition to considering the evidence presented in the published
2 literature and that reviewed in the 1986 CD, we also considered air-to-blood ratios derived from
3 the exposure assessment (73 FR 66974; 2007 REA, section 5.2.5.2). In the exposure assessment
4 (summarized in section 3.4 below), current modeling tools and information on children's activity
5 patterns, behavior and physiology were used to estimate blood Pb levels associated with
6 multimedia and multipathway Pb exposure. The results from the various case studies assessed,
7 with consideration of the context in which they were derived (e.g., the extent to which the range
8 of air-related pathways were simulated, and limitations associated with those simulations), and
9 the multiple sources of uncertainty (see section 3.4.7 below) are also informative to our
10 understanding of air-to-blood ratios. Estimates of air-to-blood ratios for the two REA case
11 studies that represent localized population exposures exhibited an increasing trend across air
12 quality scenarios representing decreasing air concentrations. For example, across the alternative
13 standard levels assessed, which ranged from a calendar quarter average of 1.5 $\mu\text{g}/\text{m}^3$ down to a
14 monthly average of 0.02 $\mu\text{g}/\text{m}^3$, the ratios ranged from 1:2 to 1:9 for the general urban case
15 study, with a similar trend although of generally higher ratio for the primary smelter case study
16 subarea. This pattern of model-derived ratios is generally consistent with the range of ratios
17 obtained from the literature. We continue to recognize a number of sources of uncertainty
18 associated with these model-derived ratios which may contribute to high or low biases.¹⁰

19 The evidence on the quantitative relationship between air Pb and air-related Pb in blood
20 is now, as in the past, limited by the circumstances (such as those related to Pb exposure) in
21 which the data were collected. Previous reviews have recognized the significant variability in
22 air-to-blood ratios for different populations exposed to Pb through different air-related exposure
23 pathways and at different air and blood levels, with the 1986 CD noting that ratios derived from
24 studies involving the higher blood and air Pb levels pertaining to workers in lead-related
25 occupations are generally smaller than ratios from studies involving lower blood and air Pb
26 levels (ISA, p. 4-129; 1986 CD, p. 11–99). Consistent with this observation, slopes in the range
27 of 3 to 5 were estimated for child population datasets assessed in the 1986 CD (ISA, p. 4-129;

¹⁰ For example, the limited number of air-related pathways (inhalation and indoor dust ingestion) simulated to change in response to changes in ambient air Pb reductions in these case studies could have implications for the air-to-blood ratios. Additionally, with regard to the urban case study, the relationship between dust loading and concentration, a key component in the hybrid dust model used in estimating indoor dust Pb levels, is based largely on a housing survey dataset reflecting dust Pb in housing constructed before 1980 (as described in the Risk Assessment Report, Volume II, Appendix G, Attachment G-1). The use of leaded paint in some housing constructed before 1980 contributes some uncertainty due to the potential role of indoor Pb paint (compared to ambient air Pb) in the relationship. The empirically-based ambient air Pb – dust Pb relationships used in the primary Pb smelter (subarea) case study may contribute to a potential for the ratios from this case study to more fully capture the impact of changes in ambient air Pb on indoor dust Pb, and consequently on blood Pb. Some have suggested, however, that the regression used may not accurately reflect the temporal relationship between reductions in ambient air Pb and indoor dust Pb and as a result may overestimate the dust Pb reduction per ambient air Pb reduction, thus contributing a potential high bias to the air-to-blood Pb ratios.

1 1986 CD p. 11–100; Brunekreef, 1984; Tripathi et al., 2004). Additional studies considered in
2 the last review and those assessed in the ISA provide evidence of ratios above this older range
3 (ISA, p. 4-130). For example a ratio of 6.5-7 is indicated by the study by Hilts (2003), one of the
4 few studies that evaluate the air Pb-blood Pb relationship in conditions that are closer to the
5 current state in the U.S. (ISA, p. 4-130). We additionally note the variety of factors identified in
6 the ISA that may potentially affect estimates of various ratios (including potentially coincident
7 reductions in nonair Pb sources during the course of the studies), and for which a lack of
8 complete information may preclude any adjustment of estimates to account for their role (ISA,
9 section 4.5).

10 In summary, as at the time of the last review of the NAAQS for Pb, the currently
11 available evidence includes estimates of air-to-blood ratios, both empirically- and model-derived,
12 with associated limitations and related uncertainties. These limitations and uncertainties, which
13 are summarized here and also noted in the ISA, usually include uncertainty associated with
14 reductions in other Pb sources during study period. The limited amount of new information
15 available in this review has not appreciably altered the scientific conclusions reached in the last
16 review regarding relationships between Pb in ambient air and Pb in children’s blood or with
17 regard to the range of ratios. The currently available evidence continues to indicate ratios
18 relevant to the population of young children in the U.S. today, reflecting multiple air-related
19 pathways in addition to inhalation, to be generally consistent with the approximate range of 1:5
20 to 1:10 given particular attention in the 2008 NAAQS decision, including the “generally central
21 estimate” of 1:7 (73 FR 67002, 67004; ISA, pp. 4-129 to 4-130).

22 **3.2 NATURE OF EFFECTS**

23 Lead has been demonstrated to exert a broad array of deleterious effects on multiple
24 organ systems as described in the assessment of the evidence available in this review and
25 consistent with conclusions of past CDs (ISA, section 2.6; 2006 CD, section 8.4.1). A sizeable
26 number of studies on Pb health effects are newly available in this review and are critically
27 assessed in the ISA as part of the full body of evidence. The newly available evidence reaffirms
28 conclusions on the broad array of effects recognized for Pb in the last review (see ISA, section
29 2.10). Consistent with those conclusions, the ISA determines that causal relationships¹¹ exist for

¹¹ Since the last Pb NAAQS review, the ISAs which have replaced CDs in documenting each review of the scientific evidence (or air quality criteria) employ a systematic framework for weighing the evidence and describing associated conclusions with regard to causality, using established descriptors (“causal” relationship with relevant exposure, “likely” to be causal, evidence is “suggestive” of causality, “inadequate” evidence to infer causality, “not likely”) (ISA, Preamble).

1 Pb¹² with effects on the nervous system (cognitive function decrements and attention-related
2 behavioral problems in children), hematological system (altered heme synthesis and decreased
3 red blood cell survival and function), and cardiovascular system (hypertension and coronary
4 heart disease), and on reproduction and development (postnatal development and male
5 reproductive function) (ISA, table 2-2). Additionally, the ISA describes relationships between
6 Pb and effects on kidney and immune system function, and with cancer¹³ as likely causal¹⁴ (ISA,
7 table 2-2, sections 2.6.3, 2.6.4 and 2.6.7).

8 In some categories of health effects, there is newly available evidence regarding some
9 aspects of the effects described in the last review or that strengthens our conclusions regarding
10 aspects of Pb toxicity on a particular physiological system. Among the nervous system effects of
11 Pb, the newly available evidence is consistent with conclusions in the previous review which
12 recognized that “[t]he neurotoxic effects of Pb exposure are among those most studied and most
13 extensively documented among human population groups” (2006 CD, p. 8-25). Nervous system
14 effects that receive prominence in the current review, as in previous reviews, include those
15 affecting cognitive function and behavior in children (ISA, section 5.3), with conclusions that are
16 consistent with findings of the last review.

17 Across the broad array of Pb effects for systems and processes other than the nervous
18 system, the evidence base has been augmented with additional epidemiological investigations in
19 a number of areas, including developmental outcomes, such as puberty onset, and adult outcomes
20 related to cardiovascular function, for which several large cohorts have been analyzed (ISA,
21 Table 2-8 and sections 5.4 and 5.8). Conclusions on these other systems and processes are
22 consistent with conclusions reached in the last review, while also extending our conclusions on
23 some aspects of these effects. For example, evidence in this review for the cardiovascular
24 system includes information on the role of interactions of cumulative Pb exposure with other
25 factors such as stress in contributing to hypertension, and on a role for Pb in contributing to
26 coronary heart disease (ISA, section 5.4 and Table 2-8).

¹² In determining that a causal relationship exists for Pb with specific health effects, EPA has concluded that “[e]vidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures (i.e., doses or exposures generally within one to two orders of magnitude of current levels)” (ISA, p. lxiv)

¹³ Lead has been classified as a probable human carcinogen by the International Agency for Research on Cancer, based mainly on sufficient animal evidence, and as reasonably anticipated to be a human carcinogen by the U.S. National Toxicology Program (ISA, section 5.10). In this assessment, EPA concludes that there is a likely causal relationship between Pb exposure and cancer, based primarily on animal studies, with limited human evidence of suggestive associations (ISA, section 5.10.6).

¹⁴ In determining a likely causal relationship exists for Pb with specific health effects, EPA has concluded that “[e]vidence is sufficient to conclude that a causal relationship is likely to exist with relevant pollutant exposures, but important uncertainties remain” (ISA, p. lxiv).

1 Based on the extensive assessment of the full body of evidence available in this review,
2 the major conclusions drawn by the ISA regarding health effects of Pb in children include the
3 following (ISA, section 1.3.1).

4 *Multiple epidemiologic studies conducted in diverse populations of children*
5 *consistently demonstrate the harmful effects of Pb exposure on IQ, academic*
6 *performance, learning and memory. Epidemiologic studies also demonstrate the*
7 *effect of Pb exposure on inattention, impulsivity, and hyperactivity in children.*
8 *The evidence in children is supported by findings in animal studies demonstrating*
9 *analogous effects and biological plausibility at relevant exposure levels. Evidence*
10 *suggests that some Pb-related cognitive effects may not be reversible and that*
11 *neurodevelopmental effects of Pb may persist into adulthood (section 2.9.4).*
12 *Lead exposure also causes hematologic effects (such as effects on blood cells or*
13 *blood producing organs) in children and is associated with an increased risk of*
14 *internalizing behaviors (e.g., withdrawn behavior and depressive symptoms),*
15 *sensory and motor function decrements, atopic and inflammatory conditions (e.g.,*
16 *asthma and allergy) in children, as well as misconduct in older children and*
17 *young adults. Uncertainties arising from the lack of information about the specific*
18 *Pb-exposure histories which contribute to observed blood Pb levels are greater in*
19 *adults and older children than in young children (section 2.9.5).*
20

21 Based on the extensive assessment of the full body of evidence available in this review,
22 the major conclusions drawn by the ISA regarding health effects of Pb in adults include the
23 following (ISA, section 1.3.1).

24 *A large body of evidence from both epidemiologic studies of adults and*
25 *experimental studies in animals demonstrates the effect of long-term Pb exposure*
26 *on increased blood pressure (BP) and hypertension (Section 2.6.2). In addition to*
27 *its effect on BP, Pb exposure leads to coronary heart disease and death from*
28 *cardiovascular causes and is likely to cause cognitive function decrements,*
29 *symptoms of depression and anxiety, reduced kidney function, and immune effects*
30 *in adults. The extent to which the effects of Pb on the cardiovascular system are*
31 *reversible is not well characterized. It is also important to note that the*
32 *frequency, timing, level and duration of Pb exposure causing the effects observed*
33 *in adults has not been pinpointed, and higher past exposures may well have*
34 *contributed to the development of health effects measured later in life.*
35

36 As in prior reviews of the Pb NAAQS, this review is focused on those effects most
37 pertinent to ambient air Pb exposures. Given the reductions in ambient air Pb levels over the
38 past decades, these effects are generally those associated with the lowest levels of Pb exposure
39 that have been evaluated. Additionally, we recognize the limitations on our ability to draw
40 conclusions regarding the exposure conditions contributing to the findings from epidemiological
41 analyses of blood Pb levels in populations of older children and adults, particularly in light of
42 their history of higher Pb exposures. In the last review, while recognizing the range of health

1 effects in variously aged populations related to Pb exposure, we focused on the health effects for
2 which the evidence was strongest with regard to relationships with the lowest exposure levels,
3 neurocognitive effects in young children. The policy-relevant questions on health effects for this
4 review (identified in the IRP) were framed in recognition of the conclusions of the last review.
5 Our consideration of the health effects evidence in this review is framed by policy-relevant
6 questions building on those identified in the IRP.

7 • **To what extent is there new scientific evidence available to improve our**
8 **understanding of the health effects associated with various time periods of Pb**
9 **exposures at various stages of life?**

10 As in the last review, we base our current understanding of health effects associated with
11 different Pb exposure circumstances at various stages of life on the full body of available
12 evidence which includes epidemiological studies of health effects associated with population Pb
13 biomarker levels as well as laboratory animal studies in which the effects of different exposures
14 on different lifestages are assessed under controlled conditions. The epidemiological evidence is
15 overwhelmingly comprised of studies that rely on blood Pb for the exposure metric, with the
16 remainder largely including a focus on bone Pb. Because these metrics reflect Pb in the body
17 (e.g., as compared to Pb exposure concentrations) and, in the case of blood Pb, reflect Pb
18 available for distribution to target sites, they strengthen the evidence base for purposes of
19 drawing causal conclusions with regard to Pb generally. The complexity of Pb exposure
20 pathways and internal dosimetry tends to limit the extent to which these types of studies inform
21 our more specific understanding of the Pb exposure circumstances (e.g., timing, duration,
22 frequency and magnitude) eliciting the various effects.

23 The specific exposure circumstances, including timing during the lifetime, that contribute
24 to the blood Pb (or bone Pb) measurements with which associations have been analyzed in
25 epidemiological studies are unknown. This is particularly the case with regard to the
26 contributing role of recent exposures for which uncertainty is greater in adults and older children
27 than in younger children (ISA, section 1.4). For example, a critical aspect of much of the
28 epidemiological evidence, particularly that focused on older adults in the U.S. today, is the
29 backdrop of generally declining environmental Pb exposure that is common across many study
30 populations. An additional complication pertaining to older children and younger adults is the
31 common behaviors of younger children (e.g., hand to mouth contact) which generally contribute
32 to relatively greater environmental exposures earlier in life (ISA, sections 4.1.1, 6.2.1). Such
33 exposure histories complicate our ability to draw conclusions regarding critical time periods and

1 lifestages for Pb exposures eliciting the effects with which associations with Pb biomarkers have
2 been observed in populations of adults and older children (e.g., ISA, section 2.9.5).¹⁵

3 As at the time of the last review, assessment of the full evidence base, including evidence
4 newly available in this review, demonstrates that Pb exposure prenatally and also in early
5 childhood can contribute to neurocognitive impacts in childhood, with evidence also indicating
6 the potential for effects persisting into adulthood (ISA, sections 2.9.5, 2.9.6, and 2.10). In
7 addition to the observed associations of prenatal and childhood blood Pb with effects at various
8 ages in childhood, there is also evidence of lead-related cognitive function effects in non-
9 occupationally exposed adults (ISA, section 5.3.12). This includes evidence of associations of
10 such effects in adulthood with childhood blood Pb levels and, in other cohorts, with concurrent
11 (adult) blood Pb levels (ISA, sections 5.3.2.1, 5.3.2.7 and 5.3.12). As the studies finding
12 associations of adult effects with childhood blood Pb levels did not examine adult blood Pb
13 levels, the relative influence of adult Pb exposure cannot be ascertained, and a corresponding
14 lack of early life exposure or biomarker measurements for the latter studies limit our ability to
15 draw conclusions regarding specific Pb exposure circumstances eliciting the observed effects
16 (5.3.12). Findings of stronger associations for adult neurocognitive effects with bone Pb,
17 however, indicate the role of historical or cumulative exposures for those effects (ISA, section
18 5.3).

19 Given the relatively short exposure histories of young children, there is relatively
20 reduced uncertainty regarding the lifestages in which exposures contribute to effects for
21 associations of early childhood effects with early childhood blood Pb levels (ISA, sections 2.9.5
22 and 5.3.12). In considering our understanding of the relative impact on neurocognitive function
23 of additional Pb exposure of children by school age or later we recognize increasing uncertainty
24 associated with limitations of the currently available evidence, including epidemiological cohorts
25 with generally similar temporal patterns of exposure. We take note, additionally, of evidence
26 from experimental animal studies and a small body of epidemiologic studies that indicates that
27 Pb exposures during different lifestages can induce cognitive impairments. The limited
28 epidemiological evidence is of populations with blood Pb levels that are not strongly correlated
29 over time and that can, accordingly, address the issue of the role of exposure subsequent to the

¹⁵ The evidence from experimental animal studies can be informative with regard to key aspects of exposure circumstances in eliciting specific effects. Accordingly, for health effects where the animal evidence base is extensive with regard to investigation of different patterns of exposure, it informs our interpretation of the epidemiological evidence. For example, the animal evidence base is particularly extensive with regard to Pb effects on blood pressure, demonstrating the etiologically-relevant role of long-term exposure (ISA, section 5.4.1). This finding then informs consideration of epidemiological studies of adult populations for whom historical exposures were likely more substantial than concurrent ones to suggest that the observed effects may be related to the past exposure (ISA, section 5.4.1). For other health effects, the animal evidence base may be less extensive with regard to the role of specific exposure circumstances and thus less informative with regard to key exposure aspects.

1 earliest lifestages (ISA, section 5.3.12; Hornung et al., 2009). Some animal evidence
2 demonstrates impaired learning with infancy only, from infancy into adulthood, and postinfancy
3 only Pb exposure (Rice, 1990; Rice and Gilbert 1990; Rice, 1992). Further, evidence that Pb
4 exposure presents a risk during different lifestages is also consistent with our broader
5 understanding that nervous system development continues throughout childhood. The limited
6 analyses of this issue that are newly available in this review do not appreciably change our
7 understanding or conclusions on this from those of the prior review (ISA, section 5.3.12).

8 As in the last review, there is also substantial evidence of other neurobehavioral effects in
9 children, such as attention-related behavioral problems, conduct problems or internalizing
10 behaviors. The evidence for many of these endpoints, as with neurocognitive effects, also
11 includes associations of effects at various ages in childhood and, for some effects, into
12 adulthood, with blood Pb levels reflective of several different lifestages (e.g., prenatal and
13 several different ages in childhood) (ISA, sections 5.3.3 and 5.3.4). There is similar or relatively
14 less extensive evidence to inform our understanding of such effects associated with specific time
15 periods of exposure at specific lifestages than is the case for effects on cognitive function.

16 Across the range of Pb effects on physiological systems and processes other than the
17 nervous system, the full body of evidence on etiologically relevant circumstances of Pb exposure
18 eliciting increases in blood pressure and hypertension is somewhat more informative than is the
19 case with regard to many other effects. In the case of lead-induced increases in blood pressure,
20 the evidence indicates an importance of long-term exposure (ISA, sections 2.6.2 and 5.4.7.1).

21 In summary, as in the last review, we continue to recognize a number of uncertainties
22 regarding the circumstances of Pb exposure, including timing or lifestages, eliciting specific
23 health effects. Consideration of the evidence newly available in this review has not appreciably
24 changed our understanding on this topic. Across the full evidence base, the effects for which our
25 understanding of relevant exposure circumstances is greatest are neurocognitive effects in
26 children, and to a lesser degree, hypertension and increased blood pressure. Thus, we continue
27 to recognize and give particular attention to the role of Pb exposures relatively early in childhood
28 in contributing to neurocognitive effects which may persist into adulthood.

- 29 • **At what levels of Pb exposure do health effects of concern occur?**
- 30 • **Is there evidence of effects at exposure levels lower than previously observed and**
31 **what are important uncertainties in that evidence?**

32 In considering the question posed here, we recognize, as discussed in section 3.1 above,
33 that the epidemiological evidence base for our consideration in this review, as in the past,
34 includes substantial focus on internal biomarkers of exposure, such as blood Pb, with relatively
35 less information specific to exposure levels, including those derived from air-related pathways.

1 Given that blood and bone Pb are integrated markers of aggregate exposure across all sources
2 and exposure pathways, our interpretation of studies relying on them is informed by what is
3 known regarding the historical context and exposure circumstances of the study populations. For
4 example, a critical aspect of much of the epidemiological evidence is the backdrop of generally
5 declining Pb exposure over the past several decades. Thus as a generality, recent
6 epidemiological studies of populations with similar characteristics as those studied in the past
7 tend to involve lower overall Pb exposures and accordingly lower blood Pb levels (e.g., ISA,
8 sections 3.5 and 4.4.1; 2006 CD, section 3.4). This has been of particular note in the evidence of
9 blood Pb associations with nervous system effects, particularly impacts on cognitive function in
10 children, for which we have seen associations with progressively lower childhood blood Pb
11 levels across past reviews (ISA, section 5.3.13; 1986 CD; USEPA, 1990; 2006 CD; 73 FR
12 66976).

13 The evidence currently available with regard to the magnitude of blood Pb levels
14 associated with neurocognitive effects in children is generally consistent with that available in
15 the review completed in 2008. Nervous system effects in children, specifically effects on
16 cognitive function, continue to be the effects that are best substantiated as occurring at the lowest
17 blood Pb concentrations (ISA, sect 1.5). Associations of blood Pb with effects on cognitive
18 function measures in children have been reported in many studies across a range of childhood
19 blood Pb levels, including study group (mean/median) levels ranging down to 2 µg/dL (e.g., ISA,
20 sections 1.5 and 5.3.2).¹⁶ Studies in which such findings were reported for childhood study
21 group blood Pb levels below 5 µg/dL are summarized in Table 3-2.¹⁷ In recognition of the
22 influence of age on blood Pb levels, the analyses in Table 3-2 are listed in order of age at which
23 the blood Pb measurements were taken. Findings for studies newly available in this review are
24 indicated in bold text.

¹⁶ The value of 2 µg/dL refers to the regression analysis of blood Pb and end-of-grade test scores, in which blood Pb was represented by categories for integer values of blood Pb from 1 µg/dL to 9 and ≥10 µg/dL from large statewide database. A significant effect estimate was reported for test scores with all blood Pb categories in comparison to the reference category (1 µg/dL), which included results at and below the limit of detection. Mean levels are not provided for any of the categories (Miranda., et al., 2009).

¹⁷ Two additional studies (both newly available) that report such associations with blood Pb levels for which the mean is equal to 5.0 µg/dL. The first is a study of 506 children in Detroit, MI (born, 1982-1984) at age 7 years which observed a significant negative association with concurrent blood Pb levels for which the mean equals 5.0 µg/dL (8.9% of children with concurrent blood Pb above 10 µg/dL) (ISA, sections 5.3.2.1 and 5.3.3.5; Chiodo et al., 2007). The second study focuses on 174 of the Rochester cohort children at age 6 years, reporting significant negative associations with FSIQ for four different blood Pb metrics: concurrent (mean = 5.0 µg/dL), lifetime average (mean=7.2 µg/dL), infancy average (mean = 7.1 µg/dL) and peak (mean = 11.4 µg/dL) (Jusko et al., 2008).

1 **Table 3-2. Associations with neurocognitive function measures in analyses with child study group blood lead levels <5 µg/dL.**

Measure ^A	Study Group Dataset Description	Blood Pb Levels			N	Additional Information on Analyses
		Age	Mean ^B (µg/dL)	Range ^B (µg/dL)		
<i>^CStudies discussed in ISA (section 5.3.2) with findings of effects on neurocognitive measures reported for childhood study group PbB ≤ 5 µg/dL (ordered by age of blood Pb measurements)</i>						
FSIQ	Boston, prospective, age 5 yr Subgroup with peak PbB <10 µg/dL Bellinger and Needleman 2003	24 mo	3.8	1 - 9.3	48	Regression, PbB as continuous variable; statistically significant negative association
BSID/MDI	Mexico City, age 24 mo, Tellez-Rojo et al., 2006 Subgroup with PbB <5 µg/dL Full dataset	24 mo " "	2.9 4.3	0.8 – 4.9 0.8 - 9.8	193 294	Regression, PbB as continuous variable; statistically significant negative association
AcadPerf	North Carolina, 4 th graders 4 th grade reading scores, Full dataset Miranda et al., 2009	9-36 mo	4.8	1-16	57,678	Linear and quantal regressions with integer PbB as categorical variable (PbB-1 =reference category, includes LOD). Linear analysis: statistically significant effect for all comparison categories. Quantal analysis: statistically significant effect in all reading score quantiles for PbB categories greater than integer PbB=3; largest effect in lower quantiles. Means not reported for PbB categories.
AcadPerf	Avon, United Kingdom, age 7-8 yr standard assessment scores, Full dataset, Chandramouli et al., 2009	30 mo	4.22	21% ≤ 2 52% 2-5 21% 5-10	488 ^D	Regressions with PbB as continuous and categorical variables: continuous analysis (statistically significant negative association); categorical (significantly reduced scores for 5-10 µg/dL vs reference group [0-2 µg/dL])
FSIQ	Rochester, prospective cohort, age 5 yr Subgroup with peak PbB <10 µg/dL Canfield et al., 2003	5 yr	3.32	0.5 – 8.4 (LOD=1)	71	Regression, PbB as continuous variable; statistically significant negative association
FSIQ	Pooled International, age 6-10 yr Subgroup with peak PbB <7.5 µg/dL Lanphear et al 2005 ^E	5-7 yr	3.24	0.9 – 7.4	103	Regression, PbB as continuous variable; statistically significant negative association
FSIQ LM, EF AcadPerf	New England, 2 areas, age 6-10, , Full dataset, Surkan et al., 2007	6-10 yr	2.3	1 – 10	389	Regression, PbB grouped into categories; statistically significant negative association for high subgroup (5-10 µg/dL, n=32) compared to reference PbB subgroup (1-2 µg/dL, n=286).
FSIQ	Korea, 4 areas, age 8-11 yr Full dataset Kim et al., 2009	8-11 yr	1.73	0.4 – 4.9	261	Regression, PbB analyzed as quartiles; statistically significant difference among quartiles; statistically significant negative association in continuous analysis of full dataset and high blood manganese group
LM AcadPerf	NHANES III (1988-1994) Lanphear et al., 2000	6-16 yr	1.9		4,853	Continuous (unadjusted) and categorical (adjusted) analysis; significant difference among PbB quartiles (< 1, 1.1-1.9, 2.0-3.0, >3.0 µg/dL), and neg assoc PbB <5.0.
LM AcadPerf	NHANES III (1991-1994) Krieg et al., 2010	12-16 yr	1.95		766-80	Regression analysis; PbB as continuous variable; statistically significant negative association.
<p>A - FSIQ = Full Scale Intelligence Quotient; BSID = Bayley Scales of Development; MDI= Mental Development Index; LM=Learning and Memory; EF= Executive Function; AcadPerf= Academic Performance.</p> <p>B - Blood Pb level (PbB) information provided here is in some cases augmented by study authors (Bellinger, 2008; Canfield, 2008a,b; Hornung, 2008a,b; Tellez-Rojo, 2008).</p> <p>C - Bolded measures and studies are newly available in this review.</p> <p>D - In practice, 337-425 cases were included in analysis (Chandramouli et al., 2009).</p> <p>E - Blood Pb measurements of subgroup with peak PbB <7.5 µg/dL comprised of 13.6% age 5 (Boston, Cleveland), 67.0% age 6 (Rochester), 19.4% age 7 (Yugoslavia, Mexico, Cincinnati). IQ assessed at age 5 for the single member of this subgroup from Cleveland cohort. This analysis includes blood Pb data from Rochester and Boston cohorts, although for different ages (6 and 5 years, respectively) than the ages analyzed in Canfield et al 2003 and Bellinger and Needleman 2003. For full dataset analysis (n=1333), IQ assessed at ages 5-10 yr, median blood Pb (at ages 5-7 yr) of 9.7 µg/dL and 5th -95th percentile of 2.5-33.2 µg/dL.</p>						

1 Among the analyses of lowest study group blood Pb levels at the youngest ages in Table
2 3-2 are analyses available in the last review of Pb associations with neurocognitive decrement in
3 study groups with mean levels on the order of 3-4 µg/dL in children aged 24 months, or ranging
4 from 5 to 7 years (73 FR 66978-66969; ISA, section 5.3.2.2; Bellinger and Needleman, 2003;
5 Canfield et al., 2003; Lanphear et al., 2005; Tellez-Rojo et al., 2006; Bellinger, 2008; Canfield,
6 2008; Hornung, 2008; Tellez-Rojo, 2008).¹⁸ Newly available in this review are two studies
7 reporting association of blood Pb levels prior to three years of age with academic performance
8 on standardized tests in primary school; mean blood Pb levels in these studies were 4.2 and 4.8
9 µg/dL (ISA, section 5.3.2.5; Chandramouli et al., 2009; Miranda et al., 2009). One of these two
10 studies, which represented integer blood Pb levels as categorical variables, indicated a small
11 effect on end-of-grade reading score of blood Pb levels as low as 2 µg/dL, after adjustment for
12 age of measurement, race, sex, enrolled in free or reduced lunch program, parental education,
13 and school type (Miranda et al., 2009).

14 In a newly available study of blood Pb levels at primary school age, a significant
15 association of blood Pb in children aged 8-11 years and concurrently measured full scale IQ
16 (FSIQ) was reported for a cross-sectional cohort in Korea with a mean blood Pb level of 1.7
17 µg/dL and range of 0.43-4.91 µg/dL (Kim et al., 2009).¹⁹ In considering the blood Pb levels in
18 this study, we note that blood Pb levels in children aged 8-11 are generally lower than those in
19 pre-school children, for reasons related to behavioral and other factors (ISA, sections 4.3.5, 4.4.1
20 and 6.2.1.1).²⁰ It is likely that the blood Pb levels of this study group at earlier ages, e.g., prior to
21 school entry, were higher and the available information does not provide a basis to judge whether
22 the blood Pb levels in this study represent lower exposure levels than those experienced by the
23 younger study groups. In still older children, a large cross-sectional investigation of blood Pb
24 association with effects on memory and learning, that was available in the last review was
25 focused on children aged 6-16 years, born during 1972-1988, with a mean blood Pb of 1.9
26 µg/dL. A study newly available in this review, focused on a subset of the earlier study cohort
27 (ages 12-16, born during 1975-1982), also reports a significant negative association of blood Pb

¹⁸ The tests for cognitive function in these studies include age-appropriate Wechsler intelligence tests (Lanphear et al., 2005; Bellinger and Needleman, 2003), the Stanford-Binet intelligence test (Canfield et al., 2003), and the Bayley Scales of Infant Development (Tellez-Rojo et al., 2006). The Wechsler and Stanford-Binet tests are widely used to assess neurocognitive function in children and adults, however, these tests are not appropriate for children under age three. For such children, studies generally use the age-appropriate Bayley Scales of Infant Development as a measure of cognitive development.

¹⁹ Limitations of this study included a lack of consideration of potential confounding by parental caregiving quality or IQ (ISA, Table 5-3).

²⁰ This study also investigated the potential role of manganese (Mn) in the blood Pb associated effects. When the cohort was subdivided based on Mn blood Pb levels, using the median Mn level (14 µg/L) as the break point, the significant association of intelligence quotient (IQ) with Pb persisted in the higher Mn group but was no longer significant in the lower Mn group (ISA, section 5.3.2). Separate analysis of the full study group found a significant negative association of IQ with blood Mn (Kim et al., 2009).

1 with learning and memory test results with mean blood Pb levels of approximately 2 µg/dL (ISA,
2 section 5.3.2.3; Lanphear et al., 2000; Krieg et al., 2010). In considering these study findings
3 with regard to the question of exposure levels eliciting effects, we recognize, however, that blood
4 Pb levels are, in general, lower among teenagers than young children and also that, for these
5 subjects specifically, the magnitude of blood Pb levels during the earlier childhood (e.g., pre-
6 school ages) was much higher. For example, the mean blood Pb levels for the 1-5 year old age
7 group in the NHANES 1976-80 sample was 15 µg/dL, declining to 3.6 µg/dL in the NHANES
8 1988-1991 sample (Pirkle et al., 1994; ISA, section 4.4.1).

9 With regard to other nervous system effects in children, the evidence base at lower blood
10 Pb levels is somewhat extended since the last review with regard to the evidence on Pb and
11 attention-related behavioral problems, such as inattention, impulsivity, hyperactivity and
12 attention deficit hyperactivity disorder (ISA, section 5.3.3 and table 5-17). Several newly
13 available studies investigating the role of blood Pb levels in older children (primary school age
14 and older) have reported significant associations of attention-related behavior or conduct
15 problem measures with concurrent blood Pb levels, with mean levels generally on the order of 5
16 µg/dL or higher (ISA, section 5.3.3). One exception is the newly available cross-sectional,
17 categorical analysis of the NHANES 2001-2004 sample of children aged 8-15 years, which
18 found higher prevalence of conduct disorder in the subgroup with concurrent blood Pb levels of
19 0.8-1.0 µg/dL as compared to the <0.8 µg/dL group (ISA, section 5.3.4 and Table 5-12). As
20 noted above, we recognize that many of these children, born between 1986 and 1996, are likely
21 to have had much higher Pb exposures (and associated blood Pb levels) in their earlier years than
22 those commonly experienced by young children today, thus precluding a conclusion regarding
23 evidence of effects associated with lower exposure levels than provided by evidence previously
24 available.

25 As summarized earlier in this section, blood Pb has been associated with a range of health
26 effects on multiple organ systems or processes. As is the case for studies of nervous system
27 effects in children, newly available studies of other effects in child and adult cohorts include
28 cohorts with similar or somewhat lower mean blood levels than in previously available studies.
29 Categories of effects for which a causal relationship has been concluded in the ISA and for
30 which there are a few newly available epidemiological studies indicating blood Pb associations
31 with effects in study groups with somewhat lower blood Pb levels than previously available for
32 these effects include effects on development (delayed puberty onset) and reproduction (male
33 reproductive function) and on the cardiovascular system (hypertension) (ISA, sections 5.4 and
34 5.8; 2006 CD, sections 6.5 and 6.6). With regard to the former category, study groups in the
35 newly available studies include groups comprised of older children ranging up to age 18 years,
36 for which there is increased uncertainty regarding historical exposures and their role in the

1 observed effects.²¹ An additional factor that handicaps our consideration of exposure levels
2 associated with these findings is the appreciable uncertainty associated with our understanding of
3 Pb biokinetics during this lifestage (ISA, sections 4.3, 5.8.6). The evidence newly available for
4 Pb relationships with cardiovascular effects in adults include some studies with somewhat lower
5 blood Pb levels than in the last review. The long exposure histories of these cohorts, as well as
6 the generally higher Pb exposures of the past complicate conclusions regarding exposure levels
7 that may be eliciting observed effects (ISA, sections 5.4.2.4 and 5.4.7).²²

8 In summary, our conclusions regarding exposure levels at which Pb health effects occur,
9 and particularly with regard to such levels that might be common in the U.S. today, are
10 complicated now, as in the last review, by several factors. These factors include the scarcity of
11 information in epidemiological studies on cohort exposure histories, as well as by the backdrop
12 of higher past exposure levels which frame the history of some study cohorts. Recognizing the
13 complexity, as well as the potential role of higher exposure levels in the past, we continue to
14 focus our consideration of this question on the evidence of effects in young children for which
15 our understanding of exposure history is less uncertain.²³ Within this evidence base, we
16 recognize the lowest study group blood Pb levels to be associated with effects on cognitive
17 function measures, indicating that to be the most sensitive endpoint. As described above, and
18 summarized in Table 3-2, the evidence available in this review is generally consistent with that
19 available in the last review with regard to blood Pb levels at which such effects had been
20 reported (ISA, section 5.3.2; 2006 CD, section 8.4.2.1; 73 FR 66976-66979). As blood Pb levels
21 are a reflection of exposure history, particularly in early childhood (ISA, section 4.3.2), we
22 conclude, by extension, that the currently available evidence does not indicate Pb effects at
23 exposure levels appreciably lower than recognized in the last review.

24 We additionally note that, as in the last review, a threshold blood Pb level with which
25 nervous system effects, and specifically neurodevelopmental effects, occur in young children
26 cannot be discerned from the currently available studies (ISA, sections 2.9.4 and 5.3.13).
27 Epidemiological analyses have reported blood Pb associations with neurocognitive effects (FSIQ
28 or BSID MDI²⁴) in young child population subgroups, which included individual blood Pb

²¹ Several of these studies involve NHANES III cohorts for which early childhood exposures were generally much higher than those common in the U.S. today (ISA, section 5.8.6).

²² Studies from the late 1960s and 1970s suggest that adult blood Pb levels during that period ranged from roughly 10 to 30 µg/m³ (ISA, section 5.4.1).

²³ In focusing on effects associated with blood Pb levels in early childhood, however, we additionally recognize the evidence across categories of effects that relate to blood Pb levels in older child study groups (for which early childhood exposure may have had an influence) which provides additional support to an emphasis nervous system effects (ISA, sections 5.3, 5.4, 5.5, 5.6, 5.7, 5.8).

²⁴ Bayley Scales of Infant Development, Mental Development Index. The Bayley MDI is a well-standardized and widely used assessment measure of infant cognitive development. Scores earlier than 24 months are not necessarily correlated with later FSIQ scores in children with normal development (ISA, section 5.3.16.1).

1 measurements as low as approximately 1 µg/dL and mean concentrations as low as 2.9 to 3.8
2 µg/dL (ISA, section 5.3.13; Bellinger and Needleman, 2003; Bellinger, 2008; Canfield et al.,
3 2003; Canfield, 2008; Tellez-Rojo et al., 2006; Tellez-Rojo et al., 2008). As concluded in the
4 ISA, however, “the current evidence does not preclude the possibility of a threshold for
5 neurodevelopmental effects in children existing with lower blood levels than those currently
6 examined” (ISA, section 5.3.13).

7 Important uncertainties associated with the evidence of effects at low exposure levels are
8 similar to those recognized in the last review, including the shape of the concentration-response
9 relationship for effects on neurocognitive function at low blood Pb levels in today’s young
10 children. Also of note is our interpretation of associations between blood Pb levels and effects in
11 epidemiological studies, with which we recognize uncertainty with regard to the specific
12 exposure circumstances (timing, duration, magnitude and frequency) that have elicited the
13 observed effects, as well as uncertainties in relating ambient air concentrations (and associated
14 air-related exposures) to blood Pb levels in early childhood, as discussed in section 3.1 above.
15 We additionally recognize uncertainties associated with conclusions drawn with regard to the
16 nature of the epidemiological associations with blood Pb (e.g., ISA, section 5.3.14), but note that
17 based on consideration of the full body of evidence for neurocognitive effects, the EPA has
18 determined a causal relationship to exist between relevant blood Pb levels and neurocognitive
19 impacts in children (ISA, section 5.3.16.1).

- 20 • **To what extent does the newly available evidence alter our understanding of the**
21 **concentration-response relationship for neurocognitive effects (IQ) with blood Pb**
22 **levels in young children?**

23 Based primarily on studies of FSIQ, the assessment of the currently available studies, as
24 was the case in the last review, continues to recognize a nonlinear relationship between blood Pb
25 and effects on cognitive function, with a greater incremental effect (greater slope) at lower
26 relative to higher blood Pb levels within the range thus far studied, extending from well above 10
27 µg/dL to below 5 µg/dL (ISA, section 5.3.13). This was supported by the evidence available in
28 the last review, including the analysis of the large pooled international dataset comprised of
29 blood Pb measurements concurrent with the IQ tests that ranged from 2.5 µg/dL to 33.2 µg/dL
30 (Lanphear et al., 2005; Rothenberg and Rothenberg, 2005; ISA, section 5.3.13). The study by
31 Lanphear et al (2005) additionally presented analyses that stratified the dataset based on peak
32 blood Pb levels (e.g., with cutpoints of 7.5 µg/dL and 10 µg/dL peak blood Pb) and found that
33 the coefficients from linear models of the association for IQ with concurrent blood Pb were
34 higher in the lower peak blood Pb level subsets than the higher groups (ISA, section 5.3.13;
35 Lanphear et al., 2005). Stratified analyses of several individual cohorts also observed higher
36 coefficients for blood Pb relationships with measures of neurocognitive function in lower as

1 compared to higher blood Pb subgroups (ISA, section 5.3.13; Canfield et al., 2003; Bellinger and
 2 Needleman, 2003; Kordas et al., 2006; Tellez-Rojo et al., 2006). Of these subgroup analyses,
 3 those involving the lowest mean blood Pb levels and closest to the current mean for U.S.
 4 preschool children are listed in Table 3-3 below (drawn from Table 3 of the 2008 final
 5 rulemaking notice [73 FR 67003]). These analyses were important inputs for the evidence-
 6 based, air-related IQ loss framework which informed decisions on a revised standard in the last
 7 review (73 FR 67005), discussed in section 4.1.1 below.

8 **Table 3-3. Summary of quantitative relationships of IQ and blood Pb for analyses with**
 9 **blood Pb levels closest to those of young children in the U.S. today.**

Blood Pb Levels (µg/dL)		Study/Analysis	Average Linear Slope ^A (IQ ^B points per µg/dL)
Geometric Mean	Range (min-max)		
2.9	0.8 – 4.9	Tellez-Rojo et al (2006) ^B , subgroup with concurrent blood Pb <5 µg/dL	-1.71
3.24	0.9 – 7.4	Lanphear et al (2005) ^C , subgroup with peak blood Pb <7.5 µg/dL	-2.94
3.32	0.5 – 8.4	Canfield et al (2003) ^C , subgroup with peak blood Pb <10 µg/dL	-1.79
3.8	1 - 9.3	Bellinger and Needleman (2003) ^C , subgroup with peak blood Pb <10 µg/dL	-1.56
Median value			-1.75
A - Average linear slope estimates here are generally for relationship with IQ assessed concurrently with blood Pb measurement. As exceptions, Bellinger & Needleman (2003) slope is relationship for 10 year old IQ with blood Pb levels at 24 months, and the data for Boston cohort included in Lanphear et al 2005 slope are relationship for 10 year old IQ with blood Pb levels at 5 years. B - The slope for Tellez-Rojo et al 2006 is for BSID (MDI), a measure of cognitive development appropriate to study population age (24-mos). C - The Lanphear et al. (2005) pooled International study also includes blood Pb data from the Rochester and Boston cohorts, although for different ages (6 and 5 years, respectively) than the ages analyzed in Canfield et al. (2003) and Bellinger and Needleman (2003). Thus, the ages at the blood Pb measurements used in derivation of the linear slope for the Lanphear et al (2005) subgroup shown here are 5 to 7 years.			

10
 11 Several studies newly available in the current review have, in all but one instance, also
 12 found a nonlinear blood Pb-cognitive function relationship in nonparametric regression analyses
 13 of the cohort blood Pb levels analyzed (ISA, section 5.3.13). These studies, however, used
 14 statistical approaches that did not produce quantitative results for each blood Pb group (ISA,
 15 section 5.3.13). Thus, newly available studies have not extended the range of observation for
 16 quantitative estimates of this relationship to lower blood Pb levels than those of the previous
 17 review. The ISA further notes that the potential for nonlinearity has not been examined in detail
 18 within a lower more narrow range of blood Pb levels than those of the full cohorts thus far
 19 studied in the currently available evidence base (ISA, section 5.3.13). Such an observation in the
 20 last review supported the consideration of linear slopes with regard to blood Pb levels at and
 21 below those represented in Table 3-3. In summary, the newly available evidence does not
 22 substantively alter our understanding of the concentration-response relationship (including
 23 quantitative aspects) for neurocognitive impact, such as IQ with blood Pb in young children.

3.3 PUBLIC HEALTH IMPLICATIONS AND AT-RISK POPULATIONS

There are several potential public health impacts associated with Pb exposure. In recognition of effects causally related to blood Pb levels somewhat near those most recently reported for today's population and for which the weight of the evidence is greatest, the potential public health impacts most prominently recognized in the ISA are population IQ impacts associated with childhood Pb exposure and prevalence of cardiovascular effects in adults (ISA, section 2.9.1). With regard to the latter category, as discussed above, the full body of evidence indicates a role of long-term cumulative exposure, with uncertainty regarding the specific exposure circumstances contributing to the effects in the epidemiological studies which focused largely on adult populations for whom historical Pb exposures were likely much higher than exposures that commonly occur today (ISA, section 5.4). There is less uncertainty regarding the exposure patterns contributing to the blood Pb levels reported in studies of younger populations (ISA, section 2.10). Accordingly, we focus the discussion of public health implications relevant to this review predominantly on nervous system effects, including IQ decrements, in children.

We focus this discussion on IQ in recognition of IQ being a well established, widely recognized and rigorously standardized measure of neurocognitive function, as well as a global measure reflecting the integration of numerous processes (ISA, section 5.3.2; 2006 CD, sections 6.2.2 and 8.4.2). We recognize, however, that IQ is one of several measures of cognitive function negatively associated with Pb exposure. Other examples include other tests of intelligence and cognitive development, and tests of other cognitive abilities, such as learning, memory, and executive functions, as well as academic performance and achievement (ISA, section 5.3.2). In considering the public health significance of neurocognitive effects of Pb in children, we recognize that, although some may be transient, some effects may persist into adulthood (ISA, section 2.9.5).²⁵ We also note that deficits in neurodevelopment early in life may have lifetime consequences as “[n]eurodevelopmental deficits measured in childhood may set affected children on trajectories more prone toward lower educational attainment and financial well-being” (ISA, section 5.3.15). Thus, population groups for which neurodevelopment is affected by Pb exposure in early childhood are at risk of related impacts on their success later in life.

There are important distinctions between population and individual risk such that “small shifts in population means are often significant from a public health perspective” (ISA, p. 2-63). For example, if lead-related decrements are manifested uniformly across the range of IQ scores

²⁵ The ISA states that the “persistence of effects appears to depend on the duration and window of exposure as well as other factors that may affect an individual's ability to recover from an insult”, with some evidence of greater recovery in children reared in households with more optimal caregiving characteristics and low concurrent blood Pb levels (ISA, section 2.9.5; Bellinger et al., 1990).

1 in a population, “a small shift in the population mean IQ may result in a substantial increase in
2 the number of individuals functioning in the low range of the IQ distribution, which is associated
3 with increased risk of educational, vocational, and social failure” as well as a decrease in the size
4 of the subgroup achieving very high scores (ISA, section 2.9.1).

5 In the discussion below, we use the term at-risk populations to recognize populations that
6 have a greater likelihood of experiencing lead-related health effects, i.e., groups with
7 characteristics that contribute to an increased risk of Pb-related health effects. These populations
8 are also sometimes referred to as sensitive groups, as in section 1.2.1 above. This increased
9 likelihood of lead-related effects can result from many factors, including lifestage or age, gender,
10 race or ethnicity, diet, pre-existing disease state, or increased exposure (ISA, chapter 6).
11 Accordingly, in identifying factors that increase risk of lead-related health effects, there has been
12 consideration of evidence regarding factors contributing to increased susceptibility (i.e.,
13 physiological or intrinsic factors contributing to a greater response for the same exposure), and
14 those contributing to increased exposure (including that resulting from behavior leading to
15 increased contact with contaminated media). As noted in the ISA, “definitions of susceptibility
16 and vulnerability vary across studies, but in most instances ‘susceptibility’ refers to biological or
17 intrinsic factors (e.g., age, sex) while ‘vulnerability’ refers to nonbiological or extrinsic factors
18 (e.g., socioeconomic status [SES])” and the terms “at-risk” and “sensitive” populations have in
19 various instances been used to encompass these concepts more generally (ISA, p. 6-1). Although
20 we emphasize the term “at-risk”, we rely on the other terms in particular instances below; in so
21 doing, our usage is consistent with these definitions.

22 Factors that increase risk of lead-related effects include, among others, behavioral
23 physiological factors. A behavioral factor of great impact on Pb exposure is the incidence of
24 hand-to-mouth activity that is prevalent in very young children and by which they transfer Pb in
25 settled particles to their mouths (ISA, sections 4.7.1 and 6.2.1.1). Physiological factors include
26 both conditions contributing to a group’s increased risk of effects at a given blood Pb level, and
27 those that contribute to blood Pb levels higher than those otherwise associated with a given Pb
28 exposure (ISA, sections 6.3 and 6.1, respectively). We also considered evidence encompassing
29 situations of elevated exposure, such as residing in old housing with Pb-containing paint or near
30 sources of ambient Pb, as well as socioeconomic factors, such as reduced access to health care or
31 low socioeconomic status (SES) that can contribute to increased risk of adverse health effects
32 from Pb (ISA, sections 2.9.7, 6.2, and 6.4).

33

1 • **Has new information altered our understanding of human populations that are**
2 **particularly at risk of health effects from Pb exposures?**

3 The information newly available in this review has not substantially altered our
4 understanding of at-risk populations. As in the last review, the factor most prominently
5 recognized to contribute to increased risk of Pb effects is age (ISA, section 2.9.7). As noted in
6 section 3.2 above, although the specific ages or lifestages of greatest susceptibility have not been
7 established (e.g., ISA, section 5.3.12), the susceptibility of young children to the
8 neurodevelopmental effects of Pb is well recognized (e.g., ISA, sections 2.9.7, 5.3, 6.2.1, 6.3.1,
9 and 6.4). The evidence indicates that prenatal blood Pb levels are causally associated with
10 nervous system effects, including mental development in very young children and can also be
11 associated with cognitive decrements in older children (ISA, section 5.3). The coincidence
12 during early childhood of behaviors that increase exposure, such as hand-to-mouth contact, and
13 the development of the nervous system contributes increased risk during this time (ISA, sections
14 5.3.2.6, 6.2.1.1, and 6.3.1.1). The evidence also indicates a causal relationship of postnatal blood
15 Pb levels (through early childhood to school age) with cognitive function decrement in older
16 children and adolescence (ISA, section 5.3). In epidemiological studies, associations have been
17 observed of neurocognitive, and some other nervous system effects, at various ages from early
18 childhood to school age with prenatal, early-childhood, lifetime average, and concurrent blood
19 Pb levels as well as with childhood tooth Pb levels (ISA, section 5.3). Consideration of
20 epidemiological study results for different lifestages of exposure, particularly later in childhood,
21 is complicated by the fact that blood Pb levels in children, although highly affected by recent
22 exposure, are also influenced by their history of Pb exposure due to rapid growth-related bone
23 turnover in children (ISA, section 4.3.5). Thus, blood Pb level in children also may reflect past
24 Pb exposures and, to some extent, maternal Pb, with relative contributions varying with child and
25 maternal exposure history (ISA, section 4.2.2.4, 4.4.1 and 5.3.16; 2006 CD, section 6.6.2).
26 Collectively, however, the evidence indicates both the susceptibility of the developing fetus and
27 early postnatal years, as well as the potential for continued susceptibility through childhood as
28 the human central nervous system continues to mature and be vulnerable to neurotoxicants (ISA,
29 sections 2.9.5 and 5.3.16; 2006 CD, section 6.2.12).

30 In the collective body of evidence of nervous system effects in children, it is difficult to
31 distinguish exposure in later lifestages (e.g., school age), and its associated risk, from prenatal
32 and early childhood (ISA, section 5.3.12). While early childhood is recognized as a time of
33 increased susceptibility, a difficulty in identifying a discrete period of susceptibility from
34 epidemiological studies has been that the period of peak exposure, reflected in peak blood Pb
35 levels, is around 18-27 months when hand-to-mouth activity is at its maximum (ISA, section
36 4.4.1 and 6.2.1.1; 2006 CD, p. 6-60). The task is additionally complicated by the role of

1 maternal exposure history in contributing Pb to the developing fetus (ISA, section 4.2.2.4.).
2 Epidemiological analyses evaluating risk of neurocognitive impacts (e.g., reduced IQ) associated
3 with different blood Pb metrics in cohorts with differing exposure patterns (including those for
4 which blood Pb levels at different ages were not highly correlated) indicate associations with
5 blood Pb measurements concurrent with FSIQ tests at ages of approximately 6-7 years, although
6 the analyses did not conclusively demonstrate stronger findings for early (e.g., age 2 years) or
7 concurrent blood Pb (ISA, section 5.3.12). The experimental animal evidence additionally
8 indicates early life susceptibility (ISA, section 5.3.16). Thus, the full evidence base continues to
9 indicate prenatal and early childhood lifestages as periods of increased lead-related risk. In
10 summary, while uncertainties remain with regard to the role of Pb exposures during a particular
11 age of life in eliciting nervous system effects, such as cognitive function decrements, the
12 evidence continues to indicate the at-risk status of pre- and postnatal childhood lifestages (ISA,
13 sections 5.3.12 and 5.3.16).

14 Several physiological factors increase risk of lead-related health effects by contributing to
15 increased blood Pb levels over those otherwise associated with a given Pb exposure (ISA,
16 sections 4.2, 4.3 and 6.1). These include nutritional status, which plays a role in Pb absorption
17 from the GI tract (ISA, section 4.2.1.2). For example, diets deficient in iron, calcium or zinc can
18 contribute to increased Pb absorption and associated higher blood Pb levels (ISA, sections
19 4.2.1.2, and 6.1). Evidence is suggestive of some genetic characteristics as potential risk factors,
20 such as presence of the δ -aminolevulinic acid dehydratase-2 (ALAD-2) allele which has been
21 indicated to increase blood Pb levels and or lead-related risk (ISA, sections 4.3.2 and 6.1).

22 Risk factors based on increased exposure include spending time in proximity to sources
23 of Pb to ambient air or other environmental media (e.g., large active metals industries or
24 locations of historical Pb contamination) (ISA, section 2.9.7, 6.2.5). Residential factors
25 associated with other sources of Pb exposure (e.g., leaded paint or plumbing with Pb pipes or
26 solder) are another exposure-related risk factor (ISA, section 2.9.7, 6.2.6). The role of
27 socioeconomic status (SES) with regard to lead-related risk is somewhat complicated. SES often
28 serves as a marker term for one or a combination of unspecified or unknown environmental or
29 behavioral variables. Lower SES has been associated with higher Pb exposure and higher blood
30 Pb concentration (ISA, sections 5.3.16, 6.2.4 and 6.4). Further, it is independently associated
31 with an adverse impact on neurocognitive development and a few studies have examined SES as
32 a potential modifier of the association of childhood Pb exposure with cognitive function with
33 inconsistent findings regarding low SES as a potential risk factor. Although the differences in
34 blood Pb levels among children of lower as compared to higher income levels have lessened,
35 blood Pb levels continue to be higher among lower income children indicating higher exposure

1 and/or greater influence of factors independent of exposure, such as nutritional factors (ISA,
2 sections 2.9.7, 6.2.1.1 and 6.4).

3 In considering risk factors associated with increased Pb exposure or increased blood Pb
4 levels, we note that the currently available evidence continues to support a nonlinear relationship
5 between neurocognitive effects and blood Pb that indicates incrementally greater impacts at
6 lower as compared to higher blood Pb levels (ISA, section 5.3.13), as described in section 3.2
7 above. An important implication of this finding is that while children with higher blood Pb
8 levels are at greater risk of lead-related effects than children with lower blood Pb levels, on an
9 incremental basis (e.g., per $\mu\text{g}/\text{dL}$), the risk is greater for children at lower blood Pb levels. This
10 was given particular attention in the last review of the Pb NAAQS, in which the standard was
11 revised with consideration of the incremental impact of air-related Pb on young children in the
12 U.S and the recognition of greater impact for those children with lower absolute blood Pb levels
13 (73 FR 67002). Such consideration included a focus on those C-R studies involving the lowest
14 blood Pb levels, as described in section 4.1.1 below.

15 Some racial or ethnic backgrounds have been identified as factors that may increase risk
16 of lead-related health effects (ISA, sections 2.9.7, 6.2.3 and 6.3.7). For example, although blood
17 Pb levels in the U.S. general population (e.g., geometric mean level in children aged 1-5) have
18 declined, mean levels reported in recent NHANES samples continue to differ among children of
19 different ethnic backgrounds, with higher levels in non-white persons as compared to whites
20 (ISA, sections 4.4.1, 6.2.1.1 and 6.2.3). Additionally, a study of lead-related risk of hypertension
21 among adults reported greater risk associated with blood Pb levels above 1 $\mu\text{g}/\text{dL}$ among
22 Mexican Americans and non-Hispanic blacks than among non-Hispanic whites (ISA, section
23 6.3.7; Muntner et al., 2005). The evidence available in the current review, consistent with that in
24 the last, also suggests that health status or pre-existing disease is potentially a physiological risk
25 factor for lead-related effects (ISA, section 2.9.7). Populations with pre-existing health
26 conditions, such as hypertension, may be more susceptible (as compared to the general
27 population) for particular Pb-associated effects (ISA, section 6.3.4). For example, increased risk
28 of lead-related renal effects and heart rate variability have been reported among hypertensive
29 individuals compared to those that are normotensive (ISA, section 2.9.7). Additionally, African
30 Americans, as a group, have a higher frequency of hypertension than the general population or
31 other ethnic groups (NCHS, 2011), and as a result may face a greater risk of adverse health
32 impact from Pb-associated cardiovascular effects.

33 Older adulthood has been identified as a lifestage of potentially greater risk of lead-
34 related health effects based primarily on the evidence of increases in blood Pb levels during this
35 lifestage, and of potential for effects, such as (ISA, sections 6.2.1.2, 6.3.1.2, and 6.4).

36 Contributing to blood Pb levels in the studied populations of older adults are likely to be their

1 exposure histories, which included younger years during the time of leaded gasoline usage and
2 other sources of Pb exposures which were more prevalent in the past than today (e.g., ISA,
3 Figure 3-1 and section 3.4.3.2). Exposure history has a contributing role to blood Pb levels
4 throughout life, and the increased rate of bone remodeling during later adulthood increases
5 contributions of Pb from bone stores into the systemic circulation during that lifestage (ISA,
6 sections 4.3.5 and 6.2.1.2). Additionally, limited animal evidence has indicated specific brain
7 pathology in older animals that had substantial Pb exposures earlier in life (ISA, sections
8 5.3.10.1 and 6.3.1). Further, the full body of evidence includes observed associations of various
9 health effects, such cardiovascular and neurological effects with bone and blood Pb in older
10 populations, with biological plausibility for the role of Pb provided by experimental animal
11 studies (ISA, sections 5.3.6, 5.3.8, 5.3.10 and 5.4).

12 In summary, the information newly available in this review has not appreciably altered
13 our understanding of human populations that are particularly sensitive to Pb exposures. In the
14 current review, as at the time of the last review of the Pb NAAQS, we recognize young children
15 as an important at-risk population, with sensitivity extending to prenatal exposures and into
16 childhood development. Additional risk factors include deficiencies in dietary minerals (iron,
17 calcium and zinc), some racial or ethnic backgrounds, and spending time in proximity to
18 environmental sources of Pb or residing in older houses. The evidence for SES continues to
19 indicate increased blood Pb levels in lower income children, although its role with regard to an
20 increased risk for same blood Pb level is unclear. Additionally, the currently available evidence
21 continues to indicate a potential for increased risk associated with several other factors, including
22 older adulthood, pre-existing disease (e.g., hypertension), variants for certain genes and
23 increased stress.

- 24 • **Is there new evidence on health effects beyond neurocognitive endpoints in**
25 **children that suggest additional at-risk populations should be given increased**
26 **focus in this review?**

27 The evidence newly available in this review supports or strengthens our previous
28 conclusions regarding the broad array of health effects of Pb (see ISA, section 2.10 which
29 compares key conclusions drawn in the last review with conclusions drawn in the current
30 assessment). Additionally, in some categories of health effects, the newly available studies
31 extend the evidence for some aspects of the health effects described in the last review. For
32 example, among the nervous system effects, the newly available evidence continues to support
33 the conclusions from the last review regarding Pb and neurocognitive and neurobehavioral
34 effects (ISA, section 5.3). Across the array of neurocognitive and behavioral effects, we
35 recognize the sensitivity of the prenatal period and several lifestages of childhood, and

1 particularly recognize young children as an important at-risk population in light of current
2 environmental exposure levels.

3 As discussed in section 3.2 above, the blood Pb levels of populations studied in newly
4 available epidemiological studies that report associations of blood Pb with effects for systems
5 and processes other than the nervous system (e.g., cardiovascular, developmental and
6 reproductive) are similar, or in a few cases, somewhat lower than those assessed in the last
7 review (ISA, sections 5.4, 5.6, 5.7, and 5.8). The greater uncertainties regarding the time,
8 duration and magnitude of exposure contributing to these observed health effects complicate
9 identification of sensitive lifestages and associated exposure patterns that might be compared
10 with our understanding of the sensitivity of young children to neurocognitive impacts of Pb.
11 Thus, while augmenting the evidence base on these additional endpoints, the newly available
12 evidence does not lead us to identify a health endpoint expected to be more sensitive to Pb
13 exposure than neurocognitive endpoints in children, leading us to continue to conclude that the
14 appropriate primary focus for our review is on neurocognitive endpoints in children.

15 In summary, there are a variety of ways in which lead-exposed populations might be
16 characterized and stratified for consideration of public health impacts. Age or lifestage was used
17 to distinguish potential groups on which to focus in the last review in recognition of its role in
18 exposure and susceptibility, and young children were selected as the priority population for the
19 risk assessment (see section 3.4 below) in consideration of the health effects evidence regarding
20 endpoints of greatest public health concern and in recognition of effects on the developing
21 nervous system as a sentinel endpoint for public health impacts of Pb. This identification
22 continues to be supported by the evidence available in the current review.

- 23 • **What does the information about air Pb concentrations available in this review**
24 **indicate with regard to the size of at-risk populations and their distribution**
25 **across the U.S.?**

26 The magnitude of a public health impact is dependent upon the size of populations
27 affected, as well as type or severity of the effect. As summarized above, the population group
28 that may be most at risk of health effects associated with exposure to Pb is young children. The
29 2010 census indicates nearly 310 million people residing in the U.S., 74 million of which
30 approximately are children under the age of 18, with some 20 million under the age of five years.
31 Children at greatest risk from air-related Pb are considered to be those children living in areas of
32 higher air Pb exposures. Accordingly, the discussion below considers the information available
33 to inform our understanding of areas of children potentially at risk from air-related Pb.

34 In considering the extent of this at-risk population, we turn first to consideration of those
35 areas in the U.S. with air Pb concentrations above the current standard (e.g., section 2.2.2.2
36 above). Using the available monitoring data and U.S. census information, Table 3-4 summarizes

1 the size of populations within 0.5 km of monitors in our current Pb NAAQS surveillance
 2 network at which Pb concentrations were higher than the current standard during the recent
 3 period from 2009-2011. The distance to which concentrations exceeding the standard might
 4 extend will vary with the magnitude of the Pb concentrations and particle size, among other
 5 factors; a half kilometer distance was selected for purposes of illustration here. This analysis
 6 indicates approximately 2,400 children aged 5 or under to reside within 0.5 km of monitors
 7 exceeding the current standard. To also account for the population in areas with air
 8 concentrations just at (or very near) the current standard, we have also identified an additional
 9 nine Pb-TSP monitors with 3-month average concentrations within 10% of the current standard
 10 (Appendix 2D). Based on the 2010 U.S. census, 265 children aged 5 or under reside within 0.5
 11 km of these additional sites.

12 **Table 3-4. Number of children aged 5 and under in areas of elevated ambient Pb**
 13 **concentrations relative to the NAAQS.**

Population within 0.5 km of monitors with maximum 3-month Pb concentration greater than 0.15 µg/m ³ (2009-2011)				
	Number of Counties	Number of States or Territories	Total population	Children, 5 and under
All sites >0.15 µg/m ³	29	20	25,344	2,416
Subset of sites >0.5 µg/m ³	11	9	11,753	1,018
Data Sources: U.S. Bureau of the Census, 2010 Census of Population and Housing; recent Pb-TSP dataset presented in Figure 2-10 above (dataset criteria and summaries included Appendices 2C and 2D, respectively).				

14
 15 As the current air quality data set (analyzed in section 2.2.2 above) may not be inclusive
 16 of all of the newly sited monitors, as discussed in section 2.2.1 above, we recognize there may be
 17 other areas of the country where concentrations are above or just meet the current standard but
 18 for which such data are not yet available. To consider the potential for there to be additional, not
 19 yet identified, areas with elevated Pb concentrations, we have separately quantified the size of
 20 young child populations residing in areas near large Pb sources in Table 3-5. In so doing, we
 21 recognize uncertainties and potential limitations associated with these emissions estimates for
 22 these purposes, uncertainties both with regard to the accuracy of such estimates and also with
 23 regard to the role of specific source characteristics and meteorology, not explicitly considered
 24 here, in influencing ambient air Pb concentrations and contributing to substantial variation in air
 25 Pb concentrations at source locations (e.g., Figure 2-11 above). Accordingly, while the summary
 26 in Table 3-5 is informative to considering the potential prevalence of airborne Pb emissions and
 27 potential exposure of human populations, it is limited with regard to its ability to identify

1 populations living in areas of elevated ambient air Pb concentrations. We interpret this analysis
 2 to indicate that fewer than about 7,800 young children (aged 5 or younger) live in areas with air
 3 Pb concentrations near or above the current standard, with the current monitoring data indicating
 4 the size of this population to be approximately 2,700.

5 **Table 3-5. Population size near larger sources of lead emissions.**

Population Within 0.5 km of Sources Emitting at Least 0.5 tpy in 2008						
Facilities (other than airports)			Airports			All
Number of Locations	Population, all ages	Population, aged 5 and younger	Number of Locations	Population, all ages	Population, aged 5 and younger	Population, aged 5 and younger
<i>Facilities/Airports estimated to emit > 5.0 tpy</i>						
8	484	61	0	0	0	61
<i>Facilities/Airports estimated to emit 1.0 - 4.9 tpy^A</i>						
53	12,143	731	6	6,261	266	997
<i>Facilities/Airports estimated to emit 0.50 - 0.95 tpy^B</i>						
63	12,934	1,143	52	76,105	6,699	7,842
A - Facilities estimated to emit at least 1.0 tpy (after rounding to 1 decimal place) and less than 5.0 (after rounding to 1 decimal place). B - Facilities estimate to emit at least 0.50 tpy (after rounding to 2 decimal places) and less than 1.0 (after rounding to 1 decimal place). Sources: Population counts from U.S. Bureau of the Census, 2010 Census of Population and Housing. Emissions estimates for facilities other than airports drawn from 2008 NEI, version 3 (December 2012); estimates for airports reflect EPA's best estimates of piston-engine aircraft emissions. Piston-engine aircraft emissions inventory is available at: http://www.epa.gov/ttn/chief/net/2008neiv2/2008_neiv2_tsd_draft.pdf .						

6
7

1 **3.4 EXPOSURE AND RISK**

2 This risk information available for this review and described here is based primarily on
3 the exposure and risk assessment developed in the last review of the Pb NAAQS (henceforth
4 referred to as the 2007 REA [USEPA, 2007a]), in the context of the evidence newly available in
5 this review, as presented in the ISA. As described in the REA Planning Document, careful
6 consideration of the information newly available in this review, with regard to designing and
7 implementing a full REA for this review, led us to conclude that performance of a new REA for
8 the review was not warranted. We did not find the information newly available in this review to
9 provide the means by which to develop an updated or enhanced risk model that would
10 substantially improve the utility of risk estimates in informing the current Pb NAAQS review
11 (REA Planning Document, section 2.3). Based on their consideration of the REA Planning
12 Document analysis, the CASAC Pb Review Panel generally concurred with the conclusion that a
13 new REA was not warranted in this review (Frey, 2011).²⁶ Accordingly, the information
14 described here is drawn primarily from the 2007 REA, augmented by a limited new case study-
15 specific analysis focused on risk associated with the current standard, as described in section
16 3.4.3.3 below.

17 The focus for the risk assessment and associated estimates presented here is on Pb
18 derived from sources emitting Pb to ambient air. As discussed in section 1.3 above (and
19 conceptually illustrated in Figure 1-1), the multimedia and persistent nature of Pb, the role of
20 multiple exposure pathways, and the contributions of nonair sources of Pb to human exposure
21 media all present challenges and contribute significant additional complexity to the health risk
22 assessment that goes far beyond the situation for similar assessments typically performed for
23 other NAAQS pollutants (e.g., that focus only on the inhalation pathway). Limitations in the
24 available data and models affected our characterization of the various complexities associated
25 with exposure to ambient air Pb. As a result, the assessment includes a number of simplifying
26 assumptions in a number of areas and, as described in section 3.4.4 below, our estimates of air-
27 related Pb risk are approximate and are characterized by upper and lower bounds.

28 The conceptual model developed to inform planning for the 2007 REA, including
29 identification of key exposure media, target population, health endpoint and risk metric is
30 described in section 3.4.1. The 2007 REA relied on a case study approach to provide estimates
31 that inform our understanding of air-related exposure and risk in different types of air Pb
32 exposure situations; the case studies included are described in section 3.4.2. In section 3.4.3, the

²⁶ In our evaluation presented in the REA Planning Document and consultation with CASAC, we indicated our conclusion that the information newly available in this review did not provide the means by which to develop an updated or enhanced risk model that would substantially improve the utility of risk estimates in informing the current Pb NAAQS review.

1 analysis approach and general aspects of exposure and risk assessment methods are summarized,
2 and the air quality scenarios simulated are described. In section 3.4.3, we also summarize the
3 2007 REA risk model and the interpolation approach used in the limited new analyses performed
4 for purposes of this review. Section 3.4.4 identifies key aspects in exposure assessment and risk
5 estimates are presented in section 3.4.5. Treatment of key sources of variability in exposure and
6 risk estimates is described in section 3.4.6 and the characterization of uncertainty is summarized
7 in 3.4.7. An updated interpretation of the risk estimates for our purposes in this Pb NAAQS
8 review section is presented in section 3.4.8.

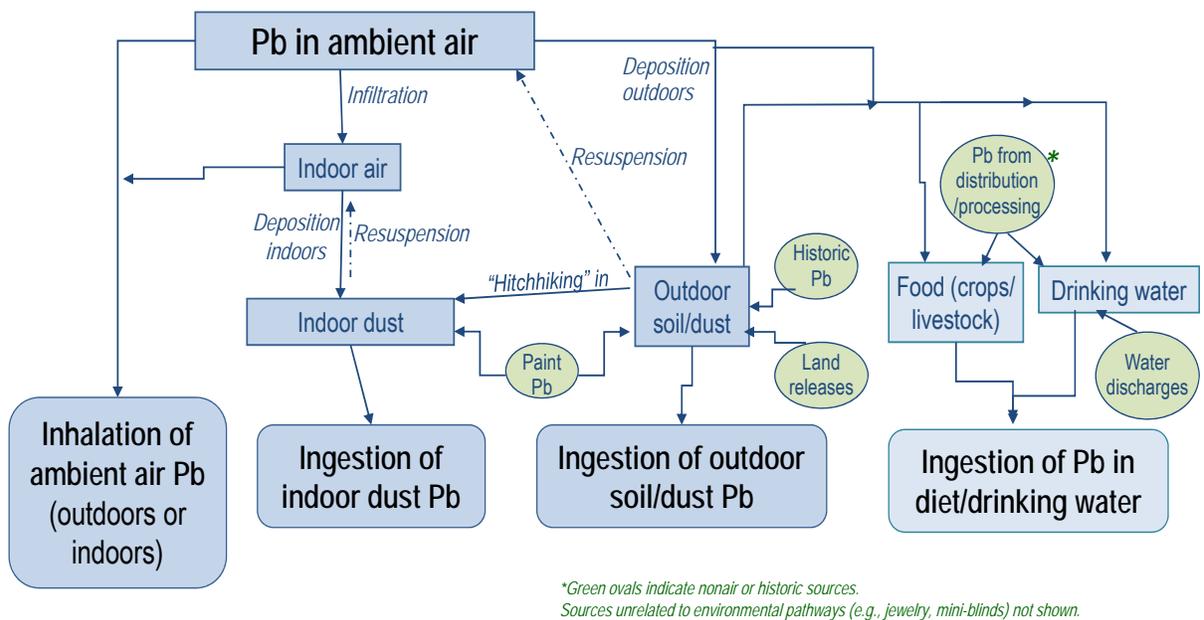
9 **3.4.1 Conceptual Model for Air-Related Lead Exposure and Risk**

10 The focus in consideration of public health risks associated with Pb from ambient air is
11 on Pb derived from those sources emitting Pb to ambient air. The multimedia and persistent
12 nature of Pb, illustrated in Figure 1-1 above, as well as the existence of many nonair sources of
13 Pb to the environment, contribute multiple complexities to the consideration of exposure and risk
14 for ambient air-related Pb. The conceptual model that informed planning for the 2007 REA
15 identified sources, pathways, routes, exposed populations, and health endpoints, focusing on
16 those aspects of Pb exposure most relevant to the review, while also recognizing the role of Pb
17 exposure pathways unrelated to Pb in ambient air (2007 REA, section 2.1).

18 As recognized in section 1.3 above, sources of human Pb exposure include current and
19 historical air emissions sources, as well as miscellaneous nonair sources, which can contribute to
20 multiple exposure media and associated pathways (e.g., inhalation of ambient air, ingestion of
21 indoor dust, outdoor soil/dust and diet or drinking water).²⁷ Figure 3-2 illustrates these human
22 exposure pathways from an analytical perspective, drawing on the conceptual model for the
23 assessment (2007 REA, Figure 2-1). As shown in Figure 3-2, in addition to airborne emissions
24 (recent or those in the past), sources of Pb to these pathways also include old leaded paint,
25 including Pb mobilized indoors during renovation/repair activities, and contaminated soils. Lead
26 in diet and drinking water may have air pathway related contributions as well as contributions
27 from nonair sources (e.g., Pb solder on water distribution pipes and Pb in materials used in food
28 processing). Limitations in our data and modeling tools handicapped our ability to separate the
29 nonair contributions to Pb exposure from estimates of air-related Pb exposure and risk.²⁸

²⁷ We did not explicitly consider Pb exposure related to consumer products (e.g., toys, cosmetics, dishes) in the 2007 REA.

²⁸ The assessment grouped the exposure and risk estimates for Pb in diet and drinking water together and combined them with the other pathways in estimates presented for “total Pb exposure”. Characterization of the risk assessment results in the rulemaking recognized the contribution, albeit unquantified, from air-related pathways within this category.



1

2 **Figure 3-2. Human exposure pathways for air-related lead.**

3 Identification of exposure populations, exposure/dose metric, health effects endpoint and
 4 risk metric to be included in the 2007 REA were based on consideration of the then-currently
 5 available evidence as assessed in detail in the 2006 CD. As discussed in the REA Planning
 6 Document (USEPA, 2011), these selections continue to be supported by the evidence now
 7 available in this review as described in the ISA.

8 In the REA, we focused on IQ loss in children exposed up to age 7 years. This focus
 9 reflected the evidence for young children with regard to air-related exposure pathways and
 10 susceptibility to Pb health impacts (e.g., ISA, sections 4.1.1, 5.3, 6.2.1, 6.3.1, and 6.4). For
 11 example, the hand-to-mouth activity of young children contributes to their Pb exposure (i.e.,
 12 incidental soil and indoor dust ingestion) and ambient air-related Pb has been shown to
 13 contribute to Pb in outdoor soil and indoor house dust. Accordingly, the Agency maintains a
 14 biokinetic blood Pb model for estimating blood Pb levels in young children (through age 7),
 15 which was used in this risk assessment.²⁹ In addition, our focus on young children reflects the
 16 evidence that the developing nervous system in children is among, if not, the most sensitive of
 17 the endpoints associated with Pb exposure (ISA, sections 2.6 and 2.10).

18 In terms of internal disposition and the biometrics used to assess Pb exposure, blood Pb
 19 (PbB) levels continue to be extensively used as an index or biomarker of exposure by national

²⁹ The pathways represented in this modeling include childhood inhalation and ingestion pathways, as well as maternal contributions to newborn body burden (2007 REA, Appendix H, Exhibit H-6).

1 and international health agencies. This reflects the association of PbB with exposure, particularly
2 recent exposure in young children, and the relative ease of collecting PbB measurement.
3 Although bone Pb measurements have become easier to collect and consequently, their use has
4 been more widespread, epidemiological and toxicological studies of Pb health effects and dose-
5 response relationships (particularly for neurodevelopmental effects in children) tend to be
6 dominated by PbB as the exposure metric. Therefore, we focused on modeling PbB in young
7 children, developing estimates for two PbB metrics: “concurrent” and “lifetime average”. For
8 the former we estimated PbB at age 7 years, while lifetime average was estimated as the average
9 of PbB levels across the 7 year period.

10 At the time of the last review, we noted that limitations precluded prediction of changes
11 in adult PbB levels (or bone Pb levels) given changes in ambient Pb levels. This reflects the fact
12 that the presence of substantial historic Pb stores in most adults introduces uncertainty into the
13 prediction of changes in blood or bone Pb in these adult populations resulting from changes in
14 ambient air Pb exposure. Additionally, in considering concentration-response relationships for
15 adult PbB and adult health outcomes, we recognized the uncertainty with regard to the role of
16 historic compared to recent exposures in eliciting the observed outcomes.

17 Based on conclusions regarding the scientific evidence available in the last review
18 (presented in the 2006 CD), the assessment focused on risk to the central nervous system in
19 childhood as the most sensitive effect that could be quantitatively assessed, with decrement in IQ
20 used as the risk metric.

21 **3.4.2 Case Studies**

22 Lead exposure and associated risk was estimated for multiple case studies that generally
23 represent two types of residential population exposures to air-related Pb (see Table 3-6): (1)
24 location-specific urban populations of children with a broad range of air-related exposures,
25 reflecting existence of urban concentration gradients; and, (2) children residing in localized areas
26 with air-related exposures representing air concentrations specifically reflecting the standard
27 level being evaluated. Thus, the two types of case studies differed with regard to the extent to
28 which they represented population variability in air-related Pb exposure (as discussed further in
29 section 3.4.7 below). Three location-specific urban case studies focused on residential areas
30 within Cleveland, Chicago, and Los Angeles, providing representations of urban populations
31 with a broad range of air-related exposures due to spatial gradients in both ambient air Pb levels
32 and population density. For example, the highest air concentrations in these case studies (i.e.,
33 those closest to the standard being assessed) were found in very small parts of the study areas,
34 while a large majority of the case study populations resided in areas with much lower air
35 concentrations. The case studies representing the children most highly exposed via air-related

1 pathways were the generalized (local) urban case study (also referred to as *general urban case*
 2 *study*) and the primary Pb smelter case study subarea. The generalized (local) urban case study
 3 was not based on a specific geographic location and reflected several simplifying assumptions in
 4 representing exposure including uniform ambient air Pb levels associated with the standard of
 5 interest across the hypothetical study area and a uniform study population. The primary Pb
 6 smelter case study provides risk estimates for children living in a specific area that, at the time of
 7 the 2007 REA, was not meeting the then current NAAQS. In addition to characterizing risks
 8 within a 10 km radius area surrounding the smelter (full area), we focused particularly on a
 9 subarea within 1.5 km of the facility, where airborne Pb concentrations were closest to the
 10 current standard and where children's air-related exposures are most impacted by emissions
 11 associated with the Pb smelter from which air Pb concentrations were estimated. Based on the
 12 nature of the population exposures represented by the two categories of case study, the
 13 generalized (local) urban and primary Pb smelter case study subarea include populations that are
 14 relatively more highly exposed by way of air pathways to air Pb concentrations near the standard
 15 level evaluated, compared with the populations in the three cities or the full area of the primary
 16 smelter case study.^{30,31}

17 **Table 3-6. Types of population exposures assessed.**

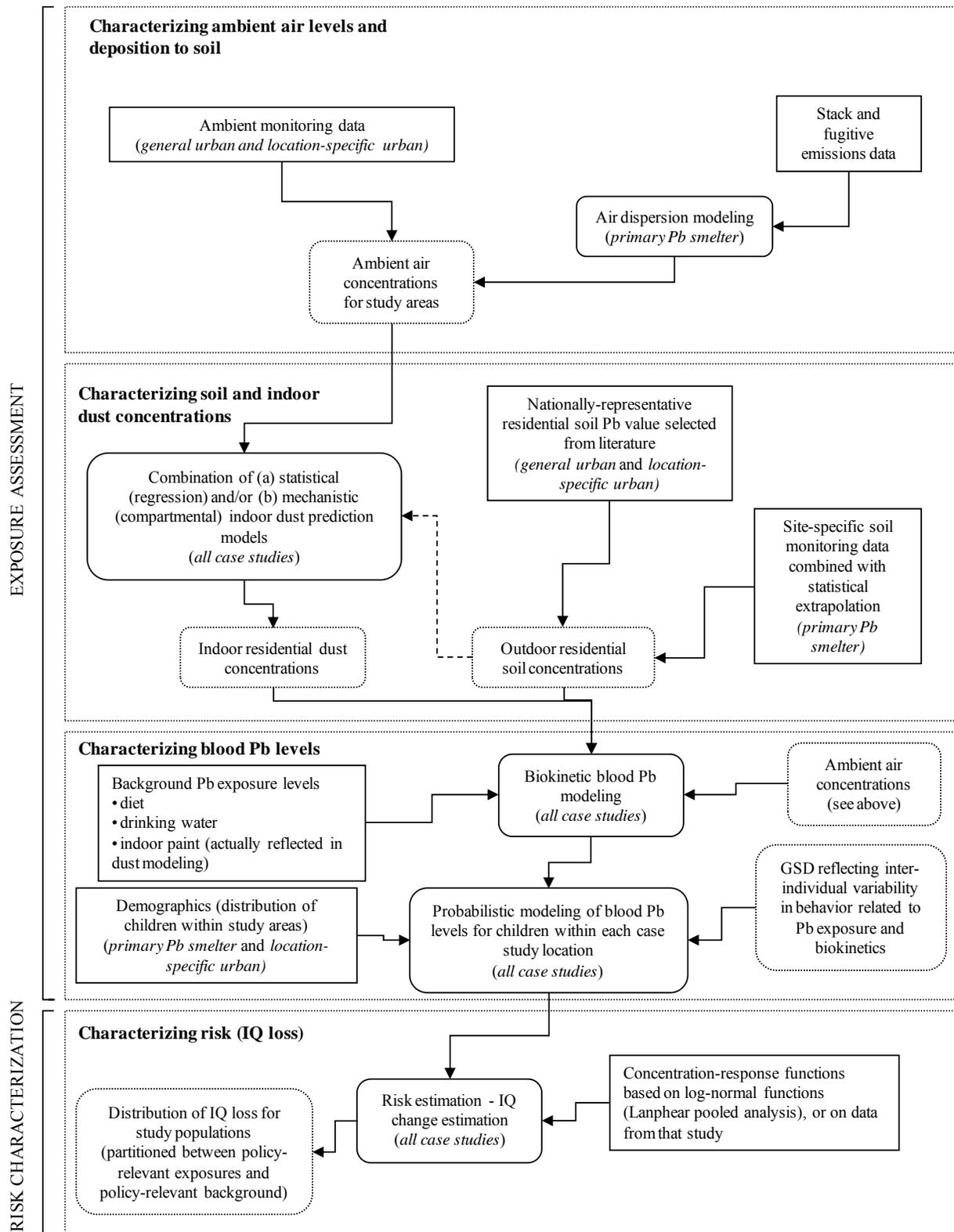
Type of Population Exposure			Case Study ¹
<i>Broad range of air-related exposures</i>	Part of metropolitan area with spatially varying air concentrations, inclusive of location at standard or conditions being evaluated	Multiple exposure zones, larger populations	Location-specific urban: Cleveland, Chicago, Los Angeles
	As above, with dominant, historically active metals industry as ambient air Pb source		Primary Pb smelter (full area)
<i>Generalized, high end of air-related exposure</i>	Localized residential area with air concentrations generally representing the standard or conditions evaluated	Single exposure zone without enumerated population	<i>Generalized (local) urban</i>
		A few exposure zones with small population	Primary Pb smelter (subarea)

³⁰ An additional case study (the secondary Pb smelter case study) was also developed in the 2007 REA, however, significant limitations associated with the use of dispersion modeling to predict ambient air, dust and soil Pb levels near the facility contributed to large uncertainties in the risk estimates (2007 REA, section 4.3.1).

³¹ In addition to the case studies included in the Risk Assessment Report, the pilot phase of the 2007 REA also included a near-roadway case study, focused on subset of urban population exposed immediately near roadways (ICF International, 2006). Based on the pilot results and advice from CASAC, however, this case study was not carried into the full-scale analysis. As an alternative, we developed the generalized (local) urban case study to represent urban residents in a localized area exposed to air-related Pb associated with the standard being assessed.

1 **3.4.3 Analysis Approach**

2 The approach to assessing exposure and risk for the two categories of case studies was
3 comprised of four main analytical steps: (a) estimation of ambient air Pb concentrations, (b)
4 estimation of Pb concentrations in other key exposure media, including outdoor soil and indoor
5 dust, (c) use of exposure media Pb concentrations, with other pathway Pb intake rates (e.g., diet),
6 to estimate PbB levels in children using biokinetic modeling, and (d) use of concentration-
7 response functions derived from epidemiology studies to estimate IQ loss associated with the
8 PbB levels. In implementing these steps for the primary smelter case study, air concentrations
9 were estimated using dispersion modeling; indoor dust concentrations were estimated for the
10 case study subarea using a site-specific regression model. The approach for the generalized
11 (local) urban case study and location-specific urban case studies is somewhat simpler, since it
12 does not involve fate and transport modeling for air concentration estimates and, instead, uses
13 ambient monitor levels to characterize the gradient in air Pb levels across the study area. All
14 steps are somewhat simpler in the generalized (local) urban case study which includes a single
15 exposure zone. Figure 3-3 identifies the key input data sets, modeling steps and intermediate
16 model output in each of the four analytical steps. The first three steps were employed in the
17 exposure assessment (discussed in section 3.4.3.1), while the fourth is the risk assessment step
18 (discussed in section 3.4.3.3).



1
2

Drawn from 2007 REA, Figure 3-3 (USEPA, 2007).

3 **Figure 3-3. Overview of analysis approach.**

3.4.3.1 Estimating Exposure

Concentrations of Pb were estimated in ambient media and indoor dust using a combination of empirical data and modeling projections. The use of empirical data brings with it uncertainty related to the potential inclusion of nonair source signals in these measurements (e.g., house paint contributions to indoor dust and outdoor soil Pb). Conversely, the use of modeling tools introduces other uncertainties (e.g., model and parameter uncertainties). The characterization of uncertainty associated with the risk assessment is discussed in section 3.4.7. Table 3-7 summarizes the exposure modeling approaches and data used to characterize Pb concentrations or input associated with each exposure pathway for each of the case studies.

Characterization of Pb in ambient air relied on (a) dispersion modeling of facility-related (including fugitive) Pb emissions for the primary Pb smelter case study, (b) the use of ambient monitor data for the location-specific urban case studies, and (c) an assumption of uniform ambient air Pb levels (matching the standard level being considered) for the generalized (local) urban case study. The use of dispersion modeling for the primary Pb smelter case study allowed us to capture more spatially refined patterns of ambient air Pb over residential areas in the vicinity of the facility, where ambient Pb levels were dominated by Pb released from the facility. For the location-specific urban case studies, we used Pb monitors within each of the urban study areas to characterize spatial gradients. By contrast, the generalized (local) urban case study is designed to assess exposure and risk for a smaller group of residents (e.g., neighborhood) exposed at the level of the standard and therefore, did not rely on monitor data in characterizing levels, since ambient air Pb was fixed at the standard being assessed. While ambient air Pb concentrations in the primary Pb smelter case study reflected only contributions from direct and fugitive emissions associated with the facility, concentrations in the location-specific urban study areas, which relied on empirical (monitor-based) data to define ambient air Pb concentrations, reflected contributions from all contributing sources, be they currently active stationary or mobile sources, resuspension of previously deposited Pb or other. Additional detail on estimation of ambient (outdoor) and indoor air concentrations is presented in section 5.2.2 and Appendices A through D of the 2007 REA.

Characterization of Pb concentrations in outdoor surface soil/dust, resulting from deposition of airborne Pb was based on the use of (a) existing site-specific measurements (primary Pb smelter case study), and (b) nationally representative residential soil measurements obtained from the literature (general and location-specific urban case studies). In the case of the primary Pb smelter case study, soil Pb concentration data were available for a zone close to the facility and statistical extrapolation from these data was used to predict soil levels for portions of the study area beyond this zone. Additional detail on estimation of Pb concentrations in outdoor surface soil or dust is presented in sections 3.1.3 and 5.2.2.2 and Appendix F of 2007 REA.

1 To predict concentrations of ambient Pb in indoor dust, we relied on a combination of (a)
 2 regression-based models that relate indoor dust to outdoor air Pb and/or outdoor soil Pb and (b)
 3 mechanistic models that predict indoor dust Pb based on key mechanisms (e.g., infiltration of
 4 outdoor air indoors, deposition rates of Pb from indoor air to indoor surfaces, house cleaning
 5 rates). For the point source case study, a combination of regression-based models obtained from
 6 the literature and developed based on site-specific data were used, and a customized hybrid
 7 empirical-mechanistic model was developed for the general and location-specific urban case
 8 studies. This reflected the fact that available regression-based models had been developed
 9 largely based on residential exposures near large point sources and were not considered
 10 representative of more general urban exposures. Consequently, a mechanistic model, augmented
 11 with empirical data, was developed for the generalized (local) urban case study. Additional
 12 detail on estimation of Pb concentrations in indoor dust is presented in sections 3.1.4 and 5.2.2.2
 13 and Appendix G of 2007 REA.

14 **Table 3-7. Summary of approaches used to estimate case study media concentrations.**

Simulation of air quality impacts	Media category	Generalized (local) urban case study	Location-specific urban case study	Primary Pb smelter case study (1.5 km subarea and 10 km full area)
Concentrations for these media were varied across air quality scenarios (see section 3.4.3.2).	<i>Ambient air Pb levels</i>	Single ambient air Pb level assumed across entire study area (single exposure zone)	Source and non-source monitors describe concentration gradient (6 to 11 exposure zones per case study)	Dispersion modeling of smelter-related emissions (22 census block groups and 115 blocks)
	<i>Indoor dust Pb levels</i>	Hybrid model: dynamic aspect relates ambient air Pb concentrations to indoor dust Pb; empirical aspect represents Pb from other sources (e.g., paint, historical air, Pb carried indoors with people)		For subarea - regression of site-specific air, dust data. For full area - regression of air, dust, soil data from other, historical locations.
Concentrations were constant across air quality scenarios (data/modeling limitations)	<i>Outdoor soil Pb levels</i>	National dataset (HUD, for houses constructed between 1940 and 1998).		Site-specific data (for subarea)
	<i>Dietary Pb intake</i>	National datasets for Pb residue data (US FDA Total Diet Study) and food consumption data (NHANES)		
	<i>Drinking water Pb intake</i>	US and Canada datasets for residential water Pb concentrations and ingestion rates		

15
 16 Blood Pb levels were predicted from estimates of Pb contained in various media (e.g.,
 17 ambient air, diet, water, indoor dust), and estimates of Pb intake from dietary and drinking water
 18 pathways, using the Integrated Exposure and Uptake Biokinetic (IEUBK) model (2007 REA,

1 sections 3.2.1.1 and 5.2.4).³² Diet and drinking water intake and concentrations, as well as other
2 model inputs, were based on the most current information (2007 REA, Appendix H). Detail on
3 methods used to characterize media Pb concentrations and all IEUBK inputs for each case study
4 are in the 2007 REA, sections 3.1, 3.2, 5.2.3 and 5.2.4 and Appendices C through H. As the
5 shortest temporal scale accepted for inputs to the IEUBK is a year, all model inputs, developed
6 for each exposure zone in each case study, were annual average values. For media concentration
7 inputs, the same values were used for each year of the seven-year simulation. Other model
8 inputs varied as appropriate with the age of the simulated child (2007 REA, Appendix H).

9 To simulate population variability in Pb intake and uptake, we used the IEUBK model to
10 first generate a central-tendency estimate of the PbB levels for the group of children within a
11 given exposure zone of a study area.³³ Outside the IEUBK model, we then combined this central-
12 tendency estimate with a geometric standard deviation (GSD) reflecting variability in PbB levels
13 for groups of children to generate a distribution of PbB levels for a study area. The procedure
14 for combining the IEUBK-based central tendency blood Pb estimate with a GSD to generate a
15 population distribution of PbB levels differs somewhat for the categories of case studies. The
16 approach for the general urban case study is fairly simple in that we have a single IEUBK-based
17 estimate of PbB levels and this is in turn, combined with the GSD selected for this study area to
18 produce a population-distribution of PbB levels. For both the primary Pb smelter and the
19 location-specific urban study areas, multiple polygons within the larger study area (e.g., US
20 Census blocks for the location specific urban study areas) are used as the basis for generating
21 distributions of PbB levels for the child population in each study area. These distributions are
22 generated using a MonteCarlo-based population-weighted sampling method with U.S. Census
23 child counts for each polygon and an adjustment factor distribution based on the chosen GSD
24 (see 2007 REA, sections 3.2.2 and 5.2.2.3).

25 The GSD reflects a number of factors which operate together to produce interindividual
26 variability in blood Pb levels, including: (a) biokinetic variability (differences in the uptake,
27 distribution or clearance of Pb), (b) differences in behavior related to Pb exposure (e.g., varying
28 hand-to-mouth activity, tap water ingestion rates, and time spent playing indoors) and (c)

³² In predicting PbB levels, we assumed that Pb concentrations in exposure media remained constant throughout the 7 year simulation period.

³³ In typical IEUBK applications, the GSD is applied within the IEUBK model as part of the modeling process in order to generate percentiles of PbB distribution for the population simulated. However, for the NAAQS REA, we used IEUBK only for generating the central-tendency PbB value for a given exposure zone and then probabilistically combined that estimate with the GSD outside of the IEUBK model. This allowed us, in the case of the primary Pb smelter and the location-specific urban case studies (as noted below) to generate individual population-level PbB distributions for each exposure zone which could then be population-weighted and combined using Monte Carlo sampling to generate a single population-distribution for each study area. This was not possible with the typical application of the GSD within the IEUBK model.

1 differences in environmental Pb exposure concentrations (e.g., spatial gradients in ambient Pb
2 levels of a resolution beyond that simulated in each case study, differences in
3 cleaning/vacuumping rates and air exchanges rates).³⁴ Note, that for all of the study areas, we
4 assumed that pathway apportionment of PbB levels based on the modeling of the central-
5 tendency PbB level (using IEUBK) holds for all percentiles of PbB levels derived by combining
6 that central-tendency with the GSD. Blood Pb modeling completed for all case studies included
7 estimates of both concurrent and lifetime-average PbB metrics, although ultimately, we focused
8 on the concurrent PbB metric in estimating risk.³⁵

9 **3.4.3.2 Air Quality Scenarios Included in 2007 Assessment**

10 The air quality scenarios assessed in the 2007 REA for the case studies identified in
11 Table 3-6 above included conditions just meeting the NAAQS that was current at the time of the
12 last review (1.5 µg/m³, maximum calendar quarter average) and conditions meeting several
13 alternative, lower standards. Additionally, for the three location-specific urban case studies,
14 scenarios were included for then-current conditions (2003-2005) in each location.³⁶ These air
15 quality scenarios are characterized by quarterly or monthly averaging times and a not-to-be-
16 exceeded form. Once the air quality dataset representing each scenario was developed, the
17 associated annual average concentrations for each exposure zone were derived for input to the
18 IEUBK model, which does not accept air quality inputs of a temporal scale shorter than a year
19 (2007 REA, Appendix H).³⁷

20 As a result of the differing then-current conditions of the location-specific case studies,
21 the specific alternative standards assessed for each differed (see Table 3-8). To simulate the

³⁴ We specified GSDs for each of the case studies that reflected differences in the study areas and underlying study populations, as well as the availability of blood Pb measurement data (see 2007 REA, sections 3.2.3 and 5.2.2.3).

³⁵ As discussed in section 2.1.5 of the 2007 REA, the concurrent PbB measurement (i.e., PbB measurements at the time of IQ test) and the lifetime-average blood level (i.e., average of measurements taken over child's first 6-7 years) were considered "stronger predictors of lead-associated intellectual deficits than was the maximal measured (peak) or early childhood blood lead concentrations" with the concurrent PbB level exhibiting the strongest relationship (2006 CD, p. 6-29).

³⁶ For the location-specific urban case studies of Cleveland, Chicago and Los Angeles, the maximum monthly average concentration was 0.56, 0.31 and 0.17 µg/m³, respectively, and the maximum calendar quarter average concentration was 0.36, 0.14 and 0.09 µg/m³, respectively, (2003-2005 data; 2007 REA, Appendix O).

³⁷ Although many different patterns of temporally varying air concentration will just meet a given potential alternative standard, the shortest time step accommodated by the blood Pb model is a year. Thus, the air Pb concentration inputs to the blood Pb model for each air quality scenario are annual average air Pb concentrations. For the generalized (local) urban case study, the national Pb-TSP monitoring dataset was analyzed to characterize the distribution of site-specific relationships between metrics reflecting the averaging time and form for the air quality scenarios being assessed (Table 3-5) and the annual average. The IEUBK annual average input was then derived by multiplying the level for a given air quality scenario by the ratio for the averaging time and form for that air quality scenario. For the location-specific case studies, the full temporally varying air Pb concentration dataset for each exposure zone was used to derive the average annual concentration for the IEUBK input.

1 previous NAAQS scenario at the primary Pb smelter location, at which then-current monitoring
2 data indicated exceedance of that standard, a proportional roll-down was performed across the
3 area to achieve conditions that just met that standard. Additionally, although it was considered
4 an extremely unlikely scenario that air concentrations in urban areas across the U.S. that were
5 well below the previous NAAQS would increase to just meet that standard (e.g., by way of
6 expansion of existing sources or congregation of multiple sources in adjacent locations), we
7 simulated this scenario in all case studies. In so doing, the air Pb concentrations were rolled up
8 proportionally across the location-specific urban study areas to conditions just meeting the
9 standard. No other scenario simulations involved rolling concentrations up. For the primary Pb
10 smelter case study, air Pb concentrations were proportionally rolled down to conditions just
11 meeting each of the potential alternative standards assessed. In the three location-specific urban
12 case studies, the temporally and spatially varying concentrations were rolled down to conditions
13 just meeting each of the potential alternative standards that they exceeded (2007 REA, section
14 5.2.2.1).³⁸

15 For the generalized (local) urban case study, which has a single exposure zone in which
16 air Pb concentrations do not vary spatially, we derived a single air Pb concentration estimate to
17 meet the standard assessed (e.g., specified maximum monthly or quarterly average). To reflect
18 the variability in air Pb concentrations that occur over time scales less than a year as a result of
19 temporal changes in meteorology and source and emission characteristics, the annual average air
20 concentration (input for IEUBK and dust model) was derived for the maximum monthly and
21 quarterly average metrics assessed using relationships based on the available Pb-TSP monitoring
22 data for large U.S. urban areas (2007 REA, Appendix A).

23

³⁸ When concentrations in the exposure zone (within the study area) that has the highest Pb concentrations (in terms of the metric being assessed) achieve a maximum quarterly or monthly average of the specified level, the potential standard is “just met”.

1 **Table 3-8. Air quality scenarios assessed.**

Air Quality Conditions Just Meeting ...		Case Studies Where Simulated				
		Generalized (local) Urban ^A	Location Specific			
Maximum Quarterly Average ($\mu\text{g}/\text{m}^3$)	Maximum Monthly Average ($\mu\text{g}/\text{m}^3$)		Primary Smelter	Cleveland	Chicago	Los Angeles
1.5 ^B		√	√	√	√	√
0.2 ^C		√	√			
	0.5	√	√	√		
	0.2	√	√	√	√	
	0.05	√	√	√	√	√
	0.02	√	√	√	√	√

A - Conditions were set to meet the standards assessed in the single exposure zone of this case study.
 B - Concentrations were proportionally rolled down to conditions just meeting the previous standard in the primary Pb smelter case study; concentrations in the three urban location-specific case studies were proportionally rolled up to just meet this standard.
 C - Concentrations were proportionally rolled down to just meet this and the other potential alternative standards.

2
 3 The approaches for estimating Pb concentrations in other media varied depending on the
 4 type of case study (see section 3.4.3.1 above). Limitations in the available data and modeling
 5 tools precluded simulation of linkages between some media and air Pb, such that the full impact
 6 of changes in air Pb conditions associated with attainment of lower standards was not simulated.
 7 For example, dietary and drinking water Pb concentrations, as well as soil Pb concentrations,
 8 were not varied across the air quality scenarios in any case study (see Table 3-7). For all case
 9 studies, however, indoor dust Pb concentrations were simulated to change with the different air
 10 quality scenarios that also provided differing ambient air Pb concentrations (outdoors and
 11 indoors).

12 **3.4.3.3 Methods for Deriving Risk Estimates**

13 In this section, we first summarize the full risk model employed in the 2007 REA for
 14 estimating risk for a broad range of air quality scenarios (section 3.4.3.3.1). Then, in section
 15 3.4.3.3.2, we summarize approaches by which we have identified risk estimates pertaining to the
 16 current standard, the second of which involves an analysis newly completed in this review in
 17 which risk estimates are interpolated for the current standard based on the 2007 REA risk
 18 estimates.

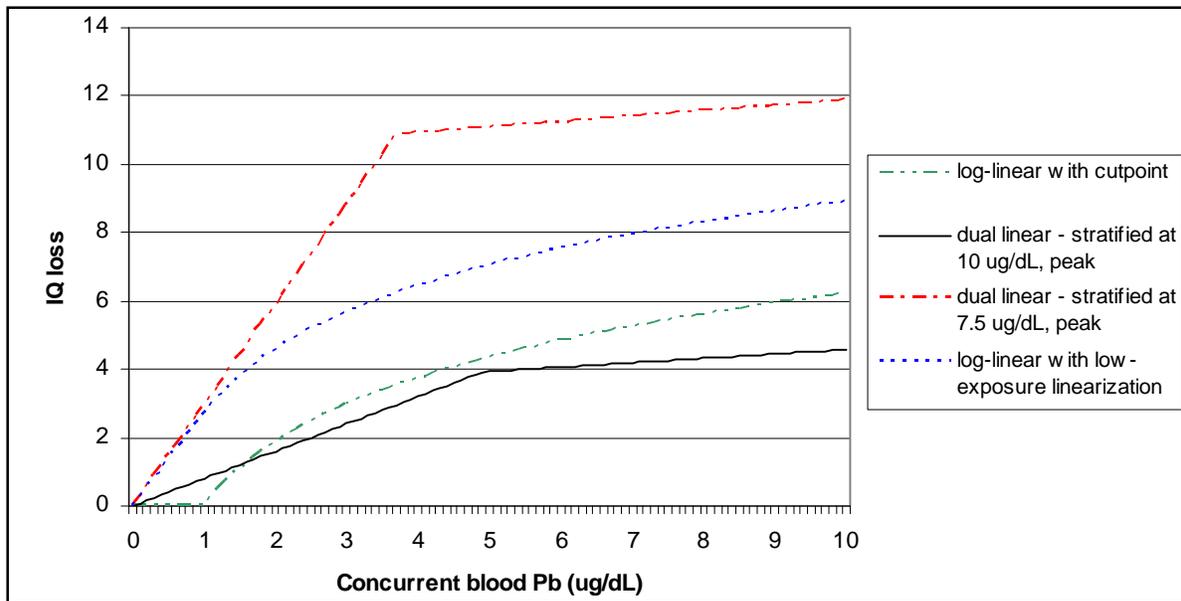
19

3.4.3.3.1 Full Risk Model in 2007 REA

The risk characterization step employed in the 2007 REA involved generating a distribution of IQ loss estimates for the set of children simulated in the exposure assessment. Specifically, estimated PbB levels (for the concurrent PbB metric)³⁹ were combined with four PbB concentration-response (C-R) functions for IQ loss based on the analysis by Lanphear et al (2005) of a pooled international dataset of blood Pb and IQ (see 2007 REA, section 5.3.1.1). Four different C-R functions were selected to provide different characterizations of behavior at low exposures. The decision to use four different functions is in recognition of uncertainty related to modeling this endpoint, particularly at lower PbB levels for which there is limited representation in the Lanphear et al (2005) pooled dataset; the 5th percentile for the concurrent PbB measurements in that dataset is 2.5 µg/dL, and the mean is 9.7 µg/dL (73 FR 66978). The four different functions are either based directly on the lognormal model described in Lanphear et al (2005), or they are derived from data presented in that study. The four functions are presented in Figure 3-4 and compared in Table 3-9 with regard to total IQ loss and incremental IQ loss (IQ loss per µg/dL blood Pb) across a range of concurrent PbB levels. A brief description of each of the functions is also here:

- Log-linear with cutpoint: log-linear function derived from the pooled analysis applied down to 1 µg/dL (concurrent PbB metric) with no IQ loss projected below that exposure level.
- Log-linear with low-exposure linearization: log-linear function applied down to 1 µg/dL (concurrent PbB metric) with linearization of the slope at that point which is used to project IQ loss down to the origin.
- Dual linear – stratified at 10 µg/dL: function developed by fitting a two-piece linear function stratified at 10 µg/dL (peak PbB metric) to the log-linear function developed from the pooled analysis.
- Dual linear-stratified at 7.5 µg/dL: as above, but based on stratification of the two-piece function at 7.5 µg/dL (peak PbB metric).

³⁹ Risk estimates were also developed for lifetime average PbB levels using concentration-response functions derived from the Lanphear et al (2005) analysis for lifetime average PbB levels. Estimates based on the concurrent PbB metric are given primary emphasis due to the slightly more significant association found for concurrent PbB with IQ by Lanphear et al (2005) in addition to advice from CASAC.



1
2 **Figure 3-4. Comparison of four concentration-response functions used in risk assessment.**

3
4 **Table 3-9. Comparison of total and incremental IQ loss estimates for blood Pb below 10**
5 **µg/dL based on the four concentration-response functions.**

Performance Metric		Concentration-Response Function			
		Log-linear with cutpoint	Log-linear with low-exposure linearization	Dual linear - stratified at 10 µg/dL peak	Dual linear - stratified at 7.5 µg/dL peak
		Points, IQ loss			
Total IQ loss	at 2 µg/dL	1.9	4.6	1.6	5.9
	at 5 µg/dL	4.3	7.0	3.9	11.1
	at 7.5 µg/dL	5.4	8.1	4.3	11.5
	at 10 µg/dL	6.2	8.9	4.6	11.9
Incremental IQ loss (average # points per µg/dL)	<2 µg/dL	0.94	2.29	0.80	2.94
	<5 µg/dL	0.87	1.41	0.80	2.24
	<7.5 µg/dL	0.73	1.09	0.58	1.55
	<10 µg/dL	0.62	0.89	0.47	1.20

6
7 Of the four C-R functions provided above, we have the greatest overall confidence in risk
8 estimates generated using the log-linear with low-exposure linearization (LLL) function because
9 this function is (a) nonlinear, describing greater response per unit blood Pb at lower blood Pb
10 levels consistent with multiple studies, (b) is based on fitting a function to the entire pooled
11 dataset (and hence uses all of the data in describing response across the range of exposures), (c)
12 is supported by sensitivity analyses showing the model coefficients to be robust (Lanphear et al.,

1 2005), and (d) provides an approach for predicting IQ loss at the lowest exposures simulated in
2 the assessment (which for some simulated children yield blood Pb levels below those studied).
3 Risk estimates generated using the other three C-R functions are also presented to provide
4 perspective on the impact of uncertainty in this key modeling step. For additional detail on the
5 rationale for placing greater emphasis on the LLL function, see section 4.2.1 of the 2007 Pb Staff
6 Paper (USEPA, 2007b).

7 Two categories of risk metrics were generated for each of the location-specific case
8 studies:

- 9 • *Population risk percentiles*: The IQ loss associated with policy-relevant exposure
10 pathways for specific percentiles of the child population (e.g., the 50th, 90th and 95th
11 percentile modeled child). This category of metric provides perspective on the
12 distribution of IQ loss resulting from policy-relevant exposure pathways, ranging
13 from the typical or average child (50th percentile, mean) to children experiencing
14 higher exposures (90th, 95th percentiles). Greater emphasis has been placed on the
15 median IQ loss estimates due to increased confidence in these estimates relative to the
16 higher percentile estimates, as noted in section 3.4.7.
- 17 • *Child frequency counts associated with specific risk percentiles*: Number of children
18 associated with each of the population percentiles (e.g., the number of children
19 predicted to have risk levels at or above the 95th percentile). This risk metric provides
20 a perspective on the number of children associated with various levels of IQ loss for a
21 particular case study.

22 For the generalized (local) urban case study, only the first type of risk metric, population
23 risk percentiles, was developed because a specific location with associated demographic data
24 was not modeled. In summarizing risk estimates from the 2007 REA in this document, we have
25 focused on the first category of risk metric, and specifically, on the median IQ loss estimates.

26 **3.4.3.3.2 Air Quality Scenarios Reflecting the Current Standard**

27 As the 2007 REA did not include an air quality scenario simulated to just meet the
28 standard selected by the 2008 decision,⁴⁰ we have considered two approaches for identifying risk
29 estimates pertaining to conditions just meeting the now-current Pb standard for our purposes in
30 this review. We first reviewed all the scenarios analyzed in the 2007 REA and recognize the
31 similarity to the current standard of the then-current conditions scenario for the Chicago case
32 study. Accordingly, we consider the risk estimates for that scenario for our purposes in this
33 review of considering risk associated with the current standard (see section 3.4.5 below).
34 Additionally, in recognition of the variation among specific locations and urban areas with

⁴⁰ The 2008 decision on the level for the revised NAAQS was based primarily on consideration of the evidence-based air-related IQ loss framework. Although the specific level, averaging time and form chosen for the new standard were not among the air quality scenarios that had been simulated in that review, the risk estimates available for the range of simulated scenarios were concluded to be roughly consistent with and generally supportive of the evidence-based air-related IQ loss estimates (73 FR 67006; see section 4.1.1 below).

1 regard to air quality patterns and exposed population, we have also newly developed estimates
2 for an air quality scenario just meeting the current Pb NAAQS in the context of the generalized
3 (local) urban case study to augment the risk information available in this current review. The
4 newly developed estimates were derived based on interpolation from the risk estimates available
5 for scenarios previously assessed for the generalized (local) urban case study. Such interpolated
6 estimates were only developed for the generalized urban case study due to its use of a single
7 exposure zone which greatly simplified the method employed, thus contributing relatively lesser
8 uncertainty from the interpolation step.⁴¹

9 In newly developing estimates for the current standard in the generalized (local) urban
10 case study, the general approach we followed was to identify the two alternative standard
11 scenarios simulated in the 2007 REA which represented air quality conditions bracketing those
12 for the current standard and then linearly interpolate an estimate of risk for the current standard
13 based on the slope created from the two bracketing estimates. In representing air quality
14 conditions for these purposes, we focused on the annual average air Pb concentration estimates
15 used as IEUBK model inputs for the various air quality scenarios. An annual average
16 concentration estimate to represent the current standard was identified in a manner consistent
17 with that employed in the 2007 REA for this case study (see section 3.4.3.2 above) with the use
18 of currently available monitoring data for relationships between air quality metrics for
19 representation of the current standard. By this method, the air quality scenario for the current
20 standard ($0.15 \mu\text{g}/\text{m}^3$, as a not-to-be-exceeded 3-month average) was found to be bracketed by
21 the scenarios for alternative standards of $0.20 \mu\text{g}/\text{m}^3$ (maximum calendar quarter average) and
22 $0.20 \mu\text{g}/\text{m}^3$ (maximum monthly average). A risk estimate for the current standard was then
23 derived using the slope relating general urban case study IQ loss to annual average Pb
24 concentration used for those two air quality scenarios. We used this interpolation approach to
25 develop median risk estimates for the current standard based on each of the four C-R functions.
26 Details on the method for the interpolation approach are provided in Appendix 3A. The
27 interpolated median estimates of risk for the current standard for the generalized (local) urban
28 case study are provided below in Table 3-9.

⁴¹ We did not complete interpolation of risk estimates for the current standard for the other case studies (i.e., the primary Pb smelter and location-specific urban case studies) because those case studies utilized a more complex, spatially-differentiated and population-based approach for which precludes application of the simple linear interpolation approach described, without introduction of substantial added uncertainty. The simplicity of the generalized (local) urban study area, however, with its single exposure zone, is amenable to the linear interpolation of risk described here.

3.4.4 Challenges in Characterizing Air-related Exposure and Risk

In estimating the portion of total (all-pathway) PbB and IQ loss attributable to air-related pathways, we faced a variety of challenges. Although we parsed total estimates into those for diet/drinking water and two air-related categories, referred to as “recent air” and “past air”, significant limitations in our modeling tools and data resulted in an inability to parse specific risk estimates into specific pathways. Although Pb in diet and drinking water sources may include Pb derived from Pb in the ambient air (as well as Pb from nonair sources), limitations precluded explicit modeling of the contribution from air pathways to these exposure pathways, such that the air-related component of these exposures was not estimated.⁴² As a result we utilized the estimates from recent and past air categories to create bounds within which we consider air-related risk to fall, as illustrated in Figure 3-5 and described further below.

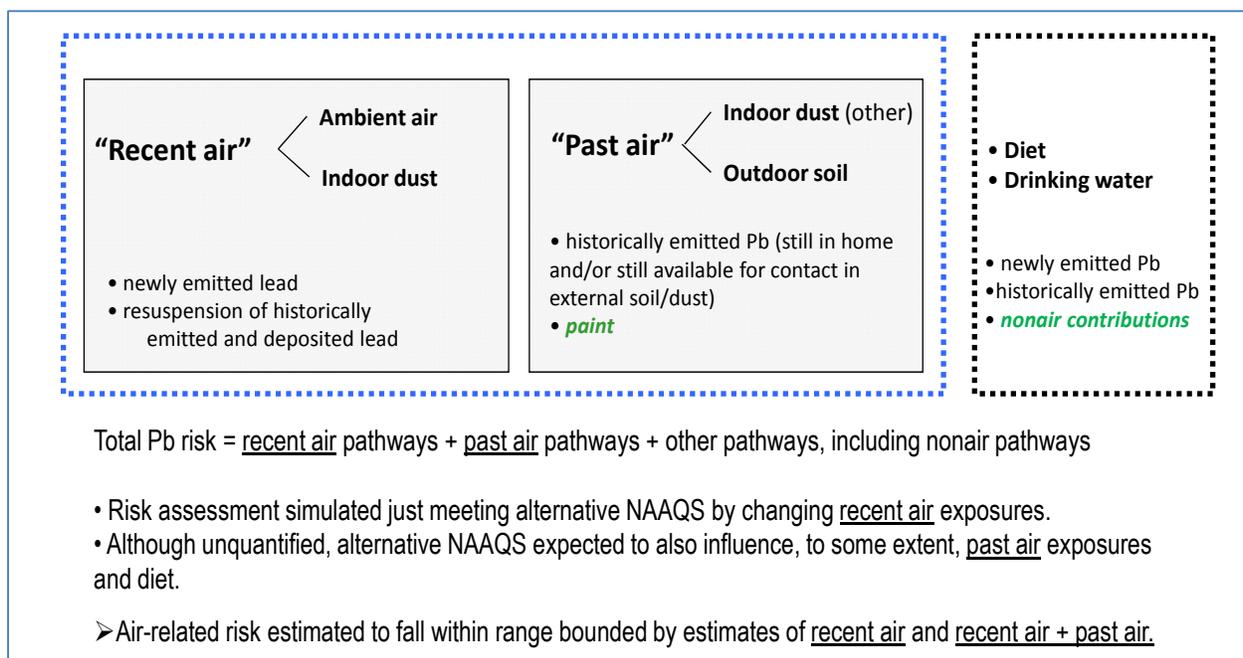


Figure 3-5. Parsing of air-related risk estimates.

Those Pb exposure pathways tied most directly to ambient air, which consequently have the potential to respond relatively more quickly to changes in air Pb (i.e., inhalation and ingestion of indoor dust Pb derived from the infiltration of ambient air Pb indoors), were placed into the “recent air” category. The other air-related Pb exposure pathways, all of which are associated with atmospheric deposition, were placed into the “past air” category. These include

⁴² Further, although paint is not an air-related source of Pb exposure, for this analysis, may be reflected somewhat in estimates developed for the “past air” category, due to modeling constraints. For example, technical limitations of the indoor dust Pb modeling may contribute to paint-related Pb in the “past air” component of indoor dust Pb and limitations in the available data and modeling may contribute to paint-related Pb in estimates of soil Pb.

1 ingestion of Pb in outdoor dust/soil and ingestion of the portion of Pb in indoor dust that after
2 deposition from ambient air outdoors is carried indoors with humans. Additionally, while we
3 recognized the potential for these other air-related exposures to be affected (over some time
4 frame) by an adjustment to the Pb NAAQS, limitations in our data and tools precluded our
5 simulation of that relationship with air Pb levels.

6 Among the limitations affecting our estimates for the air-related categories is the
7 apportionment of nonair pathways. For example, while conceptually indoor Pb paint
8 contributions to indoor dust Pb would be considered background and included in a “background”
9 category for this assessment, due to technical limitations related to indoor dust Pb modeling, dust
10 from Pb paint was included as part of "other" indoor dust Pb (i.e., as part of past air exposure).
11 The inclusion of indoor paint Pb as a component of "other" indoor dust Pb (and consequently as
12 a component of the “past air” category) represents a source of potential high bias in our
13 prediction of exposure and risk associated with the “past air” category because, indoor paint Pb
14 is a nonair Pb source. At the same time, Pb in ambient air does contribute to the drinking water
15 and diet exposure pathways, and is likely a substantial contribution to diet. We could not
16 separate the air contribution from the nonair contributions in the drinking water and diet
17 pathways. As a result, our risk estimate for the drinking water/diet category of pathways
18 includes some air-related risk representing a source of potential low bias in our predictions of
19 air-related risk.

20 Further, we note that in simulating reductions in exposure associated with reducing
21 ambient air Pb levels through alternative NAAQS (and increases in exposure if the current
22 NAAQS was reached in certain case studies) only the exposure pathways categorized as “recent
23 air” (inhalation and ingestion of that portion of indoor dust associated with outdoor ambient air)
24 were varied with changes in air concentration. The assessment from the previous review did not
25 simulate decreases in “past air” exposure pathways (e.g., reductions in outdoor soil Pb levels
26 following reduction in ambient air Pb levels and a subsequent decrease in exposure through
27 incidental soil ingestion and the contribution of outdoor soil to indoor dust). These exposures
28 were held constant across all air quality scenarios. This modeling/data limitation, accordingly,
29 precluded estimates for this category from reflecting any impact of alternative standard levels
30 simulated.

31 In summary, because of limitations in the assessment design, data and modeling tools,
32 our risk estimates for the “past air” category in the last review include both risks that are truly
33 air-related and potentially, some nonair risk. Because we could not sharply separate Pb linked to
34 ambient air from Pb from other (nonair) sources, some of the three categories of risk are
35 underestimated and others overestimated. On balance, we believe this limitation leads to a slight
36 overestimate of the risks in the “past air” category. At the same time, as discussed above, the

1 "recent air" category does not fully represent the risk associated with all air-related pathways.
2 Thus, we considered the risk attributable to air-related exposure pathways to be bounded on the
3 low end by the risk estimated for the "recent air" category and on the upper end by the risk
4 estimated for the "recent air" plus "past air" categories. With regard to the latter, we are
5 additionally cognizant of the modeling and data limitations which reduce the extent to which the
6 upper end of these bounds reflects impacts of alternative air quality conditions simulated.

7 **3.4.5 Risk Estimates**

8 This section summarizes air-related risk estimates generated for the previous review and
9 also risk estimates that have been newly derived in this review by interpolation from the previous
10 review estimates for the current standard (see section 3.4.3.3.2 above). Included in this summary
11 is consideration of the following question:

- 12 • **What is the nature and magnitude of air-related risks remaining upon just**
13 **meeting the current Pb standard?**

14 The air-related risk quantified in the 2007 REA is for IQ loss associated with Pb
15 exposure. As discussed in section 3.2 above, the evidence in the last review, as well as in the
16 current review, supports identification of neurocognitive effects in young children as a
17 particularly sensitive endpoint for the exposure circumstances relevant to this review and which
18 addresses the important at-risk population, young children. With this support in the evidence for
19 quantification, the risk assessment quantified decrements in IQ, an established indicator of
20 neurocognitive function. With regard to the nature of the risks, in addition to recognizing the
21 role of IQ as an indicator of an array of neurocognitive function impacts, we additionally take
22 note of the evidence regarding implications of neurocognitive impacts in young children with
23 regard to potential future impacts as the children age, as recognized in section 3.2 above.

24 In presenting risk estimates here, we focus on the median estimates of air-related IQ loss
25 for each case study. Estimates of air-related risk are substantially more uncertain for extremes of
26 the risk distribution, such as the 95th percentile. Those estimates and estimates for other risk
27 metrics, including population incidence for IQ loss at those case studies with population
28 enumeration, are available elsewhere (2007 REA, sections 4.2 and 5.3.2). In this section, Table
29 3-10 presents air-related IQ loss estimates derived in the 2007 REA for the full set of case
30 studies. Table 3-11 provides a subset of these risk estimates for the generalized (local) urban case
31 study in addition to estimates for air quality conditions just meeting the current standard, derived
32 by interpolation. A number of details, listed here, should be kept in mind when reviewing the
33 estimates presented in Tables 3-9 and 3-10.

- 34 • The risk estimates represent IQ loss associated with air-related lead exposure for the
35 median child (exposure is modeled through age seven years).

- 1 • Our estimation of risk attributable to air-related exposure pathways is approximate, as
2 described in sections 3.4.4 and 3.4.7. We consider the air-related risk to fall within
3 the ranges presented, bounded on the low end by estimates for the pathways
4 categorized as “recent air” and on the upper end by the sum of the estimates for both
5 the “recent” and “past air” pathways.⁴³
- 6 • The **bolded** range of risk estimates are derived using the C-R function in which we
7 have the highest overall confidence (the log-linear with low-exposure linearization –
8 see section 3.4.3.3.1). The wider range of risk estimates presented within the
9 parentheses in both tables reflects the application of all four C-R functions (see
10 section 3.4.3.3.1) to exposure estimates for the *recent* and *recent plus past air*
11 categories. Consequently, this range of risk estimates reflects both uncertainty in
12 estimation of air-related exposures as well as uncertainty in the C-R function for IQ
13 loss in children.
- 14

⁴³ The third category of pathways for which risk was estimated in the 2007 REA was comprised of diet and drinking water pathways. As other (nonair) sources of Pb can be appreciable contributors of Pb to these pathways, this category is referred to as “background” in the 2007 REA.

1 **Table 3-10. Estimates of air-related risk from 2007 risk assessment.**

Air Quality Scenario Just meeting specified maximum quarterly/monthly average ($\mu\text{g}/\text{m}^3$)	Median air-related IQ loss ^A				
	Generalized (local) urban case study	Primary Pb smelter (subarea) case study ^{B, C}	Location-specific urban case studies		
			Cleveland	Chicago	Los Angeles
Alternative Standards Scenarios					
1.5, max quarterly ^D (previous NAAQS)	3.5 - 4.8 (1.5 - 7.7)	< 6 <(3.2 - 9.4)	2.8 - 3.9 ^E (0.6 - 4.6)	3.4 - 4.7 ^E (1.4 - 7.4)	2.7 - 4.2 ^E (1.1 - 6.2)
0.5, max monthly	1.9 - 3.6 (0.7 - 4.8)	< 4.5 <(2.1 - 7.7)	0.6 - 2.9 (0.2 - 3.9)	F	F
0.2, max quarterly	1.5 - 3.4 (0.5 - 4.3)	<3.8 <(1.5 - 5.6)	0.43 - 2.8 (0.1 - 3.3)	F	F
0.2, max monthly	1.2 - 3.2 (0.4 - 4.0)	< 3.7 <(1.2 - 5.1)	0.6 - 2.8 (0.1 - 3.2)	0.6 - 2.9 (0.3 - 3.6)	0.7 - 2.9 ^G (0.2 - 3.5)
0.05, max monthly	0.5 - 2.8 (0.2 - 3.3)	< 2.8 <(0.9 - 3.4)	0.1 - 2.6 (<0.1 - 3.1)	0.2 - 2.6 (0.1 - 3.2)	0.3 - 2.7 (0.1 - 3.2)
0.02, max monthly	0.3 - 2.6 (0.1 - 3.1)	< 2.9 <(0.9 - 3.3)	<0.1 - 2.6 (<0.1 - 3.0)	0.1 - 2.6 (<0.1 - 3.1)	0.1 - 2.6 (<0.1 - 3.1)
Then-current (2003-2005) Conditions					
0.36, max quarterly			0.7 - 2.9 (0.2 - 3.6)		
0.14, max quarterly				0.6 - 2.9 (0.3 - 3.5)	
0.09, max quarterly					0.7 - 2.9 (0.2 - 3.5)
<p>A - Air-related risk is bracketed by "recent air" (lower bound of presented range) and "recent" plus "past air" (upper bound of presented range) (see section 3.4.4). Boldface estimates are generated using the log-linear with low-exposure linearization function. Values in parentheses reflect the range of estimates associated with all four concentration-response functions.</p> <p>B - In the case of the primary Pb smelter case study, only recent plus past air estimates are available.</p> <p>C - Median air-related IQ loss estimates for the primary Pb smelter (full study area) range from <1.7 to <2.9 points, with no consistent pattern across simulated NAAQS levels. This lack of a pattern reflects inclusion of a large fraction of the study population with relatively low ambient air impacts such that there is lower variation (at the population median) across standard levels (see section 4.2 of the Risk Assessment, Volume 1).</p> <p>D - This corresponds to roughly 0.7 - 1.0 $\mu\text{g}/\text{m}^3$ maximum monthly mean, across the urban case studies</p> <p>E - A "roll-up" was performed so that the highest monitor in the study area is increased to just meet this level.</p> <p>F - A "roll-up" to this level was not performed.</p> <p>G - A "roll-up" to this level was not performed; these estimates are based on current conditions in this area (0.17 max monthly).</p>					
The Information in this table is drawn from the 2007 REA, Table 5-9.					

2
3
4
5

1 **Table 3-11. Estimates of air-related risk for the generalized (local) urban case study,**
 2 **including interpolated estimates for current standard.**

Air Quality Scenario Just meeting specified metric ($\mu\text{g}/\text{m}^3$)			Median Air-related IQ Loss ^A for Generalized (local) Urban Case Study
Maximum Quarterly Average ^D	Maximum Monthly Average	Maximum 3-month Average	
1.5 (previous NAAQS)			3.5 - 4.8 (1.5 - 7.7)
	0.5		1.9 - 3.6 (0.7 - 4.8)
0.2			1.5 - 3.4 (0.5 - 4.3)
		0.15 ^B (current NAAQS)	1.5 - 3.4 (0.5 - 4.3)
	0.2		1.2 - 3.2 (0.4 - 4.0)
	0.05		0.5 - 2.8 (0.2 - 3.3)
	0.02		0.3 - 2.6 (0.1 - 3.1)
A - Air-related risk is bracketed by "recent air" (lower bound of presented range) and "recent" plus "past air" (upper bound of presented range) (see section 3.4.4 for additional detail on these categories). Boldface values are estimates generated using the log-linear with low-exposure linearization function. Values in parentheses reflect the range of estimates associated with all four concentration-response functions. B - Risk estimates interpolated - see text.			

3
 4 Key observations regarding these air-related risk estimates *across the array of air quality*
 5 *scenarios:*

6 ***All Case Studies***

- 7 • Relative to the previous Pb NAAQS, substantial reduction in estimates of air- related
 8 risk is demonstrated across the full set of potential alternative standards simulated
 9 (Table 3-10). This is particularly the case for the lower bound (the *recent air*
 10 estimates) which reflects only the pathways simulated to respond to changes in air
 11 concentrations associated with different air quality scenarios.

12 ***Generalized (local) Urban Case Study***

- 13 • As described above, the general urban case study provides risk estimates for a single
 14 group of children residing in a single area where air concentrations throughout area
 15 are near the level of the standard being simulated. In this case study, air-related
 16 median IQ loss, based on the higher confidence C-R function (bold), ranged from
 17 roughly 2 to 4 IQ points for the 0.5 $\mu\text{g}/\text{m}^3$, maximum monthly average scenario, to <1
 18 to roughly 3 IQ points for the 0.02 $\mu\text{g}/\text{m}^3$, maximum monthly average scenario.
 19 These ranges are expanded somewhat with consideration of the full range of C-R
 20 functions considered in the analysis. .

Location-specific Case Studies

- Compared to the other case studies, the air-related risk estimates for the location-specific urban case studies are lower because of the broader range of air-related exposures and the distribution of the population within the study areas. For example, the majority of the populations in each of the location-specific case studies reside in areas with ambient air Pb levels well below each standard assessed, particularly for standard levels above $0.05 \mu\text{g}/\text{m}^3$, as a maximum monthly average. Consequently, risk estimates for these case studies indicate little response to alternative standard levels above $0.05 \mu\text{g}/\text{m}^3$ maximum monthly average (as shown in Table 3-10).
- For the primary Pb smelter subarea, only an upper bound on risk attributable to air-related exposures is provided due to uncertainties associated with dust Pb model used for this case study.⁴⁴ For all air quality scenarios, the population median *recent plus past* air risk estimate is generally similar to or slightly higher than those for the general urban case study, likely due to differences in the indoor dust models used for the two case studies (discussed in Risk Assessment Report, sections 3.1.4).

Key observations regarding air-related risk estimates for **the current standard**:

- The median air-related IQ loss estimate for then current conditions in Chicago study area, which just met a level of $0.14 \mu\text{g}/\text{m}^3$ as a maximum calendar quarter average, falls somewhere within the lower and upper bounds of 0.6 and 2.9 points IQ loss, respectively (Table 3-10).
- The median air-related IQ loss estimate for the current standard in the Generalized (local) Urban Case Study, newly derived by interpolation from 2007 REA results, falls somewhere within the lower and upper bounds of 1.5 and 3.4 points IQ loss, respectively (Table 3-11). This estimate is derived by interpolation between the estimates for the $0.2 \mu\text{g}/\text{m}^3$ maximum quarterly and $0.2 \mu\text{g}/\text{m}^3$ maximum monthly average scenarios that were derived in 2007 REA. The newly interpolated estimate is essentially the same as the estimate for $0.2 \mu\text{g}/\text{m}^3$ maximum quarterly average scenario for this case study.
- Based on results from the last review for a location-specific urban study area and on those newly derived in this review based on interpolation from 2007 REA results, median air-related IQ loss for the current standard is estimated, with rounding, to generally fall above a rough lower bound of 1 point IQ loss and below a rough upper bound of 3 points IQ loss.

⁴⁴ The regression model used for estimating dust Pb concentrations in the primary Pb smelter case study does not lend itself to partitioning the *recent air* Pb from other contributions. Accordingly, this partitioning was not done for this case study (2007 REA, sections 2.4.3 and 3.1.4.2).

3.4.6 Treatment of Variability

This section discusses the degree to which the design of the previous risk assessment reflected consideration for key sources of variability associated with the scenarios evaluated (e.g., IQ loss in children associated with ambient Pb in various residential settings). In so doing, the following question is address.

- **How are key sources of variability treated in the assessment?**

Key sources of variability associated with the risk assessment include:

- *Variation in ambient air Pb levels among U.S. urban residential areas:* The location-specific urban study areas were chosen to provide coverage for diverse residential populations in U.S. urban areas with relatively elevated ambient air Pb levels (as characterized using monitoring). We believe that the three cities included in those case studies capture the variability in spatial patterns of ambient air Pb concentrations within and across such areas reasonably well. In addition, the generalized (local) urban case study provides coverage for a residential population exposed in a localized area with air concentrations somewhat near the standard being evaluated, providing coverage for a higher-end population risk scenario where a subset of urban children are exposed without any spatial gradient in ambient air Pb levels.
- *Variation in the spatial distribution of children within an urban area and assignment of ambient air Pb exposure:* Exposure of populations within the three location-specific urban study areas was characterized to the US Census block group-level characterization. Each block group was associated ambient air Pb exposure based on proximity to monitor nearest to block group centroid (Risk Assessment Report, section 5.2.2). While there is uncertainty associated with these exposures (population-weighted through use of census block groups) - for example, we did not consider time spent by children away from their residential block group - we believe that this approach provides reasonable coverage for the potential pattern of interaction between resident children located in these urban study areas and the associated spatial gradients of ambient air Pb levels.
- *Variation among children in factors other than media concentrations that influence PbB levels:* The inputs to the blood Pb model were central tendency estimates for each exposure zone within each study area (pathway-specific estimates for central-tendency child). The IEUBK model then generated PbB levels for that central-tendency child within each exposure zone. These central-tendency PbB levels were then combined with a GSD reflecting variability in PbB levels for young children (at/near age 7 years) to generate a distribution of total PbB for a group of children within each exposure zone in a given case study under the specific air quality scenario. We have reasonable confidence that this modeling approach captures variability in total Pb exposure (and consequently total PbB levels) for children modeled for a given study area. As noted in section 3.4.7 below, however, there is uncertainty associated with parsing individual pathway contributions to total PbB

1 levels (and consequently to total Pb-attributable risk). This is particularly true for
2 high-end population percentile estimates of IQ loss.

- 3 • *Inter-individual variability in IQ loss related to Pb exposure*: The use of C-R
4 functions based on a pooled analysis (Lanphear et al., 2005) which combined study
5 populations from a number of different epidemiological studies of IQ loss in children
6 likely provides reasonable coverage for variation across children regarding IQ loss
7 attributable to Pb exposure. Furthermore, our estimation of risk based on several C-R
8 functions provides further coverage for that variability, as well as also addressing
9 uncertainty in the specification of that C-R functional form (see section 3.4.7 below).

10 **3.4.7 Characterizing Uncertainty**

11 Although the risk assessment utilized a number of innovative modeling elements in order
12 to generate representative estimates of risk for the various study area populations, like all risk
13 models there was uncertainty associated with the model and its output.

- 14 • **What are the important uncertainties associated with any risk/exposure**
15 **estimates?**

16 One overarching area of uncertainty concerns the precision of our estimation of the
17 neurocognitive risk (as represented by IQ loss) associated with ambient air Pb. For example,
18 because of the evidence for a nonlinear response of PbB to Pb exposure and also for nonlinearity
19 in the C–R relationship for Pb-associated IQ loss, the assessment first estimated PbB levels and
20 associated risk for total Pb exposure (i.e., including Pb from air-related and nonair exposure
21 pathways), and then separated out estimates for pathways of interest. We separated out the
22 estimates of total (all-pathway) PbB and IQ loss into three categories that included two air-
23 related categories (“past” and “recent”), in addition to a third category for diet and drinking
24 water. However, significant limitations in our modeling tools affected our ability to develop
25 precise estimates for air-related exposure pathways. As recognized in section 3.4.4 above, we
26 believe these limitations led to a slight overestimation of the risks for the *past air* category and to
27 an under representation of air-related pathways for the *recent air* category. Thus, we
28 characterized the risk attributable to air-related exposure pathways to be bounded by the
29 estimates developed for the *past air* category and the sum of estimates for the *recent air* and *past*
30 *air* categories. For air quality scenarios other than those for the previous NAAQS, this upper
31 bound is recognized as having a potential upward bias with regard to its reflection of the
32 simulated air quality conditions because modeling and data limitations precluded simulation of
33 the influence of lower air Pb concentrations on the outdoor dust and soil exposure pathways, as
34 noted in section 3.4.4 above.

35 Additional limitations, assumptions and uncertainties, recognized in various ways in the
36 assessment and presentation of results, along with a concise characterization of their expected
37 impact on results, are listed below. The list begins with factors related to design of the

1 assessment or of the case studies, followed by those related to estimation of Pb concentrations in
2 ambient air, indoor dust, outdoor soil/dust, and blood, and estimation of Pb-related IQ loss.

- 3 • *Temporal Aspects:* During the 7-year exposure period, media concentrations remain
4 fixed and the simulated child resides at the same residence (although exposure
5 factors, including behavioral and physiological parameters, are adjusted to match the
6 aging of the child). While these aspects of simulation introduce uncertainty into the
7 risk estimates, it is not clear if there is a directional bias. For example, failure to
8 consider population mobility during the simulation period could bias the overall
9 distribution upwards unless children move to, or from a residential location with
10 higher Pb exposure, in which case this would not be the case.
- 11 • *Generalized (local) Urban Case Study:* The design for this case study employs
12 assumptions regarding uniformity that are reasonable in the context of a general
13 description of a small neighborhood population, but would contribute significant
14 uncertainty to extrapolation of these estimates to a specific urban location,
15 particularly a relatively large one. Thus, the risk estimates for this case study, while
16 generally representative of an urban residential population exposed to the specified
17 ambient air Pb levels, cannot be readily related to a specific large urban population.
18 As long as these important caveats are considered in interpreting the risk estimates
19 (i.e., these risk estimates are likely represent a relatively small portion of the resident
20 child population in any given city just meeting specific air quality conditions) then
21 the potential error of extrapolating these results to a larger child population can be
22 avoided. An additional area of uncertainty is with regard to the representation of
23 variability in air quality. Given the relatively greater variability common in areas of
24 high Pb concentrations, the approach used to reflect variability may bias the estimates
25 high, although there is uncertainty with regard to the representativeness of the
26 monitoring dataset used to characterize this.
- 27 • *Location-specific Urban Case Studies:* Limitations in the spatial density of ambient
28 air monitors in the three metropolitan areas simulated limit our characterization of
29 spatial gradients of ambient air Pb levels in these case studies. While this factor
30 introduces uncertainty into the risk estimates for this category of case study, it is not
31 clear if there is a directional bias.
- 32 • *Air Quality Simulation:* The proportional roll-up and roll-down procedures used in
33 some case studies to simulate air quality conditions just meeting the previous
34 NAAQS and alternative NAAQS, respectively, assume proportional changes in air
35 concentrations across those case study areas to create those air quality scenarios. The
36 EPA recognizes the uncertainty with our simulation of higher air Pb concentrations
37 that would just meet the previous NAAQS in the urban location-specific case studies,
38 as well as the uncertainty in simulation of conditions associated with the
39 implementation of emissions reduction actions to meet a lower standard. There is the
40 potential that the use of the proportional approach for adjusting monitor values
41 introduced a degree of high-bias into estimates of risk reduction. This could result if
42 we find that urban areas can target specific Pb sources impacting individual monitors,
43 thereby avoiding the need for more generalized reduction strategies resulting in a
44 uniform pattern of reduction across all monitors in an urban area. It is important to

- 1 point out however, that the generalized urban case study is not effected by this
2 potential bias, since, given the single exposure zone involved, the single ambient air
3 Pb level is fixed to reflect meeting the standard being considered (i.e., no spatial
4 gradient is simulated in the small, localized residential area being modeled).
- 5 • *Outdoor Soil/Dust Pb Concentrations:* Limitations in datasets on Pb levels in surface
6 soil/dust Pb in urban areas and in our ability to simulate the impact of reduced air Pb
7 levels related to lowering the NAAQS contributes uncertainty to air-related risk
8 estimates. In this case, it is likely that we have low-biased our estimates of risk
9 reduction associated with alternative (lower) Pb NAAQS levels, since we have not
10 simulated potential changes in soil Pb related to changes in ambient air Pb.
 - 11 • *Indoor Dust Pb Concentrations:* Limitations and uncertainty in modeling of indoor
12 dust Pb levels, including the impact of reductions in ambient air Pb levels, contributes
13 uncertainty to air-related risk estimates. Although our modeling of indoor dust does
14 link changes in ambient air Pb to changes in indoor dust Pb (via air exchanges and
15 indoor deposition onto surfaces), the modeling does not include a link between
16 ambient air Pb, outdoor soil Pb and subsequent changes in the level of Pb carried (or
17 “tracked”) into the house. This could introduce low-bias into our total estimates of
18 air-related Pb exposure and risk.
 - 19 • *Interindividual Variability in PbB Levels:* Uncertainty related to population
20 variability in PbB levels (i.e., interindividual variability in factors other than media
21 concentration that influence PbB) and limitations in modeling of this introduces
22 significant uncertainty into PbB and IQ loss estimates for the 95th percentile of the
23 population. We are not aware of any systematic bias introduced into the analysis from
24 this source of uncertainty.
 - 25 • *Pathway Apportionment for Higher Percentile PbB and risks:* Limitations, primarily
26 in data, prevented us from characterizing the degree of correlation among high-end
27 Pb exposures for the various pathways (e.g., the degree to which an individual
28 experiencing high drinking water Pb exposure would also experience high Pb paint
29 exposure and high ambient air-related Pb exposure). Our inability to characterize
30 potential correlations between exposure pathways (particularly at the higher
31 percentile exposure levels) limited our ability to (a) effectively model high-end Pb
32 risk and (b) apportion that risk between different exposure pathways, including
33 ambient air-related pathways.
 - 34 • *IQ Loss Concentration-response Functions:* Specification of the quantitative
35 relationship between PbB level and IQ loss is subject to greater uncertainty at lower
36 PbB levels (e.g., particularly below 2.5 µg/dL concurrent PbB). However, we believe
37 that by considering four different models (which each treat the response at low PbB
38 levels in a different manner), we have completed a reasonable characterization of this
39 source of uncertainty and its impact on risk estimates. Given comparison of risk
40 estimates generated using the four models, it would appear that this source of
41 uncertainty has a potentially significant impact on risk.

3.4.8 Updated Interpretation of Risk Estimates

As summarized in prior sections, a range of information gaps and areas of uncertainty were associated with the information available in the last review. In the REA Planning document, staff considered the degree to which information newly available since the last Pb NAAQS review, as summarized in the draft ISA, might address specific uncertainties associated with the 2007 REA, such that an updated risk model might be developed with the potential to provide new exposure and risk estimates substantially different⁴⁵ from estimates generated in 2007 (USEPA, 2011). Staff concluded that the newly available information did not provide the means by which to develop an updated or enhanced risk model that would substantially improve the utility of risk estimates in informing the Pb NAAQS review. Specifically it was concluded that none of the primary sources of uncertainty indentified to have the greatest impact on risk estimates would be substantially reduced through the use of newly available information (USEPA, 2011).

Our ongoing review of the newly available information leads us to conclude at this time, that the key observations regarding air-related Pb risk modeled for the set of standard levels covered in the 2007 REA, as well as the risk estimates interpolated for the current standard (as discussed in Section 3.4.5) are not significantly affected by the new information. Our overall characterization of uncertainty and variability associated with those estimates (as described above in sections 3.4.6 and 3.4.7) is not appreciably affected by new information. As recognized at the time of the last review, exposure and risk modeling conducted for this analysis was complex and subject to significant uncertainties due to limitations, data, and models, among other aspects. Further, limitations in the assessment design, data and modeling tools handicapped us from sharply separating Pb linked to ambient air from Pb that is not air-related.

In summary, the estimates of risk attributable to air-related exposures, with which we recognize a variety of sources of uncertainty, are considered to be approximate, falling within upper and lower bounds which over- and underestimate, respectively. In scenarios for more restrictive air quality conditions than those associated with the previous Pb standard, substantial reductions in air-related risk were demonstrated. Focusing on the results for the generalized (local) urban case study, the interpolated estimates for the scenario representing the current standard are very similar to estimates for the two 0.2 $\mu\text{g}/\text{m}^3$ scenarios (maximum monthly and quarterly averages) simulated in the 2007 REA,⁴⁶ and are appreciably lower than those

⁴⁵ In this context, “substantially different” has been intended to mean that the degree of uncertainty is substantially reduced, or bias is addressed, such that the new risk estimates could convey a different message regarding the magnitude of public health impacts associated with the current or potential alternative standards.

⁴⁶ There is uncertainty associated with judging differences between the current standard and these potential alternative standards due to the difference in air quality datasets used to estimate air concentration variability of the 2007 REA estimates versus the interpolated risk estimate.

1 associated with the previous standard. In characterizing the magnitude of air-related risk
2 associated with the current standard, we focusing on median estimates, for which we have
3 appreciably greater confidence than estimates for outer ends of risk distribution (see section
4 3.4.7) and on risks derived using the C-R function in which we have greatest confidence (see
5 sections 3.4.3.3.1 and 3.4.7). The risk results for the current standard from the last review for
6 one of the location-specific urban study area populations and those newly derived in this review
7 based on interpolation from 2007 REA results for the generalized (local) urban case study, which
8 is recognized to reflect a generalized high end of air-related exposure for localized populations,
9 indicate air-related risk for the current standard of a magnitude generally within the bounds of
10 roughly 1 and 3 points IQ loss.

1 3.5 REFERENCES

- 2 Amaral et al., 2010; Amaral, JH; Rezende, VB; Quintana, SM; Gerlach, RF; Barbosa, F, Jr; Tanus-Santos, JE.
3 (2010). The relationship between blood and serum lead levels in peripartum women and their respective
4 umbilical cords. *Basic Clin Pharmacol Toxicol* 107: 971-975. <http://dx.doi.org/10.1111/j.1742->
5 7843.2010.00616.x
- 6 Barry, PSI. (1975). A comparison of concentrations of lead in human tissues. *Occup Environ Med* 32: 119-139.
- 7 Barry, PSI. (1981). Concentrations of lead in the tissues of children. *Occup Environ Med* 38: 61-71.
- 8 Bellinger, D. C. and Needleman, H. L. (2003) Intellectual impairment and blood lead levels [letter]. *N. Engl. J. Med.*
9 349: 500.
- 10 Bellinger, D. 2008. Email message to Jee-Young Kim, U.S. EPA. February 13, 2008. Docket number EPA-HQ-
11 OAR-2006-0735.
- 12 Billick, I.H.; Curran, A.S.; Shier, D.R. (1979) Analysis of pediatric blood lead levels in New York City for 1970-
13 1976. *Environ. Health Perspect.* 31: 183-190.
- 14 Billick, I.H.; Curran, A.S.; Shier, D.R. (1980) Relation of pediatric blood lead levels to lead in gasoline. *Environ.*
15 *Health Perspect.* 34: 213-217.
- 16 Billick, I.H. (1983) Sources of lead in the environment. In: Rutter, M.; Russell Jones, R., eds. *Lead versus health:*
17 *sources and effects of low level lead exposure.* New York, NY: John Wiley and Sons, Ltd; pp. 59-77.
- 18 Brunekreef, B.; Noy, D.; Biersteker, K.; Boleij, J. (1983) Blood lead levels of Dutch city children and their
19 relationship to lead in the environment. *J. Air Pollut. Control Assoc.* 33: 872-876.
- 20 Brunekreef, B. (1984) The relationship between air lead and blood lead in children: a critical review. *Science of the*
21 *total environment*, 38: 79-123.
- 22 Canfield, R. L.; Henderson, C. R., Jr.; Cory-Slechta, D. A.; Cox, C.; Jusko, T. A.; Lanphear, B. P. (2003)
23 Intellectual impairment in children with blood lead concentrations below 10 µg per deciliter. *N. Engl. J.*
24 *Med.* 348: 1517-1526.
- 25 Canfield, R.L. 2008a. Email message to Jee-Young Kim, U.S. EPA. February 7, 2008. Docket number EPA-HQ-
26 OAR-2006-0735.
- 27 Canfield, R.L. 2008b. Email messages to Jee-Young Kim, U.S. EPA. August 11 and 12, 2008. Docket number
28 EPA-HQ-OAR-2006-0735.
- 29 Carbone, R; Laforgia, N; Crollo, E; Mautone, A; Iolascon, A. (1998). Maternal and neonatal lead exposure in
30 southern Italy. *Neonatology* 73: 362-366.
- 31 Centers for Disease Control (1991) Preventing lead poisoning in young children: a statement by the Centres for
32 Disease Control. Atlanta, GA: U.S. Department of health and Human Services, Public Health Service;
33 October 1. <http://wonder.cdc.gov/wonder/prevguid/p0000029/p0000029.asp>
- 34 Centers for Disease Control and Prevention (2005) Preventing lead poisoning in young children: a statement by the
35 Centers for Disease Control and Prevention. Atlanta, GA: U.S. Department of Health and Human Services,
36 Public Health Service. August.

- 1 Centers for Disease Control and Prevention (2012) CDC Response to Advisory Committee on Childhood Lead
2 Poisoning Prevention Recommendations in “Low Level Lead Exposure Harms Children: A Renewed Call
3 of Primary Prevention”. Atlanta, GA: U.S. Department of Health and Human Services, Public Health
4 Service. June 7.
- 5 Chandramouli, K; Steer, CD; Ellis, M; Emond, AM. (2009). Effects of early childhood lead exposure on academic
6 performance and behaviour of school age children. Arch Dis Child 94: 844-848.
7 <http://dx.doi.org/10.1136/adc.2008.149955>
- 8 Chuang, HY; Schwartz, J; Gonzales-Cossio, T; Lugo, MC; Palazuelos, E; Aro, A; Hu, H; Hernandez-Avila, M.
9 (2001). Interrelations of lead levels in bone, venous blood, and umbilical cord blood with exogenous lead
10 exposure through maternal plasma lead in peripartum women. Environ Health Perspect 109: 527-532.
11 <http://dx.doi.org/10.1289/ehp.01109527>
- 12 Franklin, CA; Inskip, MJ; Bacchanale, CL; Edwards, CM; Manton, WI; Edwards, E; O'Flaherty, EJ. (1997). Use of
13 sequentially administered stable lead isotopes to investigate changes in blood lead during pregnancy in a
14 nonhuman primate (*Macaca fascicularis*). Toxicol Sci 39: 109-119.
- 15 Frey, H.C. (2011) Letter from Dr. H. Christopher Frey, Chair, Clean Air Scientific Advisory Committee Lead
16 Review Panel, to Administrator Lisa P. Jackson. Re: Consultation on EPA’s Review of the National
17 Ambient Air Quality Standards for Lead: Risk and Exposure Assessment Planning Document. October 14,
18 2011.
- 19 Gulson, B; Mahaffey, KR; Mizon, KJ; Korsch, MJ; Cameron, MA; Vimpani, G. (1995). Contribution of tissue lead
20 to blood lead in adult female subjects based on stable lead isotope methods. Transl Res 125: 703-712.
- 21 Gulson, B; Mahaffey, KR; Jameson, CW; Patison, N; Law, AJ; Mizon, KJ; Korsch, MJ; Pederson, D. (1999).
22 Impact of diet on lead in blood and urine in female adults and relevance to mobilization of lead from bone
23 stores. Environ Health Perspect 107: 257-263. <http://dx.doi.org/10.1289/ehp.99107257>
- 24 Gulson, B; Mizon, KJ; Korsch, MJ; Palmer, JM; Donnelly, JB. (2003). Mobilization of lead from human bone tissue
25 during pregnancy and lactation: A summary of long-term research. Sci Total Environ 303: 79-104.
26 [http://dx.doi.org/10.1016/S0048-9697\(02\)00355-8](http://dx.doi.org/10.1016/S0048-9697(02)00355-8)
- 27 Gulson, B; Mizon, KJ; Palmer, JM; Korsch, MJ; Taylor, AJ; Mahaffey, KR. (2004a). Blood lead changes during
28 pregnancy and postpartum with calcium supplementation. Environ Health Perspect 112: 1499-1507.
29 <http://dx.doi.org/10.1289/ehp.6548>
- 30 Gulson, BL; Mizon, KJ; Davis, JD; Palmer, JM; Vimpani, G. (2004b). Identification of sources of lead in children in
31 a primary zinc-lead smelter environment. Environ Health Perspect 112: 52-60.
32 <http://dx.doi.org/10.1289/ehp.6465>
- 33 Harville, E. W.; Hertz-Picciotto, I.; Schramm, M.; Watt-Morse, M.; Chantala, K.; Osterloh, J.; Parsons, P. J.; Rogan,
34 W. (2005) Factors influencing the difference between maternal and cord blood lead. Occup. Environ. Med.
35 62: 263-290. Hayes, E.B.; McElvaine, M.D.; Orbach, H.G.; Fernandez, A.M.; Lyne, S.; Matte, T.D. (1994)
36 Long-term trends in blood lead levels among children in Chicago: Relationship to air lead levels. Pediatrics
37 93:195-200.
- 38 Henderson, R. (2007) Letter from Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory Committee, to
39 Administrator Stephen L. Johnson. Re: Clean Air Scientific Advisory Committee’s (CASAC) Review of
40 the 1st Draft Lead Staff Paper and Draft Lead Exposure and Risk Assessments. March 27, 2007.
- 41 Hilts, S. R. (2003) Effect of smelter emission reductions on children's blood lead levels. Sci. Total Environ. 303: 51-
42 58.

1 Hornung, R. 2008a. Email message to Jee-Young Kim, U.S. EPA. February 11, 2008. Docket number EPA-HQ-
2 OAR-2006-0735.

3 Hornung, R. 2008b. Email message to Jee-Young Kim, U.S. EPA. August 19, 2008. Docket number EPA-HQ-
4 OAR-2006-0735.

5 Hornung, RW; Lanphear, BP; Dietrich, KN. (2009). Age of greatest susceptibility to childhood lead exposure: A
6 new statistical approach. *Environ Health Perspect* 117: 1309-1312. <http://dx.doi.org/10.1289/ehp.0800426>

7 Jedrychowski, W; Perera, F; Maugeri, U; Miller, RL; Rembiasz, M; Flak, E; Mroz, E; Majewska, R; Zembala, M.
8 (2011). Intrauterine exposure to lead may enhance sensitization to common inhalant allergens in early
9 childhood: A prospective prebirth cohort study. *Environ Res* 111: 119-124.
10 <http://dx.doi.org/10.1016/j.envres.2010.11.002>

11 Kim, Y; Kim, BN; Hong, YC; Shin, MS; Yoo, HJ; Kim, JW; Bhang, SY; Cho, SC. (2009). Co-exposure to
12 environmental lead and manganese affects the intelligence of school-aged children. *Neurotoxicology* 30:
13 564-571. <http://dx.doi.org/10.1016/j.neuro.2009.03.012>

14 Krieg, EF, Jr; Butler, MA; M-h, C; Liu, T; Yesupriya, A; Dowling, N; Lindegren, ML. (2010). Lead and cognitive
15 function in VDR genotypes in the Third National Health and Nutrition Examination Survey. *Neurotoxicol*
16 *Teratol* 32: 262-272. <http://dx.doi.org/10.1016/j.ntt.2009.12.004>

17 Lagerkvist, BJ; Ekesrydh, S; Englyst, V; Nordberg, GF; Soderberg, HA; Wiklund, DE. (1996). Increased blood lead
18 and decreased calcium levels during pregnancy: A prospective study of Swedish women living near a
19 smelter. *Am J Public Health* 86: 1247-1252.

20 Lanphear, BP; Roghmann, KJ. (1997). Pathways of lead exposure in urban children. *Environ Res* 74: 67-73.

21 Lanphear, BP; Matte, TD; Rogers, J; Clickner, RP; Dietz, B; Bornschein, RL; Succop, P; Mahaffey, KR; Dixon, S;
22 Galke, W; Rabinowitz, M; Farfel, M; Rohde, C; Schwartz, J; Ashley, P; Jacobs, DE. (1998). The
23 contribution of lead-contaminated house dust and residential soil to children's blood lead levels: A pooled
24 analysis of 12 epidemiologic studies. *Environ Res* 79: 51-68. <http://dx.doi.org/10.1006/enrs.1998.3859>

25 Lanphear, BP; Dietrich, K; Auinger, P; Cox, C. (2000). Cognitive deficits associated with blood lead concentrations
26 <10 microg/dL in US children and adolescents. *Public Health Rep* 115: 521-529. Lanphear, B. P.; Hornung,
27 R.; Khoury, J.; Yolton, K.; Baghurst, P.; Bellinger, D. C.; Canfield, R. L.; Dietrich, K. N.; Bornschein, R.;
28 Greene, T.; Rothenberg, S. J.; Needleman, H. L.; Schnaas, L.; Wasserman, G.; Graziano, J.; Roberts, R.
29 (2005) Low-level environmental lead exposure and children's intellectual function: an international pooled
30 analysis. *Environ. Health Perspect.* 113: 894-899. Manton, WI. (1985). Total contribution of airborne lead
31 to blood lead. *Occup Environ Med* 42: 168-172. <http://dx.doi.org/10.1136/oem.42.3.168>

32 Miranda, ML; Kim, D; Reiter, J; Overstreet Galeano, MA; Maxson, P. (2009). Environmental contributors to the
33 achievement gap. *Neurotoxicology* 30: 1019-1024. <http://dx.doi.org/10.1016/j.neuro.2009.07.012>

34 National Center for Health Statistics. (2010) National Center for Health Statistics. (2011) Health, United States,
35 2011: With Special Feature on Socioeconomic Status and Health. Hyattsville, MD.
36 2012 <http://www.cdc.gov/nchs/data/hus/11.pdf>

37 Patel, AB; Prabhu, AS. (2009). Determinants of lead level in umbilical cord blood. *Indian Pediatr* 46: 791-
38 793. Pirkle, JL; Brody, DJ; Gunter, EW; Kramer, RA; Paschal, DC; Flegal, KM; Matte, TD. (1994). The
39 decline in blood lead levels in the United States: The National Health and Nutrition Examination Surveys
40 (NHANES). *JAMA* 272: 284-291. <http://dx.doi.org/10.1001/jama.1994.03520040046039>

- 1 Ranft, U; Delschen, T; Machtof, M; Sugiri, D; Wilhelm, M. (2008). Lead concentration in the blood of children and
2 its association with lead in soil and ambient air: Trends between 1983 and 2000 in Duisburg. *J Toxicol*
3 *Environ Health A* 71: 710-715. <http://dx.doi.org/10.1080/15287390801985117>
- 4 Rice, DC. (1990). Lead-induced behavioral impairment on a spatial discrimination reversal task in monkeys exposed
5 during different periods of development. *Toxicol Appl Pharmacol* 106: 327-333.
6 [http://dx.doi.org/10.1016/0041-008X\(90\)90251-O](http://dx.doi.org/10.1016/0041-008X(90)90251-O)
- 7 Rice, DC. (1992). Lead exposure during different developmental periods produces different effects on FI
8 performance in monkeys tested as juveniles and adults. *Neurotoxicology* 13: 757-770.
- 9 Rice, DC; Gilbert, SG. (1990). Sensitive periods for lead-induced behavioral impairment (nonspatial discrimination
10 reversal) in monkeys. *Toxicol Appl Pharmacol* 102: 101-109. [http://dx.doi.org/10.1016/0041-](http://dx.doi.org/10.1016/0041-008X(90)90087-B)
11 [008X\(90\)90087-B](http://dx.doi.org/10.1016/0041-008X(90)90087-B)Rothenberg, S. J.; Khan, F.; Manalo, M.; Jian, J.; Cuellar, R.; Reyes, S.; Acosta, S.;
12 Jauregui, M.; Diaz, M.; Sanchez, M.; Todd, A. C.; Johnson, C. (2000) Maternal bone lead contribution to
13 blood lead during and after pregnancy. *Environ. Res.* 82: 81-90.
- 14 Schnaas, L; Rothenberg, SJ; Flores, MF; Martinez, S; Hernandez, C; Osorio, E; Perroni, E. (2004). Blood lead
15 secular trend in a cohort of children in Mexico City (1987-2002). *Environ Health Perspect* 112: 1110-1115.
16 <http://dx.doi.org/10.1289/ehp.6636>
- 17 Schwartz, J., and Pitcher, H. (1989) The relationship between gasoline lead and blood lead in the United States. *J*
18 *Official Statistics* 5(4):421-431.
- 19 Schwemberger, MS, JE Mosby, MJ Doa, DE Jacobs, PJ Ashley, DJ Brody, MJ Brown, RL Jones, D Homa. (2005)
20 *Mortality and Morbidity Weekly Report* 54(20):513-516. May 27, 2005
- 21 Simon, DL; Maynard, EJ; Thomas, KD. (2007). Living in a sea of lead changes in blood- and hand-lead of infants
22 living near a smelter. *J Expo Sci Environ Epidemiol* 17: 248-259. <http://dx.doi.org/10.1038/sj.jes.7500512>
- 23 Smith, D. R.; Osterloh, J. D.; Flegal, A. R. (1996) Use of endogenous, stable lead isotopes to determine release of
24 lead from the skeleton. *Environ. Health Perspect.* 104: 60-66.
- 25 Surkan, PJ; Zhang, A; Trachtenberg, F; Daniel, DB; McKinlay, S; Bellinger, DC. (2007). Neuropsychological
26 function in children with blood lead levels <10 µg/dL. *Neurotoxicology* 28: 1170-1177.
27 <http://dx.doi.org/10.1016/j.neuro.2007.07.007>
- 28 Téllez-Rojo, M. M.; Bellinger, D. C.; Arroyo-Quiroz, C.; Lamadrid-Figueroa, H.; Mercado-García, A.; Schnaas-
29 Arrieta, L.; Wright, R. O.; Hernández-Avila, M.; Hu, H. (2006) Longitudinal associations between blood
30 lead concentrations < 10 µg/dL and neurobehavioral development in environmentally-exposed children in
31 Mexico City. *Pediatrics* 118: e323-e330.
- 32 Tellez-Rojo, M. 2008. Email message to Jee-Young Kim, U.S. EPA. February 11, 2008. Docket number EPA-HQ-
33 OAR-2006-0735.
- 34 Tripathi, R.M.; Raghunath, R.; A.V. Kumar; V.N. Sastry; S. Sadasivan. (2001) Atmospheric and children's blood
35 lead as indicators of vehicular traffic and other emission sources in Mumbai, India. *Sci Total Environ* 267:
36 101-108.
- 37 U.S. Environmental Protection Agency. (1986) Air quality criteria for lead. Research Triangle Park, NC: Office of
38 Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no.
39 EPA-600/8-83/028aF-dF. 4v. Available from: NTIS, Springfield, VA; PB87-142378.
- 40 U.S. Environmental Protection Agency. (1989) Review of the national ambient air quality standards for lead:
41 Exposure analysis methodology and validation: OAQPS staff report. Research Triangle Park, NC: Office of

- 1 Air Quality Planning and Standards; report no. EPA-450/2-89/011. Available on the web:
2 http://www.epa.gov/ttn/naaqs/standards/pb/data/rnaaqs1_eamv.pdf
- 3 U.S. Environmental Protection Agency. (1990) Air quality criteria for lead: supplement to the 1986 addendum.
4 Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and
5 Assessment Office; report no. EPA/600/8-89/049F. Available from: NTIS, Springfield, VA; PB91-138420.
- 6 U.S. Environmental Protection Agency. (2006) Air Quality Criteria for Lead. Washington, DC, EPA/600/R-
7 5/144aF. Available online at: http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_cr.html
- 8 U.S. Environmental Protection Agency. (2007a) Lead: Human Exposure and Health Risk Assessments for Selected
9 Case Studies, Volume I. Human Exposure and Health Risk Assessments – Full-Scale and Volume II.
10 Appendices. Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-452/R-07-
11 014a and EPA-452/R-07-014b.
- 12 U.S. Environmental Protection Agency. (2007b) Review of the National Ambient Air Quality Standards for Lead:
13 Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper. Office of Air Quality
14 Planning and Standards, Research Triangle Park, NC. EPA-452/R-07-013. Available at:
15 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_cr_sp.html
- 16 U.S. Environmental Protection Agency. (2011) Review of the National Ambient Air Quality Standards for Lead:
17 Risk and Exposure Assessment Planning Document. Office of Air Quality Planning and Standards,
18 Research Triangle Park, NC. EPA/452/P-11-003. Available at:
19 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_2010_pd.html
- 20 U.S. Environmental Protection Agency. (2012) Integrated Science Assessment for Lead (Third External Review
21 Draft). Washington, DC, EPA/600/R-10/075C. Available online at:
22 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_2010_isa.html
- 23 Zielhuis, R.L.; del Castilho, P.; Herber, R.F.M.; Wibowo, A.A.E.; Salle, H.J.A. (1979) Concentrations of lead and
24 other metals in blood of two and three year-old children living near a secondary smelter. Int. Arch. Occup.
25 Environ. Health 42: 231-239.

4 REVIEW OF THE PRIMARY STANDARD FOR LEAD

This chapter presents preliminary staff conclusions regarding the primary Pb standard. These preliminary staff conclusions are guided by consideration of key policy-relevant questions and based on the assessment and integrative synthesis of information presented in the ISA and by staff analyses and evaluations presented in chapters 2 and 3 herein. Final evaluations and staff conclusions, to be developed taking into consideration CASAC advice and public comment on this draft document, will be presented in the final PA. The final evaluations and staff conclusions will inform the Administrator's decisions on whether to retain or revise the existing primary standard for Pb.

Following an introductory section on the general approach for reviewing the primary standard (section 4.1), including a summary of considerations in the last review, the discussion in this chapter focuses on the central issue of whether the information available in this review supports or calls into question the adequacy of the current primary standard. Building on the responses to specific policy-relevant questions on the scientific evidence and exposure-risk information in chapter 3 above, presentation in section 4.2 is also organized into consideration of key policy-relevant questions framing evidence-based and exposure/risk-based considerations. The policy-relevant questions in this document are based on those included in the IRP (IRP, section 3.1). In section 4.3, preliminary staff conclusions are developed. Section 4.4 presents a brief overview of key uncertainties and areas for future research.

4.1 APPROACH

Staff's approach in this review of the current primary standard takes into consideration the approaches used in the previous Pb NAAQS review. The past and current approaches described below are both based, most fundamentally, on using EPA's assessment of the current scientific evidence and associated quantitative analyses to inform the Administrator's judgment regarding a primary standard for Pb that protects public health with an adequate margin of safety. We note that the final decision on the adequacy of the current standard is largely a public health policy judgment to be made by the Administrator. The Administrator's final decision must draw upon scientific information and analyses about health effects, population exposure and risks, as well as judgments about how to consider the range and magnitude of uncertainties that are inherent in the scientific evidence and analyses. Our approach to informing these judgments, discussed more fully below, is based on the recognition that the available health effects evidence generally reflects a continuum, consisting of ambient levels at which scientists generally agree that health effects are likely to occur, through lower levels at which the likelihood and magnitude of the response become increasingly uncertain. This approach is consistent with the

1 requirements of the NAAQS provisions of the Act and with how the EPA and the courts have
2 historically interpreted the Act. These provisions require the Administrator to establish primary
3 standards that, in the Administrator’s judgment, are requisite to protect public health with an
4 adequate margin of safety. In so doing, the Administrator seeks to establish standards that are
5 neither more nor less stringent than necessary for this purpose. The Act does not require that
6 primary standards be set at a zero-risk level, but rather at a level that avoids unacceptable risks to
7 public health. The four basic elements of the NAAQS (indicator, averaging time, level, and
8 form) are considered collectively in evaluating the health protection afforded by the current
9 standard.

10 The following subsections include background information on the approach used in the
11 previous review of the standard (section 4.1.1) and a discussion of the approach for the current
12 review (section 4.1.2).

13 **4.1.1 Approach Used in the Last Review**

14 The last review of the NAAQS for Pb was completed in 2008 (73 FR 66964). In
15 consideration of the much-expanded health effects evidence on neurocognitive effects of Pb in
16 children available at that time, the EPA substantially revised the primary standard from 1.5
17 $\mu\text{g}/\text{m}^3$, as a not-to-be exceeded average concentration over a calendar quarter, to a level of 0.15
18 $\mu\text{g}/\text{m}^3$, as a not-to-be-exceeded rolling 3-month average concentration. The 2008 decision to
19 revise the primary standard was based on the extensive body of scientific evidence published
20 over almost three decades, from the time the standard was originally set in 1978 through 2005-
21 2006. The 2008 decision considered the body of evidence as assessed in the 2006 CD (USEPA,
22 2006) as well as the 2007 Staff Paper assessment of the policy-relevant information contained in
23 the CD and the quantitative risk/exposure assessment (USEPA, 2007a, 2007b), the advice and
24 recommendations of CASAC (Henderson 2007a, 2007b, 2008a, 2008b), and public comment.

25 While recognizing that Pb has been demonstrated to exert “a broad array of deleterious
26 effects on multiple organ systems”, the review focused on the effects most pertinent to ambient
27 air exposures, which given ambient air Pb reductions over the past 30 years are those associated
28 with relatively lower exposures and associated blood Pb levels (73 FR 66975). In so doing, the
29 EPA recognized the general consensus that the developing nervous system in children is among,
30 if not the most sensitive health endpoint associated with Pb exposures. Thus, primary attention
31 was given to consideration of nervous system effects, including neurocognitive and
32 neurobehavioral effects, in children (73 FR 66976). The body of evidence included associations
33 of such effects in study populations of variously-aged children with mean blood Pb levels below
34 10 $\mu\text{g}/\text{dL}$, extending from 8 down to 2 $\mu\text{g}/\text{dL}$ (73 FR 66976). The public health implications of

1 effects of air-related Pb on cognitive function (e.g., IQ) in young children were given particular
2 focus in the review.

3 **4.1.1.1 Conclusion Regarding the Need for Revision**

4 The conclusions reached by the Administrator in the last review were based primarily on
5 the scientific evidence, with the risk- and exposure-based information providing support for
6 various aspects of the decision. In reaching his conclusion on the adequacy of the prior standard,
7 the Administrator placed primary consideration on the large body of scientific evidence available
8 in the review including significant new evidence concerning effects at blood Pb concentrations
9 substantially below those identified when the NAAQS for Pb was initially set in 1978 (73 FR
10 66987; 43 FR 46246). Given particular attention was the robust evidence of neurotoxic effects
11 of Pb exposure in children, recognizing: (1) that while blood Pb levels in U.S. children had
12 decreased notably since the late 1970s, newer epidemiologic studies had investigated and
13 reported associations of effects on the neurodevelopment of children with those more recent
14 lower blood Pb levels and (2) that the toxicological evidence included extensive experimental
15 laboratory animal evidence substantiating well the plausibility of the epidemiologic findings
16 observed in human children and expanding our understanding of likely mechanisms underlying
17 the neurotoxic effects (73 FR 66987). Additionally, within the range of blood Pb levels
18 investigated in the available evidence base, a threshold level for neurocognitive effects was not
19 identified (73 FR 66984; 2006 CD, p. 8-67). Further, the evidence indicated a steeper dose-
20 response relationship for effects on cognitive function at those lower blood Pb levels than at
21 higher blood Pb levels that were more common in the past, “indicating the potential for greater
22 incremental impact associated with exposure at these lower levels” (73 FR 66987). As at the
23 time when the standard was set, the health effects evidence and exposure/risk assessment
24 supported the conclusion that air-related Pb exposure pathways contribute to blood Pb levels in
25 young children by inhalation and ingestion (73 FR 66987). The available information also
26 suggested that the air-to-blood ratio was likely larger, when all inhalation and ingestion pathways
27 for air-related Pb are considered, than the air-to-blood ratio (of 2 µg/dL blood Pb to 1 µg/m³ air
28 Pb) estimated when the standard was initially set in 1978 (73 FR 66987).

29 In the Administrator’s decision on the adequacy of the 1978 standard, the Administrator
30 considered the evidence using a very specifically defined framework, referred to as an air-related
31 IQ loss evidence-based framework. This framework integrates evidence for the relationship
32 between Pb in air and Pb in young children’s blood with evidence for the relationship between
33 Pb in young children’s blood and IQ loss (73 FR 77987), as described in more detail in section
34 4.1.1.2 below. This evidence-based approach considers air-related effects on neurocognitive
35 function (using the quantitative metric of IQ loss) associated with exposure in those areas with

1 elevated air concentrations equal to potential alternative levels for the Pb standard. Thus, the
2 conceptual context for the framework is that it provides estimates of air-related IQ loss for a
3 subset of the population of U.S. children (i.e., the subset living in close proximity to air Pb
4 sources that contribute to elevated air Pb concentrations that equal the current level of the
5 standard). This is the subset expected to experience air-related Pb exposures at the high end of
6 the national distribution of such exposures, not at the average. This is the case since when a
7 standard of a particular level is just met at a monitor sited to record the highest source-oriented
8 concentration in an area, the large majority of children in the larger surrounding area would
9 likely experience exposures to concentrations well below that level.

10 The two primary inputs to the evidence-based air-related IQ loss framework are air-to-
11 blood ratios and C-R functions for the relationship between blood Pb and IQ response in young
12 children. Additionally taken into consideration in applying and drawing conclusions from the
13 framework were the uncertainties inherent in these inputs. Application of the framework also
14 entailed consideration of an appropriate level of protection from air-related IQ loss to be used in
15 conjunction with the framework. In simplest terms, the framework provides for estimation of a
16 mean air-related IQ decrement for young children in the high end of the national distribution of
17 air-related exposures by focusing on children exposed to air-related Pb in those areas with
18 elevated air Pb concentrations equal to specific potential standard levels. The framework
19 estimates of mean air-related IQ loss are derived through multiplication of the following factors:
20 standard level ($\mu\text{g}/\text{m}^3$), air-to-blood ratio in terms of $\mu\text{g}/\text{dL}$ blood Pb per $\mu\text{g}/\text{m}^3$ air concentration
21 and slope for the C-R function in terms of points IQ decrement per $\mu\text{g}/\text{dL}$ blood Pb.

22 Based on the application of the air-related IQ loss framework to the evidence, the
23 Administrator concluded that, for exposures projected for air Pb concentrations at the level of the
24 1978 standard, the quantitative estimates of IQ loss associated with air-related Pb indicated risk
25 of a magnitude that in his judgment was significant from a public health perspective, and that the
26 evidence-based framework supported a conclusion that the 1978 standard did not protect public
27 health with an adequate margin of safety (73 FR 77987). The Administrator further concluded
28 that the evidence indicated the need for a substantially lower standard level to provide increased
29 public health protection, especially for at-risk groups (most notably children), against an array of
30 effects, most importantly including effects on the developing nervous system (73 FR 77987). In
31 addition to giving primary consideration to the much expanded evidence base since the standard
32 was set, the Administrator also took into consideration the exposure/risk assessments. In so
33 doing, he observed that, while taking into consideration their inherent uncertainties and
34 limitations, the quantitative estimates of IQ loss associated with air-related Pb in air quality
35 scenarios just meeting the then-current standard also indicated risk of a magnitude that in his
36 judgment was significant from a public health perspective. Thus, the Administrator concluded

1 the exposure/risk estimates provided additional support to the evidence-based conclusion that the
2 standard needed revision (73 FR 66987).

3 **4.1.1.2 Conclusions on Elements of Revised Standard**

4 In considering appropriate revisions to the prior standard in the review completed in
5 2008, each of the four basic elements of the NAAQS (indicator, averaging time, level, and form)
6 were evaluated. The rationale for decisions on those elements is summarized below.

7 With regard to indicator, consideration was given to replacing Pb-TSP with Pb-PM₁₀.
8 The EPA recognized, however, that Pb in all particle sizes contributes to Pb in blood and
9 associated health effects, additionally noting that the difference in particulate Pb captured by
10 TSP and PM₁₀ monitors may be on the order of a factor of two in some areas (73 FR 66991).
11 Further, the Administrator recognized uncertainty with regard to whether a Pb-PM₁₀-based
12 standard would also effectively control ultra-coarse¹ Pb particles, which may have a greater
13 presence in areas near sources where Pb concentrations are highest (73 FR 66991). The
14 Administrator decided to retain Pb-TSP as the indicator to provide sufficient public health
15 protection from the range of particle sizes of ambient air Pb, including ultra-coarse particles (73
16 FR 66991). Additionally, a role was provided for Pb-PM₁₀ in the monitoring required for a Pb-
17 TSP standard (73 FR 66991) based on the conclusion that use of Pb-PM₁₀ measurements at sites
18 not influenced by sources of ultra-coarse Pb, and where Pb concentrations are well below the
19 standard, would take advantage of the increased precision of these measurements and decreased
20 spatial variation of Pb-PM₁₀ concentrations, without raising the same concerns over a lack of
21 protection against health risks from all particulate Pb emitted to the ambient air that support
22 retention of Pb-TSP as the indicator (*versus* revision to Pb-PM₁₀) (73 FR 66991). Accordingly,
23 allowance was made for the use of Pb-PM₁₀ monitoring for Pb NAAQS attainment purposes in
24 certain limited circumstances, at non-source-oriented sites, where the Pb concentrations are
25 expected to be substantially below the standard and ultra-coarse particles are not expected to be
26 present (73 FR 66991).

27 With regard to averaging time and form, consideration was given to monthly and 3-month
28 averaging times. The Administrator recognized the complexity related to the multimedia nature
29 of Pb and its multiple pathways of human exposure, in addition to uncertainty remaining over the
30 period of time needed for air Pb concentrations to lead to the health effects most at issue in this
31 review (73 FR 77995). Relevant factors pertaining to the human physiological response to
32 changes in Pb exposures and those pertaining to the response of air-related Pb exposure

¹ The term ultra-coarse is used to refer to particles collected by a TSP sampler but not by a PM₁₀ sampler. This terminology is consistent with the traditional usage of “fine” to refer to particles collected by a PM_{2.5} sampler, and “coarse” to refer to particles collected by a PM₁₀ sampler but not by a PM_{2.5} sampler, recognizing that there will be some overlap in the particle sizes in the three types of collected material.

1 pathways to changes in airborne Pb were considered in an integrated manner (73 FR 77996).
2 Key considerations included the limited evidence indicating more numerous factors influencing
3 ingestion (versus inhalation) pathways, which were considered likely to lessen the impact of
4 month-to-month variations in airborne Pb concentrations on levels of air-related Pb in children's
5 blood. Such factors were considered likely to lead to response times (e.g., for the response of
6 blood to air Pb via these pathways) extending longer than a month (73 FR 66996). The EPA
7 additionally considered the control that a rolling 3-month averaging time can provide (e.g.,
8 compared to a block calendar quarter) on month-to-month variability in air Pb concentrations
9 and in associated exposures (73 FR 66996). Based on this integrated consideration of the range
10 of relevant factors, the averaging time was revised to a rolling three-month period with a
11 maximum (not-to-be-exceeded) form, evaluated over a three-year period. As compared to the
12 previous averaging time and form of calendar quarter (not-to-be exceeded), this revision was
13 considered to be more scientifically appropriate and more health protective (73 FR 77996). The
14 rolling average gives equal weight to all three-month periods, and the new calculation method
15 gives equal weight to each month within each three-month period (73 FR 77996). Further, the
16 rolling average yields 12 three-month averages each year to be compared to the NAAQS versus
17 four averages in each year for the block calendar quarters pertaining to the previous standard (73
18 FR 77996).

19 Lastly, based on the body of scientific evidence and information available, as well as
20 CASAC recommendations and public comment, the Administrator decided on a standard level
21 that, in combination with the specified choice of indicator, averaging time, and form, he judged
22 requisite to protect public health, including the health of sensitive groups, with an adequate
23 margin of safety (73 FR 67006). In reaching the decision on level for the revised standard, the
24 Administrator considered as a useful guide the evidence-based framework developed in that
25 review. As described in section 4.1.1.1 above, that framework integrates evidence for the
26 relationship between Pb in air and Pb in children's blood and the relationship between Pb in
27 children's blood and IQ loss. Application of the air-related IQ loss evidence-based framework
28 was recognized, however, to provide "no evidence- or risk-based bright line that indicates a
29 single appropriate level" for the standard (73 FR 67006). Rather, the framework was seen as a
30 useful guide for consideration of health risks from exposure to ambient levels of Pb in the air, in
31 the context of a specified averaging time and form, with regard to the Administrator's decision
32 on a level for a revised NAAQS that provides public health protection that is sufficient but not
33 more than necessary under the Act (73 FR 67004).

34 As noted above, use of the evidence-based air-related IQ loss framework to inform
35 selection of a standard level involved consideration of the evidence with regard to two input
36 parameters. The two input parameters are an air-to-blood ratio and a C-R function for population

1 IQ response associated with blood Pb level (73 FR 67004). The evidence at the time of the last
2 review indicated a broad range of air-to-blood ratio estimates, each with limitations and
3 associated uncertainties. Based on the then-available evidence, the Administrator concluded that
4 1:5 to 1:10 represented a reasonable range to consider and identified 1:7 as a generally central
5 value on which to focus (73 FR 67004). With regard to C-R functions, in light of the evidence of
6 nonlinearity and of steeper slopes at lower blood Pb levels, the Administrator concluded it was
7 appropriate to focus on C-R analyses based on blood Pb levels that most closely reflected the
8 then-current population of children in the U.S.,² recognizing EPA's identification of four such
9 analyses and giving weight to the central estimate or median of the resultant C-R functions (73
10 FR 67003, Table 3; 73 FR 67004). The four study groups from which C-R functions were drawn
11 in 2008, and the associated C-R slopes, are summarized in Table 3-3 above.³ The median
12 estimate of -1.75 IQ points decrement per $\mu\text{g}/\text{dL}$ was selected for use with the framework. With
13 the framework, as summarized in section 4.1.1.1 above, potential alternative standard levels
14 ($\mu\text{g}/\text{m}^3$) are multiplied by estimates of air-to-blood ratio ($\mu\text{g}/\text{dL}$ blood Pb per $\mu\text{g}/\text{m}^3$ air Pb) and
15 the median slope for the C-R function (points IQ decrement per $\mu\text{g}/\text{dL}$ blood Pb), yielding
16 estimates of a mean air-related IQ decrement for a specific subset of young children (i.e., those
17 children exposed to air-related Pb in areas with elevated air Pb concentrations equal to specified
18 alternative levels). As such, the application of the framework yields estimates for the mean air-
19 related IQ decrements of the subset of children expected to experience air-related Pb exposures
20 at the high end of the distribution of such exposures. The associated mean IQ loss estimate is the
21 average for this highly exposed subset and is not the average air-related IQ loss projected for the
22 entire U.S. population of children. Uncertainties and limitations were recognized in the use of
23 the framework and in the resultant estimates (73 FR 67000).

24 In considering the use of the evidence-based air-related IQ loss framework to inform his
25 judgment as to the appropriate degree of public health protection that should be afforded by the
26 NAAQS to provide requisite protection against risk of neurocognitive effects in sensitive
27 populations, such as IQ loss in children, the Administrator recognized in the 2008 review that
28 there were no commonly accepted guidelines or criteria within the public health community that
29 would provide a clear basis for such a judgment. During the 2008 review, CASAC commented

² The geometric mean blood Pb level for U.S. children aged five years and below, reported for NHANES in 2003-04 (the most recent years for which such an estimate was available at the time of the 2008 decision) was 1.8 $\mu\text{g}/\text{dL}$ and the 5th and 95th percentiles were 0.7 $\mu\text{g}/\text{dL}$ and 5.1 $\mu\text{g}/\text{dL}$, respectively (73 FR 67002).

³ One of these four is from the analysis of the lowest blood Pb subset of the pooled international study by Lanphear et al., (2005). The nonlinear model developed from the full pooled dataset is the basis of the C-R functions used in the 2007 REA (see section 3.4.3.3 above), in which risk was estimated over a large range of blood Pb levels. Given the narrower focus of the evidence-based framework on IQ response at the end of studied blood Pb levels (closer to U.S. mean level), the C-R functions in Table 3-3 are from linear analyses (each from separate publications) for the study group subsets with blood Pb levels closest to mean for children in the U.S. today.

1 regarding the significance from a public health perspective of a 1-2 point IQ loss in the entire
2 population of children and, along with some commenters, emphasized that the NAAQS should
3 prevent air-related IQ loss of a significant magnitude, such as on the order of 1-2 IQ points, in all
4 but a small percentile of the population. Similarly, the Administrator stated that “ideally air-
5 related (as well as other) exposures to environmental Pb would be reduced to the point that no IQ
6 impact in children would occur” (73 FR 66998). The Administrator further recognized that, in
7 the case of setting a NAAQS, he was required to make a judgment as to what degree of
8 protection is requisite to protect public health with an adequate margin of safety (73 FR66998).
9 The NAAQS must be sufficient but not more stringent than necessary to achieve that result, and
10 does not require a zero-risk standard (73 FR 66998). The Administrator additionally recognized
11 that the evidence-based air-related IQ loss framework did not provide estimates pertaining to the
12 U.S. population of children as a whole. Rather, the framework provides estimates (with
13 associated uncertainties and limitations) for the mean of a subset of that population, the subset of
14 children assumed to be exposed to the level of the standard. As described in the final decision
15 “[t]he framework in effect focuses on the sensitive subpopulation that is the group of children
16 living near sources and more likely to be exposed at the level of the standard” (73 FR 67000).
17 As further noted in the final decision (73 FR 67000):

18 *EPA is unable to quantify the percentile of the U.S. population of children that*
19 *corresponds to the mean of this sensitive subpopulation. Nor is EPA confident in*
20 *its ability to develop quantified estimates of air-related IQ loss for higher*
21 *percentiles than the mean of this subpopulation. EPA expects that the mean of*
22 *this subpopulation represents a high, but not quantifiable, percentile of the U.S.*
23 *population of children. As a result, EPA expects that a standard based on*
24 *consideration of this framework would provide the same or greater protection*
25 *from estimated air-related IQ loss for a high, albeit unquantifiable, percentage of*
26 *the entire population of U.S. children.*
27

28 In reaching a judgment as to the appropriate degree of protection, the Administrator
29 considered advice and recommendations from CASAC and public comments and recognized the
30 uncertainties in the health effects evidence and related information as well as the role of, and
31 context for, a selected air-related IQ loss in the application of the framework. In so doing, the
32 Administrator identified an air-related IQ loss of 2 points to be used in conjunction with the
33 evidence-based framework in considering an appropriate level for the standard (73 FR 67005).⁴
34 Given the various uncertainties associated with the framework and the scientific evidence base,
35 and the focus of the framework on the sensitive subpopulation of more highly exposed children,

⁴ Also noted that “in determining what level of estimated IQ loss should be used for evaluating the results obtained from this specific evidence-based framework, the Administrator is not determining that such an IQ loss is appropriate for use in other contexts”. (73 FR 67005)

1 a standard level selected using this air-related IQ loss, in combination with the selected
2 averaging time and form, was expected to significantly reduce and limit for a high percentage of
3 U.S. children the risk of experiencing an air-related IQ loss of that magnitude (73 FR 67005). At
4 the standard level of $0.15 \mu\text{g}/\text{m}^3$, with the combination of the generally central estimate of air-to-
5 blood ratio of 1:7 and the median of the four C-R functions (-1.75 IQ point decrement per $\mu\text{g}/\text{dL}$
6 blood Pb), the framework estimates of air-related IQ loss were below 2 IQ points (73 FR 67005,
7 Table 4).

8 In reaching the decision in 2008 on level for the revised standard, the Administrator also
9 considered the results of the quantitative risk assessment to provide a useful perspective on risk
10 from air-related Pb. In light of important uncertainties and limitations for purposes of evaluating
11 potential standard levels, however, the Administrator placed less weight on the risk estimates
12 than on the evidence-based assessment. Nevertheless, in recognition of the general
13 comparability of quantitative risk estimates for the case studies considered most conceptually
14 similar to the scenario represented by the evidence-based framework, he judged the quantitative
15 risk estimates to be “roughly consistent with and generally supportive” of the evidence-based
16 framework estimates (73 FR 67006).

17 Based on consideration of the entire body of evidence and information available in the
18 review, as well as the recommendations of CASAC and public comments, the Administrator
19 decided that a level for the primary Pb standard of $0.15 \mu\text{g}/\text{m}^3$, in combination with the specified
20 choice of indicator, averaging time and form was requisite to protect public health, including the
21 health of sensitive groups, with an adequate margin of safety (73 FR 67006). In reaching
22 decisions on level as well as the other elements of the revised standard, the Administrator took
23 note of the complexity associated with consideration of health effects caused by different
24 ambient air concentrations of Pb and with uncertainties with regard to the relationships between
25 air concentrations, exposures, and health effects. For example, selection of a maximum, not to
26 be exceeded, form in conjunction with a rolling 3-month averaging time over a three year span
27 was expected to have the effect that the at-risk subpopulation of children would be exposed
28 below the standard most of the time (73 FR 67005). The Administrator additionally noted his
29 consideration of the provision of an adequate margin of safety in making decisions on each of the
30 elements of the standard, including, for example “selection of TSP as the indicator and the
31 rejection of the use of PM_{10} scaling factors; selection of a maximum, not to be exceeded form, in
32 conjunction with a 3-month averaging time that employs a rolling average, with the requirement
33 that each month in the 3-month period be weighted equally (rather than being averaged by
34 individual data) and that a 3-year span be used for comparison to the standard; and, the use of a
35 range of inputs for the evidence-based framework, that includes a focus on higher air-to-blood
36 ratios than the lowest ratio considered to be supportable, and steeper rather than shallower C-R

1 functions, and the consideration of these inputs in selection of 0.15 $\mu\text{g}/\text{m}^3$ as the level of the
2 standard” (73 FR 67007).

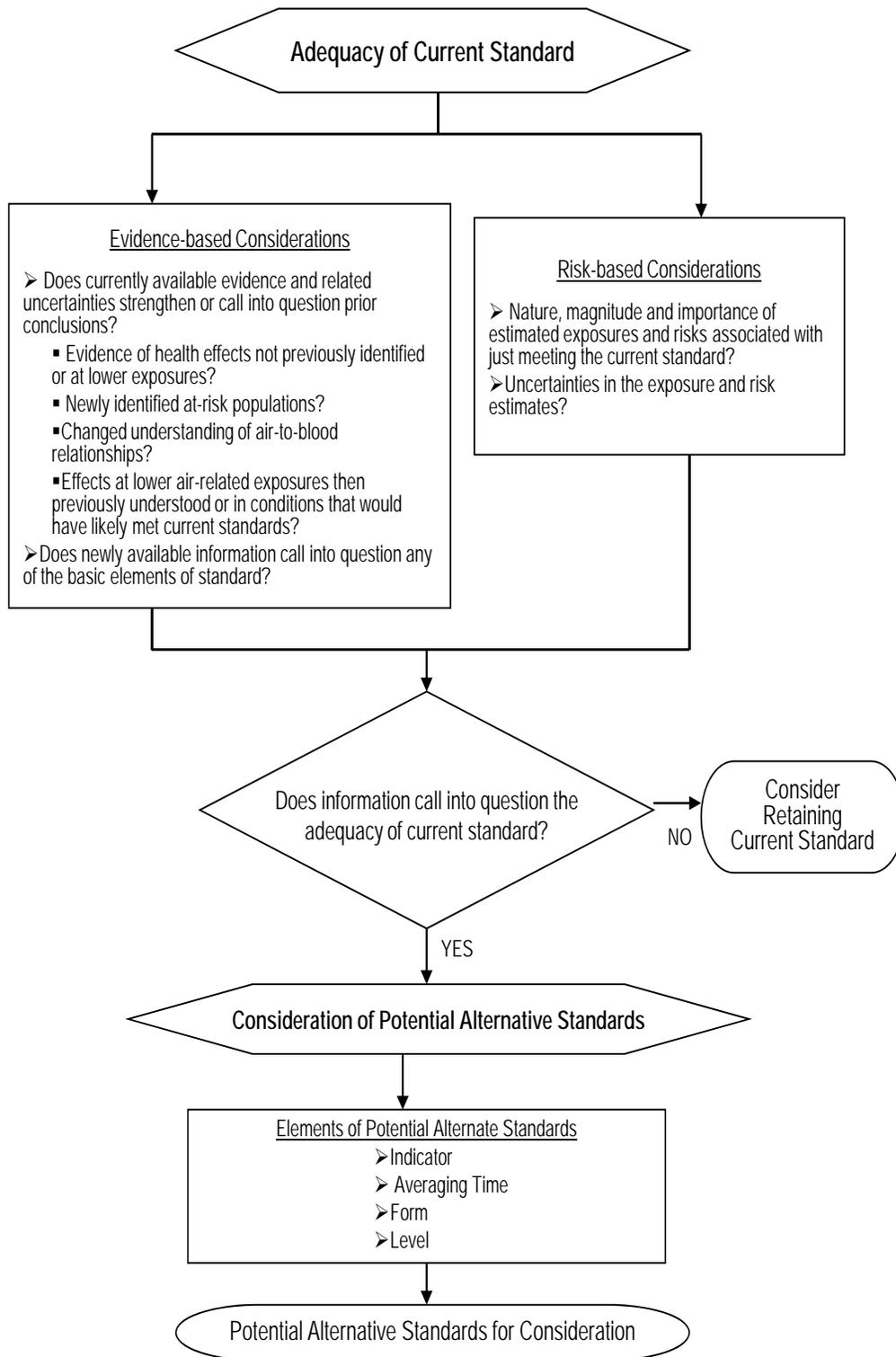
3 He additionally noted that a standard with this level would reduce the risk of a variety of
4 health effects associated with exposure to Pb, including effects indicated in the epidemiological
5 studies at lower blood Pb levels, particularly including neurological effects in children, and the
6 potential for cardiovascular and renal effects in adults (73 FR 67006). The Administrator
7 additionally considered higher and lower levels for the standard, concluding that a level of 0.15
8 $\mu\text{g}/\text{m}^3$ provides for a standard that is neither more or less stringent than necessary for this
9 purpose, recognizing that the Clean Air Act does not require that primary standards be set at a
10 zero-risk level, but rather at a level that reduces risk sufficiently so as to protect public health
11 with an adequate margin of safety (73 FR 67007). For example, the Administrator additionally
12 considered potential public health protection provided by standard levels above 0.15 $\mu\text{g}/\text{m}^3$,
13 which he concluded were insufficient to protect public health with an adequate margin of safety.
14 The Administrator also noted that in light of all of the evidence, including the evidence-based
15 framework, that the degree of public health protection likely afforded by standard levels below
16 0.15 $\mu\text{g}/\text{m}^3$ would be greater than what is necessary to protect public safety with an adequate
17 margin of safety.

18 The Administrator concluded, based on review of all of the evidence (including the
19 evidence- based framework), that when taken as a whole the selected standard, including the
20 indicator, averaging time, form, and level, would be “sufficient but not more than necessary to
21 protect public health, including the health of sensitive subpopulations, with an adequate margin
22 of safety” (73 FR 67007).

23 **4.1.2 Approach for the Current Review**

24 To evaluate whether it is appropriate to consider retaining the current primary Pb
25 standard, or whether consideration of revision is appropriate, we have adopted an approach in
26 this review that builds upon the general approach used in the last review and reflects the broader
27 body of evidence and information now available. As summarized above, the Administrator’s
28 decisions in the prior review were based on an integration of information on health effects
29 associated with exposure to Pb, relationships between ambient air Pb and blood Pb; expert
30 judgments on the adversity and public health significance of key health effects; and policy
31 judgments as to when the standard is requisite to protect public health with an adequate margin
32 of safety. These considerations were informed by air quality and related analyses, quantitative
33 exposure and risk assessments, and qualitative assessment of impacts that could not be
34 quantified.

1 In conducting this assessment, we draw on the current evidence and quantitative
2 assessments of exposure pertaining to the public health risk of Pb in ambient air. In considering
3 the scientific and technical information, we consider both the information available at the time of
4 the last review and information newly available since the last review, including the current ISA
5 (USEPA, 2012), as well as the quantitative exposure/risk assessments from the last review that
6 estimated Pb-related IQ decrements associated with different air quality conditions in simulated
7 at-risk populations in multiple case studies (USEPA, 2007a). Figure 4-1 illustrates the basic
8 construct of our two part approach in developing conclusions regarding options appropriate for
9 the Administrator to consider in this review with regard to the adequacy of the current standard
10 and, as appropriate, potential alternate standards. In the boxes of Figure 4-1, the range of
11 questions considered in chapter 3 above and section 4.2 below are represented by a summary of
12 policy-relevant questions that frame our consideration of the scientific evidence and
13 exposure/risk information.



1

2 **Figure 4-1. Overview of approach for review of current primary standard.**

1 **4.2 ADEQUACY OF THE CURRENT STANDARD**

2 In considering the adequacy of the current Pb standard, the overarching question we
3 consider is:

- 4 • **Does the currently available scientific evidence- and exposure/risk-based**
5 **information, as reflected in the ISA and REA, support or call into question the**
6 **adequacy of the protection afforded by the current Pb standard?**

7 In considering the scientific and technical information, we give our attention to both the
8 information available at the time of the last review and information newly available since the last
9 review, including most particularly that which has been critically analyzed and characterized in
10 the ISA. In chapter 3 above, attention was given to addressing specific questions on key aspects
11 of this information. To assist us in interpreting the currently available scientific evidence and
12 results of quantitative exposure/risk analyses to address the overarching question here, we draw
13 on discussions in chapter 3 above in our consideration of broader or more policy related
14 questions, posed within sections 4.2.1 and 4.2.2 below.

15 For the purposes of this draft PA, staff has drawn from EPA’s assessment and integrated
16 synthesis of the scientific evidence presented in the third draft ISA and on the quantitative
17 exposure and risk information, based on the 2007 REA (USEPA, 2007a), described in section 3.4
18 above. The evidence-based discussions presented in this chapter draw upon evidence from
19 epidemiologic studies and experimental animal studies evaluating health effects related to
20 exposures to Pb, as discussed in the ISA. The exposure/risk-based discussions have drawn from
21 the quantitative health risk analyses for Pb performed in the last Pb NAAQS review in light of
22 the currently available evidence (2007 REA, REA Planning Document). Together the evidence-
23 based and risk-based considerations inform our preliminary conclusions related to the adequacy
24 of the current Pb standard.

25 **4.2.1 Evidence-based Considerations**

26 In considering the evidence with regard to the issue of adequacy of the current standard,
27 we address several questions that build on the information summarized in chapter 3 to more
28 broadly address the extent to which the current evidence base supports the adequacy of the
29 public health protection afforded by the current primary standard. The first question addresses
30 our integrated consideration of the health effects evidence, in light of aspects described in
31 chapter 3. The second question focuses on our consideration of associated areas of uncertainty.
32 The third question then integrates our consideration of the prior two questions with a focus on
33 the standard, including each of the four elements.

- 1 • **To what extent has new information altered the scientific support for the**
2 **occurrence of health effects as a result of multimedia exposure associated with**
3 **levels of Pb occurring in the ambient air?**

4 The current evidence continues to support our conclusions from the previous review
5 regarding key aspects of the health effects evidence for Pb and the health effects of multimedia
6 exposure associated with levels of Pb occurring in ambient air in the U.S. Our conclusions in
7 this regard are based on consideration of the assessment of the currently available evidence in the
8 ISA, particularly with regard to key aspects summarized in chapter 3 of this PA, in light of the
9 assessment of the evidence in the last review as described in the 2006 CD and summarized in the
10 notice of final rulemaking (73 FR 66964). Key aspects of these conclusions are summarized
11 below.

12 As at the time of the last review, blood Pb continues to be the predominant biomarker
13 employed to assess exposure and health risk of Pb (ISA, chapters 4 and 5), as discussed in
14 section 3.1 above. This widely accepted role of blood Pb in assessing exposure and risk is
15 illustrated by its established use in programs to prevent both occupational Pb poisoning and
16 childhood Pb poisoning, with the latter program, implemented by the CDC, recently issuing
17 updated guidance on blood Pb measurement interpretation (CDC, 2012). As in the past, the
18 current evidence continues to indicate the close linkage of blood Pb levels in young children to
19 their body burden; this linkage is associated with the ongoing bone remodeling during that
20 lifestage (ISA, section 4.3.5). This tight linkage plays a role in the somewhat rapid response of
21 children's blood Pb to changes in exposure (particularly to exposure increases), which
22 contributes to its usefulness as an exposure biomarker (ISA, sections 4.2.2, 4.3.5, and 4.3.5.1).
23 Additionally, the weight of evidence documenting relationships between children's blood Pb and
24 health effects, most particularly those on the nervous and hematological systems (e.g., ISA,
25 sections 5.3 and 5.7) speaks to its usefulness in assessing health risk.

26 As in the last review, the evidence on air-to-blood relationships available today continues
27 to be comprised of studies based on an array of circumstances and population groups (of
28 different age ranges), analyzed by a variety of techniques, which together contribute to
29 appreciable variability in the associated quantitative estimates and uncertainty with regard to the
30 relationships existing in the U.S. today. Accordingly, our interpretation of this evidence base, as
31 discussed in section 3.1 above, also includes consideration of factors that may be influencing
32 various study estimates, both with regard to their usefulness for our general purpose of
33 quantitatively characterizing relationships between Pb in ambient air and air-related Pb in
34 children's blood, and with regard to their pertinence more specifically to conditions and
35 populations in the U.S. today. In so doing, we note that the current evidence, while including
36 two additional studies not available at the time of the last review, is not much changed from that

1 available in the last review. The range of estimates that can be derived from the full dataset is
2 broad and not changed by the inclusion of the newly available estimates. Further, we recognize
3 significant uncertainties regarding the air Pb to air-related blood Pb relationship for the current
4 conditions where concentrations of Pb in both ambient air and children's blood are substantially
5 lower than they have been in the past. In considering the strengths, limitations and uncertainties
6 associated with the full dataset, the currently available evidence appears to continue to support a
7 range of estimates for our purposes that is generally consistent with the range given weight in the
8 last review, 1:5 to 1:10 (ISA, section 4.7.4 and Table 4-12; 73 FR 67001-2, 67004). We
9 additionally note that the generally central estimate of 1:7 identified for this range in the last
10 review is consistent with the study involving blood Pb for pre-school children and air Pb
11 conditions near a large source of Pb to ambient air with concentrations near (and/or previously
12 above) the level of the current Pb standard (ISA, section 4.5.1; Hilts, 2003).⁵ In so noting, we
13 also recognize the general overlap of such circumstances with those represented by the evidence-
14 based, air-related IQ loss framework,⁶ for which air-to-blood ratio is a key input. In
15 characterizing the range of air-to-blood ratio estimates, we recognize uncertainty inherent in such
16 estimates as well as the variation in currently available estimates resulting from a variety of
17 factors, including differences in the populations examined, as well as in the Pb sources or
18 exposure pathways addressed in those study analyses (ISA, section 4.7.4).

19 The scientific evidence continues to recognize a broad array of health effects on multiple
20 organ systems or biological processes related to blood Pb, including Pb in blood prenatally (ISA,
21 section 2.6). The currently available evidence continues to support identification of
22 neurocognitive effects in young children as the most sensitive endpoint associated with blood Pb
23 concentrations (ISA, section 2.6.1), which as an integrated index of exposure reflect the
24 aggregate exposure to all sources of Pb through multiple pathways (inhalation and ingestion).
25 Evidence continues to indicate that neurocognitive effects in young children may not be
26 reversible and may have effects that persist into adulthood (ISA, section 2.9.6). Thus, as
27 discussed in section 3.2 above, we continue to consider the evidence of Pb effects at the low end
28 of the studied blood Pb levels (closest to those common in the U.S. today) to be strongest and of
29 greatest concern for effects on the nervous system, most particularly those on cognitive function
30 in children.

31 As in the last review, evidence on risk factors continues to support the identification of
32 young children as an important at-risk population for Pb health effects (ISA, section 6.4). The

⁵ The older study by Hayes et al (1994) during time of leaded gasoline indicated a generally similar ratio of 1:8, although the blood Pb levels in that study were much higher than those in the study by Hilts (2003). Among the studies focused on this age group, the latter study includes blood Pb levels closest to those in U.S. today.

⁶ Concentrations near air sources are higher than those at more distant sites (as described in section 2.2.2); it is near-source locations where there is the potential for concentrations at or near the current standard.

1 current evidence also continues to indicate important roles as factors that increase risk of lead-
2 related health effects for: nutritional factors, such as iron and calcium intake; elevated blood Pb
3 levels; and proximity to sources of Pb exposure, such as industrial releases or buildings with old,
4 deteriorating, leaded paint. Further, some races or ethnic groups continue to demonstrate
5 increased blood Pb levels relative to others, which may be related to these and other factors (ISA,
6 sections 6.1, 6.2 and 6.4).

7 With regard to our understanding of the relationship between exposure or blood Pb levels
8 in young children and neurocognitive effects, the evidence in this review, as in the last, does not
9 establish threshold blood Pb level for neurocognitive effects in young children (ISA, sections
10 2.9.4 and 5.3.13). The lowest blood Pb levels at which associations with neurocognitive impacts
11 have been observed in pre-school and school age children continue to range down below 5
12 $\mu\text{g}/\text{dL}$, with lowest group levels that have been associated with such effects ranging down to 2
13 $\mu\text{g}/\text{dL}$ (ISA, sections 2.6.1.1 and 5.3.16.1). Additionally, as in the last review, there is evidence
14 that the relationship of young children's blood Pb with neurocognitive impacts, such as IQ, is
15 nonlinear across a wide range of blood Pb, with greater incremental impacts at lower vs. higher
16 blood Pb levels (ISA, sections 2.9.4 and 5.3.13). Accordingly, as in the last review, we continue
17 to focus our interest on C-R relationships from study groups with blood Pb levels closest to those
18 in children in the U.S. today, which are generally lower than epidemiological study groups. The
19 currently available evidence does not identify additional C-R slopes for study groups of young
20 children (e.g., ≤ 7 years) with mean blood Pb levels below that of groups identified in the last
21 review, 2.9 – 3.8 $\mu\text{g}/\text{dL}$, as discussed in section 3.2 above (ISA, section 5.3.13). Thus, the blood
22 Pb concentration - IQ response functions or slopes identified in this review for epidemiological
23 study groups of young children with mean blood Pb levels closest to that of children in the U.S.
24 today include the same set recognized at the time of the last review (see Table 3-3 above), the
25 median of which is 1.75 points decrement per $\mu\text{g}/\text{dL}$ blood Pb (73 FR, 67003).

26 • **To what extent have important uncertainties identified in the last review been**
27 **reduced and/or have new uncertainties emerged?**

28 In our consideration of the evidence, as summarized in discussing the previous question
29 and in chapter 3 above, we have not identified any new uncertainties as emerging since the last
30 review. However, we continue to recognize important uncertainties identified in the last review
31 that remain today. Importantly, given our focus in this review, as in the last review, on
32 neurocognitive impacts associated with Pb exposure in early childhood, we recognize remaining
33 uncertainties in our understanding of the C-R relationship of neurocognitive impacts, such as IQ
34 decrements, with blood Pb level in young children, particularly across the range of blood Pb
35 levels common in the U.S. today. With regard to C-R relationships for IQ, the evidence
36 available in this review does not include studies that appreciably extend the range of blood Pb

1 levels studied beyond those available in the last review. As in the last review, the early
2 childhood (e.g., 2 to 6 or 7 years of age) blood Pb levels for which associations with IQ response
3 have been reported continue to extend at the low end of the range to study group mean blood Pb
4 levels of 2.9 to 3.8 µg/dL (e.g., 73 FR 67003, Table 3). The studies at these blood Pb levels do
5 not provide evidence of a threshold level and, as summarized in section 3.2 above, continue to
6 indicate higher C-R slopes in these groups with lower blood Pb levels than in study groups with
7 higher blood Pb levels (ISA, section 5.3.13).

8 Further, we recognize important uncertainties in our understanding of the relationship
9 between ambient air Pb concentrations and air-related Pb in children's blood. The evidence
10 newly available in this review has not reduced such key uncertainties. As in the last review, air-
11 to-blood ratios based on the available evidence continue to vary, with our conclusions based on
12 the current evidence generally consistent with the range of 1:5 to 1:10 given emphasis in the last
13 review (73 FR 67002; ISA, section 4.7.4). There continues to be uncertainty regarding the extent
14 to which this range represents the relationship between ambient air Pb and Pb in children's blood
15 (derived from the full set of air-related exposure pathways), and with regard to its reflection of
16 exposures associated with ambient air Pb levels common in the U.S. today and to circumstances
17 reflecting just meeting the current Pb standard (ISA, section 4.7.4). We note additionally the
18 significant uncertainty remaining with regard to the temporal relationships of ambient Pb levels
19 and associated exposure with occurrence of a health effect (73 FR 67005).

20 • **To what extent does newly available information support or call into question any**
21 **of the basic elements of the current Pb standard?**

22 We address this question for each of the elements of the standard in light of the health
23 effects evidence and other relevant information available in this review. As an initial matter,
24 however, we recognize the weight of the scientific evidence available in this review that
25 continues to support our focus on effects on the nervous system of young children, and
26 specifically neurocognitive decrements, as the most sensitive endpoint. Consistent with the
27 evidence available in the last review, the currently available evidence continues to indicate that a
28 standard that provides requisite public health protection against the occurrence of such effects in
29 at-risk populations would also provide the requisite public health protection against the full array
30 of health effects of Pb. Accordingly, the discussion of the elements below is framed by that
31 background.

32 ***Indicator***

33 The indicator for the current Pb standard is Pb-TSP. Exposure to Pb in all sizes of
34 particles passing through ambient air can contribute to Pb in blood and associated health effects
35 by a wide array of exposure pathways (ISA, section 4.1). These pathways include the ingestion
36 route, as well as inhalation (ISA, section 4.1), and a wide array of particle sizes play a role in

1 these pathways (ISA, section 4.1.1.1). As at the time of the last review, we continue to recognize
2 the variability of the Pb-TSP FRM in its capture of airborne Pb particles (see section 2.2.1.3.1
3 above). As in the last review, we also note that an alternative approach for collection of a
4 conceptually comparable range of particle sizes, including ultra-coarse particles, is not yet
5 available, although we take note of activities underway to remedy that situation in the future, as
6 referenced in section 2.2.1.3.1 above. Additionally, the limited available information regarding
7 relationships between Pb-TSP and Pb in other size fractions indicates appreciable variation in
8 this relationship, particularly near sources of Pb emissions where concentrations, and potential
9 exposures, are greatest. Thus, the information available in this review does not address
10 previously identified limitations and uncertainties for the current indicator. Nor does the newly
11 available information identify additional limitations or uncertainties. The evidence available in
12 this review continues to indicate the role of a range of air Pb particle sizes in contributing to Pb
13 exposure (e.g., ISA, section 4.1.1.1), that contributes to Pb in blood and associated health effects,
14 reinforcing the appropriateness of an indicator for the Pb standard that reflects a wide range of
15 airborne Pb particles.

16 ***Averaging time and form***

17 The averaging time and form of the current standard is a not-to-be-exceeded rolling
18 three-month average (CFR 50.16), derived from three monthly averages calculated in accordance
19 with the current data handling procedures (CFR, Appendix R to Part 50). As at the time of the
20 last review, evidence continues to support the importance of periods on the order of 3 months
21 and the prominent role of deposition-related exposure pathways, with uncertainty associated with
22 characterization of precise time periods associating ambient air Pb with air-related health effects.
23 Relevant factors continue to be those pertaining to the human physiological response to changes
24 in Pb exposures and those pertaining to the response of air-related Pb exposure pathways to
25 changes in airborne Pb. The newly available evidence in this review does not appreciably
26 improve our understanding of the period of time in which air Pb concentrations would lead to the
27 health effects most at issue in this review. Thus, there continue to be limitations in the evidence
28 to inform our consideration of these elements of the standard and associated uncertainty.
29 However, there is no newly available information that calls into question this element of the
30 current Pb standard.

31 ***Level***

32 The level of the current standard is 0.15 $\mu\text{g}/\text{m}^3$ (CFR 50.16). As described in section
33 4.1.1 above, this level was selected in 2008 with consideration of, among other factors, an
34 evidence-based air-related IQ loss framework, for which there are two primary inputs: air-to-
35 blood ratios and C-R functions for blood Pb – IQ response in young children. Additionally taken
36 into consideration were the uncertainties inherent in these inputs. Application of the framework

1 also entailed consideration of a magnitude of air-related IQ loss, which as further described in
2 section 4.1.1.2 above, is used in conjunction with this specific framework in light of the
3 framework context, limitations and uncertainties. Additionally, selection of a level for the
4 standard in 2008 was made in conjunction with decisions on indicator, averaging time and form.

5 As an initial matter, we consider the extent to which the evidence-based, air-related IQ
6 loss framework which informed the Administrator's decision in the last review is supported by
7 the currently available evidence and information. In so doing, we recognize the support provided
8 by the currently available evidence for the key conclusions drawn in the last review with regard
9 to health effects of greatest concern, at-risk populations, the influence of Pb in ambient air on Pb
10 in children's blood and the association between children's blood Pb and decrements in
11 neurocognitive function (e.g., IQ). We additionally note the complexity associated with
12 interpreting the scientific evidence with regard to specific levels of Pb in ambient air, given the
13 focus of the evidence on blood Pb as the key biomarker of children's aggregate exposure. The
14 need to make such interpretations in the face of the associated complexity supported use of the
15 evidence-based framework in the last review. In considering the currently available evidence for
16 the same purposes in this review, we conclude that the evidence-based framework continues to
17 provide a useful tool for consideration of the evidence with regard to the level of the standard.

18 We next turn to consideration of the primary inputs to the framework: air-to-blood ratios
19 and C-R functions for blood Pb – IQ response in young children. With regard to the former, the
20 limited newly available information assessed in the ISA, and discussed in section 3.1 above, is
21 generally consistent with the information in this area that was available at the time of the last
22 review. We additionally recognize the variability and uncertainty associated with quantitative
23 air-to-blood ratios based on this information, as also existed in the last review. As in the last
24 review, we recognize that factors contributing to the variability and uncertainty of these
25 estimates are varied, and include aspects of the study populations (e.g., age and Pb exposure
26 pathways) and the study circumstances (e.g., length of study period and variations in sources of
27 Pb exposure during the study period). We note that the full range of estimates associated with
28 the available evidence is wide, and consider it appropriate to give emphasis to estimates
29 pertaining to circumstances closest to those in the U.S. today with regard to ambient air Pb and
30 children's blood Pb concentrations, while recognizing the limitations associated with the
31 available information with regard to this emphasis. With that in mind, we consider the currently
32 available evidence to continue to support the range of estimates concluded in the last review to
33 be most appropriate for the current population of young children in the U.S., in light of the
34 multiple air-related exposure pathways by which children are exposed, in addition to inhalation
35 of ambient air, and of the levels of air and blood Pb common today. Identification of this range
36 also included consideration of the limitations associated with the available information and

1 inherent uncertainties. This range of air-to-blood ratios included 1:10 at the upper end and 1:5 at
2 the lower end. We further recognize that the limited evidence for air Pb and children's blood Pb
3 concentrations closest to those in U.S. today continues to provide support for the Administrator's
4 emphasis in the 2008 decision on the relatively central estimate of 1:7.

5 With regard to the second input to the evidence-based framework, C-R functions for the
6 relationship of young children's blood Pb with neurocognitive impacts (e.g., IQ decrements), we
7 consider several aspects of the evidence. First, as discussed in section 3.2 above, the currently
8 available information continues to provide evidence that this C-R relationship is nonlinear across
9 the range of blood Pb levels from the higher concentrations more prevalent in the past to lower
10 concentrations more common today. Thus, we continue to consider it particularly appropriate to
11 focus on the evidence from studies with blood Pb levels closest to those of today's population,
12 which in the last review included studies with study group mean blood Pb levels ranging roughly
13 from 3 to 4 $\mu\text{g}/\text{dL}$ in children aged 24 months to 7 years (Table 3-3 above). As discussed in
14 sections 3.2 above, this is also consistent with the evidence currently available for this age group
15 of young children, and the currently available evidence does not include additional C-R slopes
16 for incremental neurocognitive decrement with blood Pb levels at or below this range. In
17 considering whether this set of functions continues to be well supported by the evidence, as
18 assessed in the ISA (ISA, section 5.3.2), we note the somewhat wide range in slopes
19 encompassed by these study groups, while also noting the stability of the median. For example,
20 omission of any of the four slopes considered in the last review does not appreciably change the
21 median (e.g., the median would change from -1.75 IQ points per $\mu\text{g}/\text{dL}$ blood Pb to -1.71 or -
22 1.79). Thus, we conclude that while differing judgments might be made with regard to inclusion
23 of each of the four study groups, these estimates are generally supported by the current review of
24 the evidence in the ISA. Further, the stability of the median to modifications to this limited
25 dataset lead us to conclude that the currently available evidence continues to support
26 consideration of -1.75 IQ points per $\mu\text{g}/\text{dL}$ blood Pb as a well founded and stable estimate for
27 purposes of describing the neurocognitive impact quantitatively on this age group of U.S.
28 children.

29 In summary, in considering the evidence and information available in this review
30 pertaining to the level of the current Pb standard, we note that the evidence available in this
31 review, as summarized in the ISA, continues to support the air-related IQ loss evidence-based
32 framework, with the inputs that were used in the last review. These include estimates of air-to-
33 blood ratios ranging from 1:5 to 1:10, with a generally central estimate of 1:7. Additionally, the
34 C-R functions most relevant to blood Pb levels in U.S. children today continue to be provided by
35 the set of four analyses considered in the last review for which the median estimate is -1.75 IQ
36 points per $\mu\text{g}/\text{dL}$ Pb in young children's blood. Thus, we observe that the evidence available in

1 this review has changed little if at all with regard to the aspects given weight in the conclusion on
2 level for the new standard in the last review and would not appear to call into question any of the
3 basic elements of the standard. In so doing, we additionally recognize that the overall decision
4 on adequacy of the current standard is a public health policy judgment by the Administrator; we
5 discuss considerations that inform such judgments in section 4.3 below.

6 **4.2.2 Exposure/Risk-based Considerations**

7 Our consideration of the issue of adequacy of public health protection provided by the
8 current standard is also informed by the quantitative exposure/risk assessment completed in the
9 last review, augmented as described in section 3.4 above. We have organized the discussion that
10 follows around two questions to assist us in interpreting the results of the assessment of case
11 studies simulated to meet several different air quality conditions, including those just meeting the
12 current standard.

- 13 • **What is the level of confidence associated with estimates of air-related risk**
14 **generated for simulations just meeting the current Pb standard?**

15 As an initial matter, we recognize the significant limitations and complexity associated
16 with the risk/exposure assessment for Pb that are far beyond those associated with similar
17 assessments typically performed for other criteria pollutants. In completing the assessment we
18 were constrained by significant limitations with regard to data and tools particular to the problem
19 at hand. Further, the multimedia and persistent nature of Pb and the role of multiple exposure
20 pathways contribute significant additional complexity to the assessment as compared to other
21 assessments that focus only on the inhalation pathway. As a result, our estimates of air-related
22 exposure and risk are approximate, presented as upper and lower bounds within which we
23 consider air-related risk likely to fall. We base our description of overall confidence in this
24 characterization of air-related risk on our consideration of the overall design of the analysis (as
25 described in section 3.4 above), the degree to which key sources of variability are reflected in the
26 design of the analysis, and our characterization of key sources of uncertainty.

27 In considering the degree to which key sources of variability (discussed in section 3.4.6
28 above) are reflected in the design of the analysis, we note the following aspects addressed by the
29 risk assessment.

- 30 • *Variation in distributions of potential urban residential exposure and risk across U.S.*
31 *urban residential areas.* This is addressed by the inclusion of three different (location-
32 specific) urban study areas that reflect a diverse set of urban areas in the U.S.
- 33 • *Representation of a more highly-exposed subset of urban residents potentially exposed at*
34 *the level of the standard.* This is addressed by the inclusion of the generalized (local)
35 urban study area.

- 1 • *Variation in residential exposure to ambient air Pb within an urban area.* This is
2 addressed through the partitioning of the location-specific study areas into exposure
3 zones to provide some representation of spatial gradients in ambient air Pb and their
4 interaction with population distribution and demographics. This was done in a somewhat
5 more precise manner in the primary Pb smelter case study, which relied on dispersion
6 modeling to describe gradients, as compared with the manual assignment of gradients
7 related to air concentration differences among monitors in an area.
- 8 • *Inter-individual variability in blood Pb levels.* This is addressed though the use of
9 empirically-derived GSDs to develop blood Pb distribution for the child population in
10 each exposure zone, with GSDs selected particular to each case study populations.
- 11 • *Inter-individual variability in IQ response to blood Pb.* This is addressed through the use
12 of C-R functions for IQ loss based on a pooled analysis reflecting studies of diverse
13 populations.

14 We also considered key sources of uncertainty (discussed in section 3.4.7 above), in
15 particular the overarching area concerning the precision of the air-related risk estimates, as
16 mentioned above. Associated sources of uncertainty include our inability to simulate changes in
17 air-related Pb as a function of changes in ambient air Pb in exposure pathways other than those
18 involving inhalation of ambient air and ingestion of indoor dust. This contributes to the positive
19 bias of the upper bound for the air-related risk estimates. We additionally recognize the
20 significant uncertainty associated with estimating upper percentiles of the distribution of *air-*
21 *related* blood Pb concentration estimates (and associated IQ loss estimates) due to limitations in
22 available information. Lastly, we recognize the uncertainty associated with application of the C-
23 R function at the lower blood Pb levels in the distribution; this relates to the limited
24 representation of blood Pb levels of this magnitude in the dataset from which the C-R function is
25 derived.

26 In the quantitative risk information available in this review, we have air-related risk
27 estimates for simulations just meeting the current standard from one of the location-specific
28 urban case studies (Chicago) and from the generalized (local) urban case study. With regard to
29 the latter, we note its simplified design that does not include multiple exposure zones, thus
30 reducing the dimensions simulated. We have a reasonable degree of confidence in aspects of the
31 generalized (local) urban case study for the specific situation we consider it to represent (i.e., a
32 temporal pattern of air Pb concentrations that just meets the level of the standard), and when the
33 associated estimates are characterized as approximate, within upper and lower bounds (as
34 described in section 3.4.4 above), while also recognizing considerable associated uncertainty.

1 • **To what extent are the air-related risks remaining upon just meeting the current**
2 **Pb standard important from a public health perspective?**

3 In considering public health importance of estimated air-related risks, we consider here
4 the nature and magnitude of such estimated risks (and attendant uncertainties), including such
5 impacts on the affected population, and we additionally consider the size of the affected
6 population. Based on the evidence available in the last review and consistent with that available
7 today, the quantitative risk estimates developed in the 2007 REA and augmented slightly in this
8 review are for decrements in IQ, an established indicator of neurocognitive function. In
9 considering these estimates, we recognize that although some neurocognitive effects may be
10 transient, some effects may persist into adulthood, affecting success later in life (ISA, sections
11 2.9.5 and 5.3.15). We additionally recognize the potential population impacts of small changes
12 in population mean values of metrics such as IQ, presuming a uniform manifestation of lead-
13 related decrement across the range of population IQ (ISA, section 2.9.1), as noted in section 3.3
14 above.

15 Exposures and risks associated with air-related Pb under several different air quality
16 conditions were estimated in the 2007 REA. As summarized in section 3.4 above, limitations in
17 our modeling tools and data affected our ability to develop precise estimates for air-related
18 exposure pathways and contributed uncertainties. The results are approximate estimates which
19 we describe through the use of rough upper and lower bounds within which we estimate air-
20 related risk to fall. We have recognized a number of uncertainties in the underlying risk
21 estimates from the 2007 REA and in the interpolation approach employed in the new analyses
22 for this review. We have characterized the magnitude of air-related risk associated with the
23 current standard with a focus on median estimates, for which we have appreciably greater
24 confidence than estimates for outer ends of risk distribution (see section 3.4.7) and on risks
25 derived using the C-R function in which we have greatest confidence (see sections 3.4.3.3.1 and
26 3.4.7). These risk estimates include estimates from the last review for one of the location-
27 specific urban study area populations as well as estimates newly derived in this review based on
28 interpolation from 2007 REA results for the generalized (local) urban case study, which is
29 recognized to reflect a generalized high end of air-related exposure for localized populations.
30 Taken together, these estimates indicate air-related risk associated with just meeting the current
31 standard of a magnitude generally within the bounds of roughly 1 and 3 points IQ loss.

32 In considering the importance of such risk from a public health perspective, we also
33 consider the size of at-risk populations represented by the REA case studies. As discussed in
34 section 3.4 above, the generalized (local) urban case study is considered to represent a localized
35 urban population exposed near the level of the standard, such as a very small, compact
36 neighborhood near a source contributing to air Pb concentrations just meeting the standard. This

1 case study provides representation in the risk assessment for such small populations at the upper
2 end of the gradient in ambient air concentrations expected to occur near sources; thus estimates
3 for this case study reflect exposures nearest the standard being evaluated. While we do not have
4 precise estimates of the number of young children living in such areas of the U.S. today, we have
5 information that informs our understanding of their magnitude. For example, as discussed in
6 section 3.3 above (Table 3-3), we estimate there to be approximately 2,400 children, aged 5
7 years and younger, residing within 0.5 km of a monitor with air Pb concentrations above the
8 current standard. We further observe several additional monitors with maximum 3-month Pb
9 concentrations that fall below but within 10% of the current standard level (as noted in section
10 3.3 above); an estimated 265 young children reside near these monitors. Thus, together we
11 estimate some 2700 children, aged 5 years and younger, living in localized areas with elevated
12 air Pb concentrations that are above or near the current standard. Based on the 2010 census
13 estimates of approximately 24.3 million children in the U.S. aged five years or younger, this
14 represents approximately one hundredth of one percent of this age group in the U.S.⁷ This
15 indicates the size of the population of young children of this age living in areas in close
16 proximity to areas where air Pb concentrations may be above or near the current standard to be
17 approximately 2700, which is approximately a hundredth of one percent of the full population of
18 correspondingly aged children.⁸

19 In summary, we recognize substantial uncertainty inherent in the REA estimates of air-
20 related risk associated with conditions just meeting the current standard, which we characterize
21 as approximate and falling somewhat above the rough lower bound of 1 and below the rough
22 upper bound of 3 IQ points.⁹ This approximate estimate of risk for children living in such areas
23 is generally overlapping with and consistent with the evidence-based air-related IQ loss estimates
24 summarized in section 4.2.1 above. With regard to the the importance of the estimated risks
25 from a public health perspective, we note, based on the currently available monitoring and
26 census data, the estimated size of the affected population to be approximately 2700 young
27 children, representing slightly more than one one hundredth of a percent of the full population of

⁷ While these estimates pertain to the age group of children aged 5 years and younger, we additionally note that a focus on an alternative age range, such as inclusive of slightly older children (e.g., through age 7), while increasing the number for children living in such locations, would not be expected to appreciably change the percentage of the full U.S. age group that the subset represents.

⁸ This estimate includes children in areas where recent information indicates the standard is exceeded because areas not in attainment with the standard are required to attain the standard as expeditiously as practicable, but no later than five years after designation. Accordingly, these areas are for present purposes treated as areas with air Pb concentrations just meeting the current standard and are included for purposes of identifying the size of the at-risk population residing in areas likely to have air Pb concentrations near the current standard.

⁹ We note that the value of the upper bound is influenced by risk associated with exposure pathways that were not varied with alternative standard levels, a modeling limitation with the potential to contribute to overestimation of the upper bound with air quality scenarios involving air Pb levels below current conditions for the study area (see sections 3.4.4 and 3.4.7 above).

1 similarly aged children. In considering the exposure/risk estimates during the last review, the
2 Administrator took note of the important uncertainties and limitations associated with these
3 assessments and, while placing less weight on the assessment estimates, he recognized the
4 quantitative risk estimates to be “roughly consistent with and generally supportive” of those
5 estimated by the evidence-based framework. In our consideration of the risk estimates
6 considered in the last review, as well as the estimates for the current standard that have been
7 newly developed in this review for the generalized (local) urban case study (see section
8 3.4.3.3.2), we agree with the previous characterization. As would be expected by the use of
9 interpolation, the newly derived estimates are consistent with the estimates for similar air quality
10 scenarios that were available in the last review.

11 **4.3 PRELIMINARY STAFF CONCLUSIONS ON THE PRIMARY STANDARD**

12 This section describes preliminary staff conclusions regarding adequacy of the current
13 primary Pb standard. These conclusions are based on considerations described above and in the
14 discussion below regarding the currently available scientific evidence summarized in the third
15 draft ISA and prior CDs and the risk and exposure information drawn from the 2007 REA. The
16 final PA will draw upon the final ISA, developed after CASAC review and public comment on
17 the third draft ISA. Further, staff conclusions presented in the final PA will take into account
18 advice from CASAC and public comment on the draft PA and the preliminary conclusions.

19 Taking into consideration the discussions responding to specific questions above in this
20 and the prior chapter, this section addresses the following overarching policy question.

- 21 • **Does the currently available scientific evidence- and exposure/risk-based**
22 **information, as reflected in the ISA and REA, support or call into question the**
23 **adequacy of the protection afforded by the current Pb standard?**

24 Our response to this question takes into consideration the discussions responding to the
25 specific policy-relevant questions in prior sections of this document (see sections 3.1-3.4, 4.2.1,
26 and 4.2.2). In so doing, we focus first on consideration of the evidence, including that newly
27 available in this review, and the extent to which it alters key conclusions supporting the current
28 standard. We then turn to consideration of the quantitative risk estimates drawn from the 2007
29 REA, and associated limitations and uncertainties. We additionally consider the public health
30 policy judgments and judgments about the uncertainties inherent in the scientific evidence and
31 quantitative analyses that are inherent in decisions on the adequacy of the current primary Pb
32 standard.

33 We first recognize the complexity involved in considering the adequacy of protection in
34 the case of the primary Pb standard, which differs substantially from that involved in
35 consideration of the NAAQS for other pollutants. Unlike the case for Pb, the other pollutants for

1 which NAAQS are set involve only inhalation exposure pathways, a relatively simpler context.¹⁰
2 In addition, generally an important component of the scientific evidence base considered in
3 reviewing the adequacy of NAAQS for other pollutants is the availability of studies that have
4 investigated associations between current concentrations of the pollutant in ambient air
5 (including in circumstances where the current standard is met) and the occurrence of health
6 effects judged plausibly related to ambient air exposure to the NAAQS pollutant. While such
7 studies, targeting locations near air Pb sources such as smelters, were available at the time the Pb
8 NAAQS was initially set in 1978, and to a much more limited degree at the time of the last
9 review, such studies of health effects under air quality conditions near those reflecting the
10 current standard are not available in this review. Rather, the evidence base that supports our
11 conclusions in this review includes most prominently epidemiological studies reporting
12 associations of blood Pb levels in U.S. populations, including the particularly at-risk population
13 of young children with health effects judged plausibly related to Pb exposures. Support for our
14 conclusions regarding the plausibility for ambient air Pb to play a role in such findings derives,
15 in part, from studies linking Pb in ambient air with the occurrence of health effects. However,
16 such studies (dating from the past or from other countries) involve ambient air Pb concentrations
17 many times greater than those that would meet the current standard. Thus, in considering the
18 adequacy of the current Pb standard, rather than considering studies that have investigated
19 current concentrations of Pb in ambient air (including in locations where the current standard is
20 met) and the occurrence of health effects, we primarily consider the evidence for, and risk
21 estimated from, models based upon key relationships, such as those between ambient air Pb, Pb
22 exposure, blood Pb and health effects. This evidence, with its associated limitations and
23 uncertainties, contributes to our conclusions regarding a relationship between ambient air Pb
24 conditions under the current standard and health effects.

25 In considering the currently available evidence, staff gives great weight to the long-
26 standing body of evidence on the health effects of Pb, augmented in some aspects since the last
27 review, which continues to support identification of neurocognitive effects in young children as
28 the most sensitive endpoint associated with Pb exposure, as discussed in sections 3.2 and 3.3
29 above. The evidence continues to indicate that a standard that provides protection from
30 neurocognitive effects in young children additionally provides protection for other health effects
31 of Pb, such as cardiovascular effects later in life. Application of the evidence-based, air-related
32 IQ loss framework, developed in the last review, continues to provide a useful approach for

¹⁰ As described in sections 1.3 and 3.1 above, exposure to Pb from ambient air, unlike exposures for other NAAQS pollutants, involves both ingestion and inhalation exposure pathways. As an additional complication, other (nonair) sources of Pb also contribute to these pathways, particularly to ingestion exposure pathways. In the case of Pb, the internal biomarker, blood Pb, is an indicator of exposure across different pathways and routes (as discussed in section 3.1 above).

1 considering and integrating the evidence on relationships between Pb in ambient air and Pb in
2 children's blood and risks of neurocognitive effects (IQ loss). The currently available evidence
3 base, while somewhat expanded since the last review, is not appreciably expanded or supportive
4 of appreciably different conclusions with regard to air-to-blood ratios (section 3.1 above) or C-R
5 functions for neurocognitive decrements (section 3.2 above) in young children.

6 As in the last review, uncertainties remain in our understanding of important aspects of
7 ambient air Pb exposure and associated health effects. For example, important uncertainties
8 remain, both with regard to air-to-blood ratios that reflect the relationship between
9 concentrations of Pb in ambient air and air-related Pb in children's blood, and to estimates of the
10 slope of the C-R function for neurocognitive impacts (IQ loss) at lower blood Pb levels. With
11 regard to the former, we note particularly the limitations associated with the available studies and
12 gaps in the evidence base with regard to studies that have investigated such quantitative
13 relationships under conditions pertaining to the current standard (e.g., in localized areas near air
14 Pb sources where the standard is met in the U.S. today). Further, in considering our reliance on
15 the evidence in performing quantitative modeling of exposure and risk we additionally note
16 important uncertainties associated with relationships between ambient air Pb and outdoor
17 soil/dust Pb and indoor dust Pb that particularly affect our quantitative estimates of air-related
18 risk under conditions of lower ambient air Pb levels and lower blood Pb levels (73 FR 66981).
19 These critical exposure pathways are also inherent in the evidence-based air-related IQ loss
20 framework, encompassed within the estimates of air-to-blood ratios. Thus, we recognize
21 uncertainties related to our understanding of these and other processes that also contribute
22 uncertainty to our application of the evidence-based framework. We consider this uncertainty to
23 be increased with application of the framework for levels below the current standard given the
24 weaker linkage with existing evidence, as noted below.

25 We additionally take note of the role of public health policy judgments in the
26 Administrator's setting of the current standard in 2008, as summarized in section 4.1.1.2 above.
27 Such judgments are inherent in setting the standard, and particularly in using the evidence-based
28 framework to inform the decision on level for the standard, and in considering the exposure/risk
29 information. We recognize that public health policy judgments always play an important role in
30 each NAAQS review for each pollutant. One type of public health policy judgments focuses on
31 how to consider the nature and magnitude of the array of uncertainties that are inherent in the
32 scientific evidence and analyses. These judgments are traditionally made with a recognition that
33 our understanding of the relationships between the presence of a pollutant in ambient air and
34 associated health effects is based on a broad body of information encompassing not only more
35 established aspects of the evidence but also aspects in which there may be substantial
36 uncertainty. In the case of the Pb NAAQS review, we recognize increased uncertainty in

1 characterizing the IQ response to blood Pb levels below those represented in the evidence base.
2 We also recognize increased uncertainty in projecting the magnitude of blood Pb response to
3 ambient air Pb concentrations at and below the level of the current standard. We recognize this
4 increased uncertainty particularly in light of the multiple factors that play a role in such a
5 projection (e.g., meteorology, atmospheric dispersion and deposition, human physiology and
6 behavior), for each of which we recognize attendant uncertainties. Collectively, these aspects of
7 the evidence and associated uncertainties contribute to a recognition that for Pb, as for other
8 pollutants, the available health effects evidence generally reflects a continuum, consisting of
9 ambient levels at which scientists generally agree that health effects are likely to occur, through
10 lower levels at which the likelihood and magnitude of the response become increasingly
11 uncertain.

12 Reviews may also require judgments as to the point at which health effects become
13 important from a public health perspective. In the case of Pb, such a judgment is explicitly made
14 in considering the public health significance of one to two points IQ loss in at-risk populations,
15 such as young children. This type of judgment also includes consideration of the IQ loss
16 estimates yielded by the air-related IQ loss evidence-based framework for specific combinations
17 of standard level, air-to-blood ratio and C-R function. With regard to public health significance
18 of one to two points IQ loss in young children, staff gives weight to the comments of CASAC
19 and some public commenters in the last review which recognized such a magnitude of IQ loss to
20 be of public health significance and recommended that a very high percentage of the population
21 be protected from such a magnitude of IQ loss (73 FR 67000).¹¹ With this objective in mind, we
22 consider the extent to which the air-related IQ loss evidence-based framework informs
23 consideration of standards that might be concluded to provide such a level of protection. In so
24 doing, we first recognize that the IQ loss estimates produced with the evidence-based framework
25 do not correspond to a specific quantitative public health policy goal for air-related IQ loss that
26 would be acceptable or unacceptable for the entire population of children in the U.S. Rather, the
27 conceptual context for the evidence-based framework is that it provides estimates for the mean
28 air-related IQ loss of a subset of the population of U.S. children (i.e., the subset living in close
29 proximity to air Pb sources that contributed to elevated air Pb concentrations that equal the
30 current level of the standard). This is the subset expected to experience air-related Pb exposures
31 at the high end of the national distribution of such exposures. The associated mean IQ loss
32 estimate is the average for this highly exposed subset and is not the average air-related IQ loss

¹¹ Our focus on IQ, as noted in section 3.3 above, reflects recognition of IQ being a well established, widely recognized and rigorously standardized measure of neurocognitive function, as well as a global measure reflecting the integration of numerous processes (ISA, section 5.3.2; 2006 CD, sections 6.2.2 and 8.4.2). Use of IQ in this framework is thus considered to appropriately also reflect neurocognitive effects more generally.

1 projected for the entire U.S. population of children. Further, we recognize uncertainties
2 associated with those estimates, which increase with estimates associated with successively
3 lower standard levels.

4 For the current standard level of $0.15 \mu\text{g}/\text{m}^3$, an air-to-blood ratio estimate of $7 \mu\text{g}/\text{dL}$ per
5 $\mu\text{g}/\text{m}^3$ (which we note as reasonably representative of the range supported by the evidence) and a
6 C-R slope of -1.75 IQ points per $\mu\text{g}/\text{dL}$, the IQ loss estimate using the evidence-based
7 framework is 1.8 points.¹² As noted above, this value is considered to be an estimate, with
8 attendant uncertainties, of mean air-related IQ loss of a subset of the population of U.S. children
9 in the high end of the exposure distribution for air-related Pb.¹³ As noted in section 4.2.2 above,
10 our current information on numbers of young children living near monitors above or within 10%
11 of the current Pb standard indicates the size of this population subset to be on the order of 2700
12 children aged 5 years or younger, which would correspond to approximately one hundredth of
13 one percent of the U.S. population of this age children (estimated at approximately 24 million in
14 2010 census). A primary objective of the monitoring network for Pb is to identify and monitor
15 sites of maximum concentration in areas anticipated to be at risk of exceeding the NAAQS.
16 While we acknowledge the possibility that the monitoring data thus far available may not
17 identify every occurrence of elevated Pb concentrations, the size of such a population subset can
18 still be concluded to fall well below one tenth of one percent of the full population of children
19 aged 5 years or younger in the U.S. today. Thus, we conclude that the current evidence, as
20 considered within the conceptual and quantitative context of the evidence-based framework, and
21 current air monitoring information indicates that the current standard would be expected to
22 achieve the public health policy goal recommended by CASAC in the last Pb NAAQS review
23 that IQ loss on the order of one to two IQ points be “prevented in all but a small percentile of the
24 population” (73 FR 67000).

25 In drawing conclusions from application of the evidence based framework with regard to
26 adequacy of the current standard, we further recognize the degree to which IQ loss estimates
27 drawn from the air-related IQ loss evidence-based framework reflect mean blood Pb levels
28 below those represented in the currently available evidence for young children. For example, in
29 the case of the current standard level of $0.15 \mu\text{g}/\text{m}^3$, multiplication by the air-to-blood ratio of 7

¹² Using an air-to-blood ratio estimate of $8 \mu\text{g}/\text{dL}$ per $\mu\text{g}/\text{m}^3$ also yields an IQ loss estimate of approximately 2 IQ points.

¹³ In giving weight to consideration of the evidence within the context of the air-related IQ loss evidence-based framework, we note that the air-related IQ loss estimated by the framework is for the mean of the population subset described above. Given the lack of data on the distribution of the air-related portion of blood Pb and on the extent to which distributions of air-related blood Pb levels might differ or correlate with total blood Pb that may be more greatly influenced by other (nonair) Pb exposure pathways, estimates are not available that would correspond to upper percentiles of the IQ loss distribution for this population subset. Any such estimates would have substantial associated uncertainty.

1 yields a mean air-related blood Pb level of 1.05 $\mu\text{g}/\text{dL}$, which is half the level of the lowest blood
2 Pb subgroup of pre-school children in which neurocognitive effects have been observed (Table
3 3-2 above; Miranda et al., 2009) and well below the means of subgroups for which continuous C-
4 R functions have been estimated (Table 3-3 above).¹⁴ Such an extension below the lowest
5 studied levels may be viewed as appropriate given the lack of identified blood Pb level threshold
6 in the current evidence base for neurocognitive effects and the need for the NAAQS to provide a
7 margin of safety.¹⁵ We note, however, that the framework IQ loss estimates for still lower
8 potential standard levels represent still greater extrapolations from the current evidence base with
9 corresponding increased uncertainty.

10 In recognition of the role of public health policy judgments in drawing conclusions as to
11 adequacy of the Pb NAAQS, we consider the availability of new information or new commonly
12 accepted guidelines or criteria within the public health community with regard to the public
13 health significance of specific IQ decrements in exposed, at-risk populations that might inform
14 public health policy judgments on the appropriate degree of public health protection that should
15 be afforded to protect against risk of such neurocognitive effects in at-risk populations,¹⁶ such as
16 children living near air Pb sources. As an initial matter, we note that no such new information,
17 guidelines or criteria are described in the ISA. In further considering the occurrence of any new
18 actions by public health agencies that might indicate the availability of new information,
19 guidelines or criteria for interpreting public health significance of such effects, we note that the
20 CDC has revised the blood Pb level used to prioritize young children for whom they recommend

¹⁴ We recognize that children also have Pb in their blood derived from other (nonair) sources. The evidence-based air-related IQ loss framework is used, however, to estimate IQ loss attributable to air-related Pb because the NAAQS is intended to protect against risks from ambient air-related Pb. While children also have Pb in their blood derived from other (nonair) sources, the evidence indicates that the risk per increment of blood Pb is greater for children with the lowest blood Pb levels, as noted in section 3.2 above. Thus, the focus on estimating IQ loss attributable solely to air-related lead (i.e., assuming the presence of no other blood Pb) is a conservative approach, the use of which contributes to the margin of safety provided by the standard.

¹⁵ As noted in section 1.2.1 above, the requirement that primary standards include an adequate margin of safety was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting. It was also intended to provide a reasonable degree of protection against hazards that research has not yet identified. Both kinds of uncertainties are components of the risk associated with pollution at levels below those at which human health effects can be said to occur with reasonable scientific certainty. Thus, in selecting primary standards that includes an adequate margin of safety, the Administrator is seeking not only to prevent pollutant levels that have been demonstrated to be harmful but also to prevent lower pollutant levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree. The CAA does not require that primary standards be set at a zero-risk level or at background concentration levels, but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety.

¹⁶The term at-risk populations is used here, rather than the phrase “sensitive populations” used in the last review. In using the term at-risk, we intend the same meaning as has traditionally been intended for the term “sensitive” consistent with discussion in section 1.2.1 above.

1 particular follow-up health protective actions, as summarized in section 3.1 above.¹⁷ The CDC
2 decision, while emphasizing the critical importance of primary prevention of Pb exposure,
3 provides no new guidelines or criteria with regard to the significance of specific IQ decrements
4 or judgments on appropriate public health protection from risk of neurocognitive effects, and
5 their consideration of the evidence base at the time of their decision does not substantively differ
6 from that presented in the current draft ISA (CDC, 2012). Thus, we are aware of no new
7 information or new commonly accepted guidelines or criteria within the public health
8 community for interpreting public health significance of neurocognitive effects in the context of
9 a decision on adequacy of the current Pb standard.

10 With respect to exposure/risk-based considerations, we recognize the complexity of the
11 REA modeling analyses and the associated limitations and uncertainties. We additionally note
12 the differences among the case studies included in the REA and the extent to which they inform
13 our understanding of different aspects of the risk associated with air-related Pb in the U.S. For
14 example, the location-specific case studies indicate the distribution of population risk in urban
15 areas with differing types of Pb sources and gradients in air Pb concentrations as well as in
16 population density, while the generalized urban (local) case study indicates the magnitude of air-
17 related risk associated with those specific localized circumstances where air concentrations just
18 meet the Pb standard (regardless of source type). We agree with conclusions drawn in the 2008
19 review that the quantitative risk estimates, with a focus on those for the generalized (local) urban
20 case study, are “roughly consistent with and generally supportive” of estimates from the
21 evidence-based air-related IQ loss framework (73 FR 67006). We further take note of the
22 increasing uncertainty recognized for air quality scenarios involving air Pb concentrations
23 increasingly below the current conditions for each case study, recognizing that such uncertainty
24 is due in part to modeling limitations deriving from uncertainty regarding relationships between
25 ambient air Pb and outdoor soil/dust Pb and indoor dust Pb (as noted in section 3.4 above).

26 Based on the above considerations, we reach the preliminary conclusion that the current
27 body of evidence, in combination with the exposure/risk information, supports a primary
28 standard as protective as the current standard. Further consideration of the evidence and
29 exposure/risk information available in this review and its attendant uncertainties and limitations,
30 as well as consideration of the availability of other information that might also inform public
31 health policy judgments by the Administrator, leads us to reach the additional preliminary
32 conclusion that it is appropriate to consider retaining the current standard without revision.
33 These preliminary conclusions are based on consideration of the health effects evidence,
34 including consideration of this evidence in the context of the evidence-based, air-related IQ loss

¹⁷ Uses identified for the CDC reference level include is the identification of “ high-risk childhood populations and geographic areas most in need of primary prevention” (CDC, 2012).

1 framework, and in combination with the exposure/risk information (chapter 3 and sections 4.2.1
2 and 4.2.2 above) and the uncertainties attendant with both, recognizing the complexities and
3 limitations in the evidence base in reaching conclusions regarding the magnitude of risk
4 associated with the current standard, as well as the increasing uncertainty of risk estimates for
5 lower air Pb levels. Based on these considerations, these preliminary conclusions also recognize
6 what may be considered reasonable judgments on the public health implications of the blood Pb
7 levels and risk estimated for air-related Pb under the current standard, including the public health
8 significance of the Pb effects being considered, as well as aspects of the use of the evidence-
9 based framework that may be considered to contribute to the margin of safety.

10 In reaching these preliminary conclusions, we additionally note that different public
11 health policy judgments could lead to different conclusions regarding the extent to which the
12 current standard provides protection of public health with an adequate margin of safety. Such
13 public health policy judgments include those related to the appropriate degree of public health
14 protection that should be afforded to protect against risk of neurocognitive effects in at-risk
15 populations, such as IQ loss in young children, as well as with regard to the appropriate weight
16 to be given to differing aspects of the evidence and exposure/risk information, and how to
17 consider their associated uncertainties. For example, different judgments might give greater
18 weight to more uncertain aspects of the evidence or reflect a differing view with regard to margin
19 of safety. As noted in section 4.1 above, in establishing primary standards under the Act that, in
20 the Administrator's judgment, are requisite to protect public health with an adequate margin of
21 safety, the Administrator seeks to establish standards that are neither more nor less stringent than
22 necessary for this purpose. The Act does not require that primary standards be set at a zero-risk
23 level, but rather at a level that avoids unacceptable risks to public health, even if the risk is not
24 precisely identified as to nature or degree. The requirement that primary standards provide an
25 adequate margin of safety was intended to address uncertainties associated with inconclusive
26 scientific and technical information available at the time of standard setting. It was also intended
27 to provide a reasonable degree of protection from hazards that research has not yet identified.

28 In this context, we recognize that the uncertainties and limitations associated with the
29 many aspects of the estimated relationships between air Pb concentrations and blood Pb levels
30 and associated health effects are amplified with consideration of increasingly lower air
31 concentrations. We believe the current evidence supports the conclusion that the current
32 standard is requisite to protect public health with an adequate margin of safety. In staff's view,
33 based on the current evidence there is appreciable uncertainty associated with drawing
34 conclusions regarding whether there would be reductions in risk to public health from alternative
35 lower levels as compared to the level of the current standard. Thus, we conclude that the basis
36 for any consideration of alternative lower standard levels would reflect different public health

1 policy judgments as to the appropriate approach for weighing uncertainties in the evidence and
2 for providing requisite protection of public health with an adequate margin of safety.

3 In summary, the newly available health effects evidence, critically assessed in the ISA as
4 part of the full body of evidence, reaffirms conclusions on the broad array of effects recognized
5 for Pb in the last review. Further, staff observes the general consistency of the current evidence
6 with the evidence available in the last review with regard to key aspects on which the current
7 standard is based, including those particular to the evidence-based framework developed in the
8 last review. Staff additionally observes that quantitative risk estimates associated with the
9 current standard, based on the risk assessment performed in the last review, indicate a level of
10 risk generally consistent with conclusions drawn from the evidence using the evidence-based
11 framework. Staff additionally recognizes the limitations and uncertainties associated with the
12 currently available information. These considerations provide the basis for the preliminary staff
13 conclusion that consideration should be given to retaining the current standard, without revision.
14 Accordingly, and in light of staff preliminary conclusions that it is appropriate to consider the
15 current standard to be adequate, we have not identified any potential alternative standards for
16 consideration in this review.

17 **4.4 KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH AND DATA** 18 **COLLECTION**

19 In this section, we highlight key uncertainties associated with reviewing and establishing
20 NAAQS for Pb. Such key uncertainties and areas for future health-related research, model
21 development, and data gathering are outlined below. In some cases, research in these areas can
22 go beyond aiding standard setting to aiding in the development of more efficient and effective
23 control strategies. We note, however, that a full set of research recommendations to meet
24 standards implementation and strategy development needs is beyond the scope of this discussion.
25 Rather, listed below are key uncertainties and research questions and data gaps that have been
26 thus far highlighted in this review of the health-based primary standard.

- 27 • A critical aspect of our consideration of the evidence and the quantitative risk assessment
28 in this review is our understanding of the C-R relationship between blood Pb levels in
29 young children and neurodevelopmental effects, specifically IQ decrement. Additional
30 epidemiological research involving substantially-sized populations with mean blood Pb
31 levels closer to those common in today's population of young children, particularly those
32 less affected by higher Pb exposures earlier in childhood, would help to reduce
33 uncertainty in our estimates of IQ decrement associated with these lower blood Pb levels,
34 and accordingly, in characterizing Pb health effects.
- 35
- 36 • There remains uncertainty in the evidence base with regard to the exposure circumstances
37 (timing, duration, magnitude and frequency) eliciting effects in older children and adults.

1 Effects of particular focus include effects on the nervous system later in life,
2 cardiovascular function, and delayed onset of puberty.

- 3 • Alzheimer's-like pathology has been reported in aged laboratory animals (non-human
4 primates and rodents) exposed to Pb early in life, however uncertainty remains with
5 regard to relationships of such pathology with altered function/behavior.
- 6 • Epidemiologic studies indicate detrimental effects of Pb on sperm production and quality,
7 often in occupational cohorts. Uncertainty remains regarding these effects in otherwise
8 healthy cohorts without occupational Pb exposure or other underlying medical
9 conditions.
- 10 • Quantitative estimation of blood Pb levels in children and other at-risk subgroups in
11 response to various exposure circumstances, including air-related exposure pathways,
12 would benefit from research in a number of areas including:
 - 13 ○ Factors affecting relationships between Pb in ambient air and Pb in blood;
 - 14 ○ Temporal aspects of changes in blood Pb levels associated with changes in
15 ambient air Pb;
 - 16 ○ Interindividual variability in blood Pb levels and methods for characterizing
17 interindividual variability, including consideration of both empirical and
18 mechanistic methods;
 - 19 ○ Apportionment of blood Pb levels with regard to exposure pathway contributions,
20 particular distinctions pertinent to policy-relevant exposures and background
21 sources; and
 - 22 ○ Blood Pb model performance evaluations, with emphasis on applications
23 pertaining to blood Pb response to ambient air-related pathways and responses to
24 changes in exposures for those pathways.
- 25 • An important aspect of exposure and risk posed by ambient air Pb is the contribution of
26 ambient air Pb to indoor dust Pb. Research on the relationship between Pb in these two
27 media, both generally, as well as in specific environments, and also with regard to aspects
28 associated with mechanistic modeling (e.g., air exchange rates, home cleaning frequency
29 and efficiency) would reduce uncertainty in models and methods applied in consideration
30 of this pathway in future reviews.
- 31 • An understanding of the spatial gradient of ambient air Pb concentrations, and associated
32 particle sizes, in urban residential areas, as well as near Pb sources, is an important aspect
33 to our implementation of the NAAQS for Pb and a key element in assessing exposure and
34 risk. Additional research in this area is needed.
 - 35 ○ Current limitations in this area additionally contribute uncertainty to
36 characterization of ambient air Pb levels in the risk assessment and associated
37 exposure and risk estimates. Research in the area of characterizing spatial
38 variation in air Pb concentrations in different environments and related to different
39 air sources would help to reduce this uncertainty. Of particular interest is the
40 potential for systematic trends in the relationship between ambient air Pb
41 concentrations and distribution of urban residential populations. Examples of

1 locations of interest include neighborhoods downwind of airports with substantial
2 leaded aviation gasoline usage or in the vicinity of older roads with substantial
3 historical use of leaded gasoline, as well as inner city neighborhoods, both those in
4 which substantial reconstruction has occurred and more static areas.

- 5 • An important aspect to this review is the relationship between ambient air Pb and outdoor
6 dust and surface soil Pb concentrations, including the temporal dynamics of that
7 relationship and variation in that for different environments. Research to improve our
8 understanding of these areas would contribute to reducing associated uncertainty with
9 regard to characterization of the relationship between air and soil Pb and the impact of
10 changes in air Pb on outdoor soil Pb levels over time.

1 **4.5 REFERENCES**

2 Bellinger, D. C. and Needleman, H. L. (2003) Intellectual impairment and blood lead levels [letter]. N. Engl. J. Med.
3 349: 500.

4 Canfield, R. L.; Henderson, C. R., Jr.; Cory-Slechta, D. A.; Cox, C.; Jusko, T. A.; Lanphear, B. P. (2003)
5 Intellectual impairment in children with blood lead concentrations below 10 µg per deciliter. N. Engl. J.
6 Med. 348: 1517-1526.

7 Centers for Disease Control and Prevention (2012) CDC Response to Advisory Committee on Childhood Lead
8 Poisoning Prevention Recommendations in “Low Level Lead Exposure Harms Children: A Renewed Call
9 of Primary Prevention”. Atlanta, GA: U.S. Department of Health and Human Services, Public Health
10 Service. June 7.

11 Henderson, R. (2007a) Letter from Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory Committee, to
12 Administrator Stephen L. Johnson. Re: Clean Air Scientific Advisory Committee’s (CASAC) Review of
13 the 1st Draft Lead Staff Paper and Draft Lead Exposure and Risk Assessments. March 27, 2007.

14 Henderson, R. (2007b) Letter from Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory Committee, to
15 Administrator Stephen L. Johnson. Re: Clean Air Scientific Advisory Committee’s (CASAC) Review of
16 the 2nd Draft Lead Human Exposure and Health Risk Assessments. September 27, 2007.

17 Henderson, R. (2008a) Letter from Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory Committee, to
18 Administrator Stephen L. Johnson. Re: Clean Air Scientific Advisory Committee’s (CASAC) Review of
19 the Advance Notice of Proposed Rulemaking (ANPR) for the NAAQS for lead. January 22, 2008.

20 Henderson, R. (2008b) Letter from Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory Committee, to
21 Administrator Stephen L. Johnson. Re: Clean Air Scientific Advisory Committee’s (CASAC) Review of
22 the Notice of Proposed Rulemaking for the NAAQS for lead. July 18, 2008.

23 Hilt, S. R. (2003) Effect of smelter emission reductions on children's blood lead levels. Sci. Total Environ. 303: 51-
24 58.

25 Lanphear, B. P.; Hornung, R.; Khoury, J.; Yolton, K.; Baghurst, P.; Bellinger, D. C.; Canfield, R. L.; Dietrich, K.
26 N.; Bornschein, R.; Greene, T.; Rothenberg, S. J.; Needleman, H. L.; Schnaas, L.; Wasserman, G.;
27 Graziano, J.; Roberts, R. (2005) Low-level environmental lead exposure and children's intellectual
28 function: an international pooled analysis. Environ. Health Perspect. 113: 894-899.

29 Téllez-Rojo, M. M.; Bellinger, D. C.; Arroyo-Quiroz, C.; Lamadrid-Figueroa, H.; Mercado-García, A.; Schnaas-
30 Arrieta, L.; Wright, R. O.; Hernández-Avila, M.; Hu, H. (2006) Longitudinal associations between blood
31 lead concentrations < 10 µg/dL and neurobehavioral development in environmentally-exposed children in
32 Mexico City. Pediatrics 118: e323-e330.

33 U.S. Environmental Protection Agency. (2006) Air Quality Criteria for Lead. Washington, DC, EPA/600/R-
34 5/144aF. Available online at: www.epa.gov/ncea/

35 U.S. Environmental Protection Agency. (2007a) Lead: Human Exposure and Health Risk Assessments for Selected
36 Case Studies, Volume I. Human Exposure and Health Risk Assessments – Full-Scale and Volume II.
37 Appendices. Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-452/R-07-
38 014a and EPA-452/R-07-014b.

39 U.S. Environmental Protection Agency. (2007b) Review of the National Ambient Air Quality Standards for Lead:
40 Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper. EPA-452/R-07-013.
41 Office of Air Quality Planning and Standards, Research Triangle Park.

1 U.S. Environmental Protection Agency. (2012) Integrated Science Assessment for Lead (Third External Review
2 Draft). Washington, DC, EPA/600/R-10/075C. Available online at:
3 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_2010_isa.html

5 WELFARE EFFECTS AND EXPOSURE/RISK INFORMATION

This chapter presents key aspects of the current evidence of lead-related welfare effects, and presents exposure and risk information from the last review in the context of the currently available information. Staff has drawn from the EPA’s synthesis of the scientific evidence presented in the third draft *Integrated Science Assessment for Lead* (USEPA, 2012; henceforth referred to as the ISA) and the 2006 *Air Quality Criteria Document for Lead* (USEPA, 2006; henceforth referred to as the 2006 CD), and from the screening level risk assessment performed in the last review and described in *Lead Human Exposure and Health Risk Assessments and Ecological Risk Assessment for Selected Areas. Pilot Phase* (documented in ICF, 2006; henceforth referred to as the 2006 REA). This chapter is organized into two sections regarding the currently available welfare effects evidence (section 5.1) and the exposure and risk information (section 5.2) interpreted in light of currently available evidence. Presentation within these sections is organized to address key policy-relevant questions for this review concerning the evidence and exposure/risk information, building upon the questions included in the IRP (IRP, section 3.2).

5.1 WELFARE EFFECTS INFORMATION

Lead has been demonstrated to have harmful effects on reproduction and development, growth, and survival in many species as described in the assessment of the evidence available in this review and consistent with the conclusions drawn in past CDs (ISA, section 2.7; 2006 CD). A number of studies on Pb ecological effects are newly available in this review and are critically assessed in the ISA as part of the full body of evidence. The newly available evidence reaffirms conclusions on the array of effects recognized for Pb in the last review (ISA, section 2.7). The current ISA, unlike the 2006 CD, makes causal determinations for the effects of Pb on organisms. The ISA determines that causal or likely causal relationships¹ exist in both freshwater and terrestrial ecosystems for Pb² with effects on reproduction and development in vertebrates and invertebrates; growth in plants and invertebrates; and survival in vertebrates and invertebrates (ISA, table 2-3). Based on these determinations and a weight of evidence approach, the ISA concludes that higher level community and ecosystem effects in freshwater

¹ Since the last Pb NAAQS review, the ISAs which have replaced CDs in documenting each review of the scientific evidence (or air quality criteria) employ a systematic framework for weighing the evidence and describing associated conclusions with regard to causality, using established descriptors (“causal” relationship with relevant exposure, “likely” to be causal, evidence is “suggestive” of causality, “inadequate” evidence to infer causality, “not likely”) (ISA, Preamble).

² In determining that a causal relationship exists for Pb with specific ecological effects, the EPA has concluded that “[e]vidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures (i.e., doses or exposures generally within one to two orders of magnitude of current levels)” (ISA, p. lxiv)

1 and terrestrial systems are likely to result from the above described population-level endpoints.
2 The ISA also presents evidence for saltwater ecosystems but concludes that current evidence is
3 inadequate to make causality determinations for most organisms and communities but finds the
4 evidence to be suggestive linking Pb and effects on reproduction and development in marine
5 invertebrates (ISA, section 1.5, Table 2-3, section 7.3.12 and section 7.4.21).

6 Based on the extensive assessment of the full body of evidence available in this review,
7 the major conclusions drawn by the ISA regarding ecological effects of Pb include the following
8 (ISA, section 1.5).

9 *With regard to the ecological effects of Pb, uptake of Pb into fauna and*
10 *subsequent effects on reproduction, growth and survival are established and are*
11 *further supported by more recent evidence. These may lead to effects at the*
12 *population, community, and ecosystem level of biological organization. In both*
13 *terrestrial and aquatic organisms, gradients in response are observed with*
14 *increasing concentration of Pb and some studies report effects within the range of*
15 *Pb detected in environmental media. Specifically, observations from controlled*
16 *studies on reproduction, growth, and survival in sensitive freshwater*
17 *invertebrates are well-characterized at concentrations at or near Pb*
18 *concentrations occasionally encountered in U.S. surface waters.... However, in*
19 *natural environments, modifying factors affect Pb bioavailability and toxicity and*
20 *there are considerable uncertainties associated with generalizing effects observed*
21 *in controlled studies to effects at higher levels of biological organization.*
22 *Furthermore, available studies on community and ecosystem-level effects are*
23 *usually from contaminated areas where Pb concentrations are much higher than*
24 *typically encountered in the environment. The contribution of atmospheric Pb to*
25 *specific sites is not clear and the connection between air concentration of Pb and*
26 *ecosystem exposure continues to be poorly characterized. Furthermore, the level*
27 *at which Pb elicits a specific effect is difficult to establish in terrestrial and*
28 *aquatic systems, due to the influence of other environmental variables (e.g., pH,*
29 *organic matter) on both Pb bioavailability and toxicity, and also to substantial*
30 *species differences in Pb sensitivity.*

31
32 As in prior reviews of the Pb NAAQS, this review is focused on those effects most
33 pertinent to ambient air Pb exposures. Given the reductions in ambient air Pb levels over the
34 past decades, these effects are generally those associated with the lowest levels of Pb exposure
35 that have been evaluated. Additionally, we recognize the limitations on our ability to draw
36 conclusions about environmental exposures from ecological studies of organism-level effects as
37 most studies were conducted in laboratory settings which may not accurately represent field
38 conditions or the multiple variables that govern exposure.

39 Our consideration of welfare effects evidence in this review is framed by key policy-
40 relevant questions drawn from those included in the IRP. In the following sections, we discuss
41 the pathways by which Pb exposure occurs in ecosystems, the mechanisms that distribute Pb in

1 the environment and the bioavailability of Pb in different ecosystems. Understanding the
2 movement of Pb in the environment is important to understanding exposure and bioavailability
3 and, thus, informs the subsequent discussion of the effects of Pb on terrestrial and aquatic
4 ecosystems. Finally, we discuss the association of ambient air Pb with effects and the important
5 role critical loads play in assessing the overall ability of ecosystems to recover from past Pb
6 exposures and the degree to which newly deposited Pb may affect ecosystem function and
7 recovery.

8 • **To what extent has the newly available evidence altered our understanding of the**
9 **movement and accumulation of air-deposited Pb through ecosystems over time?**

10 There is little new information on fate and transport in ecosystems specifically related to
11 air-derived Pb. Nonetheless, some new studies are available that provide additional information,
12 briefly summarized below, on Pb cycling, flux and retention. This new information does not
13 fundamentally change our understanding from the last review of Pb movement or accumulation
14 through ecosystems over time but rather improves our understanding of some of the underlying
15 processes and mechanisms in soil, water and sediment. The newly available information also
16 adds to the evidence base with regard to the timing of ecosystem recovery from historic
17 atmospheric deposition of Pb.

18 Overall, recent studies in terrestrial ecosystems provide deposition data consistent with
19 deposition fluxes reported in the 2006 CD, and demonstrate consistently that Pb deposition to
20 soils has decreased since the phase-out of leaded on-road gasoline (ISA, section 3.3.3.1).
21 Although follow-up studies in several locations at high elevation sites indicate little change in
22 soil Pb concentrations since the phase-out of leaded on-road gasoline in surface soils, consistent
23 with the high retention reportedly associated with reduced microbial activity at lower
24 temperatures associated with high elevation sites, amounts of Pb in the surface soils at some
25 lower altitude sites were reduced over the same time period in the same study (ISA, section
26 3.3.3.1). New studies in the ISA also enhance our understanding of Pb sequestration in forest
27 soils by providing additional information on the role of leaf litter as a Pb reservoir and the effect
28 of decomposition on Pb distribution and sequestration (ISA, section 3.3.3.2).

29 Recent research on Pb transport in aquatic systems has provided a large body of
30 observations confirming that Pb transport is dominated by colloids rich in iron and organic
31 material (ISA, section 3.3.2.1). In sediments, recent research on Pb flux provides greater detail
32 on resuspension processes than was available in the 2006 CD, including research on resuspended
33 Pb largely associated with organic material or iron and manganese particles, and research on the
34 important role played by anoxic or depleted oxygen environments in Pb cycling in aquatic
35 systems. This newer research is consistent with prior evidence in indicating that appreciable
36 resuspension and release from sediments largely occurs during discrete events related to storms.

1 It has also confirmed that resuspension is an important process that strongly influences the
2 lifetime of Pb in bodies of water. Finally, there have been advances in understanding and
3 modeling of Pb partitioning between organic material and sediment in complex aquatic
4 environments (ISA, section 3.7.2).

5 In summary, the newly available evidence builds on our understanding of some specific
6 aspects of processes involved in the movement and accumulation of air-deposited Pb through
7 ecosystems over time. This new information, however, does not substantially alter our overall
8 understanding of fate and transport of Pb in ecosystems.

9 • **Does the newly available evidence further inform our understanding of the**
10 **bioavailability of Pb in different types of ecosystems and organisms?**

11 As discussed in the ISA, bioavailability of Pb is an important component of
12 understanding the effects Pb is likely to have on organisms and ecosystems (ISA, section 7.3.3).
13 It is the amount of Pb that can interact within the organism that leads to toxicity and there are
14 many factors which govern this interaction. Bioavailability is defined in the Framework for
15 Metals Risk Assessment (USEPA, 2007) as “the extent to which bioaccessible metals absorb
16 onto, or into, and across biological membranes of organisms, expressed as a fraction of the total
17 amount of metal the organism is proximately exposed to (at the sorption surface) during a given
18 time and under defined conditions.” The bioavailability of metals varies widely depending on the
19 physical, chemical, and biological conditions under which an organism is exposed (USEPA,
20 2007). Characteristics of the toxicant itself that affect bioavailability are: (1) chemical form or
21 species, (2) particle size, (3) lability, and (4) source. The bioavailability of a metal is also
22 dependent upon the fraction of metal that is bioaccessible. The bioaccessible fraction of a metal
23 is the portion (fraction or percentage) of environmentally available metal that actually interacts at
24 the organism’s contact surface and is potentially available for absorption or adsorption by the
25 organism (USEPA, 2007; ISA, section 7.3.3).

26 New studies provide additional insight into factors that influence the bioavailability of Pb
27 to specific organisms (ISA, section 7.3.3). In general, this evidence, briefly summarized below,
28 is supportive of previous conclusions and does not identify significant new variables from those
29 identified previously. Section 7.3.3 of the ISA provides a detailed discussion of bioavailability in
30 terrestrial systems. With regard to aquatic systems, a detailed discussion of bioavailability in
31 freshwater systems is provided in sections 7.4.3 and 7.4.4 of the ISA and section 7.4.14 of the
32 ISA discusses bioavailability in saltwater systems.

33 In terrestrial systems, the bioavailability of Pb has a far greater impact on the adverse
34 effects of Pb than does the total Pb burden (ISA, section 7.3.11). In such ecosystems, Pb is
35 deposited either directly onto plant surfaces or onto soil where it can bind with organic matter or

1 dissolve in pore water.³ The Pb dissolved in pore water is particularly bioavailable to organisms
2 in the soil and thereby influences the impact of soil Pb on terrestrial ecosystems to a much
3 greater extent than the total amount of Pb present (ISA, section 7.3.11). Several soil variables
4 control the amount of Pb that is dissolved in pore water, and the ISA presents new evidence that
5 has advanced scientific understanding of some of these variables (ISA, section 7.4.21). Studies
6 have shown that the two most important determinants of both Pb solubility and toxicity in soils
7 are pH and cation exchange capacity (ISA, section 2.7.1). Also, evidence newly available in this
8 review has confirmed the important influence of organic matter on Pb sequestration, leading to
9 relatively longer retention in soils with higher organic matter content, with the potential for later
10 release of deposited Pb (ISA, section 2.7.1). In general, soils with higher organic matter content
11 have the capacity for greater retention of Pb in the soil matrix and lesser availability of Pb for
12 release into pore water. Subsequent reduction in soil organic material, such as through
13 decomposition, can contribute to subsequent increased availability. Aging of Pb in soils, both
14 under natural conditions and simulated through leaching, has also been shown to reduce
15 bioavailability to plants and soil organisms (ISA, sections 7.3.2.3 and 7.3.11).

16 In aquatic systems as in terrestrial systems, the amount of Pb bioavailable to organisms is
17 a better predictor of effect on organisms than the overall amount of Pb in the system. Once
18 atmospherically-derived Pb enters surface water bodies through deposition or runoff, its fate and
19 bioavailability are influenced by many water quality characteristics, such as pH, suspended
20 solids loading and organic content (ISA, section 7.4.2). In sediments, bioavailability of Pb to
21 sediment-dwelling organisms may be influenced by the presence of other metals, sulfides, iron
22 oxides and manganese oxides and also by physical disturbance (ISA, section 2.7.2). For many
23 aquatic organisms Pb dissolved in the water column can be the primary exposure route, while for
24 others sediment ingestion is significant (ISA, section 2.7.2). As recognized in the 2006 CD and
25 further supported in the ISA, there is a body of evidence showing that uptake and elimination of
26 Pb varies widely between species.

27 Although in freshwater systems the presence of humic acid in dissolved organic material
28 (DOM) is considered to reduce the bioavailable fraction of metals in the water column, there is
29 evidence presented in the ISA that dissolved organic carbon and DOM do not have the same
30 effect on free Pb ion concentration in saltwater systems (ISA, section 7.4.24). Recent studies
31 have broadened previous understandings of the role of DOM in Pb bioavailability and have
32 shown that different components of DOM have different effects on Pb bioavailability in
33 saltwater systems. For example, the ISA discusses two new studies that suggest that in saltwater
34 aquatic systems, surface water DOM increases (rather than decreases) uptake of Pb by fish gill

³ The term “pore water” refers to the water occupying the spaces among the grains of sediment or soil.

1 structures, potentially through the alteration of membrane permeability. This recent evidence
2 supports the conclusion from the last review that factors that modify bioavailability of Pb in
3 saltwater environments are not identical to those in freshwater systems (ISA, section 7.4.14).

4 There is new evidence about bioavailability in some ecosystems, briefly summarized
5 above, which further informs our understanding of bioavailability of Pb in different ecosystems
6 and organisms, building on our fundamental understanding in the last review. Bioavailability
7 remains an important consideration in Pb toxicity and a significant source of uncertainty relating
8 ambient Pb and adverse effects.

- 9 • **Does the current evidence alter our conclusions from the previous review**
10 **regarding the ecological effects associated with exposure to Pb? Does the newly**
11 **available evidence indicate new exposure levels at which ecological systems or**
12 **receptors are expected to experience effects?**

13 There is a substantial amount of new evidence in this review regarding the ecological
14 effects of Pb on specific terrestrial and aquatic organisms. On the whole, this evidence supports
15 previous conclusions that Pb has effects on growth, reproduction and survival, and that under
16 some conditions these effects can be adverse to organisms and ecosystems. The ISA provides
17 evidence of effects in additional species, and in a few cases, at lower exposures than reported in
18 the previous review, but does not substantially alter the effects endpoints from the previous
19 review. Looking beyond organism-level evidence, the evidence of adversity in natural systems
20 remains sparse due to the difficulty in determining the effects of confounding factors such as
21 multiple metals or factors influencing bioavailability in field studies. The following is a brief
22 comparison of the newly available evidence to evidence considered in the 2006 review.

23 *Terrestrial Ecosystems*

24 The evidence available in the last review indicated a range of biological effects of Pb on
25 terrestrial organisms that varied with type of organism and life stage, duration of exposure, form
26 of Pb, and soil characteristics. New research since the 2006 CD has broadened our
27 understanding of the evidence of damage to photosynthetic ability in plants exposed to Pb, and
28 provided additional evidence of oxidative stress in response to Pb exposure (ISA, section
29 7.3.4.1). Reactive oxygen species have been found to increase in plant tissue grown in Pb-
30 contaminated soil; with increasing Pb exposure, the plant tissue responded with increased
31 antioxidant activity (ISA, section 7.3.4.1). In addition, reduced growth has been observed in
32 some experiments, as well as evidence of genotoxicity, decreased germination, and pollen
33 sterility (ISA, section 7.3.4.1).

34 In terrestrial invertebrates, previous CDs have reported adverse effects of Pb on
35 neurological and reproductive endpoints. Recently published studies have shown some
36 additional evidence of neurological changes in nematodes at relatively low concentrations of

1 soil Pb (2.5 μ M) but given the study design the relevance to environmental exposure is unclear
2 (ISA, section 7.3.4.2). Increased mortality was found in recent studies of earthworms at
3 concentrations similar to those in studies reviewed previously, but additional evidence indicated
4 that effects were strongly dependent on soil characteristics including pH, cation exchange
5 capacity, and aging (ISA, section 7.3.4.2). There is also newly available evidence for adverse
6 effects in snails and arthropods exposed through soil or diet (ISA, section 7.3.4.2). The effects
7 vary with species and exposure conditions, and include diminished growth and fecundity,
8 endocrine and reproductive anomalies, and body deformities. Increasing concentration of Pb in
9 the exposure medium generally resulted in increased effects within each study, but the
10 relationship between concentration and effects is highly variable between studies, even when
11 the same medium, e.g. soil, was used. Current evidence suggests that aging and pH are
12 important modifiers of Pb toxicity (ISA, section 7.3.11).

13 In terrestrial vertebrates, some new evidence is available for effects of Pb on amphibians
14 and reptiles as well as birds. Effects reported in reptiles and amphibians include decreased white
15 blood cell counts, decreased testis weight, and behavioral anomalies (ISA, section 7.3.4.3).
16 However, depending on various factors, studies report large differences in effects in different
17 species at the same concentration of Pb in soil, and effects were generally smaller when field-
18 collected soils were used. In some birds, recent studies have found maternal elevated blood Pb
19 level to be associated with decreased hatching success, smaller clutch size, high corticosteroid
20 level, and abnormal behavior. Studies on some species show little or no effect of elevated blood
21 Pb level. Effects of dietary exposure have been studied in several mammalian species, with
22 cognitive, endocrine, immunological, and growth effects observed (ISA, section 7.3.11) in some
23 studies.

24 Experimental evidence of organism-level effects presented in the ISA demonstrates that
25 increased exposure to Pb is generally associated with increases in observed effects in terrestrial
26 ecosystems (ISA, section 7.3.11). It also demonstrates that many factors, including species and
27 various soil physiochemical properties, interact strongly with Pb concentration to modify those
28 effects (ISA, section 7.3.11). In terrestrial ecosystems, where soil is generally the main
29 component of the exposure route, Pb aging is a particularly important factor, and one that may
30 be difficult to reproduce experimentally. Given that in natural settings, these modifying factors
31 are highly variable, the ISA notes that “[w]ithout quantitative characterization of those
32 interactions, characterizations of exposure-response relationships would likely not be
33 transferable outside of experimental settings” (ISA, section 2.7.1). The results of the few
34 exposure-response studies conducted since the 2006 CD have been inconsistent (ISA, section
35 2.7.1).

1 As reported in both the ISA and the 2006 CD, most direct evidence of community- and
2 ecosystem-level effects is from releases by near sources of Pb to the environment, where Pb
3 concentrations are much higher than typically observed environmental concentrations and often
4 derive from separate releases to multiple media. Impacts of Pb on terrestrial ecosystems near
5 smelters, mines, and other industrial sources have been studied for several decades (ISA, section
6 7.3.12.7). Emissions of Pb from smelting and other industrial activities are commonly
7 accompanied by other trace metals (e.g., Zn, Cu, Cd) and SO₂ that may cause toxic effects
8 independently or in concert with Pb. Those impacts have been long shown to include decreases
9 in species diversity and changes in floral and faunal community composition. Ecosystem-level
10 field studies are complicated by these confounding exposures and the inherent variability in
11 natural systems (ISA, section 7.3.12.7).

12 New evidence of effects of Pb at the community and ecosystem scales has been
13 reported. This evidence includes several studies of the ameliorative effects of mycorrhizal fungi
14 on plant growth with Pb exposure as well as recently published research on soil microbial
15 communities, which have been shown to be impacted by Pb in both composition and activity
16 (ISA, sections 2.7.1 and 7.3.6). Many recent studies have been conducted using mixtures of
17 metals, which have attempted to separate the effects of individual metals when possible. In
18 studies that included only Pb, or where effects of Pb could be separated, soil microbial activity
19 was generally diminished, and was shown in some of those cases to recover with time (ISA,
20 section 7.3.6). Regardless of the recovery of such microbial communities, species and
21 genotypical composition were consistently altered after Pb exposure, and findings indicate that
22 those alterations were long-lasting or permanent (ISA, section 7.3.11).

23 A recent review has examined differences in species sensitivity, using blood Pb level in
24 birds and mammals as an exposure index rather than external dose (ISA, section 7.3.9.1;
25 Buekers et al., 2009). In this analysis, variation across organisms was reduced with the blood
26 Pb index and variation of Pb absorption from the diet of the organism largely accounted for the
27 variation seen in the blood Pb. The analysis also suggests that the association between blood Pb
28 concentration and toxicity is different between birds and mammals (ISA, section 7.3.9.1).

29 Overall, new studies in this review support previous conclusions about the effects of Pb
30 on terrestrial ecosystems, namely that increasing soil Pb concentrations in areas of Pb
31 contamination (e.g. mining sites and industrial sites) can cause decreases in microorganism
32 abundance, diversity, and function. Specifically, shifts in bacterial species and fungal diversity
33 have been recently observed near long-established sources of Pb contamination (ISA, section
34 7.3.12.7). Most evidence for Pb toxicity to terrestrial plants, invertebrates and vertebrates,
35 however, is from single-species assays in laboratory studies which do not capture the

1 complexity of bioavailability and other modifiers of effect in natural systems (ISA, section
2 7.3.12.7).

3 ***Freshwater Ecosystems***

4 In fish, while there are species differences in the rate of Pb accumulation and distribution
5 of Pb within the organism, the gills and diet are the primary routes of uptake. Recent studies
6 have identified the anterior intestine as an additional site of uptake of Pb through dietary
7 exposure (ISA, section 7.4.4.3). While there are few new studies on Pb uptake through
8 freshwater exposure by amphibians and mammals, there are some new studies presented in the
9 ISA which seem to provide new information on trophic transfer of Pb (ISA, section 7.4.4.4).
10 Evidence summarized in the 2006 CD indicated that measured concentrations of Pb in the
11 tissues of aquatic organisms were generally higher in algae and benthic organisms than in
12 higher trophic-level consumers, indicating that Pb was bioconcentrated but not biomagnified
13 (ISA, section 2.7.2). Some recent studies indicate transfer of Pb in aquatic food webs; recent
14 studies that have traced Pb in freshwater aquatic food webs have found that Pb concentration
15 decreases with increasing trophic level (biodilution) (ISA, sections 2.7.2 and 7.4.4.4).

16 Evidence presented in the ISA further supports the findings of past CDs that Pb in
17 freshwater can be highly toxic to aquatic organisms, with toxicity varying with species and life
18 stage, duration of exposure, form of Pb, and water quality characteristics. The 2006 CD
19 identified evidence of adverse growth effects on several species of freshwater algae from Pb
20 exposure (2006 CD, Section 7.2.4). The ISA describes several new studies which expand the
21 list of algal species for which these adverse effects have been identified (ISA, section 7.4.5.1).
22 For vascular plants, the ISA describes additional evidence that oxidative damage, decreased
23 photosynthesis and reduced growth occur with Pb exposure (ISA, section 7.4.5.2).

24 Since the 2006 CD, there is some additional evidence for Pb effects on cellular processes
25 in aquatic invertebrates. Recent studies of reproductive and developmental effects of Pb provide
26 further support for findings in the 2006 CD and add additional species information on
27 reproductive endpoints for rotifers and freshwater snails as well as multigenerational effects of
28 Pb in mosquito larvae (ISA, section 7.4.5.2). In the 2006 CD, study concentrations cited at
29 which effects were observed in aquatic invertebrates reflected an expansive range from 5µg/L
30 (for acute toxicity to Pb nitrate in a test system at a hardness of 18 mg/L calcium carbonate) to
31 greater than 8000 µg/L (for acute toxicity to Pb chloride at a hardness of 280-300 mg/L calcium
32 carbonate) (ISA, p.2-38; 2006 CD, Table AX7-2.4.1). Recent studies cited in the ISA provide
33 additional evidence of effects from chronic exposures. These findings are generally consistent
34 with those of the previous review and reflect the variability of Pb toxicity under different
35 environmental conditions and in species with varying sensitivity.

1 Evidence of effects in aquatic vertebrates presented in the ISA reiterates the findings of
2 reproductive, behavioral, and growth effects stated in previous CDs. Some additional
3 mechanisms of Pb toxicity in the gill and the renal system of fish have been elucidated since the
4 2006 CD as well as the identification of potential new molecular targets for Pb neurotoxicity
5 (ISA, section 2.7.2). Recent studies reported sublethal effects on tadpole growth, deformity, and
6 swimming ability at concentrations within two orders of magnitude of environmental levels of
7 Pb (ISA, section 7.4.5.3) based on nominal concentrations.

8 As in terrestrial organisms, evidence presented in the ISA and prior CDs demonstrates the
9 toxicity of Pb in aquatic ecosystems and the role of many factors, including Pb speciation and
10 various water chemistry properties in modifying toxicity (ISA, section 2.7.2). Since the 2006
11 CD, additional evidence for community and ecosystem level effects of Pb is available, primarily
12 in microcosm studies or field studies with other metals present (ISA, section 7.4.11). Such
13 evidence described in previous CDs includes alteration of predator-prey dynamics, species
14 richness, species composition, and biodiversity. New studies available in this review provide
15 evidence in additional habitats for these community and ecological-scale effects, specifically in
16 aquatic plant communities and sediment-associated communities at both acute and chronic
17 exposures involving concentrations similar to previously reported (ISA, section 7.4.4.1). In
18 many cases it is difficult to characterize the nature and magnitude of effects and to quantify
19 relationships between ambient concentrations of Pb and ecosystem response due to existence of
20 multiple stressors, variability in field conditions, and to differences in Pb bioavailability at that
21 level of organization (ISA, section 2.7.3.7). In these field studies, the degree to which air
22 concentrations contribute to such effects is largely unknown.

23 ***Saltwater Ecosystems***

24 Recently available evidence in the ISA on toxicity of Pb to marine algae augments the
25 2006 CD findings of variation in sensitivity across marine species. Recent studies on Pb
26 exposure include reports of growth inhibition and oxidative stress in a few additional species of
27 marine algae (ISA, section 7.4.15).

28 Recent literature provides little new evidence of endpoints or effects in marine
29 invertebrates beyond those reported in the 2006 CD. For example, some recent studies
30 strengthen the evidence of Pb effects on enzymes and antioxidant activity in marine
31 invertebrates and have identified an additional behavioral endpoint (i.e., valve closing speed in
32 juveniles of a marine scallop was affected by 20-day exposure to 40-400 µg/L Pb nitrate) from
33 those discussed previously (ISA, section 7.4.15.2). Also, as noted in the 2006 CD and supported
34 by new studies reviewed in the ISA, Pb exposure negatively affects the growth of marine
35 invertebrates (ISA, section 7.4.15.2). Recent studies also identify several species exhibiting
36 particularly low sensitivity to high acute exposures (ISA, section 7.4.15.2).

1 Little new evidence is available of Pb effects on marine fish and mammals for
2 reproductive, growth and survival endpoints that are particularly relevant to population-level of
3 biological organization and higher (ISA, section 7.4.15). Evidence for effects at higher levels of
4 biological organization in saltwater habitats is primarily supported by observations in a small
5 number of microcosm and field studies where shifts in community structure are the most
6 commonly observed effects of Pb (ISA, section 7.4.20). These types of studies were presented
7 in the 2006 CD and while there are new studies presented in the ISA, they primarily expand the
8 types of communities for which data exist but reach similar conclusions about community
9 structure as the 2006 CD. However, there is no evidence of adverse effects in saltwater
10 environments from current ambient air concentrations and linkages between any level of
11 ambient exposure and effects are unknown in all but a few organisms.

12 New studies on organism-level effects from Pb in saltwater ecosystems provide little
13 evidence to inform our understanding of linkages between atmospheric concentrations, ambient
14 exposures in saltwater systems and such effects or our conclusions regarding the likelihood of
15 adverse effects under conditions associated with the current NAAQS for Pb. Nor does the
16 currently available evidence indicate significantly different exposure levels at which ecological
17 systems or receptors are expected to experience effects. The new evidence, as outlined above,
18 serves to support previous conclusions regarding effects evidence and provides information on
19 additional species, but does not significantly improve our understanding of ecological effects
20 levels of Pb.

21 • **To what extent is there new information that informs our understanding of**
22 **critical loads of Pb, including critical loads in sensitive ecosystems?**

23 Critical loads are an especially powerful tool that can link Pb input into an ecosystem,
24 including atmospheric deposition, with ecological impairment. Critical load calculations are
25 dependent on data relating Pb released into an ecosystem from various sources with an
26 understanding of the level of Pb in an ecosystem that is likely to result in measurable effects.
27 Importantly, with these data, critical loads can account for heterogeneity in ecosystem sensitivity
28 and exposure which often results in critical load values that vary by ecosystem (e.g., aquatic-
29 water; aquatic-sediment; terrestrial), and differ by endpoint of concern (ISA, section 7.1.3).
30 During the last review, the 2006 CD assessed the available information on critical loads for Pb
31 which was drawn primarily from Europe. Analyses were not available for U.S. locations, and
32 the European critical load values for Pb that had been developed were highly specific to the
33 bedrock geology, soil types, vegetation, and historical deposition trends in each European
34 country (2006 CD, p. E-24). As a result, the 2006 CD concluded that “[a]t this time, the methods
35 and models commonly used for the calculation of critical loads have not been validated for Pb”
36 and that “[m]any of the methods neglect the speciation of Pb when estimating critical limits, the

1 uptake of Pb into plants, and out flux of Pb in drainage water, limiting the utility of current
2 models” (2006 CD, p. 7-46). Accordingly, the quantitative assessment for the last Pb NAAQS
3 review did not involve critical load analyses.

4 There are no critical loads for Pb reported in the ISA or 2006 CD for any ecosystems in
5 the U.S.; however, as reported in the 2006 CD and further discussed in section 7.3.7 of the ISA,
6 work has been conducted in terrestrial systems in Europe and aquatic ecosystems in Canada
7 (ISA, section 7.3.7 and section 7.4.8). Given that local conditions (including historic loading, soil
8 transport and transformation processes) are key elements to critical load calculation, the utility of
9 critical loads that are developed from other countries for application to U.S. ecosystems is
10 unclear. Given the heterogeneity of ecosystems affected by Pb, and the differences in
11 expectations for ecosystem services attached to different land uses, it is expected that there
12 would be a range of critical load values for Pb for soils and freshwaters within the U.S.

13 In the short term, metal emissions generally have greater effects on biota in freshwater
14 systems than in terrestrial systems because metals are more readily immobilized in soils than in
15 sediment. However, over the longer term, terrestrial systems may be more affected by increasing
16 concentrations, particularly by those metals with a long soil residence time, such as Pb (ISA,
17 section 7.4.8). There is no new evidence regarding critical loads for Pb in saltwater ecosystems.

- 18 • **To what extent is there information that improves our understanding of the**
19 **portion of environmental Pb derived from ambient air and the associated effects**
20 **on sensitive ecosystems?**

21 As stated in the last review, the role of ambient air Pb in contributing to ecosystem Pb
22 has been declining over the past several decades. However, it remains difficult to apportion
23 exposure between air and other sources to better inform our understanding of ecosystem effects
24 that might be associated with air emissions. Considerable uncertainties also remain in drawing
25 conclusions from evidence of effects observed under laboratory conditions with regard to effects
26 expected at the ecosystem level in the environment. In many cases it is difficult to characterize
27 the nature and magnitude of effects and to quantify relationships between ambient concentrations
28 of Pb and ecosystem response due to the existence of multiple stressors, variability in field
29 conditions, and to differences in Pb bioavailability at that level of organization (ISA, section
30 7.5). There is no new evidence that substantially improves our understanding of the relationship
31 between ambient air Pb and measurable ecological effects.
32

1 **5.2 EXPOSURE AND RISK INFORMATION**

2 This risk information available for this review and described here is based primarily on
3 the pilot ecological risk assessment developed in the last review of the Pb NAAQS (henceforth
4 referred to as the 2006 REA [ICF, 2006]), which is described within the context of the evidence
5 presented in the ISA that is newly available for this review. As described in the IRP, careful
6 consideration of newly available information in this review led us to conclude that developing a
7 new REA for this review was not warranted. In light of critical limitations and uncertainties that
8 are still unresolved in the current evidence, staff concluded that currently available information
9 does not provide the basis for developing a new quantitative risk and exposure assessment with
10 substantially improved utility for informing the Agency’s consideration of welfare effects and
11 evaluation of the adequacy of the current secondary standard or alternatives (IRP, section 3.3).
12 Based on their consideration of the IRP, the CASAC Pb Review Panel generally concurred with
13 the conclusion that a new REA was not warranted in this review (Frey, 2011).⁴ Accordingly, the
14 information described here is drawn primarily from the 2006 REA.

15 The focus for the risk assessment and associated estimates presented here is on Pb
16 derived from sources emitting Pb to ambient air. While there is some new evidence that
17 improves our understanding of some of the environmental variability affecting the disposition
18 and toxicity of Pb in the environment, the information and methods to support a quantitative
19 assessment of the role of atmospheric Pb in the U.S are limited. Specific constraints include the
20 limited availability of location-specific data describing a range of U.S. ecosystems and their
21 pertinent environmental characteristics as well as the ability to apportion effects between air and
22 non air sources, and a more complete understanding of bioavailability and its modifiers. These
23 data gaps and areas of uncertainty in the current evidence restrict our ability to assess
24 quantitatively the relationship between concentrations of Pb in ambient air and terrestrial and/or
25 aquatic systems, and their effect on welfare.

26 As discussed in section 1.3 above, the multimedia and persistent nature of Pb, the role of
27 multiple exposure pathways (illustrated in Figure 1-1 above), and the contributions of nonair
28 sources of Pb to exposure media all present challenges and contribute significant additional
29 complexity to the ecological risk assessment that goes far beyond the situation for similar
30 assessments typically performed for other NAAQS pollutants (e.g., that focus only on a single
31 media pathway or for which air is the only significant source). Limitations in the available data
32 and models affected our characterization of the various complexities associated with exposure to

⁴ In our evaluation presented in the REA Planning Document and consultation with CASAC, we indicated our conclusion that the information newly available in this review did not provide the means by which to develop an updated or enhanced risk model that would substantially improve the utility of risk estimates in informing the current Pb NAAQS review.

1 ambient air Pb. As a result, the 2006 assessment was conducted as a pilot study with a number
2 of simplifying assumptions with regard to the representativeness of the case studies for
3 ecological exposures and our ability to isolate case studies where air-derived Pb was the only or
4 most significant source. Therefore, this section presents a brief summary of the screening-level
5 ecological risk assessment conducted in 2006 for the Pb NAAQS review completed in 2008 and
6 addresses several questions relating to the current evidence and understanding that may inform
7 our view of the results of that assessment. The discussion here also takes into consideration
8 CASAC recommendations in the last review with regard to interpretation of the screening-level
9 assessment (Henderson, 2007a, b), as well as comments received from the CASAC Pb Panel in
10 the current review, as part of the consultation on the REA Planning Document (Frey, 2011).

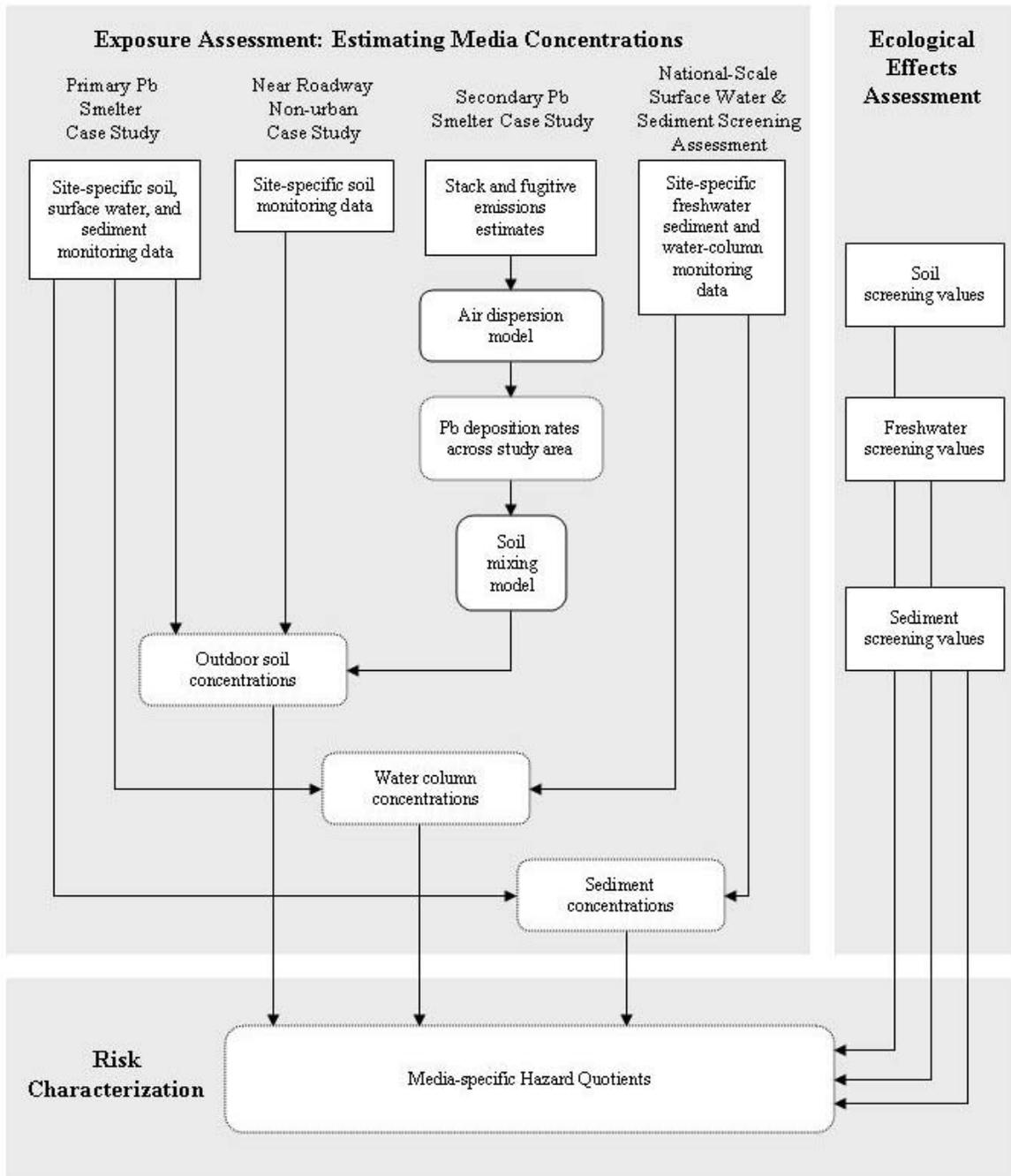
11 **5.2.1 Screening Assessment from Last Review**

12 The screening-level risk assessment performed for the last review was focused on
13 estimating the potential for ecological risks associated with ecosystem exposures to Pb emitted
14 into ambient air (2006 REA, section 7). A national-scale screen was used to evaluate surface
15 water and sediment monitoring locations across the United States for the potential for ecological
16 impacts that might be associated with atmospheric deposition of Pb (described in detail in 2006
17 REA, section 7.1.2). In addition to the national-scale screen (2006 REA, section 3.6), the
18 assessment involved a case study approach, with case studies for areas surrounding a primary Pb
19 smelter (2006 REA, section 3.1) and a secondary Pb smelter (2006 REA, section 3.2), as well as
20 a location near a non-urban roadway (2006 REA, section 3.4). An additional case study, focused
21 on consideration of gasoline-derived Pb effects on an ecologically vulnerable ecosystem
22 (Hubbard Brook Experimental Forest), was identified (2006 REA, section 3.5). The Hubbard
23 Brook Experimental Forest (HBEF), in the White Mountain National Forest, near North
24 Woodstock, New Hampshire, was selected as a fourth case study because of its location and its
25 long record of available data on concentration trends of Pb in three media (air or deposition from
26 air), soil, and surface water). While no quantitative analyses were performed, summary review of
27 the literature search was included in the assessment report (2006 REA, Appendix E). For the
28 other three case studies, exposure concentrations in soil, surface water, and/or sediment
29 concentrations were estimated from available monitoring data or modeling analysis, and then
30 compared to ecological screening benchmarks (2006 REA, section 7.1).

31 All three case studies and the national-scale assessment generally considered then-current
32 or recent environmental conditions. In all cases but the primary Pb smelter case study, current
33 air quality conditions were below the then-current NAAQS. Air Pb concentrations in the
34 primary Pb smelter case study exceeded the then-current NAAQS. A complete discussion of air
35 quality in each of the case studies can be found in section 4 of the 2006 REA.

1 An overview of the approach developed to implement the selected elements of the
2 conceptual model for the ecological risk assessment is provided in Figure 5-1. This figure shows
3 the key types of information and models involved in each part of the assessment and how they
4 are related to each other and to the other parts of the analysis. Appendix 5A gives the locations
5 and spatial resolution for each of the case studies and the national scale screen while summarizes
6 the source of the screening values used or each location and media type. As indicated in Figure
7 5-1 and Appendix 5A, the specific approach for each case study differed based on the nature of
8 the case study (e.g., type of source, locations of populations) and the site-specific measurements
9 available.
10

1 **Figure 5-1. Analytical approach for screening-level assessment in the last review (2006**
 2 **REA, Exhibit 2-6).**



3
 4
 5
 6
 7

1 To estimate the potential for ecological risk, ecological soil screening values (Eco-SSLs)
2 developed by the EPA's Superfund program (USEPA, 2003, 2005), the EPA's recommended
3 ambient water quality criteria (2006 REA, section 7.1) and sediment screening values developed
4 by MacDonald and others (2000, 2003) were used, as described in detail in section 7.1.3 of the
5 2006 REA. A hazard quotient (HQ) was calculated for adverse effects on the survival, growth,
6 and reproduction of exposed ecological receptors to determine the potential for risk to that
7 receptor. Ecological receptors used in the pilot are discussed in detail in section 7.1 of the 2006
8 REA. The HQ was calculated as the ratio of the media concentration to the ecotoxicity screening
9 value. In each case study, HQ values were calculated for each location where either modeled or
10 measured media concentrations were available. Separate soil HQ values were calculated for
11 each ecological receptor group for which an ecotoxicity screening value has been developed (i.e.,
12 birds, mammals, soil invertebrates, and plants), as described in detail in section 7.1.2 of the 2006
13 REA. HQ values less than 1.0 were concluded to suggest that Pb concentrations in a specific
14 medium were unlikely to pose significant risks to ecological receptors, while HQ values greater
15 than 1.0 indicated a potential for adverse effects.

16 **5.2.2 Screening Assessment Results and Interpretation**

17 The results for the ecological screening assessment for the three case studies and the
18 national-scale screen for surface water and for sediment indicated a potential for adverse effect
19 from ambient Pb to multiple ecological receptor groups in terrestrial and aquatic locations.
20 Below are descriptions of the location-specific case studies and the national screening
21 assessment, key findings of the risk assessment for each, and an interpretation of the results with
22 regard to past air conditions as well as the current standard (USEPA, 2007b).

23 **• What do the key findings of the 2006 screening-level assessment indicate** 24 **regarding the likelihood that adverse welfare effects would result from levels of** 25 **air-related Pb that would meet the current standard?**

26 In addressing this question, the findings of the 2006 REA are summarized below.

27 Primary Pb Smelter Case Study

- 28 • The primary Pb smelter case study location is at one of the largest and longest-
29 operating primary Pb smelters in the world (since 1892), the only one currently
30 operating in the U. S. (ICF, 2006).⁵
- 31 • Concentrations of total Pb in several of the soil and sediment locations within the case
32 study were measured in 2000 and exceeded screening values, indicating a potential for
33 adverse effects to terrestrial and sediment dwelling organisms.

⁵ As noted in section 2.1.2 above, this smelter is planning to cease existing smelting operations at this facility by April, 2014.

1 • While the contribution to these Pb concentrations from air as compared to nonair
2 sources is not quantified, air emissions from this facility are substantial (ICF, 2006). In
3 addition, this facility that has been emitting Pb for many decades, including some
4 seven decades prior to establishment of any Pb NAAQS, such that it is likely air
5 concentrations associated with the facility were substantial relative to the 1978
6 NAAQS, which it exceeded at the time of the last review. Currently, concentrations
7 monitored near this facility exceed the level of the current standard. Accordingly, this
8 case study is not informative for considering the likelihood of adverse welfare effects
9 related to Pb from air sources under air quality conditions associated with meeting the
10 current Pb standard.

11 Secondary Pb Smelter Case Study

- 12 • The secondary Pb smelter location falls within the Alabama Coastal Plain in Pike
13 County, Alabama, in an area of disturbed forests. The industrial facility in this case
14 study is much younger than the primary Pb smelter, becoming active less than ten years
15 prior to the establishment of the 1978 Pb standard.
- 16 • Estimates of total Pb concentration in soils (based on fate and transport modeling using
17 1997-2000 emissions data and data for similar locations measured in a 1995 study)
18 exceeded screening values for plants, birds and mammals, indicating the potential for
19 adverse effects to these groups.
- 20 • While the contributions from air-related Pb to the total Pb concentrations modeled in
21 soils at this location is unclear, the facility continues to emit Pb and the county where
22 this facility is located does not meet the current Pb standard (Appendix 5A). Given the
23 exceedances of the current standard, which likely extends back over 4 to 5 decades, this
24 case study also is not informative for considering the likelihood of adverse welfare
25 effects related to Pb from air sources under air quality conditions associated with
26 meeting the current Pb standard.

27 Near Roadway Non-urban Case Study

- 28 • This case study comprises two nonurban sites adjacent to established highways for
29 which soil Pb data were available: (1) in Corpus Christi, Texas (ICF, 2006) and (2) in
30 Atlee, Virginia (ICF, 2006). Measured soil concentration data were used to develop
31 estimates of Pb in soils for each location.
- 32 • Estimates of total Pb concentrations taken in 1994 and 1998 in soils in this case study
33 exceeded screening values for plants, birds and mammals, indicating the potential for
34 adverse effect to these groups.

35 These case study locations are highly impacted by past deposition of gasoline Pb. It is
36 unknown whether current conditions at these sites exceed the current Pb standard but
37 given evidence from the past of Pb concentrations near highways that ranged above the
38 previous (1978) Pb standard (1986 CD, section 7.2.1), conditions at these locations
39 during the time of leaded gasoline very likely exceeded the current standard. Similarly,
40 those conditions likely resulted in Pb deposition associated with leaded gasoline that
41 exceeds that being deposited under current air quality conditions that likely meet the
42 current Pb standard. Given this legacy, consideration of the potential for

1 environmental risks from levels of air-related Pb associated with meeting the current
2 Pb standard in these locations is highly uncertain.

3 Vulnerable Ecosystem Case Study

- 4 • This case study was focused on consideration of information available for the Hubbard
5 Brook Experimental Forest (HBEF) in the White Mountain National Forest near North
6 Woodstock, New Hampshire, which included a long record (from 1976 through 2000)
7 of available data on concentration trends of Pb in three media (air or deposition from
8 air, soil, and surface water).
- 9 • While no quantitative analyses were performed, summary review of literature search
10 indicated: (1) atmospheric Pb inputs do not directly affect stream Pb levels at HBEF
11 because deposited Pb is almost entirely retained in the soil profile; (2) soil horizon
12 analysis results showed Pb to have become more concentrated at lower soil depths over
13 time, with the soil serving as a Pb sink, appreciably reducing Pb in pore water as it
14 moves through the soil layers to streams (dissolved Pb concentrations were reduced
15 from 5 ppb to about 5 ppt from surface soil to streams). Further, the available studies
16 concluded insignificant contribution of dissolved Pb from soils to streams (ICF, 2006,
17 Appendix E). It is unlikely that conditions have changed from the previous
18 conclusions made based on soil data through 2000 and therefore, current ambient air
19 concentrations likely do not directly impact stream Pb levels under air quality
20 conditions associated with meeting the current standard.

21 National-scale Surface Water and Sediment Screen

- 22 • The national-scale screen was performed using a national database of surface water and
23 sediment monitoring data (see Appendix 5A; ICF, 2006).
 - 24 – The screen identified 15 non-mining sites at which at least one surface water
25 dissolved Pb concentration taken between 1994 and 2004 exceeded the chronic
26 screening value, indicating a potential for adverse effect if concentrations were
27 persistent over long time periods. The acute screening value was not exceeded
28 at any of these locations.
 - 29 – Analysis of the sediment concentrations for the 15 sites taken from 1991 to
30 2000 analyzed in the surface water screen identified a subset for which
31 concentrations exceeded the screening value, indicating a potential for adverse
32 effects to sediment dwelling organisms.
- 33 • The extent to which past air emissions of Pb have contributed to surface water or
34 sediment Pb concentrations at the locations identified in the screen is unclear. For
35 some of the surface water locations, nonair sources likely contributed significantly to
36 the surface water Pb concentrations. For other locations, a lack of nearby nonair
37 sources indicated a potential role for air sources to contribute to observed surface water
38 Pb concentrations. Additionally, these concentrations may have been influenced by Pb
39 in resuspended sediments and/or reflect contribution of Pb from erosion of soils with
40 Pb derived from historic as well as current air emissions.

1 There are multiple sources of uncertainty associated with different aspects of this
2 assessment (discussed in detail in 2006 REA, section 7.4). For example, there are significant
3 limitations and uncertainties associated with conclusions that can be drawn from the primary
4 smelter case study regarding the impact of atmospheric deposition under conditions associated
5 with meeting the Pb NAAQS (past or present). Additionally, while case study locations were
6 chosen with the objective of including locations for which recent Pb data were available and for
7 which Pb exposures might be influenced by air-related Pb and not be dominated by nonair
8 sources, there is significant uncertainty regarding the extent to which nonair sources and
9 conditions associated with historic emissions (e.g., prior to establishment of past or present Pb
10 NAAQS) have likely contributed to the Pb exposure estimates in some locations. The screening
11 values available and used in the assessment were also sources of uncertainty with regard to
12 conclusions as to potential for ecological risk as the selected screening values (e.g., ambient
13 water quality criteria, Eco-SSLs, sediment criteria) lacked adjustment for some critical measures
14 of bioavailability. There is also uncertainty regarding the extent to which the screening values
15 could identify potential hazard for some threatened or endangered species or unusually sensitive
16 aquatic ecosystems (ICF 2006). Thus, while the assessment results are generally consistent with
17 evidence-based observations of the potential influence of Pb on ecological systems, they are
18 limited with regard to quantitative conclusions and potential hazard or risk associated with the Pb
19 NAAQS, most particularly with regard to the current standard.

20 • **What are the important uncertainties associated with interpreting the prior**
21 **assessment in light of newly available evidence in this review?**

22 In interpreting the results from the 2006 REA, we consider newly available evidence that
23 may inform our interpretation of risk under the current standard. Factors necessary to alter our
24 interpretation of risk would include new evidence of harm at lower concentrations of Pb, new
25 linkages that enable us to draw more explicit conclusions as to the air contribution of
26 environmental exposures, and/or new methods of interpreting confounding factors that were
27 largely uncontrolled in the previous risk assessment.

28 With regard to new evidence of harm at lower concentrations, it is necessary to consider
29 that the evidence of adversity due specifically to Pb in natural systems is limited, in no small part
30 because of the difficulty in determining the effects of confounding factors such as multiple
31 metals and modifying factors influencing bioavailability in field studies. Modeling of Pb related
32 exposure and risk to ecological receptors is subject to a wide array of sources of both variability
33 and uncertainty. Variability is associated with geographic location, habitat types, physical and
34 chemical characteristics of soils and water that influence Pb bioavailability, terrestrial and
35 aquatic community composition, Pb uptake rates by invertebrates, fish, and plants by species and
36 season. For wildlife, variability also is associated with food ingestion rates by species and

1 season, prey selection, and locations of home ranges for foraging relative to the Pb
2 contamination levels (USEPA, 2006b).

3 There are significant difficulties in quantifying the role of air emissions under the current
4 standard, which is significantly lower than the previous standard. As recognized in section 1.3.2
5 above, lead deposited before the standard was enacted remains in soils and sediments,
6 complicating interpretations regarding the impact of the current standard; historic Pb emitted
7 from leaded gasoline usage continues to move slowly through systems along with more recently
8 deposited Pb and Pb derived from non air sources. The results from the location-specific case
9 studies and the surface and sediment screen performed in the last review are difficult to interpret
10 in light of the current standard and are not largely useful in informing our judgments of the
11 potential for adverse effects at levels of deposition meeting the current standard. Under such
12 constraints it is difficult to assess the merit of the risk findings from the previous review.

1 5.3 REFERENCES

- 2 Buekers, J; Redeker, ES; Smolders, E. (2009). Lead toxicity to wildlife: Derivation of a critical blood
3 concentration for wildlife monitoring based on literature data [Review]. *Sci Total Environ*
4 407: 3431-3438. <http://dx.doi.org/10.1016/j.scitotenv.2009.01.044>
- 5 Frey, C; Samet, JM. (2011). CASAC review of the EPA's integrated science assessment for lead
6 (first external review draft- May 2011). (EPA-CASAC-12-002). Washington, DC: U.S.
7 Environmental Protection Agency, Clean Air Scientific Advisory Committee.
- 8 Henderson, R. (2007a) Letter from Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory
9 Committee, to Administrator Stephen L. Johnson. Re: Clean Air Scientific Advisory
10 Committee's (CASAC) Review of the 1st Draft Lead Staff Paper and Draft Lead Exposure
11 and Risk Assessments. March 27, 2007.
- 12 Henderson, R. (2007b) Letter from Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory
13 Committee, to Administrator Stephen L. Johnson. Re: Clean Air Scientific Advisory
14 Committee's (CASAC) Review of the 2nd Draft Lead Human Exposure and Health Risk
15 Assessments. September 27, 2007.
- 16 ICF International. (2006) Lead Human Exposure and Health Risk Assessments and Ecological Risk
17 Assessment for Selected Areas. Pilot Phase. Draft Technical Report. Prepared for the U.S.
18 EPA's Office of Air Quality Planning and Standards, Research Triangle Park, NC.
19 December.
- 20 MacDonald, D.D., Ingersoll, C.G., and Berger, T.A. (2000) Development and evaluation of
21 consensus-based sediment quality guidelines for freshwater ecosystems. *Archives of*
22 *Environmental Contamination and Toxicology*. 39:20-31.
- 23
24 MacDonald, D.D., Ingersoll, C.G., Smorong, D.E., Lindskoog, R.A., Sloane, G., and Biernacki, T.
25 (2003) Development and Evaluation of Numerical Sediment Quality Assessment Guidelines
26 for Florida Inland Waters. British Columbia: MacDonald Environmental Sciences, Lt.
27 Columbia, MO: U.S. Geological Survey. Prepared for: Florida Department of Environmental
28 Protection, Tallahassee, FL. January.
- 29 U.S. Environmental Protection Agency. (2005a) Guidance for Developing Ecological Soil Screening
30 Levels. Washington, DC: Office of Solid Waste and Emergency Response. OSWER Directive
31 9285.7-55. November.
- 32 U.S. Environmental Protection Agency. (2005b) Ecological Soil Screening Levels for Lead, Interim
33 Final. Washington, DC: Office of Solid Waste and Emergency Response. OSWER Directive
34 9285.7-70. Available at http://www.epa.gov/ecotox/ecossl/pdf/eco-ssl_lead.pdf.
- 35 U.S. Environmental Protection Agency. (2006a) Air Quality Criteria for Lead. Washington, DC,
36 EPA/600/R-5/144aF. Available online at:
37 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_cr.html
- 38 U.S. Environmental Protection Agency. (2006b) Analysis Plan for Human Health and Ecological

- 1 Risk Assessment for the Review of the Lead National Ambient Air Quality Standards.
2 Office of Air Quality Planning and Standards, Research Triangle Park, NC. Available at:
3 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_cr_pd.html
- 4 U.S. Environmental Protection Agency. (2007a). Framework for metals risk assessment [EPA
5 Report]. (EPA 120/R-07/001). Washington, D.C. Available online at:
6 <http://www.epa.gov/raf/metalsframework/index.htm>
- 7 U.S. Environmental Protection Agency. (2007b). Review of the National Ambient Air Quality
8 Standards for Lead: Policy Assessment of Scientific and Technical Information OAQPS Staff
9 Paper. Washington, DC, EPA-452/R-07-013. Available online at:
10 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_cr.html
- 11 U.S. Environmental Protection Agency. (2012) Integrated Science Assessment for Lead (Third
12 External Review Draft). Washington, DC, EPA/600/R-10/075C. Available online at:
13 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_index.html

6 REVIEW OF THE SECONDARY STANDARD FOR LEAD

This chapter presents preliminary staff conclusions regarding the secondary Pb standard. These preliminary staff conclusions are guided by consideration of key policy-relevant questions and based on the assessment and integrative synthesis of information presented in the ISA and by staff analyses and evaluations presented in chapters 2 and 5 herein. Final evaluations and staff conclusions, to be developed taking into consideration CASAC advice and public comment on this draft document, will be presented in the final PA. The final evaluations and staff conclusions will inform the Administrator's decisions on whether to retain or revise the existing secondary standard for Pb.

Following an introductory section on the general approach for reviewing the secondary standard (section 6.1), including a summary of considerations in the last review, the discussion in this chapter focuses on the central issue of whether the information available in this review supports or calls into question the adequacy of the current secondary standard. Building on the responses to specific policy-relevant questions on the scientific evidence and exposure-risk information in chapter 5 above, presentation in section 6.2 is also organized into consideration of key policy-relevant questions framing evidence-based and exposure/risk-based considerations. The policy-relevant questions in this document are based on those included in the IRP (IRP, section 3.2). In section 6.3, preliminary staff conclusions are developed. Section 6.4 presents a brief overview of key uncertainties and areas for future research.

6.1 APPROACH

Staff's approach for reviewing the current secondary standard, which involves translating scientific and technical information into the basis for addressing key policy-relevant questions, takes into consideration the approaches used in the previous Pb NAAQS review. The past and current approaches described below are all based most fundamentally on using EPA's assessment of the current scientific evidence and previous quantitative analyses to inform the Administrator's judgment with regard to the secondary standard for Pb.

Staff final conclusions will be developed for the next version of this document with consideration of CASAC advice and public comment. In drawing conclusions regarding the secondary standard for the Administrator's consideration, we note that the final decision on the adequacy of the current secondary Pb standard is largely a public welfare policy judgment to be made by the Administrator. The Administrator's final decision draws upon scientific information and analyses about welfare effects, exposure and risks, as well as judgments about the appropriate response to the range of uncertainties that are inherent in the scientific evidence and analyses. This approach is consistent with the requirements of the NAAQS provisions of the

1 Act. Section 109 of the Clean Air Act requires the Administrator to establish a secondary
2 standard that, in the judgment of the Administrator, is “requisite to protect the public welfare
3 from any known or anticipated adverse effects associated with the presence of the pollutant in
4 the ambient air”. In so doing, the Administrator seeks to establish standards that are neither
5 more nor less stringent than necessary for this purpose.

6 **6.1.1 Approach Used in the Last Review**

7 The current secondary standard for Pb is equal to the primary standard for human health.
8 As summarized in section 1.2.2 and described in more detail in section 4.1.1, the primary
9 standard was substantially revised in the last review based on the much-expanded health effects
10 evidence of neurocognitive effects of Pb in children. The level of the revised NAAQS is 0.15
11 $\mu\text{g}/\text{m}^3$. The averaging time was also revised to a rolling three-month period with a maximum
12 (not-to-be-exceeded) form, evaluated over a three-year period. Compared to the previous
13 averaging time of one calendar quarter, this revision was considered to be more scientifically
14 appropriate and more protective for human health. The indicator of Pb-TSP was retained,
15 reflecting the evidence that Pb particles of all sizes pose health risks (73 FR 67007).

16 The scientific assessment for the last review is described in the 2006 Air Quality Criteria
17 for Lead (AQCD; USEPA, 2006b), multiple drafts of which received review by CASAC and the
18 public. EPA also conducted a pilot ecological risk assessment for the review, after consultation
19 with CASAC and receiving public comment on a draft analysis plan (USEPA, 2006c). The pilot
20 ecological risk assessment was released in December 2006 (ICF, 2006) and the policy
21 assessment based on these assessments, air quality analyses and key evidence from the AQCD
22 was presented in the Staff Paper (USEPA, 2007b), a draft of which also received CASAC and
23 public review. The final Staff Paper presented OAQPS staff’s evaluation of the public health and
24 welfare policy implications of the key studies and scientific information contained in the 2006
25 CD and presented and interpreted results from the pilot risk/exposure analyses conducted for this
26 review. Based on this evaluation, the Staff Paper presented OAQPS staff recommendations that
27 the Administrator give consideration to substantially revising the primary and secondary
28 standards to a range of levels at or below 0.2 $\mu\text{g}/\text{m}^3$.

29 In the previous review, the Administrator considered the evidence-based information
30 presented in the CD and the risk-based information presented in the 2006 REA. It was his
31 conclusion that the available data and evidence, while primarily qualitative, suggested that there
32 was the potential for adverse environmental impacts under the then-current standard. Given the
33 limited data on Pb effects in ecosystems, it was at the time, as it is now, necessary to look at
34 evidence of Pb effects on organisms and extrapolate to ecosystem effects. Therefore, taking into
35 account the available evidence and current media concentrations in a wide range of areas, the

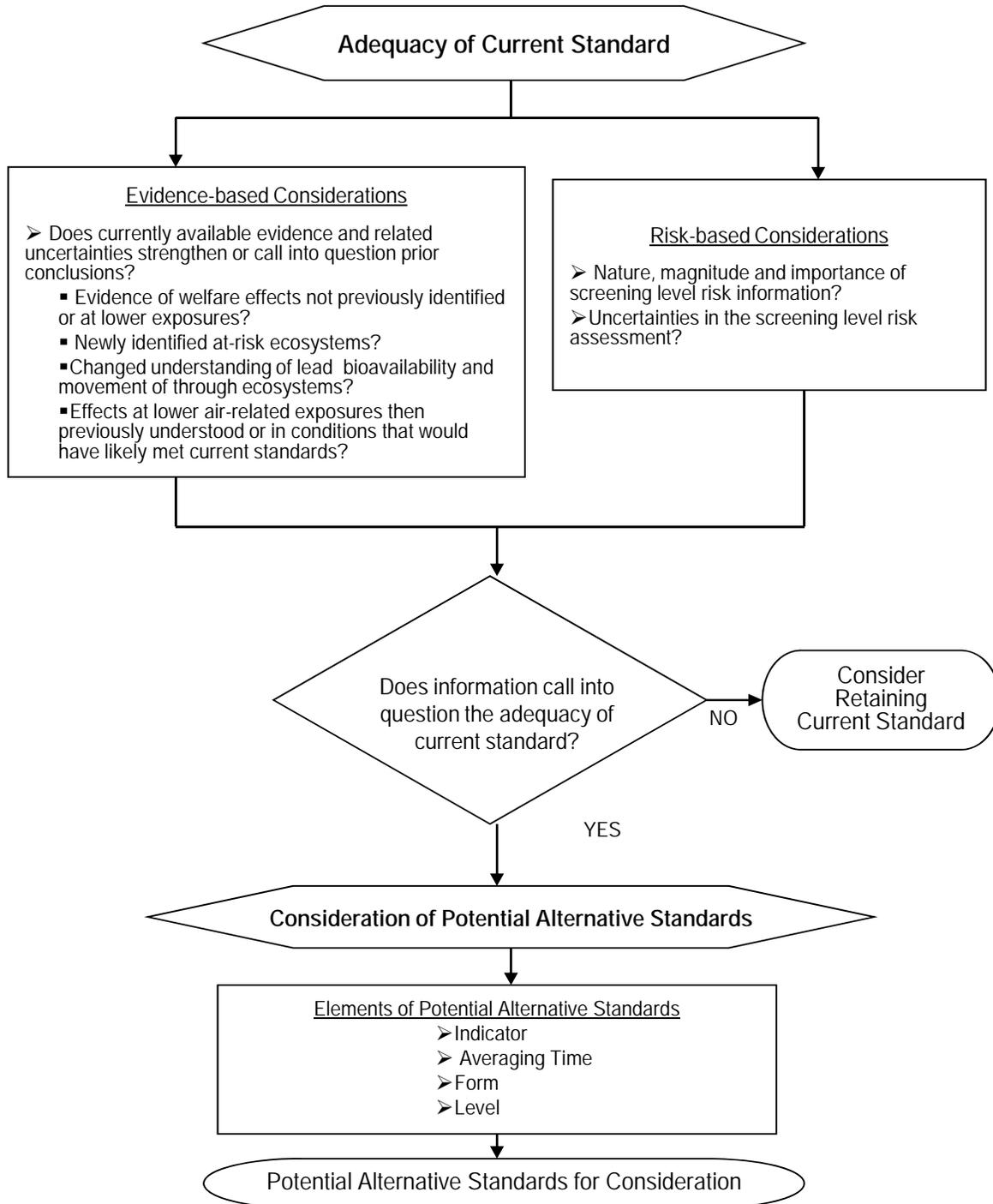
1 Administrator concluded that there was potential for adverse effects occurring under the then-
2 current standard; however there was insufficient data to provide a quantitative basis for setting a
3 secondary standard different than the primary (73 FR 67011). Therefore, citing a general lack of
4 data that would indicate the appropriate level of Pb in environmental media that may be
5 associated with adverse effects, as well as the comments of the CASAC Pb panel that a
6 significant change to current air concentrations (e.g., via a significant change to the standard)
7 was likely to have significant beneficial effects on the magnitude of Pb exposures in the
8 environment, the secondary standard was revised to be consistent with the revised primary
9 standard (73 FR 67011).

10 **6.1.2 Approach for the Current Review**

11 In evaluating whether it is appropriate to consider retaining the current secondary Pb
12 standard, or whether consideration of revision is appropriate, we have adopted an approach in
13 this review that builds on the general approach from the last review and reflects the body of
14 evidence and information now available. As summarized above, the Administrator's decisions in
15 the previous review were based on the conclusion that there was the potential for adverse
16 ecological effects under the previous standard. In our approach here, we intend to focus on
17 consideration of the extent to which a broader body of scientific evidence is now available that
18 would inform decisions on either the potential for adverse effects to ecosystems under the current
19 standard or the ability to set a more ecologically relevant secondary standard than was feasible in
20 the previous review. In conducting this assessment, we draw on the ecological effects evidence
21 presented in detail in the ISA, and aspects summarized above in chapter 5, along with the
22 information associated with the screening-level risk assessment also summarized above. Figure
23 6-1 illustrates the basic construct of our approach in developing conclusions regarding options
24 appropriate for the Administrator to consider in this review with regard to the adequacy of the
25 current secondary NAAQS standard and, as appropriate, potential alternate standards.

26 In developing preliminary conclusions in this review, we have taken into account both
27 evidence-based and risk-based considerations framed by a series of policy-relevant questions.
28 These questions are outlined in the sections below and generally discuss the extent to which we
29 are able to better characterize effects and the likelihood of adverse effects in the environment
30 under the current standard. Our approach to considering these questions recognizes that the
31 available welfare effects evidence generally reflects laboratory-based evidence of toxicological
32 effects on specific organisms exposed to concentrations of Pb at which scientists generally agree
33 that adverse effects are likely to occur. It is widely recognized, however, that environmental
34 exposures from atmospherically-derived Pb are likely to be lower and/or accompanied by

- 1 significant confounding and modifying factors (e.g., other metals, acidification), which increases
- 2 our uncertainty about the likelihood and magnitude of organism and ecosystem responses.



3 **Figure 6-1. Overview of approach for review of current secondary standard.**

1 **6.2 ADEQUACY OF THE CURRENT STANDARD**

2 In considering the adequacy of the current Pb standard, the overarching question we
3 consider is:

- 4 • **Does the currently available scientific evidence- and exposure/risk-based**
5 **information, as reflected in the ISA and REA, support or call into question the**
6 **adequacy of the protection afforded by the current Pb standard?**

7 To assist us in interpreting the currently available scientific evidence and screening-level
8 risk information to address this question, we have focused on a series of more specific questions,
9 posed within sections 6.2.1 and 6.2.2 below. In so doing, we consider both the information
10 available at the time of the last review and information newly available since the last review
11 which has been critically analyzed and characterized in the ISA.

12 **6.2.1 Evidence-based Considerations**

13 In considering the welfare effects evidence with respect to the adequacy of the current
14 standard, we must consider the array of evidence newly assessed in the ISA and the degree to
15 which the newly available evidence supports previous conclusions about the effects of Pb in the
16 environment and the extent to which it reduces areas of uncertainty recognized in decisions on
17 the secondary standard in the last review. Additionally, as the secondary standard set in 2008 is
18 significantly lower than the previous standard, which was the focus of evaluation in the last
19 review, we must consider the extent to which the current evidence supports conclusions about
20 the effects of current ambient levels under the current standard and the extent to which the
21 evidence supports or calls into question the adequacy of protection afforded by the current
22 standard.

- 23 • **To what extent does the available information indicate that Pb-related effects are**
24 **occurring as a result of multimedia pathways associated with ambient air**
25 **conditions that would meet the current standard?**

26 The current evidence continues to support our conclusions from the previous review
27 regarding key aspects of the ecological effects evidence for Pb and the effects of exposure
28 associated with levels of Pb occurring through ecological media in the U.S. Our conclusions in
29 this regard are based on consideration of the assessment of the currently available evidence in the
30 ISA, particularly with regard to key aspects summarized in chapter 5 of this PA, in light of the
31 assessment of the evidence in the last review as described in the 2006 CD and summarized in the
32 notice of final rulemaking (73 FR 67008). Key aspects of these previous conclusions are
33 summarized below.

- 34 • Deposition of gasoline-derived Pb into forest soils has produced a legacy of slow
35 moving Pb that remains bound to organic materials despite the removal of Pb from

1 most fuels and the resulting dramatic reductions in overall deposition rates (2006 CD,
2 Section AX7.1.4.3).

- 3 • For areas influenced by point sources of air Pb that meet the standard considered in the
4 previous review, concentrations of Pb in soil may exceed by many orders of magnitude
5 the concentrations which are considered harmful to laboratory organisms (2006 CD,
6 Section 3.2 and AX7.1.2.3).
- 7 • Some Pb deposited before the previous standard was enacted is still present in soils and
8 sediments; historic Pb from gasoline continues to move slowly through systems as does
9 current Pb derived from both air and nonair sources.
- 10 • The evidence of adversity in natural systems is very sparse due in no small part to the
11 difficulty in determining the effects of confounding factors such as multiple metals or
12 factors influencing bioavailability in field studies.
- 13 • Environmental conditions exist in which Pb-associated adverse effects to aquatic
14 organisms and thereby ecosystems may be anticipated given experimental results.
- 15 • Some areas with long term historical deposition of Pb to sediment from a variety of
16 sources as well as areas influenced by point sources have the potential for adverse
17 effects to aquatic communities (2006 CD, Sections AX7.2.2.2.2 and AX7.2.4).

18 The range of effects of Pb on terrestrial and aquatic organisms based on new information
19 in the current review are summarized in the ISA (ISA, section 2.7 and section 7.3 and 7.4) and
20 largely mirror the conclusions presented above from the previous review.

21 The integrated synthesis contained in the ISA conveys how effects of Pb can vary with
22 species and life stage, duration of exposure, form of Pb, and media characteristics such as soil
23 and water chemistry. A wide range of organism effects are recognized, including effects on
24 growth, development (particularly of the neurological system) and reproductive success (ISA,
25 section 7.3 and section 7.4). Lead is recognized to distribute from the air into multiple
26 environmental media, as summarized in section 1.3 above, contributing to multiple exposure
27 pathways for ecological receptors. As discussed in section 5.1 above, many factors affect the
28 bioavailability of Pb to receptors in terrestrial and aquatic ecosystems, contributing to differences
29 between laboratory-assessed toxicity and Pb toxicity in these ecosystems, challenging our
30 consideration of environmental impacts of Pb emitted to ambient air.

31 In studies in a variety of ecosystems, adverse ecosystem-level effects (including
32 decreases in species diversity, loss of vegetation, changes to community composition, decreased
33 growth of vegetation, and increased number of invasive species) have been demonstrated near
34 smelters, mines and other industries that have released Pb, among other materials (ISA, section
35 7.3.12 and 7.4.12). As noted in section 5.1 above, however, our ability to characterize the role of
36 air emissions of Pb in contributing to these effects is complicated because of coincident releases
37 to other media and of other pollutants. Co-released pollutants include a variety of other heavy
38 metals, in addition to sulfur dioxide, which may cause toxic effects in themselves and may

1 interact with Pb in the environment. There is also some uncertainty as to the extent to which Pb
2 in ambient air is contributing to the exposure. These uncertainties limit our ability to draw
3 conclusions regarding the extent to which Pb-related effects may be associated with ambient air
4 conditions that would meet the current standard.

5 The role of historically emitted Pb, as discussed in section 1.3.2 above, poses additional
6 complications in addressing this question. The vast majority of Pb in the environment today,
7 particularly in terrestrial ecosystems, was deposited in the past during the use of Pb additives in
8 gasoline (USEPA 2007b, section 6.2.1) although industrial coal use has also been documented as
9 a significant source as well (ISA, section 3.2.1). This gasoline-derived Pb was emitted in very
10 large quantities (CD, pl AX7-98 and ISA, Figure 3-8) and predominantly in small sized particles
11 which were widely dispersed and transported across large distances (ISA, section 3.2). As
12 recognized in section 2.3.1 and 2.3.3.2 above, historical records provided by sediment cores in
13 various environments document the substantially reduced Pb deposition (associated with reduced
14 Pb emissions) in many locations (ISA, section 3.2.1). As Pb is extremely persistent in the
15 environment, past environmental releases are expected to generally dominate current media
16 concentrations. There is limited evidence to relate specific ecosystem effects with current
17 ambient air concentrations of Pb through deposition to terrestrial and aquatic ecosystems and
18 subsequent movement of deposited Pb through the environment (e.g., soil, sediment, water,
19 organisms). The potential for ecosystem effects of Pb from atmospheric sources under conditions
20 meeting the current standard is difficult to assess due to limitations on available information not
21 only on bioavailability (as affected by the specific characteristics of the receiving ecosystem),
22 but also information to fully characterize the distribution of Pb from the atmosphere into
23 ecosystems in long-term exposure scenarios. Therefore, the connection between air concentration
24 and ecosystem exposure and associated potential for welfare effects continues to be poorly
25 characterized for Pb (ISA, Section 7.5), and is even harder to characterize with respect to the
26 current standard than it was in last review with respect to the previous standard.

27 • **To what extent have important uncertainties identified in the last review been**
28 **reduced and/or have new uncertainties emerged?**

29 With regard to uncertainties in effects associated with Pb in the ambient air, the last
30 review illustrated the largest of these uncertainties were the ability to determine the prevalence of
31 effects in the natural environment, the ability to apportion effects between air and nonair sources,
32 and the understanding of the effects in the context of the current standard (IRP, section 3.2).
33 While we have some new evidence with regard to bioavailability and its role in determining
34 effects, overall these remain the major sources of uncertainty in evaluating effects and exposure
35 evidence for Pb in ecosystems.

1 With regard to the prevalence of effects in the natural environment, it remains difficult to
2 assess the concentration at which Pb elicits a specific effect in terrestrial and aquatic systems,
3 due to the influence of other environmental variables on both Pb bioavailability and toxicity, and
4 also due to substantial species differences in Pb susceptibility. There is little new information
5 that would facilitate extrapolation of evidence on individual species, generally from controlled
6 laboratory studies, to conclusions regarding adversity to populations or ecosystems within the
7 natural environment. For example, in terrestrial systems, evidence reviewed in the ISA (ISA,
8 Sections 7.2.3 and 7.2.4) demonstrates that exposure to Pb is generally associated with negative
9 effects on growth, reproduction and survival in terrestrial ecosystems. Many factors, including
10 species composition and various soil physiochemical properties, interact strongly with Pb
11 concentration to modify effects. Also, Pb aging is a particularly important factor in terrestrial
12 systems, where soil is the main route of exposure, and it is difficult to reproduce experimentally.
13 Without quantitative characterization of those interactions, characterizations of exposure-
14 response relationships would likely not be transferable outside of experimental settings (ISA,
15 Section 2.7.1).

16 As reported in the ISA, there continues to be limited evidence linking current ambient
17 air sources of Pb through deposition to environmental media (e.g., soil, sediment, water, and
18 biota) and to ecological effects (ISA, section 7.3.11). The ability to apportion effects between air
19 deposited Pb and nonair sources is difficult to assess due to a lack of information not only on the
20 bioavailability of that Pb, which is greatly influenced by the specific characteristics of the
21 receiving ecosystem, but also on Pb distribution in ecosystems in long-term exposure scenarios.

22 Consideration of the environmental risks associated with the current standard is
23 complicated by the environmental burden associated with air Pb concentrations, predominantly
24 in the past, that exceeded the current standard (USEPA, 2007b, section 6.4.4.2). For example, a
25 large portion of Pb deposited before the standard was enacted is likely still present in soils and
26 sediments and historic Pb from gasoline continues to move slowly through systems as does
27 current Pb derived from both air and nonair sources. As a general matter, the currently available
28 evidence, as assessed in the ISA, does not significantly reduce any of these areas of uncertainty
29 or inform our consideration of the adequacy of the current standard.

30

6.2.2 Exposure/Risk-based Considerations

- **To what extent does risk or exposure information indicate that air-related ecosystem exposures important from a public welfare perspective are likely to occur with air Pb levels that just meet the current standard?**

The current evidence continues to support our conclusions with regard to interpreting the risk and exposure results from the previous review. Our conclusions in this regard are based on consideration of the screening-level ecological risk assessment results from the previous review in light of the currently available evidence in the ISA and as described in the 2006 REA and summarized in the notice of final rulemaking (73 FR 67009). Key aspects of these results in the context of the current standard are summarized below, drawing from the discussion of the 2006 REA results in section 5.2.2 above, which is based on the information in the 2006 REA (ICF, 2006).

Primary Pb Smelter Case Study

- While the contribution to Pb concentrations from air as compared to nonair sources is not quantified, air emissions from this facility are substantial (ICF 2006). Currently, the county where this facility is located exceeds the now current Pb NAAQS.

Secondary Pb Smelter Case Study

- While the contribution from air deposited Pb to the overall Pb concentrations modeled in soils at this location is unclear, the facility continues to emit Pb and the county where this facility is located exceeds the now current Pb NAAQS (Appendix 5-A).

Near Roadway Non-urban Case Study

- These case study locations are highly impacted by past deposition of gasoline Pb. It is unknown whether current conditions at these sites exceed the now current Pb standard but given evidence from the past of Pb concentrations near highways that ranged above the previous (1978) Pb standard (1986 CD, section 7.2.1), conditions at these locations during the time of leaded gasoline very likely exceeded the 1978 standard.

Vulnerable Ecosystem Case Study

- The previous review concluded that atmospheric Pb inputs do not directly affect stream Pb levels at Hubbard Brook Experimental Forest because deposited Pb is almost entirely retained in the soil profile. It is unlikely that conditions have changed from the previous conclusions made based on soil data through 2000 and therefore, current ambient air concentrations likely do not directly impact stream Pb levels under air quality conditions associated with meeting the now current standard.

National-scale Surface Water and Sediment Screen

- The extent to which past air emissions of Pb have contributed to surface water or sediment Pb concentrations at the locations identified in the screen is unclear. For some of the surface water locations, nonair sources likely contributed significantly to

1 the surface water Pb concentrations. For other locations, a lack of nearby nonair
2 sources indicated a potential role for air sources to contribute to observed surface water
3 Pb concentrations. Additionally, these concentrations may have been influenced by Pb
4 in resuspended sediments and/or reflect contribution of Pb from erosion of soils with
5 Pb derived from historic as well as current air emissions.

6
7 Although the available risk and exposure information continues to be sufficient to
8 conclude that the 1978 standard was not providing adequate protection to ecosystems, that
9 information, when considered with regard to air-related ecosystem exposures likely to occur with
10 air Pb levels that just meet the now current standard, does not provide evidence that the current
11 secondary standard is inadequate.

12 **6.3 PRELIMINARY STAFF CONCLUSIONS ON THE SECONDARY STANDARD**

13 This section describes preliminary staff conclusions regarding adequacy of the current
14 secondary Pb standard. These conclusions are based on considerations described above and in
15 the discussion below regarding the currently available scientific evidence summarized in the
16 third draft ISA and prior CDs and the risk and exposure information drawn from the 2006 REA.
17 The final PA will draw upon the final ISA, developed after CASAC review and public comment
18 on the third draft ISA. Staff conclusions presented in the final PA will take into account advice
19 from CASAC and public comment on this document and these draft conclusions.

20 Taking into consideration the discussions responding to specific questions above in this
21 and the prior chapter, this section addresses the following overarching policy question:

- 22 • **Does the currently available scientific evidence- and exposure/risk-based**
23 **information, as reflected in the ISA and REA, support or call into question the**
24 **adequacy of the protection afforded by the current secondary Pb standard?**

25 With respect to evidence-based considerations, staff gives weight to the body of evidence
26 on the ecological effects of Pb in the natural environment, expanded in some aspects since the
27 last review, which continues to support identification of ecological effects in organisms relating
28 to growth, reproduction, and survival as the most relevant endpoints associated with Pb
29 exposure. The currently available evidence, while somewhat expanded since the last review,
30 does not include evidence of significant effects at lower concentrations or evidence of higher
31 level ecosystem effects beyond those reported in the last review. There continues to be
32 significant difficulties in interpreting effects evidence from laboratory studies to the natural
33 environment and linking those effects to ambient air Pb concentrations. Further, we are aware of
34 no new critical loads information that would inform our interpretation of the public welfare
35 significance of the effects of Pb in various ecosystems. In summary, while new research has
36 added to the understanding of Pb biogeochemistry and expanded the list of organisms for which

1 Pb effects have been described, there remains a significant lack of knowledge about the potential
2 for adverse effects from ambient air Pb in the environment and the exposures that occur from
3 such air-derived Pb, particularly under the current standard.

4 With respect to exposure/risk-based considerations, we recognize the complexity of
5 interpreting the previous risk assessment with respect to ambient air Pb under the current
6 standard and the associated limitations and uncertainties of such assessments. For example, the
7 location-specific case studies as well as the national screen reflect both current air Pb deposition
8 as well as past air and nonair source contributions. We conclude that while the previous
9 assessment is consistent with and generally supportive of the evidence-based conclusions about
10 Pb in the environment the limitations of apportioning Pb between past and present air
11 contributions and between air and nonair sources remain significant.

12 In making the decision on revision of the secondary standard in the last review, the
13 Administrator concurred with the conclusions by CASAC that we lack the relevant data to
14 provide a clear quantitative basis for setting a secondary Pb NAAQS that differs from the
15 primary in indicator, averaging time, level or form. As a result, the Administrator revised the
16 secondary standard to be identical in all respects to the primary standard (73 FR 67012).

17 In now considering the adequacy of the current standard in this review, we have
18 considered evidence assessed in the ISA and discussed in sections 5.1, 2.3 and 6.2.1 above, as
19 well as information gained from the 2006 REA discussed in sections 5.2 and 6.2.2 above. As
20 summarized in section 6.2.1 above, the scientific evidence presented in detail in the ISA,
21 inclusive of that newly available in this review, is not substantively changed, most particularly
22 with regard to our needs in reviewing the current standard, from the information that was
23 available in the last review. Further, our updated consideration of the screening-level risk
24 information from the 2006 REA, as summarized in section 6.2.2 above, additionally does not
25 provide alternative conclusions with regard to a basis for judging the adequacy of the current
26 standard. Thus, while the current secondary standard is not expressed in elements derived from
27 ecological effects evidence, we preliminarily conclude that the currently available information
28 does not call into question the adequacy of the current standard to provide the requisite
29 protection for public welfare. In addition, as in the last review, there continues to be insufficient
30 data to provide a quantitative basis for setting a secondary standard different than the primary
31 standard. Accordingly, we reach the preliminary conclusion that it is appropriate to consider
32 retaining the current standard without revision. This preliminary conclusion is based on
33 consideration of the welfare effects evidence in combination with the screening-level
34 exposure/risk information (chapter 5 and sections 6.2.1 and 6.2.2 above) and staff judgments on
35 the associated public welfare implications.

6.4 KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH AND DATA COLLECTION

In this section, we highlight key uncertainties associated with reviewing the secondary NAAQS for Pb. Such key uncertainties and recommendations for ecosystem-related research, model development, and data gathering are outlined below. In some cases, research in these areas can go beyond aiding standard setting to aiding in the development of more efficient and effective control strategies. We note, however, that a full set of research recommendations to meet standards implementation and strategy development needs is beyond the scope of this discussion. Rather, listed below are key uncertainties and research questions and data gaps that have been thus far highlighted in this review of the health-based primary standard.

- An important source of uncertainty in characterizing the effects of air related Pb in the environment is the ability to apportion the sources of Pb to specific ecosystems.
 - It is difficult to identify the atmospheric component of Pb deposited in ecosystems from other sources of Pb. Without such information, it is not possible to determine the appropriately protective air concentrations of Pb.
 - Development of critical loads information for various ecosystems would allow for a much improved understanding of the significance of air contributions to ecosystems.
- There is significant uncertainty regarding the timeframes under which Pb currently in the environment was deposited.
 - In general, the connection between air concentration of Pb and ecosystem exposure continues to be poorly characterized.
 - There is uncertainty in characterizing the fate and transport of Pb in the environment through air, water, and soil media, especially in ecosystems. This limits our ability to account for past Pb exposures in current media concentrations and to link effects to current air contributions.
- Our understanding of the degree of adverse impact that is occurring in the environment due to Pb is incomplete and leads to uncertainty in interpreting the significance of effects at specific endpoints.
 - Critical loads are important to capture the variability of response across ecosystems to determine which ecosystems are most sensitive and additional research is needed to provide inputs for development of critical loads, particularly for a broader range of ecosystem level endpoints.
 - The available studies on community and ecosystem-level effects are sparse and usually from contaminated areas where Pb concentrations are much higher than typically encountered in the environment.
 - The role of modifying factors on Pb toxicity continues to be a source of uncertainty when determining the degree of adverse impact from Pb.

- 1 – Evidence regarding relationships between organism-level effects of Pb (e.g.,
2 reproduction, growth, and survival) and effects at the population level and higher,
3 is lacking especially in natural systems.
- 4 – Better indicators of Pb effects are needed in estuarine and marine systems to allow
5 us to understand the potential for adverse effects from Pb in these environments.
6

1 **6.5 REFERENCES**

- 2 Frey, C; Samet, JM. (2011). CASAC review of the EPA's integrated science assessment for lead
3 (first external review draft- May 2011). (EPA-CASAC-12-002). Washington, DC: U.S.
4 Environmental Protection Agency, Clean Air Scientific Advisory Committee.
- 5 Henderson, R. (2007a) Letter from Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory
6 Committee, to Administrator Stephen L. Johnson. Re: Clean Air Scientific Advisory
7 Committee's (CASAC) Review of the 1st Draft Lead Staff Paper and Draft Lead
8 Exposure and Risk Assessments. March 27, 2007.
- 9 Henderson, R. (2007b) Letter from Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory
10 Committee, to Administrator Stephen L. Johnson. Re: Clean Air Scientific Advisory
11 Committee's (CASAC) Review of the 2nd Draft Lead Human Exposure and Health Risk
12 Assessments. September 27, 2007.
- 13 ICF International. (2006) Lead Human Exposure and Health Risk Assessments and Ecological
14 Risk Assessment for Selected Areas. Pilot Phase. Draft Technical Report. Prepared for
15 the U.S. EPA's Office of Air Quality Planning and Standards, Research Triangle Park,
16 NC. December.
- 17 U.S. Environmental Protection Agency. (2006) Air Quality Criteria for Lead. Washington, DC,
18 EPA/600/R-5/144aF. Available online at:
19 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_cr.html
- 20 U.S. Environmental Protection Agency. (2012) Integrated Science Assessment for Lead (Third
21 External Review Draft). Washington, DC, EPA/600/R-10/075C. Available online at:
22 http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_index.html

APPENDICES

Appendix 2A. The 2008 NEI: Data Sources, Limitations and Confidence	2A-1
Appendix 2B. Recent Regulatory Actions on Stationary Sources of Lead	2B-1
Appendix 2C. Criteria for Air Quality Data Analysis	2C-1
Appendix 2D. Air Quality Data Analysis Summary	2D-1
Appendix 3A. Interpolated Risk Estimates for the Generalized (Local) Urban Case Study..	3A-1
Appendix 5A. Additional Detail on 2006 Ecological Screening Assessment	5A-1

APPENDIX 2A

THE 2008 NEI: DATA SOURCES, LIMITATIONS AND CONFIDENCE

The process of identifying sources that emit Pb into the air has been ongoing since before the Clean Air Act of 1970. The comprehensiveness of emission inventories generally, and the NEI, specifically, depends upon knowledge of source types emit Pb, their locations and their operating characteristics, as well as the reporting of this information to the inventory. As noted above, the NEI relies on information that is available from a variety of sources for this information. There are numerous steps, each with its own uncertainties, associated with the development of this information for use in the emissions inventory. First, the categories emitting Pb must be identified. Second, the sources' processes and control devices must be known. Third, the activity throughputs and operating schedules of these sources must be known. Finally, we must have emission factors to relate emissions to the operating throughputs, process conditions and control devices. The process, control device, throughputs and operating schedules are generally available for each source. However, the emission factors represent average emissions for a source type and average emissions may differ significantly from source to source. In some cases, emissions testing provides source-specific information. In others, emissions factors must be estimated from similar sources or source categories, or from other information. More information on emission factors and the estimation of emissions is found in the introduction to EPA's Compilation of Air Pollutant Emissions Factors.¹

The Pb emissions information presented in chapter 2 is drawn largely from EPA's NEI for 2008. The NEI is based on information submitted from State, Tribal and local air pollution agencies and data obtained during the preparation of technical support information for EPA's hazardous air pollutant regulatory programs. Data in the 2008 NEI for Pb emissions from the use of leaded aviation gasoline were developed by EPA using the Federal Aviation Administration's operations activity data, where available.² The data were then reviewed by State, Tribal, and local air pollution agencies. With some additions, the information presented in this document is primarily based on version 3 of the NEI for 2008, available on the EPA's CHIEF website at (<http://www.epa.gov/ttn/chief/net/2008inventory.html>). The NEI is limited

¹ U.S. Environmental Protection Agency. (1996-2011) . AP-42, Compilation of Air Pollutant Emission Factors, 5th Edition. Volume 1: Stationary Point and Area Sources, Chapter 13: Miscellaneous Sources. Available at: <http://www.epa.gov/ttn/chief/ap42/ch13/index.html>. Further information on emission factors is available at: <http://www.epa.gov/ttn/chief/ap42/>

² Eastern Research Group (ERG), 2011a. Project report: Documentation for Aircraft Component of the National Emissions Inventory Methodology, ERG No. 0245.03.402.011, January 27, 2011.

1 with regard to Pb emissions estimates for some sources. For example, we have not yet
2 developed estimates for the NEI of Pb emissions for the miscellaneous categories of on-road
3 emissions (e.g., combustion of fuel with Pb traces, lubricating oil, mechanical wear of vehicle
4 components, etc.) and Pb that may be emitted from wildfires, or for emissions associated with
5 resuspension of Pb residing in roadway dust and nearby surface soil in areas not otherwise
6 associated with industrial facilities (see section 2.1.2).

7 The 2008 NEI underwent extensive external review, including a review of the process for
8 developing the inventory which includes extensive quality assurance and quality control steps
9 (QA/QC). We provided feedback reports to point source data providers and we posted several
10 versions of the inventory on our website. We also conducted additional QA targeted at facilities
11 with appreciable Pb emissions in previous years to ensure that 2008 Pb emissions were reported,
12 and we augmented with TRI where facilities with annual emissions greater than 0.5 tpy in 2005
13 were not reported to NEI in 2008 due to Pb emissions reporting thresholds
14 (ftp://ftp.epa.gov/EmisInventory/20008v2/doc/2008neiv2_issues.xlsx). Further, there was
15 additional QA/QC conducted for emission inventory information for facilities that are included
16 in Risk and Technology Review source categories.³ As a result we have strong confidence in the
17 quality of the data for these facilities. In summary, generic limitations to the 2008 NEI include
18 the following.

- 19 • Consistency: The 2008 NEI for Pb is a composite of emissions estimates generated by
20 state and local regulatory agencies, industry, and EPA. Because the estimates
21 originated from a variety of sources, as well as for differing purposes, they will in turn
22 vary in quality, whether Pb is reported for particular source types, method of reporting
23 compound classes, level of detail, and geographic coverage.
- 24 • Variability in Quality and Accuracy of Emission Estimation Methods: The accuracy of
25 emission estimation techniques varies with pollutants and source categories. In some
26 cases, an estimate may be based on a few or only one emission measurement at a
27 similar source. The techniques used and quality of the estimates will vary between
28 source categories and between area, major, and mobile source sectors. Generally, the
29 more review and scrutiny given to emissions data by states and other agencies, the
30 more certainty and accuracy there is in those estimates.

31
32

³ The Risk and Technology Review is a combined effort to evaluate both risk and technology as required by the Clean Air Act (CAA) after the application of maximum achievable control technology (MACT) standards. Section 112(f)(2) of the CAA directs EPA to conduct risk assessments on each source category subject to MACT standards, and to determine if additional standards are needed to reduce residual risks. Section 112(d)(6) of the CAA requires EPA to review and revise the MACT standards, as necessary, taking into account developments in practices, processes and control technologies. For more information: <http://www.epa.gov/ttn/atw/rrisk/rtrpg.html>.

APPENDIX 2B

RECENT REGULATORY ACTIONS ON STATIONARY SOURCES OF LEAD

The table below identifies recent regulatory actions on stationary sources estimated to result in reductions in lead emissions.

Action	Source Categories Affected	Citation
NESHAP	Area Sources: Polyvinyl Chloride and Copolymers Production, Primary Copper Smelting, Secondary Copper Smelting, and Primary Nonferrous Metals (zinc, cadmium, beryllium)	76 FR 22848 (2011)
NESHAP	Area Sources: Acrylic and Modacrylic Fibers Production, Carbon Black Production, Chemical Manufacturing: Chromium Compounds, Flexible Polyurethane Foam Production and Fabrication, Lead Acid Battery Manufacturing, Wood Preserving:	72 FR 38864 (7/16/2007)
NESHAP	Area Sources: Clay Ceramics Manufacturing, Glass Manufacturing, and Secondary Nonferrous Metals Processing	72 FR 73179 (12/26/2007)
NESHAP	Area Sources: Electric Arc Furnace Steelmaking Facilities	72 FR 74088 (12/28/2007)
NESHAP	Iron and Steel Foundries Area Sources	73 FR 225 (1/2/2008)
NESHAP	Area Sources: Acrylic and Modacrylic Fibers Production, Carbon Black Production, Chemical Manufacturing: Chromium Compounds, Flexible Polyurethane Foam Production and Fabrication, Lead Acid Battery Manufacturing, Wood Preserving	73 FR 5923 (3/26/2008)
NESHAP	Area Source Standards for Plating and Polishing Operations	73 FR 37728 (7/1/2008)
NESHAP	Area Source Standards for Nine Metal Fabrication and Finishing Source Categories	73 FR 42978 (7/23/2008)
NESHAP	Area Source Standards for Aluminum, Copper, and Other Nonferrous Foundries	74 FR 30366 (6/25/2009)
NESHAP, NSPS	Portland Cement Manufacturing Industry	75 FR 54970 (9/9/2010)
EGES, NSPS	Commercial and Industrial Solid Waste Incineration Units	76 FR 15704 (3/21/2011)
EGES, NSPS	Standards of Performance for New Stationary Sources and Emission Guidelines for Existing Sources: Sewage Sludge Incineration Units	76 FR 15372 (3/21/2011)
NESHAP	Major Sources: Industrial, Commercial, and Institutional Boilers, and Process Heaters	76 FR 15608 (3/21/2011)
NESHAP	Area Sources: Industrial, Commercial, and Institutional Boilers and Process Heaters	76 FR 15554 (3/21/2011)
EGES, NSPS	Standards of Performance for New Stationary Sources and Emissions Guidelines for Existing Sources: Hospital/Medical/Infectious Waste Incinerators	76 FR 18407 (4/4/2011)
NESHAP	Ferroalloys Production	76 FR 72508 (11/23/2011)
EGES=Emissions Guidelines for Existing Sources NESHAP=National Emission Standards for Hazardous Air Pollutants NSPS= New Source Performance Standards		

APPENDIX 2C

CRITERIA FOR AIR QUALITY DATA ANALYSIS

Criteria for the 2009-2011 data analysis presented in Figures 2-10 through 2-13 are as listed below, with abbreviations defined following this list.

- Years utilized were 2009-2011. November and December 2008 data were also used for rolling 3-month average analysis.
- Data were extracted from EPA's Air Quality System in August and September of 2012.
- Data for parameters 12128 (STP) and 14129 (LC) were used for Pb-TSP analysis; data for parameters 84128 (STP) and 88128 (LC) were used for Pb-PM_{2.5} analysis; and data for parameters 82128 (STP) and 85128 (LC) were used for Pb-PM₁₀ analysis. No adjustment was made to the STP or LC data to make them more comparable to each other; based on previous analysis, the LC and STP versions of same-site-day samples are extremely similar. Collocated data – for same size cut- were combined to a site basis by using the following hierarchy, evaluated each site-day: LC parameter data takes precedence over STP parameter data and lower POC numbers take precedence over higher POC numbers.
- For maximum rolling 3-month average analysis, the 2009-2011 databases also encompassed the prior 2 months (i.e., November and December 2008 data): 3 or more observations were needed to make a valid monthly average (for Pb-TSP, the daily data had to be collected/analyzed via an FRM or FEM); 3 valid consecutive months were required to make a valid rolling 3-month period; 6 valid rolling 3-month periods were required to make a valid year; and 1, 2, or 3 valid years were required to make a valid 3-month metric. The rolling 3-month average metric (by size cut for 2009-2011) was identified as the highest valid 3-month average across the valid (1, 2, or 3) years.
- For maximum monthly analysis: 3 or more observations were needed to make a valid monthly average (for Pb-TSP, the daily data had to be collected/analyzed via an FRM or FEM); 6 valid months were required to make a valid year; and 1, 2, or 3 valid years were required to make a valid monthly metric. The maximum monthly metric (by size cut for 2009-2011) was identified as the highest valid monthly average across the valid (1, 2, or 3) years.
- For annual average analysis: monthly averages were used to construct annual averages, 3 or more observations were required to make a valid month (for Pb-TSP, the daily data had to be collected/analyzed via an FRM or FEM); 6 valid months were required to make a valid year (i.e., annual average) and 1, 2, or 3 valid years were required to make a valid annual metric. The annual metric (by size cut for 2009-2011) was identified as the average of the 1, 2, or 3 (2009, 2010, and/or 2011) valid annual means.

- 1 **Definitions for Abbreviations**
- 2 FEM – Federal equivalent method
- 3 FRM – Federal reference method
- 4 LC – Local conditions
- 5 POC – Parameter occurrence code
- 6 STP – Standard temperature and pressure

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-1. Pb-TSP concentrations at Pb-TSP sites, 2009-2011.

SITE	Max 3-month Mean (ug/m ³)	Annual Mean (ug/m ³)	Max Monthly Mean (ug/m ³)	previous source	current source	State	County
011090003	1.035	0.564	2.069	0	1	Alabama	Pike County
040071002	0.267	0.133	0.306	0	1	Arizona	Gila County
040078000	0.061	0.033	0.077	0	1	Arizona	Gila County
040134018	0.033	0.023	0.042	0	0	Arizona	Maricopa County
060250005	0.023	0.016	0.032	0	0	California	Imperial County
060371103	0.016	0.011	0.018	0	0	California	Los Angeles County
060371302	0.019	0.01	0.026	0	0	California	Los Angeles County
060371402	0.044	0.022	0.064	0	0	California	Los Angeles County
060371403	0.116	0.061	0.148	0	1	California	Los Angeles County
060371404	0.108	0.061	0.14	0	1	California	Los Angeles County
060371405	0.667	0.343	0.801	0	1	California	Los Angeles County
060371406	0.085	0.049	0.094	0	1	California	Los Angeles County
060371602	0.022	0.011	0.04	0	0	California	Los Angeles County
060374002	0.01	0.006	0.014	0	0	California	Los Angeles County
060374004	0.012	0.007	0.017	0	0	California	Los Angeles County
060375005	0.008	0.003	0.01	0	0	California	Los Angeles County
060651003	0.011	0.006	0.014	0	0	California	Riverside County
060658001	0.011	0.007	0.013	0	0	California	Riverside County
060711004	0.011	0.007	0.014	0	0	California	San Bernardino County
060719004	0.013	0.008	0.014	0	0	California	San Bernardino County
080050007	0.02	0.014	0.028	0	0	Colorado	Arapahoe County
080310025	0.01	0.006	0.013	0	0	Colorado	Denver County
120570100	0.044	0.026	0.1	0	1	Florida	Hillsborough County
120571066	0.979	0.282	1.959	0	1	Florida	Hillsborough County
120571073	0.423	0.116	0.977	0	1	Florida	Hillsborough County
130150003	0.019	0.014	0.032	0	1	Georgia	Bartow County
130890003	0.004	0.003	0.006	0	0	Georgia	DeKalb County
132150011	0.071	0.029	0.14	1	1	Georgia	Muscogee County
170310001	0.024	0.016	0.028	0	0	Illinois	Cook County
170310022	0.051	0.033	0.07	0	0	Illinois	Cook County
170310026	0.034	0.023	0.038	0	0	Illinois	Cook County
170310052	0.024	0.016	0.032	0	0	Illinois	Cook County
170310110	0.294	0.1	0.58	0	1	Illinois	Cook County
170310210	0.053	0.037	0.092	0	1	Illinois	Cook County
170313103	0.013	0.011	0.016	0	0	Illinois	Cook County
170313301	0.027	0.018	0.028	0	0	Illinois	Cook County
170314201	0.011	0.01	0.014	0	0	Illinois	Cook County
170316003	0.036	0.024	0.04	0	0	Illinois	Cook County
171150110	0.199	0.082	0.386	0	1	Illinois	Macon County
171170002	0.011	0.01	0.012	0	0	Illinois	Macoupin County
171190010	0.416	0.118	0.836	0	1	Illinois	Madison County
171193007	0.036	0.018	0.053	0	0	Illinois	Madison County
171430037	0.011	0.01	0.013	0	0	Illinois	Peoria County
171430110	0.016	0.013	0.024	0	1	Illinois	Peoria County
171430210	0.015	0.011	0.026	0	1	Illinois	Peoria County

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-1. Pb-TSP concentrations at Pb-TSP sites, 2009-2011.

SITE	Max 3-month Mean (ug/m ³)	Annual Mean (ug/m ³)	Max Monthly Mean (ug/m ³)	previous source	current source	State	County
171630010	0.029	0.019	0.038	1	0	Illinois	St. Clair County
171950110	0.028	0.02	0.04	0	1	Illinois	Whiteside County
172010110	0.063	0.031	0.118	0	1	Illinois	Winnebago County
180350008	0.11	0.079	0.141	0	1	Indiana	Delaware County
180350009	0.262	0.186	0.427	0	1	Indiana	Delaware County
180890023	0.077	0.026	0.087	0	1	Indiana	Lake County
180890032	0.06	0.027	0.144	0	1	Indiana	Lake County
180890033	0.139	0.054	0.298	0	1	Indiana	Lake County
180892008	0.037	0.016	0.049	0	0	Indiana	Lake County
180970063	0.079	0.026	0.125	0	1	Indiana	Marion County
180970076	0.02	0.01	0.029	0	1	Indiana	Marion County
180970078	0.011	0.005	0.013	0	0	Indiana	Marion County
181270023	0.022	0.013	0.032	0	1	Indiana	Porter County
181630020	0.007	0.004	0.01	0	0	Indiana	Vanderburgh County
191550011	0.263	0.122	0.282	0	1	Iowa	Pottawattamie County
191630015	0.012	0.01	0.024	0	0	Iowa	Scott County
201690004	0.421	0.18	0.488	0	1	Kansas	Saline County
210190016	0.004	0.004	0.007	0	1	Kentucky	Boyd County
211510003	0.069	0.034	0.121	0	1	Kentucky	Madison County
212070001	0.036	0.021	0.076	0	1	Kentucky	Russell County
220330014	0.005	0.004	0.007	0	1	Louisiana	East Baton Rouge Parish
220950003	0.053	0.037	0.066	0	1	Louisiana	St. John the Baptist Parish
250250042	0.01	0.007	0.012			Massachusetts	Suffolk County
260670003	0.284	0.126	0.414	0	1	Michigan	Ionia County
260810020	0.008	0.005	0.01	0	0	Michigan	Kent County
261630001	0.006	0.005	0.007	0	0	Michigan	Wayne County
261630033	0.023	0.012	0.034	0	0	Michigan	Wayne County
270036020	0.033	0.017	0.054	0	1	Minnesota	Anoka County
270072303	0.001	0	0.002	0	0	Minnesota	Beltrami County
270177416	0	0	0			Minnesota	Carlton County
270210001	0.001	0	0.002	0	0	Minnesota	Cass County
270370020	0.011	0.005	0.022	0	0	Minnesota	Dakota County
270370423	0.002	0.001	0.005	0	0	Minnesota	Dakota County
270370442	0.013	0.003	0.007	0	0	Minnesota	Dakota County
270370443	0.003	0.002	0.005	0	0	Minnesota	Dakota County
270370465	0.259	0.128	0.568	0	1	Minnesota	Dakota County
270370470	0.002	0.001	0.004	0	0	Minnesota	Dakota County
270530963	0.006	0.003	0.01	0	0	Minnesota	Hennepin County
270530966	0.016	0.004	0.044	0	0	Minnesota	Hennepin County
270531007	0.004	0.002	0.006	0	0	Minnesota	Hennepin County
270532006	0.003	0.001	0.005	0	0	Minnesota	Hennepin County
270750005	0	0	0			Minnesota	Lake County
271230866	0.016	0.012	0.023	0	0	Minnesota	Ramsey County
271230871	0.007	0.002	0.01	0	0	Minnesota	Ramsey County
271231003	0.006	0.004	0.008	0	0	Minnesota	Ramsey County

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-1. Pb-TSP concentrations at Pb-TSP sites, 2009-2011.

SITE	Max 3-month Mean (ug/m ³)	Annual Mean (ug/m ³)	Max Monthly Mean (ug/m ³)	previous source	current source	State	County
271377001	0.003	0.001	0.006	0	0	Minnesota	St. Louis County
271377555	0.006	0.002	0.008	0	0	Minnesota	St. Louis County
271453053	0.006	0.003	0.01	0	1	Minnesota	Stearns County
271630009	0.005	0.003	0.006	0	0	Minnesota	Washington County
271630438	0.005	0.002	0.008	0	0	Minnesota	Washington County
271630446	0.002	0	0.003	0	0	Minnesota	Washington County
290930016	2.07	0.49	1.392	0	1	Missouri	Iron County
290930021	1.937	0.622	2.438	0	1	Missouri	Iron County
290930027	0.083	0.038	0.121	1	1	Missouri	Iron County
290930029	0.115	0.037	0.271	1	1	Missouri	Iron County
290930033	0.057	0.027	0.105	0	1	Missouri	Iron County
290930034	0.402	0.283	0.509	0	1	Missouri	Iron County
290939007	0.581	0.392	0.844	0	1	Missouri	Iron County
290939008	0.387	0.256	0.538	0	1	Missouri	Iron County
290970005	0.019	0.013	0.024	0	0	Missouri	Jasper County
290990004	1.16	0.889	1.576	0	1	Missouri	Jefferson County
290990005	0.396	0.231	0.536	0	1	Missouri	Jefferson County
290990009	0.067	0.043	0.087	0	1	Missouri	Jefferson County
290990011	0.403	0.21	0.509	0	1	Missouri	Jefferson County
290990013	0.198	0.116	0.344	0	1	Missouri	Jefferson County
290990015	1.079	0.921	1.349	0	1	Missouri	Jefferson County
290990020	0.943	0.646	1.28	0	1	Missouri	Jefferson County
290990021	0.969	0.585	1.124	0	1	Missouri	Jefferson County
290990022	0.63	0.434	0.861	0	1	Missouri	Jefferson County
290990023	0.594	0.394	0.724	0	1	Missouri	Jefferson County
290990024	0.659	0.374	0.862	0	1	Missouri	Jefferson County
290990025	0.085	0.043	0.134	0	1	Missouri	Jefferson County
290990026	0.072	0.035	0.114	0	1	Missouri	Jefferson County
290999001	1.204	0.824	1.623	0	1	Missouri	Jefferson County
290999002	0.431	0.232	0.593	0	1	Missouri	Jefferson County
290999003	0.467	0.198	0.605	0	1	Missouri	Jefferson County
290999004	0.261	0.119	0.328	0	1	Missouri	Jefferson County
290999005	1.432	0.959	2.185	0	1	Missouri	Jefferson County
290999006	0.091	0.049	0.131	0	1	Missouri	Jefferson County
291790001	0.059	0.033	0.087	0	1	Missouri	Reynolds County
291790002	0.068	0.033	0.172	0	1	Missouri	Reynolds County
291790003	0.1	0.027	0.268	0	1	Missouri	Reynolds County
291790034	0.093	0.068	0.142	0	1	Missouri	Reynolds County
291870006	0.132	0.05	0.302	0	0	Missouri	St. Francois County
291870007	0.041	0.043	0.364	0	0	Missouri	St. Francois County
291892003	0.041	0.007	0.011	0	0	Missouri	St. Louis County
310530005	0.072	0.051	0.149	0	1	Nebraska	Dodge County
311270002	0.115	0.042	0.206	0	1	Nebraska	Nemaha County
360470122	0.019	0.014	0.02	0	0	New York	Kings County
360713001	0.101	0.031	0.134	0	1	New York	Orange County

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-1. Pb-TSP concentrations at Pb-TSP sites, 2009-2011.

SITE	Max 3-month Mean (ug/m ³)	Annual Mean (ug/m ³)	Max Monthly Mean (ug/m ³)	previous source	current source	State	County
360713002	1.027	0.128	2.821	0	1	New York	Orange County
360713004	0.012	0.005	0.016	0	1	New York	Orange County
390170015	0.009	0.006	0.013	0	0	Ohio	Butler County
390290019	0.057	0.022	0.136	0	0	Ohio	Columbiana County
390290020	0.025	0.015	0.035	0	0	Ohio	Columbiana County
390290022	0.044	0.02	0.065	0	0	Ohio	Columbiana County
390350038	0.021	0.013	0.026	0	0	Ohio	Cuyahoga County
390350042	0.03	0.014	0.044	0	0	Ohio	Cuyahoga County
390350049	0.531	0.165	0.719	0	1	Ohio	Cuyahoga County
390350061	0.023	0.015	0.03	1	0	Ohio	Cuyahoga County
390350072	0.035	0.014	0.054	0	1	Ohio	Cuyahoga County
390490025	0.011	0.008	0.012	0	0	Ohio	Franklin County
390510001	0.178	0.073	0.21	0	1	Ohio	Fulton County
390910006	0.006	0.004	0.008	1	1	Ohio	Logan County
391010003	0.056	0.035	0.097	0	1	Ohio	Marion County
391510017	0.023	0.014	0.028	0	1	Ohio	Stark County
391550012	0.011	0.007	0.017	0	1	Ohio	Trumbull County
391670008	0.007	0.005	0.01	0	0	Ohio	Washington County
391670010	0.008	0.005	0.01	0	0	Ohio	Washington County
401159006	0.015	0.011	0.022	0	0	Oklahoma	Ottawa County
401159007	0.034	0.017	0.056	0	0	Oklahoma	Ottawa County
401210416	0.004	0.003	0.006	0	1	Oklahoma	Pittsburg County
410711702	0.035	0.018	0.037	0	1	Oregon	Yamhill County
420030002	0.016	0.012	0.024	0	0	Pennsylvania	Allegheny County
420030008	0.014	0.01	0.02	0	0	Pennsylvania	Allegheny County
420030070	0.056	0.022	0.11	0	1	Pennsylvania	Allegheny County
420031009	0.138	0.043	0.149	0	1	Pennsylvania	Allegheny County
420070006	0.085	0.054	0.198	0	1	Pennsylvania	Beaver County
420070007	0.253	0.171	0.393	0	1	Pennsylvania	Beaver County
420070505	0.115	0.086	0.17	0	1	Pennsylvania	Beaver County
420110005	0.051	0.043	0.07	0	1	Pennsylvania	Berks County
420110020	0.196	0.127	0.347	0	1	Pennsylvania	Berks County
420110021	0.139	0.049	0.319	0	1	Pennsylvania	Berks County
420110022	0.118	0.043	0.261	0	1	Pennsylvania	Berks County
420110717	0.116	0.065	0.108	0	1	Pennsylvania	Berks County
420111717	0.251	0.133	0.348	0	1	Pennsylvania	Berks County
420210808	0.073	0.05	0.128	0	0	Pennsylvania	Cambria County
420450002	0.047	0.043	0.048	0	0	Pennsylvania	Delaware County
420450004	0.047	0.045	0.048	0	1	Pennsylvania	Delaware County
420550002	0.046	0.045	0.047	0	1	Pennsylvania	Franklin County
420630005	0.049	0.029	0.058	0	1	Pennsylvania	Indiana County
420730011	0.023	0.032	0.046	0	1	Pennsylvania	Lawrence County
420790036	0.137	0.095	0.268	0	1	Pennsylvania	Luzerne County
421010449	0.03	0.021	0.039	1	0	Pennsylvania	Philadelphia County
421011002	0.025	0.022	0.029	0	0	Pennsylvania	Philadelphia County

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-1. Pb-TSP concentrations at Pb-TSP sites, 2009-2011.

SITE	Max 3-month Mean (ug/m ³)	Annual Mean (ug/m ³)	Max Monthly Mean (ug/m ³)	previous source	current source	State	County
421290007	0.043	0.041	0.043	0	0	Pennsylvania	Westmoreland County
421290009	0.046	0.027	0.047	0	1	Pennsylvania	Westmoreland County
450190003	0.007	0.005	0.013	0	0	South Carolina	Charleston County
450450015	0.006	0.005	0.008	0	0	South Carolina	Greenville County
450790007	0.005	0.004	0.007	0	0	South Carolina	Richland County
450790019	0.024	0.011	0.061	0	0	South Carolina	Richland County
470930023	0.165	0.114	0.208	0	1	Tennessee	Knox County
470930027	0.042	0.02	0.078	0	1	Tennessee	Knox County
470931017	0.037	0.018	0.063	0	1	Tennessee	Knox County
471633001	0.093	0.058	0.115	1	1	Tennessee	Sullivan County
471633002	0.06	0.039	0.069	1	1	Tennessee	Sullivan County
471633003	0.06	0.041	0.066	1	1	Tennessee	Sullivan County
471633004	0.08	0.041	0.124	1	1	Tennessee	Sullivan County
471634002	0.038	0.025	0.062	1	1	Tennessee	Sullivan County
480610006	0.005	0.004	0.007	0	0	Texas	Cameron County
480850003	0.371	0.179	0.625	0	1	Texas	Collin County
480850007	0.199	0.105	0.24	0	1	Texas	Collin County
480850009	0.774	0.453	1.178	0	1	Texas	Collin County
480850029	0.18	0.076	0.335	0	1	Texas	Collin County
481410002	0.04	0.023	0.087	0	0	Texas	El Paso County
481410033	0.039	0.019	0.057	0	0	Texas	El Paso County
481410055	0.014	0.009	0.015	0	0	Texas	El Paso County
481410058	0.033	0.017	0.056	0	0	Texas	El Paso County
482011034	0.008	0.005	0.009	0	0	Texas	Harris County
482570020	0.104	0.06	0.11	0	1	Texas	Kaufman County
483750024	0.013	0.005	0.029	0	1	Texas	Potter County
484790016	0.026	0.016	0.035	0	0	Texas	Webb County
490351001	0.057	0.023	0.086	0	1	Utah	Salt Lake County
510090007	0.018	0.004	0.007	0	1	Virginia	Amherst County
510270006	0.013	0.01	0.015	0	1	Virginia	Buchanan County
510870014	0.004	0.005	0.024	0	0	Virginia	Henrico County
517700011	0.109	0.038	0.272	0	1	Virginia	Roanoke city
551170008	0.152	0.055	0.225	0	1	Wisconsin	Sheboygan County
720130001	0.339	0.188	0.416	0	1	Puerto Rico	Arecibo Municipio
720210010	0.011	0.005	0.027	0	0	Puerto Rico	Bayamón Municipio

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-2. Pb-PM₁₀ concentrations at urban PM₁₀ sites, 2009-2011.

SITE	Max 3-month Mean (ug/m ³)	Annual Mean (ug/m ³)	Max Monthly Mean (ug/m ³)	State	County
040134009		0.005	0.008	Arizona	Maricopa County
040139997	0.007	0.004	0.007	Arizona	Maricopa County
060371103	0.013	0.006	0.026	California	Los Angeles County
060658001	0.013	0.006	0.03	California	Riverside County
060850005	0.005	0.002	0.006	California	Santa Clara County
080770017	0.004	0.001	0.004	Colorado	Mesa County
110010043	0.004	0.003	0.005	District of Columbia	District of Columbia
120573002	0.003	0.001	0.003	Florida	Hillsborough County
120951004		0.003	0.008	Florida	Orange County
121030018	0.003	0.002	0.004	Florida	Pinellas County
121030026	0.003	0.002	0.003	Florida	Pinellas County
130890002	0.002	0.001	0.003	Georgia	DeKalb County
170314201	0.006	0.004	0.01	Illinois	Cook County
171639010		0.015	0.031	Illinois	St. Clair County
210190002	0.019	0.012	0.028	Kentucky	Boyd County
210670014	0.005	0.004	0.009	Kentucky	Fayette County
250250042	0.004	0.003	0.004	Massachusetts	Suffolk County
261630001	0.006	0.004	0.006	Michigan	Wayne County
261630033	0.023	0.011	0.035	Michigan	Wayne County
295100085	0.018	0.011	0.026	Missouri	St. Louis city
360050080	0.008	0.006	0.012	New York	Bronx County
360050110	0.006	0.005	0.008	New York	Bronx County
360551007	0.003	0.002	0.004	New York	Monroe County
410290133	0.003	0.002	0.005	Oregon	Jackson County
410390060	0.003	0.002	0.006	Oregon	Lane County
410390062	0.002	0.002	0.003	Oregon	Lane County
410510246	0.008	0.004	0.013	Oregon	Multnomah County
440070022	0.003	0.003	0.004	Rhode Island	Providence County
482011035	0.007	0.005	0.009	Texas	Harris County
482011039	0.003	0.002	0.004	Texas	Harris County
490110004	0.006	0.003	0.009	Utah	Davis County
500070007	0.002	0.001	0.002	Vermont	Chittenden County
510870014	0.007	0.003	0.016	Virginia	Henrico County
530330080	0.005	0.003	0.006	Washington	King County
550790010		0.006	0.01	Wisconsin	Milwaukee County

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-3. Pb-PM_{2.5} concentrations at urban CSN PM_{2.5} sites, 2009-2011.

SITE	Max 3-month Mean (ug/m ³)	Annual Mean (ug/m ³)	Max Monthly Mean (ug/m ³)	State	County
010730023	0.041	0.017	0.064	Alabama	Jefferson County
010732003	0.032	0.008	0.078	Alabama	Jefferson County
010890014	0.003	0.002	0.003	Alabama	Madison County
011011002	0.007	0.003	0.011	Alabama	Montgomery County
011130001	0.013	0.004	0.028	Alabama	Russell County
020900010	0.006	0.003	0.008	Alaska	Fairbanks North Star Borough
040134009	0.004	0.003	0.005	Arizona	Maricopa County
040139997	0.004	0.002	0.004	Arizona	Maricopa County
040191028	0.003	0.002	0.003	Arizona	Pima County
051190007	0.004	0.002	0.006	Arkansas	Pulaski County
060010007	0.005	0.004	0.005	California	Alameda County
060010011	0.012	0.005	0.023	California	Alameda County
060070002	0.003	0.002	0.003	California	Butte County
060190008	0.003	0.002	0.004	California	Fresno County
060250005	0.021	0.012	0.029	California	Imperial County
060290014	0.003	0.002	0.004	California	Kern County
060299001	0.001	0.001	0.001	California	Kern County
060371103	0.02	0.005	0.021	California	Los Angeles County
060590007	0.042	0.017	0.043	California	Orange County
060658001	0.005	0.003	0.006	California	Riverside County
060670006	0.004	0.002	0.009	California	Sacramento County
060670010	0.003	0.002	0.005	California	Sacramento County
060712002	0.037	0.018	0.038	California	San Bernardino County
060730003	0.005	0.003	0.006	California	San Diego County
060731002	0.004	0.002	0.005	California	San Diego County
060850005	0.003	0.002	0.004	California	Santa Clara County
060950004	0.007	0.005	0.009	California	Solano County
060990005	0.005	0.003	0.009	California	Stanislaus County
061072002	0.004	0.002	0.006	California	Tulare County
061112002	0.003	0.002	0.003	California	Ventura County
080010006	0.003	0.002	0.003	Colorado	Adams County
080310025	0.003	0.002	0.005	Colorado	Denver County
080770017	0.002	0.002	0.003	Colorado	Mesa County
081230008	0.003	0.002	0.003	Colorado	Weld County
090090027	0.005	0.003	0.005	Connecticut	New Haven County
100010003	0.004	0.002	0.007	Delaware	Kent County
100032004	0.004	0.003	0.006	Delaware	New Castle County
110010043	0.003	0.002	0.004	District of Columbia	District of Columbia
120111002	0.002	0.002	0.003	Florida	Broward County
120573002	0.003	0.002	0.003	Florida	Hillsborough County
120730012	0.005	0.003	0.009	Florida	Leon County
121030026	0.003	0.002	0.003	Florida	Pinellas County
130210007	0.004	0.003	0.007	Georgia	Bibb County

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-3. Pb-PM_{2.5} concentrations at urban CSN PM_{2.5} sites, 2009-2011.

SITE	Max 3-month Mean (ug/m ³)	Annual Mean (ug/m ³)	Max Monthly Mean (ug/m ³)	State	County
130590001	0.002	0.002	0.003	Georgia	Clarke County
130890002	0.003	0.002	0.003	Georgia	DeKalb County
131150003	0.006	0.003	0.009	Georgia	Floyd County
132150011	0.013	0.005	0.029	Georgia	Muscogee County
132450091	0.005	0.003	0.008	Georgia	Richmond County
132950002	0.006	0.003	0.009	Georgia	Walker County
150030010	0.002	0.002	0.002	Hawaii	Honolulu County
150032004	0.008	0.004	0.02	Hawaii	Honolulu County
160010010	0.002	0.002	0.003	Idaho	Ada County
170310057	0.07	0.011	0.199	Illinois	Cook County
170310076	0.005	0.003	0.005	Illinois	Cook County
170314201	0.004	0.003	0.006	Illinois	Cook County
170434002	0.005	0.003	0.008	Illinois	DuPage County
171190024	0.02	0.011	0.032	Illinois	Madison County
171630900	0.005	0.003	0.006	Illinois	St. Clair County
171639010	0.014	0.009	0.019	Illinois	St. Clair County
180190006	0.004	0.003	0.005	Indiana	Clark County
180390008	0.004	0.003	0.005	Indiana	Elkhart County
180890022	0.011	0.006	0.018	Indiana	Lake County
180892004	0.022	0.009	0.037	Indiana	Lake County
180970078	0.005	0.003	0.007	Indiana	Marion County
181630012	0.003	0.002	0.003	Indiana	Vanderburgh County
181630021	0.003	0.003	0.004	Indiana	Vanderburgh County
191130037	0.003	0.002	0.003	Iowa	Linn County
191530030	0.003	0.002	0.005	Iowa	Polk County
191630015	0.011	0.006	0.017	Iowa	Scott County
201730010	0.004	0.002	0.009	Kansas	Sedgwick County
202090021	0.006	0.004	0.01	Kansas	Wyandotte County
210190017	0.005	0.003	0.007	Kentucky	Boyd County
210670012	0.007	0.003	0.012	Kentucky	Fayette County
211110067	0.007	0.004	0.012	Kentucky	Jefferson County
211170007	0.006	0.004	0.01	Kentucky	Kenton County
220150008	0.006	0.004	0.009	Louisiana	Bossier Parish
220330009	0.005	0.003	0.008	Louisiana	East Baton Rouge Parish
240053001	0.006	0.003	0.007	Maryland	Baltimore County
240330030	0.003	0.002	0.005	Maryland	Prince George's County
250130008	0.003	0.002	0.004	Massachusetts	Hampden County
250250042	0.003	0.002	0.004	Massachusetts	Suffolk County
260810020	0.004	0.003	0.006	Michigan	Kent County
261150005	0.005	0.003	0.008	Michigan	Monroe County
261470005	0.016	0.004	0.03	Michigan	St. Clair County
261630001	0.004	0.003	0.005	Michigan	Wayne County
261630015	0.013	0.007	0.027	Michigan	Wayne County

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-3. Pb-PM_{2.5} concentrations at urban CSN PM_{2.5} sites, 2009-2011.

SITE	Max 3-month Mean (ug/m ³)	Annual Mean (ug/m ³)	Max Monthly Mean (ug/m ³)	State	County
261630033	0.017	0.007	0.029	Michigan	Wayne County
270031002	0.009	0.006	0.013	Minnesota	Anoka County
270530963	0.003	0.002	0.005	Minnesota	Hennepin County
271095008	0.004	0.002	0.006	Minnesota	Olmsted County
280470008	0.003	0.002	0.003	Mississippi	Harrison County
280490019	0.003	0.002	0.004	Mississippi	Hinds County
290470005	0.003	0.002	0.003	Missouri	Clay County
290990019	0.015	0.005	0.018	Missouri	Jefferson County
295100085	0.011	0.006	0.013	Missouri	St. Louis city
300630031	0.003	0.002	0.003	Montana	Missoula County
310550019	0.004	0.003	0.008	Nebraska	Douglas County
320030020	0.002	0.002	0.003	Nevada	Clark County
320030540	0.003	0.002	0.004	Nevada	Clark County
320310016	0.003	0.002	0.003	Nevada	Washoe County
340130003	0.004	0.003	0.006	New Jersey	Essex County
340230006	0.003	0.002	0.005	New Jersey	Middlesex County
340273001	0.003	0.002	0.003	New Jersey	Morris County
340390004	0.004	0.003	0.007	New Jersey	Union County
350010023	0.003	0.002	0.005	New Mexico	Bernalillo County
360010005	0.005	0.003	0.007	New York	Albany County
360050110	0.004	0.003	0.005	New York	Bronx County
360290005	0.011	0.007	0.016	New York	Erie County
360551007	0.003	0.002	0.003	New York	Monroe County
360610134	0.004	0.003	0.004	New York	New York County
360810124	0.005	0.003	0.011	New York	Queens County
370210034	0.002	0.002	0.003	North Carolina	Buncombe County
370350004	0.003	0.002	0.004	North Carolina	Catawba County
370670022	0.006	0.003	0.011	North Carolina	Forsyth County
371190041	0.003	0.002	0.003	North Carolina	Mecklenburg County
371830014	0.002	0.002	0.003	North Carolina	Wake County
380171004	0.003	0.002	0.005	North Dakota	Cass County
390171004	0.011	0.006	0.012	Ohio	Butler County
390350038	0.009	0.005	0.013	Ohio	Cuyahoga County
390350060	0.009	0.006	0.011	Ohio	Cuyahoga County
390490081	0.003	0.003	0.005	Ohio	Franklin County
390610040	0.01	0.004	0.015	Ohio	Hamilton County
390811001	0.009	0.006	0.012	Ohio	Jefferson County
390870012	0.007	0.005	0.01	Ohio	Lawrence County
390933002	0.01	0.006	0.018	Ohio	Lorain County
390950026	0.004	0.003	0.005	Ohio	Lucas County
390990014	0.012	0.006	0.018	Ohio	Mahoning County
391130032	0.014	0.004	0.034	Ohio	Montgomery County
391351001	0.003	0.003	0.004	Ohio	Preble County

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-3. Pb-PM_{2.5} concentrations at urban CSN PM_{2.5} sites, 2009-2011.

SITE	Max 3-month Mean (ug/m ³)	Annual Mean (ug/m ³)	Max Monthly Mean (ug/m ³)	State	County
391510017	0.013	0.007	0.017	Ohio	Stark County
391530023	0.006	0.004	0.01	Ohio	Summit County
401091037	0.002	0.002	0.003	Oklahoma	Oklahoma County
401431127	0.005	0.003	0.008	Oklahoma	Tulsa County
410290133	0.003	0.001	0.002	Oregon	Jackson County
410390060	0.003	0.003	0.007	Oregon	Lane County
410392013	0.002	0.002	0.004	Oregon	Lane County
410510080	0.004	0.003	0.005	Oregon	Multnomah County
420030008	0.016	0.009	0.022	Pennsylvania	Allegheny County
420030064	0.03	0.017	0.052	Pennsylvania	Allegheny County
420110011	0.012	0.006	0.014	Pennsylvania	Berks County
420210011	0.01	0.006	0.017	Pennsylvania	Cambria County
420270100	0.003	0.002	0.004	Pennsylvania	Centre County
420290100	0.004	0.003	0.005	Pennsylvania	Chester County
420430401	0.007	0.004	0.011	Pennsylvania	Dauphin County
420490003	0.005	0.003	0.007	Pennsylvania	Erie County
420692006	0.007	0.003	0.01	Pennsylvania	Lackawanna County
420710007	0.005	0.004	0.008	Pennsylvania	Lancaster County
420950025	0.008	0.004	0.018	Pennsylvania	Northampton County
421010004	0.007	0.003	0.012	Pennsylvania	Philadelphia County
421010055	0.003	0.002	0.005	Pennsylvania	Philadelphia County
421011002	0.007	0.005	0.009	Pennsylvania	Philadelphia County
421255001	0.007	0.004	0.015	Pennsylvania	Washington County
421290008	0.004	0.003	0.007	Pennsylvania	Westmoreland County
421330008	0.006	0.003	0.01	Pennsylvania	York County
440070022	0.004	0.003	0.007	Rhode Island	Providence County
440071010	0.003	0.002	0.003	Rhode Island	Providence County
450190049	0.003	0.002	0.003	South Carolina	Charleston County
450450008	0.003	0.002	0.003	South Carolina	Greenville County
450450015	0.006	0.003	0.009	South Carolina	Greenville County
450790007	0.003	0.002	0.004	South Carolina	Richland County
460990008	0.003	0.002	0.003	South Dakota	Minnehaha County
470370023	0.003	0.002	0.004	Tennessee	Davidson County
470654002	0.004	0.003	0.006	Tennessee	Hamilton County
470931020	0.006	0.003	0.007	Tennessee	Knox County
471251009	0.004	0.002	0.005	Tennessee	Montgomery County
471570024	0.004	0.002	0.005	Tennessee	Shelby County
471570075	0.004	0.003	0.006	Tennessee	Shelby County
480612004	0.002	0.002	0.003	Texas	Cameron County
481130050	0.005	0.003	0.007	Texas	Dallas County
481130069	0.004	0.003	0.008	Texas	Dallas County
481390016	0.005	0.003	0.008	Texas	Ellis County
481410044	0.006	0.003	0.008	Texas	El Paso County

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-3. Pb-PM_{2.5} concentrations at urban CSN PM_{2.5} sites, 2009-2011.

SITE	Max 3-month Mean (ug/m ³)	Annual Mean (ug/m ³)	Max Monthly Mean (ug/m ³)	State	County
481410053	0.017	0.007	0.02	Texas	El Paso County
482010024	0.006	0.004	0.008	Texas	Harris County
482011039	0.002	0.002	0.003	Texas	Harris County
482450021	0.003	0.002	0.003	Texas	Jefferson County
483030325	0.007	0.003	0.018	Texas	Lubbock County
483550034	0.014	0.003	0.036	Texas	Nueces County
484530020	0.006	0.002	0.014	Texas	Travis County
490110004	0.004	0.003	0.007	Utah	Davis County
490353006	0.008	0.003	0.016	Utah	Salt Lake County
490494001	0.007	0.003	0.012	Utah	Utah County
500070012	0.003	0.002	0.003	Vermont	Chittenden County
510870014	0.005	0.002	0.009	Virginia	Henrico County
530110013	0.003	0.002	0.004	Washington	Clark County
530330057	0.008	0.004	0.013	Washington	King County
530330080	0.003	0.003	0.004	Washington	King County
530530029	0.005	0.003	0.008	Washington	Pierce County
530530031	0.016	0.004	0.033	Washington	Pierce County
530611007	0.004	0.003	0.007	Washington	Snohomish County
530770009	0.003	0.003	0.004	Washington	Yakima County
540390011	0.004	0.002	0.006	West Virginia	Kanawha County
540391005	0.005	0.003	0.008	West Virginia	Kanawha County
540511002	0.005	0.004	0.006	West Virginia	Marshall County
540690010	0.008	0.005	0.01	West Virginia	Ohio County
550790026	0.004	0.003	0.006	Wisconsin	Milwaukee County
551330027	0.011	0.006	0.013	Wisconsin	Waukesha County
560210100	0.002	0.002	0.002	Wyoming	Laramie County

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-4. Pb-PM_{2.5} concentrations at non-urban PM_{2.5} IMPROVE sites, 2009-2011.

SITE	Max 3-mo Mean (ug/m3)	Annual Mean (ug/m3)	Max Monthly Mean (ug/m3)	State	County
020130002	0.002	0.001	0.004	Alaska	Aleutians East Borough
020680003	0.001	0	0.001	Alaska	Denali Borough
021220009	0.001	0	0.001	Alaska	Kenai Peninsula Borough
040018001	0.001	0.001	0.001	Arizona	Apache County
040038001	0.002	0.001	0.003	Arizona	Cochise County
040039000	0.009	0.004	0.012	Arizona	Cochise County
040070010	0.004	0.003	0.008	Arizona	Gila County
040078100	0.001	0.001	0.002	Arizona	Gila County
040170119	0.001	0.001	0.001	Arizona	Navajo County
051019000	0.002	0.001	0.002	Arkansas	Newton County
051130003	0.001	0.001	0.002	Arkansas	Polk County
060150002	0.001	0	0.002	California	Del Norte County
060270101	0.001	0.001	0.001	California	Inyo County
060430003	0.001	0.001	0.001	California	Mariposa County
060519000	0.001	0	0.001	California	Mono County
060930005	0.001	0	0.001	California	Siskiyou County
061059000	0.001	0	0.001	California	Trinity County
080039000	0.001	0	0.001	Colorado	Alamosa County
080579000	0.001	0	0.001	Colorado	Jackson County
080679000	0.001	0	0.001	Colorado	La Plata County
080839000	0.001	0	0.001	Colorado	Montezuma County
080979000	0.001	0	0.001	Colorado	Pitkin County
081119000	0.001	0	0.001	Colorado	San Juan County
090050005	0.002	0.001	0.002	Connecticut	Litchfield County
120179000	0.002	0.001	0.002	Florida	Citrus County
130499000	0.002	0.001	0.002	Georgia	Charlton County
150019002	0.006	0.001	0.01	Hawaii	Hawaii County
150099000	0	0	0.001	Hawaii	Maui County
160230101	0.001	0	0.001	Idaho	Butte County
160370002	0.001	0	0.001	Idaho	Custer County
191370002	0.003	0.002	0.003	Iowa	Montgomery County
191770006	0.003	0.002	0.005	Iowa	Van Buren County
200139000	0.002	0.002	0.003	Kansas	Brown County
200170001	0.002	0.001	0.002	Kansas	Chase County
201950001	0.001	0.001	0.001	Kansas	Trego County
221279000	0.002	0.001	0.002	Louisiana	Winn Parish
230031020	0.002	0.001	0.002	Maine	Aroostook County
230090103	0.002	0.001	0.002	Maine	Hancock County
230291004	0.001	0.001	0.002	Maine	Washington County
240239000	0.002	0.002	0.002	Maryland	Garrett County
250070001	0.001	0.001	0.002	Massachusetts	Dukes County
260839000	0.001	0.001	0.002	Michigan	Keweenaw County
261539000	0.002	0.001	0.002	Michigan	Schoolcraft County

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-4. Pb-PM_{2.5} concentrations at non-urban PM_{2.5} IMPROVE sites, 2009-2011.

SITE	Max 3-mo Mean (ug/m3)	Annual Mean (ug/m3)	Max Monthly Mean (ug/m3)	State	County
270759000	0.001	0.001	0.002	Minnesota	Lake County
271339000	0.002	0.002	0.003	Minnesota	Rock County
271699000	0.002	0.002	0.003	Minnesota	Winona County
290390001	0.002	0.002	0.002	Missouri	Cedar County
292070001	0.003	0.002	0.004	Missouri	Stoddard County
292130003	0.002	0.002	0.003	Missouri	Taney County
300279000	0.001	0	0.001	Montana	Fergus County
300299001	0.001	0	0.001	Montana	Flathead County
300479000	0.001	0	0.001	Montana	Lake County
300499000	0.001	0	0.001	Montana	Lewis and Clark County
300779000	0.001	0	0.001	Montana	Powell County
300819000	0.001	0	0.001	Montana	Ravalli County
300859000	0.001	0.001	0.001	Montana	Roosevelt County
300870762	0.001	0	0.001	Montana	Rosebud County
300899000	0.001	0	0.001	Montana	Sanders County
300919000	0.001	0.001	0.001	Montana	Sheridan County
310699000	0.001	0.001	0.001	Nebraska	Garden County
311719000	0.001	0.001	0.001	Nebraska	Thomas County
320079000	0.001	0	0.001	Nevada	Elko County
320339000	0.001	0	0.001	Nevada	White Pine County
330074002	0.001	0.001	0.001	New Hampshire	Coos County
350039000	0.001	0.001	0.001	New Mexico	Catron County
350059000	0.001	0.001	0.001	New Mexico	Chaves County
350279000	0.001	0.001	0.001	New Mexico	Lincoln County
350281002	0.001	0	0.001	New Mexico	Los Alamos County
350399000	0.001	0	0.001	New Mexico	Rio Arriba County
350539000	0.001	0.001	0.001	New Mexico	Socorro County
350559000	0.001	0	0.001	New Mexico	Taos County
361019000	0.003	0.002	0.006	New York	Steuben County
370110002	0.001	0.001	0.002	North Carolina	Avery County
370959000	0.002	0.001	0.002	North Carolina	Hyde County
380070002	0.001	0.001	0.001	North Dakota	Billings County
380130004	0.001	0.001	0.001	North Dakota	Burke County
391219000	0.005	0.003	0.008	Ohio	Noble County
400450890	0.001	0.001	0.001	Oklahoma	Ellis County
400719010	0.002	0.001	0.003	Oklahoma	Kay County
410330010	0.001	0.001	0.001	Oregon	Josephine County
410358001	0.001	0	0.001	Oregon	Klamath County
410610010	0.001	0	0.001	Oregon	Union County
410630002	0.001	0	0.001	Oregon	Wallowa County
420019000	0.003	0.002	0.003	Pennsylvania	Adams County
460330132	0.001	0	0.001	South Dakota	Custer County
460710001	0.001	0.001	0.001	South Dakota	Jackson County

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-4. Pb-PM_{2.5} concentrations at non-urban PM_{2.5} IMPROVE sites, 2009-2011.

SITE	Max 3-mo Mean (ug/m3)	Annual Mean (ug/m3)	Max Monthly Mean (ug/m3)	State	County
480430101	0.002	0.001	0.002	Texas	Brewster County
481099000	0.001	0.001	0.001	Texas	Culberson County
490170101	0.001	0	0.001	Utah	Garfield County
490379000	0.001	0	0.001	Utah	San Juan County
490559000	0.001	0	0.001	Utah	Wayne County
500038001	0.001	0.001	0.002	Vermont	Bennington County
511139000	0.002	0.001	0.002	Virginia	Madison County
511639000	0.004	0.003	0.006	Virginia	Rockbridge County
530090020	0.001	0.001	0.002	Washington	Clallam County
530370004	0.001	0.001	0.001	Washington	Kittitas County
530390010	0.002	0.001	0.002	Washington	Klickitat County
530390011	0.001	0.001	0.001	Washington	Klickitat County
530410007	0.001	0	0.001	Washington	Lewis County
530470012	0.001	0	0.001	Washington	Okanogan County
540939000	0.002	0.001	0.002	West Virginia	Tucker County
560050123	0.001	0	0.001	Wyoming	Campbell County
560199000	0.001	0	0.001	Wyoming	Johnson County
560299002	0.001	0	0.001	Wyoming	Park County
560359000	0.001	0	0.001	Wyoming	Sublette County
560399000	0.001	0	0.001	Wyoming	Teton County
780209000	0.001	0.001	0.001		

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-5. Distribution of maximum 3-month means, 2009-2011.

(units are ug/m3)	n	min	Pct1	Pct5	Pct10	Pct25	mean	Pct50	Pct75	Pct90	Pct95	Pct99	max
Pb-TSP all	215	0	0.001	0.004	0.006	0.013	0.154	0.04	0.116	0.421	0.943	1.432	2.07
Pb-TSP source	112	0.004	0.004	0.012	0.019	0.05	0.273	0.11	0.355	0.774	1.079	1.937	2.07
Pb-TSP prev source	12	0.006	0.006	0.006	0.023	0.0295	0.057	0.06	0.082	0.093	0.115	0.115	0.115
Pb-TSP not source	91	0	0	0.002	0.003	0.007	0.019	0.012	0.025	0.041	0.047	0.132	0.132
Pb-PM10 urban	31	0.002	0.002	0.002	0.003	0.003	0.007	0.005	0.007	0.013	0.019	0.023	0.023
Pb-PM2.5 urban CSN	198	0.001	0.002	0.002	0.003	0.003	0.007	0.004	0.007	0.013	0.02	0.042	0.07
Pb-PM2.5 non-urban													
IMPROVE	107	0	0.001	0.001	0.001	0.001	0.002	0.001	0.002	0.002	0.003	0.006	0.009

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-6. Distribution of annual mean concentrations, 2009-2011.

(units are ug/m ³)	n	min	Pct1	Pct5	Pct10	Pct25	mean	Pct50	Pct75	Pct90	Pct95	Pct99	max
Pb-TSP all	215	0	0	0.002	0.004	0.008	0.0798	0.022	0.058	0.198	0.434	0.889	0.959
Pb-TSP source	112	0.003	0.003	0.005	0.013	0.027	0.1408	0.053	0.149	0.394	0.622	0.921	0.959
Pb-TSP prev source	12	0.004	0.004	0.004	0.015	0.02	0.0306	0.033	0.04	0.041	0.058	0.058	0.058
Pb-TSP not source	91	0	0	0	0.002	0.004	0.0113	0.008	0.016	0.023	0.041	0.05	0.05
Pb-PM10 urban	35	0.001	0.001	0.001	0.001	0.002	0.0043	0.003	0.005	0.011	0.012	0.015	0.015
Pb-PM2.5 urban CSN	198	0.001	0.001	0.002	0.002	0.002	0.0036	0.003	0.004	0.006	0.009	0.017	0.018
Pb-PM2.5 non-urban													
IMPROVE	107	0	0	0	0	0	0.0008	0.001	0.001	0.002	0.002	0.003	0.004

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-7. Distribution of lead concentrations, 2009-2011.

(units are ug/m ³)	n	min	Pct1	Pct5	Pct10	Pct25	mean	Pct50	Pct75	Pct90	Pct95	Pct99	max
Pb-TSP all	215	0	0.002	0.006	0.008	0.016	0.228	0.056	0.21	0.605	1.178	2.185	2.821
Pb-TSP source	112	0.006	0.007	0.015	0.029	0.0765	0.402	0.16	0.509	1.124	1.623	2.438	2.821
Pb-TSP prev source	12	0.008	0.008	0.008	0.03	0.0385	0.09	0.068	0.1225	0.14	0.271	0.271	0.271
Pb-TSP not source	91	0	0	0.003	0.006	0.01	0.032	0.016	0.035	0.057	0.087	0.364	0.364
Pb-PM10 urban	35	0.002	0.002	0.003	0.003	0.004	0.01	0.007	0.012	0.028	0.031	0.035	0.035
Pb-PM2.5 urban CSN	198	0.001	0.002	0.003	0.003	0.004	0.011	0.007	0.011	0.021	0.033	0.078	0.199
Pb-PM2.5 non-urban													
IMPROVE	107	0.001	0.001	0.001	0.001	0.001	0.002	0.001	0.002	0.003	0.006	0.01	0.012

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-8. Pb-TSP metrics (2009-2011).

Monitoring SITE ID	CBSA_gt_1M (2010 Census)	(3-yr) - annual mean (ug/m3)	(3-yr) - max 3-month avg (ug/m3)	ratio of 3-yr max 3-month average to 3-year average annual mean	Ratios for CBSA_gt_1M (2010 Census)
011090003		0.564	1.035	1.8351	
020200051					
021880001					
040071002		0.133	0.267	2.0075	
040078000		0.033	0.061	1.8485	
060250005		0.015	0.023	1.5333	
060371103	1	0.011	0.016	1.4545	1.4545
060371302	1	0.01	0.019	1.9000	1.9000
060371402	1	0.022	0.044	2.0000	2.0000
060371403	1	0.061	0.116	1.9016	1.9016
060371404	1	0.061	0.108	1.7705	1.7705
060371405	1	0.343	0.667	1.9446	1.9446
060371406	1	0.049	0.085	1.7347	1.7347
060371602	1	0.011	0.022	2.0000	2.0000
060374002	1	0.006	0.01	1.6667	1.6667
060374004	1	0.007	0.012	1.7143	1.7143
060375005	1	0.003	0.008	2.6667	2.6667
060651003	1	0.006	0.011	1.8333	1.8333
060658001	1	0.007	0.011	1.5714	1.5714
060711004	1	0.007	0.011	1.5714	1.5714
060719004	1	0.008	0.013	1.6250	1.6250
080050007	1	0.014	0.02	1.4286	1.4286
080310025	1	0.006	0.01	1.6667	1.6667
120570100	1	0.026	0.044	1.6923	1.6923
120571066	1	0.282	0.979	3.4716	3.4716
120571073	1	0.116	0.423	3.6466	3.6466
130150003	1	0.014	0.019	1.3571	1.3571
130890003	1	0.003	0.004	1.3333	1.3333
132150010					
132150011		0.029	0.071	2.4483	
170310001	1	0.016	0.024	1.5000	1.5000
170310022	1	0.033	0.051	1.5455	1.5455
170310026	1	0.023	0.034	1.4783	1.4783
170310052	1	0.016	0.024	1.5000	1.5000
170310110	1	0.1	0.294	2.9400	2.9400
170310210	1	0.037	0.053	1.4324	1.4324
170313103	1	0.011	0.013	1.1818	1.1818
170313301	1	0.018	0.027	1.5000	1.5000
170314201	1	0.01	0.011	1.1000	1.1000
170316003	1	0.024	0.036	1.5000	1.5000
171150110		0.082	0.199	2.4268	

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-8. Pb-TSP metrics (2009-2011).

Monitoring SITE ID	CBSA_gt_1M (2010 Census)	(3-yr) - annual mean (ug/m3)	(3-yr) - max 3-month avg (ug/m3)	ratio of 3-yr max 3-month average to 3-year average annual mean	Ratios for CBSA_gt_1M (2010 Census)
171170002	1	0.01	0.011	1.1000	1.1000
171190010	1	0.118	0.416	3.5254	3.5254
171193007	1	0.018	0.036	2.0000	2.0000
171430037		0.01	0.011	1.1000	
171430110		0.013	0.016	1.2308	
171430210		0.011	0.015	1.3636	
171630010	1	0.019	0.029	1.5263	1.5263
171950110		0.02	0.028	1.4000	
172010110		0.031	0.063	2.0323	
180350008		0.079	0.11	1.3924	
180350009		0.186	0.262	1.4086	
180890023	1	0.026	0.077	2.9615	2.9615
180890032	1	0.025	0.06	2.4000	2.4000
180890033	1	0.054	0.139	2.5741	2.5741
180892008	1	0.016	0.037	2.3125	2.3125
180970063	1	0.026	0.079	3.0385	3.0385
180970076	1	0.01	0.02	2.0000	2.0000
180970078	1	0.005	0.011	2.2000	2.2000
181270023	1	0.013	0.022	1.6923	1.6923
181270027					
181630006					
181630020		0.004	0.007	1.7500	
191550011		0.122	0.263	2.1557	
191630015		0.01	0.012	1.2000	
201690004		0.18	0.421	2.3389	
210190016		0.003	0.004	1.3333	
211510003		0.034	0.069	2.0294	
212070001		0.021	0.036	1.7143	
220330014		0.004	0.005	1.2500	
220950003	1	0.037	0.053	1.4324	1.4324
250250002					
260290011					
260670002					
260670003		0.126	0.284	2.2540	
260810020		0.005	0.008	1.6000	
261250013					
261570001					
261630001	1	0.005	0.006	1.2000	1.2000
261630033	1	0.012	0.023	1.9167	1.9167
270036020	1	0.017	0.033	1.9412	1.9412
270072303		0	0.001	0.0000	

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-8. Pb-TSP metrics (2009-2011).

Monitoring SITE ID	CBSA_gt_1M (2010 Census)	(3-yr) - annual mean (ug/m3)	(3-yr) - max 3-month avg (ug/m3)	ratio of 3-yr max 3-month average to 3-year average annual mean	Ratios for CBSA_gt_1M (2010 Census)
270210001		0	0.001	0.0000	
270370020	1	0.005	0.011	2.2000	2.2000
270370423	1	0.001	0.002	2.0000	2.0000
270370442	1	0.003	0.013	4.3333	4.3333
270370443	1	0.002	0.003	1.5000	1.5000
270370465	1	0.128	0.259	2.0234	2.0234
270370470	1	0.001	0.002	2.0000	2.0000
270530963	1	0.003	0.006	2.0000	2.0000
270530966	1	0.004	0.016	4.0000	4.0000
270531007	1	0.002	0.004	2.0000	2.0000
270532006	1	0.001	0.003	3.0000	3.0000
271230866	1	0.012	0.016	1.3333	1.3333
271230871	1	0.002	0.007	3.5000	3.5000
271231003	1	0.004	0.006	1.5000	1.5000
271377001		0.001	0.003	3.0000	
271377555		0.002	0.006	3.0000	
271453053		0.003	0.006	2.0000	
271630009	1	0.003	0.005	1.6667	1.6667
271630438	1	0.002	0.005	2.5000	2.5000
271630446	1	0	0.002	0.0000	0.0000
290930016		0.49	2.07	4.2245	
290930021		0.622	1.937	3.1141	
290930027		0.038	0.083	2.1842	
290930029		0.037	0.115	3.1081	
290930032					
290930033		0.027	0.057	2.1111	
290930034		0.283	0.402	1.4205	
290939007		0.392	0.581	1.4821	
290939008		0.256	0.387	1.5117	
290970005		0.013	0.019	1.4615	
290990004	1	0.889	1.16	1.3048	1.3048
290990005	1	0.231	0.396	1.7143	1.7143
290990009	1	0.043	0.067	1.5581	1.5581
290990011	1	0.21	0.403	1.9190	1.9190
290990013	1	0.116	0.198	1.7069	1.7069
290990015	1	0.921	1.079	1.1716	1.1716
290990020	1	0.646	0.943	1.4598	1.4598
290990021	1	0.585	0.969	1.6564	1.6564
290990022	1	0.434	0.63	1.4516	1.4516
290990023	1	0.394	0.594	1.5076	1.5076
290990024	1	0.374	0.659	1.7620	1.7620

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-8. Pb-TSP metrics (2009-2011).

Monitoring SITE ID	CBSA_gt_1M (2010 Census)	(3-yr) - annual mean (ug/m3)	(3-yr) - max 3-month avg (ug/m3)	ratio of 3-yr max 3-month average to 3-year average annual mean	Ratios for CBSA_gt_1M (2010 Census)
290990025	1	0.043	0.085	1.9767	1.9767
290990026	1	0.035	0.072	2.0571	2.0571
290990027					
290999001	1	0.824	1.204	1.4612	1.4612
290999002	1	0.232	0.431	1.8578	1.8578
290999003	1	0.198	0.467	2.3586	2.3586
290999004	1	0.119	0.261	2.1933	2.1933
290999005	1	0.959	1.432	1.4932	1.4932
290999006	1	0.049	0.091	1.8571	1.8571
291790001		0.033	0.059	1.7879	
291790002		0.033	0.068	2.0606	
291790003		0.027	0.1	3.7037	
291790034		0.068	0.093	1.3676	
291870006		0.05	0.132	2.6400	
291870007		0.027	0.041	1.5185	
291892003	1	0.007	0.041	5.8571	5.8571
295100085					
310530005		0.051	0.072	1.4118	
311270002		0.042	0.115	2.7381	
360470122	1	0.014	0.019	1.3571	1.3571
360713001		0.031	0.101	3.2581	
360713002		0.128	1.027	8.0234	
360713004		0.005	0.012	2.4000	
361030024					
390170015	1	0.006	0.009	1.5000	1.5000
390290019		0.022	0.057	2.5909	
390290020		0.015	0.025	1.6667	
390290022		0.02	0.044	2.2000	
390350038	1	0.013	0.021	1.6154	1.6154
390350042	1	0.014	0.03	2.1429	2.1429
390350049	1	0.194	0.531	2.7371	2.7371
390350060					
390350061	1	0.015	0.023	1.5333	1.5333
390350072	1	0.014	0.035	2.5000	2.5000
390490025	1	0.008	0.011	1.3750	1.3750
390510001		0.073	0.178	2.4384	
390910006		0.004	0.006	1.5000	
391010003		0.035	0.056	1.6000	
391137001					
391510017		0.014	0.023	1.6429	
391550012		0.007	0.011	1.5714	

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-8. Pb-TSP metrics (2009-2011).

Monitoring SITE ID	CBSA_gt_1M (2010 Census)	(3-yr) - annual mean (ug/m3)	(3-yr) - max 3-month avg (ug/m3)	ratio of 3-yr max 3-month average to 3-year average annual mean	Ratios for CBSA_gt_1M (2010 Census)
391670008		0.005	0.007	1.4000	
391670010		0.005	0.008	1.6000	
401159006		0.011	0.015	1.3636	
401159007		0.017	0.034	2.0000	
401210416		0.003	0.004	1.3333	
401431127		0.004	0.005	1.2500	
410711702	1	0.018	0.035	1.9444	1.9444
420030002	1	0.012	0.016	1.3333	1.3333
420030008	1	0.01	0.014	1.4000	1.4000
420030070	1	0.022	0.056	2.5455	2.5455
420031009	1	0.043	0.138	3.2093	3.2093
420070006	1	0.054	0.085	1.5741	1.5741
420070007	1	0.171	0.253	1.4795	1.4795
420070505	1	0.09	0.115	1.2778	1.2778
420110005		0.043	0.051	1.1860	
420110020		0.127	0.196	1.5433	
420110021		0.049	0.139	2.8367	
420110022		0.043	0.118	2.7442	
420110717		0.065	0.116	1.7846	
420111717		0.133	0.251	1.8872	
420210808		0.05	0.073	1.4600	
420450002	1	0.043	0.047	1.0930	1.0930
420450004	1	0.045	0.047	1.0444	1.0444
420550002		0.045	0.046	1.0222	
420630005		0.045	0.049	1.0889	
420730011		0.019	0.023	1.2105	
420790036		0.095	0.137	1.4421	
421010449	1	0.021	0.03	1.4286	1.4286
421011002	1	0.022	0.025	1.1364	1.1364
421290007	1	0.041	0.043	1.0488	1.0488
421290009	1	0.027	0.046	1.7037	1.7037
450190003		0.003	0.007	2.3333	
450450015		0.003	0.007	2.3333	
450790007		0.004	0.005	1.2500	
450790019		0.005	0.024	4.8000	
470930023		0.114	0.165	1.4474	
470930027		0.02	0.042	2.1000	
470931017		0.018	0.037	2.0556	
471633001		0.058	0.093	1.6034	
471633002		0.039	0.06	1.5385	
471633003		0.041	0.06	1.4634	

Appendix 2D - Air Quality Data Analysis Summary

Table 2D-8. Pb-TSP metrics (2009-2011).

Monitoring SITE ID	CBSA_gt_1M (2010 Census)	(3-yr) - annual mean (ug/m3)	(3-yr) - max 3-month avg (ug/m3)	ratio of 3-yr max 3-month average to 3-year average annual mean	Ratios for CBSA_gt_1M (2010 Census)
471633004		0.041	0.08	1.9512	
471634002		0.025	0.038	1.5200	
480610006		0.004	0.005	1.2500	
480850003	1	0.179	0.371	2.0726	2.0726
480850007	1	0.105	0.199	1.8952	1.8952
480850009	1	0.453	0.774	1.7086	1.7086
480850029	1	0.076	0.18	2.3684	2.3684
481130069					
481410002		0.023	0.04	1.7391	
481410033		0.019	0.039	2.0526	
481410055		0.009	0.014	1.5556	
481410058		0.017	0.033	1.9412	
482011034	1	0.005	0.008	1.6000	1.6000
482011039					
482570020	1	0.06	0.104	1.7333	1.7333
483750024		0.006	0.013	2.1667	
484790016		0.016	0.026	1.6250	
490351001	1	0.023	0.057	2.4783	2.4783
510090007		0.004	0.018	4.5000	
510270006		0.01	0.013	1.3000	
510870014	1	0.004	0.011	2.7500	2.7500
517700011		0.038	0.109	2.8684	
530330029					
530610013					
551170008		0.055	0.152	2.7636	
720130001		0.188	0.339	1.8032	
720210010		0.005	0.011	2.2000	
counts	114	212	212		
average --->				1.9463	1.9203

APPENDIX 3A

INTERPOLATED RISK ESTIMATES FOR THE GENERALIZED (LOCAL) URBAN CASE STUDY

This Appendix describes the method used to develop risk estimates for conditions just meeting the current standard ($0.15 \mu\text{g}/\text{m}^3$, as a maximum 3-month average) for the generalized (local) urban case study. These risk estimates were developed by interpolation from the 2007 REA results for this case study. The general approach was to identify the two alternative standard scenarios simulated in the 2007 REA which represented air quality conditions bracketing those for the current standard and then linearly interpolate an estimate of risk for the current standard based on the slope created from the two bracketing estimates. In representing air quality conditions for these purposes, we focused on the annual average air Pb concentration estimates as that is the metric which had been the IEUBK model inputs for the various air quality scenarios (IEUBK does not accept air quality inputs of a temporal scale shorter than a year).¹ An annual average concentration estimate to represent the current standard was identified in a manner consistent with that employed in the 2007 REA for this case study (see section 3.4.3.2 above, use of 2003-2005 data) with the use of currently available monitoring data (2009-2011) for relationships between air quality metrics for representation of the current standard. By this method, the air quality scenario for the current standard ($0.15 \mu\text{g}/\text{m}^3$, as a not-to-be-exceeded 3-month average) was found to be bracketed by the scenarios for alternative standards of 0.5 and $0.20 \mu\text{g}/\text{m}^3$ (maximum monthly averages). A risk estimate for the current standard was then derived using the slope relating generalized (local) urban case study IQ loss to the annual average Pb concentration used for those two air quality scenarios. We used this interpolation approach to develop median risk estimates for the current standard based on each of the four C-R functions. Details on the method for the interpolation approach are provided below.

1. *Identify an estimate of annual average air Pb concentration to represent each air quality scenario.* For the alternative scenarios, this was done in the 2007 REA using the 2003-2005 Pb-TSP dataset for urban areas of population greater than one million. For analysis

¹ Although many different patterns of temporally varying air concentration will just meet a given potential alternative standard, the shortest time step accommodated by the blood Pb model is a year. Thus, the air Pb concentration inputs to the blood Pb model for each air quality scenario are annual average air Pb concentrations. For the generalized (local) urban case study, the national Pb-TSP monitoring dataset was analyzed to characterize the distribution of site-specific relationships between metrics reflecting the averaging time and form for the air quality scenarios being assessed (Table 3-8 of this document) and the annual average. The IEUBK annual average input was then derived by multiplying the level for a given air quality scenario by the ratio for the averaging time and form for that air quality scenario. For the location-specific case studies, however, the full temporally varying air Pb concentration dataset for each exposure zone was used to derive the average annual concentration for the IEUBK input.

of the current standard here, this uses the recent Pb-TSP dataset (2009-2011) for urban areas of population greater than one million. The concentration in terms of the metric being assessed (e.g., maximum 3-month or calendar quarter average) was derived per monitoring site, as was the annual average concentration and also the ratio of the two (2007 REA, Appendix A; Appendix 2D of this document). From the average of the monitor-specific ratios we derived the annual average estimate. [In the 2007 REA, this was the IEUBK air quality input for each AQ scenario.] With the 2009-2011 data, the average, across monitors in urban areas greater than 1 million population, of the ratio of maximum 3-month average to annual average is 1.92 (Appendix 2D in this document).

- This ratio was used to derive an annual average air Pb-TSP concentration estimate to represent the current standard scenario (annual value = $0.15 \mu\text{g}/\text{m}^3 * 1/1.92 = 0.078 \mu\text{g}/\text{m}^3$). The annual average values for the scenarios included in the 2007 REA and also for the current standard scenario considered in this interpolation are shown in Table 3A-1 below.

Table 3A-1. Annual average air Pb-TSP concentration estimates for different air quality scenarios.

Air Quality Scenarios			Annual Average Estimate ($\mu\text{g}/\text{m}^3$)
Maximum Quarterly Average ^D ($\mu\text{g}/\text{m}^3$)	Maximum Monthly Average ($\mu\text{g}/\text{m}^3$)	Maximum 3-month Average ($\mu\text{g}/\text{m}^3$)	
1.5 (previous NAAQS)			0.60
	0.5		0.130
0.2			0.08
		0.15	0.078*
	0.2		0.05
	0.05		0.013
	0.02		0.005

* Derived as described in step 1 using 2009-2011 air quality dataset.

- Identify the two “bounding” alternative standard levels that will be used to derive a slope for the risk interpolation for the current standard level. Based on comparison of the annual ambient air Pb estimates for each of the 2007 REA air quality scenarios and for the current standard we determine which scenarios “bound” the current standard (in terms of the annual average ambient Pb estimate). As Table 3A-1 above shows, the bounding scenarios are the scenarios for just meeting maximum monthly average concentrations of 0.5 and $0.2 \mu\text{g}/\text{m}^3$.
- Calculate the slopes of generalized (local) urban case study IQ loss per unit annual average Pb estimate for each risk estimate of interest for the two bounding scenarios. This calculation is $(\text{IQ LOSS}_{\text{Scenario X}} - \text{IQ LOSS}_{\text{Scenario Y}}) / (\text{annual average}_{\text{Scenario X}} - \text{annual average}_{\text{Scenario Y}})$. The risk estimates for the 4 different C-R functions and two exposure pathway categories of interest (*Recent Air* and *Recent + Past Air*), with the derived slopes, are in Table 3A-2.

1 **Table 3A-2. Risk estimates for bounding air quality scenarios and associated slopes.**

	Risk Estimates for different C-R functions and exposure pathway categories							
	Log-linear with low-exposure linearization		Dual linear – stratified at 10 µg/dL peak		Log-linear with cutpoint		Dual linear - stratified at 7.5 µg/dL peak	
	Recent air	Recent + past air	Recent air	Recent + past air	Recent air	Recent + past air	Recent air	Recent + past air
0.2 µg/m ³ , max quarterly average	1.53	3.37	0.54	1.18	0.63	1.38	1.97	4.34
0.2 µg/m ³ max monthly average	1.21	3.16	0.41	1.08	0.47	1.22	1.53	3.99
Slope *	10.69	7.03	4.10	3.38	5.31	5.28	14.77	11.66
* In terms of points IQ loss per unit annual average air Pb in generalized (local) urban case study (see texts for explanation).								

2

3 4. *Derive interpolated risk estimates for the current standard scenario.* Using the
4 slopes presented in Table 3A-2 and the lower bounding air quality scenario risk
5 estimates, derive corresponding risk estimates for the current standard scenario:

6 Risk = lower bounding risk + (increment annual average Pb * slope)

7 Where:

- 8 ■ lower bounding risk = risk for the lower bound air quality scenario (i.e.,
9 for 0.2 µg/m³ maximum monthly average)
- 10 ■ increment annual average Pb is the difference between the annual average
11 estimates for the current standard and the lower bounding scenario.
- 12 ■ slope is the value described in Step 4, which differs across the 8
13 combinations of C-R functions and exposure pathway categories.

14 The resulting interpolated risk estimates are presented in Table 3A-3.

15 **Table 3A-3. Interpolated risk estimates for the current NAAQS scenario for the**
16 **generalized (local) urban case study.**

	Risk Estimates for different C-R functions and exposure pathway categories							
	Log-linear with low-exposure linearization		Dual linear – stratified at 10 µg/dL peak		Log-linear with cutpoint		Dual linear - stratified at 7.5 µg/dL peak	
	Recent air	Recent + past air	Recent air	Recent + past air	Recent air	Recent + past air	Recent air	Recent + past air
Interpolated Estimate*	1.51	3.36	0.53	1.17	0.62	1.37	1.94	4.32
* Points IQ loss in generalized (local) urban case study (see text for explanation).								

17

1
2
3

APPENDIX 5A

ADDITIONAL DETAIL ON 2006 ECOLOGICAL SCREENING ASSESSMENT

	Setting and spatial extent of dataset or modeling analysis	Media Screened and Screening Levels Used		
		Soil	Freshwater water column	Freshwater Sediment
Primary Smelter Case Study	Herculaneum, Missouri: soil and waterbody samples from study area 6 km diameter, centered on point source	Soil screening values developed based on U.S. EPA Superfund methodology for developing ecological soil screening levels (USEPA, 2005a,b)	U.S. EPA freshwater AWQC for aquatic life adjusted for site-specific water hardness	Sediment screening values based on MacDonald et al. (2000) sediment quality assessment guidelines
Secondary Smelter Case Study	Troy, Alabama: soil concentrations in census blocks near facility predicted from dispersion and soil mixing model		NA	NA
Near Roadway Non-Urban Case Study	Two datasets: Corpus Christi, Texas (within 4 m from road), and Atlee, Virginia (within 2 to 30 m distance from road)			
Vulnerable Ecosystem Case Study	Hubbard Brook Experimental Forest, New Hampshire: forest in oblong basin about 8 km long by 5 km wide	While no quantitative analyses were performed, summary review of literature search indicated: (1) atmospheric Pb inputs do not directly affect stream Pb levels at HBEF because deposited Pb is almost entirely retained in the soil profile; (2) soil horizon analysis results show Pb has become more concentrated at lower depths over time and that the soil profile serves as a Pb sink, appreciably reducing Pb in porewater as it moves through the soil layers to streams, (3) dissolved Pb concentrations were reduced (5 ppb to about 5 ppt) as Pb moves from the Oa horizon to streams. Studies concluded insignificant contribution of dissolved Pb from soils to streams (less than $0.2 \text{ g} \cdot \text{ha}^{-1} \cdot \text{yr}^{-1}$). (ICF, 2006, Appendix E)		
National Surface Water Screen	Surface water bodies in the 47 basin study units from all regions of the United States, covering approx. 50% of U.S. land base	NA	U.S. EPA freshwater AWQC for aquatic life adjusted for hardness at site or nearby water body	Sediment screening values based on MacDonald et al. (2000) sediment quality assessment guidelines

AWQC= Ambient water quality criteria. NA = Not applicable; medium not part of case study.
NOTE: Information here is drawn from ICF, 2006 and EPA, 2007b.

4

United States
Environmental Protection
Agency

Office of Air Quality Planning and Standards
Health and Environmental Impacts Division
Research Triangle Park, NC

Publication No. EPA-452/P-13-001
January 2013
