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

U.S. Environmental Protection Agency
Office of Air Quality Planning and Standards
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ACKNOWLEDGMENTS

This 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. 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.

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EXECUTIVE SUMMARY

This 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 staff conclusions regarding the policy implications of the key scientific and technical information that informs this review. The 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 PA are based on the scientific assessment presented in the *Integrated Science Assessment for Lead* (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. For the purpose of review of the NAAQS 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 PA to address such questions lead to 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 the draft PA, have informed staff conclusions and the presentation of information in this final document.

Current Lead NAAQS and Scope of Review

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

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

Characterization of Ambient Air Lead

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

As at the time of the last review, the majority of Pb emissions nationally is associated with combustion of leaded aviation gasoline by piston-driven aircraft. The largest sources on a local scale are generally associated with metals industries. As a result of revisions to monitoring regulations stemming from the last Pb NAAQS review, Pb NAAQS monitors are required near the largest Pb emissions sources, as well as at sites distant from such sources in large population areas. Ambient air Pb monitoring data available thus far from this expanded network continue to illustrate the source-related aspect of airborne Pb, with highest concentrations near large sources and lowest in areas removed from sources. In addition, Pb monitoring data are also being collected over at least a one-year period near a set of airports identified as most likely to have elevated Pb concentrations due to leaded aviation gasoline usage. These data will improve our understanding of Pb concentrations in ambient air near airports and conditions influencing these concentrations. These data will also inform EPA's ongoing investigation into Pb emissions from piston-engine aircraft under Section 231 of the CAA, separate from this Pb NAAQS review.

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

Health Effects and Review of the Primary Standard

Lead has long been recognized to exert an array of deleterious effects on multiple organ systems as described in the ISA for this review and consistent with conclusions of prior scientific assessments. Over the three decades from the time the standard was initially set in 1978 through its revision with the NAAQS review completed in 2008, the evidence base expanded considerably in a number of areas, including with regard to effects on neurocognitive function in young children at increasingly lower blood Pb levels. These effects formed the primary basis for the 2008 revisions to the primary standard. The current standard was set most specifically to provide appropriate public health protection from the effects of air-related Pb on cognitive function (e.g., IQ loss) in young children. In so doing, the standard was judged to provide the requisite public health protection from the full array of health effects of Pb, consistent with the CAA requirement that the primary standard, in the judgment of the Administrator, based on the latest scientific knowledge, be requisite to protect public health with an adequate margin of safety.

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

In also considering the quantitative risk estimates associated with the current standard, based on the risk assessment conducted in the last review, staff observes that these estimates indicate a level of risk that is roughly consistent with and generally supportive of conclusions drawn from the evidence using the evidence-based air-related IQ loss framework. Staff

additionally recognizes the complexity of the modeling done as part of that assessment and the substantial limitations and uncertainties in the resulting risk estimates.

Based on the above considerations, staff concludes that the currently available information supports a primary standard as protective as the current standard and that it is appropriate to consider retaining the current standard without revision. In so doing, staff additionally notes that the final decision on the adequacy of the current standard is largely a public health policy judgment to be made by the Administrator, drawing upon the scientific information as well as judgments about how to consider the range and magnitude of uncertainties that are inherent in the scientific evidence and technical analyses. In this context, staff recognizes that the uncertainties and limitations associated with the many aspects of the relationship between air Pb concentrations and blood Pb levels and associated health effects are amplified with consideration of increasingly lower air concentrations. In staff's view, based on the current evidence there is appreciable uncertainty associated with drawing conclusions regarding whether there would be reductions in blood Pb levels and risk to public health from alternative lower levels as compared to the level of the current standard. Thus, staff concludes that the basis for any consideration of alternative lower standard levels would reflect different public health policy judgments as to the appropriate approach for weighing uncertainties in the evidence and for providing requisite protection of public health with an adequate margin of safety. Accordingly, and in light of the staff conclusion that it is appropriate to consider the current standard to be adequate, this document does not identify potential alternative standards for consideration in this review.

Welfare Effects and Review of the Secondary Standard

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

In assessing the currently available scientific evidence and the exposure/risk information with regard to support for the adequacy of the protection afforded by the current standard, staff observes the general consistency of the current evidence with that available in the last review, including that with regard to ecological effects associated with Pb exposure and the substantial limitations in the current evidence that complicate conclusions regarding the potential for Pb emissions under the current, much lower standard to contribute to welfare effects. Such complications include the significant difficulties in interpreting effects evidence from laboratory

studies to the natural environment and linking those effects to ambient air Pb concentrations. Based on staff analysis, including the above considerations, staff concludes that the currently available evidence and exposure/risk information do not call into question the adequacy of the current standard to provide the requisite protection for public welfare. Thus, staff concludes that consideration should be given to retaining the current standard, without revision, and this document does not identify potential alternative standards for consideration in this review.

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.

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 current standards 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.

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[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 NAAQS for fine particulate matter ($PM_{2.5}$) is the average of annual mean $PM_{2.5}$ concentrations for three consecutive years, while the form of the 8-hour NAAQS for carbon monoxide is the second-highest 8-hour average in a year.

The development of the PA is also intended to facilitate advice to the Agency and recommendations to the Administrator from an independent scientific review committee, the Clean Air Scientific Advisory Committee (CASAC), as provided for in the Clean Air Act. As discussed below in section 1.2.1, the CASAC is to advise not only on the Agency's assessment of the relevant scientific information, but also on the adequacy of the existing standards, and to make recommendations as to any revisions of the standards that may be appropriate. The EPA facilitates CASAC advice and recommendations, as well as public input and comment, by requesting CASAC review and public comment on one or more drafts of the PA.[4] In this PA for this review of the Pb NAAQS, we consider the scientific and technical information available in this review as assessed in the *Integrated Science Assessment for Lead* (henceforth referred to as the ISA [USEPA, 2013a]), prepared by EPA's National Center for Environmental Assessment (NCEA), and the quantitative human exposure and health risk and screening-level ecological risk assessments performed in the last review. As discussed below in section 1.2.3, upon consideration of the evidence newly available in this review with regard to risk and exposure assessment, staff concluded that new health and welfare REAs were not warranted. Accordingly, the quantitative risk information considered in this PA is based on the quantitative human exposure and health risk and screening-level ecological risk assessments performed in the last review (the 2007 Health Risk Assessment Report or 2007 REA [USEPA, 2007a] and the 2006 screening-level Ecological Risk Assessment or 2006 REA [ICF, 2006]) and is interpreted in the context of newly available evidence in this review. The evaluation and staff conclusions presented in this PA for the Pb NAAQS have been informed by comments and advice received from CASAC in their reviews of the draft PA and of other draft Agency documents prepared in this NAAQS review.

Beyond informing the EPA Administrator and facilitating the advice and recommendations of CASAC and the public, the PA is also intended to be a useful reference to all parties interested in the Pb NAAQS review. In these roles, it is intended to serve as a single source of the most policy-relevant information that informs the Agency's review of the Pb NAAQS, and it is written to be understandable to a broad audience.

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

1.2 BACKGROUND

1.2.1 Legislative Requirements

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

The requirement that primary standards provide 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. See *Lead Industries Association v. EPA*, 647 F.2d 1130, 1154 (D.C. Cir 1980), *cert. denied*, 449 U.S. 1042 (1980); *American Petroleum Institute v. Costle*, 665 F.2d 1176, 1186 (D.C. Cir. 1981), *cert. denied*, 455 U.S. 1034 (1982); *American Farm Bureau Federation v. EPA,* 559 F. 3d 512, 533 (D.C. Cir. 2009); *Association of Battery Recyclers v. EPA*, 604 F. 3d 613, 617-18 (D.C. Cir. 2010). Both kinds of uncertainties are components of the risk associated with pollution at levels below those

[5] 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).

[6] 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."

at which human health effects can be said to occur with reasonable scientific certainty. Thus, in selecting primary standards that provide an adequate margin of safety, the Administrator is seeking not only to prevent pollution 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 the Administrator to establish a primary NAAQS at a zero-risk level or at background concentration levels, see *Lead Industries v. EPA*, 647 F.2d at 1156 n.51, but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety.

In addressing the requirement for an adequate margin of safety, the EPA considers such factors as the nature and severity of the health effects involved, the size of sensitive population(s) at risk,[7] and the kind and degree of the uncertainties that must be addressed. The selection of any particular approach to providing an adequate margin of safety is a policy choice left specifically to the Administrator's judgment. See *Lead Industries Association v. EPA*, 647 F.2d at 1161-62.

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

Section 109(d)(1) requires that "not later than December 31, 1980, and at 5-year intervals thereafter, the Administrator shall complete a thorough review of the criteria published under section 108 and the national ambient air quality standards . . . and shall make such revisions in such criteria and standards and promulgate such new standards as may be appropriate . . . ." Section 109(d)(2) requires that an independent scientific review committee "shall complete a review of the criteria . . . and the national primary and secondary ambient air quality standards . . . and shall recommend to the Administrator any new . . . standards and revisions of existing criteria and standards as may be appropriate . . . ." Since the early 1980s, this independent review function has been performed by the Clean Air Scientific Advisory Committee (CASAC).[8]

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[7] As used here and similarly throughout this document, the term population (or group) refers to persons having a quality or characteristic in common, such as a specific pre-existing illness or a specific age or life stage. As discussed more fully in section 3.3 below, the identification of sensitive groups (called at-risk groups or at-risk populations) involves consideration of susceptibility and vulnerability.

[8] 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.

1.2.2 History of Lead NAAQS Reviews

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

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

The first review of the Pb standards was initiated in the mid-1980s. The scientific assessment for that review is described in the 1986 Air Quality Criteria for Lead (USEPA, 1986a), the associated Addendum (USEPA, 1986b) and the 1990 Supplement (USEPA, 1990a). As part of the review, the Agency designed and performed human exposure and health risk analyses (USEPA, 1989), the results of which were presented in a 1990 Staff Paper (USEPA, 1990b). Based on the scientific assessment and the human exposure and health risk analyses, the 1990 Staff Paper presented recommendations for consideration by the Administrator (USEPA, 1990b). After consideration of the documents developed during the review and the significantly changed circumstances since Pb was listed in 1976, the Agency did not propose any revisions to the 1978 Pb NAAQS. In a parallel effort, the Agency developed the broad, multi-program, multimedia, integrated *U.S. Strategy for Reducing Lead Exposure* (USEPA, 1991). As part of implementing this strategy, the Agency focused efforts primarily on regulatory and remedial clean-up actions aimed at reducing Pb exposures from a variety of nonair sources judged to pose more extensive public health risks to U.S. populations, as well as on actions to reduce Pb emissions to air, such as bringing more areas into compliance with the existing Pb NAAQS

(USEPA, 1991). EPA continues this broad, multi-program, multimedia approach to reducing Pb exposures today, as described in section 1.2.4 below.

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

The scientific assessment for the review is described in the 2006 *Air Quality Criteria for Lead* (USEPA, 2006b; henceforth referred to as the 2006 CD), multiple drafts of which received review by CASAC and the public. The EPA also conducted human exposure and health risk assessments and a pilot ecological risk assessment for the review, after consultation with CASAC and receiving public comment on a draft analysis plan (USEPA, 2006c). Drafts of these quantitative assessments were reviewed by CASAC and the public. The pilot ecological risk assessment was released in December 2006 (ICF, 2006) and the final health risk assessment report was released in November 2007 (USEPA, 2007a). The policy assessment, based on both of these assessments, air quality analyses and key evidence from the 2006 CD, was presented in the Staff Paper (USEPA, 2007b), a draft of which also received CASAC and public review. The final Staff Paper presented OAQPS staff's evaluation of the public health and welfare policy implications of the key studies and scientific information contained in the 2006 CD and presented and interpreted results from the quantitative risk/exposure analyses conducted for this review. Based on this evaluation, the Staff Paper presented OAQPS staff recommendations that the Administrator give consideration to substantially revising the primary and secondary standards to a range of levels at or below 0.2 $\mu g/m^3$.

Immediately subsequent to completion of the Staff Paper, the EPA issued an advance notice of proposed rulemaking (ANPR) that was signed by the Administrator on December 5, 2007 (72 FR 71488).[10] CASAC provided advice and recommendations to the Administrator with regard to the Pb NAAQS based on its review of the ANPR and the previously released final Staff Paper and risk assessment reports. The proposed decision on revisions to the Pb NAAQS was signed on May 1, 2008 and published in the *Federal Register* on May 20, 2008 (73 FR 29184). Members of the public provided both written and, at two public hearings, oral comments and the

[9] In the current review, these two documents have been combined in the IRP.

[10] 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 a policy assessment prepared by OAQPS staff (previously termed the OAQPS Staff Paper).

CASAC Pb Panel also provided advice and recommendations to the Administrator based on its review of the proposal notice. The final decision on revisions to the Pb NAAQS was signed on October 15, 2008 and published in the *Federal Register* on November 12, 2008 (73 FR 66964).

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

1.2.3 Current Lead NAAQS Review

On February 26, 2010, the EPA formally initiated its current review of the air quality criteria and standards for Pb, requesting the submission of recent scientific information on specified topics (75 FR 8934). Soon after this, the EPA held a science policy workshop to discuss the policy-relevant scientific information, which informed identification of key policy issues and questions to frame the review of the Pb NAAQS (75 FR 20843). Drawing from the workshop discussions, the EPA developed the draft IRP (USEPA, 2011b). The draft IRP was made available in late March 2011 for consultation with the CASAC Pb Review Panel and for public comment (76 FR 20347). This document was discussed by the Panel via a publicly accessible teleconference consultation on May 5, 2011 (76 FR 21346; Frey, 2011a). The final IRP, developed in consideration of the CASAC consultation and public comment, was released in November, 2011 (USEPA, 2011a; 76 FR 76972).

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[11] The current NAAQS for Pb are specified at 40 CFR 50.16.

[12] 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).

In developing the ISA for this review, the EPA held a workshop in December 2010 to discuss with invited scientific experts preliminary draft materials and released the first external review draft of the document for CASAC review and public comment on May 6, 2011 (USEPA, 2011c; 76 FR 26284). The CASAC Pb Review Panel met at a public meeting on July 20, 2011 to review the draft ISA (76 FR 36120). The CASAC provided comments in a December 9, 2011 letter to the EPA Administrator (Frey and Samet, 2011). The second external review draft ISA was released for CASAC review and public comment in February 2012 (USEPA, 2012b; 77 FR 5247) and was the subject of a public meeting on April 10-11, 2012 (77 FR 14783). The CASAC provided comments in a July 20, 2012 letter (Samet and Frey, 2012). The third external review draft was released for CASAC review and public comment in November 2012 (USEPA, 2012a; 77 FR 70776) and was the subject of a public meeting on February 5-6, 2013 (78 FR 938). The CASAC provided comments in a June 4, 2013 letter (Frey, 2013a). The final ISA was released in late June 2013 (USEPA, 2013a; 78 FR 38318).

In June 2011, the EPA developed and released the REA Planning Document for consultation with CASAC and public comment (USEPA, 2011d; 76 FR 58509). This document presented a critical evaluation of the information related to Pb human and ecological exposure and risk (e.g., data, modeling approaches) newly available in this review, with a focus on consideration of the extent to which new or substantially revised REAs for health and ecological risk are warranted by the newly available evidence. Evaluation of the newly available information with regard to designing and implementing health and ecological REAs for this review led us to conclude that the currently available information did not provide a basis for developing new quantitative risk and exposure assessments that would have substantially improved utility for informing the Agency's consideration of health and welfare effects and evaluation of the adequacy of the current primary and secondary standards, respectively (REA Planning Document, sections 2.3 and 3.3, respectively). The CASAC Pb Panel provided consultative advice on that document and its conclusions at a public meeting on July 21, 2011 (76 FR 36120; Frey, 2011b). Based on their consideration of the REA Planning Document analysis, the CASAC Pb Review Panel generally concurred with the conclusion that a new REA was not warranted in this review (Frey, 2011b; Frey, 2013b). In consideration of the conclusions reached in the REA Planning Document and CASAC's consultative advice, the EPA has not developed REAs for health and ecological risk for this review. Accordingly, this Policy Assessment considers the risk assessment findings from the last review with regard to any appropriate further interpretation in light of the evidence newly available in this review.

A draft of this Policy Assessment was released for public comment and review by CASAC in January 2013 (USEPA, 2013b; 77 FR 70776) and was the subject of a public meeting on February 5-6, 2013 (78 FR 938). Comments provided by the CASAC in a June 4, 2013 letter

(Frey, 2013b), as well as public comments received on the draft PA were considered in preparing the final PA.

1.2.4 Related Lead Control Programs

States are primarily responsible for ensuring attainment and maintenance of the NAAQS. Under section 110 of the Act (42 U.S.C. 7410) and related provisions, states are to submit, for EPA approval, state implementation plans (SIPs) that provide for the attainment and maintenance of such standards through control programs directed to sources of the pollutants involved. The states, in conjunction with EPA, also administer the prevention of significant deterioration program (42 U.S.C. 7470–7479) for these pollutants. In addition, federal programs provide for nationwide reductions in emissions of these and other air pollutants through the Federal Motor Vehicle Control Program under Title II of the Act (42 U.S.C. 7521–7574), which involves controls for automobile, truck, bus, motorcycle, nonroad engine, and aircraft emissions; the new source performance standards under section 111 of the Act (42 U.S.C. 7411); and the national emission standards for hazardous air pollutants under section 112 of the Act (42 U.S.C. 7412).

As noted in section 1.2.2 above, the NAAQS is only one component of EPA's programs to address Pb in the environment. Some recent actions expected to result in air Pb emissions reductions that are associated with other EPA programs, such as those recognized above, are more specifically recognized in section 2.1. The presentation below briefly summarizes additional ongoing activities that, although not directly pertinent to the review of the NAAQS, are associated with controlling environmental Pb levels and human Pb exposures more broadly. Among those identified are EPA programs intended to encourage exposure reduction programs in other countries.

Reducing Pb exposures has been recognized as a federal priority as environmental and public health agencies continue to grapple with soil and dust Pb levels from the historical use of Pb in paint and gasoline and from other sources.[13] A broad range of federal programs beyond those that focus on air pollution control provide for nationwide reductions in environmental releases and human exposures. For example, pursuant to Section 1412 of the Safe Drinking Water Act (SDWA), EPA regulates lead in public drinking water systems through corrosion

[13] In 1991, the Secretary of the Health and Human Services (HHS) characterized Pb poisoning as the "number one environmental threat to the health of children in the United States" (Alliance to End Childhood Lead Poisoning. 1991). In 1997, President Clinton created, by Executive Order 13045, the President's Task Force on Environmental Health Risks and Safety Risks to Children in response to increased awareness that children face disproportionate risks from environmental health and safety hazards (62 FR 19885). By Executive Orders issued in October 2001 and April 2003, President Bush extended the work for the Task Force beyond its original charter (66 FR 52013 and 68 FR 19931). Reducing Pb poisoning in children was identified as the Task Force's top priority.

control and other utility actions which work together to minimize lead levels at the tap. (40 CFR 141.80-141.91). In addition, under Section 1417 of the Safe Drinking Water Act, pipes, fittings and fixtures for potable water applications may not be used or introduced into commerce unless they are considered "lead free" as defined by that Act (40 CFR 141.43).[14] Additionally, federal Pb abatement programs provide for the reduction in human exposures and environmental releases from in-place materials containing Pb (e.g., Pb-based paint, urban soil and dust, and contaminated waste sites). Federal regulations on disposal of Pb-based paint waste help facilitate the removal of Pb-based paint from residences (68 FR 36487).

Federal programs to reduce exposure to Pb in paint, dust, and soil are specified under the comprehensive federal regulatory framework developed under the Residential Lead-Based Paint Hazard Reduction Act (Title X). Under Title X (codified as Title IV of the Toxic Substances Control Act [TSCA]), EPA has established regulations and associated programs in the following six categories: (1) training, certification and work practice requirements for persons engaged in lead-based paint activities (abatement, inspection and risk assessment); accreditation of training providers; and authorization of state and tribal lead-based paint programs; (2) training, certification, and work practice requirements for persons engaged in home renovation, repair and painting (RRP) activities; accreditation of RRP training providers; and authorization of state and tribal RRP programs; (3) ensuring that, for most housing constructed before 1978, information about lead-based paint and lead-based paint hazards flows from sellers to purchasers, from landlords to tenants, and from renovators to owners and occupants; (4) establishing standards for identifying dangerous levels of Pb in paint, dust and soil; (5) providing grant funding to establish and maintain state and tribal lead-based paint programs; and, (6) providing information on Pb hazards to the public, including steps that people can take to protect themselves and their families from lead-based paint hazards.

Under Title IV of TSCA, EPA established standards identifying hazardous levels of Pb in residential paint, dust, and soil in 2001. This regulation supports the implementation of other regulations which deal with worker training and certification, Pb hazard disclosure in real estate transactions, Pb hazard evaluation and control in federally-owned housing prior to sale and housing receiving federal assistance, and U.S. Department of Housing and Urban Development grants to local jurisdictions to perform Pb hazard control. The TSCA Title IV term "lead-based paint hazard" implemented through this regulation identifies lead-based paint and all residential lead-containing dust and soil regardless of the source of Pb, which, due to their condition and

[14] In 2011, revisions to this section of the Safe Drinking Water Act lowered the amount of Pb permitted in pipes, fittings, and fixtures; the changes became effective in January 2014. More information is provided in "Summary of the Reduction of Lead in Drinking Water Act and Frequently Asked Questions" at http://water.epa.gov/drink/info/lead/index.cfm)

location, would result in adverse human health effects. One of the underlying principles of Title X is to move the focus of public and private decision makers away from the mere presence of lead-based paint to the presence of lead-based paint hazards, for which more substantive action should be undertaken to control exposures, especially to young children. In addition, the success of the program relies on the voluntary participation of states and tribes as well as counties and cities to implement the programs and on property owners to follow the standards and EPA's requirements.

On March 31, 2008, the EPA issued a new rule (Lead: Renovation, Repair and Painting [RRP] Program, 73 FR 21692) to protect children from lead-based paint hazards. This rule applies to compensated renovators and maintenance professionals who perform renovation, repair, or painting in housing and child-care facilities built prior to 1978. Among its requirements is one that requires that firms that conduct RRP activities be certified; that their employees be trained; and that they follow protective work practice standards. These standards prohibit certain dangerous work practices, such as open flame burning or torching of lead-based paint. The required work practices also include posting warning signs, restricting occupants from work areas, containing work areas to prevent dust and debris from spreading, conducting a thorough cleanup, and verifying that cleanup was effective. The rule became fully effective in April 2010. The rule also specifies procedures for the authorization of states, territories, and tribes to administer and enforce these standards and regulations in lieu of a federal program. In announcing this rule, EPA noted that approximately 37 million homes in the United States contain some lead-based paint, and that this rule's requirements are key components of a comprehensive effort to eliminate childhood Pb poisoning. To foster adoption of the rule's measures, EPA has been conducting an extensive education and outreach campaign to promote awareness of these new requirements among both the regulated entities and the consumers who hire them. In addition, the EPA is investigating whether lead hazards are also created by RRP activities in public and commercial buildings, and if appropriate, to issue RRP requirements for this class of buildings.

Programs associated with the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) and Resource Conservation Recovery Act (RCRA) also implement abatement programs, reducing exposures to Pb and other pollutants. For example, EPA determines and implements protective levels for Pb in soil at Superfund sites and RCRA corrective action facilities. Federal programs, including those implementing RCRA, provide for management of hazardous substances in hazardous and municipal solid waste (see, e.g., 66 FR 58258). Federal regulations concerning batteries in municipal solid waste facilitate

the collection and recycling or proper disposal of batteries containing Pb.[15] Similarly, federal programs provide for the reduction in environmental releases of hazardous substances such as Pb in the management of wastewater (http://www.epa.gov/owm/).

A variety of federal nonregulatory programs also provide for reduced environmental release of Pb-containing materials by encouraging pollution prevention, promotion of reuse and recycling, reduction of priority and toxic chemicals in products and waste, and conservation of energy and materials. These include the Resource Conservation Challenge (http://www.epa.gov/epaoswer/osw/conserve/index.htm), the National Waste Minimization Program (http://www.epa.gov/epaoswer/hazwaste/minimize/leadtire.htm), "Plug in to eCycling" (a partnership between EPA and consumer electronics manufacturers and retailers; http://www.epa.gov/epaoswer/hazwaste/recycle/electron/crt.htm#crts), and activities to reduce the practice of backyard trash burning (http://www.epa.gov/msw/backyard/pubs.htm).

In addition to the Pb control programs summarized above, EPA's research program identifies, encourages and conducts research needed to locate and assess serious risks and to develop methods and tools to characterize and help reduce risks. For example, EPA's Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK model) is widely used and accepted as a tool that informs the evaluation of site-specific data. More recently, in recognition of the need for a single model that predicts Pb concentrations in tissues for children and adults, EPA has been developing the All Ages Lead Model (AALM) to provide researchers and risk assessors with a pharmacokinetic model capable of estimating blood, tissue, and bone concentrations of Pb based on estimates of exposure over the lifetime of the individual (ISA, section 3.6). EPA research activities on substances including Pb, such as those identified here, focus on improving our characterization of health and environmental effects, exposure, and control or management of environmental releases (see http://www.epa.gov/research/).

Other federal agencies also participate in programs intended to reduce Pb exposures. For examples programs of the Centers for Disease Control and Prevention (CDC) provide for the tracking of children's blood Pb levels in the U.S. and provide guidance on levels at which medical and environmental case management activities should be implemented (CDC, 2012; ACCLPP, 2012).[16] As a result of coordinated, intensive efforts at the national, state and local levels, including those programs described above, blood Pb levels in all segments of the population have continued to decline from levels observed in the past. For example, blood Pb

[15] See, e.g., "Implementation of the Mercury-Containing and Rechargeable Battery Management Act" http://www.epa.gov/epaoswer/hazwaste/recycle/battery.pdf and "Municipal Solid Waste Generation, Recycling, and Disposal in the United States: Facts and Figures for 2005" http://www.epa.gov/epaoswer/osw/conserve/resources/msw-2005.pdf.

[16] The CDC guidance on blood Pb levels is described further in section 3.1 below.

levels for the general population of children 1 to 5 years of age have dropped to a geometric mean level of 1.17 μg/dL in the 2009-2010 National Health and Nutrition Examination Survey (NHANES) as compared to the geometric mean in 1999-2000 of 2.23 μg/dL and in 1988-1991 of 3.6 μg/dL (ISA, section 3.4.1; CD, AX4-2). Similarly, blood Pb levels in non-hispanic black, Mexican American and lower socioeconomic groups, which are generally higher than those for the general population, have also declined (ISA, section 3.4.1; Jones et al, 2009).

The EPA also participates in a broad range of international programs focused on reducing environmental releases and human exposures in other countries. For example, the *Partnership for Clean Fuels and Vehicles* program engages governments and stakeholders in developing countries to eliminate Pb in gasoline globally.[17] From 2007 to 2011, the number of countries known to still be using leaded gasoline was reduced from just over 20 to six, with three of the six also offering unleaded fuel. All six were expected to eliminate Pb from fuel in the subsequent few years (USEPA, 2011e). The U.S. EPA is a contributor to the *Global Alliance to Eliminate Lead Paint*, which is a cooperative initiative jointly led by the World Health Organization and the United Nations Environment Programme (UNEP) to focus and catalyze the efforts to achieve international goals to prevent children's Pb exposure from paints containing Pb and to minimize occupational exposures to Pb paint. This alliance has the broad objective of promoting a phase-out of the manufacture and sale of paints containing Pb and eventually to eliminate the risks that such paints pose. The UNEP is also engaged on the problem of managing wastes containing Pb, including lead-containing batteries. The Governing Council of the UNEP, of which the U.S. is a member, has adopted decisions focused on promoting the environmentally sound management of products, wastes and contaminated sites containing Pb and reducing risks to human health and the environment from Pb and cadmium throughout the life cycles of those substances (UNEP Governing Council, 2011, 2013). EPA is also engaged in the issue of environmental impacts of spent lead-acid batteries internationally through the Commission for Environmental Cooperation (CEC), where the EPA Administrator along with the cabinet-level or equivalent representatives of Mexico and Canada comprise the CEC's senior governing body (CEC Council).[18]

[17] International programs in which the U.S. participates, including those identified here, are described at several web sites: http://epa.gov/international/air/pcfv.html, http://www.unep.org/transport/pcfv/, http://www.unep.org/hazardoussubstances/Home/tabid/197/hazardoussubstances/LeadCadmium/PrioritiesforAction/GAELP/tabid/6176/Default.aspx

[18] The CEC was established to support cooperation among the North American Free Trade Agreement partners to address environmental issues of continental concern, including the environmental challenges and opportunities presented by continent-wide free trade.

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

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

1.3.1 Environmental Distribution and Exposure Pathways

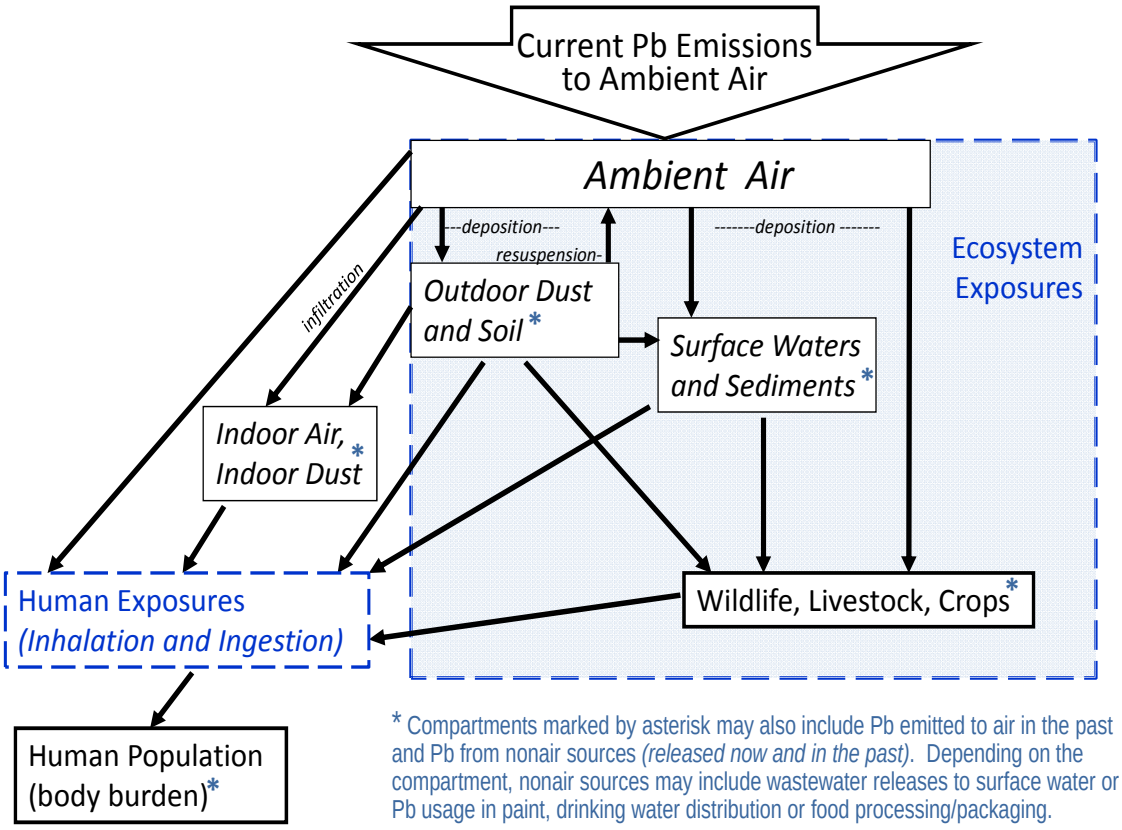
Lead emitted to ambient air is transported through the air and is also distributed to other media through the process of deposition, which may occur in dry conditions or in association with precipitation, as summarized further in section 2.3 below (ISA, section 2.7.2). Once deposited, the fate of Pb is influenced by the type of surface onto which the particles deposit and by the type and activity level of transport processes in that location. Precipitation and other natural, as well as human-influenced, processes contribute to the fate of such particles, which affects the likelihood of subsequent human and ecological exposures, e.g., tracking into nearby houses or transport with surface runoff into nearby water bodies (ISA, sections 2.3 and 3.1). For

[19] Some aspects of the review of the secondary standard for oxides of nitrogen and sulfur (completed in 2012), 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.

example, Pb particles deposited onto impervious surfaces, such as roadway, sidewalk or other urban surfaces, may be more available for human contact while they remain on such surfaces or are transferred to other human environments, such as on clothing or through resuspension and infiltration (ISA, section 3.1.1.1). Deposited Pb can also be transported (by direct deposition or stormwater runoff) to water bodies and into associated sediments, which may provide a storage function for Pb in aquatic ecosystems (ISA, sections 2.3.2 and 6.2.1). Lead deposited in terrestrial ecosystems can also be incorporated into vegetation and soil matrices (ISA, sections 2.3.3 and 6.2.1).

Figure 1-1 illustrates, in summary fashion, the pathways by which Pb emitted into ambient air can be distributed in the environment, contributing to human and ecosystem exposures. As shown in this figure, the multimedia distribution of Pb emitted into ambient air (air-related Pb) contributes to multiple air-related pathways of human and ecosystem exposure (ISA, sections 3.1.1 and 3.7.1).[20] Figure 1-1 additionally illustrates that air-related pathways may involve media other than air, including indoor and outdoor dust, soil, surface water and sediments, vegetation and biota. As recognized by the figure and discussed more completely in the subsections below, Pb occurring in indoor and outdoor environments that has not passed through ambient air (nonair Pb) may complicate our consideration of ambient air related Pb exposures. Further, the persistence of Pb and the associated environmental legacy of historical releases pose an additional complication, also discussed below, with regard to consideration of exposures associated with current Pb emissions.

[20] 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 associated health 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 3.1.1.1). 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. The various inhalation and ingestion air-related pathways may vary with regard to the time in which they respond to changes in air Pb concentrations. For example, human exposure pathways most directly involving Pb in ambient air and exchanges of ambient air with indoor air (e.g., inhalation) can respond most quickly, while pathways involving

exposure to Pb deposited from ambient air into the environment (e.g., diet) may be expected to respond more slowly. The extent of this will be influenced by the magnitude of change, as well as – for deposition-related pathways - the extent of prior deposition and environment characteristics influencing availability of prior deposited Pb (section 1.3.1.2 below).

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 3.7.1). For example, Pb in dust inside some houses or outdoors in some urban areas may derive from the common past usage of leaded paint, while Pb in drinking water may derive from the use of leaded pipe or solder in drinking water distribution systems (ISA, section 3.1.3.3). We also recognize the history of much greater air emissions of Pb in the past, such as that associated with leaded gasoline usage and higher industrial emissions (as summarized in section 2.1.1 below) which have left a legacy of Pb in other (nonair) media.

The relative importance of different pathways of human exposure to Pb, as well as the relative contributions from Pb resulting from recent and historic air emissions and from nonair sources, vary across the U.S. population as a result of both extrinsic factors, such as a home's proximity to industrial Pb sources or its history of leaded paint usage, and intrinsic factors, such as a person's age and nutritional status (ISA, sections 5.1, 5.2, 5.2.1, 5.2.5 and 5.2.6). Thus, the relative contributions from specific pathways is situation specific (ISA, p. 1-11), although a predominant Pb exposure pathway for very young children is the incidental ingestion of indoor dust by hand-to-mouth activity (ISA, section 3.1.1.1). For adults, however, diet may be the primary Pb exposure pathway (2006 CD, section 3.4). Similarly, the relative importance of air-related and nonair-related Pb also varies with the relative magnitudes of exposure by those pathways, which may vary with different circumstances. For example, relative contributions to a child's total Pb exposure from air-related exposure pathways compared to other (nonair) Pb exposures depend on many factors, including ambient air concentrations and air deposition in the area where the child resides (as well as in the area from which the child's food derives), as well as access to other sources of Pb exposure such as Pb paint, tap water affected by plumbing containing Pb, and lead-tainted products. Studies indicate that in the absence of paint-related exposures, Pb from other sources such as nearby stationary sources of Pb emissions may dominate a child's Pb exposures (ISA, sections 3.1 and 3.1.3.2; 2006 CD, section 3.2.3). In other cases, such as children living in older housing with peeling paint or where renovations have occurred, the dominant source of Pb exposure may be dust from leaded paint used in the house in the past. Depending on Pb levels in a home's tap water, drinking water can sometimes be a significant source. Lead exposure may also be the result of a mixture of contributions from multiple sources, with no one source dominating. Our understanding of the relative contribution of air-related Pb to ingestion exposure pathways is limited by the paucity of studies that parse

ingestion exposure pathways with regard to air-related and nonair Pb. Our understanding of the relative contribution of air-related Pb associated with historical emissions and that from recent emissions is similarly limited, as discussed further in section 1.3.2 below.

1.3.1.2 Ecosystem Exposure Pathways

The distribution of Pb from ambient air to other environmental media also influences the exposure pathways in terrestrial and aquatic ecosystems. Exposure of terrestrial animals and vegetation to air-related Pb can occur by contact with ambient air or by contact with soil, water or food items that have been contaminated by Pb from ambient air (ISA, section 6.2). Transport of Pb into aquatic systems similarly provides for exposure of biota in those systems, and exposures may vary among systems as a result of differences in sources and levels of contamination, as well as characteristics of the systems themselves. In addition to Pb contributed by current atmospheric deposition, Pb may occur in aquatic systems as a result of nonair sources such as industrial discharges or mine-related drainage, of historical air Pb emissions (e.g., contributing to deposition to a water body or via runoff from soils near historical air sources) or combinations of different types of sources (e.g., resuspension of sediments contaminated by urban runoff and surface water discharges).

The persistence of Pb contributes an important temporal aspect to lead's environmental pathways, and the time (or lag) associated with realization of the impact of air Pb concentrations on concentrations in other media can vary with the media (e.g., ISA, section 6.2.2). For example, exposure pathways most directly involving Pb in ambient air or surface waters can respond more quickly to changes in ambient air Pb concentrations while pathways involving exposure to Pb in soil or sediments generally respond more slowly. An additional influence on the response time for nonair media is the environmental presence of Pb associated with past, generally higher, air concentrations. For example, after a reduction in air Pb concentrations, the time needed for sediment or surface soil concentrations to indicate a response to reduced air Pb concentrations might be expected to be longer in areas of more substantial past contamination than in areas with lesser past contamination. Thus, considering the Pb concentrations occurring in nonair media as a result of air quality conditions that meet the current NAAQS is a complexity of this review, as it also was, although to a lesser degree, with regard to the prior standard in the last review.

1.3.2 Considerations Related to Historically Emitted Lead

In reviews of NAAQS, the overarching consideration of each review is first focused on the general question as to whether the currently available information supports or calls into question the adequacy of the current standard(s). In addressing that consideration for the NAAQS for Pb, our focus is on Pb emitted to ambient air under conditions meeting the current standard and its potential to cause health or welfare effects as a result of exposures to Pb in air or

in other media. Our framing of the focus in this way differs from that for NAAQS reviews involving other pollutants. We frame the focus in this way in consideration of the multiple exposure pathways for Pb currently being emitted and in consideration of the persistence of Pb in the environment over time, a characteristic which does not affect reviews of other NAAQS. In considering the case for Pb, however, we recognize that, because of its persistence, both recent emissions to ambient air and the substantial emissions of the past contribute to current Pb exposures (via multiple exposure pathways, as summarized in section 1.4.1 above). And we recognize that past Pb emissions contributed to many situations where air concentrations were well in excess of the current Pb standard, and may continue to contribute to exposures even where air concentrations are below the current standard. Yet our task in this and every NAAQS review is focused on assessing the adequacy of the current standard.

The substantial historical emissions to air and releases to nonair media contributed to human and environmental exposures in the past (e.g., 1978 CD, 1986 CD, 2006 CD, ISA). Because of the persistence of Pb, historical exposures associated with both air-related and nonair-related sources have contributed a legacy of Pb that is now stored within older humans and ecosystems. For example, concentrations of Pb in the bone and blood of older members of the U.S. population, who lived during the time of widespread air emissions associated with leaded gasoline usage (as well as higher industrial emissions) under the previous Pb NAAQS or prior to establishment of any Pb NAAQS, are greater than what would result from air quality conditions allowed by the current, more restrictive NAAQS. Epidemiological studies of these populations, in which this exposure history is represented by current bone or blood Pb concentrations, contribute to the overall evidence base regarding lead-related health effects (as discussed in chapter 3 below). Such studies of these historically exposed populations, however, are generally less informative in judging the adequacy of the current primary standard (as discussed in chapter 3 below). This is in contrast to epidemiological studies of very young populations with much shorter and more recent exposure histories (also discussed in chapter 3).

Substantial and widespread atmospheric emissions of Pb in the U.S. (as well as releases to other media) extend back through the 19th century (Yohn et al, 2004; Jackson et al., 2004; Graney et al., 1995). These historical emissions contributed to the distribution of Pb within and well beyond the U.S. (ISA, section 2.5.5; Reuer and Weiss, 2002; McConnell and Edwards, 2008). Although it has not been completely and totally characterized, multiple aspects of the legacy of this widespread distribution have been documented and described. The current concentrations of Pb in U.S. ecosystems thus reflect the influence of greater air emissions that occurred in the past, under the prior Pb NAAQS and prior to establishment of any Pb NAAQS.[21]

[21] The current distribution of Pb in U.S. ecosystems also reflects historical nonair releases.

As described further in section 2.3 below, media in ecosystems across the U.S. are still recovering from the past period of greater atmospheric emissions and deposition that is documented in media such as soil, aquatic sediments and peat bogs (2006 CD, section 2.3.1; ISA, section 2.6.2). Core samples of these media show a pattern of Pb concentrations in deposited material that peaks around the 1970s followed by marked decline in more recent years (2006 CD, section 2.3.1; ISA section 2.6.2). Individual ecosystem responses differ with regard to their rates of change in media concentrations due to the extent of contamination in individual systems as well as the influence of ecosystem-specific characteristics (further described in section 2.3 below). Thus, the time required for these ecosystem media to come to a condition that is relatively stable with time (i.e., "steady-state") will also vary, and the resulting range of "steady-state" media concentrations in U.S. ecosystems under ambient air concentrations consistent with the current NAAQS are unknown. Future research may better inform our understanding in this area.

In considering the array of ecological effects evidence in this review, we recognize that evidence of effects pertaining to the concentrations associated with the past, higher emissions (as well as from nonair sources), while generally informative to our understanding of welfare effects associated with environmental Pb, does not directly inform our consideration of welfare effects that might be anticipated under the current secondary standard and thus may be generally less informative in judging the adequacy of the current standard. The availability of information on whether adverse effects could be anticipated from the Pb in terrestrial and aquatic ecosystems that results from air quality conditions allowed by the current, more restrictive NAAQS is discussed in chapter 5, with consideration of the current NAAQS discussed in chapter 6.

1.4 GENERAL ORGANIZATION OF THE DOCUMENT

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

Chapters 3 and 4 comprise the second main part of this document, dealing with human health and the primary standard. These chapters are organized around a series of questions,

building on those identified in the IRP, that address the key policy-relevant issues related to the primary standard. Chapter 3 presents an overview of key policy-relevant health effects evidence and major health-related conclusions from the ISA; an examination of issues related to the quantitative assessment of health risks; key results from quantitative assessments together with a discussion of uncertainty and variability in the results; and discussion of the public health implications of the evidence and exposure/risk information. Chapter 4 includes staff's consideration of the scientific evidence and exposure/risk information related to the primary standard and associated conclusions related to the adequacy of the current primary standard.

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

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2 AMBIENT AIR LEAD

The focus for this Pb NAAQS review is on Pb derived from 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 nonair media is enhanced by the persistent nature of Pb. Consequently this chapter discusses our current understanding of both 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 2.3).[1] Once emitted, particle-bound Pb can be transported long or short distances depending on particle 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 PM_{10}) confirms the transport of airborne Pb in smaller particles 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/m^3 to western U.S. Pb concentrations (ISA, sections 2.3.1 and 2.5.5; Murphy et al., 2007). In general, 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 1.2.1). 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 on sources and emissions of Pb to ambient air (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

[1] While in some circumstances Pb can be emitted in gaseous form, the Pb compounds that may be produced initially in vapor phase can be expected to condense into particles upon cooling to ambient temperature and/or upon oxidizing with mixing into the atmosphere (ISA, section 2.2.2.1; Gidney et al., 2010).

on a national or local scale, and generally describe their emissions and distribution within the U.S.

The primary data source for this discussion is the National Emissions Inventory (NEI) for 2008[2]. The NEI is a comprehensive and detailed estimate of air emissions of both criteria and hazardous air pollutants from air emissions sources. The NEI is generally prepared at three-year intervals, such that the next NEI, for 2011, is currently under development. The NEI is prepared by the EPA based primarily upon emission estimates and emission model inputs provided by state, local, and tribal air agencies for sources in their jurisdictions, supplemented by data developed by the EPA. Some of these estimates are required by regulation, while some are voluntarily reported. For example, states are required to report Pb emissions from facilities emitting more than 5 tons of Pb per year (tpy) and from facilities emitting greater than threshold amounts for other criteria pollutants (e.g., 100 tpy of particulate matter or volatile organic compounds; CFR 51, subpart A). Estimates of Pb emissions presented in this document (and in the ISA) are drawn from the 2008 NEI version 3.[3] As a result of various Clean Air Act requirements, emissions standards implemented since 2008 for a number of industrial source categories represented in the NEI are projected to result in considerably lower emissions at the current time or in the near future. (Appendix 2A)

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

2.1.1 Temporal Trends on a National Scale

Figure 2-1 shows the substantial downward trend in Pb emissions that has occurred over the past several decades. The most dramatic reductions in Pb emissions occurred prior to 1990 in the transportation sector due to the removal of Pb from gasoline used in on-road vehicles. Lead emissions were further reduced substantially between 1990 and 2008, with significant

[2] http://www.epa.gov/ttn/chief/net/2008inventory.html

[3] 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.

reductions occurring in the metals industries at least in part as a result of national emissions standards for hazardous air pollutants.

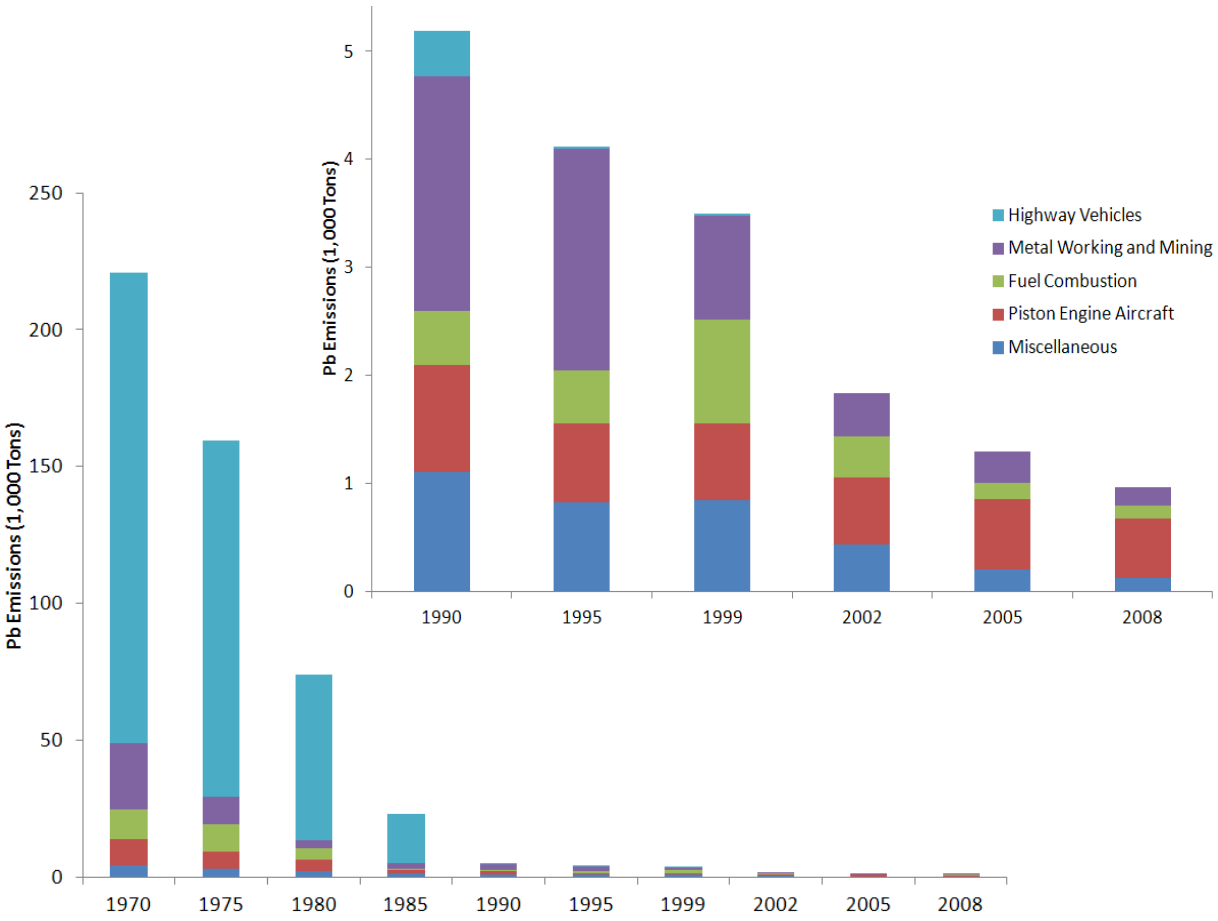

Figure 2-1. Temporal trend in U.S. air emissions of Pb: 1970-2008.

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). The latter individually are generally small sources, which comprise a large category 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.

Table 2-1. U.S. Pb 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	63
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	8
Secondary Non-ferrous Metals (other)	7
Primary Non-ferrous Metals (other)	7
Carbon Manufacturing	6
Pulp and Paper Production	6
Secondary Copper Smelting	5
Commercial Marine Vessels	5
Fabricated Metal Products Manufacturing	5
Residential Heating	5
Municipal Waste Incineration	4
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 Pb 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 ceased smelter operations at this site at the end of 2013 (USEPA, 2012).

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.

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 Pb 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 Pb emissions reduction of 13.6 tpy across the category (a 68% reduction). Also, the NESHAPs for Primary and Secondary Lead Smelting were revised in 2011 and 2012, respectively (76 FR 70834, 77 FR 555), as well as 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 2A).

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.[4] 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.[5] contributing an estimated 550 tons of Pb to the air that comprise 58% of the national Pb inventory.[6] Leaded avgas is used at approximately 20,000 airport facilities in the United States.

The EPA is currently collecting and evaluating information regarding emissions and air concentrations of Pb resulting from avgas combustion by piston-engine aircraft (including monitoring data described in section 2.2.1.1 below). 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

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

[5] 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.

[6] 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

Administration would be required to promulgate regulations addressing the fuel used by those aircraft. More information about EPA's actions is available at www.epa.gov/otaq/aviation.htm.

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

Due to the presence of Pb as a trace contaminant in gasoline, diesel fuel and lubricating oil, cars, trucks, and engines operating in nonroad equipment, marine engines and jet aircraft emit small amounts of Pb (ISA, Section 2.2.2.6). Additional mobile sources of Pb include brake wear, tire wear, and loss of Pb wheel weights (ISA, Section 2.2.2.6).

2.1.2.3 Natural Sources and Long-range Transport

Some amount of Pb in the air in the U.S. derives from natural sources, such as volcanoes, sea salt, and windborne soil particles from areas free of anthropogenic activity and some may also derive from anthropogenic sources of airborne Pb located outside of the U.S. (ISA, section 2.5.5). Emissions estimates for these sources, as well as forest wildfires and biogenic sources, have not been developed for the NEI. Quantitative estimates for these processes remain an area of significant uncertainty. Based on several different approaches, the ISA identifies several estimates of the concentration of airborne Pb derived from natural sources. The estimates extend no higher than 1 nanogram per cubic meter (ng/m^3), and extend down as low as 0.02 ng/m^3 (ISA, section 2.5.5). The data available to derive such an estimate are limited and such a value might be expected to vary geographically with the natural distribution of Pb.

Another contribution to U.S. airborne Pb concentrations is long-range transport such as that associated with air masses carrying Pb from sources in Asia, where controls on Pb emissions have lagged those in the U.S. and Canada (ISA, section 2.5.5; Osterberg et al., 2008). The most recent estimates of contributions from Asia, however, conclude that the Asian contribution to U.S. airborne Pb concentrations is generally less than 1 ng/m^3 (ISA, section 2.5.5; Murphy, 2007; Ewing et al., 2010).

2.1.2.4 Previously Deposited Lead

Lead-bearing particles that occur in outdoor dust on soil or built surfaces (e.g., streets and sidewalks) can be a source of airborne Pb as a result of particle resuspension (ISA, section 2.3.1.3). Proximity to major sources of Pb emissions, the extent of previous or historic deposition, and the effectiveness of natural and human removal processes dictate how important resuspension may be as a contribution to air Pb concentrations. In addition to resuspension and

subsequent dispersion, surface water runoff (e.g., associated with rainfall) also plays a role in the movement of Pb-bearing particles from outdoor surfaces to human-made or natural stormwater sediment catchments (ISA, sections 1.2.1, 2.3.1.3, 2.3.2 and 2.3.2.4; Wong et al., 2006). Mean residence time of street dust has been estimated for a low-traffic street (approximately 30 vehicles/hour) to be several months but less than a year (ISA, section 2.3.1.3; Allott et al 1990).

Outdoor dust may be resuspended into the air by wind or human-induced mechanical forces, such that the main drivers of particle resuspension are typically mechanical stressors such as vehicular traffic, construction and agricultural operations, and, generally to a lesser extent, the wind (ISA, section 2.3.1.3; 2006 CD, section 2.3.3). Wind resuspension, often defined in terms of a resuspension rate (the fraction of a surface contaminant released per unit time) is dependent on many factors, including wind speed, soil/surface moisture, particle size, presence of saltating particles and presence of vegetation; typical values range over several orders of magnitude (ISA, section 3.3.1.3; 2006 CD, section 2.3.3). Vehicular resuspension results from shearing stress of tires or turbulence generated by a passing vehicle and can be affected by a number of factors including vehicle size, vehicle speed, soil or surface moisture, and particle size (ISA, section 2.3.1.3; 2006 CD, section 2.3.3). Variability and uncertainty in these factors, and with regard to surface soil/dust composition, affect quantitative emissions estimates for these processes (2006 CD, section 2.3.3).[7]

The relative importance of resuspension of previously deposited dust particles as an influence on airborne Pb concentrations will depend on site-specific circumstances, such as the magnitude of Pb concentrations in the surface dust and air Pb contributions from nearby sources of new Pb emissions, as well as with variation in the forces that influence particle resuspension. For example, the highest air Pb concentrations associated with resuspension appear to occur in areas of highly contaminated surface dust associated with historically active industrial sites, which may or may not be currently active (ISA, sections 2.3.1.3). Air concentrations near such sites with currently active facilities (e.g., metals industries) will reflect the impact of emissions from the current industrial activity in addition to that from resuspension of any previously deposited material (often a component of "fugitive" emissions estimates[8]). Accordingly, as might be expected, the limited data available for comparison indicate the relative magnitude of

[7] 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.

[8] 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).

air Pb concentrations associated with no-longer-active industrial sites to be generally lower than that for active sites yet greater than that in locations somewhat removed from industrial sources (section 2.2.2.2 below).

Lead-bearing particulate matter in other, non-industrial, locations of appreciable historic Pb contamination, such as in soils or on surfaces in older urban areas or near older transportation corridors may, if disturbed, also become suspended into the air and contribute to air Pb concentrations, although the availability of such material for resuspension might be expected to decline over time in most locations due to transport and removal processes. In older transportation corridors or other locations not influenced by active industries, the significance of resuspension (e.g., in terms of resultant air Pb concentrations) appears to be much less than that associated with active industries or now-closed industries with substantial emissions in the past.[9] For example, the available data indicate that current Pb concentrations near roadways are substantially lower than those near large, currently active industrial sources (see ISA, sections 2.3.1.3 and 2.5.1.2 and Figures 2-9 and 2-11 below). In general, air Pb concentrations at sites described as not influenced by an active industry are much lower than those near active sources (see ISA, section 2.5.1.2 and Figure 2-11 below).

2.1.3 Sources and Emissions on Local Scale

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

[9] 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).

Table 2-2. Facilities estimated to emit at least 0.50 tpy of Pb 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	4	3	2.3 - 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 or Graphite Plant	1	1	6.3		
Primary Non-ferrous Metal Smelting/Refining Plant (not Lead, Gold, Aluminum, or Copper)	1	1	5.4		
Mines/Quarries	5	2	1.6 - 3.8		
Ethanol Biorefineries	2	2	1.5 - 3.8		
Portland Cement Manufacturing	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 Board Manufacturing Plant	1	1	2.3		
Foundries, Iron and Steel	3	3	1.1 - 2.3	10	8
Foundries, Non-ferrous	3	2	1.2 - 2.0	2	2
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	2	2
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
Glass Plant				2	2
Fabricated Metal Products Plant				5	4
Municipal Waste Combustor				3	3
Lumber/Sawmill				2	1
Plastic, Resin, or Rubber Products Plant				1	1
Taconite Processing Plant				1	1
Petroleum Storage Facility (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 additional uncertainty regarding this total emissions estimate for this facility, the only remaining operational primary Pb smelter in the U.S., which ceased smelter operations at this site at the end of 2013 (USEPA, 2012).

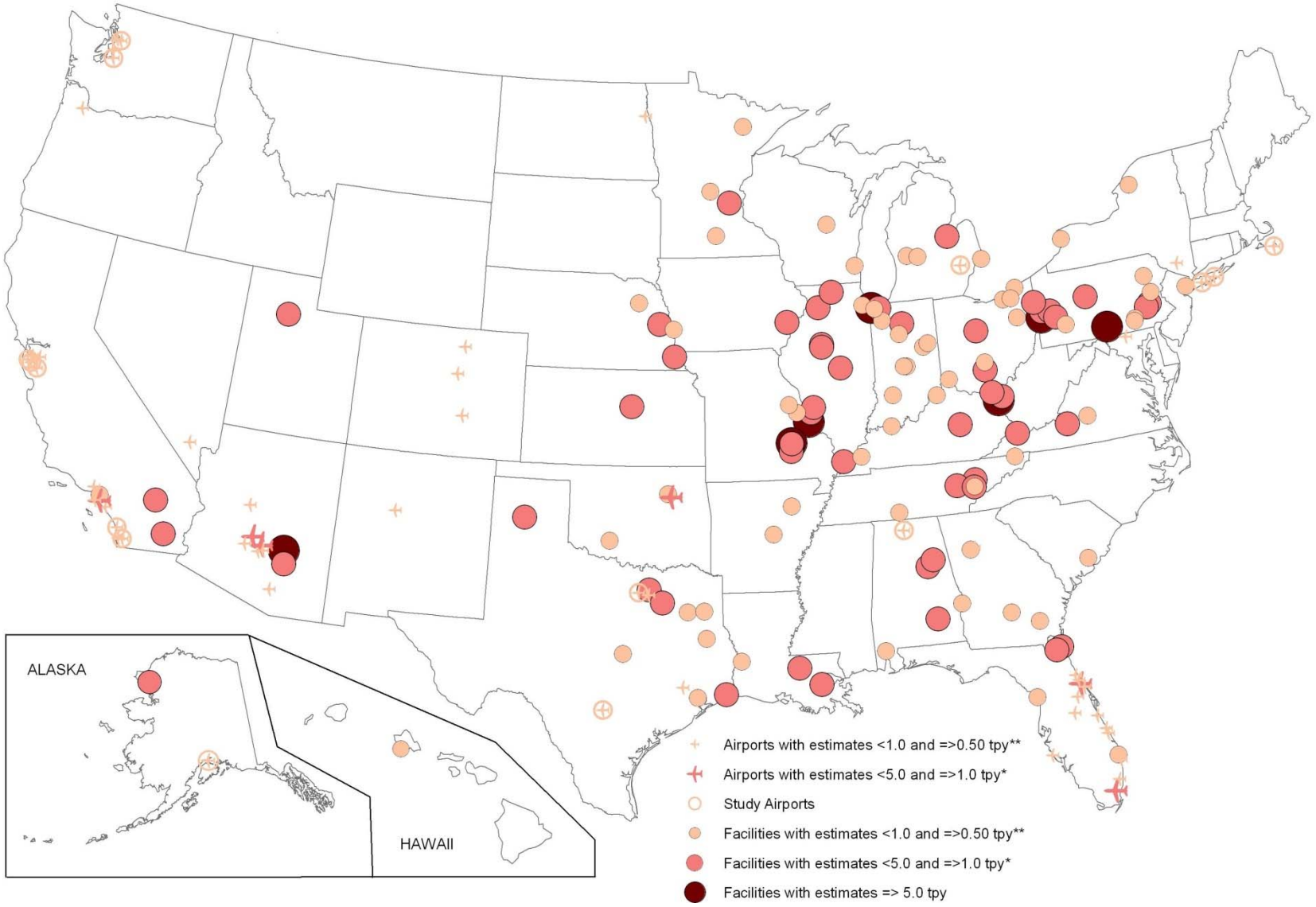

Figure 2-2. Geographic distribution of facilities and airports estimated to emit at least 0.50 tpy of Pb in 2008.

2.2 AMBIENT AIR QUALITY

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

2.2.1 Air Monitoring

Ambient air Pb concentrations are measured by five national monitoring networks. The networks include the State and Local Air Monitoring Sites (SLAMS) intended for Pb NAAQS surveillance, the $PM_{2.5}$ Chemical Speciation Network (CSN), the Interagency Monitoring of Protected Visual Environments (IMPROVE) network, the National Air Toxics Trends Stations (NATTS) network, and the Urban Air Toxics Monitoring program. All of the data from these networks are accessible via EPA's Air Quality System (AQS): http://www.epa.gov/ttn/airs/airsaqs/. In addition to these networks, various environmental organizations have operated other sampling sites yielding data (which may or may not be accessible via AQS) on ambient air concentrations of Pb, often for limited periods and/or for primary purposes other than quantification of Pb itself. The subsections below describe each network and the Pb measurements made at these sites.

2.2.1.1 Lead NAAQS Surveillance Network

This section describes sample collection, analysis and network aspects for the Pb SLAMS network, the main purpose of which is surveillance for the Pb NAAQS. As such, the EPA regulates how this monitoring is conducted in order to ensure accurate and comparable data for determining compliance with the NAAQS. The code of federal regulations (CFR) at parts 50, 53 and 58 specifies required aspects of the ambient monitoring program for NAAQS pollutants.[10] In order to be used in NAAQS attainment designations, ambient Pb concentration data must be obtained using either the federal reference method (FRM) or a federal equivalent method (FEM). The indicator for the current Pb NAAQS is Pb-TSP. However, in some situations,[11] ambient Pb-

[10] The 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.

[11] The Pb-PM_{10} measurements may be used for NAAQS monitoring as an alternative to Pb-TSP measurements in certain conditions defined in 40 CFR part 58 Appendix C, section 2.10.1.2. These conditions include where Pb concentrations are not expected to equal or exceed 0.10 micrograms per cubic meter as an

PM_{10} concentrations may be used in judging nonattainment. Accordingly, FRMs have been established for Pb-TSP and for Pb-PM_{10}. The current FRM for the measurement of Pb-TSP is provided in 40 CFR part 50 Appendix G. This FRM includes sampling using a high-volume TSP sampler that meets the design criteria identified in 40 CFR part 50 Appendix B and sample analysis for Pb content using flame atomic absorption. There are 24 FEMs currently approved for Pb-TSP.[12] All 24 FEMs are based on the use of high-volume TSP samplers and a variety of approved equivalent analysis methods.

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

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

Monitoring agencies are also required, under 40 CFR, part 58, Appendix D, to conduct non-source-oriented Pb monitoring at the NCore sites required in metropolitan areas with a population of 500,000 or more.[14] NCore is a network of multipollutant monitoring stations

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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 PM_{10} monitors.

[12] 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

[13] 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)).

[14] Defined by the US Census Bureau - http://www.census.gov/population/www/metroareas/metroarea.html

intended to meet multiple monitoring objectives that formally began in January 2011. The NCore stations are a subset of the state and local air monitoring stations network and are intended to support long-term trends analysis, model evaluation, health and ecosystem studies, as well as NAAQS compliance. The complete NCore network consists of approximately 60 urban and 20 rural stations, including some existing SLAMS sites that have been modified for additional measurements. Each state will contain at least one NCore station, and 46 of the states plus Washington, DC, will have at least one urban station.

Either Pb-TSP or Pb-PM_{10} monitoring may be performed at these sites. Of 50 NCore sites measuring Pb concentrations as of April 2014, 28 are measuring Pb in TSP and 24 are measuring Pb in PM_{10} (two sites are measuring both Pb in TSP and Pb in PM_{10}). While non-source-oriented monitoring data can be used for purposes of NAAQS attainment designations, a main objective for non-source-oriented monitoring is to gather information on neighborhood-scale Pb concentrations that are typical in urban areas in order to better understand ambient air-related Pb exposures for populations in these areas.

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

[15] This figure reflects Pb-TSP monitors in AQS as of September 2012.

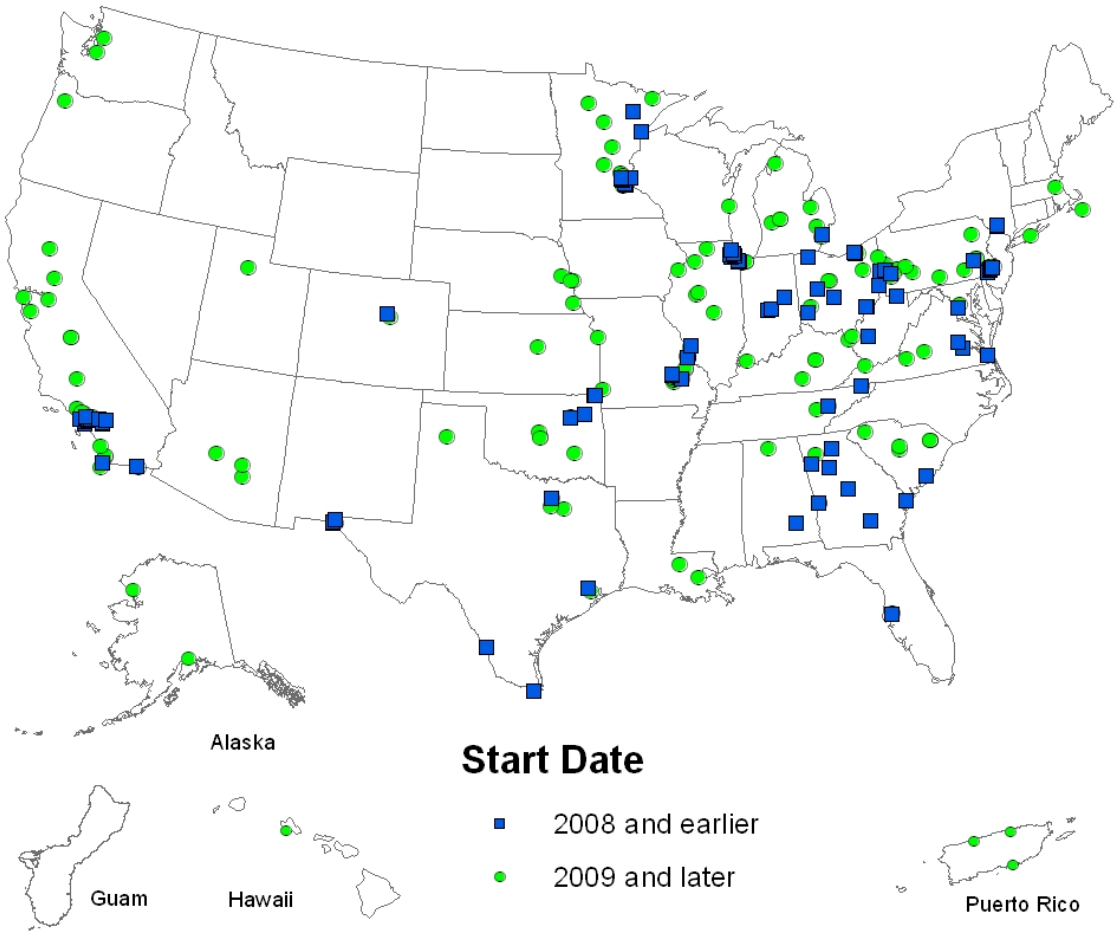


Figure 2-3. Map of Pb-TSP monitoring sites in current Pb NAAQS monitoring network.

The current regulations also required one year of Pb-TSP monitoring (using FRM or FEM methods) near 15 specific airports in order to gather additional information on the likelihood of NAAQS exceedances near airports due to the combustion of leaded aviation gasoline (75 FR 81126). These airports were selected based on three criteria: annual Pb inventory between 0.5 ton/year and 1.0 ton/year, ambient air within 150 meters of the location of maximum emissions (e.g., the end of the runway or run-up location), and airport configuration and meteorological scenario that leads to a greater frequency of operations from one runway. These characteristics were selected because they are expected, collectively, to identify airports with the highest potential to have ambient Pb concentrations approaching or exceeding the Pb NAAQS. Data from this monitoring study will be used to assess the need for additional Pb monitoring at airports. These 15 sites (Figure 2-4) were intended to be operational no later than December 27, 2011, although delays in monitor siting extended the installation of some of these monitors into late 2012.

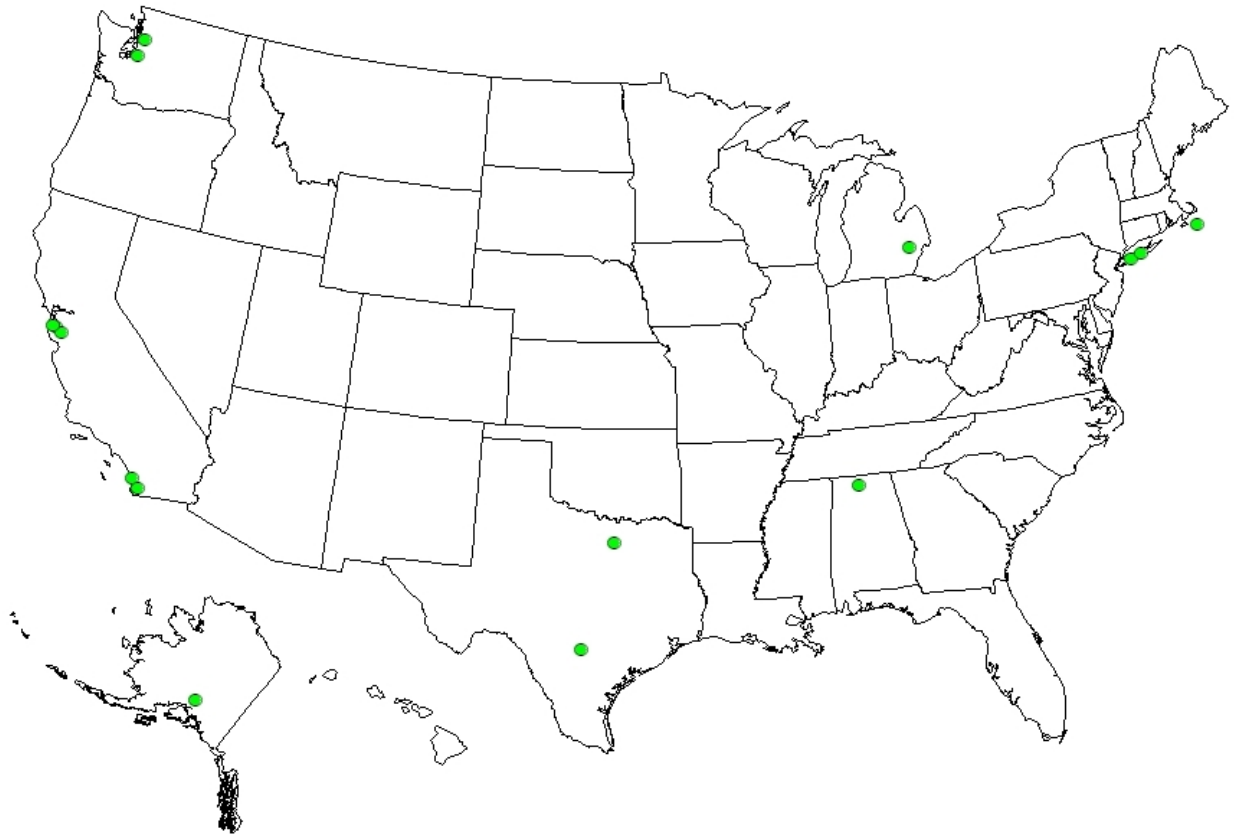


Figure 2-4. Sites near airports at which one year of Pb-TSP monitoring is required.[16]

2.2.1.2 Other Lead Monitoring Networks

The NATTS network, designed to monitor concentrations of hazardous air pollutants including Pb compounds, included 25 sites measuring Pb-PM_{10} as of 2012. Most of these sites (20) are in urban areas (Figure 2-5). In addition to the NATTS network, as of 2012, states were collecting Pb-PM_{10} at an additional 22 sites (most as part of the Urban Air Toxics Monitoring program). All collect particulate matter as PM_{10} for toxic metals analysis using either a high-volume PM_{10} sampler or a low volume PM_{10} sampler, typically on a 1 in 6 day sampling schedule. Most of these monitoring locations are not measuring using FRM/FEM methods at this time. Lead in the collected sample is generally quantified via an ICP/MS method. The standard operating procedure for metals by ICP/MS is available at:

[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).

http://www.epa.gov/ttn/amtic/airtox.html . As noted in section 2.2.1.1 above, non-source-oriented Pb monitoring using FRM/FEMs that is required at the 51 NCore sites with a population of 500,000 or more (shown in Figure 2-5) may be Pb-PM_{10}. As shown in Figure 2-5, there are some cases where states have addressed their NATTS and NCore Pb monitoring needs with the use of a single monitoring site or may have nearby sites.

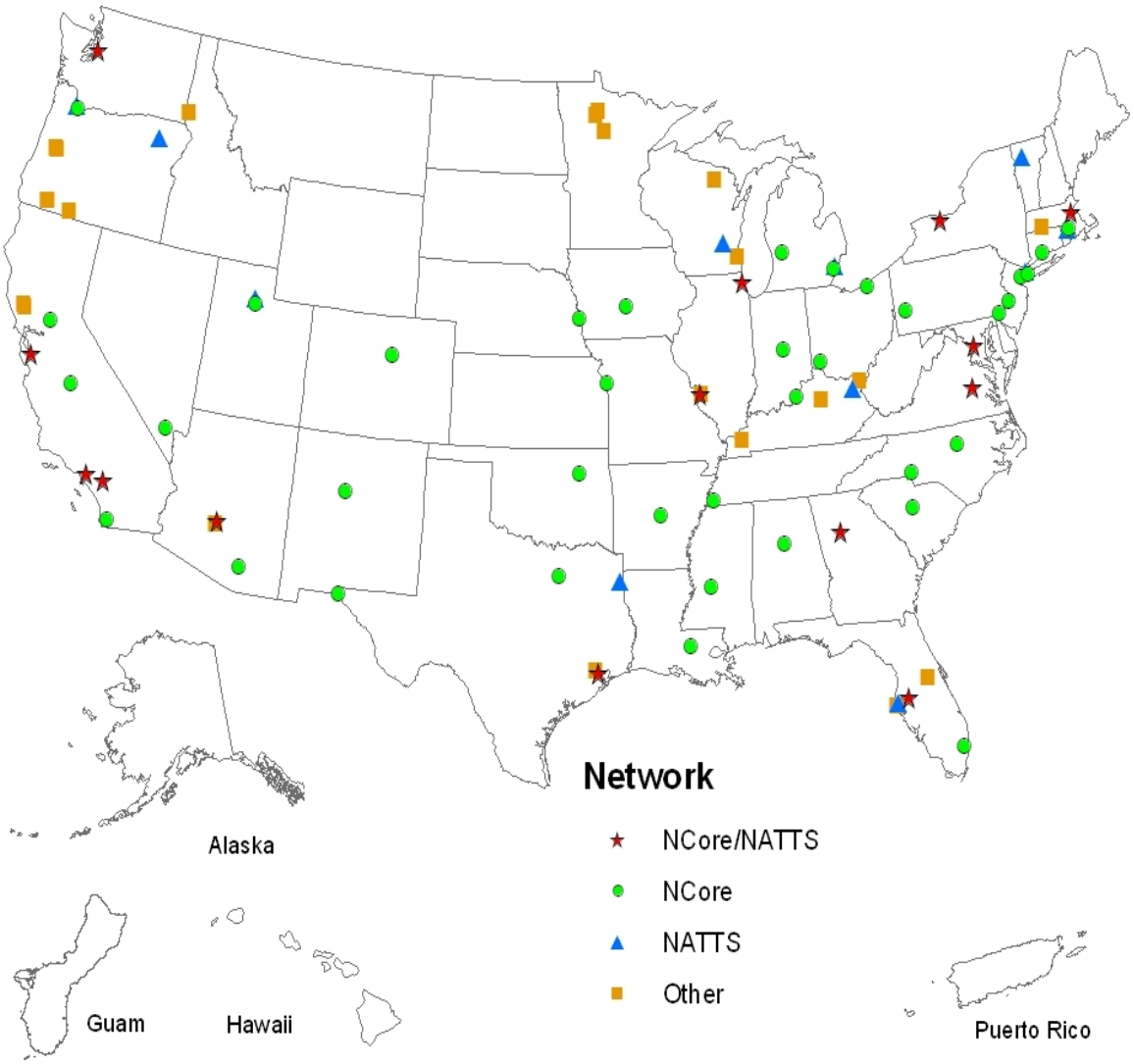


Figure 2-5. Pb-PM_{10} monitoring sites.[17]

[17] Presented on this map are all NCore sites where FRM/FEM Pb monitoring is required along with other sites actively monitoring Pb-PM_{10} (by any method) based on having 2012 data in AQS as of September 2012.

Two networks measure Pb in $PM_{2.5}$, the EPA CSN and the IMPROVE network. The CSN consists of 53 long-term trends sites (commonly referred to as the Speciation Trends Network or STN sites) and approximately 150 supplemental sites, all operated by state and local monitoring agencies. Most STN sites operate on a 1 in 3 day sampling schedule, while most supplemental sites operate on a 1 in 6 day sampling schedule. All sites in the CSN network determine Pb concentrations in $PM_{2.5}$ samples. Lead is quantified via the XRF method.[18] Data are accessible through AQS. The locations of the CSN are shown in Figure 2-6. Nearly all of the CSN sites are in urban areas, often at the location of highest known $PM_{2.5}$ concentrations. The first CSN sites began operation around 2000.

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

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

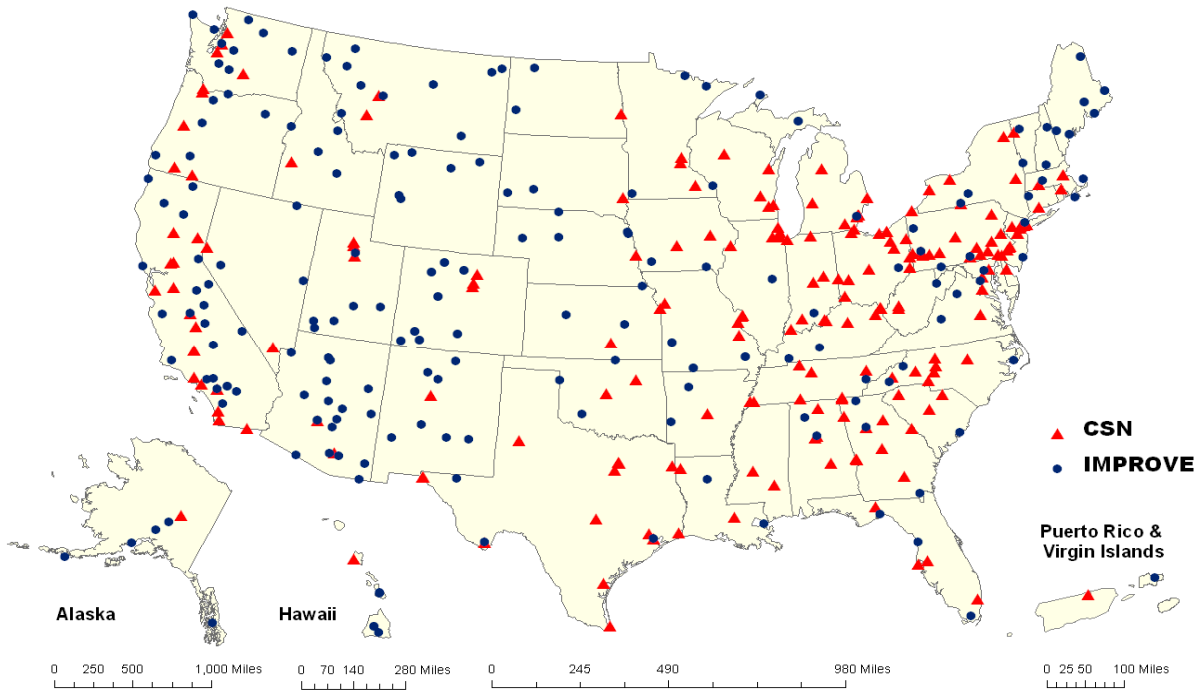


Figure 2-6. Pb-$PM_{2.5}$ monitoring sites in CSN and IMPROVE networks (2012).

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 are 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.[19]

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

[19] The code of federal regulations (CFR) at parts 50, 53 and 58 specifies required aspects of the ambient monitoring program for NAAQS pollutants. The FRMs for sample collection and analysis are specified in 40 CFR part 50, the procedures for approval of FRMs and 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.

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.

The EPA has initiated efforts to characterize the collection efficiency of alternative sampler designs through wind tunnel testing as a necessary step towards the development of a new sampler capable of sampling particles larger than PM_{10} without the noted wind speed and wind direction biases. Important considerations in specifying a sampler design with these desired features include the feasibility and challenges to collection of particles much larger than 10 μm; some existing alternative sample collection methods are discussed in ISA (ISA, section 2.4.1.1). Also important to consider are physical limitations in the ability to generate and transport ultra-coarse particles that affect the ability to adequately characterize the new sampler. Although all sizes of airborne Pb particles are of interest, the factors identified above are expected to ultimately limit the upper cut-point of potential samplers to the range of 18-20 micrometers. Following characterization in a wind tunnel, field testing of promising candidates would be required to evaluate performance and to make comparisons to the existing Pb-TSP samplers. This effort is expected to take several years to complete, and, as such, it is unlikely that this new sampler will be available in time for consideration during this NAAQS review due to the activities which will need to be completed to adequately characterize its performance both in the laboratory and the field. We expect the new sampler to be available for consideration in a future review and consequently do not expect to consider new alternatives for sampling methods for Pb-TSP as part of this review.

In addition to the FRM for Pb-TSP, there is also, as noted in section 2.2.1.1 above, a FRM for Pb-PM_{10} (40 CFR part 50 Appendix Q), based on the PM_{10} sampler defined in 40 CFR part 50 Appendix J coupled with XRF analysis. The Pb-PM_{10} measurements may be used as an alternative to Pb-TSP measurements in certain conditions defined in 40 CFR part 58 Appendix C paragraph 2.10. These conditions include where Pb concentrations are not expected to equal or exceed 0.10 micrograms per cubic meter on an arithmetic 3-month mean and where the source of Pb emissions is expected to emit a substantial majority of its Pb in the PM_{10} size fraction. At this time, we believe the low-volume FRM sampler for Pb-PM_{10} to be adequate for this application. Hence, we do not expect to consider new sampling methods for Pb-PM_{10} as part of this review.

2.2.1.3.2 Analysis Considerations

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

With regard to Pb-PM_{10} samples, in addition to the FRM analysis method (XRF), two FEMs have been accepted for Pb-PM_{10} analysis since the 2008 Pb NAAQS rulemaking. These methods are based on ICP-MS and are consistent with analysis methods used for the NATTS network. The EPA will continue to consider new FEMs for analysis of Pb-PM_{10} and Pb-TSP as applications are received, although no new FRMs (beyond the Pb-TSP FRM for ICP-MS discussed above) are expected during this NAAQS review.

2.2.1.3.3 Network Design Considerations

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

Source-Oriented Monitoring. Since the phase out of Pb in on-road gasoline, Pb is widely recognized as a source-oriented air pollutant. As summarized in the ISA, variability in air Pb concentrations is highest in areas including a Pb source, "with high concentrations

downwind of the sources and low concentration at areas far from sources" (ISA, p. 2-92). Recent data summarized in section 2.2.2.2 below indicates that the highest ambient Pb concentrations are found near large Pb sources, usually metals industries (see, for example, Figures 2-11 through 2-13 below). Analysis of the monitoring network during the last Pb NAAQS review with regard to adequacy of monitoring near such sources found that monitors were lacking near many of the larger Pb emissions sources, leading the EPA to conclude that the monitoring network existing at that time was inadequate to determine compliance with the revised Pb NAAQS (73 FR 29262). Findings and conclusions of that analysis led to revisions of network design requirements for source-oriented monitoring, begun with the 2008 Pb NAAQS rulemaking (73 FR 66964). Additional revisions were completed as part of a reconsideration of the monitoring requirements in December 2010 (75 FR 81126).

The current requirements for source-oriented monitoring include placement of monitor sites near sources of air Pb emissions which are expected to or have been shown to contribute to ambient air Pb concentrations in excess of the NAAQS. At a minimum, there must be one source-oriented site located to measure the maximum Pb concentration in ambient air resulting from each non-airport Pb source which emits 0.50 or more tons of Pb per year and from each airport which emits 1.0 or more tons of Pb per year.[20] The expansion of the network, including these source-oriented sites, is shown in Figure 2-3. Comparison of Figure 2-3 (monitors) with Figure 2-2 (sources) illustrates the coverage which the monitoring network provides, as of 2012, of large Pb emissions sources.

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

As more recent data become available, analysis of the updated and expanded dataset will inform evaluation of the appropriateness of the current requirements. Since the analysis

[20] 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).

performed during the 2008 review, over 150 additional source-oriented monitors have been installed. At this time, the full set of these new monitors have not collected a complete 3-year dataset, which is needed for the development of complete design values for the new source-oriented monitors. For monitors installed in response to the 2008 revisions (near sources estimated to emit 1.0 tpy or more of Pb), 3-years of certified data would be expected to have been available in spring 2013. Three-years of certified data for the monitors required in the 2010 regulations will be available in spring 2015. As is seen from Figures 2-11 through 2-13 below, Pb concentrations very widely across the source oriented monitors. This variation is not unexpected given potential for appreciable differences in source characteristics beyond estimated annual Pb emissions that would be expected to affect airborne Pb concentrations. Such characteristics include source or industry type and associated processes and work practices, proximity of emissions points to source boundaries, extent of fugitive emissions.

One-year Airport Monitoring. In addition to the above source-oriented monitoring requirement for airports estimated to emit more than 1 tpy of Pb (40 CFR part 58, Appendix D, 4.5(a)) and the Regional authority to require monitoring near airports where piston-engine aircraft emit Pb (40 CFR part 58, Appendix D, 4.5(c)), one year of monitoring was required near 15 specific airports in order to gather additional information on the likelihood of NAAQS exceedances due to the combustion of leaded aviation gasoline (75 FR 81126; 40 CFR part 58, Appendix D, 4.5(a)(iii)). Given delays in monitor siting, monitor installation at some of these sites extended into late 2012. Accordingly, the timing for completion of the year of monitoring has varied across the 15 locations. As described further in section 2.1.2.2 above, these airport monitoring data along with other data gathering and analyses will inform EPA's ongoing investigation into the potential for Pb emissions from piston-engine aircraft to cause or contribute to air pollution that may reasonably be anticipated to endanger public health or welfare. This investigation is occurring under Section 231 of the Clean Air Act (CAA), separate from the Pb NAAQS review. As a whole, the various data gathering and analyses are expected to improve our understanding of Pb concentrations in ambient air near airports and conditions influencing these concentrations.

Non-Source-Oriented Monitoring. Monitoring agencies are also required to conduct non-source-oriented monitoring NCore sites with a population of 500,000 or more, as noted above.[21] Currently, all 50 NCore sites are operational and measuring Pb concentrations, with 28 measuring Pb in TSP and 24 measuring Pb in PM_{10} (2 sites are measuring both Pb in TSP and Pb in PM_{10}). While non-source-oriented monitoring data can be used for designation purposes, an alternative objective stated for these sites is the collection of data on neighborhood-scale Pb

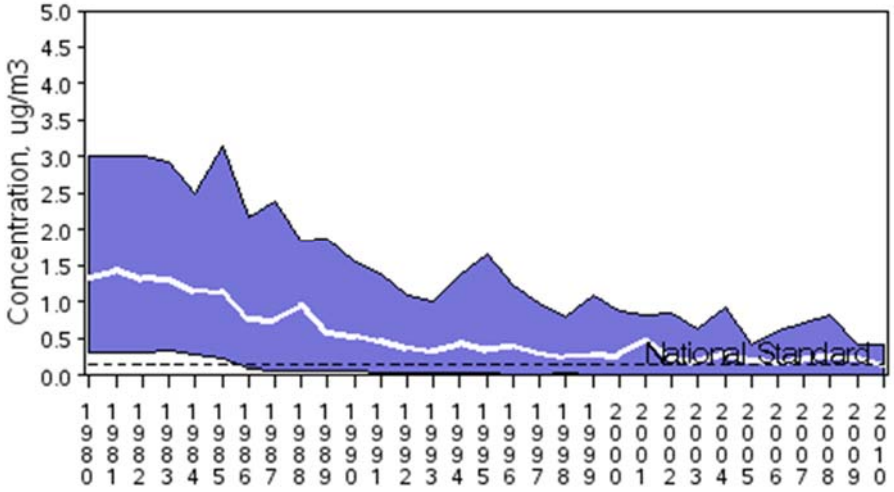
[21] Defined by the US Census Bureau - http://www.census.gov/population/www/metroareas/metroarea.html

concentrations that are typical in urban areas to inform our understanding of ambient air-related Pb exposures for the general population. The data available as of April 2014 for these sites indicate little variation in concentrations, with a range from less than 0.01 to 0.06 $\mu g/m^3$ in terms of maximum 3-month average concentration (of Pb-PM_{10} or Pb-TSP), with the vast majority of sites showing concentrations less than 0.03 $\mu g/m^3$. In recognition of the limited extent of this data analysis, we have not drawn conclusions here on the usefulness of these data for purposes of characterizing neighborhood-scale concentrations. We additionally note the existence of other monitoring networks that, although not required by regulation, provide data on Pb in PM_{10}, and also in $PM_{2.5}$, at non-source-oriented urban sites. These include the NATTS for PM_{10} and the CSN for $PM_{2.5}$, as described in section 2.2.1.2 above.

2.2.2 Ambient Concentrations

2.2.2.1 Temporal Trends

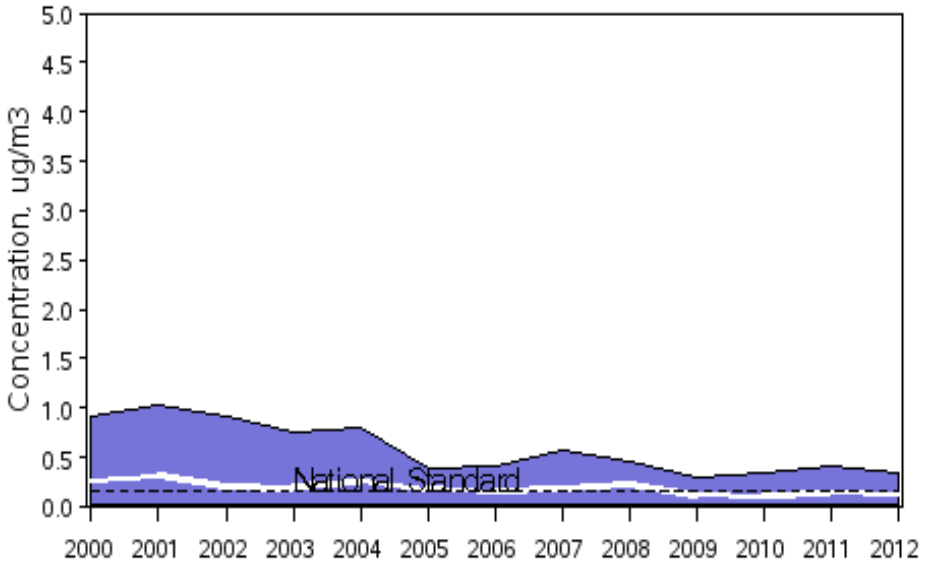
Ambient air concentrations of Pb in the U.S. have declined substantially over the past 30 years. Figure 2-7 illustrates this decline in terms of site-specific maximum 3-month average concentrations at the set of 31 monitoring sites that have been operating across this period. The median of this dataset has declined by more than 90% over the 30-year period, and the average by 89%. Over the past 12 years, a larger dataset of 50 sites operating across that period also indicates a decline, which is on the order of 50% for the average of that dataset (Figure 2-8).[22]



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

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

[22] In Figures 2-7 and 2-8, the top of the blue band is the 90th percentile among sites, the bottom is the 10th percentile, and the white line is the average. http://www.epa.gov/air/airtrends/lead.html

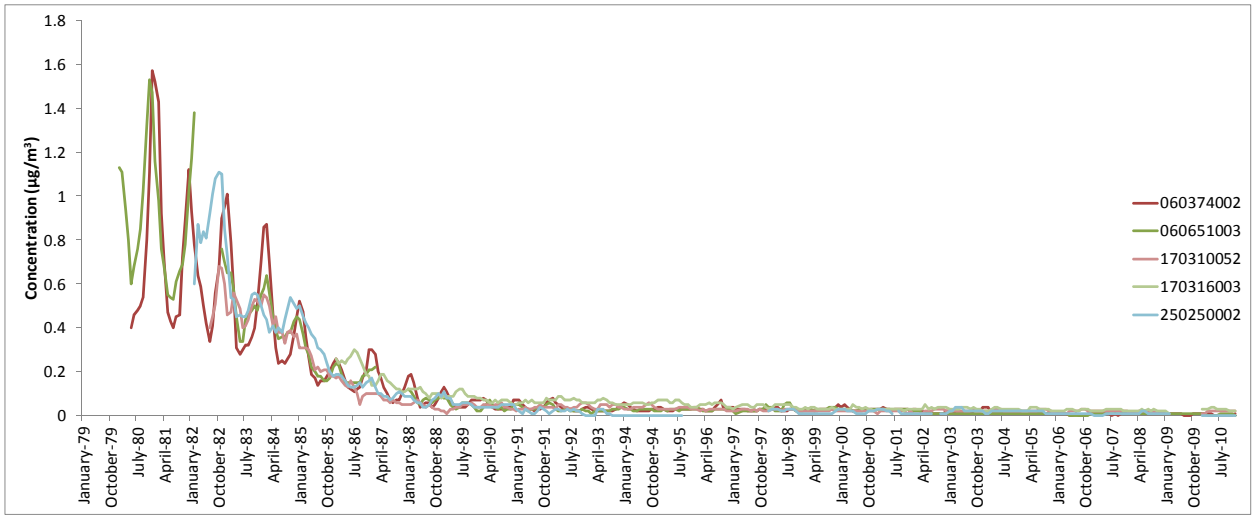


Note: Based on annual maximum 3-month average at 50 sites.

Figure 2-8. Temporal trend in Pb-TSP concentrations: 2000-2012 (50 sites).

The role of the phase-out of leaded gasoline for on-road vehicles on declining concentrations is evident from temporal trends in air Pb concentrations near roadways, as illustrated in Figure 2-9 which presents data for five monitors sited near roadways during the years 1979 through 2010.[23] These sites additionally indicate the concentrations currently common at such sites, with the maximum 3-month average concentrations at all five sites falling below 0.03 $\mu g/m^3$ in the most recent years.

[23] 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.



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).

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

2.2.2.2 Current Concentrations

As a result of revisions to the Pb NAAQS surveillance monitoring requirements (described in section 2.2.1 above), Pb monitoring sites have been in transition over the last few years as indicated by Figure 2-3. For presentation in this document, we have focused on the most recent period for which adequately complete data are available, 2010-2012, recognizing that the dataset developed for this period includes many but not all of the monitors newly required by the December 2010 regulations described in section 2.2.1.1 above.

Lead concentrations, in terms of maximum 3-month average Pb-TSP concentration, at monitoring sites active across the U.S. during the period 2010-2012, and for which sufficient data are available to meet completeness criteria described in Appendix 2C, are presented in Figure 2-10.[24] Highest concentrations occur in the vicinity of large metals industries, as discussed in section 2.1.2.1.[25] Note that due to differences in data completeness criteria which reflect the different purposes of the analyses (characterization of air quality at different types of monitoring sites vs identification of sites exceeding the NAAQS), Figure 2-10 (and Figures 2-11 through 2-13) do not include some sites which may have been reported elsewhere (e.g., with "design values" which are used for comparison to the NAAQS level).[26]

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[24] Criteria for development of the 2010-2012 air Pb-TSP, Pb-PM_{10} and Pb-$PM_{2.5}$ datasets discussed in this section are described in Appendix 2C. Data summaries are included in Appendix 2D.

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

[26] Design values for the 2010-2012 period are available at: http://www.epa.gov/airtrends/values.html

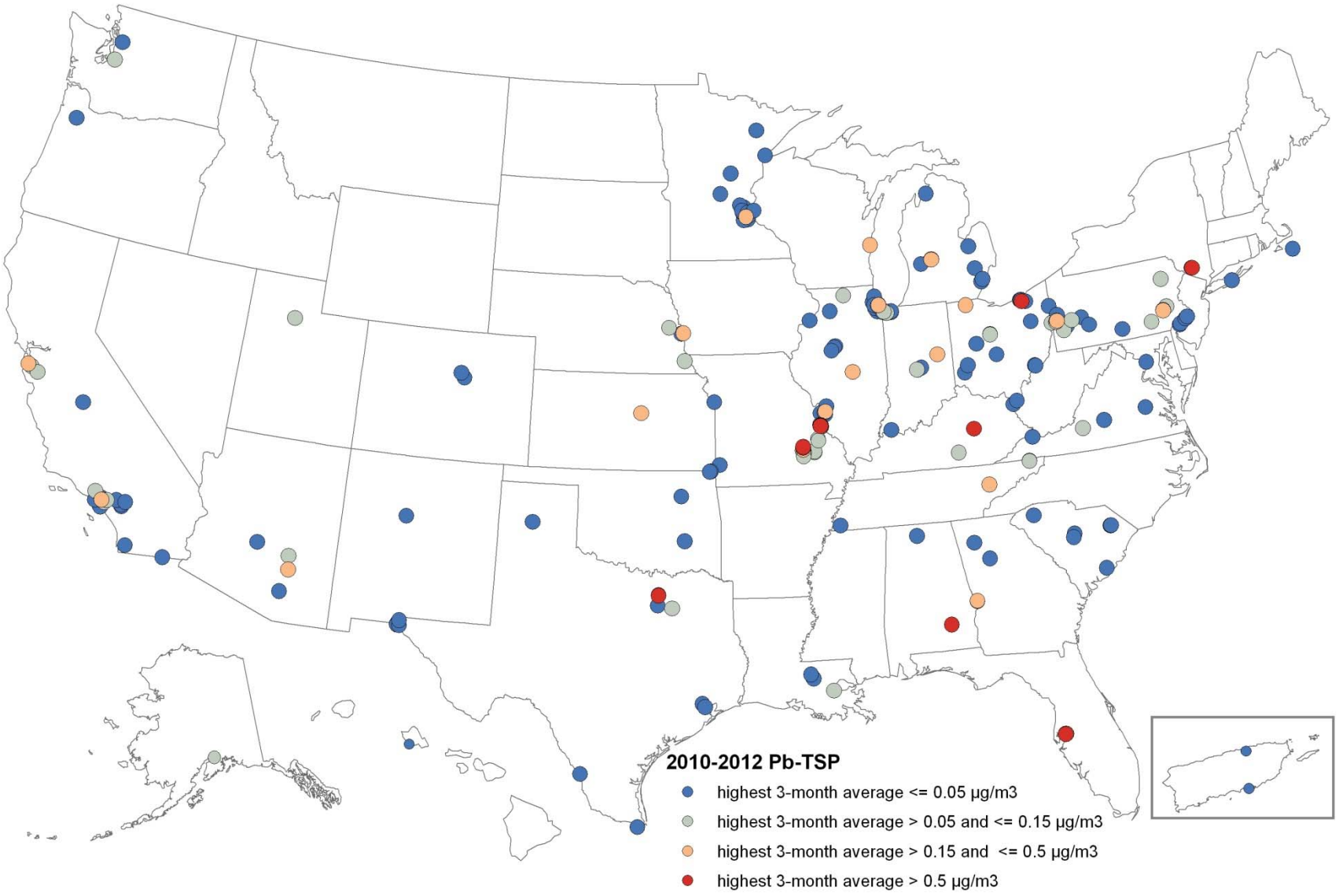

Figure 2-10. Pb-TSP maximum 3-month means (215 sites), 2010-2012.

Figures 2-11 through 2-13 illustrate differences in Pb-TSP concentrations, in terms of three different metrics (maximum 3-month mean, annual mean, and maximum monthly mean), among sites near and more distant from emissions sources.[27] The Pb-TSP sites indicate the much greater site and temporal variability in concentrations at source-oriented and previous source-oriented sites as compared to non-source-oriented sites. Across the Pb-TSP sites, as would be expected, the highest concentrations are observed at the source-oriented sites, followed by the previous source-oriented sites. This is the case for all three metrics analyzed (maximum 3-month mean, maximum monthly mean and annual mean).

Figures 2-11 through 2-13 additionally present distributions of Pb-PM_{10} and Pb-$PM_{2.5}$ concentrations in urban and rural locations where they are monitored. The Pb-PM_{10} and Pb-$PM_{2.5}$ networks are described in section 2.2.1.2 and shown in Figures 2-5 and 2-6, respectively. It is important to note that there are few sites in these recent datasets with colocated monitors for the different size fractions. As described in the ISA, at 18 urban sites (without specification as to proximity to Pb sources) with at least 30 co-located Pb-TSP and Pb-PM_{10} samples collected during various time periods (nearly all between 1990 and 2000), approximately 80% of the Pb mass, on average, is captured by the Pb-PM_{10} measurements (ISA, sections 2.5.3.1 and 2.8.4). The data distributions in Figures 2-11 through 2-13 indicate reduced variability in concentration for non-source-oriented sites and for particulate Pb of smaller size fractions.[28]

[27] In the Figures 2-11 through 2-13, the whiskers indicate the 5th and 95th percentiles, the box indicates the 25th 50th and 75th percentiles and the star indicates the arithmetic mean.

[28] The number of observations for some categories of monitoring site varies among the three figures due to the impact of data completeness criteria used for the three different concentration metrics (see Appendix 2C).

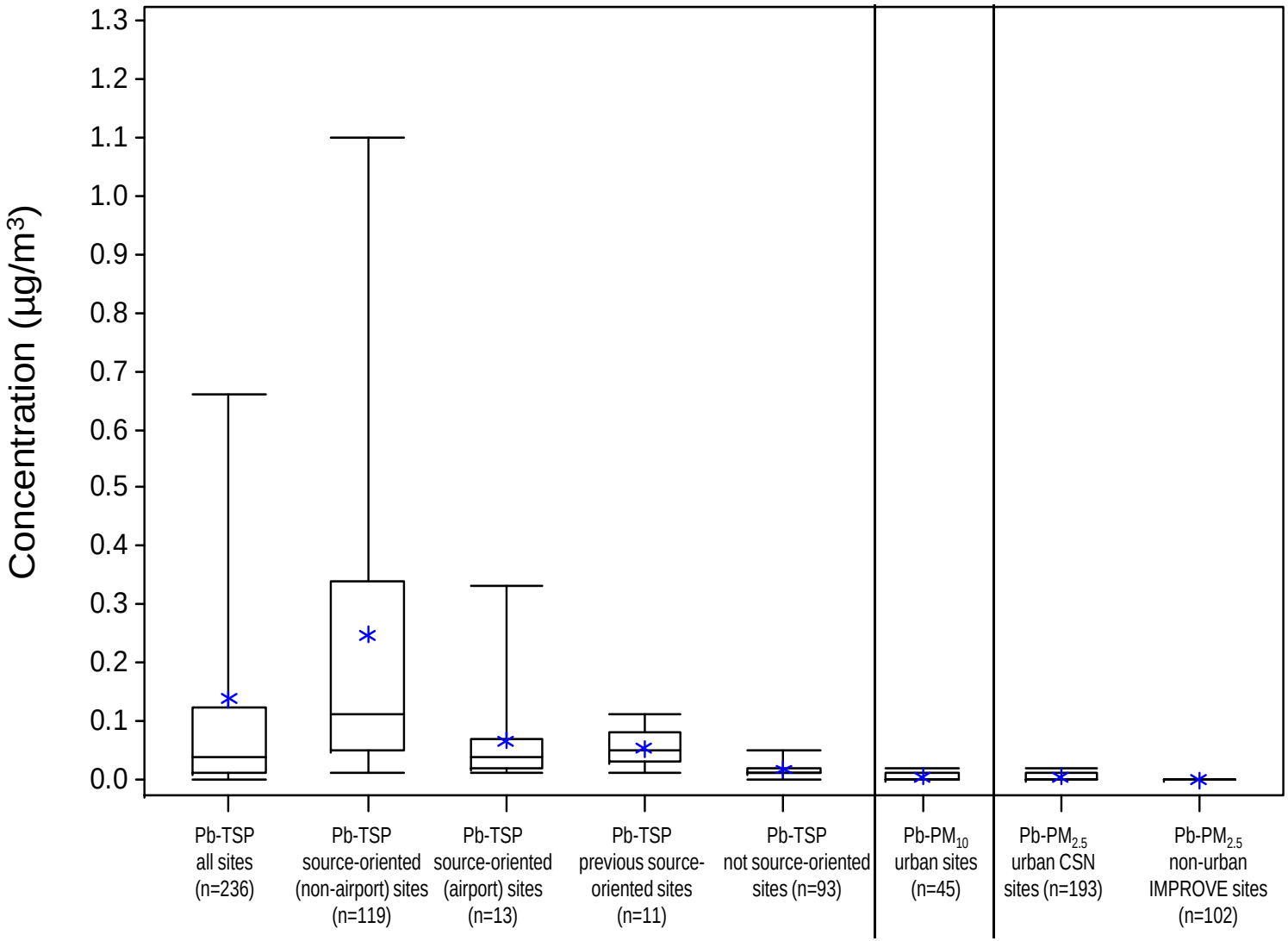

Figure 2-11. Distribution of maximum 3-month mean concentrations of Pb-TSP, Pb-PM_{10} and Pb-$PM_{2.5}$ at different site types, 2010-2012.

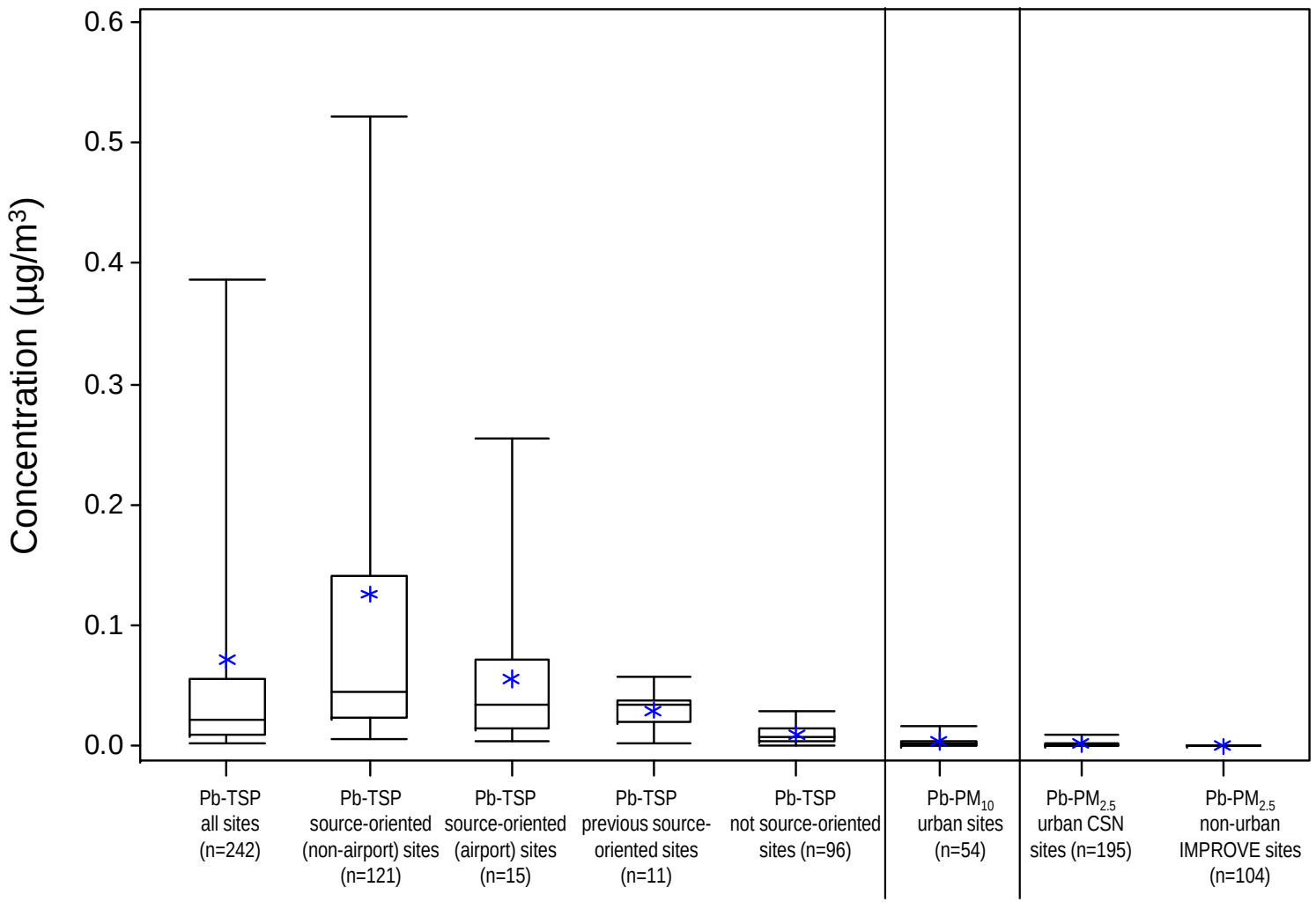

Figure 2-12. Distribution of annual mean concentrations of Pb-TSP, Pb-PM_{10} and Pb-$PM_{2.5}$ at different site types, 2010-2012.

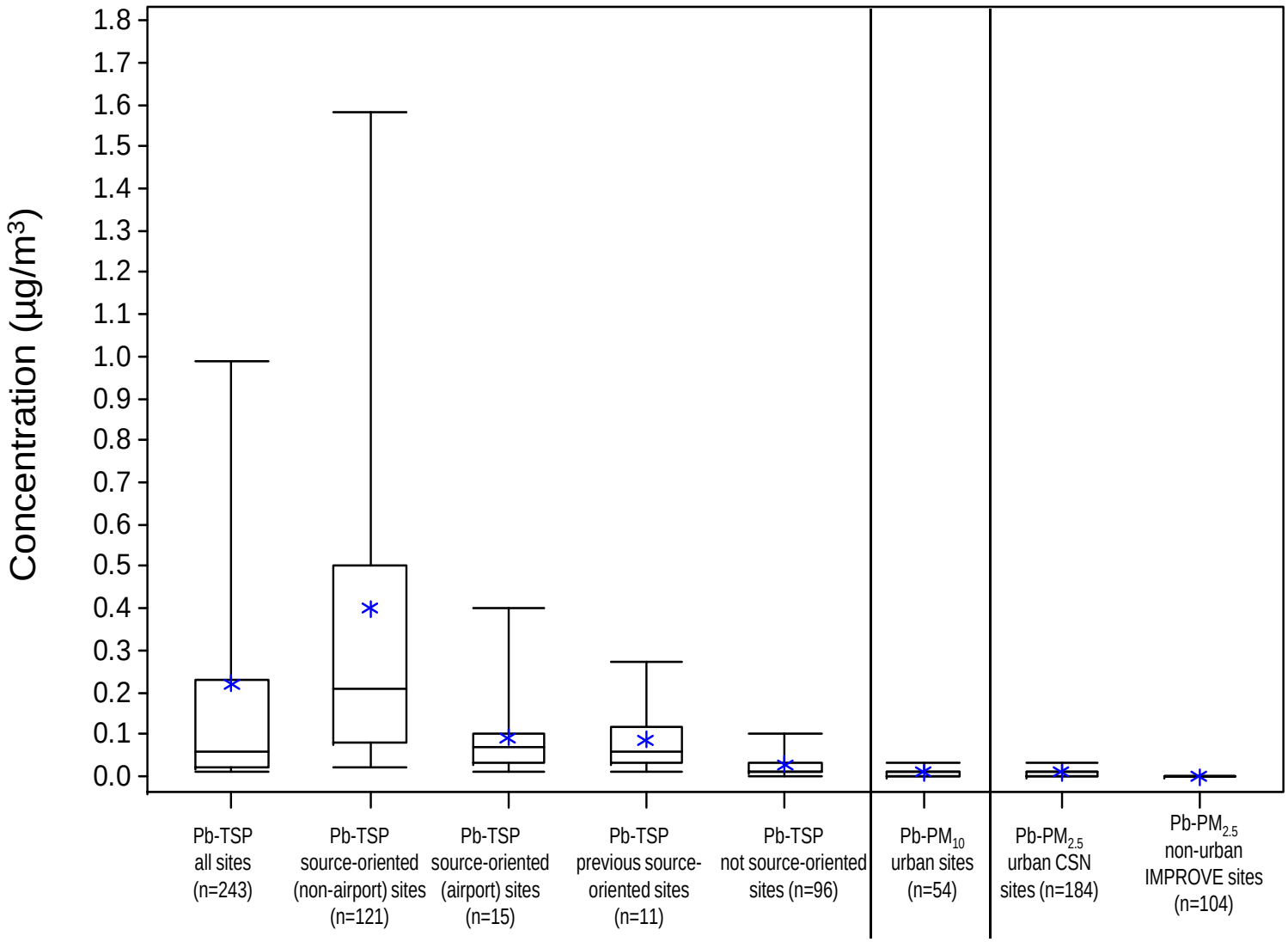

Figure 2-13. Distribution of maximum monthly mean concentrations of Pb-TSP, Pb-PM_{10} and Pb-$PM_{2.5}$ at different site types, 2010-2012.

2.3 AMBIENT AIR LEAD IN OTHER MEDIA

Lead emitted into the ambient air, depending on its chemical and physical characteristics, deposits out of air onto surfaces in the environment. The environmental fate of atmospherically emitted Pb, once deposited, is influenced by the type of surface onto which the particles deposit and by the type and activity level of transport forces in that location. Deposited Pb can be incorporated into soil matrices in terrestrial ecosystems or transported (by direct deposition or stormwater runoff) to water bodies and into aquatic sediments, which may serve as aquatic ecosystem sinks. Particles deposited onto impervious surfaces, such as urban surfaces or roadways, are more available for human contact while they remain on such surfaces. For example, particles on urban surfaces may be available for adherence onto skin or they may be resuspended into the air and inhaled. Precipitation and other natural, as well as human-influenced, processes contribute to the fate of such particles and their potential transport to areas or environments of lesser or greater likelihood for human contact, e.g., transport with surface runoff into nearby water bodies or tracking into nearby houses (ISA, section 2.3).

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

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

2.3.1 Atmospheric Deposition

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

dry periods (dry deposition); dry deposition is also estimated from measurements of airborne Pb particles coupled with estimates of deposition velocity (see 2006 CD, Section 2.3.2). Less direct approaches include monitoring changes in Pb concentration in various biological or physical media such as lichen or mosses, snowpack, soil and aquatic sediments (ISA, sections 2.6.2, 2.6.4 and 2.6.6). Approaches for the latter three media may involve repeated measurements of surface samples over time or core samples coupled with isotope dating. To gain information on atmospheric deposition unaffected by contributions from direct nonair environmental releases, such studies are generally conducted in somewhat remote areas. As there are currently no nationwide Pb atmospheric deposition monitoring programs, the discussion of atmospheric deposition in this and subsequent sections is drawn from this range of approaches (2006 CD, sections 2.3, 8.2.2 and AX7.1.2.3; ISA, sections 2.3.1.2 and 2.6). Geographic differences in deposition generally relate to differences in the amount and size distribution of airborne Pb particles in that location, as well as meteorology and other site-specific factors. For example, the size of particles, as well as solubility in rainwater, can also influence wet deposition rates (ISA, section 2.3.1.2; 2006 CD, p. 2-59). Factors that particularly influence dry deposition are the level of atmospheric turbulence, especially in the layer nearest the ground, particle size distributions and density, and the nature of the surface itself, such as smooth or rough (2006 CD, pp. 2-55 to 2-57).

Evidence for the temporal pattern of U.S. Pb deposition generally indicates a peak during the middle twentieth century (e.g., 1940s through 1970s) followed by substantial declines in the latter part of the century (ISA, section 2.3.1; 2006 CD section 2.3.1; Jackson et al., 2004). This pattern reflects the increased use of Pb in the industrial age (including its use in gasoline in the twentieth century) and declines in response to environmental controls on gasoline and metals industries in the latter part of the twentieth century. Studies continue to document declines in atmospheric deposition since the 1980s in locations remote from industrial areas (e.g., Watmough and Dillon, 2007). As would be expected, stationary sources of Pb continue to contribute to relatively higher deposition rates in industrial areas as compared to other areas, although rates in those areas appear to have also declined since the 1970s-80s (ISA, section 2.3.1.2; Sabin and Schiff, 2008).

In addition to records of atmospheric deposition in the U.S., evidence also documents the long-range atmospheric transport and distribution of the substantial emissions of Pb from the U.S. and other developed countries to regions well beyond those source countries. For example, evidence provided by isotopic analyses of rainwater and surface water in the North Atlantic and ice cores in Greenland have documented the long-range transport and distribution of Pb from North American and European sources (ISA, section 2.5.5; 2006 CD, p. AX7-141; 1977 CD, section 6.3.1; Veron et al., 1998). The historical record provided by those cores indicate the

period of most substantial deposition to that remote region during the 1960s and 1970s followed by a dramatic decline (ISA, section 2.5.5; McConnell and Edwards, 2008). These and other records of long-range transport indicate the widespread distribution of historically emitted Pb (e.g., 2006 CD, section 2.3.1).

2.3.2 Terrestrial Media

Lead in the terrestrial media discussed here may originate from current or historical air emissions or other sources. Other sources to these media include historical use of indoor and outdoor leaded paint, more prevalent in areas with older buildings, and the processing of lead-containing materials. Further, the concentrations of Pb in each media, as well as the relative contributions from various types of sources to those concentrations, vary among media and among locations for any one media. Differences among locations in media concentrations of air-related Pb are generally related to the current and historical magnitude of Pb emissions and deposition at that location, as well as location- and media-specific removal factors.

2.3.2.1 Indoor Household Dust

Household dust arises from particulate matter generated indoors and outdoors, and Pb in household dust can reflect a combination of these sources.[29] Airborne particles can be transported indoors with ambient air, while particles deposited outdoors from ambient air may be carried in on humans and their clothing or other items transported indoors. Depending on factors such as the proximity of the residence to current or historical metals industries, leaded paint usage, and age of the residence (which informs consideration of potential for presence of leaded paint), Pb in indoor residential dust can reflect current or historical atmospheric Pb or nonair-related sources, such as leaded paint. Another indoor source is tobacco smoking (ISA, section 3.1.3.2; Gaitens et al., 2009; 2006 CD, p. 3-15; Mannino et al., 2003). The age of the residence may be an indicator of the potential for the presence of leaded paint and, in areas of significant levels of airborne Pb in the past, such as near mining or smelting industries, may also indicate the potential for the residual presence of historically emitted Pb (ISA, sections 3.1.1 and 3.1.3.2; 2006 CD section 3.2.3). A study of households in the Baltimore metropolitan area (in which there are no areas designated non-attainment for the current Pb NAAQS)[30] indicated indoor air Pb concentrations to be significantly associated with outdoor air Pb concentrations but did not show a statistically significant relationship of outdoor air Pb with indoor dust Pb (ISA, 3.1.3.2; Egeghy et al., 2005). Rather, indoor dust Pb was associated with Pb concentration in soil and

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

[30] http://www.epa.gov/air/oaqps/greenbk/mnc.html

several factors related to housing age, perhaps indicating the greater role of sources other than air Pb for those households (Egeghy et al., 2005).

2.3.2.2 Outdoor Dust in Areas of Human Activity

Lead in particulate matter occurring on outdoor surfaces (outdoor dust) may reflect current or historical Pb emissions, as well as the historical uses of Pb in products on buildings and infrastructure in surrounding areas. The concentrations of air-related Pb in outdoor dust and the relative contribution of air-related Pb to the total Pb concentrations in outdoor dust vary depending on the location-specific characteristics of air–related (and other) Pb sources. The role of air-related sources in outdoor dust Pb has been documented in areas near industrial sources, such as smelters, where Pb concentrations in outdoor dust have been documented to decline in response to reduced emissions. For example, outdoor dust Pb concentrations in a long-time Canadian smelter town were found to track smelter Pb emissions, as a new smelting technology which reduced airborne Pb concentrations by 75% resulted in a 50% reduction in outdoor dust Pb loading rate and concentrations (2006 CD, p. 3-23; Hilts, 2003). The residence time of settled dust Pb (e.g., on sidewalks and other public surfaces) and thus the response of settled dust Pb concentrations to changes in emissions and associated atmospheric deposition of Pb particles are expected to reflect site-specific rates of transport or removal processes, such as stormwater runoff and atmospheric resuspension and transport (ISA, sections 2.3.1.3 and 2.3.2.4; Allott et al., 1990; Wong et al., 2006).

Rates of dry deposition of Pb in large metropolitan areas during the past decade are much lower than those reported for the 1970s (ISA, sections 2.3.1 and 2.6.1; Table 2-4, below). For example, dry deposition of Pb into Los Angeles harbor during 2002-2006 was more than an order of magnitude lower than rates reported for the same location in 1975 (ISA section 2.3.1.2; Sabin and Schiff, 2008; Lim et al., 2006). Across the Los Angeles metropolitan area, average rates generally ranged from 10 to 32 micrograms per square meter per day ($\mu g/m^2$/day) and down to 0.3 $\mu g/m^2$/day for the non-urban site of Malibu during 2002-2003 (Lim et al., 2006). Rates within this range are reported from studies in Manhattan and a non-industrial area of New Jersey, with a somewhat higher rate reported for a major industrial location within the New York City metropolitan area (Yi et al., 2006; Caravanos et al., 2006a). Although rates have not been reported recently for smelter locations in the U.S., the outdoor dustfall deposition rate reported in a smelter town in British Columbia, Canada, after the installation of new technology that reduced average airborne Pb-TSP concentrations to a level still at least twice the current U.S. NAAQS (0.3 $\mu g/m^3$ from prior average of 1.1 $\mu g/m^3$) was several orders of magnitude higher than those reported for New York, Los Angeles and Chicago urban areas (Hilts, 2003; Lim et al., 2006; Sabin et al., 2006; Yi et al., 2006; Caravanos et al., 2006a).

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

Table 2-3. Dry deposition of Pb in large metropolitan areas.

Description	**Average** (or Median*) **Dry Deposition Rate** (μg Pb/m^2/day) [A]	**Study**
Los Angeles Harbor, June-Nov 2006	14*[B]	Sabin and Schiff, 2008
Los Angeles Harbor, 2002-2003[C]	15	Lim et al., 2006
Los Angeles Harbor, 1975	300*	Sabin and Schiff, 2008
Los Angeles, metropolitan area urban sites, 2002-2003 [C]	10-32	Lim et al., 2006
Los Angeles, metropolitan area, non-urban site Malibu, 2002- 2003 [C]	0.3	"
Los Angeles, I-405, near Westwood (downwind side), spring 2003 [D]	24	Sabin et al., 2006
Los Angeles, I-405, near Westwood (upwind side), spring 2003 [D]	7.3	"
Jersey City, NJ, 2001- 2002	50	Yi et al., 2006
New Brunswick, NJ, 2001- 2002	8	"
Manhattan, NYC, 2nd story rooftop (unprotected), 2003-2005	27	Caravanos et al., 2006a
Chicago, 1993-95[E]	38-71	Yi et al 2001; Paode et al., 1998

A – Methods generally involved collection and analysis of samples of deposited particulate matter; details provided in references cited.
B - Rates for rest of off-shore transact from Santa Barbara to San Diego Bay ranged from 0.52 (Oxnard) – 3.3 (San Diego Bay) μg/m^3.
C - Average Pb-TSP range= 0.0056-0.017 μg/m^3 for urban sites; 0.0022 at Malibu site.
D - Average Pb-TSP at downwind site over sampling period ~0.02 μg/m^3.
E – This time period is the most recent for which such data are reported for this metropolitan area.

2.3.2.3 Soil

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

In forested areas away from old urban areas, the role of leaded gasoline in soil contamination is illustrated by the documented reductions in Pb in surface soils subsequent to the phase-out of leaded gasoline for on-road vehicles. For example, forest surface soil (litter) concentrations in a transect from Vermont through Maine and up to Gaspe in Quebec, which in 1979 exhibited a significant spatial trend ranging from 200 milligrams per kilogram, dry weight

(mg/kg dw) at its southernmost point down to 60 mg/kg dw at its northernmost and most remote point in Quebec, declined to 32-66 mg/kg dw (with no spatial trend) by 1996 (ISA, section 2.6.1; Evans, 2005). In forests from the mid-Atlantic to southern New England, a reduction in litter Pb concentration was observed between 1978 and 2004-05 (ISA, section 2.6.1; Johnson and Richter, 2010). In the latter study, the authors observed less change in concentration in the more northern sampling sites, which they attribute to reduced rates of organic matter decomposition in those colder temperature areas. Similarly, an additional New England study by Kaste et al (2006) also documented a pattern in temporal reductions in soil Pb (O-horizon) related to decomposition activity. These recent findings are generally consistent with findings reported in the last review which indicated the gradual migration of deposited Pb into mineral soils (e.g., Miller and Friedland, 1994; Kaste et al., 2003; Wang and Benoit, 1997; Johnson et al., 1995; Zhang, 2003).

Few studies have investigated temporal trends of surface soil Pb concentrations in more populated areas in relation to reductions in usage of leaded gasoline and paint. Current concentrations in areas of past heavy traffic powered by leaded gasoline are generally elevated above areas more distant from these areas (ISA, section 2.6.1). Current roadway-related sources of Pb (e.g., wear of vehicle parts), while substantially less significant than the historic use of leaded gasoline, may continue to provide some contribution to surface dust/soil in these areas (ISA, section 2.2.2.6). Surface soil and dust concentrations are much higher in such areas in large, older cities than surrounding suburban areas (ISA, section 2.6.1). In older residential areas, the presence of leaded house paint is another contributor to surface soil concentrations (ISA, sections 2.6.1; Yesilonis et al., 2008; Brown et al., 2008; Clark et al., 2006).

Areas of long-term Pb emissions from point sources appear to be the areas of highest surface soil Pb concentrations (ISA, sections 2.6 and 2.6.1). For example, Pb surface soil concentrations within approximately 100-250 meters of long-established Pb smelters have been five to ten times higher than those at 3-5 km distance (ISA, section 2.6.1; 2006 CD, Table 3-4). Surface soil concentrations of Pb near U.S. mines that are no longer active have also been found to be elevated above more distant areas (2006 CD, Table 3-6). Information described in the 2006 CD for areas surrounding smelters after implementation of pollution controls, although showing declines in Pb concentrations in outdoor dustfall, street dust and indoor dustfall, has not indicated a noticeable decline in soil Pb concentrations (2006 CD, pp. 3-23 to 3-24). The continued Pb emissions in such industrial areas likely influence the dynamics of Pb concentrations in the soil, affecting any response to emissions reductions. Estimates of associated steady-state surface soil Pb concentrations or the expected longer-term temporal pattern for this situation have not been made.

In summary, findings to date indicate that many of those systems less influenced by current point sources have and may still be responding to reduced Pb deposition rates associated

with reduced atmospheric emissions of Pb, including those associated with the phase-out of leaded gasoline for on-road vehicles, while potential responses of soils near point sources and those involving historically deposited Pb near roadways are less well characterized, but might be expected to have longer time horizons.

2.3.2.4 Biota

Terrestrial plants can take up Pb from soils into roots and then transport it to other plant tissues, as well as absorb deposited Pb into above-ground plant tissues (ISA, section 3.1.3.3; 2006 CD, section 2.3.7; 1986 CD, sections 6.5.3 and 7.2.2.2.1). The relative contributions of these pathways to the different plant tissues differ with the physiology of different plants, the chemical and physical characteristics of the soil, and the relative levels of Pb in soil and atmospheric deposition (ISA, section 3.1.3.3). Terrestrial animals may take in Pb through ingestion of vegetation, vegetation-consuming animals or soil, as well as directly from air. The relative contributions from these terrestrial animal exposure pathways vary with contamination level of the different items, as well as with species-specific factors, including intake rates.

The availability of data to document temporal trends in Pb concentrations in terrestrial biota is somewhat limited. Measurements of Pb in some biota indicate reductions in biologically available Pb over the past 30 years in some remote locations. For example, measurements of Pb in lichen from Golden Lake in Mount Ranier National Park (2005) and Emerald Lake basin in Sequoia/Kings Canyon National Parks (2004) indicate significant reductions (approximately 3-5-fold) since samples were previously collected in those locations in 1984 (ISA, section 2.6.6; Landers et al., 2008). Further, Pb in teeth of juvenile and adult moose in Isle Royale National Park in northern Michigan have also declined substantially (ISA, section 2.6.8; Vucetich et al., 2009).

Few recent data are available to characterize terrestrial biota Pb contamination levels associated with current air Pb deposition. In one example, however, Pb deposition associated with hauling mining materials has substantially increased Pb concentrations in moss within 10 meters of a road in the remote wilderness areas of Cape Krusenstern National Monument, Alaska.[31] In this example, the median Pb concentration measured within 10 meters of the haul road in summer 2001 was more than 20 times higher than at sites at greater distance from the road and nearly three orders of magnitude greater than those reported for this moss in other Alaska locations in 1990-1992 (ISA, section 2.3.1.2; Hasselbach et al., 2005).

The composition of human and wildlife diets (and associated contamination levels) influences the relative magnitude of dietary Pb intake, which may derive from currently existing

[31] 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.

or historic air sources in the U.S. or in countries that export food to the U.S. As noted in section 1.3.1.1 above, U.S. dietary Pb may also derive from nonair sources, such as through processing steps.

2.3.3 Aquatic Media

2.3.3.1 Surface Waters

In addition to delivery by atmospheric deposition directly to surface waters, Pb is also carried into surface waters via wastewater effluent from municipalities and industry, stormwater runoff, erosion, and accidental discharges (2006 CD, p. AX7-142; ISA section 2.3.2). As a result of the phasing out of leaded gasoline for use in on-road vehicles, reductions in Pb concentrations have been documented in surface waters of the North Atlantic Ocean, as well as the relatively less remote areas of the Great Lakes (2006 CD, p. 7-23). The availability of studies investigating historical trends in surface waters is limited, in part due to analytical issues that challenged many monitoring programs in the past (2006 CD, AX7.2.2.2). Thus, temporal trends reported in many aquatic systems are based on sediment analyses (see section 2.3.3.2 below).

Most Pb occurring in aquatic systems is associated with particles, with the distribution between particle-bound and dissolved form being influenced by water chemistry as well as suspended sediment levels (ISA, 2.3.2; 2006 CD, pp. AX7-117 to AX7-118, Section AX7.2.2). Water columns have been described as "transient reservoirs" for pollutants (2006 CD, p. 2-75). Once deposited to sediments, whether Pb is available for resuspension back into the water column with potential transport further down a watershed versus being buried into deeper sediments depends on the aquatic system (ISA, section 2.7.2). In open ocean waters (generally characterized by depth and distance from continental sources), resuspension to surface waters is unlikely. In more shallow systems, and those influenced by land sources (e.g., stormwater runoff as well as point sources), resuspension may play a role in water column concentrations. For example, studies in San Francisco Bay, the southern arm of which has an average depth of 2 m, have indicated that Pb particles may be remobilized from surface sediments into the water column (2006 CD, AX7-141).

The distribution of Pb dissolved in many U.S. surface waters has been reported by the United States Geological Survey (USGS) National Water-Quality Assessment (NAWQA) program. The NAWQA data set referenced in the ISA (ISA, section 2.6) encompasses data collected from 1991-2003 on Pb concentrations in flowing surface waters for more than 50 river basins and aquifers throughout the country (2006 CD, Section AX7.2.2.3). These data indicate a mean dissolved Pb concentration in U.S. surface waters of 0.66 μg/L (range 0.04 to 30 μg/L) in waters affected by a combined contribution of natural and anthropogenic sources, as compared to a mean of 0.52 μg/L (range 0.04 to 8.4 μg/L) for waters in "forest", "rangeland", and "reference"

sites (2006 CD, Section AX7.2.2.3). The highest surface water Pb concentrations were observed in sites impacted by land uses such as mining (2006 CD, p. AX7-131). The role of surface runoff in delivering contamination to waters near such land uses, as well as near metals industries, presents a challenge to the task of disentangling the relative contributions from atmospheric deposition as compared to those associated with surface runoff.

2.3.3.2 Sediments

Many studies have investigated temporal trends in sediment Pb concentrations, using sediment cores or surface sediment Pb concentration, with declines documented in many systems and usually attributed to the phasing out of leaded gasoline for on-road vehicles and industrial emissions reductions (ISA, sections 2.6.2 and 2.7.5; 2006 CD, section AX7.2.2). Several studies documenting the increased Pb deposition of the industrial age, including specifically leaded gasoline usage, and the subsequent declines associated with on-road leaded gasoline phase-out were reported in the 2006 CD. They include investigations involving sediment cores from the Okefenokee Swamp in Georgia as well as from 35 reservoirs and lakes in urban and reference locations (ISA, section 2.6.2; 2006 CD, p. AX7-141). In the latter, the median reduction in Pb mass accumulation rate in the cores, adjusted for background concentrations, was 246%, with the largest decreases in lakes located in dense urban watersheds (ISA, section 2.6.2; 2006 CD, p. AX7-141; Mahler et al, 2006). A third study of sediment cores in 12 lakes in the Great Lakes area also documented a peak in Pb concentrations consistent with peak use of leaded gasoline in the U.S. in the mid 1970s and declining concentrations in most lake sediments through the mid 1990s (2006 CD, p. 2-55; Yohn et al., 2004). Sediment surveys by the USGS NAWQA have reported the highest Pb concentrations in Idaho, Utah, and Colorado, with seven of the highest concentrations at sites classified as mining land use (2006 CD, p. AX7-133).

Among the more recent investigation of temporal trends in aquatic sediments described in the ISA is that associated with the Western Airborne Contaminants Assessment Project which documented Pb in sediment cores from 14 lakes in eight U.S. national parks in western states, including three in Alaska (ISA, section 2.6.2; Landers et al., 2010). Among the Alaska cores, in which concentrations were generally on the order of 20 μg/kg dw, there was little variation in Pb concentration, flux or enrichment factor. The other park cores, with few exceptions, generally exhibited an increase in concentration commencing in the mid-nineteenth century, which transitioned to declining trends in the past few decades, with lower concentrations in more recent, surface material. The highest concentration was recorded at core depth corresponding to

the mid-1970s, in one of the Sequoia/Kings Canyon lake cores (Figure 2-16; ISA, section 2.6.2; Landers et al., 2010[32]).

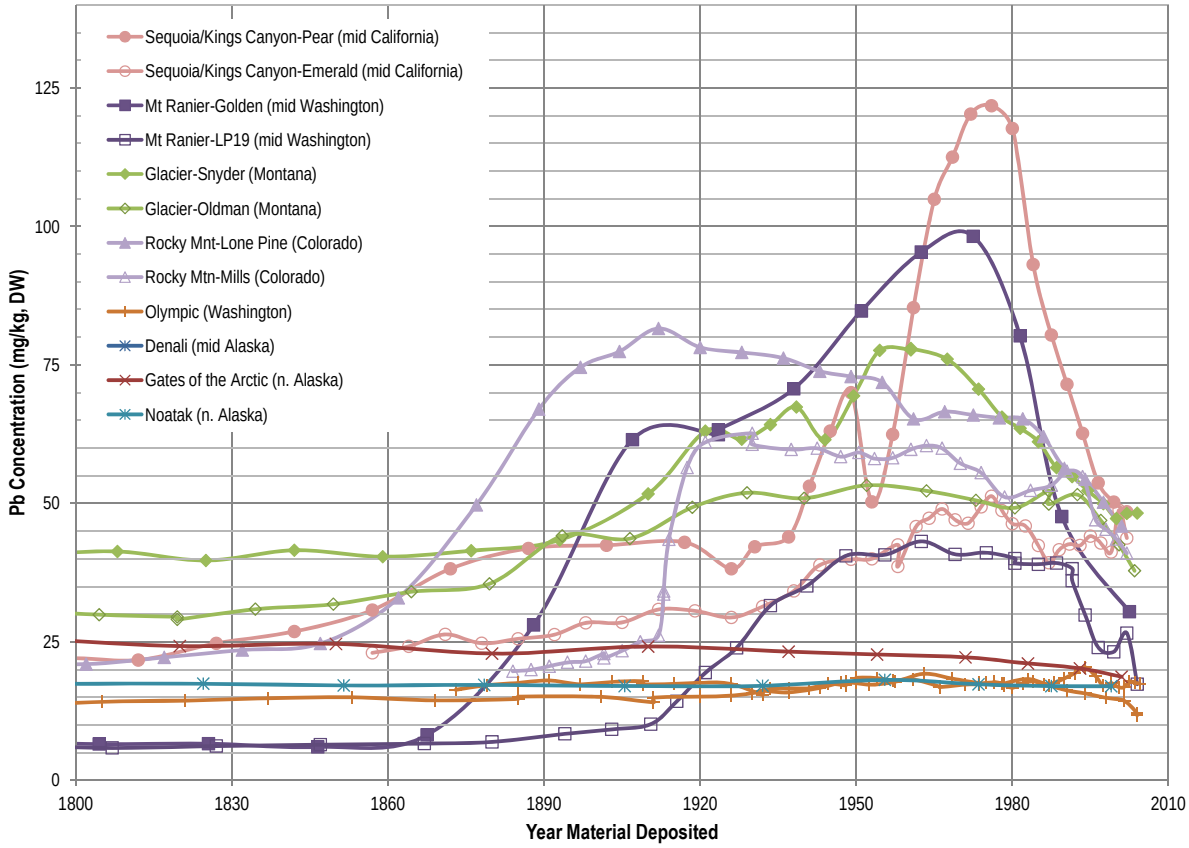

Figure 2-14. Temporal trend in sediment Pb concentration from core samples in twelve lakes at eight National Parks or Preserves.

Analyses of cores taken in several lakes and reservoirs along the Apalachicola, Chattahoochee, and Flint River Basin from north of the Atlanta, GA metropolitan area to the Gulf of Mexico indicate changes in sediment concentration both with time and in relation to influence of the large metropolitan area of Atlanta (ISA, section 2.6.2; Callender and Rice, 2000). Highest concentrations were documented just downstream from Atlanta at the core depth corresponding to the 1975-1980 time period; the corresponding concentration for the most recent time period (1990-1995) at that location was approximately 50% lower (ISA, section 2.6.2; Callender and Rice, 2000). These data may reflect changes in surface water discharges of Pb in the Atlanta area, during this time period, as well as changes in air deposition. The smaller

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

reduction (on the order of 20%) observed across the 20-year time period at the upstream site is likely more generally reflective of changes in air deposition.

2.3.3.3 Biota

Aquatic biota can contain Pb that may be derived from historic or current atmospheric deposition in the watershed, from historic or current direct water discharges or from natural sources (ISA, section 6.2.1). In near-shore systems and populated watersheds, all of these sources may play a role in observed Pb concentrations, complicating consideration of the role of ambient air Pb deposition. Additional challenges are presented by available data for mobile species such as fish (e.g., ISA, section 2.6.8; 2006 CD, section AX7.2.2.2). A 20-year record of Pb concentrations in blue and zebra mussels and oysters in U.S. coastal waters provides varying evidence on temporal trends that may reflect patterns in site-specific environmental releases as well as other ecosystem impacts on Pb fate and transport over that period (ISA, section 2.6.7; Kimbrough et al., 2008).

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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 *Integrated Science Assessment for Lead* (USEPA, 2013; 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. As discussed below, blood Pb has traditionally been used as a biomarker of Pb exposure and of internal dose, with relationships between air Pb concentrations and blood Pb concentrations informing consideration of the NAAQS for Pb. Information available in this review continues to support conclusions in the last review with regard to these relationships.

Lead associated with inhaled particles may, depending on particle size and Pb solubility, be absorbed into the systemic circulation or transported with particles to the gastrointestinal tract (ISA, section 3.2.1.1). 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 3.2.1.2). 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 3.2.2). Skeletal tissue serves as the largest

storage compartment, with much less Pb stored in soft tissues (e.g., kidney, liver, brain, etc.) (ISA, section 3.2.2.2).

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 3.2.2.2). In infants less than a year old, the bone is estimated to contain approximately 60% of the total body burden of Pb (ISA, section 3.2.2.2; Barry, 1975). Circulating Pb is taken up into the bone regions of active calcification. Accordingly, during early childhood there is rapid uptake of Pb into mineralizing bone, with somewhat more than 70% of total body burden Pb estimated to reside in bone of children aged 2 to 16, increasing up to more than 90% by adulthood (ISA, section 3.2.2.2; Barry, 1975). The net accumulation of Pb in bone over a person's lifetime results in bone lead concentrations generally increasing with age (ISA, section 3.2.2.2).

The distribution of Pb in the body is dynamic. Throughout life, Pb in the body is exchanged between blood and bone and between blood and soft tissues (ISA, sections 3.3.5 and 3.2.2; 2006 CD, section 4.3.2). The rates of these exchanges vary with age, exposure and various physiological variables. For example, resorption of bone, which results in the mobilization of Pb from bone into the blood, is a somewhat rapid and ongoing process during childhood and a more gradual process in later adulthood (ISA, sections 3.2.2.2, 3.3.5 and 3.7.2). Resorption rate is appreciably increased in pregnant or nursing women and in association with osteoporosis in postmenopausal women or, to a lesser magnitude, in older men (ISA, sections 3.3.5.2). Changes in Pb exposure circumstances can also influence these exchanges, e.g., when exposure levels are substantially reduced, the relative contribution of Pb from bone to blood Pb concentration increases (ISA, section 3.3.5). The studies that address the relative contributions of bone Pb and current Pb exposure to blood Pb are limited to a few human studies during the 1980s and early 1990s, when leaded gasoline usage was common, and a study in non-human primates. These studies indicate an appreciable contribution from bone Pb stores to Pb in blood, on the order of 40-70% of blood Pb contributed from bone, under circumstances with higher concurrent exposure than exposures common today (ISA, section 3.3.5; Smith et al., 1996; Gulson et al., 1995; Manton, 1985; Franklin et al., 1997).

During bone resorption that occurs in pregnancy, Pb is released from bone, increasing the bone contribution to maternal blood Pb levels, and maternal Pb circulates across the placenta, posing risk to the developing fetus and providing the fetal body burden (ISA, sections 3.2.2.4, 3.3.5, 3.7.2 and 3.7.3; 2006 CD, 6.6.2; Chuang et al., 2001). The relative size of contributions of maternal bone Pb and current maternal exposure to maternal blood Pb and fetal body burden are influenced by the relative magnitude of current and historical exposures. In various study populations with mean maternal blood Pb levels ranging from 1.7 to 8.6 μg/dL, average blood Pb

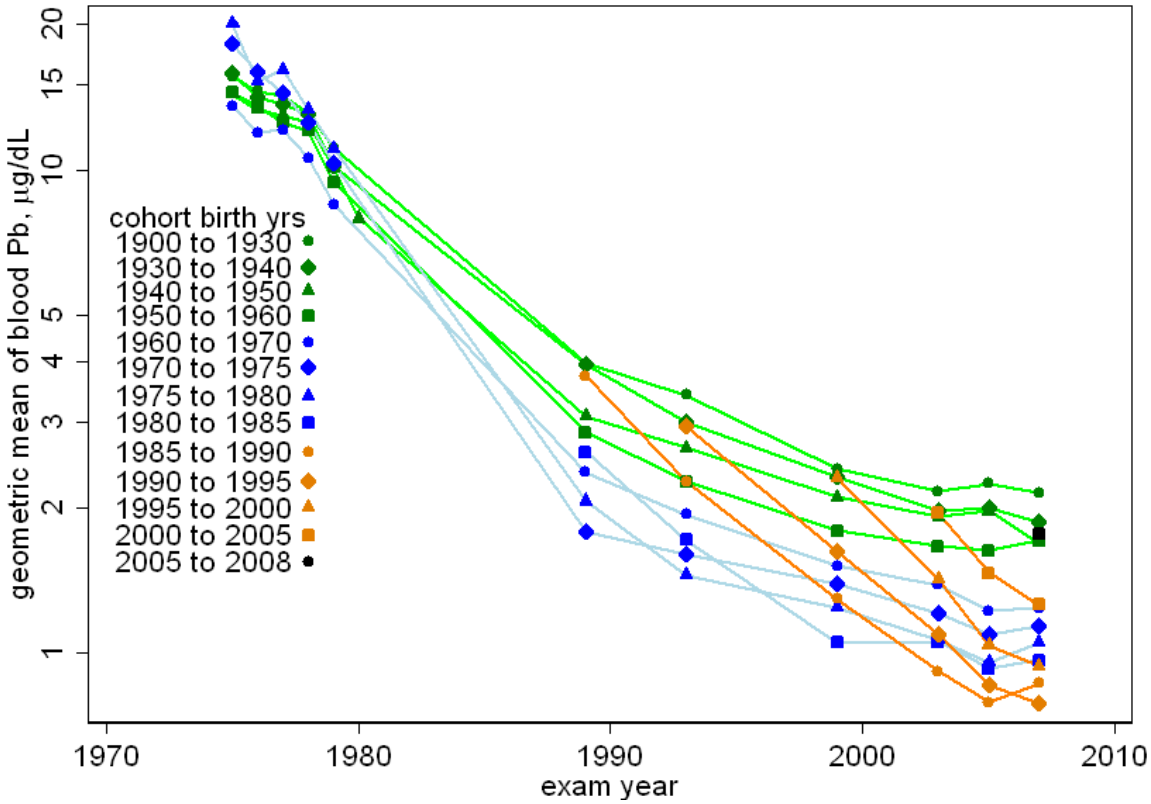
concentration in the umbilical cord (as representative of newborn blood) has been reported to range from 70% to 100% of average maternal blood Pb (ISA, section 3.2.2.4); the range is similar for four study populations with mean maternal blood Pb level below 4 μg/dL (Amaral et al., 2010; Patel and Prabhu, 2009; Lagerkvist et al., 1996; Jedrychowski et al., 2011). The relationship between cord blood Pb and maternal blood Pb concentrations is more variable for individual mother-child pairs. In a low-income population with mean maternal blood Pb of 1.9 μg/dL, factors associated with occurrences of relatively higher cord-to-maternal blood Pb ratios included higher maternal blood pressure and alcohol consumption, while factors associated with relatively lower cord-to-maternal blood Pb ratios included higher maternal hemoglobin and sickle cell trait presence (Harville et al., 2005). As a result of the contributions of maternal bone Pb to maternal blood Pb during pregnancy, the contribution to fetal body burden from maternal bone Pb relative to maternal concurrent exposure can be appreciable. This maternal bone Pb contribution to fetal body burden is likely to vary, however, in response to differences between maternal historical exposures and those during pregnancy, among other factors (2006 CD, sections 4.3.1.5 and 4.3.2.5; ISA, section 5.1; Chuang et al., 2001; Gulson et al., 1999; Gulson et al., 2003; Gulson et al., 2004a; Rothenberg et al., 2000).

Limited data indicate that blood Pb levels in some newborns may decline during the period extending through the first few months of life; the extent to which this may occur may be influenced by the magnitude of blood Pb level at birth and of subsequent exposure during early infancy (ISA, section 3.4.1; Carbone et al., 1998; Simon et al., 2007; Gulson et al., 1999, 2001). As infants become more mobile and engage in hand-to-mouth behavior, blood Pb levels then commonly increase to a peak around one to two years of age (ISA, sections 3.4.1 and 5.2.1.1).

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

As discussed in past CDs and in the ISA, blood Pb is the most commonly used indicator of human Pb exposure. Given the association with exposure and the relative ease of collection, blood Pb levels are extensively used as an index or biomarker of exposure by national and international health agencies, as well as in epidemiological and toxicological studies of Pb health effects and dose-response relationships (ISA, sections 3.3.2, 3.4.1, 4.3, 4.4, 4.5, 4.6, 4.7, and 4.8). While bone Pb measurements are also used in epidemiological studies as an indicator of cumulative Pb exposure, blood Pb measurements remain the predominant, well-established and well-characterized exposure approach.

Since 1976, the U.S. Centers for Disease Control and Prevention (CDC) has been monitoring blood Pb levels nationally through the National Health and Nutrition Examination Survey (NHANES). This survey has documented the dramatic decline in mean blood Pb levels in all ages of the U.S. population that has occurred since the 1970s, as shown in Figure 3-1, and that coincides with actions on leaded fuels, leaded paint, lead in food packaging, and lead-containing plumbing materials that have reduced Pb exposure in the U.S. (ISA, section 3.4.1; Pirkle et al., 1994; Schwemberger et al., 2005). This decline has continued over the more recent past. For example, the 2009-2010 geometric mean blood Pb level in U.S. children aged 1-5 years is 1.17 µg/dL, as compared to 1.51 µg/dL in 2007-2008 (ISA, section 3.4.1) and 1.8 µg/dL in 2003-2004, the most recent data available at the time of the last review (73 FR 67002). Somewhat less dramatic declines have been reported in the upper tails of the distribution and in different groups with higher blood Pb levels than the general child population (ISA, Figures 3-17 and 3-19).



Source: Adapted from data from the NHANES (NCHS, 2010)
Note: The means of log blood Pb were weighted to represent national averages. Data were from the publically available NHANES II, NHANES III (1988-1991 and 1992-1994), and the continuous NHANES (1999-2000, 2003-2004, 2005-2006, 2007-2008). Continuous NHANES data from 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 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 the continuous NHANES.

Figure 3-1. Temporal trend in mean blood Pb levels for NHANES cohorts (ISA, Figure 3-18).

The CDC, and its predecessor agencies, have for many years used blood Pb level as a metric for identifying highly exposed children at risk of adverse health effects for whom recommendations might be made for their protection (e.g., CDC, 1991; CDC, 2005). In 1978, when the Pb NAAQS was initially established, the CDC recognized a blood Pb level of 30 μg/dL as a level warranting individual intervention (CDC, 1991). In 1985, the CDC recognized a level of 25 μg/dL for intervention, and in 1991, they adopted a multi-tier approach that recommended individual intervention actions at a level of 15 μg/dL and implementation of more general community-wide prevention activities if many children in a community have blood Pb at or above 10 μg/dL (CDC, 1991; CDC, 2005). In 2005, CDC revised their statement on *Preventing Lead Poisoning in Young Children*, specifically recognizing the evidence of adverse health effects in children with blood Pb levels below 10 μg/dL and emphasizing the importance of preventive measures (CDC, 2005).[1] Consistent with this statement, the CDC revised their approach in 2012 to one that relies on the 97.5th percentile of blood Pb concentrations in U.S. children aged one to five years of age (currently 5 μg/dL)[2] as a reference level for identifying young children for whom they recommend particular follow-up actions (CDC, 2012).

Unless influenced by a recent elevation in exposure, blood Pb measurements are a reflection of total body burden. Accordingly, Figure 3-1 illustrates the reduction in Pb body burden in the U.S. population over the past 40 years. Associations of health effects with blood Pb measurements indicate relationships with body burden and may, in some cases, indicate relationships with recent exposures. The extent to which blood Pb measurements represent current or recent exposure circumstances, however, may be uncertain, particularly where histories include exposures largely different from those occurring more recently (ISA, sections 3.3 and 3.3.2). This uncertainty may be greater for blood Pb measurements taken in older children[3] and adults than those for young children as a result of their longer exposure histories. For example, the Pb in bone of adults may have accumulated over decades of Pb exposure (with past exposures often greater than current ones for current U.S. populations) such that the bone may be a significant source of Pb in blood in later years of life, after exposure has ended or been appreciably reduced. Thus, in adult and older child populations with past exposures that were

[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 μg/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).

[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 3.3).

appreciably elevated in comparison to more recent exposures, current blood Pb levels may predominantly reflect their exposure history rather than their current exposures (ISA, sections 3.3.5, 3.3.5.2 and 3.4.1). Accordingly, the extent to which studies using cross-sectional blood Pb measurements as the exposure biomarker inform our understanding of relationships between recent Pb exposures and various health effects can differ across age groups and is greatest for very young children, as discussed in section 3.3 below.

The relationship of children's blood Pb to recent exposure may reflect their labile bone pool, with their rapid bone turnover in response to rapid childhood growth rates (ISA, section 3.3.5). The relatively smaller skeletal compartment of Pb in children (particularly very young children) compared to adults is subject to more rapid turnover. As a result the blood Pb concentration of children can more quickly reflect changes in their total body burden (associated with their shorter exposure history) and can also reflect changes in recent exposures (ISA, section 3.3.5).

Multiple studies of the relationship between Pb exposure and blood Pb in children (e.g., Lanphear and Roghmann 1997; Lanphear et al., 1998) have shown young children's blood Pb levels to reflect Pb exposures, including exposures to Pb in surface dust. These and studies of child populations near sources of air Pb emissions, such as metal smelters, further demonstrate the effect of airborne Pb on interior dust and on blood Pb (ISA, sections 3.4.1, 3.5.1 and 3.5.3; Hilts, 2003; Gulson et al., 2004b). Accordingly, blood Pb level was the index of exposure for children in the risk assessment performed in the last Pb NAAQS review (described in section 3.4 below).

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

Alternative biomarkers of Pb exposure include bone Pb, teeth Pb, and hair Pb (ISA, section 3.3.1, 3.3.3 and 3.3.4). Given the role of bone as the repository of more than 90% of an adult body's Pb burden, bone Pb levels are recognized as indicators of body burden and cumulative lifetime exposure (ISA, sections 3.2.2.2, 3.3 and 3.3.5). The currently available evidence does not, however, indicate these or any other alternatives to be superior to blood Pb in young children or as commonly used for the purposes of tracking recent Pb exposures and for assessing potential health risk for this age group (ISA, section 3.7.3). In summary, the current evidence continues to support our conclusions from the last review regarding the use of blood Pb levels as an internal biomarker of Pb exposure and dose informative to characterize Pb health effects in young children.

- **To what extent has new information altered scientific conclusions regarding the relationships between Pb in ambient air and Pb in children's blood?**

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

Lead in ambient air contributes to Pb in blood by multiple exposure pathways by both inhalation and ingestion exposure routes (ISA, section 3.1.1). The quantitative relationship between ambient air Pb and blood Pb, which is often termed a slope or ratio, describes the increase in blood Pb (in µg/dL) estimated to be associated with each unit increase of air Pb (in $\mu g/m^3$). Ratios are presented in the form of 1:x, with the 1 representing air Pb (in $\mu g/m^3$) and x representing blood Pb (in µg/dL). Description of ratios as higher or lower refers to the values for x (i.e., the change in blood Pb per unit of air Pb). Slopes are presented as simply the value of x.

At the conclusion of the last review in 2008, the EPA interpreted the evidence as providing support for use (in informing the Administrator's decision on standard level) of a range "inclusive at the upper end of estimates on the order of 1:10 and at the lower end on the order of 1:5" (73 FR 67002). This conclusion reflected consideration of the air-to-blood ratios presented in the 1986 CD[4] and associated observations regarding factors contributing to variation in such ratios, ratios reported subsequently and ratios estimated based on modeling performed in the REA, as well as advice from CASAC (73 FR 66973-66975, 67001-67002). The information

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

available in this review, which is assessed in the ISA and largely, although not completely, comprises studies that were available in the last review, does not alter the primary scientific conclusions drawn in the last review regarding the relationships between Pb in ambient air and Pb in children's blood. The ratios summarized in the ISA in this review span a range generally consistent with the range concluded in 2008 (ISA, section 3.5.1).

The air-to-blood ratios for children, or for populations inclusive of children, discussed in the ISA for this review are summarized in Table 3-1. The evidence pertaining to this quantitative relationship between air Pb and children's blood Pb is now, as in the past, limited by the circumstances in which the data are collected. These estimates are generally developed from studies of populations in a variety of Pb exposure circumstances. Accordingly, there is significant variability in air-to-blood ratios among the different study populations exposed to Pb through different air-related exposure pathways and at different exposure levels. This variability in air-to-blood estimates can relate to the representation of air-related pathways and study populations, including, for example, relatively narrow age ranges for the population in order to reduce age-related variability in blood Pb, or including populations with narrowly specified dietary sources. It can relate to the study population exposure and blood Pb levels (ISA, section 3.7.4). It can also relate to the precision of air and blood measurements and of the study circumstances, such as with regard to spatial and temporal aspects. Additionally, in situations where exposure to nonair sources covaries with air-related exposures that are not accounted for in deriving ratio estimates, uncertainties may relate to the potential for confounding by nonair exposure covariance (ISA, section 3.5). Most studies have reported the relationship as either log-log or linear (Table 3-1; ISA, section 3.7.4).

As was noted in the last review, age is an important influence on the magnitude of air-to-blood ratio estimates derived. Ratios for children are generally higher than those for adults, and higher for young children than older children, perhaps due to behavioral differences between the age groups, as well as their shorter exposure history. Similarly, given the common pattern of higher blood Pb levels in pre-school aged children than during the rest of childhood, related to behaviors that increase environmental exposures (e.g., hand-to-mouth activity), ratios would be expected to be highest in earlier childhood. Additionally, estimates of air-to-blood ratios that include air-related ingestion pathways in addition to the inhalation pathway are "necessarily higher," in terms of blood Pb response, than those estimates based on inhalation alone (USEPA 1986, p. 11-106). Thus, the extent to which studies account for the full set of air-related inhalation and ingestion exposure pathways affects the magnitude of the resultant air-to-blood estimates, such that including fewer pathways as "air-related" yields lower ratios. Estimates of air-to-blood ratios can also be influenced by population characteristics that may influence blood

Pb; accordingly, some analyses include adjustments. Most of the studies in Table 3-1 include ratios derived from linear analyses, while a subset are derived from nonlinear models.

Given the recognition of young children as a key at-risk population in section 3.3 below, as well as the influence of age on blood Pb levels, the studies presented in Table 3-1 are grouped with regard to the extent of their inclusion of children younger than or barely school age (less than or equal to five years of age). Among the first group of studies, focused exclusively on young children, only one study dates from the end of or after the phase-out of leaded gasoline usage (namely, Hilts, 2003). This study reports changes in children's blood Pb levels associated with reduced Pb emissions and associated air concentrations near a Pb smelter in Canada (for children through age five). Given the timing of this study, after the leaded gasoline phase-out, and its setting near a smelter, the ambient air Pb in this study may be somewhat more comparable to that near sources in the U.S. today than other studies discussed herein. The study authors report an air-to-blood ratio of 1:6.[5] An EPA analysis of the air and blood data reported for 1996, 1999 and 2001 results in a ratio of 1:6.5, and the analysis focused only on the 1996 and 1999 data (pre- and post- the new technology) yields a ratio of 1:7 (ISA, section 3.5.1; Hilts, 2003).[6] The two other studies that focused on children of age 5 or younger analyzed variations in air Pb as a result of variations in leaded gasoline usage in Chicago, Illinois. The study by Hayes et al. (1994) compared patterns of ambient air Pb reductions and blood Pb reductions for large numbers of children, aged 6 months to 5 years, in Chicago between 1974 and 1988, a period when significant reductions occurred in both measures. The study reported a better fit for the log-log model which describes a pattern of higher ratios with lower ambient air Pb and blood Pb levels (Hayes et al., 1994). Based on the log-log model, the ratio derived for the relationship of quarterly mean air concentration with blood Pb during the period is 1:8 (Table 3-1). Another analysis for a Chicago dataset, performed by Schwartz and Pitcher (1989) focused on the

[5] Sources of uncertainty include the role of factors other than ambient air Pb reduction in influencing decreases in blood Pb (ISA, section 3.5.1). 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 the author 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 (Hilts, 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 earlier years of the study (Hilts, 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 3.5.1).

association of blood Pb in black children (aged $\leq$ 5 years) with the use of leaded gasoline from 1976 through 1980. Given that leaded gasoline exposure occurs by air-related pathways, additional analyses have related the leaded gasoline usage for this period to air concentrations. The resulting relationship of blood Pb with air Pb (adjusted for age and a number of other covariates) yields a ratio of 1:8.6 (ISA, table 3-12, section 3.5.1). The blood Pb concentrations in the two leaded gasoline studies are appreciably higher (a factor of two or more) than those in the study near the smelter (Hilts, 2003).

The second group of studies in Table 3-1 (comprising studies including but not limited to children less than or equal to five years of age) includes a complex statistical analysis and associated dataset for a cohort of children born in Mexico City from 1987 through 1992 (Schnaas et al., 2004). This study, which was not assessed in the last review, encompasses the period of leaded gasoline usage and further informs our understanding of factors influencing the quantitative relationship between air Pb and children's blood Pb. Air-to-blood ratios developed from this study are influenced by a number of factors and appear to range from roughly 2 to 6, in addition to an estimate of 9 (ISA, section 3.5.1), although the latter is derived from a data set restricted to the latter years of the study when little change in air Pb concentration occurred, such that the role of air Pb may be more uncertain. Estimates associated with the developmental period of highest exposure (e.g., age 2 years) range up to approximately 6, illustrating the influence of age on the ratio (ISA, section 3.5.1). Also in the second group in Table 3-1 are two much older studies of populations with age ranges extending well beyond 6 years. The first is the review and meta-analysis by Brunekreef (1984) using datasets available at the time for variously aged children as old as 18 years with identified air monitoring methods and reliable blood Pb data for 18 locations in the U.S. and internationally.[7] The author discusses potential confounders of the relationship between air Pb and blood Pb, recognizing the desirability of taking them into account when deriving an air-to-blood relationship from a community study but noting that was not feasible in such an analysis. Two models were produced, one based on the full pooled dataset and a second limited to blood Pb-air Pb data pairs with blood Pb levels below 20 μg/dL (r^2 values were 0.692 and 0.331, respectively). From these two $\log_n$-$\log_n$ models, air-to-blood ratio estimates were derived for air concentrations corresponding to the geometric means of the two sets of data pairs. At those concentrations (1.5 and 0.54 $\mu g/m^3$, respectively), the resultant ratios both round to 5. The study by Schwartz and Pitcher (1989) described above also analyzed the relationship between U.S. NHANES II blood Pb levels for white subjects, aged

[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 (1984) used multiple regression to control for several confounders (73 FR 66974).

≤74 years, and national usage of leaded gasoline. A separate, less specifically described, air Pb dataset was used to convert the relationship of blood Pb with gas Pb to one for blood Pb with air Pb, with a resultant ratio on the order of 9, adjusted for age and other covariates (Henderson, 2007a, pp. D-2 to D-3; ISA, Table 3-12).

The last two studies included in Table 3-1 are focused on older children (ages 6-11). The methods for characterization of air Pb concentrations (and soil Pb for Ranft et al., 2008) also differ from other studies in Table 3-1. The first study regressed average blood Pb concentrations for multiple locations around Mumbai, India on average air Pb concentrations at those locations (Tripathi et al., 2001). The values in the linear regression were 13 pairs of location-specific geometric means of all the data collected over the 13-year period from 1984 to 1996; the reported slope was 3.6 (Tripathi et al., 2001). The location-specific geometric mean blood Pb levels in this study (8.6-14.4 μg/dL) indicate blood Pb distributions in this age group much higher than those pertinent to similarly aged children in the U.S. today. The second study analyzed air, soil and children's blood Pb concentrations in Duisburg, Germany, during the leaded gasoline phase-out (Ranft et al., 2008). Average blood Pb levels declined over the nearly 20-year study period from 9 μg/dL in 1983 (345 children average age of 9 years) to 3 μg/dL in 2000 (162 children average of 6 years).[8] Average air Pb concentration declined from 0.45 $\mu g/m^3$ to 0.06 $\mu g/m^3$ over the same period, with the largest reduction occurring between the first study year (derived from two monitoring sites for full study area) and the second study year, 1991, for which air concentrations were derived from a combination of dispersion modeling and the two monitoring sites.[9] For a mean air Pb concentration of 0.1 $\mu g/m^3$, the study's multivariate log-linear regression model predicted air-to-blood ratios of 3.2 and 6.4 for "background" blood Pb concentrations of 1.5 and 3 μg/dL, respectively. In this study, background referred to Pb in blood from other sources; the blood Pb distribution over the study period, including levels when air Pb concentrations are lowest, indicates 3 μg/dL may be the better estimate of background for this study population. Inclusion of soil Pb as a variable in the model may have contributed to an underestimation of the these blood Pb-air Pb ratios for this study because some of the Pb in soil likely originated in air and the blood Pb-air Pb slope does not include the portion of the soil/dust Pb ingestion pathway that derives from air Pb. Using univariate linear, log-log and log-linear models on the median air and blood Pb concentrations reported for the five years included in this study, the ISA also derived air-to-blood ratio estimates ranging from 9 to 17 (ISA, p. 3-126;

[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).

Ranft et al., 2008, Table 2). Uncertainties related to this study's estimates include those related to the bulk of air concentration reduction occurring between the first two time points (1983 and 1991) and the difference among the year's air datasets (e.g., two data sources [air monitors] in 1983 and multiple geographical points from a combination of the monitors and modeling in subsequent years).

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)	$\log_n$-$\log_n$ regression: quarterly median PbB and quarterly mean PbA [unadjusted] PbB: 10-28 µg/dL (quarterly median) 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)	linear 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)	linear 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 (1984)	Meta-analyses ($\log_n$-$\log_n$ 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 Average 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)	Linear , $\log_n$-$\log_n$ 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)	Linear 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)	Linear 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); average: ~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)	Linear, $\log_n$-$\log_n$, log-linear 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

A - Predicted change in blood Pb (µg/dL per µg/m3) over range ± 0.01 µg/m^3 from study's central air Pb, which is provided in µg/m^3 in parentheses. For linear models, this is simply the air Pb coefficient.
B - ln(PbB) = ln(PbA) x 0.24 +3.17 (ISA, Table 3-12; Hayes et al., 1994)
C - See discussion in text and ISA, section 3.5.1.
D - Based on data for U.S. (1986 CD). See ISA, section 3.5.1. Log-lin = log-linear model.
E - ln(PbB) = ln(PbA) x 0.3485 +2.853
F - ln(PbB) = ln(PbA) x 0.2159 + 2.620
G -Derived from regressions with separate soil Pb variable, contributing to underestimation of contribution from air Pb (see text and ISA, section 3.5.1).
GM, geometric mean; AM, arithmetic mean; GSD, geometric standard deviation; PbB, blood Pb concentration, µg/dL; PbA, air Pb concentration, µg/m^3

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

The evidence on the quantitative relationship between air Pb and air-related Pb in blood is now, as in the past, limited by the circumstances (such as those related to Pb exposure) in which the data were collected. Previous reviews have recognized the significant variability in air-to-blood ratios for different populations exposed to Pb through different air-related exposure pathways and at different air and blood levels, with the 1986 CD noting that ratios derived from studies involving the higher blood and air Pb levels pertaining to occupationally exposed workers are generally smaller than ratios from studies involving lower blood and air Pb levels (ISA, p. 3-132; 1986 CD, p. 11–99). Consistent with this observation, slopes in the range of 3 to

[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 2007 REA, 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.

5 were estimated for child population datasets assessed in the 1986 CD (ISA, p. 3-132; 1986 CD p. 11–100; Brunekreef, 1984; Tripathi et al., 2004). Additional studies considered in the last review and those assessed in the ISA provide evidence of ratios above this older range (ISA, p. 3-133). For example a ratio of 6.5-7 is indicated by the study by Hilts (2003), one of the few studies that evaluate the air Pb-blood Pb relationship in conditions that are closer to the current state in the U.S. (ISA, p. 3-132). We additionally note the variety of factors identified in the ISA that may potentially affect estimates of various ratios (including potentially coincident reductions in nonair Pb sources during the course of the studies), and for which a lack of complete information may preclude any adjustment of estimates to account for their role (ISA, section 3.5).

In summary, as at the time of the last review of the NAAQS for Pb, the currently available evidence includes estimates of air-to-blood ratios, both empirically- and model-derived, with associated limitations and related uncertainties. These limitations and uncertainties, which are summarized here and also noted in the ISA, usually include uncertainty associated with reductions in other Pb sources during the study period. The limited amount of new information available in this review has not appreciably altered the scientific conclusions reached in the last review regarding relationships between Pb in ambient air and Pb in children's blood or with regard to the range of ratios. The currently available evidence continues to indicate ratios relevant to the population of young children in the U.S. today, reflecting multiple air-related pathways in addition to inhalation, to be generally consistent with the approximate range of 1:5 to 1:10 given particular attention in the 2008 NAAQS decision, including the "generally central estimate" of 1:7 (73 FR 67002, 67004; ISA, pp. 3-132 to 3-133).

3.2 NATURE OF EFFECTS

Lead has been demonstrated to exert a broad array of deleterious effects on multiple organ systems as described in the assessment of the evidence available in this review and consistent with conclusions of past CDs (ISA, section 1.6; 2006 CD, section 8.4.1). A sizeable number of studies on Pb health 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 broad array of effects recognized for Pb in the last review (see ISA, section 1.10).[11] Consistent with those conclusions, in the context of pollutant exposures considered

[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).

relevant to the Pb NAAQS review,[12] the ISA determines that causal relationships[13] exist for Pbwith effects on the nervous system in children (cognitive function decrements and the group of externalizing behaviors comprising attention, impulsivity and hyperactivity), the hematological system (altered heme synthesis and decreased red blood cell survival and function), and the cardiovascular system (hypertension and coronary heart disease), and on reproduction and development (postnatal development and male reproductive function) (ISA, table 1-2). Additionally, the ISA describes relationships between Pb and effects on the nervous system in adults, on immune system function and with cancer[14] as likely to be causal[15] (ISA, table 1-2, sections 1.6.4 and 1.6.7).

In some categories of health effects, there is newly available evidence regarding some aspects of the effects described in the last review or that strengthens our conclusions regarding aspects of Pb toxicity on a particular physiological system. Among the nervous system effects of Pb, the newly available evidence is consistent with conclusions in the previous review which recognized that "[t]he neurotoxic effects of Pb exposure are among those most studied and most extensively documented among human population groups" (2006 CD, p. 8-25). Nervous system effects that receive prominence in the current review, as in previous reviews, include those

[12] With regard to consideration of pollutant exposures for studies included in the ISA, the ISA states the following (ISA, pp. lx-lxi).

> *In drawing judgments regarding causality for the criteria air pollutants, the ISA focuses on evidence of effects in the range of relevant pollutant exposures or doses, and not on determination of causality at any dose. Emphasis is placed on evidence of effects at doses (e.g., blood Pb concentration) or exposures (e.g., air concentrations) that are relevant to, or somewhat above, those currently experienced by the population. The extent to which studies of higher concentrations are considered varies by pollutant and major outcome category, but generally includes those with doses or exposures in the range of one to two orders of magnitude above current or ambient conditions. Studies that use higher doses or exposures may also be considered to the extent that they provide useful information to inform understanding of mode of action, interspecies differences, or factors that may increase risk of effects for a population. Thus, a causality determination is based on weight of evidence evaluation for health, ecological or welfare effects, focusing on the evidence from exposures or doses generally ranging from current levels to one or two orders of magnitude above current levels.*

[13] In determining there to be a causal relationship 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. lxii)

[14] 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 4.10). In this assessment, EPA concludes that a causal relationship is likely to exist between Pb exposure and cancer, based primarily on consistent, strong evidence from experimental animal studies, but inconsistent epidemiological evidence (ISA, section 4.10.5).

[15] In determining that there is likely to be a causal relationship 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. lxii).

affecting cognitive function and behavior in children (ISA, section 4.3), with conclusions that are consistent with findings of the last review.

Across the broad array of Pb effects for systems and processes other than the nervous system, the evidence base has been augmented with additional epidemiological investigations in a number of areas, including developmental outcomes, such as puberty onset, and adult outcomes related to cardiovascular function, for which several large cohorts have been analyzed (ISA, Table 1-8 and sections 4.4 and 4.8). Conclusions on these other systems and processes are consistent with conclusions reached in the last review, while also extending our conclusions on some aspects of these effects. For example, evidence in this review for the cardiovascular system includes information on the role of interactions of cumulative Pb exposure with other factors, such as stress in contributing to hypertension, and on a role for Pb in contributing to coronary heart disease (ISA, section 4.4 and Table 1-8).

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

> *Multiple epidemiologic studies conducted in diverse populations of children consistently demonstrate the harmful effects of Pb exposure on cognitive function (as measured by IQ decrements, decreased academic performance and poorer performance on tests of executive function).… Evidence suggests that some Pb-related cognitive effects may be irreversible and that the neurodevelopmental effects of Pb exposure may persist into adulthood (Section 1.9.4). Epidemiologic studies also demonstrate that Pb exposure is associated with decreased attention, and increased impulsivity and hyperactivity in children (externalizing behaviors). This is supported by findings in animal studies demonstrating both analogous effects and biological plausibility at relevant exposure levels. Pb exposure can also exert harmful effects on blood cells and blood producing organs, and is likely to cause an increased risk of symptoms of depression and anxiety and withdrawn behavior (internalizing behaviors),decreases in auditory and motor function, asthma and allergy, as well as conduct disorders in children and young adults. There is some uncertainty about the Pb exposures contributing to the effects and blood Pb levels observed in epidemiologic studies; however, these uncertainties are greater in studies of older children and adults than in studies of young children (Section 1.9.5).*

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

> *A large body of evidence from both epidemiologic studies of adults and experimental studies in animals demonstrates the effect of long-term Pb exposure on increased blood pressure (BP) and hypertension (Section 1.6.2). In addition to its effect on BP, Pb exposure can also lead to coronary heart disease and death*

from cardiovascular causes and is associated with cognitive function decrements, symptoms of depression and anxiety, and immune effects in adult humans. The extent to which the effects of Pb on the cardiovascular system are reversible is not well-characterized. Additionally, the frequency, timing, level, and duration of Pb exposure causing the effects observed in adults has not been pinpointed, and higher past exposures may contribute to the development of health effects measured later in life.

As in prior reviews of the Pb NAAQS, this review is focused on those effects most pertinent to ambient air Pb exposures. Given the reductions in ambient air Pb levels over the past decades, these effects are generally those associated with the lowest levels of Pb exposure that have been evaluated. Additionally, we recognize the limitations on our ability to draw conclusions regarding the exposure conditions contributing to the findings from epidemiological analyses of blood Pb levels in populations of older children and adults, particularly in light of their history of higher Pb exposures. In the last review, while recognizing the range of health effects in variously aged populations related to Pb exposure, we focused on the health effects for which the evidence was strongest with regard to relationships with the lowest exposure levels, neurocognitive effects in young children. The policy-relevant questions on health effects for this review (identified in the IRP) were framed in recognition of the conclusions of the last review. Our consideration of the health effects evidence in this review is framed by policy-relevant questions building on those identified in the IRP.

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

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

The specific exposure circumstances, including timing during the lifetime, that contribute to the blood Pb (or bone Pb) measurements with which associations have been analyzed in epidemiological studies are unknown. This is particularly the case with regard to the contributing role of recent exposures for which uncertainty is greater in adults and older children than in younger children (ISA, sections 1.9.4). For example, a critical aspect of much of the epidemiological evidence, particularly that focused on older adults in the U.S. today, is the backdrop of generally declining environmental Pb exposure (from higher exposures during their younger years) that is common across many study populations (ISA, p. 4-2). An additional factor complicating the interpretation of health effect associations with blood Pb measurements in older children and younger adults is the common behaviors of younger children (e.g., hand-to-mouth contact) which generally contribute to relatively greater environmental exposures earlier in life (ISA, sections 3.1.1, 4.2.1). Such exposure histories complicate our ability to draw conclusions regarding critical time periods and lifestages for Pb exposures eliciting the effects with which associations with Pb biomarkers have been observed in populations of adults and older children (e.g., ISA, section 1.9.6).[16]

As at the time of the last review, assessment of the full evidence base, including evidence newly available in this review, demonstrates that Pb exposure prenatally and also in early childhood can contribute to neurocognitive impacts in childhood, with evidence also indicating the potential for effects persisting into adulthood (ISA, sections 1.9.5, 1.9.6, and 1.10). In addition to the observed associations of prenatal and childhood blood Pb with effects at various ages in childhood, there is also evidence of lead-related cognitive function effects in non-occupationally exposed adults (ISA, section 4.3.11). This includes evidence of associations of such effects in adulthood with childhood blood Pb levels and, in other cohorts, with concurrent (adult) blood Pb levels (ISA, sections 4.3.2.1, 4.3.2.7 and 4.3.11). As the studies finding associations of adult effects with childhood blood Pb levels did not examine adult blood Pb levels, the relative influence of adult Pb exposure cannot be ascertained, and a corresponding lack of early life exposure or biomarker measurements for the latter studies limit our ability to draw conclusions regarding specific Pb exposure circumstances eliciting the observed effects (4.3.11). Findings of stronger associations for adult neurocognitive effects with bone Pb,

[16] The evidence from experimental animal studies can be informative with regard to key aspects of exposure circumstances in eliciting specific effects which can inform our interpretation of the epidemiological evidence. For example, the animal evidence base with regard to Pb effects on blood pressure demonstrates the etiologically-relevant role of long-term (as compared to short-term) exposure (ISA, section 4.4.1). 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 4.4.1). For other health effects, the animal evidence base may or may not be informative with regard to the role of specific exposure circumstances in eliciting those effects.

however, indicate the role of historical or cumulative exposures for those effects (ISA, section 4.3).

Given the relatively short exposure histories of young children, there is relatively reduced uncertainty regarding the lifestages in which exposures contribute to effects for associations of early childhood effects with early childhood blood Pb levels (ISA, sections 1.9.4, 1.9.6 and 4.3.11). In considering our understanding of the relative impact on neurocognitive function of additional Pb exposure of children by school age or later we recognize increasing uncertainty associated with limitations of the currently available evidence, including epidemiological cohorts with generally similar temporal patterns of exposure. We take note, additionally, of evidence from experimental animal studies and a small body of epidemiological studies that indicates that Pb exposures during different lifestages can induce cognitive impairments. The limited epidemiological evidence is of populations with blood Pb levels that are not strongly correlated over time and that can, accordingly, address the issue of the role of exposure subsequent to the earliest lifestages (ISA, section 4.3.11; Hornung et al., 2009). Some animal evidence demonstrates impaired learning with infancy only, from infancy into adulthood, and postinfancy only Pb exposure (Rice, 1990; Rice and Gilbert 1990; Rice, 1992). Further, evidence that Pb exposure presents a risk during different lifestages is also consistent with our broader understanding that nervous system development continues throughout childhood. The limited analyses of this issue that are newly available in this review do not appreciably change our understanding or conclusions on this from those of the prior review (ISA, section 4.3.11).

As in the last review, there is also substantial evidence of other neurobehavioral effects in children, such as reduced attention span, increased impulsivity, hyperactivity, conduct disorders and effects on internalizing behaviors. The evidence for many of these endpoints, as with neurocognitive effects, also includes associations of effects at various ages in childhood and, for some effects, into adulthood, with blood Pb levels reflective of several different lifestages (e.g., prenatal and several different ages in childhood) (ISA, sections 4.3.3 and 4.3.4). There is similar or relatively less extensive evidence to inform our understanding of such effects associated with specific time periods of exposure at specific lifestages than is the case for effects on cognitive function.

Across the range of Pb effects on physiological systems and processes other than the nervous system, the full body of evidence on etiologically relevant circumstances of Pb exposure eliciting increases in blood pressure and hypertension is somewhat more informative than is the case with regard to many other effects. In the case of lead-induced increases in blood pressure, the evidence indicates an importance of long-term exposure (ISA, sections 1.6.2 and 4.4.7.1).

In summary, as in the last review, we continue to recognize a number of uncertainties regarding the circumstances of Pb exposure, including timing or lifestages, eliciting specific

health effects. Consideration of the evidence newly available in this review has not appreciably changed our understanding on this topic. The relationship of long-term exposure to Pb with hypertension and increased blood pressure in adults is substantiated despite some uncertainty regarding the exposures circumstances (e.g., magnitude and timing) contributing to blood Pb levels measured in epidemiological studies. Across the full evidence base, the effects for which our understanding of relevant exposure circumstances is greatest are neurocognitive effects in young children. Thus, we continue to recognize and give particular attention to the role of Pb exposures relatively early in childhood in contributing to neurocognitive effects which may persist into adulthood.

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

In considering the question posed here, we recognize, as discussed in section 3.1 above, that the epidemiological evidence base for our consideration in this review, as in the past, includes substantial focus on internal biomarkers of exposure, such as blood Pb, with relatively less information specific to exposure levels, including those derived from air-related pathways. Given that blood and bone Pb are integrated markers of aggregate exposure across all sources and exposure pathways, our interpretation of studies relying on them is informed by what is known regarding the historical context and exposure circumstances of the study populations. For example, a critical aspect of much of the epidemiological evidence is the backdrop of generally declining Pb exposure over the past several decades. Thus as a generality, recent epidemiological studies of populations with similar characteristics as those studied in the past tend to involve lower overall Pb exposures and accordingly lower blood Pb levels (e.g., ISA, sections 2.5 and 3.4.1; 2006 CD, section 3.4). This has been of particular note in the evidence of blood Pb associations with nervous system effects, particularly impacts on cognitive function in children, for which we have seen associations with progressively lower childhood blood Pb levels across past reviews (ISA, section 4.3.12; 1986 CD; USEPA, 1990; 2006 CD; 73 FR 66976).

The evidence currently available with regard to the magnitude of blood Pb levels associated with neurocognitive effects in children is generally consistent with that available in the review completed in 2008. Nervous system effects in children, specifically effects on cognitive function, continue to be the effects that are best substantiated as occurring at the lowest blood Pb concentrations (ISA, pp. lxxxvii-lxxxviii). Associations of blood Pb with effects on cognitive function measures in children have been reported in many studies across a range of

childhood blood Pb levels, including study group (mean/median) levels ranging down to 2 µg/dL (e.g., ISA, p. lxxxvii and section 4.3.2).[17]

Studies in which such findings were reported for childhood study group blood Pb levels below 5 µg/dL are summarized in Table 3-2.[18] In recognition of the influence of age on blood Pb levels, the analyses in Table 3-2 are listed in order of age at which the blood Pb measurements were taken. Findings for studies newly available in this review are indicated in bold text. Although the analyses from Lanphear et al (2005) listed in Table 3-2 were available in the last review, the information presented in Table 3-2 for sample size and mean blood Pb concentration has been updated to reflect recalculations using data corrected for recently identified errors, as described further in the next section below (Kirrane and Patel, 2014).

[17] 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).

[18] 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 childen with concurrent blood Pb above 10 µg/dL) (ISA, sections 4.3.2.1 and 4.3.3.1; 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).

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

Measure[A]	Study Group Dataset Description	Blood Pb Levels				Additional Information on Analyses
		Age	Mean[B] (µg/dL)	Range[B] (µg/dL)	N	
[C]*Studies discussed in ISA (section 4.3.2) with findings of effects on neurocognitive measures reported for childhood study group PbB ≤ 5 µg/dL (ordered by age of blood Pb measurements)*						
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, 4th graders 4th 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	3.3	0.9 – 7.4	118	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.

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.
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; Kirrane and Patel, 2014).
C - Bolded measures and studies are newly available in this review. D - In practice, 337-425 cases were included in analysis (Chandramouli et al., 2009).
E - Blood Pb measurements of subgroup with peak PbB <7.5 µg/dL comprised of 24.6% age 5 (Boston, Cleveland), 58.5% age 6 (Rochester), 16.9% 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.

Among the analyses of lowest study group blood Pb levels at the youngest ages in Table 3-2 are analyses available in the last review of Pb associations with neurocognitive decrement in study groups with mean levels on the order of 3-4 μg/dL in children aged 24 months or ranging from 5 to 7 years (73 FR 66978-66969; ISA, sections 4.3.2.1 and 4.3.2.2; Bellinger and Needleman, 2003; Canfield et al., 2003; Lanphear et al., 2005; Tellez-Rojo et al., 2006; Bellinger, 2008; Canfield, 2008; Tellez-Rojo, 2008; Kirrane and Patel, 2014).[19] Newly available in this review are two studies reporting association of blood Pb levels prior to three years of age with academic performance on standardized tests in primary school; mean blood Pb levels in these studies were 4.2 and 4.8 μg/dL (ISA, section 4.3.2.5; Chandramouli et al., 2009; Miranda et al., 2009). One of these two studies, which represented integer blood Pb levels as categorical variables, indicated a small effect on end-of-grade reading score of blood Pb levels as low as 2 μg/dL, after adjustment for age of measurement, race, sex, enrollment in free or reduced lunch program, parental education, and school type (Miranda et al., 2009).

In a newly available study of blood Pb levels at primary school age, a significant association of blood Pb in children aged 8-11 years and concurrently measured full scale IQ (FSIQ) was reported for a cross-sectional cohort in Korea with a mean blood Pb level of 1.7 μg/dL and range of 0.43-4.91 μg/dL (Kim et al., 2009).[20] In considering the blood Pb levels in this study, we note that blood Pb levels in children aged 8-11 are generally lower than those in pre-school children, for reasons related to behavioral and other factors (ISA, sections 3.3.5, 3.4.1 and 5.2.1.1).[21] It is likely that the blood Pb levels of this study group at earlier ages, e.g., prior to school entry, were higher and the available information does not provide a basis to judge whether the blood Pb levels in this study represent lower exposure levels than those experienced by the younger study groups. In still older children, a large cross-sectional investigation of blood Pb association with effects on memory and learning that was available in the last review was focused on children aged 6-16 years, born during 1972-1988, with a mean blood Pb of 1.9 μg/dL. A study newly available in this review, focused on a subset of the earlier study cohort

[19] 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. These tests,however, 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.

[20] Limitations of this study included a lack of consideration of potential confounding by parental caregiving quality or IQ (ISA, Table 4-3).

[21] 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 4.3.2). Separate analysis of the full study group found a significant negative association of IQ with blood Mn (Kim et al., 2009).

(ages 12-16, born during 1975-1982), also reports a significant negative association of blood Pb with learning and memory test results with mean blood Pb levels of approximately 2 μg/dL (ISA, section 4.3.2.3; Lanphear et al., 2000; Krieg et al., 2010). In considering these study findings with regard to the question of exposure levels eliciting effects, we recognize, however, that blood Pb levels are, in general, lower among teenagers than young children and also that, for these subjects specifically, the magnitude of blood Pb levels during the earlier childhood (e.g., pre-school ages) was much higher. For example, the mean blood Pb levels for the 1-5 year old age group in the NHANES 1976-80 sample was 15 μg/dL, declining to 3.6 μg/dL in the NHANES 1988-1991 sample (Pirkle et al., 1994; ISA, section 3.4.1).

With regard to other nervous system effects in children, the evidence base at lower blood Pb levels is somewhat extended since the last review with regard to the evidence on Pb and effects on externalizing behaviors, such as attention, impulsivity, hyperactivity and conduct disorders (ISA, section 4.3.3 and table 4-17). Several newly available studies investigating the role of blood Pb levels in older children (primary school age and older) have reported significant associations for these effects with concurrent blood Pb levels, with mean levels generally on the order of 5 μg/dL or higher (ISA, section 4.3.3). One exception is the newly available cross-sectional, categorical analysis of the NHANES 2001-2004 sample of children aged 8-15 years, which found higher prevalence of conduct disorder in the subgroup with concurrent blood Pb levels of 0.8-1.0 μg/dL as compared to the <0.8 μg/dL group (ISA, section 4.3.4 and Table 4-12). As noted above, we recognize that many of these children, born between 1986 and 1996, are likely to have had much higher Pb exposures (and associated blood Pb levels) in their earlier years than those commonly experienced by young children today, thus precluding a conclusion regarding evidence of effects associated with lower exposure levels than provided by evidence previously available.

As summarized earlier in this section, blood Pb has been associated with a range of health effects on multiple organ systems or processes. As is the case for studies of nervous system effects in children, newly available studies of other effects in child and adult cohorts include cohorts with similar or somewhat lower mean blood levels than in previously available studies. Categories of effects for which a causal relationship has been concluded in the ISA and for which there are a few newly available epidemiological studies indicating blood Pb associations with effects in study groups with somewhat lower blood Pb levels than previously available for these effects include effects on development (delayed puberty onset) and reproduction (male reproductive function) and on the cardiovascular system (hypertension) (ISA, sections 4.4 and 4.8; 2006 CD, sections 6.5 and 6.6). With regard to the former category, study groups in the newly available studies include groups comprised of older children ranging up to age 18 years, for which there is increased uncertainty regarding historical exposures and their role in the

observed effects.[22] An additional factor that handicaps our consideration of exposure levels associated with these findings is the appreciable uncertainty associated with our understanding of Pb biokinetics during this lifestage (ISA, sections 3.2, 3.3, and 4.8.6). The evidence newly available for Pb relationships with cardiovascular effects in adults include some studies with somewhat lower blood Pb levels than in the last review. The long exposure histories of these cohorts, as well as the generally higher Pb exposures of the past complicate conclusions regarding exposure levels that may be eliciting observed effects (ISA, sections 4.4.2.4 and 4.4.7).[23]

In summary, our conclusions regarding exposure levels at which Pb health effects occur, particularly with regard to such levels that might be common in the U.S. today, are complicated now, as in the last review, by several factors. These factors include the scarcity of information in epidemiological studies on cohort exposure histories, as well as by the backdrop of higher past exposure levels which frame the history of some study cohorts. Recognizing the complexity, as well as the potential role of higher exposure levels in the past, we continue to focus our consideration of this question on the evidence of effects in young children for which our understanding of exposure history is less uncertain.[24] Within this evidence base, we recognize the lowest study group blood Pb levels to be associated with effects on cognitive function measures, indicating that to be the most sensitive endpoint. As described above, and summarized in Table 3-2, the evidence available in this review is generally consistent with that available in the last review with regard to blood Pb levels at which such effects had been reported (ISA, section 4.3.2; 2006 CD, section 8.4.2.1; 73 FR 66976-66979). As blood Pb levels are a reflection of exposure history, particularly in early childhood (ISA, section 3.3.2), we conclude, by extension, that the currently available evidence does not indicate Pb effects at exposure levels appreciably lower than recognized in the last review.

We additionally note that, as in the last review, a threshold blood Pb level with which nervous system effects, and specifically neurocognitive effects, occur in young children cannot be discerned from the currently available studies (ISA, sections 1.9.3 and 4.3.12). Epidemiological analyses have reported blood Pb associations with neurocognitive effects (FSIQ

[22] 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 4.8.5).

[23] Studies from the late 1960s and 1970s suggest that adult blood Pb levels during that period ranged from roughly 13 to 16 μg/m^3 and from 15 to 30 μg/dL in children aged six and younger (ISA, section 4.4.1).

[24] 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 childhoood exposure may have had an influence) which provides additional support to an emphasis nervous system effects (ISA, sections 4.3, 4.4, 4.5, 4.6, 4.7, 4.8).

or BSID MDI[25]) for young child population subgroups (age five years or younger) with individual blood Pb measurements as low as approximately 1 µg/dL and mean concentrations as low as 2.9 to 3.8 µg/dL (ISA, section 4.3.12; Bellinger and Needleman, 2003; Bellinger, 2008; Canfield el al., 2003; Canfield, 2008; Tellez-Rojo et al., 2006; Tellez-Rojo et al., 2008). As concluded in the ISA, however, "the current evidence does not preclude the possibility of a threshold for neurodevelopmental effects in children existing with lower blood levels than those currently examined" (ISA, section 4.3.13).

Important uncertainties associated with the evidence of effects at low exposure levels are similar to those recognized in the last review, including the shape of the concentration-response relationship for effects on neurocognitive function at low blood Pb levels in today's young children. Also of note is our interpretation of associations between blood Pb levels and effects in epidemiological studies, with which we recognize uncertainty with regard to the specific exposure circumstances (timing, duration, magnitude and frequency) that have elicited the observed effects, as well as uncertainties in relating ambient air concentrations (and associated air-related exposures) to blood Pb levels in early childhood, as discussed in section 3.1 above. We additionally recognize uncertainties associated with conclusions drawn with regard to the nature of the epidemiological associations with blood Pb (e.g., ISA, section 4.3.13), but note that, based on consideration of the full body of evidence for neurocognitive effects, the EPA has determined a causal relationship to exist between relevant blood Pb levels and neurocognitive impacts in children (ISA, section 4.3.15.1).

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

Based primarily on studies of FSIQ, the assessment of the currently available studies, as was the case in the last review, continues to recognize a nonlinear relationship between blood Pb and effects on cognitive function, with a greater incremental effect (greater slope) at lower relative to higher blood Pb levels within the range thus far studied, extending from well above 10 µg/dL to below 5 µg/dL (ISA, section 4.3.12). This was supported by the evidence available in the last review, including the analysis of the large pooled international dataset comprised of blood Pb measurements and IQ test results from seven prospective cohorts (Lanphear et al., 2005; Rothenberg and Rothenberg, 2005; ISA, section 4.3.12). The blood Pb measurements in this pooled dataset that were concurrent with the IQ tests ranged from 2.5 µg/dL to 33.2 µg/dL. The study by Lanphear et al (2005) additionally presented analyses that stratified the dataset

[25] 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 4.3.15.1).

based on peak blood Pb levels (e.g., with cutpoints of 7.5 µg/dL and 10 µg/dL peak blood Pb) and found that the coefficients from linear models of the association for IQ with concurrent blood Pb were higher in the lower peak blood Pb level subsets than the higher groups (ISA, section 4.3.12; Lanphear et al., 2005).

We note that since the completion of the ISA, two errors have been identified with the pooled dataset analyzed by Lanphear et al (2005) (Kirrane and Patel, 2014). A recent publication and EPA have separately recalculated the statistics and mathematical models of Lanphear et al (2005) using the corrected pooled dataset (Kirrane and Patel, 2014). While the magnitude of the loglinear and linear regression coefficients are modified slightly based on the corrections, the conclusions drawn from these coefficients, including the finding of a steeper slope at lower (as compared to higher) blood Pb concentrations are not affected (Kirrane and Patel, 2014).

In other publications, stratified analyses of several individual cohorts also observed higher coefficients for blood Pb relationships with measures of neurocognitive function in lower as compared to higher blood Pb subgroups (ISA, section 4.3.12; Canfield et al., 2003; Bellinger and Needleman, 2003; Kordas et al., 2006; Tellez-Rojo et al., 2006). Of these subgroup analyses, those involving the lowest mean blood Pb levels and closest to the current mean for U.S. preschool children are listed in Table 3-3 below (drawn from Table 3 of the 2008 final rulemaking notice [73 FR 67003], and Kirrane and Patel, 2014). These analyses were important inputs for the evidence-based, air-related IQ loss framework which informed decisions on a revised standard in the last review (73 FR 67005), discussed in section 4.1.1 below. As the framework focused on the median of the four slopes in Table 3-3, the change to the one from Lanphear et al (2005) based on the recent recalculation described above has no impact.

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.

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
3.3	0.9 – 7.4	Lanphear et al (2005)[C], subgroup with peak blood Pb <7.5 μg/dL	-2.53
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. The blood Pb levels and coefficient presented here reflect the recalculation using the corrected pooled dataset (Kirrane and Patel, 2014).

Several studies newly available in the current review have, in all but one instance, also found a nonlinear blood Pb-cognitive function relationship in nonparametric regression analyses of the cohort blood Pb levels analyzed (ISA, section 4.3.12). These studies, however, used statistical approaches that did not produce quantitative results for each blood Pb group (ISA, section 4.3.12). Thus, newly available studies have not extended the range of observation for quantitative estimates of this relationship to lower blood Pb levels than those of the previous review. The ISA further notes that the potential for nonlinearity has not been examined in detail within a lower, narrower range of blood Pb levels than those of the full cohorts thus far studied in the currently available evidence base (ISA, section 4.3.12). Such an observation in the last review supported the consideration of linear slopes with regard to blood Pb levels at and below those represented in Table 3-3. In summary, the newly available evidence does not substantively alter our understanding of the concentration-response relationship (including 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 1.9.1). 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 of adult populations, for whom historical Pb exposures were likely much higher than exposures that commonly occur today (ISA, section 4.4). There is less uncertainty regarding the exposure patterns contributing to the blood Pb levels reported in studies of younger populations (ISA, sections 1.9.4, 1.10). 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 4.3.2; 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 4.3.2). 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 1.9.5).[26] 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 4.3.14). 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 "[s]mall shifts in the population mean IQ can be highly significant from a public health perspective" (ISA, p. xciii). For example, if lead-related decrements are manifested uniformly across the range of IQ scores in a population, "a small shift in the population mean IQ may be significant from a public health perspective because such a shift could yield a larger proportion of individuals functioning in the low range of the IQ distribution, which is associated with increased risk of educational, vocational, and social failure" as well as a decrease in the proportion with high IQ scores (ISA, section 1.9.1).

[26] 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, p. 1-77; Bellinger et al., 1990).

In the discussion below, we use the term at-risk populations[27] to recognize populations that have a greater likelihood of experiencing lead-related health effects, i.e., groups with characteristics that contribute to an increased risk of Pb-related health effects. These populations are also sometimes referred to as sensitive groups, as in section 1.2.1 above. This increased likelihood of lead-related effects can result from many factors, including lifestage or age, sex, race or ethnicity, diet, pre-existing disease state, or increased exposure (ISA, chapter 5). Accordingly, in identifying factors that increase risk of lead-related health effects, there has been consideration of evidence regarding factors contributing to increased susceptibility (i.e., physiological or intrinsic factors contributing to a greater response for the same exposure), and those contributing to increased exposure (including that resulting from behavior leading to increased contact with contaminated media). As noted in the ISA, "definitions of susceptibility and vulnerability vary across studies, but in most instances 'susceptibility' refers to biological or intrinsic factors (e.g., age, sex) while 'vulnerability' refers to nonbiological or extrinsic factors (e.g., socioeconomic status [SES])" and the terms "at-risk" and "sensitive" populations have in various instances been used to encompass these concepts more generally (ISA, p. 5-1). Although we emphasize the term "at-risk", we rely on the other terms in particular instances below; in so doing, our usage is consistent with these definitions.

Factors that increase risk of lead-related effects include, among others, behavioral and physiological factors. A behavioral factor of great impact on Pb exposure is the incidence of hand-to-mouth activity that is prevalent in very young children and by which they transfer Pb in settled particles to their mouths (ISA, sections 3.7.1 and 5.2.1.1). Physiological factors include both conditions contributing to a group's increased risk of effects at a given blood Pb level, and those that contribute to blood Pb levels higher than those otherwise associated with a given Pb exposure (ISA, sections 5.3 and 5.1, respectively). We also considered evidence encompassing situations of elevated exposure, such as residing in old housing with Pb-containing paint or near sources of ambient Pb, as well as socioeconomic factors, such as reduced access to health care or low socioeconomic status (SES) that can contribute to increased risk of adverse health effects from Pb (ISA, sections 1.9.7, 5.2, and 5.4).

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

The information newly available in this review has not substantially altered our understanding of at-risk populations. As in the last review, the factor most prominently

[27] In the context of "at-risk populations", the term population refers to persons having one or more qualities or characteristics including, for example, a specific pre-existing illness or a specific age or lifestage, with lifestage referring to a distinguishable time frame in an individual's life characterized by unique and relatively stable behavioral and/or physiological characteristics that are associated with development and growth.

recognized to contribute to increased risk of Pb effects is age (ISA, section 1.9.6). As noted in section 3.2 above, although the specific ages or lifestages of greatest susceptibility or risk have not been established (e.g., ISA, section 4.3.11), the at-risk status of young children to the neurodevelopmental effects of Pb is well recognized (e.g., ISA, sections 1.9.6, 4.3, 5.2.1, 5.3.1, and 5.4). The evidence indicates that prenatal blood Pb levels are associated with nervous system effects, including mental development in very young children and can also be associated with cognitive decrements in older children (ISA, section 4.3). The coincidence during early childhood of behaviors that increase exposure, such as hand-to-mouth contact, and the development of the nervous system contributes increased risk during this time (ISA, sections 4.3.2.6, 5.2.1.1, and 5.3.1.1). The evidence also indicates a relationship of postnatal blood Pb levels (through early childhood to school age) with cognitive function decrement in older children and adolescents (ISA, section 4.3). In epidemiological studies, associations have been observed of neurocognitive, and some other nervous system effects, at various ages from early childhood to school age with prenatal, early-childhood, lifetime average, and concurrent blood Pb levels as well as with childhood tooth Pb levels (ISA, section 4.3). Consideration of epidemiological study results for different lifestages of exposure, particularly later in childhood, is complicated by the fact that blood Pb levels in children, although highly affected by recent exposure, are also influenced by their history of Pb exposure due to rapid growth-related bone turnover in children (ISA, section 3.3.5). Thus, blood Pb level in children also may reflect past Pb exposures and, to some extent, maternal Pb, with relative contributions varying with child and maternal exposure history (ISA, section 3.2.2.4, 3.4.1 and 4.3.15; 2006 CD, section 6.6.2). Collectively, however, the evidence indicates both the susceptibility of the developing fetus and early postnatal years, as well as the potential for continued susceptibility through childhood as the human central nervous system continues to mature and be vulnerable to neurotoxicants (ISA, sections 1.9.5 and 4.3.15; 2006 CD, section 6.2.12).

In the collective body of evidence of nervous system effects in children, it is difficult to distinguish exposure in later lifestages (e.g., school age) and its associated risk from risks resulting from exposure in prenatal and early childhood (ISA, section 4.3.11). While early childhood is recognized as a time of increased susceptibility, a difficulty in identifying a discrete period of susceptibility from epidemiological studies has been that the period of peak exposure, reflected in peak blood Pb levels, is around 18-27 months when hand-to-mouth activity is at its maximum (ISA, section 3.4.1 and 5.2.1.1; 2006 CD, p. 6-60). The task is additionally complicated by the role of maternal exposure history in contributing Pb to the developing fetus (ISA, section 3.2.2.4.). Epidemiological analyses evaluating risk of neurocognitive impacts (e.g., reduced IQ) associated with different blood Pb metrics in cohorts with differing exposure patterns (including those for which blood Pb levels at different ages were not highly correlated)

indicate associations with blood Pb measurements concurrent with FSIQ tests at ages of approximately 6-7 years, although the analyses did not conclusively demonstrate stronger findings for early (e.g., age 2 years) or concurrent blood Pb (ISA, section 4.3.11). The experimental animal evidence additionally indicates early life susceptibility (ISA, section 4.3.15 and p. 5-21). Thus, the full evidence base continues to indicate prenatal and early childhood lifestages as periods of increased lead-related risk. In summary, while uncertainties remain with regard to the role of Pb exposures during a particular age of life in eliciting nervous system effects, such as cognitive function decrements, the evidence continues to indicate the at-risk status of pre- and postnatal childhood lifestages (ISA, sections 4.3.11 and 4.3.15).

Several physiological factors increase risk of lead-related health effects by contributing to increased blood Pb levels over those otherwise associated with a given Pb exposure (ISA, sections 3.2, 3.3 and 5.1). These include nutritional status, which plays a role in Pb absorption from the GI tract (ISA, section 3.2.1.2). For example, diets deficient in iron, calcium or zinc can contribute to increased Pb absorption and associated higher blood Pb levels (ISA, sections 3.2.1.2, and 5.1). Evidence is suggestive of some genetic characteristics as potential risk factors, such as presence of the δ-aminolevulinic acid dehydratase-2 (ALAD-2) allele which has been indicated to increase blood Pb levels or lead-related risk (ISA, sections 3.3.2 and 5.1).

Risk factors based on increased exposure include spending time in proximity to sources of Pb to ambient air or other environmental media (e.g., large active metals industries or locations of historical Pb contamination) (ISA, sections 3.7.1 and 5.2.5). Residential factors associated with other sources of Pb exposure (e.g., leaded paint or plumbing with Pb pipes or solder) are another exposure-related risk factor (ISA, sections 3.7.1 and 5.2.6). The role of socioeconomic status (SES) with regard to lead-related risk is somewhat complicated. SES often serves as a marker term for one or a combination of unspecified or unknown environmental or behavioral variables. Lower SES has been associated with higher Pb exposure and higher blood Pb concentration (ISA, sections 5.3.16, 6.2.4 and 6.4). Further, it is independently associated with an adverse impact on neurocognitive development, and a few studies have examined SES as a potential modifier of the association of childhood Pb exposure with cognitive function with inconsistent findings regarding low SES as a potential risk factor. Although the differences in blood Pb levels among children of lower as compared to higher income levels have lessened, blood Pb levels continue to be higher among lower-income children indicating higher exposure and/or greater influence of factors independent of exposure, such as nutritional factors (ISA, sections 1.9.6, 5.2.1.1 and 5.4).

In considering risk factors associated with increased Pb exposure or increased blood Pb levels, we note that the currently available evidence continues to support a nonlinear relationship between neurocognitive effects and blood Pb that indicates incrementally greater impacts at

lower as compared to higher blood Pb levels (ISA, section 4.3.12), as described in section 3.2 above. An important implication of this finding is that while children with higher blood Pb levels are at greater risk of lead-related effects than children with lower blood Pb levels, on an incremental basis (e.g., per μg/dL), the risk is greater for children at lower blood Pb levels. This was given particular attention in the last review of the Pb NAAQS, in which the standard was revised with consideration of the incremental impact of air-related Pb on young children in the U.S and the recognition of greater impact for those children with lower absolute blood Pb levels (73 FR 67002). Such consideration included a focus on those C-R studies involving the lowest blood Pb levels, as described in section 4.1.1 below.

Some racial or ethnic backgrounds have been identified as factors that may increase risk of lead-related health effects (ISA, sections 1.9.6, 5.2.3 and 5.3.7). For example, although blood Pb levels in the U.S. general population (e.g., geometric mean level in children aged 1-5) have declined, mean levels reported in recent NHANES samples continue to differ among children of different ethnic backgrounds, with higher levels in non-white persons as compared to whites (ISA, sections 3.4.1, 5.2.1.1 and 5.2.3). Additionally, a study of lead-related risk of hypertension among adults reported greater risk associated with blood Pb levels above 1 μg/dL among Mexican Americans and non-Hispanic blacks than among non-Hispanic whites (ISA, section 5.3.7; Muntner et al., 2005). The evidence available in the current review, consistent with that in the last, also suggests that health status or pre-existing disease is potentially a physiological risk factor for lead-related effects (ISA, section 1.9.6). Populations with pre-existing health conditions, such as hypertension, may be more susceptible than those without such conditions for particular Pb-associated effects (ISA, section 5.3.4). For example, increased risks of lead-related renal effects and heart rate variability have been reported among hypertensive individuals compared to those that are normotensive (ISA, section 1.9.6). Additionally, African Americans, as a group, have a higher frequency of hypertension than the larger U.S. population as a whole and than other ethnic groups (NCHS, 2011) and, as a result, may face a greater risk of adverse health impact from Pb-associated cardiovascular effects.

Older adulthood has been identified as a lifestage of potentially greater risk of lead-related health effects based primarily on the evidence of increases in blood Pb levels during this lifestage (ISA, sections 5.2.1.2, 5.3.1.2, and 5.4). Contributing to blood Pb levels in the studied populations of older adults are likely to be their exposure histories, which included younger years during the time of leaded gasoline usage and other sources of Pb exposures which were more prevalent in the past than today (e.g., ISA, Figure 2-1 and section 2.5.2). Exposure history has a contributing role to blood Pb levels throughout life, and the increased rate of bone remodeling during later adulthood increases contributions of Pb from bone stores into the systemic circulation during that lifestage (ISA, sections 3.3.5 and 5.2.1.2). Additionally, limited

animal evidence has indicated specific brain pathology in older animals that had substantial Pb exposures earlier in life (ISA, sections 4.3.9.1 and 5.3.1). Further, the full body of evidence includes observed associations of some cardiovascular and neurological effects with bone and blood Pb in older populations, with biological plausibility for the role of Pb provided by experimental animal studies (ISA, sections 4.3.5, 4.3.7 and 4.4).

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

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

The evidence newly available in this review supports or strengthens our previous conclusions regarding the broad array of health effects of Pb (see ISA, section 1.10 which compares key conclusions drawn in the last review with conclusions drawn in the current assessment). Additionally, in some categories of health effects, the newly available studies extend the evidence for some aspects of the health effects described in the last review. For example, among the nervous system effects, the newly available evidence continues to support the conclusions from the last review regarding Pb and neurocognitive and behavioral effects (ISA, section 4.3). Across the array of neurocognitive and behavioral effects, we recognize the sensitivity of the prenatal period and several lifestages of childhood, and we particularly recognize young children as an important at-risk population in light of current environmental exposure levels.

As discussed in section 3.2 above, the blood Pb levels of populations studied in newly available epidemiological studies that report associations of blood Pb with effects for systems and processes other than the nervous system (e.g., cardiovascular, developmental and reproductive) are similar to or, in a few cases, somewhat lower than those assessed in the last review (ISA, sections 4.4, 4.6, 4.7, and 4.8). The greater uncertainties regarding the time,

duration and magnitude of exposure contributing to these observed health effects complicate identification of sensitive lifestages and associated exposure patterns that might be compared with our understanding of the sensitivity of young children to neurocognitive impacts of Pb. Thus, while augmenting the evidence base on these additional endpoints, the newly available evidence does not lead us to identify a health endpoint expected to be more sensitive to Pb exposure than neurocognitive endpoints in children, leading us to continue to conclude that the appropriate primary focus for our review is on neurocognitive endpoints in children.

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

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

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

In considering the extent of this at-risk population, we turn first to consideration of those areas in the U.S. with air Pb concentrations above the current standard (e.g., section 2.2.2.2 above). Using the available monitoring data and U.S. census information, Table 3-4 summarizes the size of populations within 0.5 km of monitors in our current Pb NAAQS surveillance network at which Pb concentrations were higher than the current standard during the recent period from 2009-2011. The distance to which concentrations exceeding the standard might extend will vary with the magnitude of the Pb concentrations and particle size, among other factors; a half-kilometer distance was selected for purposes of illustration here. This analysis indicates that approximately 2,400 children aged 5 or under reside within 0.5 km of monitors

exceeding the current standard. To also account for the population in areas with air concentrations just at (or very near) the current standard, we have also identified an additional nine Pb-TSP monitors with 3-month average concentrations within 10% of, but not exceeding, the current standard (Appendix 2D). Based on the 2010 U.S. census, 265 children aged 5 or under reside within 0.5 km of these additional sites.

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

Population within 0.5 km of monitors with maximum 3-month Pb concentration greater than 0.15 μg/m³ (2009-2011)				
	Number of Counties	Number of States or Territories	Total population	Children, 5 and under
All sites >0.15 μg/m³	29	20	25,344	2,416
Subset of sites >0.5 μg/m³	11	9	11,753	1,018
Data Sources: U.S. Bureau of the Census, 2010 Census of Population and Housing and recent Pb-TSP dataset presented in Figure 2-10 above (dataset criteria and summaries included Appendices 2C and 2D, respectively). Section 2.2.1.1 above describes the surveillance monitoring network required for identifying locations with the potential to exceed the NAAQS.				

As the air quality data set analyzed in section 2.2.2 above may not be inclusive of all of the newly sited monitors, as discussed in section 2.2.1 above, we recognize there may be other areas of the country where concentrations are above or just meet the current standard but for which such data are not yet available. To consider the potential for there to be additional, not yet identified, areas with elevated Pb concentrations, we have separately quantified the size of young child populations residing in areas near large Pb sources in Table 3-5. In so doing, we recognize uncertainties and potential limitations associated with these emissions estimates for these purposes, uncertainties both with regard to the accuracy of such estimates and also with regard to the role of specific source characteristics and meteorology, not explicitly considered here, in influencing ambient air Pb concentrations and contributing to substantial variation in air Pb concentrations at source locations (e.g., Figure 2-11 above). Accordingly, while the summary in Table 3-5 is informative in considering the potential prevalence of airborne Pb emissions and potential exposure of human populations, it is limited with regard to its ability to identify populations living in areas of elevated ambient air Pb concentrations. We interpret this analysis to indicate that fewer than about 7,800 young children (aged 5 or younger) live in areas with air Pb concentrations near or above the current standard, with the current monitoring data indicating the size of this population to be approximately 2,700.

Table 3-5. Population size near larger sources of Pb 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
Facilities/Airports estimated to emit > 5.0 tpy						
8	484	61	0	0	0	61
Facilities/Airports estimated to emit 1.0 - 4.9 tpy [A]						
53	12,143	731	6	6,261	266	997
Facilities/Airports estimated to emit 0.50 – 0.95 tpy [B]						
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.

3.4 EXPOSURE AND RISK

The risk information available for this review and described here is based primarily on the exposure and risk assessment developed in the last review of the Pb NAAQS (henceforth referred to as the 2007 REA [USEPA, 2007a]), as considered in the context of the evidence newly available in this review (as presented in the ISA). As described in the REA Planning Document, careful consideration of the information newly available in this review, with regard to designing and implementing a full REA for this review, led us to conclude that performance of a new REA for this review was not warranted. We did not find the information newly available in this review to 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 (REA Planning Document, section 2.3). Based on their consideration of the REA Planning Document analysis, the CASAC Pb Review Panel generally concurred with the conclusion that a new REA was not warranted in this review (Frey, 2011).[28] Accordingly, the information described here is drawn primarily from the 2007 REA, augmented by a limited new case study-specific analysis focused on risk associated with the current standard, as described in section 3.4.3.3 below.

[28] In our evaluation presented in the REA Planning Document and in 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. In their review of the draft PA, the CASAC Pb Review Panel reenforced their concurrence with EPA's decision not to develop a new REA (Frey, 2013).

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

The conceptual model developed to inform planning for the 2007 REA, including identification of key exposure media, target population, health endpoint and risk metric is described in section 3.4.1. The 2007 REA relied on a case study approach to provide estimates that inform our understanding of air-related exposure and risk in different types of air Pb exposure situations; the case studies included are described in section 3.4.2. In section 3.4.3, the analysis approach and general aspects of exposure and risk assessment methods are summarized, and the air quality scenarios simulated are described. In section 3.4.3, we also summarize the 2007 REA risk model and the interpolation approach used in the limited new analyses performed for purposes of this review. Section 3.4.4 identifies key aspects of the exposure assessment and risk estimates are presented in section 3.4.5. Treatment of key sources of variability in exposure and risk estimates is described in section 3.4.6 and the characterization of uncertainty is summarized in 3.4.7. An updated interpretation of the risk estimates for our purposes in this Pb NAAQS review section is presented in section 3.4.8.

3.4.1 Conceptual Model for Air-Related Lead Exposure and Risk

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

As recognized in section 1.3 above, sources of human Pb exposure include current and historical air emissions sources, as well as miscellaneous nonair sources, which can contribute to multiple exposure media and associated pathways (e.g., inhalation of ambient air, ingestion of indoor dust, outdoor soil/dust and diet or drinking water).[29] Figure 3-2 illustrates these human exposure pathways from an analytical perspective, drawing on the conceptual model for the assessment (2007 REA, Figure 2-1).[30] As shown in Figure 3-2, in addition to airborne emissions (recent or those in the past), sources of Pb to these pathways also include old leaded paint, including Pb mobilized indoors during renovation/repair activities, and contaminated soils. Lead in diet and drinking water may have air pathway-related contributions as well as contributions from nonair sources (e.g., Pb solder on water distribution pipes and Pb in materials used in food processing). Limitations in our data and modeling tools handicapped our ability to separate the nonair contributions to Pb exposure from estimates of air-related Pb exposure and risk.[31]

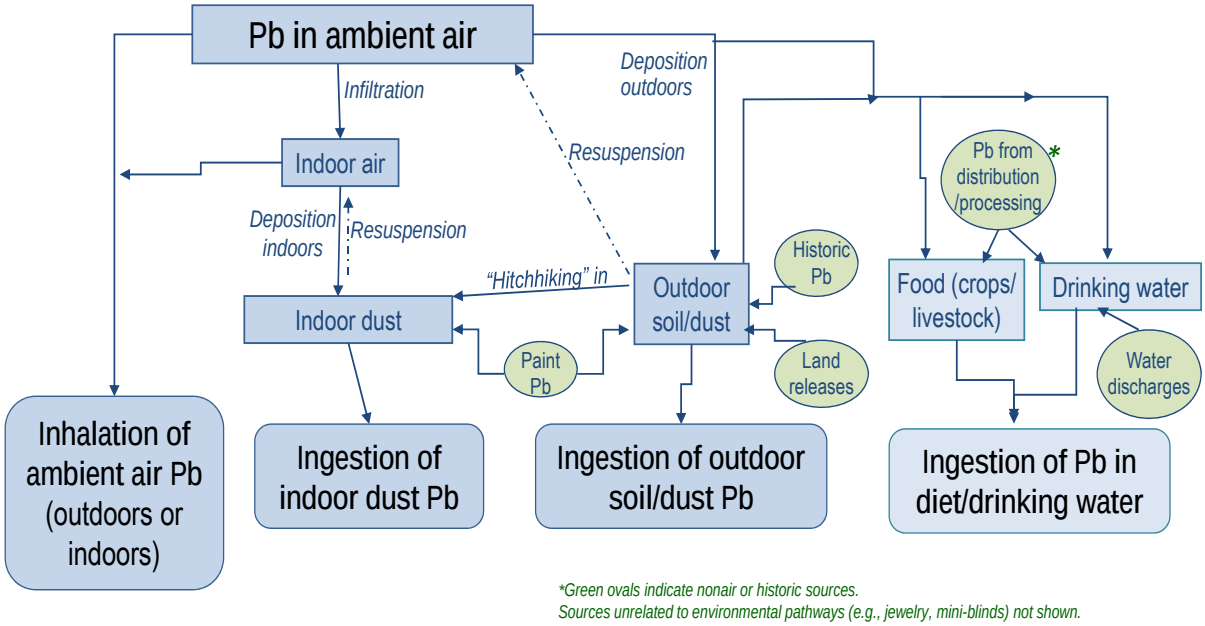


Figure 3-2. Human exposure pathways for air-related Pb.

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

[30] We additionally note that Pb in children at birth is from maternal exposures, recent or historical, as recognized in section 3.1 above, and that ingestion of maternal breast milk may be a Pb exposure pathway for infants in some cases (ISA, section 3.1.3.3).

[31] 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.

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

In the REA, we focused on IQ loss in children exposed up to age 7 years. This focus reflected the evidence for young children with regard to air-related exposure pathways and susceptibility to Pb health impacts (e.g., ISA, sections 3.1.1, 4.3, 5.2.1.1, 5.3.1.1, and 5.4). For example, the hand-to-mouth activity of young children contributes to their Pb exposure (i.e., incidental soil and indoor dust ingestion) and ambient air-related Pb has been shown to contribute to Pb in outdoor soil and indoor house dust (ISA, sections 3.1.1 and 3.4.1; 2006 CD, section 3.2.3).

Blood Pb is commonly used as an integrated index or biomarker of exposure due to both its association with exposure, particularly recent exposure in young children, and the relative ease of the measurements, as discussed in section 3.1 above. Although bone Pb measurements have become easier to collect and consequently, their use has been more widespread, epidemiological and toxicological studies of Pb health effects and dose-response relationships (particularly for neurodevelopmental effects in children) tend to be dominated by blood Pb as the exposure metric. Therefore, we focused on modeling blood Pb in young children, developing estimates for two blood Pb metrics: "concurrent" and "lifetime average". For the former we estimated blood Pb at age 7 years, while lifetime average was estimated as the average across the 7-year period.[32]

In addition, our focus on young children reflects the evidence that the developing nervous system in children is among, if not, the most sensitive of the endpoints associated with Pb exposure (ISA, sections 1.6 and 1.10). At the time of the last review, we noted that limitations precluded prediction of changes in adult blood Pb levels (or bone Pb levels) given changes in ambient Pb levels. This reflects the fact that the presence of substantial historic Pb stores in most adults introduces uncertainty into the prediction of changes in blood or bone Pb in these adult populations resulting from changes in ambient air Pb exposure. Additionally, in considering concentration-response relationships for adult blood Pb and adult health outcomes, we recognized the uncertainty with regard to the role of historic compared to recent exposures in eliciting the observed outcomes. Based on conclusions regarding the scientific evidence available in the last review (presented in the 2006 CD), the assessment focused on risk to the

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

central nervous system in childhood as the most sensitive effect that could be quantitatively assessed, with decrement in IQ used as the risk metric.

3.4.2 Case Studies

Lead exposure and associated risk were estimated for multiple case studies that generally represent two types of residential population exposures to air-related Pb (see Table 3-6): (1) location-specific urban populations of children with a broad range of air-related exposures, reflecting existence of urban concentration gradients; and, (2) children residing in localized areas with air-related exposures representing air concentrations specifically reflecting the standard level being evaluated. Thus, the two types of case studies differed with regard to the extent to which they represented population variability in air-related Pb exposure (as discussed further in section 3.4.7 below). Three location-specific urban case studies focused on residential areas within Cleveland, Chicago, and Los Angeles, providing representations of urban populations with a broad range of air-related exposures due to spatial gradients in both ambient air Pb levels and population density. For example, the highest air concentrations in these case studies (i.e., those closest to the standard being assessed) were found in very small parts of the study areas, while a large majority of the case study populations resided in areas with much lower air concentrations. The case studies representing the children most highly exposed via air-related pathways were the generalized (local) urban case study (also referred to as *general urban case study*) and the primary Pb smelter case study subarea. The generalized (local) urban case study was not based on a specific geographic location and reflected several simplifying assumptions in representing exposure including uniform ambient air Pb levels associated with the standard of interest across the hypothetical study area and a uniform study population. The primary Pb smelter case study provides risk estimates for children living in a specific area within which some locations, at the time of the 2007 REA, were not meeting the then-current standard. In addition to characterizing risks within a 10 km radius area surrounding the smelter (full area), we focused particularly on a subarea within 1.5 km of the facility, where airborne Pb concentrations were closest to the then-current standard (a maximum calendar quarter average concentration of 1.5 $\mu g/m^3$ Pb-TSP) and where children's air-related exposures are most impacted by emissions associated with the Pb smelter from which air Pb concentrations were estimated. Based on the nature of the population exposures represented by the two categories of case study, the generalized (local) urban and primary Pb smelter case study subarea include populations that are relatively more highly exposed by way of air pathways to air Pb concentrations near the standard

level evaluated, compared with the populations in the three cities or the full area of the primary smelter case study.[33,34]

Table 3-6. Types of population exposures assessed.

Type of Population Exposure			Case Study[1]
Broad range of air-related exposures	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)
Generalized, high end of air-related exposure	Localized residential area with air concentrations generally representing the standard or conditions evaluated	Single exposure zone without enumerated population	*Generalized (local)* urban
		A few exposure zones with small population	Primary Pb smelter (subarea)

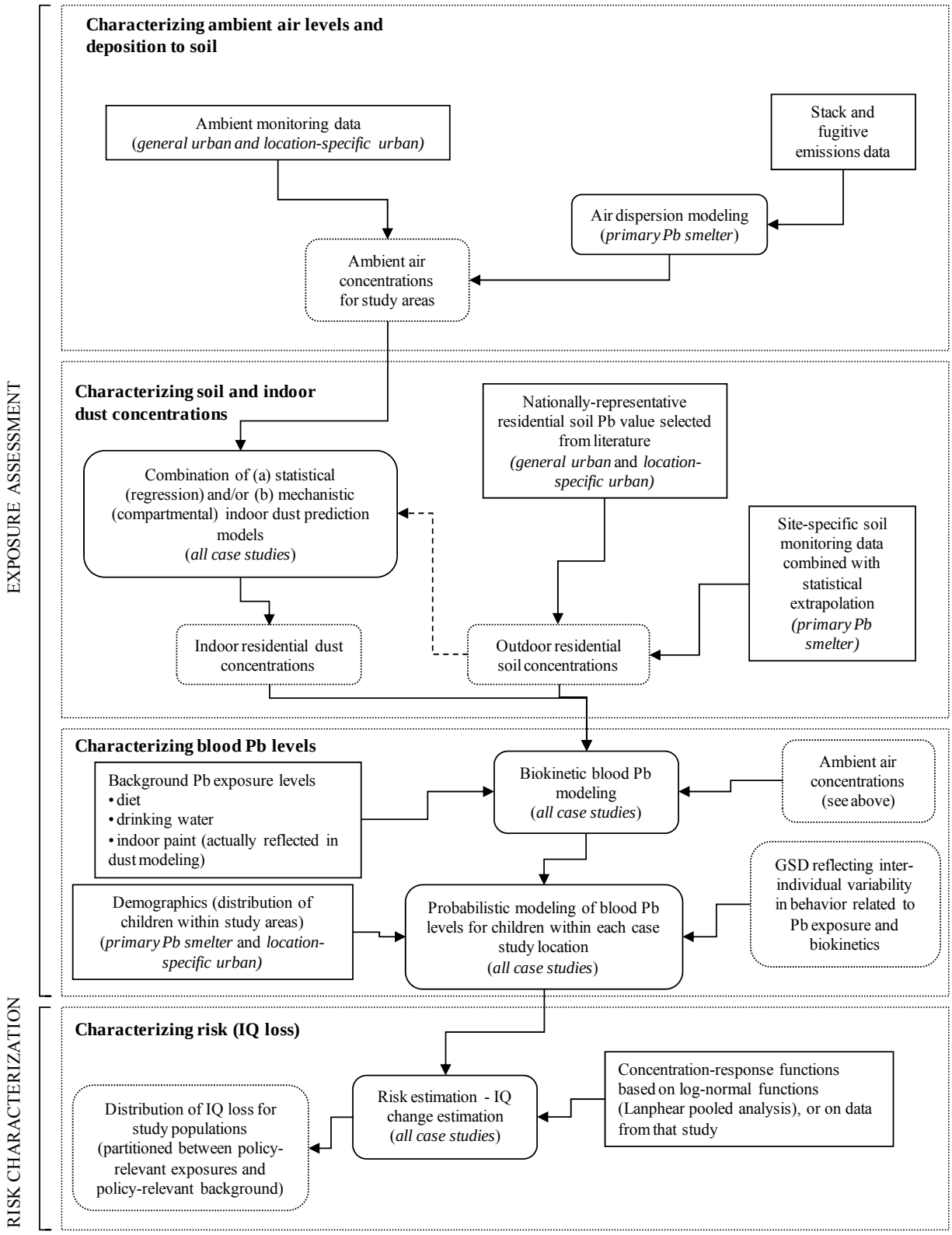
3.4.3 Analysis Approach

The approach to assessing exposure and risk for the two categories of case studies was comprised of four main analytical steps: (a) estimation of ambient air Pb concentrations, (b) estimation of Pb concentrations in other key exposure media, including outdoor soil and indoor dust, (c) use of exposure media Pb concentrations, with other pathway Pb intake rates (e.g., diet), to estimate blood Pb levels in children using biokinetic modeling, and (d) use of concentration-response functions derived from epidemiology studies to estimate IQ loss associated with the blood Pb levels. In implementing these steps for the primary smelter case study, air concentrations were estimated using dispersion modeling; indoor dust concentrations were estimated for the case study subarea using a site-specific regression model. The approach for the generalized (local) urban case study and location-specific urban case studies was somewhat

[33] 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).

[34] In addition to the case studies included in the 2007 REA, 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.

simpler, since it did not involve fate and transport modeling for air concentration estimates and, instead, used ambient monitor levels to characterize the gradient in air Pb levels across the study area. All steps are somewhat simpler in the generalized (local) urban case study which included a single exposure zone. Figure 3-3 identifies the key input data sets, modeling steps and intermediate model output in each of the four analytical steps. The first three steps were employed in the exposure assessment (discussed in section 3.4.3.1), while the fourth is the risk assessment step (discussed in section 3.4.3.3).



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

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.

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

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

<table>
<tr><th>Simulation of air quality impacts</th><th>Media category</th><th>Generalized (local) urban case study</th><th>Location-specific urban case study</th><th>Primary Pb smelter case study (1.5 km subarea and 10 km full area)</th></tr>
<tr><td rowspan="2">Concentrations for these media <u>were varied</u> across air quality scenarios (see section 3.4.3.2).</td><td>Ambient air Pb levels</td><td>Single ambient air Pb level assumed across entire study area (single exposure zone)</td><td>Source and non-source monitors describe concentration gradient (6 to 11 exposure zones per case study)</td><td>Dispersion modeling of smelter-related emissions (22 census block groups and 115 blocks)</td></tr>
<tr><td>Indoor dust Pb levels</td><td colspan="2">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)</td><td>For subarea - regression of site-specific air, dust data. For full area - regression of air, dust, soil data from other, historical locations.</td></tr>
<tr><td rowspan="3">Concentrations <u>were constant</u> across air quality scenarios (data/modeling limitations)</td><td>Outdoor soil Pb levels</td><td colspan="2">National dataset (HUD, for houses constructed between 1940 and 1998).</td><td>Site-specific data (for subarea)</td></tr>
<tr><td>Dietary Pb intake</td><td colspan="3">National datasets for Pb residue data (US FDA Total Diet Study) and food consumption data (NHANES)</td></tr>
<tr><td>Drinking water Pb intake</td><td colspan="3">US and Canada datasets for residential water Pb concentrations and ingestion rates</td></tr>
</table>

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

sections 3.2.1.1 and 5.2.4).[35] Diet and drinking water intake and concentrations, as well as other model inputs, were based on the most current information (2007 REA, Appendix H). Detail on methods used to characterize media Pb concentrations and all IEUBK inputs for each case study are in the 2007 REA, sections 3.1, 3.2, 5.2.3 and 5.2.4 and appendices C through H. As the shortest temporal scale accepted for inputs to the IEUBK is a year, all model inputs, developed for each exposure zone in each case study, were annual average values. For media concentration inputs, the same values were used for each year of the seven-year simulation. Other model inputs varied as appropriate with the age of the simulated child (2007 REA, Appendix H).

To simulate population variability in Pb intake and uptake, we used the IEUBK model to first generate a central-tendency estimate of the blood Pb levels for the group of children within a given exposure zone of a study area.[36] Outside the IEUBK model, we then combined this central-tendency estimate with a geometric standard deviation (GSD) reflecting variability in blood Pb levels for groups of children to generate a distribution of blood Pb levels for a study area. The procedure for combining the IEUBK-based central tendency blood Pb estimate with a GSD to generate a population distribution of blood Pb levels differs somewhat for the categories of case studies. The approach for the general urban case study is fairly simple in that we have a single IEUBK-based estimate of blood Pb levels, and this is, in turn, combined with the GSD selected for this study area to produce a population distribution of blood Pb levels. For both the primary Pb smelter and the location-specific urban study areas, multiple polygons within the larger study area (e.g., U.S. Census blocks for the location specific urban study areas) are used as the basis for generating distributions of blood Pb levels for the child population in each study area. These distributions are generated using a Monte Carlo-based population-weighted sampling method with U.S. Census child counts for each polygon and an adjustment factor distribution based on the chosen GSD (see 2007 REA, sections 3.2.2 and 5.2.2.3).

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

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

[36] 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.

differences in environmental Pb exposure concentrations (e.g., spatial gradients in ambient Pb levels of a resolution beyond that simulated in each case study, differences in cleaning/vacuuming rates and air exchanges rates).[37] For all of the study areas, we assumed that pathway apportionment of blood Pb levels based on the modeling of the central-tendency blood Pb level (using IEUBK) holds for all percentiles of blood Pb levels derived by combining that central tendency estimate with the GSD. Blood Pb modeling completed for all case studies included estimates of both concurrent and lifetime-average blood Pb metrics, although ultimately we focused on the concurrent blood Pb metric in estimating risk (2007 REA, section 2.1.5).[38]

3.4.3.2 Air Quality Scenarios Included in 2007 Assessment

The air quality scenarios assessed in the 2007 REA for the case studies identified in Table 3-6 above included conditions just meeting the NAAQS that was current at the time of the last review (1.5 $\mu g/m^3$, maximum calendar quarter average) and conditions meeting several alternative, lower standards. Additionally, scenarios for current conditions (2003-2005) were also included for the three location-specific urban case studies.[39] These air quality scenarios are characterized by quarterly or monthly averaging times and a not-to-be-exceeded form. Once the air quality dataset representing each scenario was developed, the associated annual average concentrations for each exposure zone were derived for input to the IEUBK model, which does not accept air quality inputs of a temporal scale shorter than a year (2007 REA, Appendix H). [40]

As a result of the differing air quality conditions of the location-specific case studies, there were differences among them with regard to the scenarios assessing the then-existing or alternative standards (see Table 3-8). To simulate the previous (1978) standard at the primary Pb

[37] 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).

[38] 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's effect on IQ than were peak and early childhood levels.

[39] 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 $\mu g/m^3$, respectively, and the maximum calendar quarter average concentration was 0.36, 0.14 and 0.09 $\mu g/m^3$, respectively, (2003-2005 data; 2007 REA, Appendix O).

[40] 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.

smelter location, at which then-current monitoring data indicated exceedance of that standard, a proportional roll-down was performed across the area to achieve conditions that just met that standard. Additionally, although it was considered an extremely unlikely scenario that air concentrations in urban areas across the U.S. that were well below the previous NAAQS would increase to just meet that standard (e.g., by way of expansion of existing sources or congregation of multiple sources in adjacent locations), we simulated this scenario in all case studies. In so doing, the air Pb concentrations were rolled up proportionally across the location-specific urban study areas to conditions just meeting the standard. No other scenario simulations involved rolling concentrations up. For the primary Pb smelter case study, air Pb concentrations were proportionally rolled down to conditions just meeting each of the potential alternative standards assessed. In the three location-specific urban case studies, the temporally and spatially varying concentrations were rolled down to conditions just meeting each of the potential alternative standards that they exceeded (2007 REA, section 5.2.2.1).[41]

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

[41] 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".

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 ($\mu g/m^3$)	Maximum Monthly Average ($\mu g/m^3$)		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.

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

3.4.3.3 Methods for Deriving Risk Estimates

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

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 blood Pb levels for the concurrent blood Pb metric[42] were combined with four blood Pb 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 blood Pb levels for which there is limited representation in the Lanphear et al (2005) pooled dataset; the 5th percentile for the concurrent blood Pb 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.[43] 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 blood Pb 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 blood Pb 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 blood Pb 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 blood Pb concentration) 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 blood Pb concentration).

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[42] 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, consistent with advice from CASAC (Henderson, 2007b).

[43] The two log-linear C-R functions rely on the loglinear model in Lanphear et al (2005). The two dual linear C-R functions rely on the linear models reported in Lanphear et al (2005) for concurrent blood Pb in four groupings of children based on whether peak blood Pb levels were below or at/above 10 or 7.5 μg/dL. In order to utilize the linear models in the REA, we considered the relationship between peak and concurrent blood Pb levels in the pooled dataset, as well as in the cohort comprising the bulk of the low blood Pb subsets. In both cases, the difference was approximately a factor of two (2007 REA, section 5.3.1.1). The "dual linear –stratified at 10 μg/dL" function applies the Lanphear et al (2005) linear coefficient for children with peak blood Pb below 10 μg/dL to REA concurrent blood Pb estimates below 5 ug/dL and the linear coefficient for children with peak blood Pb at or above 10 μg/dL to REA concurrent blood Pb estimates at or above 5 μg/dL (Figure 3-4). The "dual linear –stratified at 7.5 μg/dL" function applies the Lanphear et al (2005) linear coefficient for children with peak blood Pb below 7.5μg/dL to REA concurrent blood Pb estimates below 3.75 μg/dL and the linear coefficient for children with peak blood Pb at or above 7.5 μg/dL to REA concurrent blood Pb estimates at or above 3.75 μg/dL.

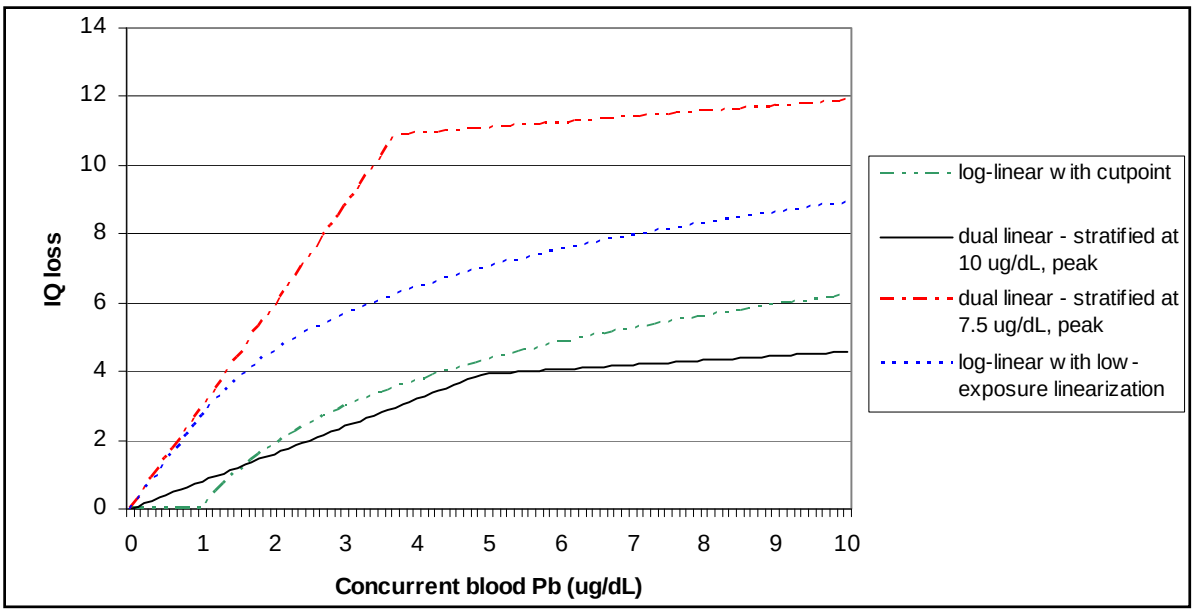

Figure 3-4. Comparison of four concentration-response functions used in risk assessment.

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.

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

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

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

As noted in section 3.2 above, since the completion of the ISA in the current review, two errors have been identified with the pooled dataset analyzed by Lanphear et al (2005) (Kirrane and Patel, 2014). A recent publication and EPA have separately recalculated the statistics and mathematical models of Lanphear et al (2005) using the corrected pooled dataset (Kirrane and Patel, 2014). While the conclusions drawn from these coefficients, including the finding of a steeper slope at lower (as compared to higher) blood Pb concentrations are unaffected, the magnitude of the loglinear and linear regression coefficients are somewhat lower based on the corrections. For example, the loglinear model coefficient used for the LLL function, which we focused on in the last review and also focus on here, changed only negligibly from -2.7 to -2.65 when recalculated using the corrected pooled dataset (Kirrane and Patel, 2014). As a result, the risk estimates for this function described below and presented in Tables 3-10 and 3-11 would be expected to be very similar although slightly lower if derived using the recalculated loglinear model coefficient for the corrected dataset.[44, 45]

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

- *Population risk percentiles:* The IQ loss associated with policy-relevant exposure pathways for specific percentiles of the child population (e.g., the 50th, 90th and 95th percentile modeled child). This category of metric provides perspective on the distribution of IQ loss resulting from policy-relevant exposure pathways, ranging from the typical or average child (50th percentile, mean) to children experiencing higher exposures (90th, 95th percentiles). Greater emphasis has been placed on the median IQ loss estimates due to increased confidence in these estimates relative to the higher percentile estimates, as noted in section 3.4.7.

[44] Since the loglinear model coefficient calculated from the corrected dataset is unchanged at two significant figures from that original reported, any change to the risk estimates would be very small and, particularly in light of other uncertainties in the analysis, does not materially affect staff's consideration of the results.

[45] We also note that risk estimates for the other three C-R functions would also be expected to change as a result of corrections to two of the linear model coefficients (Kirrane and Patel, 2014), such that the upper end of the risk estimates range presented parenthetically in Tables 3-10 and 3-11 for all four functions would also be expected to be somewhat lower (the upper end is generally based on estimates from the dual linear-stratified at 7.5 μg/dL, peak, function). As was the case in the last review, the ranges reflecting all four functions are not a focus in this review.

- *Child frequency counts associated with specific risk percentiles*: Number of children associated with each of the population percentiles (e.g., the number of children predicted to have risk levels at or above the 95th percentile). This risk metric provides a perspective on the number of children associated with various levels of IQ loss for a particular case study.

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

3.4.3.3.2 Air Quality Scenarios Reflecting the Current Standard

As the 2007 REA did not include an air quality scenario simulated to just meet the standard selected by the 2008 decision,[46] we have considered two approaches for identifying risk estimates pertaining to conditions just meeting the current Pb standard (set in 2008) for our purposes in this review. We first reviewed all the scenarios analyzed in the 2007 REA and recognize the similarity to the current standard of the then-current conditions scenario for the Chicago case study. Accordingly, we consider the risk estimates for that scenario for our purposes in this review of considering risk associated with the current standard (see section 3.4.5 below). Additionally, in recognition of the variation among specific locations and urban areas with regard to air quality patterns and exposed population, we have also newly developed estimates for an air quality scenario just meeting the current Pb NAAQS in the context of the generalized (local) urban case study to augment the risk information available in this current review. The newly developed estimates were derived based on interpolation from the risk estimates available for scenarios previously assessed for the generalized (local) urban case study. Such interpolated estimates were only developed for the generalized urban case study due to its use of a single exposure zone which greatly simplified the method employed, thus contributing relatively lesser uncertainty from the interpolation step.[47]

In newly developing estimates for the current standard in the generalized (local) urban case study, the general approach we followed was to identify the two alternative standard

[46] 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).

[47] 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.

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 used as IEUBK model inputs for the various air quality scenarios. 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) with the use of currently available monitoring data 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 $\mu g/m^3$, as a not-to-be-exceeded 3-month average) was found to be bracketed by the scenarios for alternative standards of 0.20 $\mu g/m^3$ (maximum calendar quarter average) and 0.20 $\mu g/m^3$ (maximum monthly average). A risk estimate for the current standard was then derived using the slope relating general urban case study IQ loss to 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 in Appendix 3A. The interpolated median estimates of risk for the current standard for the generalized (local) urban case study are provided below in Table 3-9.

3.4.4 Challenges in Characterizing Air-related Exposure and Risk

In estimating the portion of total (all-pathway) blood Pb 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.[48] 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.

[48] 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. Fore 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.

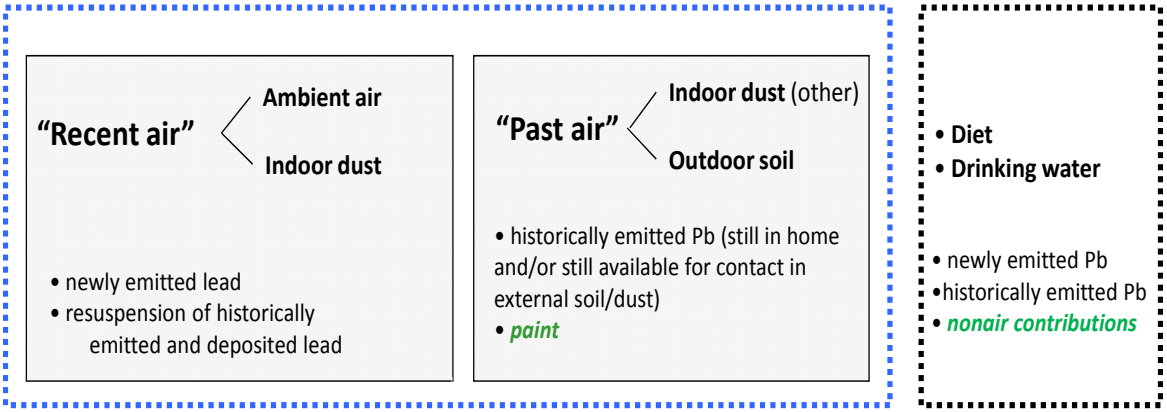

Figure 3-5. Parsing of air-related risk estimates.

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

Among the limitations affecting our estimates for the air-related categories is the apportionment of nonair pathways. For example, while conceptually indoor Pb paint contributions to indoor dust Pb would be considered background and included in a "background" category for this assessment, due to technical limitations related to indoor dust Pb modeling, dust from Pb paint was included as part of "other" indoor dust Pb (i.e., as part of past air exposure). The inclusion of indoor paint Pb as a component of "other" indoor dust Pb (and consequently as a component of the "past air" category) represents a source of potential high bias in our prediction of exposure and risk associated with the "past air" category because indoor paint Pb is a nonair Pb source. At the same time, Pb in ambient air does contribute to the drinking water

and diet exposure pathways and is likely a substantial contribution to diet. We could not separate the air contribution from the nonair contributions in the drinking water and diet pathways. As a result, our risk estimate for the drinking water/diet category of pathways includes some air-related risk representing a source of potential low bias in our predictions of air-related risk.

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

In summary, because of limitations in the assessment design, data and modeling tools, our risk estimates for the "past air" category in the last review include both risks that are truly air-related and potentially, some nonair risk. Because we could not sharply separate Pb linked to ambient air from Pb from other (nonair) sources, some of the three categories of risk are underestimated and others overestimated. On balance, we believe this limitation leads to a slight overestimate of the risks in the "past air" category. At the same time, as discussed above, the "recent air" category does not fully represent the risk associated with all air-related pathways. Thus, we considered the risk attributable to air-related exposure pathways to be bounded on the low end by the risk estimated for the "recent air" category and on the upper end by the risk estimated for the "recent air" plus "past air" categories. With regard to the latter, we are additionally cognizant of the modeling and data limitations which reduce the extent to which the upper end of these bounds reflects impacts of alternative air quality conditions simulated.

3.4.5 Risk Estimates

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

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

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

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

- The risk estimates represent IQ loss associated with air-related Pb exposure for the median child (exposure is modeled through age seven years).
- Our estimation of risk attributable to air-related exposure pathways is approximate, as described in sections 3.4.4 and 3.4.7. We consider the air-related risk to fall within the ranges presented, bounded on the low end by estimates for the pathways categorized as "recent air" and on the upper end by the sum of the estimates for both the "recent" and "past air" pathways. [49]
- The bolded range of risk estimates is derived using the C-R function in which we have the highest overall confidence (the log-linear with low-exposure linearization – see section 3.4.3.3.1). The wider range of risk estimates presented within the parentheses in both tables reflects the application of all four C-R functions (see discussion in section 3.4.3.3.1) to exposure estimates for the "recent" and "recent" plus "past" air categories.

[49] The third category of pathways for which risk was estimated in the 2007 REA comprised 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.

Consequently, this range of risk estimates reflects both uncertainty in estimation of air-related exposures as well as uncertainty in the C-R function for IQ loss in children.

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

Air Quality Scenario Just meeting specified maximum quarterly/monthly average (μg/m³)	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)

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 C-R function in which we have the highest overall confidence (the log-linear with low-exposure linearization). Values in parentheses reflect the range of estimates associated with all four concentration-response functions (see discussion in section 3.4.3.3.1).

B – In the case of the primary Pb smelter case study, only recent plus past air estimates are available.

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).

D – This corresponds to roughly 0.7 - 1.0 μg/m³ maximum monthly mean, across the urban case studies

E - A "roll-up" was performed so that the highest monitor in the study area is increased to just meet this level.

F – A "roll-up" to this level was not performed.

G – A "roll-up" to this level was not performed; these estimates are based on current conditions in this area (0.17 max monthly).

The Information in this table is drawn from the 2007 REA, Table 5-9.

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

Air Quality Scenario Just meeting specified metric (μg/m³)			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 estimates are generated using the C-R function in which we have the highest overall confidence (the log-linear with low-exposure linearization). Values in parentheses reflect the range of estimates associated with all four concentration-response functions (see discussion in section 3.4.3.3.1). Values in parentheses reflect the range of estimates associated with all four concentration-response functions.
B – Risk estimates interpolated - see text.

Key observations regarding these air-related risk estimates **across the array of air quality scenarios** include the following:

All Case Studies

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

Generalized (local) Urban Case Study

- As described above, the general urban case study provides risk estimates for a single group of children residing in a single area where air concentrations throughout area are near the level of the standard being simulated. In this case study, air-related median IQ

loss, based on the higher confidence C-R function (bold), ranged from roughly 2 to 4 IQ points for the 0.5 $\mu g/m^3$, maximum monthly average scenario, to <1 to roughly 3 IQ points for the 0.02 $\mu g/m^3$, maximum monthly average scenario. These ranges are expanded somewhat with consideration of the full range of C-R 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 $\mu g/m^3$, as a maximum monthly average. Consequently, risk estimates for these case studies indicate little response to alternative standard levels above 0.05$\mu g/m^3$ 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 the dust Pb model used for this case study.[50] 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 the 2007 REA, sections 3.1.4).

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

- The median air-related IQ loss estimate for then-current conditions in the Chicago study area, which just met a level of 0.14 $\mu g/m^3$ 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 $\mu g/m^3$ maximum quarterly and 0.2 $\mu g/m^3$ maximum monthly average scenarios that were derived in 2007 REA. The newly interpolated estimate is essentially the same as the estimate for 0.2 $\mu g/m^3$ 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.

[50] 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, we address the following question.

- **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 U.S. Census block group level. Each block group was associated ambient air Pb exposure based on proximity to the monitor nearest to block group centroid (2007 REA, 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 Blood Pb 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 blood Pb levels for that central-tendency child within each exposure zone. These central-tendency blood Pb levels were then combined with a GSD reflecting variability in blood Pb levels for young children (at/near age 7 years) to generate a distribution of total blood Pb 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 blood Pb 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 blood Pb levels (and consequently to

total Pb-attributable risk). This is particularly true for high-end population percentile estimates of IQ loss.

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

3.4.7 Characterizing Uncertainty

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

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

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

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

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

- *Temporal Aspects:* During the 7-year exposure period, media concentrations remain fixed and the simulated child resides at the same residence (although exposure factors, including behavioral and physiological parameters, are adjusted to match the aging of the child). While these aspects of simulation introduce uncertainty into the risk estimates, it is not clear whether there is a directional bias. For example, failure to consider population mobility during the simulation period could bias the overall risk distribution upwards unless children move to or from a residential location with higher Pb exposure, in which case this would not be the case.
- *Generalized (local) Urban Case Study:* The design for this case study employs assumptions regarding uniformity that are reasonable in the context of a general description of a small neighborhood population but would contribute significant uncertainty to extrapolation of these estimates to a specific urban location, particularly a relatively large one. Thus, the risk estimates for this case study, while generally representative of an urban residential population exposed to the specified ambient air Pb levels, cannot be readily related to a specific large urban population. As long as these important caveats are considered in interpreting the risk estimates (i.e., these risk estimates likely represent a relatively small portion of the resident child population in any given city just meeting specific air quality conditions), then the potential error of extrapolating these results to a larger child population can be avoided. An additional area of uncertainty is with regard to the representation of variability in air quality. Given the relatively greater variability common in areas of high Pb concentrations, the approach used to reflect variability may bias the estimates high, although there is uncertainty with regard to the representativeness of the monitoring dataset used to characterize this.
- *Location-specific Urban Case Studies:* Limitations in the spatial density of ambient air monitors in the three metropolitan areas simulated limit our characterization of spatial gradients of ambient air Pb levels in these case studies. While this factor introduces uncertainty into the risk estimates for this category of case study, it is not clear whether there is a directional bias.
- *Air Quality Simulation:* The proportional roll-up and roll-down procedures used in some case studies to simulate air quality conditions just meeting the previous NAAQS and alternative NAAQS, respectively, assume proportional changes in air concentrations across those case study areas to create those air quality scenarios. The EPA recognizes the uncertainty with our simulation of higher air Pb concentrations that would just meet the previous (1978) NAAQS in the urban location-specific case studies, as well as the uncertainty in simulation of conditions associated with the implementation of emissions reduction actions to meet a lower standard. There is the potential that the use of the proportional approach for adjusting monitor values introduced a degree of high bias into estimates of risk reduction. This could result if we find that urban areas can target specific Pb sources impacting individual monitors, thereby avoiding the need for more generalized reduction strategies resulting in a uniform pattern of reduction across all monitors in an urban area. It is important to point out, however, that the generalized urban case study is not affected by this potential bias, since, given the single exposure

zone involved, the single ambient air Pb level is fixed to reflect meeting the standard being considered (i.e., no spatial gradient is simulated in the small, localized residential area being modeled).

- *Outdoor Soil/Dust Pb Concentrations:* Limitations in datasets on Pb levels in surface soil/dust Pb in urban areas and in our ability to simulate the impact of reduced air Pb levels related to lowering the NAAQS contribute uncertainty to air-related risk estimates. In this case, it is likely that we have low biased our estimates of risk reduction associated with alternative (lower) Pb NAAQS levels, since we have not simulated potential changes in soil Pb related to changes in ambient air Pb.
- *Indoor Dust Pb Concentrations:* Limitations and uncertainty in modeling of indoor dust Pb levels, including the impact of reductions in ambient air Pb levels, contributes uncertainty to air-related risk estimates. Although our modeling of indoor dust does link changes in ambient air Pb to changes in indoor dust Pb (via air exchanges and indoor deposition onto surfaces), the modeling does not include a link between ambient air Pb, outdoor soil Pb and subsequent changes in the level of Pb carried (or "tracked") into the house. This could introduce low bias into our total estimates of air-related Pb exposure and risk.
- *Interindividual Variability in Blood Pb Levels:* Uncertainty related to population variability in blood Pb levels (i.e., interindividual variability in factors other than media concentration that influence blood Pb) and limitations in modeling of this introduces significant uncertainty into blood Pb and IQ loss estimates for the 95th percentile of the population. We are not aware of any systematic bias introduced into the analysis from this source of uncertainty.
- *Pathway Apportionment for Higher Percentile Blood Pb and risks:* Limitations, primarily in data, prevented us from characterizing the degree of correlation among high-end Pb exposures for the various pathways (e.g., the degree to which an individual experiencing high drinking water Pb exposure would also experience high Pb paint exposure and high ambient air-related Pb exposure). Our inability to characterize potential correlations between exposure pathways (particularly at the higher percentile exposure levels) limited our ability to (a) effectively model high-end Pb risk and (b) apportion that risk between different exposure pathways, including ambient air-related pathways.
- *IQ Loss Concentration-response Functions:* Specification of the quantitative relationship between blood Pb level and IQ loss is subject to greater uncertainty at lower blood Pb levels (e.g., particularly below 2.5 µg/dL concurrent blood Pb). However, we believe that by considering four different models (which each treat the response at low blood Pb levels in a different manner), we have completed a reasonable characterization of this source of uncertainty and its impact on risk estimates. Given comparison of risk estimates generated using the four models, it would appear that this source of 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 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 different[51] 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 identified 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 in the 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, roughly estimated as 3 and 1 IQ points, which over- and underestimate risk, 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 $\mu g/m^3$ scenarios (maximum monthly and quarterly averages) simulated in the 2007 REA[52] and are

[51] 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.

[52] 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.

appreciably lower than those associated with the previous standard. In characterizing the magnitude of air-related risk associated with the current standard, we focus on median estimates, for which we have appreciably greater confidence than estimates for outer ends of risk distribution (see section 3.4.7) and on risks derived using the C-R function in which we have greatest confidence (see sections 3.4.3.3.1 and 3.4.7). The risk results for the current standard estimated in the last review for one of the location-specific urban study area populations and those newly derived in this review using interpolation of the estimates from the last review for the generalized (local) urban case study, which is recognized to reflect a generalized high end of air-related exposure for localized populations, provide approximate bounds for air-related risk, with attendant uncertainties described above.

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4 REVIEW OF THE PRIMARY STANDARD FOR LEAD

This chapter presents staff conclusions regarding the primary Pb standard. These 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. The evaluations and staff conclusions presented in this chapter have also been developed with consideration of CASAC advice and public comment on the external review draft of this document. These 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, 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 last Pb NAAQS review addressing key policy-relevant questions in light of currently available scientific and technical information. 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. In drawing conclusions for consideration with regard to the primary standard, 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 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 requirements of the NAAQS provisions of the Act and with how the EPA and the courts have historically interpreted the Act. These provisions require the Administrator to establish primary standards that, in the judgment of the Administrator, are requisite to protect public health with an adequate margin of safety. In so doing, the Administrator seeks to establish standards that are neither more nor less stringent than necessary for this purpose. The Act does not require that primary standards be set at a zero-risk level, but rather at a level that avoids unacceptable risks to public health including the health of sensitive groups.[1] The four basic elements of the NAAQS (indicator, averaging time, level, and form) are considered collectively in evaluating the health protection afforded by the current standard.

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

4.1.1 Approach Used in the Last Review

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

While recognizing that Pb has been demonstrated to exert "a broad array of deleterious effects on multiple organ systems", the review focused on the effects most pertinent to ambient air exposures, which given ambient air Pb reductions over the past 30 years are those associated with relatively lower exposures and associated blood Pb levels (73 FR 66975). In so doing, the

[1] The at-risk population groups identified in a NAAQS review may include low income or minority groups. Where low income/minority groups are among the at-risk populations, the rulemaking decision will be based on providing protection for these and other at-risk populations and lifestages (e.g., children, older adults, persons with pre-existing heart and lung disease). To the extent that low income/minority groups are not among the at-risk populations identified in the ISA, a decision based on providing protection of the at-risk lifestages and populations would be expected to provide protection for the low income/minority groups.

EPA recognized the general consensus that the developing nervous system in children is among, if not the most sensitive health endpoint associated with Pb exposures. Thus, primary attention was given to consideration of nervous system effects, including neurocognitive and neurobehavioral effects, in children (73 FR 66976). The body of evidence included associations of such effects in study populations of variously-aged children with mean blood Pb levels below 10 µg/dL, extending from 8 down to 2 µg/dL (73 FR 66976). The public health implications of effects of air-related Pb on cognitive function (e.g., IQ) in young children were given particular focus in the review.

4.1.1.1 Approach Regarding the Need for Revision

The conclusions reached by the Administrator in the last review were based primarily on the scientific evidence, with the risk- and exposure-based information providing support for various aspects of the decision. In reaching his conclusion on the adequacy of the then-current standard, which was set in 1978, the Administrator placed primary consideration on the large body of scientific evidence available in the review including significant new evidence concerning effects at blood Pb concentrations substantially below those identified when the standard was initially set (73 FR 66987; 43 FR 46246). Given particular attention was the robust evidence of neurotoxic effects of Pb exposure in children, recognizing: (1) that while blood Pb levels in U.S. children had decreased notably since the late 1970s, newer epidemiological studies had investigated and reported associations of effects on the neurodevelopment of children with those more recent lower blood Pb levels and (2) that the toxicological evidence included extensive experimental laboratory animal evidence substantiating well the plausibility of the epidemiological findings observed in human children and expanding our understanding of likely mechanisms underlying the neurotoxic effects (73 FR 66987). Additionally, within the range of blood Pb levels investigated in the available evidence base, a threshold level for neurocognitive effects was not identified (73 FR 66984; 2006 CD, p. 8-67). Further, the evidence indicated a steeper dose-response relationship for effects on cognitive function at those lower blood Pb levels than at higher blood Pb levels that were more common in the past, "indicating the potential for greater incremental impact associated with exposure at these lower levels" (73 FR 66987). As at the time when the standard was initially set in 1978, the health effects evidence and exposure/risk assessment available in the last review supported the conclusion that air-related Pb exposure pathways contribute to blood Pb levels in young children by inhalation and ingestion (73 FR 66987). The available information in the last review also indicated, however, that the air-to-blood ratio was likely larger than the air-to-blood ratio (of 2 µg/dL blood Pb to 1 $\mu g/m^3$ air Pb) estimated when the standard was initially set (73 FR 66987).

In the Administrator's decision on the adequacy of the 1978 standard, the Administrator considered the evidence using a very specifically defined framework, referred to as an air-related IQ loss evidence-based framework. This framework integrates evidence for the relationship between Pb in air and Pb in young children's blood with evidence for the relationship between Pb in young children's blood and IQ loss (73 FR 77987), as described in more detail in section 4.1.1.2 below. This evidence-based approach considers air-related effects on neurocognitive function (using the quantitative metric of IQ loss) associated with exposure in those areas with elevated air concentrations equal to potential alternative levels for the Pb standard. Thus, the conceptual context for the framework is that it provides estimates of air-related IQ loss for a subset of the population of U.S. children (i.e., the subset living in close proximity to air Pb sources that contribute to elevated air Pb concentrations that equal the current level of the standard). This is the subset expected to experience air-related Pb exposures at the high end of the national distribution of such exposures, not at the average. This is the case since when a standard of a particular level is just met at a monitor sited to record the highest source-oriented concentration in an area, the large majority of children in the larger surrounding area would likely experience exposures to concentrations well below that level.

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

Based on the application of the air-related IQ loss framework to the evidence, the Administrator concluded that, for exposures projected for air Pb concentrations at the level of the 1978 standard, the quantitative estimates of IQ loss associated with air-related Pb indicated risk of a magnitude that in his judgment was significant from a public health perspective, and that the evidence-based framework supported a conclusion that the 1978 standard did not protect public health with an adequate margin of safety (73 FR 77987). The Administrator further concluded that the evidence indicated the need for a substantially lower standard level to provide increased public health protection, especially for at-risk groups (most notably children), against an array of

effects, most importantly including effects on the developing nervous system (73 FR 77987). In addition to giving primary consideration to the much expanded evidence base since the standard was set, the Administrator also took into consideration the exposure/risk assessments. In so doing, he observed that, while taking into consideration their inherent uncertainties and limitations, the quantitative estimates of IQ loss associated with air-related Pb in air quality scenarios just meeting the then-current standard also indicated risk of a magnitude that in his judgment was significant from a public health perspective. Thus, the Administrator concluded the exposure/risk estimates provided additional support to the evidence-based conclusion that the standard needed revision (73 FR 66987).

4.1.1.2 Approach Regarding Elements of Revised Standard

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

With regard to indicator, consideration was given to replacing Pb-TSP with Pb-PM_{10}. The EPA recognized, however, that Pb in all particle sizes contributes to Pb in blood and associated health effects, additionally noting that the difference in particulate Pb captured by TSP and PM_{10} monitors may be on the order of a factor of two in some areas (73 FR 66991). Further, the Administrator recognized uncertainty with regard to whether a Pb-PM_{10}-based standard would also effectively control ultra-coarse[2] Pb particles, which may have a greater presence in areas near sources where Pb concentrations are highest (73 FR 66991). The Administrator decided to retain Pb-TSP as the indicator to provide sufficient public health protection from the range of particle sizes of ambient air Pb, including ultra-coarse particles (73 FR 66991). Additionally, a role was provided for Pb-PM_{10} in the monitoring required for a Pb-TSP standard (73 FR 66991) based on the conclusion that use of Pb-PM_{10} measurements at sites not influenced by sources of ultra-coarse Pb, and where Pb concentrations are well below the standard, would take advantage of the increased precision of these measurements and decreased spatial variation of Pb-PM_{10} concentrations, without raising the same concerns over a lack of protection against health risks from all particulate Pb emitted to the ambient air that support retention of Pb-TSP as the indicator (*versus* revision to Pb-PM_{10}) (73 FR 66991). Accordingly, allowance was made for the use of Pb-PM_{10} monitoring for Pb NAAQS attainment purposes in certain limited circumstances, at non-source-oriented sites, where the Pb concentrations are

[2] The term ultra-coarse is used to refer to particles collected by a TSP sampler but not by a PM_{10} 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 PM_{10} 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.

expected to be substantially below the standard and ultra-coarse particles are not expected to be present (73 FR 66991).

With regard to averaging time and form for the revised standard, consideration was given to a monthly averaging time, with a form of second maximum, and a 3-month calendar quarter averaging time, with not-to-be exceeded forms. While the Administrator recognized that there were some factors that might imply support for a period as short as a month for averaging time, he also noted other factors supporting use of a longer time. He additionally took note of the complexity inherent in this consideration for the primary Pb standard, which is greater than in the case of other criteria pollutants due to the multimedia nature of Pb and its multiple pathways of human exposure.

In this situation for Pb, the Administrator emphasized the importance of considering all of the relevant factors, both those pertaining to the human physiological response to changes in Pb exposures and those pertaining to the response of air-related Pb exposure pathways to changes in airborne Pb, in an integrated manner. With regard to the human physiological response to changes in Pb exposures, the evidence discussed in the CD indicated that children's blood Pb levels respond quickly to increased Pb exposures, such that an abrupt increase in Pb uptake results in increased blood Pb levels. Contributing to this response is the absorption through the lungs and the gastrointestinal tract and the rapid distribution, once absorbed, throughout the body. With regard to the relationship between airborne Pb and children's blood Pb, evidence collected during the time of leaded gasoline usage when airborne Pb was a prominent Pb exposure pathway across the population indicated children's blood Pb to respond to changes in airborne Pb over a month's time lag. The phase-out of on-road leaded gasoline, however, has resulted in changed circumstances with regard to children's exposure pathways, with accompanying temporal implications. Accordingly, EPA considered the limited evidence indicating the more numerous factors influencing ingestion (versus inhalation) pathways, which were considered likely to lessen the impact of month-to-month variations in airborne Pb concentrations on levels of air-related Pb in children's blood. Such factors were considered likely to lead to response times (e.g., for the response of blood to air Pb via these pathways) extending longer than a month (73 FR 66996). The Administrator also recognized limitations and uncertainties in the evidence including the limited available evidence specific to the consideration of the particular duration of sustained airborne Pb levels having the potential to contribute to the adverse health effects identified as most relevant to this review, as well as variability in the response time of indoor dust Pb loading to ambient airborne Pb. Based on these various considerations, the Administrator concluded that the information provided support for an averaging time no longer than a 3-month period.

With regard to a three-month averaging time, the EPA recognized that a rolling three-month averaging time can provide (e.g., compared to a block calendar quarter) control on month-to-month variability in air Pb concentrations and in associated exposures (73 FR 66996). The rolling three-month average eliminates the possibility for two consecutive "high" months falling in two separate calendar quarters to be considered independently (perhaps being mitigated by "low" months falling in each of the same calendar quarters). Rather, in the rolling 3-month approach, the same month would contribute to three different 3-month periods through separate combinations with three different pairs of months, thus providing a more complete consideration of air quality during that month and the 3-month periods in which it falls. The Administrator additionally concluded it appropriate to modify the method by which the 3-month average metric is derived, to be the average of three monthly average concentrations, as compared to the then-current practice by which the average was derived across the full dataset for a quarter without equally weighting each month within the quarter. Thus, in consideration of the uncertainty associated with the evidence pertinent to averaging time discussed above, the Administrator noted that the two changes in form for the standard (to a rolling 3-month average and to providing equal weighting to each month in deriving the 3-month average) both afford greater weight to each individual month than did the calendar quarter form of the 1978 standard, tending to control both the likelihood that any month will exceed the level of the standard and the magnitude of any such exceedance.

Based on this integrated consideration of the range of relevant factors, the averaging time was revised to a rolling three-month period with a maximum (not-to-be-exceeded) form, evaluated over a three-year period. As compared to the previous averaging time and form of calendar quarter (not-to-be exceeded), this revision was considered to be more scientifically appropriate and more health protective (73 FR 77996). The rolling average gives equal weight to all three-month periods, and the new calculation method gives equal weight to each month within each three-month period (73 FR 77996). Further, the rolling average yields 12 three-month averages each year to be compared to the NAAQS versus four averages in each year for the block calendar quarters pertaining to the previous standard (73 FR 77996).

Lastly, based on the body of scientific evidence and information available, as well as CASAC recommendations and public comment, the Administrator decided on a standard level that, in combination with the specified choice of indicator, averaging time, and form, he judged requisite to protect public health, including the health of sensitive groups, with an adequate margin of safety (73 FR 67006). In reaching the decision on level for the revised standard, the Administrator considered as a useful guide the evidence-based framework developed in that review. As described in section 4.1.1.1 above, that framework integrates evidence for the relationship between Pb in air and Pb in children's blood and the relationship between Pb in

children's blood and IQ loss. Application of the air-related IQ loss evidence-based framework was recognized, however, to provide "no evidence- or risk-based bright line that indicates a single appropriate level" for the standard (73 FR 67006). Rather, the framework was seen as a useful guide for consideration of health risks from exposure to ambient levels of Pb in the air, in the context of a specified averaging time and form, with regard to the Administrator's decision on a level for a revised NAAQS that provides public health protection that is sufficient but not more than necessary under the Act (73 FR 67004).

As noted above, use of the evidence-based air-related IQ loss framework to inform selection of a standard level involved consideration of the evidence with regard to two input parameters. The two input parameters are an air-to-blood ratio and a C-R function for population IQ response associated with blood Pb level (73 FR 67004). The evidence at the time of the last review indicated a broad range of air-to-blood ratio estimates, each with limitations and associated uncertainties. Based on the then-available evidence, the Administrator concluded that 1:5 to 1:10 represented a reasonable range to consider and identified 1:7 as a generally central value on which to focus (73 FR 67004). With regard to C-R functions, in light of the evidence of nonlinearity and of steeper slopes at lower blood Pb levels, the Administrator concluded it was appropriate to focus on C-R analyses based on blood Pb levels that most closely reflected the then-current population of children in the U.S.,[3] recognizing EPA's identification of four such analyses and giving weight to the central estimate or median of the resultant C-R functions (73 FR 67003, Table 3; 73 FR 67004). The four study groups from which C-R functions were drawn in 2008, and the associated C-R slopes, are summarized in Table 3-3 above.[4] The median estimate of -1.75 IQ points decrement per µg/dL was selected for use with the framework. With the framework, as summarized in section 4.1.1.1 above, potential alternative standard levels (µg/m^3) are multiplied by estimates of air-to-blood ratio (µg/dL blood Pb per µg/m^3 air Pb) and the median slope for the C-R function (points IQ decrement per µg/dL blood Pb), yielding estimates of a mean air-related IQ decrement for a specific subset of young children (i.e., those children exposed to air-related Pb in areas with elevated air Pb concentrations equal to specified alternative levels). As such, the application of the framework yields estimates for the mean air-related IQ decrements of the subset of children expected to experience air-related Pb exposures

[3] 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 µg/dL and the 5th and 95th percentiles were 0.7 µg/dL and 5.1 µg/dL, respectively (73 FR 67002).

[4] 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.

at the high end of the distribution of such exposures. The associated mean IQ loss estimate is the average for this highly exposed subset and is not the average air-related IQ loss projected for the entire U.S. population of children. Uncertainties and limitations were recognized in the use of the framework and in the resultant estimates (73 FR 67000).

In considering the use of the evidence-based air-related IQ loss framework to inform his judgment as to the appropriate degree of public health protection that should be afforded by the NAAQS to provide requisite protection against risk of neurocognitive effects in sensitive populations, such as IQ loss in children, the Administrator recognized in the 2008 review that there were no commonly accepted guidelines or criteria within the public health community that would provide a clear basis for such a judgment. During the 2008 review, CASAC commented regarding the significance from a public health perspective of a 1-2 point IQ loss in the entire population of children and, along with some commenters, emphasized that the NAAQS should prevent air-related IQ loss of a significant magnitude, such as on the order of 1-2 IQ points, in all but a small percentile of the population. Similarly, the Administrator stated that "ideally air-related (as well as other) exposures to environmental Pb would be reduced to the point that no IQ impact in children would occur" (73 FR 66998). The Administrator further recognized that, in the case of setting a NAAQS, he was required to make a judgment as to what degree of protection is requisite to protect public health with an adequate margin of safety (73 FR66998). The NAAQS must be sufficient but not more stringent than necessary to achieve that result, and the Act does not require a zero-risk standard (73 FR 66998). The Administrator additionally recognized that the evidence-based air-related IQ loss framework did not provide estimates pertaining to the U.S. population of children as a whole. Rather, the framework provides estimates (with associated uncertainties and limitations) for the mean of a subset of that population, the subset of children assumed to be exposed to the level of the standard. As described in the final decision "[t]he framework in effect focuses on the sensitive subpopulation that is the group of children living near sources and more likely to be exposed at the level of the standard" (73 FR 67000). As further noted in the final decision (73 FR 67000):

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

In reaching a judgment as to the appropriate degree of protection, the Administrator considered advice and recommendations from CASAC and public comments and recognized the

uncertainties in the health effects evidence and related information as well as the role of, and context for, a selected air-related IQ loss in the application of the framework, as described above. Based on these considerations, the Administrator identified an air-related IQ loss of 2 points for use with the framework, as a tool for considering the evidence with regard to the level for the standard (73 FR 67005). In so doing, the Administrator was not determining that such an IQ decrement value was appropriate in other contexts (73 FR 67005). Given the various uncertainties associated with the framework and the scientific evidence base, and the focus of the framework on the sensitive subpopulation of children that are more highly exposed to air-related Pb, a standard level selected in this way, in combination with the selected averaging time and form, was expected to significantly reduce and limit for a high percentage of U.S. children the risk of experiencing an air-related IQ loss of that magnitude (73 FR 67005). At the standard level of 0.15 $\mu g/m^3$, with the combination of the generally central estimate of air-to-blood ratio of 1:7 and the median of the four C-R functions (-1.75 IQ point decrement per µg/dL blood Pb), the framework estimates of air-related IQ loss were below 2 IQ points (73 FR 67005, Table 4).

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

Based on consideration of the entire body of evidence and information available in the review, as well as the recommendations of CASAC and public comments, the Administrator decided that a level for the primary Pb standard of 0.15 $\mu g/m^3$, in combination with the specified choice of indicator, averaging time and form was requisite to protect public health, including the health of sensitive groups, with an adequate margin of safety (73 FR 67006). In reaching decisions on level as well as the other elements of the revised standard, the Administrator took note of the complexity associated with consideration of health effects caused by different ambient air concentrations of Pb and with uncertainties with regard to the relationships between air concentrations, exposures, and health effects For example, selection of a maximum, not to be exceeded, form in conjunction with a rolling 3-month averaging time over a 3-year span was expected to have the effect that the at-risk population of children would be exposed below the standard most of the time (73 FR 67005). The Administrator additionally considered the provision of an adequate margin of safety in making decisions on each of the elements of the

standard, including, for example "selection of TSP as the indicator and the rejection of the use of PM_{10} scaling factors; selection of a maximum, not to be exceeded form, in conjunction with a 3-month averaging time that employs a rolling average, with the requirement that each month in the 3-month period be weighted equally (rather than being averaged by individual data) and that a 3-year span be used for comparison to the standard; and the use of a range of inputs for the evidence-based framework, that includes a focus on higher air-to-blood ratios than the lowest ratio considered to be supportable, and steeper rather than shallower C-R functions, and the consideration of these inputs in selection of 0.15 $\mu g/m^3$ as the level of the standard" (73 FR 67007).

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

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

4.1.2 Approach for the Current Review

To evaluate whether it is appropriate to consider retaining the current primary Pb standard, or whether consideration of revision is appropriate, we have adopted an approach in this review that builds upon the general approach used in the last review and reflects the broader body of evidence and information now available. As summarized above, the Administrator's decisions in the prior review were based on an integration of information on health effects

associated with exposure to Pb, relationships between ambient air Pb and blood Pb; expert judgments on the adversity and public health significance of key health effects; and policy judgments as to when the standard is requisite to protect public health with an adequate margin of safety. These considerations were informed by air quality and related analyses, quantitative exposure and risk assessments, and qualitative assessment of impacts that could not be quantified.

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

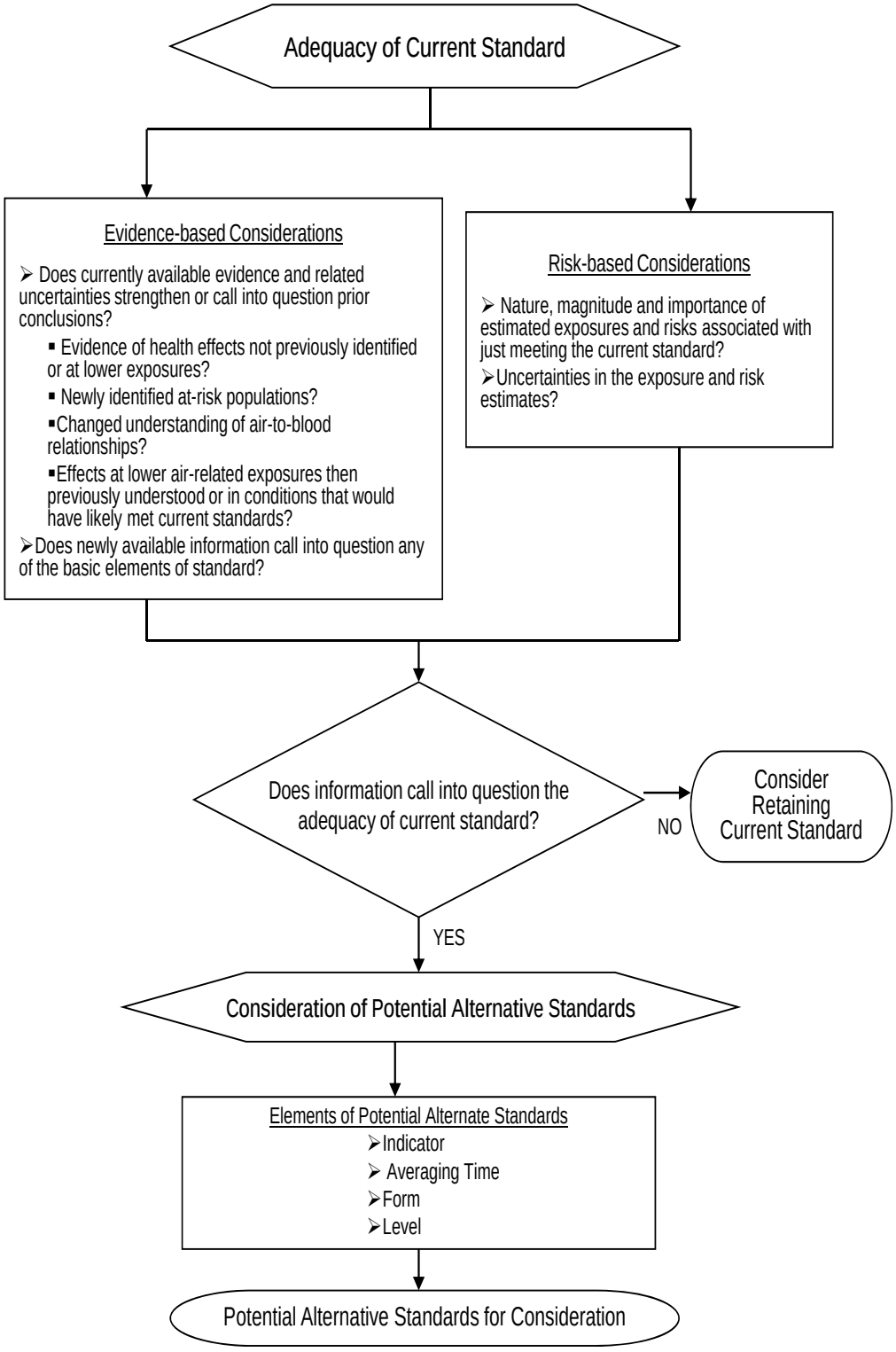

Figure 4-1. Overview of approach for review of current primary standard.

4.2 ADEQUACY OF THE CURRENT STANDARD

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

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

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

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

4.2.1 Evidence-based Considerations

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

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

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

As at the time of the last review, blood Pb continues to be the predominant biomarker employed to assess exposure and health risk of Pb (ISA, chapters 3 and 4), as discussed in section 3.1 above. This widely accepted role of blood Pb in assessing exposure and risk is illustrated by its established use in programs to prevent both occupational Pb poisoning and childhood Pb poisoning, with the latter program, implemented by the CDC, recently issuing updated guidance on blood Pb measurement interpretation (CDC, 2012). As in the past, the current evidence continues to indicate the close linkage of blood Pb levels in young children to their body burden; this linkage is associated with the ongoing bone remodeling during that lifestage (ISA, section 3.3.5). This tight linkage plays a role in the somewhat rapid response of children's blood Pb to changes in exposure (particularly to exposure increases), which contributes to its usefulness as an exposure biomarker (ISA, sections 3.2.2, 3.3.5, and 3.3.5.1). Additionally, the weight of evidence documenting relationships between children's blood Pb and health effects, most particularly those on the nervous and hematological systems (e.g., ISA, sections 4.3 and 4.7), speaks to its usefulness in assessing health risk.

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

available in the last review. The range of estimates that can be derived from the full dataset is broad and not changed by the inclusion of the newly available estimates. Further, we recognize significant uncertainties regarding the air Pb to air-related blood Pb relationship for the current conditions where concentrations of Pb in both ambient air and children's blood are substantially lower than they have been in the past. In considering the strengths, limitations and uncertainties associated with the full dataset, the currently available evidence appears to continue to support a range of estimates for our purposes that is generally consistent with the range given weight in the last review, 1:5 to 1:10 (ISA, section 3.7.4 and Table 3-12; 73 FR 67001-2, 67004). We additionally note that the generally central estimate of 1:7 identified for this range in the last review is consistent with the study involving blood Pb for pre-school children and air Pb conditions near a large source of Pb to ambient air with concentrations near (and/or previously above) the level of the current Pb standard (ISA, section 3.5.1; Hilts, 2003).[5] In so noting, we also recognize the general overlap of such circumstances with those represented by the evidence-based, air-related IQ loss framework,[6] for which air-to-blood ratio is a key input. In characterizing the range of air-to-blood ratio estimates, we recognize uncertainty inherent in such estimates as well as the variation in currently available estimates resulting from a variety of factors, including differences in the populations examined, as well as in the Pb sources or exposure pathways addressed in those study analyses (ISA, section 3.7.4).

The scientific evidence continues to recognize a broad array of health effects on multiple organ systems or biological processes related to blood Pb, including Pb in blood prenatally (ISA, section 1.6). The currently available evidence continues to support identification of neurocognitive effects in young children as the most sensitive endpoint associated with blood Pb concentrations (ISA, section 1.6.1), which as an integrated index of exposure reflects the aggregate exposure to all sources of Pb through multiple pathways (inhalation and ingestion). Evidence continues to indicate that neurocognitive effects in young children may not be reversible and may have effects that persist into adulthood (ISA, section 1.9.6). Thus, as discussed in section 3.2 above, we continue to consider the evidence of Pb effects at the low end of the studied blood Pb levels (closest to those common in the U.S. today) to be strongest and of greatest concern for effects on the nervous system, most particularly those on cognitive function in children.

As in the last review, evidence on risk factors continues to support the identification of young children as an important at-risk population for Pb health effects (ISA, section 5.4). 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.

current evidence also continues to indicate important roles as factors that increase risk of lead-related health effects for the following: nutritional factors, such as iron and calcium intake; elevated blood Pb levels; and proximity to sources of Pb exposure, such as industrial releases or buildings with old, deteriorating, leaded paint. Further, some races or ethnic groups continue to demonstrate increased blood Pb levels relative to others, which may be related to these and other factors (ISA, sections 5.1, 5.2 and 5.4).

With regard to our understanding of the relationship between exposure or blood Pb levels in young children and neurocognitive effects, the evidence in this review, as in the last, does not establish a threshold blood Pb level for neurocognitive effects in young children (ISA, sections 1.9.4 and 4.3.12). The lowest blood Pb levels at which associations with neurocognitive impacts have been observed in pre-school and school age children continue to range down below 5 μg/dL, with the lowest group levels that have been associated with such effects ranging down to 2 μg/dL (ISA, sections 1.6.1 and 4.3.15.1). Additionally, as in the last review, there is evidence that the relationship of young children's blood Pb with neurocognitive impacts, such as IQ, is nonlinear across a wide range of blood Pb, with greater incremental impacts at lower vs. higher blood Pb levels (ISA, sections 1.9.4 and 4.3.12). Accordingly, as in the last review, we continue to focus our interest on C-R relationships from study groups with blood Pb levels closest to those in children in the U.S. today, which are generally lower than epidemiological study groups. The currently available evidence does not identify additional C-R slopes for study groups of young children (e.g., $\leq$ 7 years) with mean blood Pb levels below that of groups identified in the last review, 2.9 – 3.8 μg/dL, as discussed in section 3.2 above (ISA, section 4.3.12). Thus, the blood Pb concentration - IQ response functions or slopes identified in this review for epidemiological study groups of young children with mean blood Pb levels closest to that of children in the U.S. today include the same set recognized at the time of the last review (see Table 3-3 above), the median of which is 1.75 points decrement per μg/dL blood Pb (73 FR, 67003).

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

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

levels studied beyond those available in the last review. As in the last review, the early childhood (e.g., 2 to 6 or 7 years of age) blood Pb levels for which associations with IQ response have been reported continue to extend at the low end of the range to study group mean blood Pb levels of 2.9 to 3.8 μg/dL (e.g., 73 FR 67003, Table 3). The lack of studies considering concentration-response relationships for Pb effects on IQ at lower blood Pb levels contributes to uncertainty regarding the quantitative relationship between blood Pb and IQ response in populations with mean blood Pb levels closer to the most recently available mean for children aged one to five years of age (e.g., 1.17 μg/dL in 2009-2010 [ISA, p. 3-85]). The studies at the blood Pb levels studied, as summarized in section 3.2 above, continue to indicate higher C-R slopes in these groups with lower blood Pb levels than in study groups with higher blood Pb levels (ISA, section 4.3.12).

Further, we recognize important uncertainties in our understanding of the relationship between ambient air Pb concentrations and air-related Pb in children's blood. The evidence newly available in this review has not reduced such key uncertainties. As in the last review, air-to-blood ratios based on the available evidence continue to vary, with our conclusions based on the current evidence generally consistent with the range of 1:5 to 1:10 given emphasis in the last review (73 FR 67002; ISA, section 3.7.4). There continues to be uncertainty regarding the extent to which this range represents the relationship between ambient air Pb and Pb in children's blood (derived from the full set of air-related exposure pathways) and with regard to its reflection of exposures associated with ambient air Pb levels common in the U.S. today and to circumstances reflecting just meeting the current Pb standard (ISA, section 3.7.4). We note additionally the significant uncertainty remaining with regard to the temporal relationships of ambient Pb levels and associated exposure with occurrence of a health effect (73 FR 67005).

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

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

Indicator

The indicator for the current Pb standard is Pb-TSP. Key considerations in retaining this indicator in the last review are summarized in section 4.1.1.2 above. Exposure to Pb in all sizes of particles passing through ambient air can contribute to Pb in blood and associated health effects by a wide array of exposure pathways (ISA, section 3.1). These pathways include the ingestion route, as well as inhalation (ISA, section 3.1), and a wide array of particle sizes play a role in these pathways (ISA, section 3.1.1.1). As at the time of the last review, we continue to recognize the variability of the Pb-TSP FRM in its capture of airborne Pb particles (see section 2.2.1.3.1 above). As in the last review, we also note that an alternative approach for collection of a conceptually comparable range of particle sizes, including ultra-coarse particles, is not yet available, although we take note of activities underway that may remedy that situation in the future, as referenced in section 2.2.1.3.1 above. Additionally, the limited available information regarding relationships between Pb-TSP and Pb in other size fractions indicates appreciable variation in this relationship, particularly near sources of Pb emissions where concentrations and potential exposures are greatest. Thus, the information available in this review does not address previously identified limitations and uncertainties for the current indicator. Nor does the newly available information identify additional limitations or uncertainties.

The evidence available in this review continues to indicate the role of a range of air Pb particle sizes in contributing to Pb exposure (e.g., ISA, section 3.1.1.1) that contributes to Pb in blood and associated health effects. For example, the evidence indicates larger particle sizes for Pb that occurs in soil and house dust and may be ingested as compared to Pb particles commonly occurring in the atmosphere and the size fraction of the latter that may be inhaled (ISA, section 3.1.1.1). Taken together the evidence currently available reinforces the appropriateness of an indicator for the Pb standard that reflects a wide range of airborne Pb particles.

Averaging time and form

The averaging time and form of the standard were revised in the last Pb NAAQS review, based on considerations summarized in section 4.1.1.2 above. The current standard is a not-to-be-exceeded rolling three-month average (CFR 50.16), derived from three monthly averages calculated in accordance with the current data handling procedures (CFR, Appendix R to Part 50). The form is a maximum, evaluated within a three-year period (CFR 50.16). As at the time of the last review, evidence continues to support the importance of periods on the order of three months and the prominent role of deposition-related exposure pathways, with uncertainty associated with characterization of precise time periods associating ambient air Pb with air-related health effects. Relevant factors continue to be those pertaining to the human physiological response to changes in Pb exposures and those pertaining to the response of air-related Pb exposure pathways to changes in airborne Pb. The newly available evidence in this

review does not appreciably improve our understanding of the period of time in which air Pb concentrations would lead to the health effects most at issue in this review. Thus, there continue to be limitations in the evidence to inform our consideration of these elements of the standard and associated uncertainty. However, there is no newly available information that calls into question this element of the current Pb standard.

Level

The level of the current standard is 0.15 $\mu g/m^3$ (CFR 50.16). As described in section 4.1.1 above, this level was selected in 2008 with consideration of, among other factors, an evidence-based air-related IQ loss framework, for which there are two primary inputs: air-to-blood ratios and C-R functions for blood Pb – IQ response in young children. Additionally taken into consideration were the uncertainties inherent in these inputs. Application of the framework also entailed consideration of a magnitude of air-related IQ loss, which as further described in section 4.1.1.2 above, is used in conjunction with this specific framework in light of the framework context, limitations and uncertainties. Additionally, selection of a level for the standard in 2008 was made in conjunction with decisions on indicator, averaging time and form.

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

We next turn to consideration of the primary inputs to the framework: air-to-blood ratios and C-R functions for blood Pb – IQ response in young children. With regard to the former, the limited newly available information assessed in the ISA and discussed in section 3.1 above, is generally consistent with the information in this area that was available at the time of the last review. We additionally recognize the variability and uncertainty associated with quantitative air-to-blood ratios based on this information, as also existed in the last review. As in the last review, we recognize that factors contributing to the variability and uncertainty of these estimates are varied and include aspects of the study populations (e.g., age and Pb exposure

pathways) and the study circumstances (e.g., length of study period and variations in sources of Pb exposure during the study period). We note that the full range of estimates associated with the available evidence is wide and consider it appropriate to give emphasis to estimates pertaining to circumstances closest to those in the U.S. today with regard to ambient air Pb and children's blood Pb concentrations, while recognizing the limitations associated with the available information with regard to this emphasis. With that in mind, we consider the currently available evidence to continue to support the range of estimates concluded in the last review to be most appropriate for the current population of young children in the U.S., in light of the multiple air-related exposure pathways by which children are exposed, in addition to inhalation of ambient air, and of the levels of air and blood Pb common today. Identification of this range also included consideration of the limitations associated with the available information and inherent uncertainties. This range of air-to-blood ratios included 1:10 at the upper end and 1:5 at the lower end. We further recognize that the limited evidence for air Pb and children's blood Pb concentrations closest to those in U.S. today continues to provide support for the Administrator's emphasis in the 2008 decision on the relatively central estimate of 1:7.

With regard to the second input to the evidence-based framework, C-R functions for the relationship of young children's blood Pb with neurocognitive impacts (e.g., IQ decrements), we consider several aspects of the evidence. First, as discussed in section 3.2 above, the currently available information continues to provide evidence that this C-R relationship is nonlinear across the range of blood Pb levels from the higher concentrations more prevalent in the past to lower concentrations more common today. Thus, we continue to consider it particularly appropriate to focus on the evidence from studies with blood Pb levels closest to those of today's population, which in the last review included studies with study group mean blood Pb levels ranging roughly from 3 to 4 μg/dL in children aged 24 months to 7 years (Table 3-3 above). As discussed in sections 3.2 above, this is also consistent with the evidence currently available for this age group of young children, and the currently available evidence does not include additional C-R slopes for incremental neurocognitive decrement with blood Pb levels at or below this range. In considering whether this set of functions continues to be well supported by the evidence, as assessed in the ISA (ISA, section 4.3.2), we note the somewhat wide range in slopes encompassed by these study groups, while also noting the stability of the median. For example, omission of any of the four slopes considered in the last review does not appreciably change the median (e.g., the median would change from -1.75 IQ points per μg/dL blood Pb to -1.71 or -1.79). Thus, we conclude that while differing judgments might be made with regard to inclusion of each of the four study groups, these estimates are generally supported by the current review of the evidence in the ISA. Further, the stability of the median to modifications to this limited dataset lead us to conclude that the currently available evidence continues to support

consideration of -1.75 IQ points per μg/dL blood Pb as a well-founded and stable estimate for purposes of describing the neurocognitive impact quantitatively on this age group of U.S. children.

In summary, in considering the evidence and information available in this review pertaining to the level of the current Pb standard, we note that the evidence available in this review, as summarized in the ISA, continues to support the air-related IQ loss evidence-based framework, with the inputs that were used in the last review. These include estimates of air-to-blood ratios ranging from 1:5 to 1:10, with a generally central estimate of 1:7. Additionally, the C-R functions most relevant to blood Pb levels in U.S. children today continue to be provided by the set of four analyses considered in the last review for which the median estimate is -1.75 IQ points per μg/dL Pb in young children's blood. Thus, we observe that the evidence available in this review has changed little if at all with regard to the aspects given weight in the conclusion on level for the new standard in the last review and would not appear to call into question any of the basic elements of the standard. In so doing, we additionally recognize that the overall decision on adequacy of the current standard is a public health policy judgment by the Administrator; we discuss considerations that inform such judgments in section 4.3 below.

4.2.2 Exposure/Risk-based Considerations

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

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

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

described in section 3.4 above), the degree to which key sources of variability are reflected in the design of the analysis, and our characterization of key sources of uncertainty.

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

- *Variation in distributions of potential urban residential exposure and risk across U.S. urban residential areas.* This is addressed by the inclusion of three different (location-specific) urban study areas that reflect a diverse set of urban areas in the U.S.
- *Representation of a more highly exposed subset of urban residents potentially exposed at the level of the standard.* This is addressed by the inclusion of the generalized (local) urban study area.
- *Variation in residential exposure to ambient air Pb within an urban area.* This is addressed through the partitioning of the location-specific study areas into exposure zones to provide some representation of spatial gradients in ambient air Pb and their interaction with population distribution and demographics. This was done in a somewhat more precise manner in the primary Pb smelter case study, which relied on dispersion modeling to describe gradients, as compared with the manual assignment of gradients related to air concentration differences among monitors in an area.
- *Inter-individual variability in blood Pb levels.* This is addressed though the use of empirically-derived GSDs to develop blood Pb distribution for the child population in each exposure zone, with GSDs selected particular to each case study population.
- *Inter-individual variability in IQ response to blood Pb.* This is addressed through the use of C-R functions for IQ loss based on a pooled analysis reflecting studies of diverse populations.

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

In the quantitative risk information available in this review, we have air-related risk estimates for simulations just meeting the current standard from one of the location-specific urban case studies (Chicago) and from the generalized (local) urban case study. With regard to

the latter, we note its simplified design that does not include multiple exposure zones, thus reducing the dimensions simulated. We have a reasonable degree of confidence in aspects of the generalized (local) urban case study for the specific situation we consider it to represent (i.e., a temporal pattern of air Pb concentrations that just meets the level of the standard), and when the associated estimates are characterized as approximate, within upper and lower bounds (as described in section 3.4.4 above), while also recognizing considerable associated uncertainty.

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

In considering public health importance of estimated air-related risks, we consider here the nature and magnitude of such estimated risks (and attendant uncertainties), including such impacts on the affected population, and we additionally consider the size of the affected population. Based on the evidence available in the last review and consistent with that available today, the quantitative risk estimates developed in the 2007 REA and augmented slightly in this review are for decrements in IQ, an established indicator of neurocognitive function. In considering these estimates, we recognize that although some neurocognitive effects may be transient, some effects may persist into adulthood, affecting success later in life (ISA, sections 1.9.5 and 4.3.14). We additionally recognize the potential population impacts of small changes in population mean values of metrics such as IQ, presuming a uniform manifestation of lead-related decrement across the range of population IQ (ISA, section 1.9.1), as noted in section 3.3 above.

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

risk estimate for air-related Pb of a magnitude generally bounded by roughly 1 and 3 points IQ loss, with attendant uncertainties, and with appreciably lower risks with increasing distance from the highest exposure locations

In considering the importance of such risk from a public health perspective, we also consider the size of at-risk populations represented by the REA case studies. As discussed in section 3.4 above, the generalized (local) urban case study is considered to represent a localized urban population exposed near the level of the standard, such as a very small, compact neighborhood near a source contributing to air Pb concentrations just meeting the standard. This case study provides representation in the risk assessment for such small populations at the upper end of the gradient in ambient air concentrations expected to occur near sources; thus estimates for this case study reflect exposures nearest the standard being evaluated. While we do not have precise estimates of the number of young children living in such areas of the U.S. today, we have information that informs our understanding of their magnitude. For example, as discussed in section 3.3 above (Table 3-4), we estimate there to be approximately 2,400 children, aged 5 years and younger, residing within 0.5 km of a monitor with air Pb concentrations above the current standard. We further observe several additional monitors with maximum 3-month Pb concentrations that fall below but within 10% of the current standard level (as noted in section 3.3 above); an estimated 265 young children reside near these monitors. Thus, together we estimate some 2700 children, aged 5 years and younger, living in localized areas with elevated air Pb concentrations that are above or near the current standard. Based on the 2010 census estimates of approximately 24.3 million children in the U.S. aged five years or younger, this represents approximately one hundredth of one percent of this age group in the U.S.[7] This indicates the size of the population of young children of this age living in areas in close proximity to areas where air Pb concentrations may be above or near the current standard approximately a hundredth of one percent of the full population of correspondingly aged children.[8]

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[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. Further, to the extent some of the source-oriented monitors required by the 2010 monitoring regulation summarized in section 2.2.1.1 above were not yet represented in the analysis of monitoring data summarized in Table 3-4 above and might report such elevated concentrations, consideration of the analysis summarized in Table 3-5 indicates the potential for only small changes in the percentage of the full age group represented by the subset (e.g., potential for less than an additional 0.01%).

[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.

In summary, we recognize substantial uncertainty inherent in the REA estimates of air-related risk associated with localized conditions just meeting the current standard, which we have characterized as approximate and falling near or somewhat above the rough lower bound of 1 and below the rough upper bound of 3 IQ points.[9] This approximate estimate of risk for children living in such areas is generally overlapping with and consistent with the evidence-based air-related IQ loss estimates summarized in section 4.2.1 above. With regard to the importance of the estimated risks from a public health perspective, we note, based on the currently available monitoring and census data, the estimated size of the population at risk in such areas to be approximately 2700 young children, representing slightly more than one one hundredth of a percent of the full population of similarly aged children. In considering the exposure/risk estimates during the last review, the Administrator took note of the important uncertainties and limitations associated with these assessments and, while placing less weight on the assessment estimates, he recognized the quantitative risk estimates to be "roughly consistent with and generally supportive" of those estimated by the evidence-based framework. In our consideration of the risk estimates considered in the last review, as well as the estimates for the current standard that have been newly developed in this review for the generalized (local) urban case study (see section 3.4.3.3.2), we agree with the previous characterization. As would be expected by the use of interpolation, the newly derived estimates are consistent with the estimates for similar air quality scenarios that were available in the last review.

4.2.3 CASAC Advice

In our consideration of the adequacy of the current standard, in addition to the evidence- and risk/exposure-based information discussed above, we have also considered the advice and recommendations of CASAC, based on their review of the ISA, the REA Planning Document, and the earlier draft of this document, as well as comments from the public on the earlier draft of this document.

A limited number of public comments have been received on this review to date, including comments focused on the draft IRP, the draft REA Planning Document or the draft PA. Of the three commenters that addressed adequacy of the current primary Pb standard, two are in agreement with staff conclusions in the draft PA. A third expressed the view that the 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).

should be revised to be more restrictive given the evidence of Pb effects in populations with mean blood Pb levels below 10 μg/dL.[10]

In their comments on the draft PA, the CASAC Pb Panel concurred with staff's overall preliminary conclusions that it is appropriate to consider retaining the current primary standard without revision, stating that "the current scientific literature does not support a revision to the Primary Lead (Pb) National Ambient Air Quality Standard (NAAQS)" (Frey, 2013). They further noted that "[a]lthough the current review incorporates a substantial body of new scientific literature, the new literature does not justify a revision to the standards because it does not significantly reduce substantial data gaps and uncertainties (e.g., air-blood Pb relationship at low levels; sources contributing to current population blood Pb levels, especially in children; the relationship between Pb and childhood neurocognitive function at current population exposure levels; the relationship between ambient air Pb and outdoor dust and surface soil Pb concentrations)". In recognition of these limitations in the available information, the CASAC provided recommendations on research to address these data gaps and uncertainties so as to inform future Pb NAAQS reviews (Frey, 2013).

The CASAC comments indicated agreements with key aspects of staff's consideration of the exposure/risk information and currently available evidence in this review (Frey, 2013, Consensus Response to Charge Questions, p. 7).

> *The use of exposure/risk information from the previous Pb NAAQS review appears appropriate given the absence of significant new information that could fundamentally change the interpretation of the exposure/risk information. This interpretation is reasonable given that information supporting the current standard is largely unchanged since the current standard was issued.*
>
> *The CASAC agrees that the adverse impact of low levels of Pb exposure on neurocognitive function and development in children remains the most sensitive health endpoint, and that a primary Pb NAAQS designed to protect against that effect will offer satisfactory protection against the many other health impacts associated with Pb exposure.*
>
> *The CASAC concurs with the draft PA that the scientific findings pertaining to air-to-blood Pb ratios and the C-R relationships between blood Pb and childhood IQ decrements that formed the basis of the current Pb NAAQS remain valid and are consistent with current data.*

The CASAC concurred with the appropriateness of the application of the evidence-based framework from the last Pb NAAQS review. With regard to the key inputs to that framework, CASAC concluded that "[t]he new literature published since the previous review provides

[10] All written comments submitted to the Agency will be available in the docket for this rulemaking, as will be transcripts of the public meetings held in conjunction with CASAC's review of the earlier draft of this document, of the REA Planning Document and of drafts of the ISA.

further support for the health effect conclusions presented in that review" and that the studies newly available in this review "do not fundamentally alter the uncertainties for air-to-blood ratios or C-R functions for IQ decrements in young children (Frey, 2013, Consensus Response to Charge Questions, p. 6).

The comments from CASAC took note of the uncertainties that remain in this review, which contribute to the uncertainties associated with drawing conclusions regarding air-related exposures and associated health risk at or below the level of the current standard (Frey, 2013, Consensus Response to Charge Questions, p. 6).

> *This draft PA is constrained by the absence of new observational and experimental data that address, at least in part, limitations and uncertainties in the evidence that was present at the time of the last update of the Pb NAAQS. Until evidence is available to assess Pb exposure and health risks related to air Pb levels reflecting the current standard, a substantive refinement and update of the PA will not be possible. The obvious uncertainty underlying evaluation of this PA is whether lowering the standard would (or would not) impact exposure and thus risk. The CASAC agrees with the EPA conclusion that "there is appreciable uncertainty associated with drawing conclusions regarding whether there would be reductions in blood Pb levels from alternative lower levels as compared to the level of the current standard."*

4.3 STAFF CONCLUSIONS ON THE PRIMARY STANDARD

This section describes staff conclusions regarding adequacy of the current primary Pb standard. These conclusions are based on considerations described above and in the discussion below regarding the currently available scientific evidence summarized in the ISA and prior CDs and the risk and exposure information drawn from the 2007 REA. Further, these staff conclusions have taken into account advice from CASAC and public comment on the draft PA and preliminary staff conclusions.

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

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

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

quantitative analyses that are inherent in decisions on the adequacy of the current primary Pb standard.

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

In considering the currently available evidence, staff gives great weight to the long-standing body of evidence on the health effects of Pb, augmented in some aspects since the last review, which continues to support identification of neurocognitive effects in young children as

[11] 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).

the most sensitive endpoint associated with Pb exposure, as discussed in sections 3.2 and 3.3 above. The evidence continues to indicate that a standard that provides protection from neurocognitive effects in young children additionally provides protection for other health effects of Pb, such as cardiovascular effects later in life. Application of the evidence-based, air-related IQ loss framework, developed in the last review, continues to provide a useful approach for considering and integrating the evidence on relationships between Pb in ambient air and Pb in children's blood and risks of neurocognitive effects (IQ loss). The currently available evidence base, while somewhat expanded since the last review, is not appreciably expanded or supportive of appreciably different conclusions with regard to air-to-blood ratios (section 3.1 above) or C-R functions for neurocognitive decrements (section 3.2 above) in young children.

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

We additionally take note of the role of public health policy judgments in considering the evidence-based framework and the exposure/risk information to inform the Administrator's decision in 2008 to set the current standard, as summarized in section 4.1.1.2 above. We recognize that public health policy judgments always play an important role in each NAAQS review for each pollutant. One type of public health policy judgment focuses on how to consider the nature and magnitude of the array of uncertainties that are inherent in the scientific evidence and analyses. These judgments are traditionally made with a recognition that our understanding

of the relationships between the presence of a pollutant in ambient air and associated health effects is based on a broad body of information encompassing not only more established aspects of the evidence but also aspects in which there may be substantial uncertainty. In the case of the Pb NAAQS review, we recognize increased uncertainty in characterizing the relationship of effects on IQ with blood Pb levels below those represented in the evidence base. We also recognize increased uncertainty in projecting the magnitude of blood Pb response to ambient air Pb concentrations at and below the level of the current standard. We recognize this increased uncertainty particularly in light of the multiple factors that play a role in such a projection (e.g., meteorology, atmospheric dispersion and deposition, human physiology and behavior), for each of which we recognize attendant uncertainties. Collectively, these aspects of the evidence and associated uncertainties contribute to a recognition that for Pb, as for other pollutants, the available health effects evidence generally reflects a continuum, consisting of 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.

Reviews may also require judgments as to the point at which health effects become important from a public health perspective. In the case of Pb, such a judgment includes consideration of the public health significance of one to two points IQ loss in at-risk populations, such as young children, in light of associated uncertainties. This type of judgment also includes consideration of the IQ loss estimates yielded by the air-related IQ loss evidence-based framework for specific combinations of standard level, air-to-blood ratio and C-R function. With regard to public health significance of one to two points IQ loss in young children, staff gives weight to the comments of CASAC and some public commenters in the last review which recognized such a magnitude of IQ loss to be of public health significance and recommended that a very high percentage of the population be protected from such a magnitude of IQ loss (73 FR 67000).[12] With this objective in mind, we consider the extent to which the air-related IQ loss evidence-based framework informs consideration of standards that might be concluded to provide such a level of protection. In so doing, we first recognize that the IQ loss estimates produced with the evidence-based framework do not correspond to a specific quantitative public health policy goal for air-related IQ loss that would be acceptable or unacceptable for the entire population of children in the U.S. Rather, the conceptual context for the evidence-based framework is that it provides estimates for the mean air-related IQ loss of a subset of the population of U.S. children (i.e., the subset living in close proximity to air Pb sources that

[12] 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 4.3.2; 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.

contributed to elevated air Pb concentrations that equal the current level of the standard). This is the subset expected to experience air-related Pb exposures at the high end of the national distribution of such exposures. The associated mean IQ loss estimate is the average for this highly exposed subset and is not the average air-related IQ loss projected for the entire U.S. population of children. Further, we recognize uncertainties associated with those estimates, which increase with estimates associated with successively lower standard levels.

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

In drawing conclusions from application of the evidence-based framework with regard to adequacy of the current standard, we further recognize the degree to which IQ loss estimates

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[13] Using an air-to-blood ratio estimate of 8 μg/dL per $\mu g/m^3$ yields a similar, but just slightly higher IQ loss estimate (of approximately 2 IQ points).

[14] 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.

drawn from the air-related IQ loss evidence-based framework reflect mean blood Pb levels below those represented in the currently available evidence for young children. For example, in the case of the current standard level of 0.15 μg/m^3, multiplication by the air-to-blood ratio of 7 yields a mean air-related blood Pb level of 1.05 μg/dL, which is half the level of the lowest blood Pb subgroup of pre-school children in which neurocognitive effects have been observed (Table 3-2 above; Miranda et al., 2009) and well below the means of subgroups for which continuous C-R functions have been estimated (Table 3-3 above).[15] Such an extension below the lowest studied levels may be viewed as appropriate given the lack of identified blood Pb level threshold in the current evidence base for neurocognitive effects and the need for the NAAQS to provide a margin of safety.[16] We note, however, that the framework IQ loss estimates for still lower potential standard levels represent still greater extrapolations from the current evidence base with corresponding increased uncertainty.

In recognition of the role of public health policy judgments in drawing conclusions as to adequacy of the Pb NAAQS, we consider the availability of new information or new commonly accepted guidelines or criteria within the public health community with regard to the public health significance of specific IQ decrements in exposed, at-risk populations that might inform public health policy judgments on the appropriate degree of public health protection that should be afforded to protect against risk of such neurocognitive effects in at-risk populations,[17] such as children living near air Pb sources. As an initial matter, we note that no such new information, guidelines or criteria are described in the ISA. In further considering the occurrence of any new actions by public health agencies that might indicate the availability of new information,

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[15] 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 neurocognitive risk per increment of blood Pb is greater for children with the lowest blood Pb levels, as noted in section 3.2 above. Since the evidence indicates Pb exposure to pose the greatest risk on an incremental basis at lower blood Pb levels, 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.

[16] 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.

[17]The term at-risk populations is used here, rather then 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.

guidelines or criteria for interpreting public health significance of such effects, we note that the CDC has revised the blood Pb level used to prioritize young children for whom they recommend particular follow-up health-protective actions, as summarized in section 3.1 above.[18] The CDC decision, while emphasizing the critical importance of primary prevention of Pb exposure, provides no new guidelines or criteria with regard to the significance of specific IQ decrements or judgments on appropriate public health protection from risk of neurocognitive effects, and their consideration of the evidence base at the time of their decision does not substantively differ from that presented in the ISA (CDC, 2012). Thus, we are aware of no new information or new commonly accepted guidelines or criteria within the public health community for interpreting public health significance of neurocognitive effects in the context of a decision on adequacy of the current Pb standard.

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

Based on the above considerations and with consideration of advice from CASAC, we reach the conclusion that the current body of evidence, in combination with the exposure/risk information, supports a primary standard as protective as the current standard. Further consideration of the evidence and exposure/risk information available in this review and its attendant uncertainties and limitations, advice from CASAC and consideration of the availability of other information that might also inform public health policy judgments by the Administrator, leads us to reach the additional conclusion that it is appropriate to consider retaining the current

[18] 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).

standard without revision. We base these conclusions on consideration of the health effects evidence, including consideration of this evidence in the context of the evidence-based, air-related IQ loss framework, and in combination with the exposure/risk information (chapter 3 and sections 4.2.1 and 4.2.2 above) and the uncertainties attendant with both. In so doing, we recognize the complexities and limitations in the evidence base in reaching conclusions regarding the magnitude of risk associated with the current standard, as well as the increasing uncertainty of risk estimates for lower air Pb concentrations. Based on these considerations, these conclusions also recognize what may be considered reasonable judgments on the public health implications of the blood Pb levels and risk estimated for air-related Pb under the current standard, including the public health significance of the Pb effects being considered, as well as aspects of the use of the evidence-based framework that may be considered to contribute to the margin of safety.

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

In this context, we recognize that the uncertainties and limitations associated with the many aspects of the estimated relationships between air Pb concentrations and blood Pb levels and associated health effects are amplified with consideration of increasingly lower air concentrations. We believe the current evidence supports the conclusion that the current standard is requisite to protect public health with an adequate margin of safety. In staff's view, with which CASAC has agreed (Frey, 2013, p. 6) based on the current evidence, there is

appreciable uncertainty associated with drawing conclusions regarding whether there would be reductions in blood Pb levels and risk to public health from alternative lower levels of the standard as compared to the level of the current standard. Thus, we conclude that the basis for any consideration of alternative lower standard levels would reflect different public health policy judgments as to the appropriate approach for weighing uncertainties in the evidence and for providing requisite protection of public health with an adequate margin of safety.

In summary, the newly available health effects evidence, critically assessed in the ISA as part of the full body of evidence, reaffirms conclusions on the broad array of effects recognized for Pb in the last review. Further, we observe the general consistency of the current evidence with the evidence available in the last review with regard to key aspects on which the current standard is based, including those particular to the evidence-based framework developed in the last review. We additionally observe that quantitative risk estimates associated with the current standard, based on the risk assessment performed in the last review, indicate a level of risk generally consistent with conclusions drawn from the evidence using the evidence-based framework. In so doing, we also recognize the limitations and uncertainties associated with the currently available information. These considerations and the advice from CASAC provide the basis for the staff conclusion that consideration should be given to retaining the current standard, without revision. In light of this conclusion, we have not identified any potential alternative standards for consideration in this review.

4.4 KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH AND DATA COLLECTION

In this section, we highlight key uncertainties associated with reviewing and establishing NAAQS for Pb. Such key uncertainties and areas for future health-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.

- A critical aspect of our consideration of the evidence and the quantitative risk assessment in this review is our understanding of the C-R relationship between blood Pb levels in young children and neurodevelopmental effects, specifically IQ decrement. An important area of uncertainty in the Pb NAAQS review concerns interpretation of neurocognitive impact risks and the shape of the C-R relationship at blood Pb levels similar to and below those common in today's U.S. young child population. Accordingly, additional epidemiological research involving substantially sized populations with mean blood Pb levels closer to those common in today's population of young children, particularly those

less affected by higher Pb exposures earlier in childhood, would help to reduce uncertainty in our estimates of IQ decrement associated with these lower blood Pb levels and, accordingly, in characterizing Pb health effects.

- Key uncertainties with regard to other aspects of the health effects evidence include the following.
  - There remains uncertainty in the evidence base with regard to the exposure circumstances (pre- and postnatal timing, duration, magnitude and frequency) eliciting effects in older children and adults. Effects of particular focus include effects on the nervous system later in life, cardiovascular function, and delayed onset of puberty.
  - Alzheimer's-like pathology has been reported in aged laboratory animals (non-human primates and rodents) exposed to Pb early in life. Uncertainty remains, however, regarding the relationships of such pathology with altered function or behavior.
  - Epidemiological studies indicate detrimental effects of Pb on sperm production and quality, often in occupational cohorts. Uncertainty remains regarding these effects in otherwise healthy cohorts without occupational Pb exposure or other underlying medical conditions.
  - An additional area of uncertainty is that related to the inverse association observed in some studies between low blood Pb and renal function.
- A key consideration in the Pb NAAQS review concerns the relationship between air Pb concentrations and blood Pb levels, most particularly those in young children. Our quantitative estimation of blood Pb levels in response to various exposure circumstances, including air-related exposure pathways associated with current ambient air Pb concentrations, would benefit from research into this relationship.
  - Information is limited with regard to the temporal aspects of the relationship between ambient air Pb and levels of air-related Pb in blood in young children, and our understanding of the factors influencing this relationship is incomplete, particularly in the Pb exposure circumstances common in the U.S. today.
  - Important aspects of (or influence on) the air-to-blood relationship include the relationships (1) between ambient air Pb and outdoor dust and surface soil Pb concentrations and (2) between ambient air Pb and indoor dust Pb. Additional information would help to reduce uncertainty in our understanding of these relationships and in models and methods used to characterize these pathways in future reviews. Specific mechanistic aspects for indoor dust modeling include air exchange rates, home cleaning frequency and efficiency, among other factors. The impact of changes in ambient air Pb on Pb in indoor dust, outdoor dust and soil, including the temporal dynamics of these relationships, and variations for different environments, are important aspects of the Pb NAAQS review, yet current information on multiple aspects of these pathways is limited.
  - There is appreciable uncertainty regarding the magnitude of the contribution of air-related Pb to diet. Additional information is needed regarding sources of Pb to

the diet of different age groups of the U.S. population, including particularly those sources that relate to current ambient air Pb, as well as those related to the legacy of historic ambient air Pb emissions.

  - Information is lacking on toxicokinetics of Pb during adolescence which could inform interpretation of epidemiological studies of relationships between blood Pb and health effects in this lifestage.

- Future quantitative blood Pb modeling would benefit from additional research into several aspects of population blood Pb levels, including contributing exposure pathways. Examples include:

  - Interindividual variability in blood Pb levels and methods for characterizing interindividual variability, including consideration of both empirical and mechanistic methods;

  - Apportionment of blood Pb levels with regard to exposure pathway contributions, with particular focus on understanding exposure pathways and sources that cause the more elevated blood Pb levels among children today, as well as those related to ambient air Pb; and

  - Blood Pb model performance evaluations, with emphasis on applications pertaining to blood Pb response to ambient air-related pathways and responses to changes in exposures for those pathways.

- An understanding of the spatial gradient of ambient air Pb concentrations and associated particle sizes in urban residential areas, as well as near Pb sources, is an important aspect to our implementation of the NAAQS for Pb and a key element in assessing exposure and risk. Additional research in this area is needed. Current limitations in this area additionally contribute uncertainty to characterization of ambient air Pb levels in the risk assessment and the resulting exposure and risk estimates. Research in the characterization of spatial variation in ambient air Pb concentrations in different environments and related to different air sources would help to reduce this uncertainty. The potential for systematic trends in the relationship between ambient air Pb concentrations and distribution of urban residential populations is of interest. Example locations of interest include neighborhoods downwind of airports with substantial leaded aviation gasoline usage, as well as those in the vicinity of older roads with a substantial historical use of leaded gasoline, including inner city neighborhoods (with and without substantial reconstruction).

- Another area of uncertainty relates to our understanding of concentrations of relatively larger airborne particles carrying Pb that occur in areas near sources and where exposure may occur.

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5 WELFARE EFFECTS AND EXPOSURE/RISK INFORMATION

This chapter presents key aspects of the current evidence of Pb-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 *Integrated Science Assessment for Lead* (USEPA, 2013; henceforth referred to as the ISA) and the 2006 *Air Quality Criteria Document for Lead* (USEPA, 2006a; 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 International, 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 Integrated Review Plan (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 the last review (ISA, section 1.7; 2006 CD). A number of studies on ecological effects of Pb are newly available in this review and are critically assessed in the ISA as part of the full body of evidence. The full body of currently available evidence reaffirms conclusions on the array of effects recognized for Pb in the last review (ISA, section 1.7). In so doing, in the context of pollutant exposures considered relevant,[1] the ISA determines[2] that causal[3] or likely causal[4] relationships exist in both freshwater

[1] With regard to consideration of pollutant exposures for studies included in the ISA, the ISA states the following (ISA, pp. lx-lxi).

> *In drawing judgments regarding causality for the criteria air pollutants, the ISA focuses on evidence of effects in the range of relevant pollutant exposures or doses, and not on determination of causality at any dose. Emphasis is placed on evidence of effects at doses (e.g., blood Pb concentration) or exposures (e.g., air concentrations) that are relevant to, or somewhat above, those currently experienced by the population. The extent to which studies of higher concentrations are considered varies by pollutant and major outcome category, but generally includes those with doses or exposures in the range of one to two orders of magnitude above current or ambient conditions. Studies that use higher doses or exposures may also be considered to the extent that they provide useful information to inform understanding of mode of action, interspecies differences, or factors that may increase risk of effects for a population. Thus, a causality determination is based on weight of evidence evaluation for health, ecological or welfare effects, focusing on the evidence from exposures or doses generally ranging from current levels to one or two orders of magnitude above current levels.*

and terrestrial ecosystems for Pb with effects on reproduction and development in vertebrates and invertebrates; growth in plants and invertebrates; and survival in vertebrates and invertebrates (ISA, table 1-3). Although considerable uncertainties are recognized in generalizing effects observed under particular, small-scale conditions, up to the ecosystem level of biological organization, the ISA determines that the cumulative evidence reported for Pb effects at higher levels of biological organization and for the above described population-level endpoints is sufficient to conclude that a causal relationship is likely to exist between Pb exposures and community and ecosystem-level effects in freshwater and terrestrial systems (ISA, section 1.7.3.7). The ISA also presents evidence for saltwater ecosystems, concluding that current evidence is inadequate to make causality determinations for most population-level responses, as well as community and ecosystem effects, while finding the evidence to be suggestive linking Pb and effects on reproduction and development in marine invertebrates (ISA, Table 1-3, section 6.3.12 and section 6.4.21).

Based on the extensive assessment of the full body of evidence available in this review, the major conclusions drawn by the ISA regarding ecological effects of Pb include the following (ISA, Executive Summary, p. xcvi).

> *With regard to the ecological effects of Pb, uptake of Pb into fauna and subsequent effects on reproduction, growth and survival are established and are further supported by more recent evidence. These may lead to effects at the population, community, and ecosystem level of biological organization. In both terrestrial and aquatic organisms, gradients in response are observed with increasing concentration of Pb and some studies report effects within the range of Pb detected in environmental media over the last several decades. Specifically, observations from controlled studies on reproduction, growth, and survival in sensitive freshwater invertebrates are well-characterized at concentrations at or near Pb concentrations occasionally encountered in U.S. fresh surface waters…. However, in natural environments, modifying factors affect Pb bioavailability and toxicity and there are considerable uncertainties associated with generalizing effects observed in controlled studies to effects at higher levels of biological organization. Furthermore, available studies on community and ecosystem-level*

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[2] 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).

[3] In determining that a causal relationship exists for Pb with specific ecological 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. lxii)

[4] In determining a likely causal relationship exists for Pb with specific ecological effects, EPA has concluded that "[e]vidence is sufficient to conclude that there is a likely causal relationship with relevant pollutant exposures ... important uncertainties remain" (ISA, p. lxii).

effects are usually from contaminated areas where Pb concentrations are much higher than typically encountered in the environment. The contribution of atmospheric Pb to specific sites is not clear and the connection between air concentration of Pb and ecosystem exposure continues to be poorly characterized. Furthermore, the level at which Pb elicits a specific effect is difficult to establish in terrestrial and aquatic systems, due to the influence of other environmental variables (e.g., pH, organic matter) on both Pb bioavailability and toxicity, and also to substantial species differences in Pb sensitivity.

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

Our consideration of welfare effects evidence in this review is framed by key policy-relevant questions drawn from those included in the IRP. In the following sections, we discuss the pathways by which Pb exposure occurs in ecosystems, the mechanisms that distribute Pb in the environment and the bioavailability of Pb in different ecosystems. Understanding the movement of Pb in the environment is important to understanding exposure and bioavailability and, thus, informs the subsequent discussion of the effects of Pb on terrestrial and aquatic ecosystems. Finally, we discuss the association of ambient air Pb with effects and the important role a "critical loads" approach[5] might play in assessing the overall ability of ecosystems to recover from past Pb exposures and the degree to which newly deposited Pb may affect ecosystem function and recovery.

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

The extensive history of Pb uses in developed countries coupled with atmospheric transport processes has left a legacy of Pb in ecosystems globally (e.g., 1977 CD, section 6.3.1). Records of U.S. atmospheric emissions of Pb in the twentieth and late nineteenth centuries have been documented in sediment cores, as noted in section 2.3 above (ISA, section 2.6.2; Landers et

[5] As discussed further in subsequent text of this section, the phrase "critical loads" is generally used to describe loading (i.e. addition of a pollutant) to a system that can occur without causing a critical impact, e.g., deposition rates of air pollutants that current knowledge indicates will not cause long-term adverse effects to ecosystem structure and function. A critical loads analysis approach takes into account what is known about the release of a specific chemical into the environment, its distribution and cycling within and across ecosystems and each ecosystem's sensitivity to the chemical (ISA, section 6.1.3).

al., 2010). Once deposited, Pb can be transported by stormwater runoff or resuspension to catchments and nearby waterbodies or stored in soil layers in forested areas, its further movement influenced by soil or sediment composition and chemistry and physical processes. Some new studies are available that provide additional information, briefly summarized below, on Pb cycling, flux and retention within terrestrial and aquatic systems. This new information does not fundamentally change our understanding from the last review of Pb movement through or accumulation in ecosystems over time but rather improves our understanding of some of the underlying processes and mechanisms in soil, water and sediment. There is little new information, however, on fate and transport in ecosystems specifically related to air-derived Pb (ISA, section 2.3). There is limited newly available information with regard to the timing of ecosystem recovery from historic atmospheric deposition of Pb.

Overall, recent studies in terrestrial ecosystems provide deposition data consistent with deposition fluxes reported in the 2006 CD, and demonstrate consistently that atmospheric deposition of Pb has decreased since the phase-out of leaded on-road gasoline, as described in section 2.3.2.2 above (ISA, section 2.3.3). Follow-up studies in several locations at high elevation sites indicate little change in soil Pb concentrations since the phase-out of leaded on-road gasoline in surface soils, consistent with the high retention reportedly associated with reduced microbial activity at lower temperatures associated with high elevation sites. However, amounts of Pb in the surface soils at some lower altitude sites were reduced over the same time period in the same study (ISA, section 2.3.3). New studies in the ISA also enhance our understanding of Pb sequestration in forest soils by providing additional information on the role of leaf litter as a Pb reservoir in some situations and the effect of litter decomposition on Pb distribution (ISA, section 2.3.3).

Recent research on Pb transport in aquatic systems has provided a large body of observations confirming that such transport is dominated by colloids rich in iron and organic material (ISA, section 2.3.2). Recent research on Pb flux in sediments provides greater detail on resuspension processes than was available in the 2006 CD, including research on resuspended Pb largely associated with organic material or iron and manganese particles and research on the important role played by anoxic or depleted oxygen environments in Pb cycling in aquatic systems. This newer research is consistent with prior evidence in indicating that appreciable resuspension and release from sediments largely occurs during discrete events related to storms. It has also confirmed that resuspension is an important process that strongly influences the lifetime of Pb in bodies of water. Finally, there have been advances in understanding and modeling of Pb partitioning between organic material and sediment in aquatic environments (ISA, section 2.7.2).

In summary, the newly available evidence builds on our understanding of some specific aspects of processes involved in the movement and accumulation of Pb through ecosystems over time. The new information, however, does not substantially alter our overall understanding of the fate and transport of Pb in ecosystems or provide a broad quantitative understanding of U.S. ecosystem responses to atmospheric Pb deposition.

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

As discussed in the ISA, bioavailability of Pb is an important component of understanding the effects Pb is likely to have on organisms and ecosystems (ISA, section 6.3.3). It is the amount of Pb that can interact within the organism that leads to toxicity, and there are many factors which govern this interaction (ISA, sections 6.2.1 and 6.3.3; USEPA, 2007a). The bioavailability of metals varies widely depending on the physical, chemical, and biological conditions under which an organism is exposed (USEPA, 2007a). In both aquatic and terrestrial systems, a primary factor influencing the bioavailability of Pb, as well as its fate and tranport is solubility (ISA, p. 6-63). Additionally important characteristics that affect bioavailability are (1) chemical form or species, (2) particle size, (3) lability, and (4) source. The bioavailability of a metal is also dependent upon the fraction of metal that is bioaccessible. The bioaccessible fraction of a metal is the portion (fraction or percentage) of environmentally available metal that actually interacts at the organism's contact surface and is potentially available for absorption or adsorption by the organism (USEPA, 2007a; ISA, section 6.3.3).

Studies newly available since the last Pb NAAQS review provide additional insight into factors that influence the bioavailability of Pb to specific organisms (ISA, section 6.3.3). In general, this evidence, briefly summarized below, is supportive of previous conclusions and does not identify significant new variables from those identified previously. Section 6.3.3 of the ISA provides a detailed discussion of bioavailability in terrestrial systems. With regard to aquatic systems, a detailed discussion of bioavailability in freshwater systems is provided in sections 6.4.3 and 6.4.4 of the ISA, and section 6.4.14 of the ISA discusses bioavailability in saltwater systems.

In terrestrial systems, the amount of bioavailable Pb present determines the impact of soil Pb to a much greater extent than does the total amount present (ISA, section 6.3.11). In such ecosystems, Pb is deposited either directly onto plant surfaces or onto soil where it can bind with organic matter or dissolve in pore water.[6] The Pb dissolved in pore water is particularly bioavailable to organisms in the soil and thereby influences the impact of soil Pb on terrestrial ecosystems to a much greater extent than the total amount of Pb present (ISA, section 6.3.11).

[6] The term "pore water" refers to the water occupying the spaces among the grains of sediment or soil.

Several soil characteristics control the amount of Pb that is dissolved in pore water, and the ISA presents evidence that has advanced scientific understanding of some of these variables (ISA, section 6.4.21). Studies have shown that the two most important determinants of both Pb solubility and toxicity in soils are pH and cation exchange capacity (ISA, section 2.6.1). Also, evidence newly available in this review has confirmed the important influence of organic matter on Pb sequestration, leading to relatively longer retention in soils with higher organic matter content (ISA, section 2.6.1). When soils are amended with soluble metals, aging, both under natural conditions and simulated through leaching, reduces the bioavailability of Pb to plants and soil organisms (ISA, sections 6.3.2, 6.3.9 and 6.3.11; USEPA, 2007a). In general, soils with higher organic matter content have the capacity for greater retention of Pb in the soil matrix and lesser availability of Pb for release into pore water. Reduction in soil organic material, such as through decomposition, can contribute to subsequent increased availability of Pb (ISA, section 6.3.9; 2006 CD, section 6.1.5).

In aquatic systems as in terrestrial systems, the amount of Pb bioavailable to organisms is a better predictor of effect on organisms than the overall amount of Pb in the system. Once atmospherically derived Pb enters surface water bodies through deposition or runoff, its fate and bioavailability are influenced by many water quality characteristics, such as pH, suspended solids levels and organic content (ISA, section 6.4.2). In sediments, bioavailability of Pb to sediment-dwelling organisms may be influenced by the presence of other metals, sulfides, iron oxides and manganese oxides and also by physical disturbance (ISA, section 2.6.2). For many aquatic organisms Pb dissolved in the water column can be the primary exposure route, while for others sediment ingestion is significant (ISA, section 2.6.2). As recognized in the 2006 CD and further supported in the ISA, there is a body of evidence showing that uptake and elimination of Pb vary widely among aquatic species.

Although in freshwater systems the presence of humic acid in dissolved organic material (DOM) is considered to reduce the bioavailable fraction of metals in the water column, there is evidence presented in the ISA that DOM does not have the same effect on free Pb ion concentration and toxicity in seawater (ISA, section 6.4.2). For example, the ISA discusses two new studies, performed in artificial seawater systems, that suggest that in saltwater, the presence of DOM increases (rather than decreases) uptake of Pb by mussel gill structures, potentially through the alteration of membrane permeability (ISA, section 6.4.2.4). These studies also investigated the individual roles of some DOM components. This recent evidence supports the conclusion from the last review that factors that modify bioavailability of Pb in saltwater environments are not identical to those in freshwater systems (ISA, section 6.4.14).

The newly available evidence about bioavailability from experimental systems, briefly summarized above, builds on our fundamental understanding of Pb bioavailability in aquatic and

terrestrial systems and species from the last review. Bioavailability remains an important consideration in Pb toxicity and a significant source of uncertainty in relating ambient Pb and adverse effects.

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

There is a substantial amount of new evidence in this review regarding the ecological effects of Pb on individual terrestrial and aquatic species. On the whole, this evidence supports previous conclusions that Pb has effects on growth, reproduction and survival, and that under some conditions these effects can be adverse to organisms and ecosystems. The ISA provides evidence of effects in additional species, and in a few cases, at lower exposures than reported in the previous review, but does not substantially alter our understanding of the ecological endpoints affected by Pb from the previous review. Looking beyond organism-level evidence, the evidence of adversity in natural systems remains sparse due to the difficulty in determining the effects of confounding factors such as co-occurring metals or system characteristics that influence bioavailability of Pb in field studies. The following is a brief comparison of the newly available evidence to evidence considered in the 2006 review.

Terrestrial Ecosystems

The evidence available in the last review indicated a range of biological effects of Pb on terrestrial organisms that varied with type of organism and life stage, duration of exposure, form of Pb, and soil characteristics. New research since the 2006 CD has broadened our understanding of the evidence of damage to photosynthetic ability in plants exposed to Pb and provided additional evidence of oxidative stress in response to Pb exposure (ISA, section 6.3.4). For example, reactive oxygen species have been found to increase in plant tissue grown in Pb-contaminated soil, and, with increasing Pb exposure, the plant tissue responded with increased antioxidant activity (ISA, section 6.3.4). In addition, recent studies have reported reduced growth of plants with increased Pb concentration in soil in some experiments, as well as evidence of genotoxicity, decreased germination, and pollen sterility (ISA, section 6.3.4).

In terrestrial invertebrates, previous CDs have reported adverse effects of Pb on neurological and reproductive endpoints. Recently published studies have further explored the potential for neurotoxic action of Pb, albeit under artificial liquid media conditions (ISA, section 6.3.4.2). Increased mortality was found in recent studies of earthworms at concentrations similar to those in studies reviewed previously, with additional evidence indicating the strong dependence of effects on soil characteristics including pH, cation exchange capacity, and aging (ISA, section 6.3.4.2). There is also newly available evidence for adverse

effects in terrestrial snails and arthropods exposed through soil or diet (ISA, section 6.3.4.2). The effects vary with species and exposure conditions, and they include diminished growth and fecundity, endocrine and reproductive anomalies, and body deformities. Increasing concentration of Pb in the exposure medium generally resulted in increased effects within each study, but the relationship between concentration and effects is highly variable between studies, even when the same medium, e.g. soil, was used. Current evidence suggests that soil aging and pH are important modifiers of Pb toxicity in these studies (ISA, section 6.3.11).

In terrestrial vertebrates, some new evidence is available for effects of Pb on amphibians and reptiles as well as birds.[7] Effects reported in reptiles and amphibians include decreased white blood cell counts, decreased testis weight, and behavioral anomalies (ISA, section 6.3.4.3). However, depending on various factors, studies report large differences in effects in different species at the same concentration of Pb in soil, and effects were generally smaller when field-collected soils were used. In some birds, recent studies have found maternal elevated blood Pb level to be associated with decreased hatching success, smaller clutch size, high corticosteroid level, and abnormal behavior in offspring. Studies on some species show little or no effect of elevated blood Pb level. Effects of dietary exposure have been studied in several mammalian species, with cognitive, endocrine, immunological, and growth effects observed (ISA, section 6.3.11) in some studies.

Experimental evidence of organism-level effects presented in the ISA demonstrates that increased exposure to Pb is generally associated with increases in observed effects in terrestrial species (ISA, section 6.3.11). It also demonstrates that many factors, including the form of Pb and various soil physiochemical properties, influence the Pb concentration-response relationship (ISA, section 6.3.11). Further, results from amended soil exposures generally do not reflect the important effect of environmental aging on bioavailability and associated toxicity of such soil Pb amendments and therefore make interpretation of the results in an ecosystem context difficult. Given that in natural settings, these modifying factors are highly variable, the ISA notes that without quantitatively accounting for these factors, laboratory-derived "characterizations of exposure-response relationships would likely not be transferable outside of experimental settings" (ISA, section 1.7.1). Newly available soil invertebrate studies of multiple Pb concentrations in different soil systems also provide inconsistent results with respect to exposure-response relationships (ISA, sections 1.7.1 and 6.3.5). In consideration of these results, the ISA notes that "laboratory-amended artificial soils provide a poor model for predicting the toxicity of Pb-contaminated field soils, because aging and leaching processes,

[7] Elevated blood and tissue concentrations of Pb reported in avian species in some areas have been indicated to be a result of nonair pathways, including ingestion of lead-containing materials such as paint chips in urban areas and Pb ammunition fragments in other areas (ISA, sections 6.3.3.3 and 6.3.4.3).

along with variations in physiochemical proterties … influence metal bioavailability" (ISA, section 6.3.5)

As reported in both the ISA and the 2006 CD, direct evidence for community- and ecosystem-level effects comes from locations near sources of Pb to the environment, where Pb concentrations are much higher than typically observed environmental concentrations and often derive from multiple sources to multiple media (e.g. soil, water, air). Impacts of Pb on terrestrial ecosystems near smelters, mines, and other industrial sources have been studied for several decades (ISA, section 6.3.12.7). Atmospheric emissions of Pb from smelting and other industrial activities are commonly accompanied by other trace metals (e.g., Zn, Cu, Cd) and SO_2 that may cause toxic effects independently or in concert with Pb. Those impacts have been shown to include decreases in species diversity and changes in floral and faunal community composition in locations with histories of substantial Pb emissions affecting air concentrations that were likely well in exceedance of the current NAAQS, (e.g., 1977 CD, section 8.8; 1986 CD, section 8.1.1.3; 2006 CD, p. 7-15). Interpretation of ecosystem-level field studies with regard to ambient Pb levels associated with ecosystem and community effects is complicated by these confounding factors and the inherent variability in natural systems (ISA, section 6.3.12.7).

Limited new evidence of effects of Pb at the community scale has been reported. This evidence includes several studies of the ameliorative effects of mycorrhizal fungi on plant growth with Pb exposure as well as recently published research on soil microbial communities, which have been shown to be impacted by Pb in both composition and activity (ISA, sections 1.7.1 and 6.3.6). Many recent studies have been conducted using mixtures of metals, which have attempted to separate the effects of individual metals when possible. In studies that included only Pb, or where effects of Pb could be separated, soil microbial activity was generally diminished with increased Pb concentration and was shown in some of those cases to recover with time (ISA, section 6.3.6). In studies involving heavily contaminated soils from historic smelters, mining sites, or natural Pb deposits, microbial species and genotype composition were consistently altered after Pb exposure, and findings indicate that those alterations were long-lasting or permanent (ISA, section 6.3.6, pg 6-120).

A recent review has examined differences in species sensitivity, using blood Pb level in birds and mammals as an exposure index rather than external dose (ISA, section 6.3.9.1; Buekers et al., 2009). In this analysis, variation across organisms was lower with the blood Pb index compared to use of media concentration, and variation of Pb absorption from the diet of the organism largely accounted for the variation seen in the blood Pb. The analysis also suggests a larger variation in sensitivity across avian species tested compared to mammalian species with regard to the association between blood Pb concentration and toxicity (ISA, section 6.3.9.1).

Overall, recent studies cited in this review support previous conclusions about the effects of Pb on terrestrial ecosystems, namely that increasing soil Pb concentrations in areas of Pb contamination (e.g. mining sites and industrial sites) can cause decreases in microorganism abundance, diversity, and function. Previous reviews have also reported on effects on bird and plant communities (2006 CD, section AX7.1.3). The shifts in bacterial species and fungal diversity have been observed near long-established sources of Pb contamination (ISA, section 6.3.12.7). Most recent evidence for Pb toxicity to terrestrial plants, invertebrates and vertebrates is from single-species assays in laboratory studies which do not capture the complexity of bioavailability and other modifiers of effect in natural systems (ISA, section 6.3.12.7). Further, models that might account for modifiers of bioavailability have proven difficult to develop (ISA, p. 6-16).

Freshwater Ecosystems

Studies newly available in this review address trophic transfer of Pb in freshwater ecosystems (ISA, section 6.4.4.4). Evidence summarized in the 2006 CD indicated that measured concentrations of Pb in the tissues of aquatic organisms were generally higher in algae and benthic organisms than in higher trophic-level consumers, indicating that Pb was bioconcentrated but not biomagnified (ISA, section 1.7.2). Some recent studies indicate transfer of Pb in aquatic food webs; while other recent studies that have traced Pb in freshwater aquatic food webs have found, similar to the 2006 CD findings, that Pb concentration decreases with increasing trophic level (biodilution) (ISA, sections 1.7.2 and 6.4.4.4).

Evidence presented in the ISA further supports the findings of past CDs that Pb in freshwater can be highly toxic to aquatic organisms, with toxicity varying with species and life stage, duration of exposure, form of Pb, and water quality characteristics. The 2006 CD identified evidence of adverse growth effects on several species of freshwater algae from Pb exposure (2006 CD, Section 7.2.4). The ISA describes several new studies which expand the list of algal species for which these adverse effects have been identified (ISA, section 6.4.5.1). For vascular plants, the ISA describes additional evidence that oxidative damage, decreased photosynthesis and reduced growth occur with elevated Pb exposure (ISA, section 6.4.5.2).

Since the 2006 CD, there is some additional evidence for Pb effects on cellular processes in aquatic invertebrates. Recent studies of reproductive and developmental effects of Pb augment similar findings in the 2006 CD and add additional species information on reproductive endpoints for rotifers and freshwater snails as well as multigenerational effects of Pb in mosquito larvae (ISA, section 6.4.5.2). In the 2006 CD, study concentrations cited at which effects were observed in various aquatic invertebrates reflected an expansive range from 5μg/L (for acute toxicity to Pb nitrate in a test system at a hardness of 18 mg/L calcium carbonate) to greater than 8000 μg/L (for acute toxicity to Pb chloride at a hardness of 280-300

mg/L calcium carbonate) (ISA, p.1-45; 2006 CD, Table AX7-2.4.1). Recent studies cited in the ISA provide additional evidence of effects from chronic exposures. These findings are generally consistent with those of the previous review and reflect the variability of Pb toxicity under different environmental conditions and in species with varying sensitivity.

Evidence of effects in aquatic vertebrates presented in the ISA reiterates the findings of reproductive, behavioral, and growth effects stated in previous CDs. Some additional mechanisms of Pb toxicity in the gill and the renal system of fish have been elucidated since the 2006 CD as well as the identification of potential new molecular targets for Pb neurotoxicity (ISA, section 1.7.2).

As in terrestrial organisms, evidence presented in the ISA and prior CDs demonstrates the toxicity of Pb in aquatic ecosystems and the role of many factors, including Pb speciation and various water chemistry properties, in modifying toxicity (ISA, section 1.7.2). Since the 2006 CD, additional evidence for community and ecosystem level effects of Pb is available, primarily in microcosm studies or field studies with other metals present (ISA, section 6.4.11). Such evidence described in previous CDs includes alteration of predator-prey dynamics, species richness, species composition, and biodiversity. New studies available in this review provide evidence in additional habitats for these community and ecological-scale effects, specifically in aquatic plant communities and sediment-associated communities at both acute and chronic exposures involving concentrations similar to those previously reported (ISA, section 6.4.7). In many cases it is difficult to characterize the nature and magnitude of effects and to quantify relationships between ambient concentrations of Pb and ecosystem response due to existence of multiple ecosystem-level stressors, variability in field conditions, and differences in Pb bioavailability (ISA, sections 1.7.3.7 and 6.4.7). Additionally, the degree to which air concentrations have contributed to such effects in freshwater ecosystems is largely unknown.

Saltwater Ecosystems

As assessed in the ISA, the evidence for saltwater species and ecosystems is generally inadequate to draw conclusions regarding the effect of Pb (ISA, Tables 1-3 and II). The extent of newly available evidence in the context of prior available evidence is summarized here.

With regard to evidence in marine plants, recently available evidence on the toxicity of Pb to marine algae augments the 2006 CD findings of variation in sensitivity across marine species. Recent studies on Pb exposure include reports of growth inhibition and oxidative stress in a few additional species of marine algae (ISA, section 6.4.15).

Recent literature provides little new evidence of endpoints or effects in marine invertebrates beyond those reported in the 2006 CD. For example, some recent studies strengthen the evidence of Pb effects on enzymes and antioxidant activity in marine invertebrates and have identified an additional behavioral endpoint (i.e., valve closing speed in

juveniles of a marine scallop affected by 20 days exposure to 40-400 μg/L Pb nitrate) from those discussed previously (ISA, section 6.4.15.2). Also, as noted in the 2006 CD and supported by new studies reviewed in the ISA, Pb exposure negatively affects the growth of marine invertebrates (ISA, section 6.4.15.2). Recent studies also identify several species exhibiting particularly low sensitivity to high acute exposures (ISA, section 6.4.15.2).

Little new evidence is available of Pb effects on marine fish and mammals for reproductive, growth and survival endpoints that are particularly relevant to the population level of biological organization and higher (ISA, section 6.4.15). Evidence for effects at higher levels of biological organization in saltwater habitats is primarily supported by observations in a small number of microcosm and field studies where shifts in nematode community structure are the most commonly observed effects of Pb in experimentally contaminated sediments (ISA, sections 6.4.17 and Table 6-6). These types of studies were presented in the 2006 CD, and while there are new studies presented in the ISA, they primarily expand the types of communities (seagrass and amphipods) for which data exist but reach similar conclusions about community structure as the 2006 CD. However, there is no evidence of adverse effects in saltwater environments associated with current ambient air Pb concentrations, and linkages between any level of ambient air deposition and effects are unknown in all but a few organisms.

New studies on organism-level effects from Pb in saltwater ecosystems (ISA, section 6.4.15) provide little evidence to inform our understanding of linkages between atmospheric concentrations, ambient exposures in saltwater systems and such effects or our conclusions regarding the likelihood of adverse effects under conditions associated with the current NAAQS for Pb. Nor does the currently available evidence indicate significantly different exposure levels from the previous review at which ecological systems or receptors are expected to experience effects.

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

Critical loads analyses are a method that facilitates the assessment of ecosystem impacts as a result of pollutant loading which may arise from multiple pathways. A critical loads analysis approach can take into account what is known about the release of a specific chemical into the environment, its distribution and cycling within (and, as appropriate, across) ecosystems and each ecosystem's sensitivity to the chemical (ISA, section 6.1.3). Thus, this approach provides a conceptual framework for linking atmospheric deposition to environmental media concentration target values related to ecological endpoints associated with impairment (termed "critical limits"). Given the potential relevance to consideration of the secondary NAAQS for Pb, research in this area has been assessed in the current ISA and the 2006 CD.

The following generally accepted definition for a critical load of atmospheric pollutant deposition was developed from a pair of international workshops in the late 1980s (USEPA, 2008).

> *A quantitative estimate of an exposure to one or more pollutants below which significant harmful effects on specified sensitive elements of the environment do not occur according to present knowledge.*

As critical loads estimates reflect the current state of knowledge and policy priorities, as well as scientific and science policy judgments pertaining to the context for their use, there is no single "definitive" critical load for a natural resource. The state of scientific knowledge in this area can change in reflection of new information in various areas including, for example, new information about dose-response relationships; more comprehensive and detailed characterization of ecosystem resources (e.g., improved maps, inventories and survey datasets); more detailed information on pollutant concentrations (e.g., continuing time-series monitoring); and improved numerical models of pollutant fate and transport and ecosystem response. Science policy judgments reflect their context (e.g., goal for protection) as well as the state of scientific knowledge and consideration of its attendant strengths, limitations and uncertainties. Additionally inherent in critical loads analyses, due to their predictive nature, are a variety of assumptions, which may be relatively more or less numerous depending on the extent of knowledge on environmental processes for a particular application.

Some of the earliest uses of critical loads analyses date to consideration of the evidence for air pollutant deposition impacts on acidification of sensitive lake systems (2006 CD, section 7.3.2). Accordingly, the integrated science assessment of the evidence for the air pollutants nitrogen oxides (NOx) and sulfur oxides (SOx) described in detail the critical loads concept and the substantial evidence base pertaining to its application in consideration of acidification and eutrophication of aquatic ecosystems (USEPA, 2008). In this case, the substantial evidence of these ecosystem effects and the role of NOx/SOx atmospheric deposition was assessed (USEPA, 2008), and multiple alternative biological indicators, critical biological responses, chemical indicators, and critical chemical limits were presented that could be used to determine appropriate critical loads for aquatic and terrestrial ecosystems with regard to acidification and eutrophication (e.g., USEPA, 2008, Table 3-1, sections 3.2 and 3.3 and Annexes B, C and D).

A comparable evidence base does not exist with regard to impacts of air deposition of Pb on ecosystems. Nonetheless, the potential exists for critical loads to be an especially powerful tool allowing us to assess Pb atmospheric input into an ecosystem (e.g., deposition) and the potential ecological impairment resulting from that input. Critical load analyses are dependent on data relating pollutant release into an ecosystem (or multiple connected systems) from various sources with an understanding of the ecosystem concentrations likely to result in significant

harmful effects. In the context of Pb, a persistent, naturally occurring material, the bioavailability and mobility are influenced by multiple ecosystem characteristics; the data types for such analysis are numerous and may include information in the following areas:

- Pathways and rates by which Pb is released into ecosystems;
- Current Pb presence within and among ecosystems,[8] including spatial distribution, bioavailability and mobility;
- Fate and transport processes, within and among ecosystem components, as well as associated rates and factors influencing them;
- Forms and concentrations of Pb in ecosystem exposure media that likely result in critical ecosystem effects (e.g., concentrations that might relate to critical limits).

Additional information pertaining to the consideration of critical loads analyses in the context of ambient air standard setting for Pb includes factors that influence the transport and spatial pattern of deposition of airborne Pb, such as particle size (ISA, section 2.3.1).

During the last review, the 2006 CD assessed the available information on critical loads for Pb (2006 CD, section 7.3). This information included publications on methods and example applications, primarily in Europe, specific to the bedrock geology, soil types, vegetation, and historical deposition trends in each European country (2006 CD, p. E-24), with no analyses available for U.S. locations (2006 CD, sections 7.3.4-7.3.6).[9] As a result, the 2006 CD concluded that "[c]onsiderable research is necessary before critical load estimates can be formulated for ecosystems extant in the United States" (2006 CD, p. E-24). More generally, the 2006 CD identified the largest sources of uncertainty to include: derivation of the critical limit, Pb speciation, and soil runoff as an input to aquatic ecosystems (2006 CD, p. 7-45). Overarching conclusions reached included the following (2006 CD, p. 7-46).

> … *At this time, the methods and models commonly used for the calculation of critical loads have not been validated for Pb. Many of the methods neglect the speciation of Pb when estimating critical limits, the uptake of Pb into plants, and out flux of Pb in drainage water, limiting the utility of current models. Future efforts should focus on fully incorporating the role of Pb speciation into critical load models, and validating the assumptions used by the models.*

[8] In the case of Pb, in addition to the role of current air-related pathways, its presence in the ecosystem results from larger historical air emissions as well as contributions from nonair pathways which may be appreciable in some areas (e.g., industrial discharges to surface waters, contaminated waste disposal, drainage from metals mining sites, Pb ammunition near outdoor shooting areas).

[9] The work in Europe is largely responsive to developments, beginning in the late 1980s, associated with the consideration of the critical-load concept for a range of air pollutants in future international agreements limiting air pollutant emissions (2006 CD, section 7.3.2). A key impetus for such international cooperation efforts was research in the 1960s and 1970s indicating the role of long-range trans-boundary transport on ecosystems (e.g., sulfur emissions on the European continent contributing to acidification of Scandinavian lakes). Since about 1990, research has been exploring the use of critical loads in Europe for other air pollutants, including metals (2006 CD, section 7.3.2).

Accordingly, the quantitative assessment for the last Pb NAAQS review (ICF, 2006) did not involve critical load analyses.

Newly available evidence pertaining to critical loads analysis in this review includes limited recent research on consideration of bioavailability in characterizing Pb effect concentrations or indices and on modeling approaches to incorporate chemistry effects on Pb speciation and bioavailability (ISA, sections 6.3.7 and 6.4.8). With consideration of this information and the four critical load analysis studies newly available in this review (none of which are for U.S. ecosystems), the ISA does not modify the conclusions noted above from the 2006 CD (ISA, sections 6.1.3, 6.3.7 and 6.4.8). In summary, the new information in this review does not appreciably change our evidence base or further inform our understanding of critical loads of Pb, including critical loads in sensitive ecosystems.

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

There is no new evidence since the last review that substantially improves our understanding of the relationship between ambient air Pb and measurable ecological effects. As stated in the last review, the role of ambient air Pb in contributing to ecosystem Pb has been declining over the past several decades. It remains difficult to apportion exposure between air and other sources to better inform our understanding of the potential for ecosystem effects that might be associated with air emissions. As noted in the ISA, "[t]he amount of Pb in ecosystems is a result of a number of inputs and it is not currently possible to determine the contribution of atmospherically-derived Pb from total Pb in terrestrial, freshwater or saltwater systems" (ISA, section 6.5). Further, considerable uncertainties also remain in drawing conclusions from evidence of effects observed under laboratory conditions with regard to effects expected at the ecosystem level in the environment. In many cases it is difficult to characterize the nature and magnitude of effects and to quantify relationships between ambient concentrations of Pb and ecosystem response due to the existence of multiple stressors, variability in field conditions, and differences in Pb bioavailability at that level of organization (ISA, section 6.5). In summary, the ISA concludes that "[r]ecent information available since the 2006 Pb AQCD, includes additional field studies in both terrestrial and aquatic ecosystems, but the connection between air concentration and ecosystem exposure continues to be poorly characterized for Pb and the contribution of atmospheric Pb to specific sites is not clear" (ISA, section 6.5).

5.2 EXPOSURE AND RISK INFORMATION

The risk information available for this review and described here is based primarily on the pilot ecological risk assessment developed in the last review of the Pb NAAQS (henceforth referred to as the 2006 REA [ICF, 2006]). This information is described within the context of the evidence presented in the ISA that is newly available for this review. As described in the IRP, careful consideration of newly available information in this review led us to conclude that developing a new REA for this review was not warranted. In light of critical limitations and uncertainties that are still unresolved in the current evidence, staff concluded that currently available information does not provide the basis for developing a new quantitative risk and exposure assessment with substantially improved utility for informing the Agency's consideration of welfare effects and evaluation of the adequacy of the current secondary standard or alternatives (REA Planning Document, section 3.3). More specificallly, we also 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 (REA Planning Document, section 3.3). Based on their consideration of the REA Planning Document, the CASAC Pb Review Panel generally concurred with the conclusion that a new REA was not warranted in this review (Frey, 2011; Frey, 2013). Accordingly, the information described here is drawn primarily from the 2006 REA.

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

As discussed in section 1.3 above, the multimedia and persistent nature of Pb, the role of multiple exposure pathways (illustrated in Figure 1-1 above), and the contributions of nonair sources of Pb to exposure media all present challenges and contribute significant additional complexity to the ecological risk assessment that goes far beyond the situation for similar assessments typically performed for other NAAQS pollutants (e.g., that focus only on a single

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

5.2.1 Screening Assessment from Last Review

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

All three case studies and the national-scale assessment generally considered then-current or recent environmental conditions. In all cases but the primary Pb smelter case study, current air quality conditions were below the then-current NAAQS. Air Pb concentrations in the

primary Pb smelter case study exceeded the then-current NAAQS. A complete discussion of air quality in each of the case studies can be found in section 4 of the 2006 REA.

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

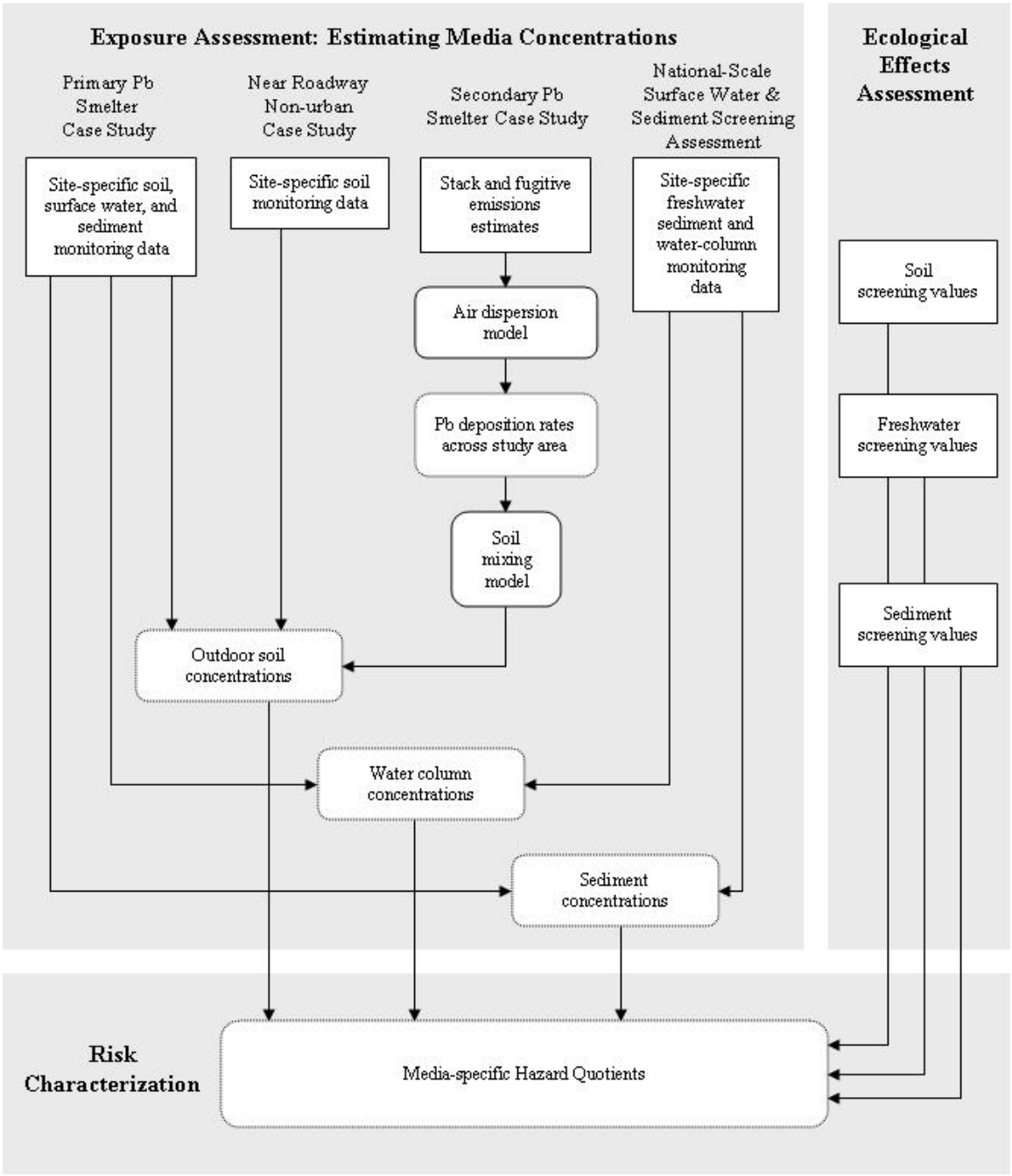

Figure 5-1. Analytical approach for screening-level ecological risk assessment in the last review (2006 REA, Exhibit 2-6).

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

5.2.2 Screening Assessment Results and Interpretation

The results for the ecological screening assessment for the three case studies and the national-scale screen for surface water and for sediment indicated a potential for adverse effect from ambient Pb to multiple ecological receptor groups in terrestrial and aquatic locations. [10] Below are descriptions of the location-specific case studies and the national screening assessment, key findings of the risk assessment for each, and an interpretation of the results with regard to past air conditions as well as the current standard (USEPA, 2007b).

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

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

Primary Pb Smelter Case Study

- The primary Pb smelter case study location is at one of the largest and longest-operating primary Pb smelters in the world (since 1892), the only one currently operating in the U. S. (ICF, 2006).[11]
- Concentrations of total Pb in several of the soil and sediment locations within the case study were measured in 2000 and exceeded screening values, indicating a potential for adverse effects to terrestrial and sediment-dwelling organisms.

[10] It is important to note that the screening values available and used in the assessment lacked adjustment for some critical measures of bioavailability. See uncertainty discussion below.

[11] As noted in section 2.1.2 above, this smelter ceased smelting operations at this facility at the end of 2013.

- While the contribution to these Pb concentrations from air as compared to nonair sources is not quantified, air emissions from this facility are substantial (ICF, 2006). In addition, this facility that has been emitting Pb for many decades, including some seven decades prior to establishment of any Pb NAAQS, such that it is likely air concentrations associated with the facility were substantial relative to the 1978 NAAQS, which it exceeded at the time of the last review. At the time of the previous review (2006) and also since the adoption of the current standard, concentrations monitored near this facility exceeded the level of the applicable NAAQS (USEPA 2007b, Appendix 5A and Appendix 2D of this document). Accordingly, this case study is not informative for considering the likelihood of adverse welfare effects related to Pb from air sources under air quality conditions associated with meeting the current Pb standard.

Secondary Pb Smelter Case Study

- The secondary Pb smelter location falls within the Alabama Coastal Plain in Pike County, Alabama, in an area of disturbed forests. The industrial facility in this case study is much younger than the primary Pb smelter, becoming active less than ten years prior to the establishment of the 1978 Pb standard.
- Estimates of total Pb concentration in soils (based on fate and transport modeling using 1997-2000 emissions data and data for similar locations measured in a 1995 study) exceeded screening values for plants, birds and mammals, indicating the potential for adverse effects to these groups.
- While the contributions from air-related Pb to the total Pb concentrations modeled in soils at this location are unclear, the facility continues to emit Pb, and the county where this facility is located does not meet the current Pb standard (Appendix 2D and 5A). Given the exceedances of the current standard, which likely extend back over 4 to 5 decades, this case study also is not informative for considering the likelihood of adverse welfare effects related to Pb from air sources under air quality conditions associated with meeting the current Pb standard.

Near-Roadway Non-urban Case Study

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

  These case study locations are highly impacted by past deposition of gasoline Pb. It is unknown whether current conditions at these sites exceed the 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 current standard. Similarly, those conditions likely resulted in Pb deposition associated with leaded gasoline that exceeds that being deposited under air quality conditions that would meet the current

Pb standard. Given this legacy, consideration of the potential for environmental risks from levels of air–related Pb associated with meeting the current Pb standard in these locations is highly uncertain.

Vulnerable Ecosystem Case Study

- This case study was focused on consideration of information available for the Hubbard Brook Experimental Forest (HBEF) in the White Mountain National Forest near North Woodstock, New Hampshire, which included a long record (from 1976 through 2000) of available data on concentration trends of Pb in three media (air or deposition from air, soil, and surface water).
- While no quantitative analyses were performed, a summary review of literature published on HBEF was developed. This literature 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; and (2) soil horizon analysis results showed Pb to have become more concentrated at lower soil depths over time, with the soil serving as a Pb sink, appreciably reducing Pb in pore water as it moves through the soil layers to streams (dissolved Pb concentrations were reduced from 5 μg/L to about 5 ng/L from surface soil to streams). Further, the available studies concluded insignificant contribution of dissolved Pb from soils to streams (ICF, 2006, Appendix E). 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 current standard.

National-scale Surface Water and Sediment Screen

- The national-scale screen was performed using a national database of surface water and sediment monitoring data (see Appendix 5A; ICF, 2006).
  - The screen identified 15 non-mining sites at which at least one surface water dissolved Pb concentration taken between 1994 and 2004 exceeded the chronic screening value, indicating a potential for adverse effect if concentrations were persistent over long time periods. The acute screening value was not exceeded at any of these locations.
  - Analysis of the sediment concentrations for the 15 sites taken from 1991 to 2000 analyzed in the surface water screen identified a subset for which concentrations exceeded the screening value, indicating a potential for adverse effects to sediment dwelling organisms.
- 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 the surface water Pb concentrations. For other locations, a lack of nearby nonair sources indicated a potential role for air sources to contribute to observed surface water Pb concentrations. Additionally, these concentrations may have been influenced by Pb in resuspended sediments and may reflect contribution of Pb from erosion of soils with Pb derived from historic as well as current air emissions.

There are multiple sources of uncertainty associated with different aspects of this assessment (discussed in detail in 2006 REA, section 7.4). For example, there are significant limitations and uncertainties associated with conclusions that can be drawn from the primary Pb smelter case study regarding the impact of atmospheric deposition under conditions associated with meeting the Pb NAAQS (past or present). Additionally, while case study locations were chosen with the objective of including locations for which recent Pb data were available and for which Pb exposures might be influenced by air-related Pb and not be dominated by nonair sources, there is significant uncertainty regarding the extent to which nonair sources and conditions associated with historic emissions (e.g., prior to establishment of past or present Pb NAAQS) have likely contributed to the Pb exposure estimates in some locations. The screening values available and used in the assessment (e.g., ambient water quality criteria, Eco-SSLs, sediment criteria) lacked adjustment for some critical measures of bioavailability and were sources of uncertainty with regard to potential for ecological risk. There is also uncertainty regarding the extent to which the screening values could identify potential hazards of Pb for some threatened or endangered species or unusually sensitive aquatic ecosystems (ICF 2006). Thus, while the assessment results are generally consistent with evidence-based observations of the potential influence of Pb on ecological systems, they are limited with regard to quantitative conclusions and potential hazard or risk associated with the Pb NAAQS, most particularly with regard to the current standard. The Hubbard Brook case study represents the most applicable case study for current conditions, and based on the previous analysis undertaken in the last review, current ambient air concentrations likely do not directly impact stream Pb levels under air quality conditions associated with meeting the current standard. With data limitations and the difficulties in apportioning Pb in ecosystems, there are no additional known locations in the U.S. that would likely provide additional insight into the effects of ambient Pb under the current standard, therefore no additional case study analyses were undertaken in this review.

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

In interpreting the results from the 2006 REA, we consider newly available evidence that may inform our interpretation of risk under the current standard. Factors necessary to alter our interpretation of risk would include new evidence of harm at lower concentrations of Pb, new linkages that enable us to draw more explicit conclusions as to the air contribution of environmental exposures, and/or new methods of interpreting confounding factors that were largely uncontrolled in the previous risk assessment. In general, however, the key uncertainties identified in the last review remain today, as described below.

With regard to new evidence of harm at lower concentrations, it is necessary to consider that the evidence of adversity due specifically to Pb in natural systems is limited, in no small part

because of the difficulty in determining the effects of confounding factors such as multiple metals and modifying factors influencing bioavailability in field studies. Modeling of Pb-related exposure and risk to ecological receptors is subject to a wide array of sources of both variability and uncertainty. Variability is associated with geographic location, habitat types, physical and chemical characteristics of soils and water that influence Pb bioavailability, terrestrial and aquatic community composition. Furthermore Pb uptake rates by invertebrates, fish, and plants may vary by species and season. For wildlife, variability also is associated with food ingestion rates by species and season, prey selection, and locations of home ranges for foraging relative to the Pb contamination levels (USEPA, 2005b).

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

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6 REVIEW OF THE SECONDARY STANDARD FOR LEAD

This chapter presents staff conclusions regarding the secondary Pb standard. These 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. These evaluations and staff conclusions have also taken into consideration CASAC advice and public comment on the draft PA and 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 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, 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). Staff conclusions are reported in Section 6.3. 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 takes into consideration the approaches used in the last Pb NAAQS review and involves addressing key policy-relevant questions in light of currently available scientific and technical information. 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. In drawing conclusions for the Administrator's consideration with regard to the secondary standard, 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 must draw 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 Clean Air Act. These provisions require the Administrator to establish a secondary standard that, in the judgment of the Administrator, is "requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of

the pollutant in the ambient air". In so doing, the Administrator seeks to establish standards that are neither more nor less stringent than necessary for this purpose.

6.1.1 Approach Used in the Last Review

In the last review, completed in 2008, the current secondary standard for Pb was set equal to the primary standard (73 FR 66964). As summarized in section 1.2.2 and described in more detail in section 4.1.1, the primary standard was substantially revised in the last review based on the much-expanded health effects evidence of neurocognitive effects of Pb in children. The level of the revised NAAQS is 0.15 $\mu g/m^3$. The averaging time was also revised to a rolling three-month period with a maximum (not-to-be-exceeded) form, evaluated over a three-year period. Compared to the previous averaging time of one calendar quarter, this revision was considered to be more scientifically appropriate and more protective for human health. The indicator of Pb-TSP was retained, reflecting the evidence that Pb particles of all sizes pose health risks (73 FR 67007). The 2008 decision considered the body of evidence as assessed in the 2006 CD (USEPA, 2006) as well as the 2007 Staff Paper assessment of the policy-relevant information contained in the 2006 CD and the screening-level ecological risk assessment (ICF, 2006; USEPA, 2007), the advice and recommendations of CASAC (Henderson 2007a, 2007b, 2008a, 2008b), and public comment.

In the 2008 review, the Staff Paper concluded, based on laboratory studies and current media concentrations in a wide range of locations, that it seemed likely that adverse effects were occurring, particularly near point sources, under the then-current standard (73 FR 67010). Given the limited data on Pb effects in ecosystems, and associated uncertainties, such as those with regard to factors such as the presence of multiple metals and historic environmental burdens, it was at the time, as it is now, necessary to look at evidence of Pb effects on organisms and extrapolate to ecosystem effects. Taking into account the available evidence and current media concentrations in a wide range of locations, the Administrator concluded that there was potential for adverse effects occurring under the then-current standard; however there were insufficient data to provide a quantitative basis for setting a secondary standard different than the primary (73 FR 67011). Therefore, citing a general lack of data that would indicate the appropriate level of Pb in environmental media that may be associated with adverse effects, as well as the comments of the CASAC Pb panel that a significant change to current air concentrations (e.g., via a significant change to the standard) was likely to have significant beneficial effects on the magnitude of Pb exposures in the environment, the secondary standard was revised to be consistent with the revised primary standard (73 FR 67011).

6.1.2 Approach for the Current Review

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

In developing conclusions in this review, we have taken into account both evidence-based and risk-based considerations framed by a series of policy-relevant questions. These questions are outlined in the sections below and generally discuss the extent to which we are able to better characterize effects and the likelihood of adverse effects in the environment under the current standard. Our approach to considering these questions recognizes that the available welfare effects evidence generally reflects laboratory-based evidence of toxicological effects on specific organisms exposed to concentrations of Pb. It is widely recognized, however, that environmental exposures from atmospherically derived Pb are likely to be lower and/or accompanied by significant confounding and modifying factors (e.g., other metals, acidification), which increases our uncertainty about the likelihood and magnitude of organism and ecosystem responses.

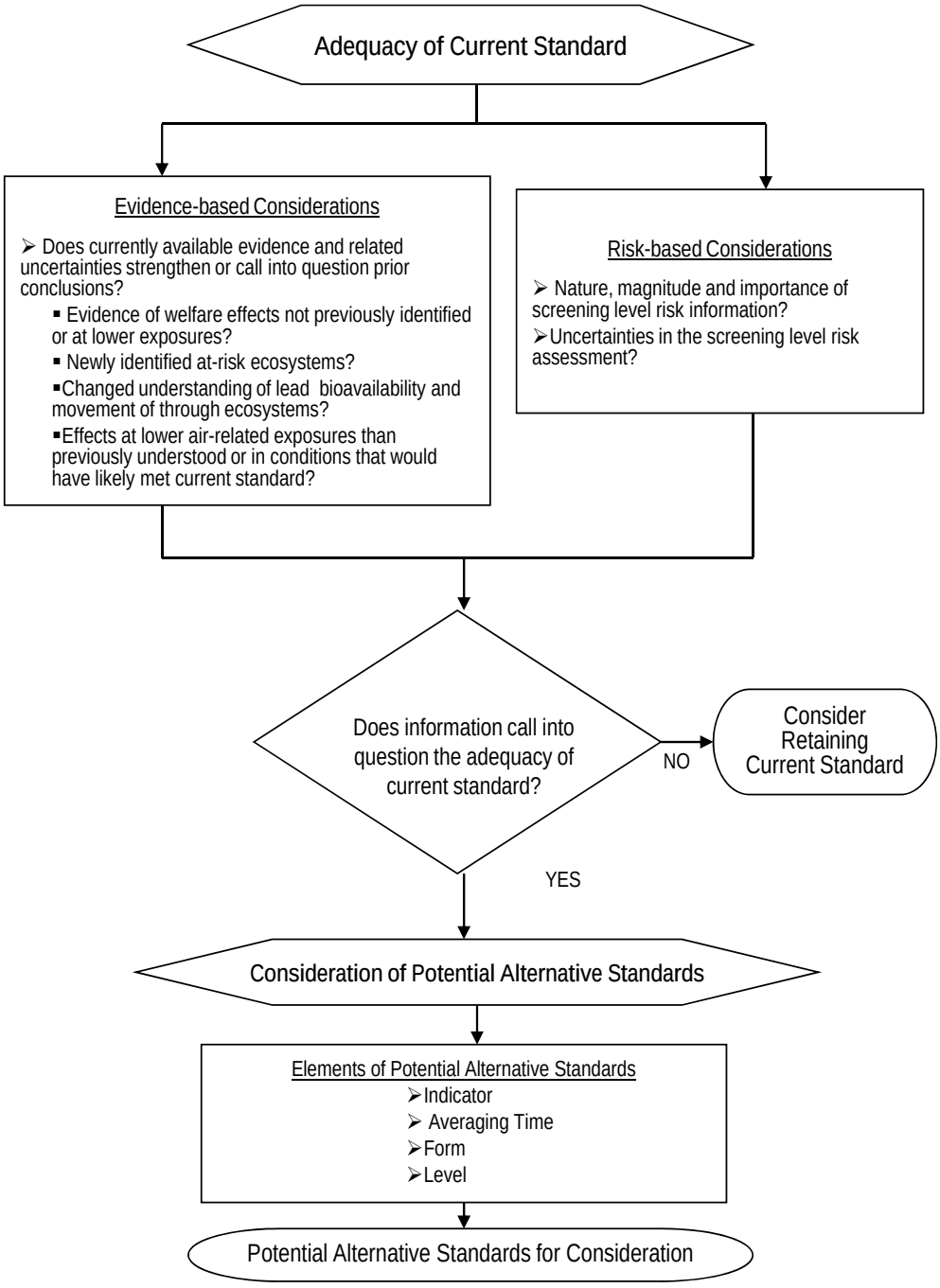

Figure 6-1. Overview of approach for review of current secondary standard.

6.2 ADEQUACY OF THE CURRENT STANDARD

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

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

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

6.2.1 Evidence-based Considerations

In considering the welfare effects evidence with respect to the adequacy of the current standard, we consider the array of evidence newly assessed in the ISA with regard to the degree to which this evidence supports conclusions about the effects of Pb in the environment that were drawn in the last review and the extent to which it reduces previously recognized areas of uncertainty. Further, we consider the current evidence and associated conclusions about the potential for effects to occur as a result of the much lower ambient Pb concentrations allowed by the current secondary standard (set in 2008) than those allowed by the prior standard, which was the focus of the last review. These considerations, discussed in the context of the specific questions below, inform our conclusions regarding the extent to which the evidence supports or calls into question the adequacy of protection afforded by the current standard.

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

The current evidence continues to support our conclusions from the previous review regarding key aspects of the ecological effects evidence for Pb and the effects of exposure associated with levels of Pb occurring in ecological media in the U.S. Our conclusions in this regard are based on consideration of the assessment of the currently available evidence in the ISA, particularly with regard to key aspects summarized in chapter 5 of this PA, in light of the assessment of the evidence in the last review as described in the 2006 CD and summarized in the notice of final rulemaking (73 FR 67008). Key aspects of conclusions drawn in the last review (73 FR 67010) are summarized below.

- There are several difficulties to quantifying the role of recent air emissions of Pb in the environment:

  - While the removal of Pb from on-road gasoline and reductions in industrial emissions have resulted in dramatic reductions in overall deposition rates (2006 CD, pp. AX7-34 and AX7-103), Pb deposited before the 1978 standard, and the relatively lesser amount deposited since, is still present in soils and sediments.
  - Historic Pb from gasoline-derived and other air emissions as well as nonair sources has produced a legacy of Pb in forest soils that continues to move slowly through these systems (2006 CD, section AX7.1.4.3).
- The evidence of adversity in natural systems is very sparse due in no small part to the difficulty in determining the effects of confounding factors such as multiple metals or factors influencing bioavailability of Pb in field studies (2006 CD, E-19).
- For areas influenced by stationary sources of air Pb that met the standard considered in the last review at the time of the last review (although they likely did not throughout their active period), concentrations of Pb in soil may exceed by many orders of magnitude the concentrations which are considered harmful to laboratory organisms (2006 CD, sections 3.2 and AX7.1.2.3).
- Environmental conditions exist in which Pb-associated adverse effects to aquatic organisms and thereby ecosystems may be anticipated given experimental results. While the evidence does not indicate that dissolved Pb in surface water constitutes a threat to those ecosystems that are not directly influenced by point sources, the evidence regarding Pb in sediment is less clear. Some areas with long term historical deposition of Pb to sediment from a variety of sources as well as areas influenced by point sources have the potential for adverse effects to aquatic communities (2006 CD, sections AX7.2.2.2 and AX7.2.4).

The range of effects that Pb can exert on terrestrial and aquatic organisms indicated by information available in the current review is summarized in the ISA (ISA, sections 1.7, 6.3 and 6.4) and largely mirror the findings of from the previous review (summarized above). The integrated synthesis contained in the ISA conveys how effects of Pb can vary with species and life stage, duration of exposure, form of Pb, and media characteristics such as soil and water chemistry. A wide range of organism effects are recognized, including effects on growth, development (particularly of the neurological system) and reproductive success (ISA, section 6.3 and 6.4). Lead is recognized to distribute from the air into multiple environmental media, as summarized in section 1.3 above, contributing to multiple exposure pathways for ecological receptors. As discussed in section 5.1 above, many factors affect the bioavailability of Pb to receptors in terrestrial and aquatic ecosystems, contributing to differences between laboratory-

assessed toxicity and Pb toxicity in these ecosystems, challenging our consideration of environmental impacts of Pb emitted to ambient air.

In studies in a variety of ecosystems, adverse ecosystem-level effects (including decreases in species diversity, loss of vegetation, changes to community composition, primarily in soil microbes and plants, decreased growth of vegetation, and increased number of invasive species) have been demonstrated near smelters, mines and other industries that have released substantial amounts of Pb, among other materials, to the environment (ISA, sections 6.3.12 and 6.4.12). As noted in section 5.1 above, however, our ability to characterize the role of air emissions of Pb in contributing to these effects is complicated because of coincident releases to other media and of other pollutants. Co-released pollutants include a variety of other heavy metals, in addition to sulfur dioxide, which may cause toxic effects in themselves and may interact with Pb in the environment, contributing uncertainty to characterization of the role of Pb from ambient air with regard to the reported effects. These uncertainties limit our ability to draw conclusions regarding the extent to which Pb-related effects may be associated with ambient air conditions that would meet the current standard.

The role of historically emitted Pb, as discussed in section 1.3.2 above, poses additional complications in addressing this question. The vast majority of Pb in the U.S. environment today, particularly in terrestrial ecosystems, was deposited in the past during the use of Pb additives in gasoline (2006 CD, pp. 2-82, AX7-36 to AX7-38, AX7-98; Johnson et al., 2004), although contributions from industrial activities, including metals industries have also been documented (ISA, section 2.2.2.3, Jackson et al., 2004). The gasoline-derived Pb was emitted in very large quantities (2006 CD, p. AX7-98 and ISA, Figure 2-8) and predominantly in small sized particles which were widely dispersed and transported across large distances, within and beyond the U.S. (ISA, section 2.2). As recognized in sections 2.3.1 and 2.3.3.2 above, historical records provided by sediment cores in various environments document the substantially reduced Pb deposition (associated with reduced Pb emissions) in many locations (ISA, section 2.2.1). As Pb is persistent in the environment, these substantial past environmental releases are expected to generally dominate current nonair media concentrations. There is limited evidence to relate specific ecosystem effects with current ambient air concentrations of Pb through deposition to terrestrial and aquatic ecosystems and subsequent movement of deposited Pb through the environment (e.g., soil, sediment, water, organisms). The potential for ecosystem effects of Pb from atmospheric sources under conditions meeting the current standard is difficult to assess due to limitations on the availability of information to fully characterize the distribution of Pb from the atmosphere into ecosystems over the long term, as well as limitations on information on the bioavailability of atmospherically deposited Pb (as affected by the specific characteristics of the receiving ecosystem). Therefore, while information available since the 2006 CD includes

additional terrestrial and aquatic field studies, "the connection between air concentration and ecosystem exposure and associated potential for welfare effects continues to be poorly characterized for Pb" (ISA, section 6.5). Such a connection is even harder to characterize with respect to the current standard than it was in the last review with respect to the previous, much higher, standard.

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

In the characterization of effects associated with Pb in ambient air for the last review, the areas of largest uncertainty were identified to be associated with our abilities to determine the extent of lead-related effects in the natural environment and to apportion effects between air and nonair sources, and with the relationship between effects and environmental conditions associated with the standard. While we have some new evidence with regard to bioavailability and its role in influencing the potential for effects in the environment, overall these remain the major sources of uncertainty in our evaluation of effects and exposure evidence for Pb in ecosystems. Moreover, the impact of these uncertainties is larger in this review given our current focus on a secondary standard much lower than that which was the focus of the last review.

With regard to the extent of lead-related effects in the natural environment, it remains difficult to assess the concentrations at which Pb elicits specific effects in terrestrial and aquatic systems, due to the influence of other environmental variables on both Pb bioavailability and toxicity and also due to substantial species differences in Pb susceptibility (ISA, sections 6.3.2-6.3.4 and 6.4.2 -6.4.5). There is little new information that would facilitate extrapolation of evidence on individual species, generally from controlled laboratory studies, to conclusions regarding adversity to populations or ecosystems within the natural environment. For example, in terrestrial systems, evidence reviewed in the ISA (ISA, sections 6.2.3 and 6.2.4) demonstrates that exposure to Pb is generally associated with negative effects on growth, reproduction and survival in terrestrial ecosystems. Many factors, including species composition and various soil physiochemical properties, interact strongly with Pb concentration to modify effects. Also, the changes associated with environmental aging of lead-contaminated material is a particularly important factor in terrestrial systems, where soil is the main route of exposure, however, aging of lead-contaminated soil is difficult to reproduce experimentally (ISA, sections 6.3.9.3 and 6.3.11). Without quantitatively accounting for these factors, laboratory-derived "characterizations of exposure-response relationships would likely not be transferable outside of experimental settings" (ISA, p. 1-42).

With regard to the role of air-related Pb, as discussed above and noted in the ISA, "the connection between air concentration and ecosystem exposures continues to be poorly

characterized for Pb and the contribution of atmospheric Pb to specific sites is not clear" (ISA, p. 6-264). Thus, the limited evidence and associated uncertainties connecting air concentrations to ecosystem exposures and possible effects, in combination with those that influence Pb bioavailability and ecosystem mobility, are areas particularly limiting the extent to which critical loads analyses might otherwise be useful to our assessment in this review, as discussed in section 5.1. Thus, the evidence is lacking in quantitative relationships between Pb in ambient air and welfare effects, including identification of a specific exposure metric or index (e.g., mathematical formulation of averaging time and concentration) by which to consider ambient air Pb concentrations with regard to the potential for adverse effects to public welfare. In the decision in the last review to set the secondary standard equal to the primary standard, EPA similarly recognized a lack of evidence that related welfare effects to Pb in ambient air and from which differing standard elements (e.g., averaging time and concentration) might be derived for the secondary standard.[1] These limitations in the evidence base remain in the current review.

As in the last review, consideration of the environmental risks and potential welfare effects associated with the current standard is complicated by the environmental burden associated with air Pb concentrations, predominantly in the past, that exceeded both the previously existing standard and the current standard (USEPA, 2007, section 6.4.4.2). For example, a large portion of Pb deposited before the current standard was enacted is likely still present in soils and sediments, and historic Pb from gasoline (and other historic sources) continues to move slowly through systems as does Pb derived from current air and nonair sources. As a general matter, the currently available evidence, as assessed in the ISA, does not significantly reduce any of the areas of uncertainty described here which, given the standard considered in the present review (with its much lower level than the previous standard), weigh heavily in our consideration of the adequacy of the current standard.

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

[1] As described in section 6.1.1 above, the EPA concluded in the last review that the then-current secondary standard did not provide requisite protection from effects adverse to public welfare. In considering the appropriate revision, EPA recognized that the Agency lacked the relevant data to provide a clear quantitative basis for setting a secondary Pb NAAQS that differs from the primary standard, yet recognized that significant beneficial effects were likely to result in terms of reduced magnitude of Pb exposures in the environment and associated toxicity impacts on ecosystems from a significant change to the level for the secondary standard, such as the order-of-magnitude change which was achieved by setting the secondary standard identical to the revised primary standard (73 FR 67012).

consideration of the screening-level ecological risk assessment results from the previous review as described in the 2006 REA and summarized in the notice of final rulemaking (73 FR 67009) and in light of the currently available evidence in the ISA. 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 (Appendices 2D and 5A).

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 and that there was "little evidence that sites affected primarily by long-range Pb transport [such as this one] have experienced significant effects on ecosystem structure or function" (2006 CD, p. AX-98). Further, it is unlikely that conditions have changed from the data through 2000 on which the previous conclusions were based, 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 the surface water Pb concentrations. For other locations, a lack of nearby nonair sources indicated a potential role for air sources to contribute to observed surface water Pb concentrations. Additionally, these concentrations may have been influenced by Pb

in resuspended sediments and/or reflect contribution of Pb from erosion of soils with Pb derived from historic as well as current air emissions.

Although the available risk and exposure information continues to be sufficient to conclude that the 1978 standard was not providing adequate protection to ecosystems, that information, when considered with regard to air-related ecosystem exposures likely to occur with air Pb levels that just meet the now-current standard, does not provide evidence that the current secondary standard (set in 2008) is inadequate.

6.2.3 CASAC Advice

In our consideration of the adequacy of the current standards, in addition to the evidence- and risk/exposure-based information discussed above, we have also considered the advice and recommendations of CASAC, based on their review of the ISA and the earlier draft of this document. Comments from the public on earlier drafts of this document did not address the adequacy of the current secondary standard.[2]

In their advice and comments conveyed in the context of their review of the draft PA, the CASAC agreed with staff's preliminary conclusions that the available information since the last review is not sufficient to warrant revision to the secondary standard (Frey, 2013). On this subject, the CASAC letter said the following.

> *Overall, the CASAC concurs with the EPA that the current scientific literature does not support a revision to the Primary Lead (Pb) National Ambient Air quality Standard (NAAQS) nor the Secondary Pb NAAQS.*

The CASAC also recognized the many uncertainties and data gaps in the new scientific literature and recommended that research be performed in the future to address these limitations.

> *Given the existing scientific data, the CASAC concurs with retaining the current secondary standard without revision. However, the CASAC also notes that important research gaps remain. For example questions remain regarding the relevance of the primary standard's indicator, level, averaging time, and form for the secondary standard. Other areas for additional research to address data gaps and uncertainty include developing a critical loads approach for U.S. conditions and a multi-media approach to account for legacy Pb and contributions from different sources. Addressing these gaps may require reconsideration of the secondary standard in future assessments.*

[2] All written comments submitted to the Agency will be available in the docket for this rulemaking, as will be transcripts of the public meetings held in conjunction with CASAC's review of the earlier draft of this document, of the REA Planning Document and of drafts of the ISA.

6.3 STAFF CONCLUSIONS ON THE SECONDARY STANDARD

This section describes staff conclusions regarding adequacy of the current secondary Pb standard. These conclusions are based on considerations described above and in the discussion below regarding the currently available scientific evidence summarized in the ISA and prior CDs and the risk and exposure information drawn from the 2006 REA. These conclusions also take into account advice from CASAC and public comment on the draft PA and preliminary conclusions.

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

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

With respect to evidence-based considerations, the body of evidence on the ecological effects of Pb, expanded in some aspects since the last review, continues to support identification of ecological effects in organisms relating to growth, reproduction, and survival as the most relevant endpoints associated with Pb exposure. In consideration of the appreciable influence of site-specific environmental characteristics on the bioavailability and toxicity of environmental Pb in our assessment here, we give greater weight to studies conducted under conditions most closely reflecting the natural environment as compared to laboratory-based exposures. The currently available evidence, while somewhat expanded since the last review, does not include evidence of significant effects at lower concentrations or evidence of higher level ecosystem effects beyond those reported in the last review. There continue to be significant difficulties in interpreting effects evidence from laboratory studies to the natural environment and linking those effects to ambient air Pb concentrations. Further, we are aware of no new critical loads information that would inform our interpretation of the public welfare significance of the effects of Pb in various ecosystems (as discussed in section 5.1). In summary, while new research has added to the understanding of Pb biogeochemistry and expanded the list of organisms for which Pb effects have been described, there remains a significant lack of knowledge about the potential for adverse effects on public welfare from ambient air Pb in the environment and the exposures that occur from such air-derived Pb, particularly under the current standard.

With respect to exposure/risk-based considerations, we recognize the complexity of interpreting the previous risk assessment with regard to the ecological risk of ambient air Pb associated with conditions meeting the current standard and the associated limitations and uncertainties of such assessments. For example, the location-specific case studies as well as the national screen conducted in the last review reflect both current air Pb deposition as well as past air and nonair source contributions. We conclude that while the previous assessment is

consistent with and generally supportive of the evidence-based conclusions about Pb in the environment, the limitations of apportioning Pb between past and present air contributions and between air and nonair sources remain significant.

In now considering the adequacy of the current standard in this review, we have considered the ecological and welfare effects evidence assessed in the ISA and discussed in sections 5.1, 2.3 and 6.2.1 above, as well screening-level exposure;/risk information gained from the 2006 REA discussed in sections 5.2 and 6.2.2 above, and staff judgments on associated implications with regard to public welfare. As summarized, in section 6.2.1 above, the scientific evidence presented in detail in the ISA, inclusive of that newly available in this review, is not substantively changed, most particularly with regard to our needs in reviewing the current standard, from the information that was available in the last review. The evidence base does not indicate that the current standard is inadequate. Further, our updated consideration of the screening-level risk information from the 2006 REA, as summarized in section 6.2.2 above, additionally does not provide evidence that the current secondary standard is inadequate. Thus, we conclude that the currently available evidence and exposure/risk information do not call into question the adequacy of the current standard to provide the requisite protection for public welfare. Accordingly, we reach the conclusion that it is appropriate to consider retaining the current secondary standard without revision.

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 supporting 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 welfare-based secondary standard for Pb.

- Our understanding of the extent and degree of adverse impact occurring in the environment due to Pb (from atmospheric and other sources) is incomplete and leads to uncertainty in characterizing environmental effects of Pb, which is necessary to assess potential associated effects on public welfare.
  - 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 as a result of atmospheric input and are usually co-occurring with elevated concentrations of other pollutants.

- Many ecosystem-specific characteristics affect bioavailability and associated toxicity, contributing uncertainty to our interpretation of study results from laboratory test-systems. For example, the role of modifying factors such as soil and water chemistry on Pb toxicity continues to be a source of uncertainty when determining the degree of adverse impact from Pb. [3]

- Evidence regarding relationships between organism-level effects of Pb (e.g., reproduction, growth, and survival) and effects at the population level and higher is lacking especially in natural systems.

- A better understanding of the potential for adverse effects from Pb in estuarine and marine systems is needed.

- There is appreciable uncertainty in our understanding of the current dynamics of Pb in U.S. ecosystems.

  - In general, the connection between air concentration of Pb and ecosystem exposure continues to be poorly characterized and is complicated by the legacy of historical inputs both from air and, in some systems, also from other sources.

  - There is uncertainty in characterizing the fate and transport of Pb in the environment through air, water, and soil media. This limits our ability to account for the role of past Pb releases in current media concentrations and to link effects to current air contributions.

  - An important source of uncertainty in characterizing the effects of air-related Pb in some U.S. ecosystems is that associated with distinguishing air-related Pb from other sources of Pb. It is difficult to determine the portion of Pb occurring in some ecosystems that derives from the atmosphere as compared to that from other sources.

- Development of critical loads information for U.S. ecosystems would allow for a much improved understanding of the significance of air contributions to ecosystems and ecological receptor exposures, as well as that of contributions from other sources. Development of a critical loads approach, with sensitivity analysis, would inform identification of processes critical to ecological receptor exposures and toxicity.

  - Such critical loads analyses could inform characterization of the variability of response across ecosystems to determine which ecosystems are most sensitive.

  - Additional research is needed to provide inputs for development of critical loads, particularly for a broader range of ecosystem-level endpoints.

- Information is currently lacking to inform identification of standard elements, such as averaging time and form, specifically reflecting a ecological and public welfare context for ambient air Pb.

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[3] Further uncertainty accompanies the interpretation of toxicity data from test systems involving artificial media such as hydroponic systems, agar or culture media.

6.5 REFERENCES

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APPENDICES

APPENDIX 2A

RECENT REGULATORY ACTIONS ON STATIONARY SOURCES OF LEAD

The table below identifies recent actions projected to result in reductions in Pb emissions from stationary sources.

Action	Source Categories Affected	Citation	Estimated Pb Emissions Reduction (tons per year)	Year of Compliance (year in which reductions estimated to begin)
NESHAP	Secondary Lead Smelting	77 FR 555 (1/5/2012)	13.6	2014
NESHAP	Primary Lead Smelting	76 FR 70834 (11/15/2011)	10	2013
NESHAP	Area Sources: Clay Ceramics Manufacturing, Glass Manufacturing, and Secondary Nonferrous Metals Processing	72 FR 73179 (12/26/2007)	Unquantified*	2007, 2009, 2007
NESHAP	Area Sources: Electric Arc Furnace Steelmaking Facilities	72 FR 74088 (12/28/2007)	Unquantified*	2008 or 2010
NESHAP	Iron and Steel Foundries Area Sources	73 FR 225 (1/2/2008)	Unquantified*	2010
NESHAP	Area Source Standards for Aluminum, Copper, and Other Nonferrous Foundries	74 FR 30366 (6/25/2009)	Unquantified*	2011
NESHAP, NSPS	Portland Cement Manufacturing Industry	78 FR 10006 (2/12/2013)	6.72	2015
EGES, NSPS	Commercial and Industrial Solid Waste Incineration Units	76 FR 9112 (02/07/2013)	2.51	2018
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)	1.2-1.5	2016
NESHAP	Major Sources: Industrial, Commercial, and Institutional Boilers, and Process Heaters	78 FR 7138 (1/31/2013)	Unquantified*	2016
EGES, NSPS	Standards of Performance for New Stationary Sources and Emissions Guidelines for Existing Sources: Hospital/Medical/Infectious Waste Incinerators	74 FR 51367 (10/6/2009)	0.16	2014

EGES = Emissions Guidelines for Existing Sources
NESHAP = National Emission Standards for Hazardous Air Pollutants
NSPS = New Source Performance Standards
*Unquantified = Reductions in Pb emissions expected from controls in response to regulatory action have not been quantified.

APPENDIX 2B

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.

with regard to Pb emissions estimates for some sources. For example, we have not yet developed estimates for the NEI of Pb emissions for the miscellaneous categories of on-road emissions (e.g., combustion of fuel with Pb traces, lubricating oil, mechanical wear of vehicle components, etc.) and Pb that may be emitted from wildfires, or for emissions associated with resuspension of Pb residing in roadway dust and nearby surface soil in areas not otherwise associated with industrial facilities (see section 2.1.2).

The 2008 NEI underwent extensive external review, including a review of the process for developing the inventory which includes extensive quality assurance and quality control steps (QA/QC). We provided feedback reports to point source data providers and we posted several versions of the inventory on our website. We also conducted additional QA targeted at facilities with appreciable Pb emissions in previous years to ensure that 2008 Pb emissions were reported, and we augmented with TRI where facilities with annual emissions greater than 0.5 tpy in 2005 were not reported to NEI in 2008 due to Pb emissions reporting thresholds (ftp://ftp.epa.gov/EmisInventory/20008v2/doc/2008neiv2_issues.xlsx). Further, there was additional QA/QC conducted for emission inventory information for facilities that are included in Risk and Technology Review source categories.[3] As a result we have strong confidence in the quality of the data for these facilities. In summary, generic limitations to the 2008 NEI include the following.

- Consistency: The 2008 NEI for Pb is a composite of emissions estimates generated by state and local regulatory agencies, industry, and EPA. Because the estimates originated from a variety of sources, as well as for differing purposes, they will in turn vary in quality, whether Pb is reported for particular source types, method of reporting compound classes, level of detail, and geographic coverage.
- Variability in Quality and Accuracy of Emission Estimation Methods: The accuracy of emission estimation techniques varies with pollutants and source categories. In some cases, an estimate may be based on a few or only one emission measurement at a similar source. The techniques used and quality of the estimates will vary between source categories and between area, major, and mobile source sectors. Generally, the more review and scrutiny given to emissions data by states and other agencies, the more certainty and accuracy there is in those estimates.

[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 2C

CRITERIA FOR AIR QUALITY DATA ANALYSIS

Criteria for the 2010-2012 data analysis presented in Figures 2-10 through 2-13 are as listed below, with abbreviations defined following this list.

- Years utilized were 2010-2012. November and December 2009 data were also used for the rolling 3-month average analysis.
- Data were extracted from EPA's Air Quality System in June of 2013.
- Data for parameter 14129 (Pb-TSP FRM/FEM LC) were used for Pb-TSP analysis; data for parameters 84128 (Pb-$PM_{2.5}$ STP) and 88128 (Pb-$PM_{2.5}$ LC) were used for Pb-PM2.5 analysis; and data for parameters 82128 (Pb-PM_{10} STP), 85128 (Pb-PM_{10} LC), and 85129 (Pb-PM_{10} FRM/FEM 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 FRM/FEM parameter data takes precedence over LC (non-FRM/FEM) parameter data, and those data take precedence overSTP parameter data; also, within those categories, lower POC numbers take precedence over higher POC numbers.
- For maximum rolling 3-month average analysis, the 2010-2012 databases also encompassed the prior 2 months (i.e., November and December 2009 data): 3 or more observations were needed to make a valid monthly; 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-year metric. Thus, the shortest duration that could potentially meet these criteria for the maximum 3-month average analysis is 8 months. The 3-year maximum rolling 3-month average metric (by size cut for 2010-2012) 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; 6 valid months were required to make a valid year; and 1, 2, or 3 valid years were required to make a valid 3-year metric. Thus, the shortest duration that could potentially meet these criteria for the maximum monthly analysis is 6 months. The 3-year maximum monthly metric (by size cut for 2010-2012) 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; 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. Thus, the shortest duration that could potentially meet these criteria for the annual average analysis is 6 months. The 3-year annual mean metric (by size cut for 2010-2012) was identified as the average of the 1, 2, or 3 (2010, 2011, and/or 2012) valid annual means.

Definitions for Abbreviations

FEM – Federal equivalent method

FRM – Federal reference method

LC – Local conditions

POC – Parameter occurrence code

STP – Standard temperature and pressure

The process for classifying monitors as "source oriented" or "non-source-oriented" began with the application of these criteria: (a) a minimum of 0.5 cumulative tons per year of Pb emissions within 1 mile of the monitoring site based on 2008 NEI and/or (b) monitors identified as "source-oriented" within AQS based on the same-name Monitoring Objective tag. Next, the Regional monitoring leads reviewed the listings and provided local, and often monitor-specific information and lastly Google Earth and more focused queries were applied.

The 14 monitors sited for the airport monitoring study, along with the 3 monitors sited at airports estimated to emit at least 1 tpy were classified as source-oriented (airport) sites and the remaining source-oriented sites were classified as source-oriented (non-airport) sites.

Among the remaining sites that were not classified as "source-oriented", previous source-oriented sites were those that had previously been active source sites but for which current information indicated the impacting facility had closed. The particular circumstances related to the emission sources associated with these nine monitoring sites vary considerably. In some instances the emission sources have been closed for more than a decade and the facility locations have undergone remediation. For other sources, production and clean-up status was not fully ascertained.

Non-source sites were what was left after making the airport source-oriented, non-airport source-oriented, and previous source-oriented designations.

APPENDIX 2D

Table 2D-1. Pb-TSP concentrations at Pb-TSP sites, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)	Non-airport source-oriented (during 2010-2012)	Airport source-oriented	Previous source-oriented
Alabama	Limestone	010830005	0.009	0.007	0.012	0	1	0
Alabama	Pike	011090003	1.296	0.613	2.262	1	0	0
Alaska	Anchorage Municipali	020200051	0.067	0.054	0.073	0	1	0
Arizona	Gila	040071002	0.267	0.131	0.306	1	0	0
Arizona	Gila	040078000	0.061	0.030	0.077	1	0	0
Arizona	Maricopa	040134018	0.038	0.031	0.049	0	1	0
Arizona	Pima	040191028	0.002	0.002	0.003	0	0	0
California	Fresno	060190011	0.005	0.004	0.005	0	0	0
California	Imperial	060250005	0.026	0.017	0.031	0	0	0
California	Los Angeles	060371103	0.013	0.009	0.014	0	0	0
California	Los Angeles	060371302	0.015	0.007	0.014	0	0	0
California	Los Angeles	060371402	0.061	0.033	0.092	0	1	0
California	Los Angeles	060371403	0.110	0.055	0.124	1	0	0
California	Los Angeles	060371404	0.108	0.042	0.140	1	0	0
California	Los Angeles	060371405	0.450	0.241	0.523	1	0	0
California	Los Angeles	060371406	0.072	0.035	0.085	1	0	0
California	Los Angeles	060371602	0.013	0.008	0.017	0	0	0
California	Los Angeles	060374002	0.009	0.005	0.010	0	0	0
California	Los Angeles	060374004	0.010	0.006	0.010	0	0	0
California	Los Angeles	060375005	0.006	0.003	0.008	0	0	0
California	Riverside	060651003	0.008	0.005	0.010	0	0	0
California	Riverside	060658001	0.008	0.006	0.010	0	0	0
California	San Bernardino	060711004	0.010	0.006	0.012	0	0	0
California	San Bernardino	060719004	0.011	0.007	0.013	0	0	0
California	San Diego	060730003	0.006	0.005	0.007	0	0	0
California	San Diego	060731020		0.127	0.234	0	1	0
California	San Diego	060731021		0.058	0.074	0	1	0
California	San Mateo	060812002	0.331	0.255	0.401	0	1	0
California	Santa Clara	060852010	0.119	0.090	0.151	0	1	0
California	Santa Clara	060852011	0.093	0.070	0.102	0	1	0
Colorado	Arapahoe	080050007	0.023	0.014	0.035	0	1	0

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Table 2D-1. Pb-TSP concentrations at Pb-TSP sites, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)	Non-airport source-oriented (during 2010-2012)	Airport source-oriented	Previous source-oriented
Colorado	Denver	080310025	0.010	0.006	0.013	0	0	0
District of Columb	District of Columbia	110010043	0.004	0.003	0.005	0	0	0
Florida	Hillsborough	120570100	0.044	0.025	0.100	1	0	0
Florida	Hillsborough	120571066	0.984	0.374	1.959	1	0	0
Florida	Hillsborough	120571073	0.423	0.151	0.977	1	0	0
Georgia	Bartow	130150003	0.027	0.015	0.039	1	0	0
Georgia	DeKalb	130890003	0.004	0.003	0.006	0	0	0
Georgia	Muscogee	132150009	0.230	0.128	0.424	1	0	0
Georgia	Muscogee	132150010	0.170	0.114	0.234	1	0	0
Georgia	Muscogee	132150011	0.071	0.023	0.140	0	0	1
Hawaii	Honolulu	150030010	0.001	0.001	0.001	0	0	0
Illinois	Cook	170310001	0.024	0.018	0.028	0	0	0
Illinois	Cook	170310022	0.047	0.034	0.064	0	0	0
Illinois	Cook	170310026	0.034	0.023	0.038	0	0	0
Illinois	Cook	170310052	0.024	0.017	0.032	0	0	0
Illinois	Cook	170310110	0.294	0.077	0.580	1	0	0
Illinois	Cook	170310210	0.053	0.032	0.092	1	0	0
Illinois	Cook	170313103	0.041	0.014	0.103	0	0	0
Illinois	Cook	170313301	0.027	0.019	0.028	0	0	0
Illinois	Cook	170314201	0.011	0.010	0.012	0	0	0
Illinois	Cook	170316003	0.042	0.024	0.080	0	0	0
Illinois	Macon	171150110	0.199	0.073	0.386	1	0	0
Illinois	Madison	171190010	0.416	0.140	0.848	1	0	0
Illinois	Madison	171193007	0.036	0.020	0.053	0	0	0
Illinois	Peoria	171430037	0.011	0.010	0.013	0	0	0
Illinois	Peoria	171430110	0.016	0.012	0.024	1	0	0
Illinois	Peoria	171430210	0.015	0.011	0.026	1	0	0
Illinois	St. Clair	171630010	0.029	0.020	0.038	0	0	1
Illinois	Whiteside	171950110	0.028	0.020	0.040	1	0	0
Illinois	Winnebago	172010110	0.063	0.028	0.118	1	0	0
Indiana	Delaware	180350009	0.338	0.159	0.476	1	0	0

APPENDIX 2D

Table 2D-1. Pb-TSP concentrations at Pb-TSP sites, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)	Non-airport source-oriented (during 2010-2012)	Airport source-oriented	Previous source-oriented
Indiana	Lake	180890023	0.077	0.031	0.087	1	0	0
Indiana	Lake	180890032	0.060	0.023	0.144	1	0	0
Indiana	Lake	180890033	0.139	0.055	0.298	1	0	0
Indiana	Lake	180892008	0.046	0.020	0.103	0	0	0
Indiana	Marion	180970063	0.079	0.028	0.125	1	0	0
Indiana	Marion	180970076	0.020	0.010	0.029	1	0	0
Indiana	Marion	180970078	0.011	0.006	0.021	0	0	0
Indiana	Porter	181270023	0.022	0.013	0.032	1	0	0
Indiana	Porter	181270027	0.041	0.027	0.085	1	0	0
Indiana	Vanderburgh	181630020	0.006	0.004	0.008	0	0	0
Iowa	Pottawattamie	191550011	0.263	0.123	0.282	1	0	0
Iowa	Scott	191630015	0.012	0.010	0.024	0	0	0
Kansas	Saline	201690004	0.421	0.164	0.488	1	0	0
Kansas	Wyandotte	202090021	0.011	0.008	0.012	0	0	0
Kentucky	Boyd	210190016	0.004	0.003	0.007	1	0	0
Kentucky	Madison	211510003	0.248	0.057	0.281	1	0	0
Kentucky	Madison	211510005	1.584	0.703	2.244	1	0	0
Kentucky	Russell	212070001	0.063	0.027	0.109	1	0	0
Louisiana	East Baton Rouge Pa	220330009	0.002	0.002	0.003	0	0	0
Louisiana	East Baton Rouge Pa	220330014	0.011	0.004	0.027	1	0	0
Louisiana	St. John the Baptist P	220950003	0.053	0.025	0.066	1	0	0
Massachusetts	Nantucket County	250190001	0.010	0.009	0.020	0	1	0
Michigan	Charlevoix	260290011	0.006	0.005	0.010	1	0	0
Michigan	Ionia	260670002	0.049	0.071	0.298	1	0	0
Michigan	Ionia	260670003	0.284	0.096	0.414	1	0	0
Michigan	Kent	260810020	0.008	0.005	0.010	0	0	0
Michigan	Oakland	261250013	0.022	0.019	0.027	0	1	0
Michigan	Tuscola	261570001	0.034	0.021	0.063	1	0	0
Michigan	Wayne	261630001	0.006	0.005	0.007	0	0	0
Michigan	Wayne	261630033	0.023	0.012	0.034	0	0	0
Minnesota	Anoka	270031002	0.011	0.006	0.012	0	0	0

APPENDIX 2D

Table 2D-1. Pb-TSP concentrations at Pb-TSP sites, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)	Non-airport source-oriented (during 2010-2012)	Airport source-oriented	Previous source-oriented
Minnesota	Anoka	270036020	0.038	0.017	0.078	1	0	0
Minnesota	Beltrami	270072303		0.001	0.002	0	0	0
Minnesota	Cass	270210001		0.001	0.002	0	0	0
Minnesota	Dakota	270370020	0.004	0.004	0.018	0	0	0
Minnesota	Dakota	270370465	0.259	0.096	0.568	1	0	0
Minnesota	Dakota	270370470	0.002	0.001	0.004	0	0	0
Minnesota	Hennepin	270530963	0.004	0.003	0.006	0	0	0
Minnesota	Hennepin	270530966	0.005	0.004	0.008	0	0	0
Minnesota	Hennepin	270531007	0.004	0.003	0.007	0	0	0
Minnesota	Mille Lacs	270953051	0.001	0.000	0.002	0	0	0
Minnesota	Ramsey	271230871	0.007	0.005	0.010	0	0	0
Minnesota	St. Louis	271377001	0.008	0.002	0.022	0	0	0
Minnesota	St. Louis	271377555	0.004	0.003	0.008	0	0	0
Minnesota	Stearns	271453053	0.008	0.004	0.012	1	0	0
Minnesota	Washington	271630438	0.005	0.003	0.008	0	0	0
Minnesota	Washington	271630446	0.001	0.000	0.003	0	0	0
Missouri	Iron	290930016	0.882	0.484	1.436	1	0	0
Missouri	Iron	290930021	1.147	0.522	1.698	1	0	0
Missouri	Iron	290930027	0.083	0.033	0.107	0	0	1
Missouri	Iron	290930029	0.115	0.038	0.271	0	0	1
Missouri	Iron	290930032		0.354	0.987	1	0	0
Missouri	Iron	290930033	0.057	0.026	0.105	1	0	0
Missouri	Iron	290930034	0.402	0.241	0.615	1	0	0
Missouri	Iron	290939007	0.581	0.392	0.844	1	0	0
Missouri	Iron	290939008	0.387	0.209	0.538	1	0	0
Missouri	Jasper	290970005	0.019	0.014	0.024	0	0	0
Missouri	Jefferson	290990004	1.122	0.888	1.576	1	0	0
Missouri	Jefferson	290990005	0.396	0.223	0.498	1	0	0
Missouri	Jefferson	290990009	0.191	0.055	0.280	1	0	0
Missouri	Jefferson	290990013	0.305	0.135	0.418	1	0	0
Missouri	Jefferson	290990020	0.865	0.546	1.233	1	0	0

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Table 2D-1. Pb-TSP concentrations at Pb-TSP sites, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)	Non-airport source-oriented (during 2010-2012)	Airport source-oriented	Previous source-oriented
Missouri	Jefferson	290990022	0.630	0.469	0.861	1	0	0
Missouri	Jefferson	290990023	0.563	0.386	0.700	1	0	0
Missouri	Jefferson	290990024	0.659	0.340	0.862	1	0	0
Missouri	Jefferson	290990025	0.085	0.042	0.134	1	0	0
Missouri	Jefferson	290990026	0.080	0.036	0.138	1	0	0
Missouri	Jefferson	290990027	0.592	0.454	0.857	1	0	0
Missouri	Jefferson	290999001	1.100	0.812	1.558	1	0	0
Missouri	Jefferson	290999002	0.431	0.228	0.593	1	0	0
Missouri	Jefferson	290999003	0.327	0.175	0.551	1	0	0
Missouri	Jefferson	290999004	0.442	0.156	0.545	1	0	0
Missouri	Jefferson	290999005	1.432	0.913	2.185	1	0	0
Missouri	Jefferson	290999006	0.103	0.049	0.136	1	0	0
Missouri	Reynolds	291790001	0.059	0.031	0.087	1	0	0
Missouri	Reynolds	291790002	0.068	0.034	0.172	1	0	0
Missouri	Reynolds	291790003	0.100	0.027	0.268	1	0	0
Missouri	Reynolds	291790034	0.161	0.078	0.297	1	0	0
Missouri	St. Francois	291870006	0.132	0.043	0.302	0	0	0
Missouri	St. Francois	291870007	0.134	0.053	0.364	0	0	0
Missouri	St. Louis	291892003	0.008	0.007	0.009	0	0	0
Missouri	St. Louis city	295100085	0.028	0.026	0.045	0	0	0
Nebraska	Dodge	310530005	0.139	0.055	0.197	1	0	0
Nebraska	Douglas	310550019	0.006	0.005	0.008	0	0	0
Nebraska	Nemaha	311270002	0.115	0.034	0.206	1	0	0
Nevada	Clark	320030540		0.003	0.004	0	0	0
New Mexico	Bernalillo	350010023	0.006	0.003	0.012	0	0	0
New York	Orange	360713001	0.101	0.025	0.134	1	0	0
New York	Orange	360713002	1.027	0.123	2.821	1	0	0
New York	Orange	360713004	0.007	0.005	0.011	1	0	0
New York	Suffolk	361030024	0.027	0.017	0.031	0	1	0
Ohio	Butler	390170015	0.009	0.006	0.013	0	0	0
Ohio	Columbiana	390290019	0.057	0.020	0.136	0	0	0

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Table 2D-1. Pb-TSP concentrations at Pb-TSP sites, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)	Non-airport source-oriented (during 2010-2012)	Airport source-oriented	Previous source-oriented
Ohio	Columbiana	390290020	0.025	0.015	0.035	0	0	0
Ohio	Columbiana	390290022	0.044	0.019	0.065	0	0	0
Ohio	Cuyahoga	390350038	0.021	0.013	0.026	0	0	0
Ohio	Cuyahoga	390350042	0.030	0.013	0.044	0	0	0
Ohio	Cuyahoga	390350049	0.531	0.136	0.719	1	0	0
Ohio	Cuyahoga	390350060	0.030	0.020	0.035	0	0	0
Ohio	Cuyahoga	390350061	0.023	0.015	0.030	0	0	1
Ohio	Cuyahoga	390350072	0.035	0.014	0.054	1	0	0
Ohio	Franklin	390490025	0.011	0.009	0.016	0	0	0
Ohio	Fulton	390510001	0.178	0.070	0.210	1	0	0
Ohio	Logan	390910006	0.006	0.004	0.008	0	0	1
Ohio	Marion	391010003	0.088	0.041	0.155	1	0	0
Ohio	Marion	391010004	0.017	0.013	0.028	1	0	0
Ohio	Montgomery	391137001	0.008	0.007	0.012	0	0	0
Ohio	Stark	391510017	0.023	0.015	0.032	1	0	0
Ohio	Trumbull	391550012	0.011	0.007	0.017	1	0	0
Ohio	Washington	391670008	0.007	0.005	0.010	0	0	0
Ohio	Washington	391670010	0.008	0.005	0.010	0	0	0
Oklahoma	Ottawa	401159006	0.022	0.012	0.049	0	0	0
Oklahoma	Ottawa	401159007	0.034	0.018	0.056	0	0	0
Oklahoma	Pittsburg	401210416	0.004	0.003	0.006	1	0	0
Oklahoma	Tulsa	401431127	0.008	0.006	0.009	0	0	0
Oregon	Yamhill	410711702	0.045	0.021	0.078	1	0	0
Pennsylvania	Allegheny	420030002	0.016	0.014	0.024	0	0	0
Pennsylvania	Allegheny	420030008	0.014	0.008	0.020	0	0	0
Pennsylvania	Allegheny	420030070	0.056	0.018	0.110	1	0	0
Pennsylvania	Allegheny	420031009	0.138	0.035	0.149	1	0	0
Pennsylvania	Beaver	420070006	0.085	0.051	0.198	1	0	0
Pennsylvania	Beaver	420070007	0.253	0.166	0.393	1	0	0
Pennsylvania	Beaver	420070505	0.151	0.089	0.291	1	0	0
Pennsylvania	Berks	420110020	0.509	0.158	1.064	1	0	0

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Table 2D-1. Pb-TSP concentrations at Pb-TSP sites, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)	Non-airport source-oriented (during 2010-2012)	Airport source-oriented	Previous source-oriented
Pennsylvania	Berks	420110021	0.139	0.044	0.319	1	0	0
Pennsylvania	Berks	420110022	0.118	0.035	0.261	1	0	0
Pennsylvania	Berks	420111717	0.217	0.146	0.285	1	0	0
Pennsylvania	Carbon	420250214		0.104	0.321	1	0	0
Pennsylvania	Delaware	420450002	0.047	0.028	0.048	0	0	0
Pennsylvania	Delaware	420450004	0.047	0.028	0.048	1	0	0
Pennsylvania	Franklin	420550002	0.046	0.028	0.047	1	0	0
Pennsylvania	Indiana	420630005	0.049	0.025	0.058	1	0	0
Pennsylvania	Lancaster	420710009	0.068	0.039	0.093	1	0	0
Pennsylvania	Lawrence	420730011	0.023	0.026	0.046	1	0	0
Pennsylvania	Luzerne	420790036	0.137	0.054	0.268	1	0	0
Pennsylvania	Philadelphia	421010449	0.029	0.021	0.029	0	0	1
Pennsylvania	Philadelphia	421011002	0.051	0.030	0.069	0	0	0
Pennsylvania	Westmoreland	421290009	0.046	0.023	0.047	1	0	0
South Carolina	Charleston	450190003	0.007	0.005	0.013	0	0	0
South Carolina	Florence	450418001	0.044	0.013	0.101	1	0	0
South Carolina	Florence	450418002	0.015	0.006	0.024	1	0	0
South Carolina	Florence	450418003	0.011	0.005	0.013	0	0	0
South Carolina	Greenville	450450015	0.006	0.005	0.008	0	0	0
South Carolina	Richland	450790007	0.005	0.004	0.007	0	0	0
South Carolina	Richland	450790019	0.024	0.011	0.061	0	0	0
Tennessee	Knox	470930023	0.165	0.108	0.214	1	0	0
Tennessee	Knox	470930027	0.042	0.020	0.078	1	0	0
Tennessee	Knox	470931017	0.037	0.018	0.058	1	0	0
Tennessee	Shelby	471570075	0.005	0.004	0.005	0	0	0
Tennessee	Sullivan	471633001	0.076	0.056	0.107	0	0	1
Tennessee	Sullivan	471633002	0.044	0.036	0.052	0	0	1
Tennessee	Sullivan	471633003	0.053	0.038	0.056	0	0	1
Tennessee	Sullivan	471633004	0.080	0.044	0.124	0	0	1
Texas	Cameron	480610006	0.008	0.004	0.018	0	0	0
Texas	Collin	480850003	0.371	0.136	0.625	1	0	0

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Table 2D-1. Pb-TSP concentrations at Pb-TSP sites, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)	Non-airport source-oriented (during 2010-2012)	Airport source-oriented	Previous source-oriented
Texas	Collin	480850007	0.199	0.093	0.240	1	0	0
Texas	Collin	480850009	0.774	0.388	1.178	1	0	0
Texas	Collin	480850029	0.180	0.052	0.335	1	0	0
Texas	Dallas	481130069	0.013	0.009	0.022	0	0	0
Texas	El Paso	481410002	0.040	0.022	0.087	0	0	0
Texas	El Paso	481410033	0.019	0.017	0.023	0	0	0
Texas	El Paso	481410037	0.025	0.022	0.032	1	0	0
Texas	El Paso	481410055	0.019	0.010	0.032	0	0	0
Texas	El Paso	481410058	0.022	0.016	0.022	0	0	0
Texas	Harris	482011034	0.008	0.005	0.009	0	0	0
Texas	Harris	482011039	0.004	0.003	0.008	0	0	0
Texas	Kaufman	482570020	0.104	0.044	0.110	1	0	0
Texas	Potter	483750024	0.020	0.007	0.029	1	0	0
Texas	Webb	484790016	0.026	0.018	0.035	0	0	0
Utah	Salt Lake	490351001	0.057	0.024	0.086	1	0	0
Virginia	Amherst	510090007	0.018	0.006	0.016	1	0	0
Virginia	Buchanan	510270006	0.014	0.012	0.021	1	0	0
Virginia	Henrico	510870014	0.005	0.005	0.024	0	0	0
Virginia	Roanoke city	517700011	0.109	0.033	0.272	1	0	0
Washington	King	530330029	0.055	0.036	0.087	0	1	0
Washington	Snohomish	530610013	0.023	0.013	0.032	0	1	0
West Virginia	Cabell	540110006	0.012	0.010	0.013	0	0	0
Wisconsin	Sheboygan	551170008	0.152	0.056	0.225	1	0	0
Puerto Rico	Arecibo Municipio, Pu	720130001	0.339	0.171	0.416	1	0	0
Puerto Rico	Bayamón Municipio, P	720210010	0.011	0.004	0.027	0	0	0
Puerto Rico	Salinas Municipio, Pu	721230002	0.017	0.008	0.042	0	0	0
		counts →	236	243	243	121	15	11

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Table 2D-2. Pb-PM_{10} concentrations at urban Pb-PM_{10} sites, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)
North Carolin	Wake	371830014		0.002	0.002
Georgia	DeKalb	130890002	0.002	0.001	0.003
Massachusetts	Suffolk	250250002	0.002	0.002	0.003
New Hampshi	Rockingham	330150018	0.002	0.002	0.003
North Carolin	Mecklenburg	371190041		0.002	0.003
Oregon	Jackson	410290133	0.003	0.002	0.003
Oregon	Jackson	410292129		0.002	0.003
Oregon	Lane	410390060	0.002	0.001	0.003
Oregon	Lane	410390062	0.002	0.002	0.003
Vermont	Chittenden	500070007	0.002	0.001	0.003
Florida	Hillsborough	120573002	0.003	0.002	0.004
Florida	Pinellas	121030018	0.003	0.002	0.004
Kentucky	Henderson	211010014	0.003	0.003	0.004
Nevada	Clark	320030540		0.003	0.004
New York	Monroe	360551007	0.003	0.003	0.004
Arkansas	Pulaski	051190007	0.004	0.003	0.005
California	Sacramento	060670006	0.003	0.002	0.005
Delaware	New Castle	100032004	0.004	0.003	0.005
District of Col	District of Colum	110010043	0.004	0.003	0.005
Florida	Orange	120951004		0.002	0.005
Idaho	Ada	160010010	0.002	0.002	0.005
Massachusetts	Suffolk	250250042	0.005	0.003	0.005
Rhode Island	Providence	440070022	0.003	0.003	0.005
Washington	King	530330080	0.004	0.003	0.005
California	Santa Clara	060850005	0.006	0.002	0.006
Florida	Pinellas	121030026	0.004	0.002	0.006
Kentucky	Fayette	210670012	0.004	0.003	0.006
Kentucky	Fayette	210670014	0.005	0.004	0.006
Michigan	Wayne	261630001	0.006	0.004	0.006
New Jersey	Essex	340130003	0.005	0.003	0.006
Arizona	Maricopa	040139997	0.006	0.004	0.007
Ohio	Hamilton	390610040	0.005	0.005	0.007
Texas	Harris	482011039	0.004	0.002	0.007
Arizona	Maricopa	040134009		0.005	0.008
Kentucky	Jefferson	211110067	0.006	0.005	0.008
Massachusetts	Hampden	250132009	0.006	0.004	0.008
New York	Bronx	360050110	0.006	0.006	0.008
Utah	Davis	490110004	0.006	0.003	0.009

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Table 2D-2. Pb-PM_{10} concentrations at urban Pb-PM_{10} sites, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)
Illinois	Cook	170314201	0.006	0.004	0.010
Wisconsin	Milwaukee	550790010		0.006	0.010
New York	Bronx	360050080	0.008	0.006	0.012
Oregon	Multnomah	410510246	0.006	0.004	0.012
Connecticut	New Haven	090090027	0.008	0.004	0.013
Maryland	Prince George's	240330030	0.008	0.004	0.016
Texas	Harris	482011035	0.008	0.005	0.016
Virginia	Henrico	510870014	0.007	0.003	0.016
California	Los Angeles	060371103	0.013	0.007	0.026
Kentucky	Boyd	210190002	0.020	0.013	0.029
California	Riverside	060658001	0.013	0.006	0.030
Illinois	St. Clair	171639010		0.015	0.031
Missouri	St. Louis city	295100085	0.017	0.012	0.032
Michigan	Wayne	261630033	0.023	0.011	0.035
Alabama	Jefferson	010730023	0.029	0.019	0.040
Mississippi	Hinds	280490019		0.074	0.082
		counts →	45	54	54

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Table 2D-3. Pb-$PM_{2.5}$ concentrations at urban CSN $PM_{2.5}$ sites, 2010-2012.

state_name	county_name	SITE	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)
California	Kern	060299001	0.001	0.001	0.001
Hawaii	Honolulu	150030010	0.001	0.000	0.001
Wyoming	Laramie	560210100	0.000	0.000	0.001
California	Butte	060070008		0.002	0.002
Colorado	Adams	080010006	0.002	0.001	0.002
Florida	Broward	120111002	0.001	0.001	0.002
Idaho	Ada	160010010	0.001	0.001	0.002
North Carolina	Buncombe	370210034	0.001	0.001	0.002
North Carolina	Wake	371830014	0.002	0.001	0.002
Oklahoma	Oklahoma	401091037	0.001	0.001	0.002
Rhode Island	Providence	440071010	0.002	0.002	0.002
South Carolina	Charleston	450190049	0.002	0.001	0.002
South Carolina	Richland	450790007	0.002	0.001	0.002
Texas	Harris	482011039	0.001	0.001	0.002
Vermont	Chittenden	500070012	0.002	0.001	0.002
Wisconsin	Brown	550090005	0.001	0.001	0.002
Alabama	Madison	010890014	0.002	0.001	0.003
Arizona	Pima	040191028	0.002	0.001	0.003
California	Butte	060070002	0.003	0.002	0.003
California	Fresno	060190008	0.002	0.001	0.003
California	Ventura	061112002	0.002	0.001	0.003
Colorado	Weld	081230008	0.002	0.001	0.003
District of Colum	District of Columbia	110010043	0.002	0.001	0.003
Florida	Hillsborough	120573002	0.002	0.001	0.003
Florida	Pinellas	121030026	0.002	0.001	0.003
Georgia	Clarke	130590001	0.002	0.001	0.003
Georgia	DeKalb	130890002	0.002	0.001	0.003
Iowa	Linn	191130037	0.002	0.002	0.003
Iowa	Polk	191530030	0.002	0.001	0.003
Louisiana	East Baton Rouge Parish	220330009	0.003	0.002	0.003
Minnesota	Hennepin	270530963	0.002	0.002	0.003
Mississippi	Hinds	280490019	0.003	0.001	0.003
Missouri	Clay	290470005	0.002	0.001	0.003
Nevada	Washoe	320310016	0.002	0.001	0.003
New Jersey	Morris	340273001	0.002	0.001	0.003
New York	Monroe	360551007	0.002	0.002	0.003
North Carolina	Catawba	370350004	0.002	0.002	0.003

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Table 2D-3. Pb-$PM_{2.5}$ concentrations at urban CSN $PM_{2.5}$ sites, 2010-2012.

state_name	county_name	SITE	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)
North Carolina	Mecklenburg	371190041	0.002	0.001	0.003
North Carolina	Rowan	371590021	0.006	0.001	0.003
Oregon	Lane	410392013	0.002	0.001	0.003
Pennsylvania	Centre	420270100	0.002	0.001	0.003
South Dakota	Minnehaha	460990008	0.002	0.001	0.003
Tennessee	Roane	471451001	0.002	0.002	0.003
Texas	Cameron	480612004	0.002	0.001	0.003
Texas	Jefferson	482450021	0.002	0.002	0.003
Texas	Travis	484530020	0.002	0.001	0.003
Washington	Yakima	530770009	0.002	0.001	0.003
California	Fresno	060190011	0.002	0.001	0.004
California	Santa Clara	060850005	0.003	0.001	0.004
Colorado	Denver	080310025	0.002	0.001	0.004
Georgia	Bibb	130210007	0.003	0.001	0.004
Illinois	Madison	171199010		0.002	0.004
Indiana	Elkhart	180390008	0.003	0.002	0.004
Indiana	Vanderburgh	181630021	0.003	0.002	0.004
Maryland	Prince George's	240330030	0.002	0.001	0.004
Massachusetts	Hampden	250130008	0.003	0.002	0.004
Michigan	Wayne	261630001	0.003	0.002	0.004
Nevada	Clark	320030540	0.003	0.001	0.004
New Jersey	Middlesex	340230006	0.003	0.002	0.004
New Jersey	Union	340390004	0.004	0.002	0.004
New Mexico	Bernalillo	350010023	0.002	0.001	0.004
New York	New York	360610134	0.003	0.002	0.004
North Dakota	Cass	380171004	0.002	0.001	0.004
Ohio	Franklin	390490081	0.003	0.002	0.004
Ohio	Lucas	390950026	0.003	0.002	0.004
Oklahoma	Tulsa	401431127	0.003	0.001	0.004
Pennsylvania	Adams	420010001	0.002	0.001	0.004
Tennessee	Shelby	471570024	0.003	0.002	0.004
Washington	Clark	530110013	0.003	0.001	0.004
Arizona	Maricopa	040134009	0.004	0.003	0.005
Arizona	Maricopa	040139997	0.003	0.002	0.005
California	Kern	060290014	0.004	0.001	0.005
California	Riverside	060658001	0.003	0.002	0.005
California	Sacramento	060670010	0.003	0.002	0.005

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Table 2D-3. Pb-$PM_{2.5}$ concentrations at urban CSN $PM_{2.5}$ sites, 2010-2012.

state_name	county_name	SITE	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)
California	San Diego	060730003	0.004	0.002	0.005
California	San Diego	060731002	0.004	0.002	0.005
California	Tulare	061072002	0.004	0.002	0.005
Delaware	New Castle	100032004	0.004	0.002	0.005
Illinois	Cook	170314201	0.003	0.002	0.005
Indiana	Clark	180190006	0.003	0.002	0.005
Minnesota	Olmsted	271095008	0.003	0.001	0.005
New Jersey	Essex	340130003	0.003	0.002	0.005
New York	Bronx	360050110	0.004	0.003	0.005
North Carolina	Davidson	370570002	0.003	0.001	0.005
Pennsylvania	Chester	420290100	0.004	0.002	0.005
Pennsylvania	Philadelphia	421010055	0.003	0.002	0.005
Tennessee	Davidson	470370023	0.003	0.002	0.005
Tennessee	Hamilton	470654002	0.003	0.002	0.005
Tennessee	Montgomery	471251009	0.003	0.001	0.005
Texas	Nueces	483550034	0.003	0.002	0.005
Virginia	Henrico	510870014	0.002	0.001	0.005
Washington	Snohomish	530611007	0.004	0.001	0.005
West Virginia	Kanawha	540390011	0.003	0.002	0.005
California	Alameda	060010007	0.005	0.002	0.006
Delaware	Kent	100010003	0.003	0.001	0.006
Illinois	Cook	170310076	0.004	0.003	0.006
Illinois	St. Clair	171630900	0.005	0.003	0.006
Iowa	Linn	191130040	0.003	0.002	0.006
Michigan	Kent	260810020	0.004	0.002	0.006
Nebraska	Douglas	310550019	0.004	0.001	0.006
New York	Albany	360010005	0.004	0.002	0.006
Pennsylvania	Westmoreland	421290008	0.004	0.003	0.006
Rhode Island	Providence	440070022	0.004	0.003	0.006
Tennessee	Shelby	471570075	0.003	0.002	0.006
Wisconsin	Milwaukee	550790026	0.004	0.003	0.006
Connecticut	New Haven	090090027	0.004	0.002	0.007
Indiana	Marion	180970078	0.004	0.003	0.007
Kentucky	Boyd	210190017	0.005	0.003	0.007
Maryland	Baltimore	240053001	0.006	0.002	0.007
Michigan	Monroe	261150005	0.005	0.002	0.007
Ohio	Summit	391530023	0.005	0.004	0.007

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Table 2D-3. Pb-$PM_{2.5}$ concentrations at urban CSN $PM_{2.5}$ sites, 2010-2012.

state_name	county_name	SITE	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)
Pennsylvania	Erie	420490003	0.004	0.002	0.007
Tennessee	Knox	470931020	0.006	0.003	0.007
Texas	Dallas	481130050	0.005	0.003	0.007
Utah	Davis	490110004	0.003	0.002	0.007
Washington	King	530330080	0.003	0.001	0.007
West Virginia	Kanawha	540391005	0.005	0.002	0.007
West Virginia	Marshall	540511002	0.007	0.004	0.007
Alaska	Fairbanks North Star Borough	020900010	0.005	0.002	0.008
California	Solano	060950004	0.006	0.003	0.008
Florida	Leon	120730012	0.004	0.002	0.008
Georgia	Richmond	132450091	0.004	0.001	0.008
Georgia	Walker	132950002	0.005	0.002	0.008
Illinois	DuPage	170434002	0.004	0.002	0.008
Kansas	Sedgwick	201730010	0.004	0.002	0.008
Kansas	Wyandotte	202090021	0.006	0.004	0.008
Oregon	Multnomah	410510080	0.005	0.003	0.008
Pennsylvania	Washington	421255001	0.005	0.003	0.008
South Carolina	Greenville	450450015	0.006	0.002	0.008
Texas	Ellis	481390016	0.006	0.003	0.008
Texas	El Paso	481410044	0.006	0.003	0.008
Texas	Harris	482010024	0.006	0.003	0.008
Washington	Pierce	530530029	0.005	0.003	0.008
California	Sacramento	060670006	0.004	0.001	0.009
California	Stanislaus	060990005	0.005	0.003	0.009
Georgia	Floyd	131150003	0.005	0.002	0.009
Louisiana	Bossier Parish	220150008	0.006	0.003	0.009
Ohio	Hamilton	390610040	0.005	0.002	0.009
Pennsylvania	Lancaster	420710007	0.007	0.003	0.009
Pennsylvania	York	421330008	0.006	0.002	0.009
New York	Queens	360810124	0.005	0.002	0.010
Pennsylvania	Lackawanna	420692006	0.006	0.003	0.010
West Virginia	Ohio	540690010	0.008	0.005	0.010
Kentucky	Fayette	210670012	0.006	0.002	0.011
North Carolina	Forsyth	370670022	0.006	0.002	0.011
Ohio	Cuyahoga	390350060	0.009	0.006	0.011
Pennsylvania	Dauphin	420430401	0.007	0.003	0.011
Pennsylvania	Philadelphia	421010004	0.006	0.003	0.011

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Table 2D-3. Pb-$PM_{2.5}$ concentrations at urban CSN $PM_{2.5}$ sites, 2010-2012.

state_name	county_name	SITE	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)
Kentucky	Jefferson	211110067	0.007	0.004	0.012
Massachusetts	Suffolk	250250042	0.005	0.002	0.012
Ohio	Cuyahoga	390350038	0.008	0.004	0.012
Ohio	Jefferson	390811001	0.009	0.005	0.012
Ohio	Lawrence	390870012	0.009	0.005	0.012
Texas	Dallas	481130069	0.007	0.003	0.012
Utah	Utah	490494001	0.006	0.002	0.012
Washington	King	530330057	0.008	0.004	0.013
Wisconsin	Waukesha	551330027	0.010	0.005	0.013
Alabama	Montgomery	011011002	0.008	0.003	0.014
Arkansas	Pulaski	051190007	0.006	0.002	0.014
California	Los Angeles	060371103	0.015	0.002	0.015
Missouri	St. Louis city	295100085	0.010	0.006	0.016
New York	Erie	360290005	0.012	0.007	0.016
Pennsylvania	Berks	420110011	0.013	0.007	0.016
Utah	Salt Lake	490353006	0.007	0.002	0.016
Iowa	Scott	191630015	0.010	0.005	0.017
Missouri	Jefferson	290990019	0.009	0.004	0.017
Ohio	Mahoning	390990014	0.012	0.007	0.017
Texas	Lubbock	483030325	0.007	0.003	0.017
Indiana	Lake	180890022	0.011	0.005	0.018
Pennsylvania	Northampton	420950025	0.008	0.003	0.018
Texas	El Paso	481410053	0.017	0.006	0.018
Illinois	St. Clair	171639010	0.014	0.010	0.019
Minnesota	Anoka	270031002	0.012	0.006	0.019
Ohio	Stark	391510017	0.012	0.007	0.019
Pennsylvania	Philadelphia	421011002	0.012	0.006	0.020
California	Alameda	060010011	0.011	0.004	0.021
Pennsylvania	Allegheny	420030008	0.016	0.008	0.022
Alabama	Jefferson	010732003	0.018	0.006	0.023
Ohio	Lorain	390933002	0.012	0.006	0.023
Pennsylvania	Cambria	420210011	0.013	0.007	0.023
California	Imperial	060250005	0.018	0.012	0.025
Michigan	Wayne	261630015	0.012	0.006	0.027
Alabama	Russell	011130001	0.016	0.004	0.028
Michigan	Wayne	261630033	0.017	0.006	0.028
Georgia	Muscogee	132150011	0.014	0.004	0.029

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Table 2D-3. Pb-$PM_{2.5}$ concentrations at urban CSN $PM_{2.5}$ sites, 2010-2012.

state_name	county_name	SITE	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)
Michigan	St. Clair	261470005	0.015	0.004	0.030
Illinois	Madison	171190024	0.020	0.010	0.032
Washington	Pierce	530530031	0.015	0.004	0.032
Ohio	Montgomery	391130032	0.014	0.003	0.033
California	San Bernardino	060712002	0.037	0.017	0.037
Indiana	Lake	180892004	0.022	0.010	0.037
California	Orange	060590007	0.042	0.013	0.043
Pennsylvania	Allegheny	420030064	0.038	0.018	0.051
Alabama	Jefferson	010730023	0.041	0.020	0.063
Illinois	Cook	170310057	0.069	0.011	0.199
		counts →	193	195	195

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Table 2D-4. Pb-$PM_{2.5}$ concentrations at non-urban IMPROVE $PM_{2.5}$ sites, 2010-2012.

state_name	county_name	SITE	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)
Alaska	Aleutians East Borough	020130002	0.002	0.001	0.002
Alaska	Denali Borough	020680003	0.001	0.000	0.001
Alaska	Kenai Peninsula Borough	021220009	0.001	0.000	0.002
Alaska	Northwest Arctic Borough	021889000			
Alaska	Yukon-Koyukuk Census Are	022909000	0.001	0.000	0.001
Arizona	Apache	040018001	0.001	0.001	0.002
Arizona	Gila	040070010	0.005	0.003	0.008
Arizona	Gila	040078100	0.001	0.001	0.002
Arizona	Navajo	040170119	0.001	0.001	0.002
Arkansas	Newton	051019000	0.002	0.001	0.003
Arkansas	Polk	051130003	0.002	0.001	0.003
California	Del Norte	060150002	0.001	0.000	0.001
California	Inyo	060270101	0.001	0.001	0.002
California	Mariposa	060430003	0.001	0.001	0.002
California	Mono	060519000	0.001	0.000	0.001
California	Siskiyou	060930005	0.001	0.000	0.001
California	Trinity	061059000	0.001	0.001	0.001
Colorado	Alamosa	080039000	0.001	0.000	0.002
Colorado	Garfield	080450015		0.001	0.002
Colorado	Jackson	080579000	0.001	0.000	0.001
Colorado	La Plata	080679000	0.001	0.001	0.001
Colorado	Montezuma	080839000	0.001	0.001	0.001
Colorado	Pitkin	080979000	0.001	0.000	0.001
Colorado	Rio Blanco	081039000			
Colorado	San Juan	081119000	0.001	0.001	0.001
Connecticut	Litchfield	090050005	0.002	0.001	0.002
Georgia	Charlton	130499000	0.002	0.001	0.002
Hawaii	Hawaii	150019000			
Hawaii	Hawaii	150019001			
Hawaii	Hawaii	150019002	0.005	0.001	0.010
Idaho	Custer	160370002	0.001	0.000	0.001
Idaho	Lemhi	160590007			
Iowa	Montgomery	191370002	0.002	0.002	0.003
Iowa	Van Buren	191770006	0.003	0.002	0.004
Iowa	Van Buren	191779000			
Kansas	Brown	200139000	0.003	0.002	0.003
Kansas	Chase	200170001	0.002	0.001	0.003

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Table 2D-4. Pb-$PM_{2.5}$ concentrations at non-urban IMPROVE $PM_{2.5}$ sites, 2010-2012.

state_name	county_name	SITE	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)
Kansas	Trego	201950001	0.001	0.001	0.001
Louisiana	Winn Parish	221279000	0.001	0.001	0.002
Maine	Aroostook	230031020	0.002	0.001	0.004
Maine	Hancock	230090103	0.001	0.001	0.002
Maine	Washington	230291004	0.001	0.001	0.002
Maryland	Garrett	240239000	0.002	0.002	0.003
Massachusetts	Dukes	250070001	0.001	0.001	0.002
Michigan	Keweenaw	260839000	0.001	0.001	0.002
Michigan	Keweenaw	260839001			
Michigan	Schoolcraft	261539000	0.002	0.001	0.002
Minnesota	Lake	270759000	0.001	0.001	0.002
Minnesota	Rock	271339000	0.002	0.002	0.003
Minnesota	Winona	271699000	0.002	0.002	0.002
Missouri	Cedar	290390001	0.002	0.002	0.003
Missouri	Stoddard	292070001	0.004	0.003	0.005
Missouri	Taney	292130003	0.002	0.002	0.003
Montana	Fergus	300279000	0.001	0.001	0.001
Montana	Flathead	300299001	0.001	0.000	0.001
Montana	Lake	300479000	0.001	0.000	0.001
Montana	Lewis and Clark	300499000	0.001	0.000	0.001
Montana	Powell	300779000	0.001	0.000	0.001
Montana	Ravalli	300819000	0.001	0.000	0.001
Montana	Roosevelt	300859000	0.001	0.001	0.001
Montana	Rosebud	300870762	0.001	0.000	0.001
Montana	Sanders	300899000	0.001	0.000	0.001
Montana	Sheridan	300919000	0.001	0.001	0.001
Nebraska	Garden	310699000	0.001	0.001	0.001
Nebraska	Thomas	311719000	0.001	0.001	0.001
Nebraska	Thurston	311739000			
Nevada	Elko	320079000	0.001	0.000	0.002
Nevada	Mineral	320219000			
Nevada	White Pine	320339000	0.001	0.000	0.001
New Hampshire	Coos	330074002	0.001	0.001	0.001
New Mexico	Catron	350039000	0.001	0.001	0.001
New Mexico	Chaves	350059000	0.001	0.001	0.002
New Mexico	Lincoln	350279000	0.001	0.001	0.002
New Mexico	Los Alamos	350281002	0.001	0.001	0.002

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Table 2D-4. Pb-$PM_{2.5}$ concentrations at non-urban IMPROVE $PM_{2.5}$ sites, 2010-2012.

state_name	county_name	SITE	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)
New Mexico	Rio Arriba	350399000	0.001	0.001	0.002
New Mexico	Socorro	350539000	0.001	0.001	0.002
New Mexico	Taos	350559000	0.001	0.000	0.001
New York	Steuben	361019000		0.002	0.002
North Carolina	Avery	370110002	0.001	0.001	0.002
North Carolina	Hyde	370959000	0.002	0.001	0.002
North Dakota	Billings	380070002	0.001	0.001	0.001
North Dakota	Burke	380130004	0.001	0.001	0.001
Ohio	Noble	391219000	0.005	0.003	0.008
Oklahoma	Adair	400019009	0.002	0.002	0.003
Oklahoma	Ellis	400450890	0.001	0.001	0.001
Oklahoma	Kay	400719010	0.002	0.001	0.003
Oregon	Klamath	410358001	0.001	0.000	0.001
Oregon	Union	410610010	0.001	0.000	0.001
Oregon	Wallowa	410630002	0.002	0.001	0.003
South Dakota	Jackson	460710001	0.001	0.001	0.001
Texas	Brewster	480430101	0.002	0.001	0.002
Texas	Culberson	481099000	0.001	0.001	0.002
Utah	Garfield	490170101	0.001	0.001	0.001
Utah	San Juan	490379000	0.001	0.001	0.001
Utah	Wayne	490559000	0.001	0.000	0.001
Vermont	Bennington	500038001	0.001	0.001	0.002
Virginia	Madison	511139000	0.002	0.001	0.002
Virginia	Rockbridge	511639000	0.004	0.003	0.006
Washington	Clallam	530090013	0.000	0.000	0.001
Washington	Clallam	530090014			
Washington	Clallam	530090020	0.001	0.001	0.002
Washington	Kittitas	530370004	0.001	0.001	0.001
Washington	Klickitat	530390010	0.002	0.001	0.002
Washington	Klickitat	530390011	0.001	0.001	0.001
Washington	Lewis	530410007	0.001	0.000	0.001
Washington	Okanogan	530470012	0.001	0.000	0.001
West Virginia	Tucker	540939000	0.002	0.001	0.002
Wyoming	Albany	560019000			
Wyoming	Campbell	560050123	0.000	0.000	0.001
Wyoming	Johnson	560199000	0.001	0.000	0.001
Wyoming	Park	560299002	0.001	0.000	0.001

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Table 2D-4. Pb-$PM_{2.5}$ concentrations at non-urban IMPROVE $PM_{2.5}$ sites, 2010-2012.

state_name	county_name	SITE	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Maximum monthly mean ($\mu g/m^3$)
Wyoming	Sublette	560359000	0.001	0.000	0.001
Wyoming	Sublette	560359001	0.001	0.000	0.001
Wyoming	Teton	560399000	0.001	0.000	0.001
Virgin Islands	St John	780209000	0.001	0.001	0.001
		counts →	102	104	104

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Table 2D-5. Distribution of maximum 3-month means, 2010-2012.

(units are µg/m^3)	n	min	Pct1	Pct5	Pct10	Pct25	mean	Pct50	Pct75	Pct90	Pct95	Pct99	max
All Pb-TSP sites	235	0.001	0.001	0.004	0.006	0.011	0.1396	0.040	0.132	0.416	0.659	1.296	1.584
Pb-TSP non-airport source-oriented sites	119	0.004	0.004	0.011	0.017	0.045	0.2493	0.109	0.338	0.659	1.100	1.432	1.584
Pb-TSP airport source-oriented sites	13	0.010	0.010	0.010	0.010	0.020	0.0677	0.040	0.070	0.120	0.330	0.330	0.330
Pb-TSP previous source-oriented sites	11	0.006	0.006	0.006	0.023	0.029	0.0554	0.053	0.080	0.083	0.115	0.115	0.115
Pb-TSP not source-oriented sites	93	0.001	0.001	0.002	0.004	0.006	0.0180	0.011	0.024	0.041	0.047	0.134	0.134
Pb-PM10 urban sites	45	0.002	0.002	0.002	0.002	0.003	0.0065	0.005	0.006	0.013	0.020	0.029	0.029
Pb-PM2.5 urban CSN sites	193	0.000	0.001	0.002	0.002	0.002	0.0064	0.004	0.007	0.013	0.017	0.042	0.069
Pb-PM2.5 non-urban IMPROVE sites	102	0.000	0.000	0.001	0.001	0.001	0.0014	0.001	0.002	0.002	0.003	0.005	0.005

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Table 2D-6. Distribution of annual means, 2010-2012.

(units are µg/m^3)	n	min	Pct1	Pct5	Pct10	Pct25	mean	Pct50	Pct75	Pct90	Pct95	Pct99	max
All Pb-TSP sites	242	0.000	0.001	0.003	0.004	0.007	0.0718	0.021	0.055	0.166	0.386	0.812	0.913
Pb-TSP non-airport source-oriented sites	121	0.003	0.003	0.006	0.012	0.024	0.1261	0.044	0.140	0.386	0.522	0.888	0.913
Pb-TSP airport source-oriented sites	15	0.004	0.004	0.004	0.009	0.014	0.0557	0.033	0.071	0.128	0.254	0.254	0.254
Pb-TSP previous source-oriented sites	11	0.004	0.004	0.004	0.015	0.020	0.0298	0.033	0.038	0.044	0.056	0.056	0.056
Pb-TSP not source-oriented sites	96	0.000	0.000	0.001	0.003	0.004	0.0101	0.006	0.014	0.020	0.028	0.053	0.053
Pb-PM10 urban sites	54	0.001	0.001	0.001	0.002	0.002	0.0055	0.003	0.005	0.011	0.015	0.074	0.074
Pb-PM2.5 urban CSN sites	195	0.000	0.000	0.001	0.001	0.001	0.0029	0.002	0.003	0.006	0.008	0.018	0.020
Pb-PM2.5 non-urban IMPROVE sites	104	0.000	0.000	0.000	0.000	0.000	0.0009	0.001	0.001	0.002	0.002	0.003	0.003

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Table 2D-7. Distribution of maximum monthly means, 2010-2012.

(units are µg/m^3)	n	min	Pct1	Pct5	Pct10	Pct25	mean	Pct50	Pct75	Pct90	Pct95	Pct99	max
All Pb-TSP sites	243	0.000	0.000	0.010	0.010	0.020	0.2213	0.060	0.230	0.580	0.990	2.240	2.820
Pb-TSP non-airport source-oriented sites	121	0.010	0.010	0.020	0.030	0.080	0.4012	0.210	0.500	0.990	1.580	2.260	2.820
Pb-TSP airport source-oriented sites	15	0.010	0.010	0.010	0.020	0.030	0.0933	0.070	0.100	0.230	0.400	0.400	0.400
Pb-TSP previous source-oriented sites	11	0.010	0.010	0.010	0.030	0.030	0.0882	0.060	0.120	0.140	0.270	0.270	0.270
Pb-TSP not source-oriented sites	96	0.000	0.000	0.000	0.010	0.010	0.0299	0.010	0.030	0.060	0.100	0.360	0.360
Pb-PM10 urban sites	54	0.000	0.000	0.000	0.000	0.000	0.0109	0.010	0.010	0.030	0.030	0.080	0.080
Pb-PM2.5 urban CSN sites	184	0.000	0.000	0.000	0.000	0.000	0.0098	0.010	0.010	0.020	0.030	0.050	0.200
Pb-PM2.5 non-urban IMPROVE sites	104	0.000	0.000	0.000	0.000	0.000	0.0005	0.000	0.000	0.000	0.000	0.010	0.010

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Table 2D-8. Pb-TSP metric ratios, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Ratio of max 3-month mean to annual mean	Ratio of max 3-month mean to annual mean for CBSAs > 1M population
Alabama	Limestone	010830005	0.009	0.007	1.2857	
Alabama	Pike	011090003	1.296	0.613	2.1142	
Alaska	Anchorage Municipality	020200051	0.067	0.054	1.2407	
Arizona	Gila	040071002	0.267	0.131	2.0382	
Arizona	Gila	040078000	0.061	0.030	2.0333	
Arizona	Maricopa	040134018	0.038	0.031	1.2258	1.2258
Arizona	Pima	040191028	0.002	0.002	1.0000	
California	Fresno	060190011	0.005	0.004	1.2500	
California	Imperial	060250005	0.026	0.017	1.5294	
California	Los Angeles	060371103	0.013	0.009	1.4444	1.4444
California	Los Angeles	060371302	0.015	0.007	2.1429	2.1429
California	Los Angeles	060371402	0.061	0.033	1.8485	1.8485
California	Los Angeles	060371403	0.110	0.055	2.0000	2.0000
California	Los Angeles	060371404	0.108	0.042	2.5714	2.5714
California	Los Angeles	060371405	0.450	0.241	1.8672	1.8672
California	Los Angeles	060371406	0.072	0.035	2.0571	2.0571
California	Los Angeles	060371602	0.013	0.008	1.6250	1.6250
California	Los Angeles	060374002	0.009	0.005	1.8000	1.8000
California	Los Angeles	060374004	0.010	0.006	1.6667	1.6667
California	Los Angeles	060375005	0.006	0.003	2.0000	2.0000
California	Riverside	060651003	0.008	0.005	1.6000	1.6000
California	Riverside	060658001	0.008	0.006	1.3333	1.3333
California	San Bernardino	060711004	0.010	0.006	1.6667	1.6667
California	San Bernardino	060719004	0.011	0.007	1.5714	1.5714
California	San Diego	060730003	0.006	0.005	1.2000	1.2000
California	San Diego	060731020		0.127		
California	San Diego	060731021		0.058		
California	San Mateo	060812002	0.331	0.255	1.2980	1.2980
California	Santa Clara	060852010	0.119	0.090	1.3222	1.3222
California	Santa Clara	060852011	0.093	0.070	1.3286	1.3286
Colorado	Arapahoe	080050007	0.023	0.014	1.6429	1.6429
Colorado	Denver	080310025	0.010	0.006	1.6667	1.6667
District of Columbia	District of Columbia	110010043	0.004	0.003	1.3333	1.3333
Florida	Hillsborough	120570100	0.044	0.025	1.7600	1.7600
Florida	Hillsborough	120571066	0.984	0.374	2.6310	2.6310

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Table 2D-8. Pb-TSP metric ratios, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Ratio of max 3-month mean to annual mean	Ratio of max 3-month mean to annual mean for CBSAs > 1M population
Florida	Hillsborough	120571073	0.423	0.151	2.8013	2.8013
Georgia	Bartow	130150003	0.027	0.015	1.8000	1.8000
Georgia	DeKalb	130890003	0.004	0.003	1.3333	1.3333
Georgia	Muscogee	132150009	0.230	0.128	1.7969	
Georgia	Muscogee	132150010	0.170	0.114	1.4912	
Georgia	Muscogee	132150011	0.071	0.023	3.0870	
Hawaii	Honolulu	150030010	0.001	0.001	1.0000	
Illinois	Cook	170310001	0.024	0.018	1.3333	1.3333
Illinois	Cook	170310022	0.047	0.034	1.3824	1.3824
Illinois	Cook	170310026	0.034	0.023	1.4783	1.4783
Illinois	Cook	170310052	0.024	0.017	1.4118	1.4118
Illinois	Cook	170310110	0.294	0.077	3.8182	3.8182
Illinois	Cook	170310210	0.053	0.032	1.6563	1.6563
Illinois	Cook	170313103	0.041	0.014	2.9286	2.9286
Illinois	Cook	170313301	0.027	0.019	1.4211	1.4211
Illinois	Cook	170314201	0.011	0.010	1.1000	1.1000
Illinois	Cook	170316003	0.042	0.024	1.7500	1.7500
Illinois	Macon	171150110	0.199	0.073	2.7260	
Illinois	Madison	171190010	0.416	0.140	2.9714	2.9714
Illinois	Madison	171193007	0.036	0.020	1.8000	1.8000
Illinois	Peoria	171430037	0.011	0.010	1.1000	
Illinois	Peoria	171430110	0.016	0.012	1.3333	
Illinois	Peoria	171430210	0.015	0.011	1.3636	
Illinois	St. Clair	171630010	0.029	0.020	1.4500	1.4500
Illinois	Whiteside	171950110	0.028	0.020	1.4000	
Illinois	Winnebago	172010110	0.063	0.028	2.2500	
Indiana	Delaware	180350009	0.338	0.159	2.1258	
Indiana	Lake	180890023	0.077	0.031	2.4839	2.4839
Indiana	Lake	180890032	0.060	0.023	2.6087	2.6087
Indiana	Lake	180890033	0.139	0.055	2.5273	2.5273
Indiana	Lake	180892008	0.046	0.020	2.3000	2.3000
Indiana	Marion	180970063	0.079	0.028	2.8214	2.8214
Indiana	Marion	180970076	0.020	0.010	2.0000	2.0000
Indiana	Marion	180970078	0.011	0.006	1.8333	1.8333
Indiana	Porter	181270023	0.022	0.013	1.6923	1.6923

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Table 2D-8. Pb-TSP metric ratios, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Ratio of max 3-month mean to annual mean	Ratio of max 3-month mean to annual mean for CBSAs > 1M population
Indiana	Porter	181270027	0.041	0.027	1.5185	1.5185
Indiana	Vanderburgh	181630020	0.006	0.004	1.5000	
Iowa	Pottawattamie	191550011	0.263	0.123	2.1382	
Iowa	Scott	191630015	0.012	0.010	1.2000	
Kansas	Saline	201690004	0.421	0.164	2.5671	
Kansas	Wyandotte	202090021	0.011	0.008	1.3750	1.3750
Kentucky	Boyd	210190016	0.004	0.003	1.3333	
Kentucky	Madison	211510003	0.248	0.057	4.3509	
Kentucky	Madison	211510005	1.584	0.703	2.2532	
Kentucky	Russell	212070001	0.063	0.027	2.3333	
Louisiana	East Baton Rouge Parish	220330009	0.002	0.002	1.0000	
Louisiana	East Baton Rouge Parish	220330014	0.011	0.004	2.7500	
Louisiana	St. John the Baptist Parish	220950003	0.053	0.025	2.1200	2.1200
Massachusetts	Nantucket County	250190001	0.010	0.009	1.1111	
Michigan	Charlevoix	260290011	0.006	0.005	1.2000	
Michigan	Ionia	260670002	0.049	0.071	0.6901	
Michigan	Ionia	260670003	0.284	0.096	2.9583	
Michigan	Kent	260810020	0.008	0.005	1.6000	
Michigan	Oakland	261250013	0.022	0.019	1.1579	1.1579
Michigan	Tuscola	261570001	0.034	0.021	1.6190	
Michigan	Wayne	261630001	0.006	0.005	1.2000	1.2000
Michigan	Wayne	261630033	0.023	0.012	1.9167	1.9167
Minnesota	Anoka	270031002	0.011	0.006	1.8333	1.8333
Minnesota	Anoka	270036020	0.038	0.017	2.2353	2.2353
Minnesota	Beltrami	270072303		0.001		
Minnesota	Cass	270210001		0.001		
Minnesota	Dakota	270370020	0.004	0.004	1.0000	1.0000
Minnesota	Dakota	270370465	0.259	0.096	2.6979	2.6979
Minnesota	Dakota	270370470	0.002	0.001	2.0000	2.0000
Minnesota	Hennepin	270530963	0.004	0.003	1.3333	1.3333
Minnesota	Hennepin	270530966	0.005	0.004	1.2500	1.2500
Minnesota	Hennepin	270531007	0.004	0.003	1.3333	1.3333
Minnesota	Mille Lacs	270953051	0.001	0.000		
Minnesota	Ramsey	271230871	0.007	0.005	1.4000	1.4000
Minnesota	St. Louis	271377001	0.008	0.002	4.0000	

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Table 2D-8. Pb-TSP metric ratios, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Ratio of max 3-month mean to annual mean	Ratio of max 3-month mean to annual mean for CBSAs > 1M population
Minnesota	St. Louis	271377555	0.004	0.003	1.3333	
Minnesota	Stearns	271453053	0.008	0.004	2.0000	
Minnesota	Washington	271630438	0.005	0.003	1.6667	1.6667
Minnesota	Washington	271630446	0.001	0.000		
Missouri	Iron	290930016	0.882	0.484	1.8223	
Missouri	Iron	290930021	1.147	0.522	2.1973	
Missouri	Iron	290930027	0.083	0.033	2.5152	
Missouri	Iron	290930029	0.115	0.038	3.0263	
Missouri	Iron	290930032		0.354		
Missouri	Iron	290930033	0.057	0.026	2.1923	
Missouri	Iron	290930034	0.402	0.241	1.6680	
Missouri	Iron	290939007	0.581	0.392	1.4821	
Missouri	Iron	290939008	0.387	0.209	1.8517	
Missouri	Jasper	290970005	0.019	0.014	1.3571	
Missouri	Jefferson	290990004	1.122	0.888	1.2635	1.2635
Missouri	Jefferson	290990005	0.396	0.223	1.7758	1.7758
Missouri	Jefferson	290990009	0.191	0.055	3.4727	3.4727
Missouri	Jefferson	290990013	0.305	0.135	2.2593	2.2593
Missouri	Jefferson	290990020	0.865	0.546	1.5842	1.5842
Missouri	Jefferson	290990022	0.630	0.469	1.3433	1.3433
Missouri	Jefferson	290990023	0.563	0.386	1.4585	1.4585
Missouri	Jefferson	290990024	0.659	0.340	1.9382	1.9382
Missouri	Jefferson	290990025	0.085	0.042	2.0238	2.0238
Missouri	Jefferson	290990026	0.080	0.036	2.2222	2.2222
Missouri	Jefferson	290990027	0.592	0.454	1.3040	1.3040
Missouri	Jefferson	290999001	1.100	0.812	1.3547	1.3547
Missouri	Jefferson	290999002	0.431	0.228	1.8904	1.8904
Missouri	Jefferson	290999003	0.327	0.175	1.8686	1.8686
Missouri	Jefferson	290999004	0.442	0.156	2.8333	2.8333
Missouri	Jefferson	290999005	1.432	0.913	1.5685	1.5685
Missouri	Jefferson	290999006	0.103	0.049	2.1020	2.1020
Missouri	Reynolds	291790001	0.059	0.031	1.9032	
Missouri	Reynolds	291790002	0.068	0.034	2.0000	
Missouri	Reynolds	291790003	0.100	0.027	3.7037	
Missouri	Reynolds	291790034	0.161	0.078	2.0641	

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Table 2D-8. Pb-TSP metric ratios, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Ratio of max 3-month mean to annual mean	Ratio of max 3-month mean to annual mean for CBSAs > 1M population
Missouri	St. Francois	291870006	0.132	0.043	3.0698	
Missouri	St. Francois	291870007	0.134	0.053	2.5283	
Missouri	St. Louis	291892003	0.008	0.007	1.1429	1.1429
Missouri	St. Louis city	295100085	0.028	0.026	1.0769	1.0769
Nebraska	Dodge	310530005	0.139	0.055	2.5273	
Nebraska	Douglas	310550019	0.006	0.005	1.2000	
Nebraska	Nemaha	311270002	0.115	0.034	3.3824	
Nevada	Clark	320030540		0.003		
New Mexico	Bernalillo	350010023	0.006	0.003	2.0000	
New York	Orange	360713001	0.101	0.025	4.0400	4.0400
New York	Orange	360713002	1.027	0.123	8.3496	8.3496
New York	Orange	360713004	0.007	0.005	1.4000	1.4000
New York	Suffolk	361030024	0.027	0.017	1.5882	1.5882
Ohio	Butler	390170015	0.009	0.006	1.5000	1.5000
Ohio	Columbiana	390290019	0.057	0.020	2.8500	
Ohio	Columbiana	390290020	0.025	0.015	1.6667	
Ohio	Columbiana	390290022	0.044	0.019	2.3158	
Ohio	Cuyahoga	390350038	0.021	0.013	1.6154	1.6154
Ohio	Cuyahoga	390350042	0.030	0.013	2.3077	2.3077
Ohio	Cuyahoga	390350049	0.531	0.136	3.9044	3.9044
Ohio	Cuyahoga	390350060	0.030	0.020	1.5000	1.5000
Ohio	Cuyahoga	390350061	0.023	0.015	1.5333	1.5333
Ohio	Cuyahoga	390350072	0.035	0.014	2.5000	2.5000
Ohio	Franklin	390490025	0.011	0.009	1.2222	1.2222
Ohio	Fulton	390510001	0.178	0.070	2.5429	
Ohio	Logan	390910006	0.006	0.004	1.5000	
Ohio	Marion	391010003	0.088	0.041	2.1463	
Ohio	Marion	391010004	0.017	0.013	1.3077	
Ohio	Montgomery	391137001	0.008	0.007	1.1429	
Ohio	Stark	391510017	0.023	0.015	1.5333	
Ohio	Trumbull	391550012	0.011	0.007	1.5714	
Ohio	Washington	391670008	0.007	0.005	1.4000	
Ohio	Washington	391670010	0.008	0.005	1.6000	
Oklahoma	Ottawa	401159006	0.022	0.012	1.8333	
Oklahoma	Ottawa	401159007	0.034	0.018	1.8889	

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Table 2D-8. Pb-TSP metric ratios, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Ratio of max 3-month mean to annual mean	Ratio of max 3-month mean to annual mean for CBSAs > 1M population
Oklahoma	Pittsburg	401210416	0.004	0.003	1.3333	
Oklahoma	Tulsa	401431127	0.008	0.006	1.3333	
Oregon	Yamhill	410711702	0.045	0.021	2.1429	2.1429
Pennsylvania	Allegheny	420030002	0.016	0.014	1.1429	1.1429
Pennsylvania	Allegheny	420030008	0.014	0.008	1.7500	1.7500
Pennsylvania	Allegheny	420030070	0.056	0.018	3.1111	3.1111
Pennsylvania	Allegheny	420031009	0.138	0.035	3.9429	3.9429
Pennsylvania	Beaver	420070006	0.085	0.051	1.6667	1.6667
Pennsylvania	Beaver	420070007	0.253	0.166	1.5241	1.5241
Pennsylvania	Beaver	420070505	0.151	0.089	1.6966	1.6966
Pennsylvania	Berks	420110020	0.509	0.158	3.2215	
Pennsylvania	Berks	420110021	0.139	0.044	3.1591	
Pennsylvania	Berks	420110022	0.118	0.035	3.3714	
Pennsylvania	Berks	420111717	0.217	0.146	1.4863	
Pennsylvania	Carbon	420250214		0.104		
Pennsylvania	Delaware	420450002	0.047	0.028	1.6786	1.6786
Pennsylvania	Delaware	420450004	0.047	0.028	1.6786	1.6786
Pennsylvania	Franklin	420550002	0.046	0.028	1.6429	
Pennsylvania	Indiana	420630005	0.049	0.025	1.9600	
Pennsylvania	Lancaster	420710009	0.068	0.039	1.7436	
Pennsylvania	Lawrence	420730011	0.023	0.026	0.8846	
Pennsylvania	Luzerne	420790036	0.137	0.054	2.5370	
Pennsylvania	Philadelphia	421010449	0.029	0.021	1.3810	1.3810
Pennsylvania	Philadelphia	421011002	0.051	0.030	1.7000	1.7000
Pennsylvania	Westmoreland	421290009	0.046	0.023	2.0000	2.0000
South Carolina	Charleston	450190003	0.007	0.005	1.4000	
South Carolina	Florence	450418001	0.044	0.013	3.3846	
South Carolina	Florence	450418002	0.015	0.006	2.5000	
South Carolina	Florence	450418003	0.011	0.005	2.2000	
South Carolina	Greenville	450450015	0.006	0.005	1.2000	
South Carolina	Richland	450790007	0.005	0.004	1.2500	
South Carolina	Richland	450790019	0.024	0.011	2.1818	
Tennessee	Knox	470930023	0.165	0.108	1.5278	
Tennessee	Knox	470930027	0.042	0.020	2.1000	
Tennessee	Knox	470931017	0.037	0.018	2.0556	

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Table 2D-8. Pb-TSP metric ratios, 2010-2012.

State	County	Site	Maximum 3-month mean ($\mu g/m^3$)	Annual average ($\mu g/m^3$)	Ratio of max 3-month mean to annual mean	Ratio of max 3-month mean to annual mean for CBSAs > 1M population
Tennessee	Shelby	471570075	0.005	0.004	1.2500	1.2500
Tennessee	Sullivan	471633001	0.076	0.056	1.3571	
Tennessee	Sullivan	471633002	0.044	0.036	1.2222	
Tennessee	Sullivan	471633003	0.053	0.038	1.3947	
Tennessee	Sullivan	471633004	0.080	0.044	1.8182	
Texas	Cameron	480610006	0.008	0.004	2.0000	
Texas	Collin	480850003	0.371	0.136	2.7279	2.7279
Texas	Collin	480850007	0.199	0.093	2.1398	2.1398
Texas	Collin	480850009	0.774	0.388	1.9948	1.9948
Texas	Collin	480850029	0.180	0.052	3.4615	3.4615
Texas	Dallas	481130069	0.013	0.009	1.4444	1.4444
Texas	El Paso	481410002	0.040	0.022	1.8182	
Texas	El Paso	481410033	0.019	0.017	1.1176	
Texas	El Paso	481410037	0.025	0.022	1.1364	
Texas	El Paso	481410055	0.019	0.010	1.9000	
Texas	El Paso	481410058	0.022	0.016	1.3750	
Texas	Harris	482011034	0.008	0.005	1.6000	1.6000
Texas	Harris	482011039	0.004	0.003	1.3333	1.3333
Texas	Kaufman	482570020	0.104	0.044	2.3636	2.3636
Texas	Potter	483750024	0.020	0.007	2.8571	
Texas	Webb	484790016	0.026	0.018	1.4444	
Utah	Salt Lake	490351001	0.057	0.024	2.3750	2.3750
Virginia	Amherst	510090007	0.018	0.006	3.0000	
Virginia	Buchanan	510270006	0.014	0.012	1.1667	
Virginia	Henrico	510870014	0.005	0.005	1.0000	1.0000
Virginia	Roanoke city	517700011	0.109	0.033	3.3030	
Washington	King	530330029	0.055	0.036	1.5278	1.5278
Washington	Snohomish	530610013	0.023	0.013	1.7692	1.7692
West Virginia	Cabell	540110006	0.012	0.010	1.2000	
Wisconsin	Sheboygan	551170008	0.152	0.056	2.7143	
Puerto Rico	Arecibo Municipio, Puerto Rico	720130001	0.339	0.171	1.9825	
Puerto Rico	Bayamón Municipio, Puerto Rico	720210010	0.011	0.004	2.7500	2.7500
Puerto Rico	Salinas Municipio, Puerto Rico	721230002	0.017	0.008	2.1250	

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 $\mu g/m^3$, 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 (2010-2012) 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 $\mu g/m^3$, 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 $\mu g/m^3$ (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 (2010-2012) 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 2010-2012 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 $\mu g/m^3$ * 1/1.92 = 0.078 $\mu g/m^3$). 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 ($\mu g/m^3$)
Maximum Quarterly Average [D] ($\mu g/m^3$)	Maximum Monthly Average ($\mu g/m^3$)	Maximum 3-month Average ($\mu g/m^3$)	
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 2010-2012 air quality dataset.

2. *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 $\mu g/m^3$.

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 (IQ $Loss_{Scenario\ X}$ – IQ $Loss_{Scenario\ Y}$)/ (annual $average_{Scenario\ X}$ – annual $average_{Scenario\ Y}$). 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.

Table 3A-2. Risk estimates for bounding air quality scenarios and associated slopes.

	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).

4. *Derive interpolated risk estimates for the current standard scenario.* Using the slopes presented in Table 3A-2 and the lower bounding air quality scenario risk estimates, derive corresponding risk estimates for the current standard scenario:

   Risk = lower bounding risk + (increment annual average Pb * slope)

   Where:

   - lower bounding risk = risk for the lower bound air quality scenario (i.e., for 0.2 µg/m^3 maximum monthly average)
   - increment annual average Pb is the difference between the annual average estimates for the current standard and the lower bounding scenario.
   - slope is the value described in Step 4, which differs across the 8 combinations of C-R functions and exposure pathway categories.

   The resulting interpolated risk estimates are presented in Table 3A-3.

Table 3A-3. Interpolated risk estimates for the current NAAQS scenario for the 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).

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
Seconday Smelter Case Study	Troy (Pike County), 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 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 $g \cdot ha^{-1} \cdot yr^{-1}$). (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.

ATTACHMENT

Clean Air Scientific Advisory Committee Letter (June 4, 2013)

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

June 4, 2013

EPA-CASAC-13-005

The Honorable Bob Perciasepe
Acting Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, D.C. 20460

Subject: CASAC Review of the EPA's *Policy Assessment for the Review of the Lead National Ambient Air Quality Standards (External Review Draft – January 2013)*

Dear Acting Administrator Perciasepe:

The Clean Air Scientific Advisory Committee (CASAC) Lead Review Panel met on February 5 - 6, 2013, to peer review the EPA's *Policy Assessment for the Review of the Lead National Ambient Air Quality Standards (External Review Draft – January 2013)*, hereafter referred to as the PA. The CASAC's consensus responses to the agency's charge questions and the individual review comments from the CASAC Lead Review Panel are enclosed. The CASAC's key points are highlighted below.

Overall, the CASAC concurs with the EPA that the current scientific literature does not support a revision to the Primary Lead (Pb) National Ambient Air Quality Standard (NAAQS) nor the Secondary Pb NAAQS. Although the current review incorporates a substantial body of new scientific literature, the new literature does not justify a revision to the standards because it does not significantly reduce substantial data gaps and uncertainties (e.g., air-blood Pb relationship at low levels; sources contributing to current population blood Pb levels, especially in children; the relationship between Pb and childhood neurocognitive function at current population exposure levels; the relationship between ambient air Pb and outdoor dust and surface soil Pb concentrations). Further details on these and other research needs are provided in the consensus responses. The CASAC recommends that research be performed to address these data gaps and uncertainties to inform future Pb NAAQS reviews.

The CASAC has additional comments and recommendations on improving the document. With the completion of the recommended revisions outlined below and in the consensus responses, the PA will serve its intended purpose. Another CASAC review of the document is not needed.

The PA should include a discussion that Pb is a unique pollutant in many ways. Unlike other criteria air pollutants, Pb is of concern from a multimedia perspective. Millions of tons of Pb are present in the environment from legacy sources. The distribution of this substantial reservoir of Pb is not known. Thus, the extent of current human exposure from this legacy cannot be reliably estimated.
The PA generally captures the key aspects of the health effects evidence presented in the Integrated Science Assessment, but can be made more concise and clear by providing summary conclusions

regarding the health effects evidence at the beginning of the sections. The risk and exposure information from the previous Pb NAAQS review is adequately presented. The CASAC concurs that a new risk and exposure assessment (REA) is not needed due the lack of sufficient new scientific information to warrant revision of the prior REA data and methods.

The application of the evidence-based framework and the use of the health risk and exposure information from the previous Pb NAAQS review seem appropriate and provide a sufficient rationale to support retaining the current primary standard without revision. However, there should be more description in the PA of the data and rationale behind the averaging time and form of the current primary standard. Additionally, research is needed to address uncertainties and data gaps in the evidence-based framework and in the health risk and exposure information for future Pb NAAQS reviews.

Given the existing scientific data, the CASAC concurs with retaining the current secondary standard without revision. However, the CASAC also notes that important research gaps remain. For example questions remain regarding the relevance of the primary standard's indicator, level, averaging time, and form for the secondary standard. Other areas for additional research to address data gaps and uncertainty include developing a critical loads approach for U.S. conditions and a multi-media approach to account for legacy Pb and contributions from different sources. Addressing these gaps may require reconsideration of the secondary standard in future assessments.

The CASAC also wishes to highlight the importance of a separate but related policy issue. The CASAC notes that it has not considered this separate policy issue in providing advice on the NAAQS. The decrease in childhood lead poisoning in the United States over the last three decades is a great public health success story. Concurrently, there is a trend of increased relocation of Pb production, recycling, and recovery to other nations. One example is the export of spent lead acid batteries (SLAB) to Mexico. As detailed in a report by the Secretariat of the Commission for Environmental Cooperation (CEC), environmental and health protections in the secondary lead industry in other countries are not functionally equivalent to those in the United States. The CEC offers recommendations to avoid development of pollution havens and for the United States to work with Mexico and Canada to foster adoption of best practices throughout North America. The CASAC recognizes the role of the EPA Administrator as a member of the CEC Council and strongly urges the EPA to carefully consider the recommendations of the CEC report, and to support decisive action that will enable the success of the Pb NAAQS in the United States to be a role model for development of best practices internationally that avoid adverse public health impacts abroad. For example, the considerable effort by the EPA to develop the ISA and the PA could be a valuable starting point for other countries to develop or revise their own ambient standards for lead.

The CASAC appreciates the opportunity to provide advice and looks forward to receiving the EPA's response.

Sincerely,

/signed/

Dr. H. Christopher Frey, Chair
Clean Air Scientific Advisory Committee

Enclosures

NOTICE

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

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

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Consensus Responses to Charge Questions on
EPA's Policy Assessment for the Review of the Lead National Ambient Air Quality Standards
(External Review Draft – January 2013)

Chapter 1 – Introduction

This chapter provides context for the review, including the background of past reviews, as well as the scope for the current review. This includes discussion of fate and multimedia pathways of ambient air Pb and other nonair sources of Pb in the environment.

Does the Panel find the introductory and background material, including that pertaining to previous reviews of the Pb standard and the scope of the current review to be appropriately characterized and clearly communicated?

Chapter 1 of the Policy Assessment (PA) is reasonably well characterized and for the most part clearly communicated, although there are several improvements that should be made (see below). The CASAC concurs with the EPA that the current scientific evidence does not justify a change to the current Lead (Pb) National Ambient Air Quality Standards (NAAQS), with the caveats stated below.

Chapter 1 should emphasize Pb as a unique pollutant in many ways. Historically, anthropogenic emissions of Pb to the air around the globe exceeded natural emissions by a huge margin, more than any other trace metal. Natural emissions come from unpolluted soil, seaspray, and other natural sources. Removal of Pb from gasoline, paint, solder, and other anthropogenic sources constitutes what is arguably the biggest environmental success story for any pollutant to-date. Unlike other criteria air pollutants, Pb is of concern from a multimedia perspective; human exposure to Pb comes from inhalation of air and also from ingestion of food, water, and dust. In addition, Pb may be the pollutant with the biggest legacy problem: millions of tons of Pb are now present in the environment as a result of discharges from years ago. The distribution of this huge reservoir of Pb is not known, and thus current human exposure from this legacy cannot reliably be estimated. What is known, however, is that human activities have on average substantially elevated the Pb content of soil at numerous locations around the United States, more so than other trace metals processed in large quantities. Although air Pb levels are much lower because of reduced emissions, soil Pb can be expected to remain elevated for many years. These unique aspects of Pb, especially the problem of not knowing the distribution of legacy Pb, should be clearly discussed in the PA.

This chapter concludes that there is no information published in the last five years justifying reconsideration of the current NAAQS. Although that is true, this conclusion should be conveyed with an assessment of the adequacy of the old information. In particular, there were significant unknowns and uncertainties associated with a lack of information five years ago; those unknowns and uncertainties still remain.

There are no statements in the PA that the Integrated Science Assessment (ISA) is limited only to exposures and data sources considered currently relevant to the U.S. population, as opposed to populations outside of the United States. Furthermore, the literature is considered only to assess how new studies relate to conclusions drawn in the past review, and then only to studies in the peer-reviewed literature. This has resulted in an ISA that is dedicated predominately to toxicology, health effects,

biokinetics, and causal determinations. These are areas that were data rich in the last review, and continue to produce volumes of new peer-reviewed information. In contrast, in areas where the least is known and EPA relies on past findings, uncertainty is becoming greater as the existing information becomes outdated. Additionally, the current assessment *excludes* consideration of impacts on populations outside of the United States.

There is considerable discussion dedicated to the reduction of Pb in air and other media in the United States over the last four decades. Most of the reduction was achieved through the elimination of tetra-ethyl Pb gasoline additives. Another major component of the reduction was substantial decreases in emissions from primary and secondary smelters, and metals processing industries. In the case of motor vehicle gasoline-related emissions, these ceased and other non-Pb products were substituted in commerce. This resulted in decreased Pb emissions and health and environmental effects in both the United States and globally. Within the United States, Pb continues to be used as an octane boosting fuel additive for very high octane fuels used in general aviation for piston engine aircraft. In the case of Pb production and secondary recovery, however, this production and recovery were exported overseas. There is no mention of the impact of the "avoided emissions" in their new locales.

Thus, overall, there is no reason to change the current airborne Pb standard. However, although airborne Pb is far more limited as a problem within the United States, potential exposure to Pb in other environmental media in the United States is likely to be more significant. Furthermore, there may be increasing Pb exposures overseas (e.g., as described in CEC, 2013).

There are a few areas that need revision for clarity and accuracy. On page ES-1, line 25, the following sentence should be added to the end of the paragraph: "This approach was taken to aid in the decision to retain or revise the current standards." On page 1-13 line 35, it states: "And we recognize that past Pb emissions in many situations were well in excess of the current Pb standard." One cannot compare emissions to an airborne standard. This sentence can be revised to state: "We recognize that past Pb emissions in many situations caused airborne Pb concentrations far in excess of the current Pb standard."

Chapter 2 – Ambient Air Lead

This chapter provides an overview of current information on air Pb emissions and monitoring data, consideration of the current air Pb monitoring requirements and an overview of current information on Pb in nonair media.

To what extent does the Panel agree that the most relevant information on emissions (section 2.1), air quality (section 2.2.2), and Pb concentrations in other media (section 2.3) is presented, and to what extent is the information presented appropriately characterized and clearly communicated?

With a few minor exceptions (see specific individual panel member comments), the information on Pb emissions, air quality and concentrations in other media is appropriately characterized and clearly presented. Historical and recent (2008) emissions data are summarized quantitatively in clear charts and tables, with additional detail on the 2008 National Emissions Inventory (NEI) data sources and limitations provided in Appendix 2A. There are also qualitative discussions and an informative Appendix 2B on recent regulatory actions, indicating that current emissions have declined since 2008, with additional reductions pending. However, quantitative estimates of emissions reductions would be

informative. To the extent that these or other historical controls of U.S. Pb source categories have shifted Pb emissions to other countries, it would be informative to include discussion of those displaced emissions.

The information on ambient air concentrations is clearly presented (for sites with 1 to 3 years of valid data for 2009 to 2011) in maps, charts and in a detailed appendix (Appendix 2D). More recent measurements from sites (including near airports) initiated since the previous Pb NAAQS review would help inform the current Pb NAAQS review, as well as the separate Section 231 aviation gasoline ("avgas") review.

Given the "not to be exceeded" 3-month rolling average form of the Pb NAAQS, an exceedance could be determined with as few as 3 months of new data. Therefore, it would be useful if more recent data are summarized in the next Pb PA, without being limited to sites with multiple years of valid data.

The information on Pb concentrations in other media is clearly presented. The sections (2.3.2.1 and 2.3.2.2) on indoor and outdoor dust are highly relevant to exposure assessments and would benefit from some added discussion of how dust Pb concentration, loading, and loading rates are measured. In particular, information relating to the differences in particle sizes in dust samples and ambient air samples would be helpful. More information on changes, if any, over time in the availability of historically deposited soil Pb for resuspension to the air or for direct uptake through ingestion could help clarify the significance of this potentially important source category.

With regard to information on ambient Pb monitoring (section 2.2.1), to what extent is this information appropriately characterized and clearly communicated?

The information on ambient Pb monitoring is appropriately characterized and clearly communicated. It is understood that the high-volume (Hi-Vol) Total Suspended Particulates (TSP) sampler is an imperfect historical artifact, and that there is not time for this review cycle of the Pb NAAQS to develop, fully test, and deploy alternative samplers that would consistently capture particles (less than and) greater than 10 microns with appropriate collection efficiencies and size ranges under varying wind speeds and directions. The draft PA notes that the EPA expects a new, improved sampler to be "available for consideration in a future review." Toward this goal, discussion is needed regarding the desirable cut size characteristics of, and practical constraints on, an alternative sampler. Information from the ISA could be cited here, such as material currently on page 3-67 of the 3rd external draft of the ISA that may be revised for the final ISA regarding discussion of the desirable cut size.

If an alternative low-volume sampler could be developed with an upper 50% particle cut size in the range of 15 to 20 microns, and without the wind speed and direction biases of the Hi-Vol TSP sampler, it seems likely that such a sampler would typically capture as much (or occasionally more) Pb as the Hi-Vol TSP sampler. The CASAC has previously recommended the development of a new air Pb sampler that collects larger particle sizes, that could improve the quality of sampling in the National Air Toxics Trends Station (NATTS) network, and that could serve as an Federal Reference Method (FRM) or Federal Equivalent Method (FEM) for Pb. Filters collected by this sampler also would be amenable to multi-elemental analyses by lower-cost analytical methods and could be useful for more accurate and precise assessments of other particulate pollutants with significant coarse mode concentrations, including chemical contaminants (like hexavalent chromium, silica, and cadmium) and biological components like pollen, fungi, and endotoxins.

Chapter 3 – Health Effects and Exposure/Risk Information

This chapter discusses key policy relevant aspects of the health effects evidence and exposure/risk information.

To what extent does the information in sections 3.1 (Internal Disposition and Biomarkers of Exposure and Dose), 3.2 (Nature of Effects) and 3.3 (Public Health Implications and At-Risk Populations) capture and appropriately characterize the key aspects of the evidence assessed and integrated in the ISA?

Chapter 3 of the draft PA generally captures the key aspects of the evidence presented in the ISA; therefore, concerns about content stem from the content of the ISA and not its condensation in the PA.

Recognizing the degree of condensation needed to summarize the ISA in only a few pages, this format presents a great writing challenge, and the writing in the chapter lacks clarity. Wordiness should be reduced to directly and efficiently convey the meaning. For example, sentences such as the following could be shortened and recast in a more active voice: "The results from the various case studies assessed, with consideration of the context in which they were derived (e.g., the extent to which the range of air-related pathways were simulated, and limitations associated with those simulations), and the multiple sources of uncertainty (see section 3.4.7 below) are also informative to our understanding of air-to-blood ratios." (Please refer to Dr. Canfield's individual comments for details on the sentences in need of streamlining and clarification.)

In addition, there are a few places where legacy text from the previous PA remains, so extraneous words need to be deleted and tenses updated. What needs the most attention is the length and complexity of some sentences.

To what extent is the newly available evidence on air-to-blood ratios appropriately characterized and considered in light of information previously available in past reviews?

The new information on air-to-blood ratios is presented in context of previous information and no change in the estimate is justified at this time.

When revising this section (3.1) it would be helpful to first read the last (summary paragraph) on page 3-14. Those conclusions should be included in the first paragraph of the section and then restated at the end of the section when the reader will be in a position to understand the context.

To what extent is the newly available evidence on concentration-response functions for IQ decrements in young children appropriately characterized and considered in light of information previously available in past reviews?

The newly available evidence is appropriately characterized. Parametric information could be extracted from these data to produce quantitative results for blood Pb subgroups, but the quality of the data would not likely provide a useful basis for altering the conclusions reached from the data available prior to 2008.

There is a critical need for more information about effects of Pb at levels in the 0-5 µg/dL range. Studies about effects of Pb at these low levels are a future research need.

With regard to the exposure and risk information, to what extent is the information drawn from the human exposure and health risk assessment in the last review sufficiently characterized and clearly communicated? To what extent is the information appropriately interpreted in light of the currently available information and for the purpose of assessing the adequacy of the now current standard?

Information from the last review is well-characterized and appropriately interpreted, but the clarity of communication should be improved. Putting the conclusion in the first paragraph of the section was very helpful. The CASAC concurs that a new risk and exposure assessment (REA) is not warranted at this time.

Are the limitations and uncertainties in the exposure/risk information appropriately characterized and considered in our interpretation of the information in the context of this current review?

The limitations and uncertainties in the exposure/risk information are well characterized. Epidemiological data are inherently limited; there is a lack of data on the effects of Pb exposure at the levels that are common today and thus concentration-response (C-R) estimates require extrapolation. Parameter choices for biokinetic models typically require restrictive assumptions. No single air quality scenario is adequate.

There are specific areas of the chapter that should be revised for clarity:

- **Page 3-2, Line 15,** referring to distribution of Pb from bone to blood, includes the sentence: "Changes in Pb exposure circumstances also can influence these exchanges, e.g., substantial reductions in exposure levels contribute to increased release of Pb from the bone into the blood (ISA, section 4.3.5)." This sentence should be revised, as it appears to incorrectly characterize the information in section 4.3.5 of the ISA. A reduction in external Pb exposure does not, from a pharmacodynamic standpoint, induce an increase in the release of Pb from bone. In the context of the paragraph, the intended point could be expressed as follows: "When there are substantial reductions in external Pb exposure, the relative contribution of Pb from bone to the concentration of Pb in blood increases."

- **Page 3-5, line 29 to Page 3-6, line 9:** The italicized sentence in this paragraph, reproduced below, should be revised to improve clarity:

  "The response of adult blood Pb levels to appreciable changes in exposure circumstances is generally slower than that of blood Pb levels in young children. For example, simulations using biokinetic models indicate that blood Pb levels in adults achieve a new quasi-steady state within 75-100 days (approximately 3-4 times the blood elimination half-life) subsequent to abrupt increases in Pb intake (ISA, section 4.3.5.2); similar models indicate a much quicker response of blood Pb levels in children both with regard to abrupt increases and reductions in Pb exposure (ISA, section 4.3.5.1). *The response in young children may reflect their much more labile bone pool associated with the rapid turnover of bone mineral in response to their rapid growth rates (ISA, section 4.3.5*). As a result of these physiological processes in young children, their blood Pb levels tend to more quickly reflect changes in their total body burden (associated with their shorter exposure history), and also can reflect changes in recent exposures (ISA, section 4.3.5)."

Instead of the italicized sentence above, substitution of a sentence such as the following might enhance clarity: "Because the skeletal compartment of Pb is relatively smaller and subject to more rapid turnover in children compared to adults, the blood Pb concentration of children is more reflective of their recent external exposure."

- **Page 3-19 lines 23-24**: It might be clearer to use the phrase "qualitatively change" rather than "appreciably change" to allow for appreciable strengthening of the previous conclusion but no change of consequence.

- **Page 3-22 lines 17-23:** This is a helpful discussion of the problem with unknown earlier exposures in adults and older children. The topic comes up multiple times throughout this document and becomes redundant. Maybe this should be covered fully (including this as a general critique of cross-sectional studies), early in the chapter and then simply referred back to when the context warrants.

Chapter 4 – Review of the Primary Standard for Lead

This chapter describes the basis for the current primary standard and consideration of the current evidence and exposure/risk-based information with regard to reaching preliminary staff conclusions about the adequacy of the current standard.

In this chapter, staff applies the same evidence-based air-related IQ loss framework as developed and used in the last review, which has fundamentally two key inputs: an air-to-blood ratio and the slope of a concentration-response (C-R) function for IQ decrements in young children.

This draft PA is constrained by the absence of new observational and experimental data that address, at least in part, limitations and uncertainties in the evidence that was present at the time of the last update of the Pb NAAQS. Until evidence is available to assess Pb exposure and health risks related to air Pb levels reflecting the current standard, a substantive refinement and update of the PA will not be possible. The obvious uncertainty underlying evaluation of this PA is whether lowering the standard would (or would not) impact exposure and thus risk. The CASAC agrees with the EPA conclusion that "there is appreciable uncertainty associated with drawing conclusions regarding whether there would be reductions in blood Pb levels from alternative lower levels as compared to the level of the current standard." If lowering the primary standard would lower blood Pb levels amongst the U.S. population, then there would be potential public health benefits from a lower standard. Research priorities discussed below are designed to help inform these uncertainties.

To what extent does the Panel agree with application of the evidence-based framework from the last review, particularly with regard to consideration of the currently available information, and related limitations and uncertainties, for air-to-blood ratios and C-R functions for IQ decrements in young children?

The application of the evidence-based framework from the previous Pb NAAQS review seems appropriate. The new literature published since the previous review provides further support for the health effect conclusions presented in that review. Additionally, the new studies do not fundamentally alter the uncertainties for air-to-blood ratios or C-R functions for IQ decrements in young children.

As previously discussed with CASAC, staff concluded that the current information does not warrant development of a new REA in this review. Thus, exposure/risk information was drawn from the REA conducted in the last review.

What are the Panel's views on staff's interpretation of the exposure/risk information, and on staff's conclusions that the information is generally supportive of conclusions drawn from the evidence-based framework as to the adequacy of the current standard?

The use of exposure/risk information from the previous Pb NAAQS review appears appropriate given the absence of significant new information that could fundamentally change the interpretation of the exposure/risk information. This interpretation is reasonable given that information supporting the current standard is largely unchanged since the current standard was issued.

The CASAC agrees that the adverse impact of low levels of Pb exposure on neurocognitive function and development in children remains the most sensitive health endpoint, and that a primary Pb NAAQS designed to protect against that effect will offer satisfactory protection against the many other health impacts associated with Pb exposure.

The CASAC concurs with the draft PA that the scientific findings pertaining to air-to-blood Pb ratios and the C-R relationships between blood Pb and childhood IQ decrements that formed the basis of the current Pb NAAQS remain valid and are consistent with current data.

In reaching preliminary staff conclusions, staff notes that, like any NAAQS review, this Pb NAAQS review requires public health policy judgments. The public health policy judgments for this review include the public health significance of a given magnitude of IQ loss in a small subset of highly exposed children (i.e., those likely to experience air-related Pb exposures at the level of the standard), as well as how to consider the nature and magnitude of the array of uncertainties that are inherent in the evidence and in the application of this specific framework.

What are the Panel's views on public health policy judgments that inform staff's preliminary conclusions with regard to the adequacy of current standard and a lack of support for consideration of potential alternative standards?

The PA states repeatedly that no threshold for Pb effects on IQ can be identified. In some respects, the ability to define a threshold may already be a moot issue. Reductions in IQ in children are being reported at blood Pb values as low as 2 µg/dL. In essence, these effects are being reported at the lowest levels of Pb in blood that can be reliably measured by most laboratories doing such analyses.
Child IQ is the Pb-sensitive health endpoint on which this PA (and the previous one) is based. Thus, the discussion of health policy judgment needs to be carefully considered in light of the far-reaching public health value of childhood cognitive and neurobehavioral health. For example, the 2012 Centers for Disease Control and Prevention (CDC) update of recommendations regarding childhood Pb poisoning acknowledges that there is no blood Pb level in childhood that has been shown to be without deleterious effects. In this context, defining the threshold for "unacceptable risks to public health" or "sufficient public health protection" is difficult. Indeed, such language - with its implicit use of a threshold approach to a process that presumably has no threshold - may no longer be appropriate. Although there is evidence that even very low Pb levels are related to measurable reductions in IQ in children, the extent to which the blood Pb levels observed in children are linked to ambient air Pb levels below the

current standard (as opposed to other sources of Pb in the environment) has not been established. Therefore there is not justification for modifying the current standard based on these data at this point in time. However additional research on air to blood Pb at low levels may require reconsideration of this decision in the future.

In the Panel's view, does the discussion in section 4.3 provide an appropriate and sufficient rationale to support staff's preliminary conclusion that it is appropriate to consider retaining the current standard (including the indicator, level, averaging time, and form) without revision?

Given the evidence-based framework, the discussion in section 4.3 provides an appropriate and sufficient rationale to support retaining the current standard without revision. For example, there is discussion in the PA regarding the choice of indicator level. However, there should be more description of supporting data and rationale behind the recommendation for the averaging time and form of the current standard.

The CASAC concurs that the new science does not support lowering the Pb NAAQS from its current level (0.15 $\mu g/m^3$). Additionally, the CASAC concurs with the caveats provided about the uncertainty in the science behind the NAAQS for Pb. In particular it appreciates and affirms a key point on page 4-28, lines 2-3: "We also recognize increased uncertainty in projecting the magnitude of blood Pb response to ambient air Pb concentrations at and below the level of the current standard." Likewise, the key idea on page 4-32, lines 32-35 is important, but a clarifying revision is recommended as follows:

> Page 4-32, lines 32-35, current text: "In staff's view, based on current evidence there is appreciable uncertainty associated with drawing conclusions regarding whether there would be reductions in risk to public health from alternative lower levels as compared to the level of the current standard." This should be re-written to read "In staff's view, based on current evidence there is appreciable uncertainty associated with drawing conclusions regarding whether there would be reductions in ***blood lead levels*** from alternative lower levels as compared to the level of the current standard."

Does the Panel have any recommendations regarding additional interpretations and conclusions based on the available information that would be appropriate for consideration beyond those discussed in this chapter?

As noted above, repeated statements about a threshold do not seem warranted given that IQ reductions now occur at the lowest blood Pb levels that can be reliably measured in most laboratories. It is for this reason that the Advisory Committee on Childhood Lead Poisoning Prevention recommended to CDC a complete elimination of the phrase 'level of concern' and stated that no blood Pb level in children has been shown to be without deleterious effects.

The EPA should encourage development of research programs to address those limitations and uncertainties in currently available evidence (and exposure/risk information) that are critical to the identification of "sufficiently health protective" air Pb standards in the future.

There are some areas that need revision for clarity:

- Page 4-34, line 13: The statement should be edited to: "Factors affecting relationships between Pb in ambient air and Pb in blood ***at low exposures experienced in the general population today"***
- Page 4-34, lines 19-21: This research need is profound, but as written is too vague to be appreciated by the average reader. This research need should be revised to: "Apportionment of blood Pb levels with regard to exposure pathways, with particular focus on understanding exposure pathways and sources that cause the more elevated blood Pb levels among children today."

Research Needs

Several areas of research that could assist in further refinement of the Pb NAAQS include:

1. For the purposes of policy and decision making, ***key*** research priorities should be studies that elucidate: (1) the air-blood Pb relationship at low levels; (2) sources (exposure pathways) contributing to current population blood Pb levels, especially in children; (3) the relationship between Pb and childhood neurocognitive function at current population exposure levels; and (4) the relationship between ambient air Pb and outdoor dust and surface soil Pb concentrations, *including the temporal dynamics of that relationship*. These research priorities are of particular interest because of the prominent contribution of past (as opposed to recent) Pb emissions to Pb in soil and dust, and the significant contribution of dust and soil matrices to the Pb exposure of children.

2. For the typical American adult not subject to current or past point sources or occupational Pb exposure, Pb in the diet is likely to constitute the largest fraction of daily Pb exposure. Therefore, another research need of considerable interest is to determine the source of contemporary dietary Pb, including the indirect contribution of historical air Pb emissions (i.e. "legacy Pb"). Further, there remains a need to determine how much of dietary Pb is from legacy and how much can be amenable to interventions.

3. The shape of the C-R curve for IQ reductions at extremely low levels requires further clarification. In addition, studies on more sensitive endpoints in the domain of emotion and behavior regulation are warranted, given that they may yield specific and sensitive measures and thereby assist in defining appropriate intervention strategies for children.

4. There has been a long-term reliance on the Integrated Exposure Uptake Biokinetic (IEUBK) model. However, greater understanding of inter-individual variability, as quantified in the Geometric Standard Deviation (GSD) input parameter, is needed. Information about toxicokinetics during adolescence remains limited. The All Ages Lead Model could be utilized to improve this understanding. There is also the need to know more about gene-environment interactions, particularly in driving inter-individual susceptibility and vulnerability.

5. Characterization and better understanding of Pb exposure hotspots/sources will give better representation of significant exposure risks.

6. Understanding of the impacts of Pb exposures during critical developmental windows and their contribution to adverse outcomes; e.g., little is known about the specific effects of prenatal exposure.

7. The effect of contemporary Pb exposure (i.e., that resulting in blood Pb concentrations on the order of 5 µg/dL or lower) on the future risk of hypertension and cardiovascular morbidity and mortality, and on age-related neurodegeneration, as these could lead to additional information related to most sensitive health outcomes.

8. A further understanding of the role of "reverse causation" in the inverse association observed in some studies between low blood Pb concentration and renal function (e.g., glomerular filtration rate).

9. The extent to which product substitution, i.e., replacement of Pb with less hazardous alternative substances in contemporary commerce, may result in reduction of human Pb exposure. The EPA might consider supporting studies on Pb potentially undertaken by programs such as the Toxic Use Reduction Program in Massachusetts, and the Green Chemistry Initiative in California.

10. As yet, the extent to which global warming will influence exposure to Pb (e.g., through soil erosion, resuspension) has not been evaluated.

Chapter 5 – Welfare Effects and Exposure/Risk Information

This chapter discusses key policy relevant aspects of the environmental evidence and exposure/risk information.

Chapter 5 of the PA is a well-written synthesis of the findings related to ecological effects in the ISA. The ISA supports the conclusion that recent research has not changed our fundamental understanding of Pb fate, transport and toxicity in the environment.

To what extent does the information in section 5.1 (Welfare Effects Information) capture and appropriately characterize the key aspects of the evidence assessed and integrated in the ISA?

Section 5.1 does a good job of summarizing the evidence for ecological effects from the ISA. The general conclusion is that recent research has added depth and nuance to the understanding of the fate and transport of Pb in ecological systems, and to the understanding of effects on organisms in terrestrial and aquatic ecosystems, but has not changed the understanding in a way that merits reconsideration of the relationships used to assess risk.

A persistent theme in the ecological effects sections of the ISA and this PA document is that it is difficult to isolate the effects of air Pb on ecosystems from other Pb sources, including "legacy" Pb accumulated in soils and sediments. The threat of release of legacy Pb in soils and sediments, whatever the original source, may necessitate a lower secondary air quality standard than would be warranted in the absence of the legacy Pb. With respect to critical loads, it is recommended that Chapter 5 acknowledge the impact on raptors and water fowl of Pb in spent ammunition; these are the ecological

receptors most heavily exposed to Pb, and thus potentially the most susceptible to the incremental contribution of Pb in air.

With regard to the exposure and risk information in section 5.2 (Exposure and Risk Information), to what extent is the information drawn from the screening-level risk assessment in the last review sufficiently characterized and clearly communicated? To what extent is the information appropriately interpreted in light of the currently available information and for the purpose of assessing the adequacy of the current standard?

The results of the 2006 REA are summarized in section 5.2. The summary is concise and clear, both in the explanation of the model employed and in the descriptions of the case studies used in the assessment.

The interpretation of the results from the 2006 REA is appropriate insofar as it re-states the conclusions from that document, and there have been no fundamental changes to our understanding of key thresholds or ecological receptors in the intervening years. Of four terrestrial case studies employed, the results from two (the primary and secondary smelter cases) are judged to be "not informative." The relevance of a third case study (non-urban near-roadway conditions) is deemed "highly uncertain" due to the presence of legacy Pb in roadside soils. The only terrestrial case study that is deemed relevant is the Hubbard Brook case, where ambient Pb concentrations are far below the current (and proposed) standard. Results from analysis of surface water and sediment data are judged to be inconclusive because of possible non-air sources to waters and legacy Pb in sediments. Therefore, overall, four of the five major efforts in the 2006 REA are judged to be of limited or no value for the purposes of this PA. Given that there is little field research underway on Pb in U.S. ecosystems that are not impacted by point sources, it would appear to be unlikely that data for new REA case studies is forthcoming. A robust critical loads approach, which is a research priority to support a future review, is needed to fill this gap.

Are the limitations and uncertainties in the exposure/risk information appropriately characterized and considered in our interpretation of the information in the context of this current review?

The discussion of limitations and uncertainties is generally good. Issues such as legacy Pb, multi-stressor effects, and lab-to-field applicability create considerable uncertainty. The use of conservative screening levels in the calculation of hazard quotients is particularly useful because the calculated risks are overstated.

Chapter 6 – Review of the Secondary Standard for Lead

This chapter describes the basis for the current secondary standard and consideration of the current evidence and exposure/risk-based information with regard to reaching preliminary staff conclusions about the adequacy of the current standard.

Does the Panel agree with preliminary staff conclusions about the evidence and previous risk assessment in light of current standards as presented in section 6.2 (Adequacy of the Current Standard)?

The preliminary staff conclusions provide a good assessment of the available evidence and the previous risk assessment in light of the current secondary standard. The CASAC notes, however, the concerns raised in the response to the chapter 5 charge questions regarding the previous risk assessment.

In the Panel's view, does the discussion in this chapter provide an appropriate and sufficient rationale to support preliminary staff conclusions that it is appropriate to consider retaining the current standard (including the indicator, level, averaging time, and form) without revision?

The discussion provides appropriate and sufficient rationale to support retaining the current secondary standard without revision. A general lack of new data that would indicate the appropriate level of Pb in environmental media that may be associated with adverse effects suggests that the secondary standard should be retained. Questions remain regarding the relevance of the indicator, level, averaging time and form to the secondary standard (ecological context). A multi-media approach may be necessary to account for legacy Pb and contributions from different sources for a secondary standard.

Does the Panel have any recommendations regarding additional interpretations and conclusions based on the available information that would be appropriate for consideration beyond those discussed in this chapter?

The CASAC does not have any recommendations regarding additional interpretations and conclusions beyond what is contained in the chapter. Developing a critical loads approach for U.S. conditions would be an important area for additional research. The discussion of uncertainties at the end of the chapter is excellent. It should include mention of the use and/or relevance of toxicity data that are generated in test systems that deploy exposures to media other than soil or water for appropriate organisms (e.g., plants in hydroponic systems, soil nematodes in agar or culture medium).

Research Needs

Application of a critical loads approach with sensitivity analysis will help to determine which processes are most important in determining Pb exposure to ecological receptors. This would be an integrated, holistic, multi-media approach that could be used to examine the contributions of current aerial Pb deposition to historical aerial deposition as well as Pb from other sources. Current critical loads models are largely qualitative and empirical. Mechanistic sub-models need to be incorporated into the critical loads model to provide an adequate means to predict Pb bioavailability, exposure, and toxicity. This critical loads approach could be integrated to include other aerial pollutants such as oxides of nitrogen and oxides of sulfur.

Reference

Secretariat of the Commission for Environmental Cooperation (CEC), (2013), *Hazardous Trade? An Examination of US-generated Spent Lead-acid Battery Exports and Secondary Lead Recycling in Mexico, the United States and Canada*, Final Report, 15 April.

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