Review of the National Ambient Air Quality Standards for Lead:

Risk and Exposure Assessment Planning Document

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1 INTRODUCTION

The U.S. Environmental Protection Agency (EPA) is conducting a review of the air quality criteria and the national ambient air quality standards (NAAQS) for lead (Pb). The purpose of this planning document is to describe the consideration of the extent to which newly available scientific evidence and tools or methodologies warrant the conduct of quantitative risk and exposure assessments (REAs) that might inform this review. Also considered is the extent to which newly available evidence may refine our characterization of exposure and risk estimates provided by the assessments conducted for the last review.

The previous Pb NAAQS review, completed in fall of 2008, resulted in substantial revision to the standards (73 FR 66964). In consideration of the much-expanded health effects evidence on neurocognitive effects of Pb in children, EPA substantially revised the primary standard from a level of 1.5 micrograms per cubic meter ($\mu g/m^3$) to a level of 0.15 $\mu g/m^3$. EPA's decision on the level for the standard was based on the weight of the scientific evidence and guided by an evidence-based framework that integrates evidence for relationships between Pb in air and Pb in children's blood and between Pb in children's blood and IQ loss. The level of 0.15 $\mu g/m^3$ was estimated to protect against air Pb-related IQ loss in the most highly exposed children, those exposed at the level of the standard. Results of the quantitative risk assessment were judged supportive of the evidence-based framework estimates. The averaging time was revised to a rolling 3-month period with a maximum (not-to-be-exceeded) form, evaluated over a 3-year period.[1] 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 standards.[2]

As part of the previous Pb NAAQS review, EPA completed a quantitative assessment which estimated air-related Pb exposure and the associated risk of IQ loss in children for a series of study areas. Risks were estimated for then-current air quality conditions and for air quality conditions associated with just meeting the then-current standard and a series of alternative (lower) standards. Risk estimates generated as part of that analysis informed the EPA's decision to lower the level of the standard in the last review.

[1] As compared to the previous averaging time of calendar quarter, this revision was considered to be more scientifically appropriate and more health protective. 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. 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.

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

To estimate the potential ecological risks associated with exposures to Pb emitted into ambient air in the last review, a screening level risk assessment was performed. Hazard quotients were developed for three case studies for which media concentrations had also been estimated for the health risk assessment as well as for a national scale surface water and sediment assessment. While these analyses indicated the potential for air-related Pb to pose ecological risks of concern, limitations in available information and tools precluded our ability to parse out estimates of air-related ecological risk associated with the then-current or alternative Pb NAAQS.

This document (titled *Risk and Exposure Assessment Planning Document* – hereafter referred to as *REA Planning Document*) presents a critical evaluation of information related to Pb human and ecological exposure and risk (e.g., data, modeling approaches) newly available in this review as identified in the first draft *Integrated Science Assessment for Lead* (draft ISA; USEPA, 2011a). The focus of this evaluation is consideration of the extent to which new or substantially revised REAs for health and ecological risk are warranted by the newly available evidence.

This document is intended to facilitate consultation with the Clean Air Scientific Advisory Committee (CASAC), as well as public review, for the purpose of obtaining advice on EPA's consideration of the recently available evidence (information, methods, etc) with regard to its potential impact on quantitative exposure and risk analyses, both with regard to consideration of the extent to which new assessments are warranted in this review and with regard to our consideration of the last assessment in evaluating risk and exposure-related considerations in our Policy Assessment for this review. The discussion in this document is intended to build upon the exposure and risk assessment approaches employed for the last review, and on Agency experience with Pb exposure and risk assessment since that time, while also drawing from information presented in the May 2011 draft of the ISA for the current review.

Background

Sections 108 and 109 of the Clean Air Act (Act) govern the establishment and periodic review of the NAAQS. These standards are established for pollutants that may reasonably be anticipated to endanger public health and welfare, and whose presence in the ambient air results from numerous or diverse mobile or stationary sources. The NAAQS are to be based on air quality criteria, which are to accurately reflect the latest scientific knowledge useful in indicating the kind and extent of identifiable effects on public health or welfare which may be expected from the presence of the pollutant in ambient air. The EPA Administrator is to promulgate and periodically review, at five-year intervals, "primary" (health-based) and "secondary" (welfare-based)[1] NAAQS for such pollutants.[2] Based on periodic reviews of the air quality criteria and standards, the Administrator is to make revisions in the criteria and standards, and promulgate any new standards, as may be appropriate. The Act also requires that an independent scientific review committee advise the Administrator as part of this NAAQS review process, a function now performed by the CASAC.

EPA's overall draft plan and schedule for this Pb NAAQS review is presented in the draft *Integrated Review Plan for the National Ambient Air Quality Standards for Lead*.[3] That plan discusses the preparation of key documents in the NAAQS review process including an Integrated Science Assessment (ISA) and a Policy Assessment. The ISA provides a critical assessment of the latest available scientific information upon which the NAAQS are to be based, and the Policy Assessment evaluates the policy implications of the information contained in the ISA and of any policy-relevant quantitative analyses, such as quantitative human and/or ecological risk and exposure assessments, that were performed for the review or for past reviews. Based on this evaluation, the Policy Assessment presents staff conclusions regarding standard-setting options for the Administrator to consider in reaching decisions on the NAAQS.[4]

[1] Welfare effects, as defined in section 302(h) of the Act 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.

[2]Section 109(b)(1) [42 U.S.C. 7409] of the Act 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." Section 109(b)(2) of the Act directs that a secondary standard is to Aspecify 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.

[3]*Integrated Review Plan for the National Ambient Air Quality Standards for Lead,* March, 2011, available at: http://www.epa.gov/ttn/naaqs/standards/pb/data/20110331pbirpdraftcasac.pdf.

[4]NAAQS decisions involve consideration of the four basic elements of a standard: indicator, averaging time, form, and level. The indicator defines the pollutant to be measured in the ambient air for the purpose of determining compliance with the standard. The averaging time defines the time period over which air quality measurements are to be obtained and averaged, considering evidence of effects associated with various time periods of exposure. 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.

2 HUMAN EXPOSURE AND HEALTH RISK ASSESSMENT

This evaluation of newly available information is structured around consideration of two key questions: (a) is there newly available information relevant to critical uncertainties or limitations associated with the human exposure and health risk assessments completed for the last review; and, (b) to what extent would an updated risk model generate risk estimates that are substantially different from estimates generated in the previous review (i.e., enhanced precision and/or confidence, coverage for additional sensitive subpopulations and/or health endpoints)?

In designing and implementing an assessment of exposure and risk associated with Pb in ambient air, we are faced with significant limitations and complexity that go far beyond the situation for similar assessments typically performed for other criteria pollutants. For example, unlike other criteria pollutants, risk associated with exposure to Pb originally released to ambient air is multi-pathway in nature (i.e., not only an inhalation hazard), with exposure occurring through a range of ingestion pathways associated with deposition of ambient air Pb onto surfaces in indoor and outdoor environments, as well as inhalation of ambient air outdoors and of ambient (outdoor) air that has infiltrated indoors. The ingestion pathways include incidental ingestion through hand-to-mouth contact as well as dietary and drinking water ingestion. In addition, given the persistent nature of Pb, exposure to ambient air Pb can result from Pb originally emitted into the environment recently or at some point in the past (e.g., Pb from current or historic sources). Furthermore, uses of Pb in house paint and solder used in water distribution systems can also contribute to Pb exposures.

The multiple and potentially intertwined exposure pathways add substantial complexity to designing and implementing risk and exposure assessments for Pb. For example, because of nonlinearity in the relationship between Pb exposure and the level of Pb in blood (i.e., blood lead, or PbB - the commonly used Pb dose metric) as well as nonlinearity that has been identified in the concentration-response relationship for IQ loss, it is necessary to consider total Pb exposure in modeling risk for a child scenario and not only exposure related to Pb released into ambient air. In addition, modeling some of the key pathways involved in Pb exposure (e.g., Pb loading of indoor residential dust by ambient air Pb that penetrates indoors and settles onto surface), can be complicated. These are some of the factors that make modeling exposure and risk to Pb released into ambient air a technically challenging task that often requires the use of simplifying assumptions and that is subject to a range of technical limitations and considerable uncertainty. This chapter first describes salient aspects and details of the previous assessment (section 2.1) and then considers information newly available in this review and the extent to

which it might appreciably impact a revised or updated assessment (section 2.2). Key observations and conclusions drawn from this evaluation are presented in section 2.3.

2.1 OVERVIEW OF THE PREVIOUS ASSESSMENT

This section provides an overview of the human exposure and health risk assessments completed as part of the previous Pb NAAQS review (USEPA 2007a,b). The risk assessment completed for the previous review focused on estimating IQ loss in children due to exposure to Pb over the first seven years of life from a variety of sources, including, most importantly for purposes of this review, Pb released into ambient air. The risk assessment focused on a set of case studies. Due to limited data and models for characterizing all of the various complexities associated with Pb exposures, our efforts to focus on and characterize risk associated with ambient air-related sources and exposures in the last assessment included a number of simplifying assumptions in a number of areas. Moreover, while Pb in diet and some sources of drinking water may derive at least in part from Pb emitted into ambient air, the contribution of these air-related exposure pathways was not explicitly separated from that of nonair pathways due to limitations in the tools and data (as summarized in section 2.1.4 below).

This section begins with an overview of the conceptual model for our assessment of air-related Pb exposure and risk in the last Pb NAAQS review (section 2.1.1), followed by a discussion of the study areas on which the last assessment focused (section 2.1.2). Section 2.1.3 describes the risk model used, including key modeling elements (e.g., monitor data, air quality models, indoor dust models, input datasets, concentration-response functions), and section 2.1.4 describes particular challenges associated with differentiating air-related exposure and risk from those associated with nonair pathways (section 2.1.4). Lastly in section 2.1.5, we describe key uncertainties and limitations associated with the risk model.

2.1.1 Conceptual Model for Risk Associated with Air-related Lead

This section describes the conceptual model developed for the last review for assessment of public health risks associated with Pb from ambient air. The model identifies sources, pathways, routes, exposed populations, and health endpoints, with specific attention to those that were explicitly addressed in the risk assessment completed for the previous review. This is summarized in Figure 2-1, with boxes outlined in bold indicating items included in the quantitative risk assessment and sources and pathways for which ambient air has played a role ("air-related") identified in bold text. In this conceptualization, these are exposures with the potential to be affected (over some time frame) by an adjustment to the Pb NAAQS. Those pathways not associated with Pb originally emitted to the ambient air are considered policy-relevant background since an adjustment to the Pb NAAQS is not likely to have an impact on these exposures; these pathways are not bolded in Figure 2-1.

Sources of Pb exposure include current and historical air emissions sources, as well as miscellaneous nonair sources (e.g., land disposal of wastes). Such sources can contribute to Pb in outdoor dust and soil, which may play a substantial role in human exposures, particularly for children. Additionally, Pb in house dust, which may be derived from Pb in outdoor dust and soil, as well as from ambient air Pb is another source of children's exposure.

In addition to airborne emissions (recent or those in the past), sources of Pb exposure also included old leaded paint, including Pb mobilized indoors during renovation/repair activities, Pb in drinking water and Pb in the diet (Figure 2-1).[3] Pb in diet and drinking water may have air pathway related contributions as well as contributions from background 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 these contributions in the risk assessment performed for the last review.[4] Other pathways shown in Figure 2-1 as air-related include inhalation of newly or previously emitted Pb, ingestion of outdoor soil/dust containing previously deposited Pb, and ingestion of indoor dust containing newly or previously emitted Pb.

Regarding exposed populations and health effects endpoints, the previous risk assessment focused on IQ loss in children up to age 7 years. This reflected consideration of the evidence which indicated that children received elevated Pb exposure due to hand-to-mouth activity (i.e., incidental soil and indoor dust ingestion) and that ambient air-related Pb has been shown to contribute to Pb in outdoor soil and indoor residential dust. In addition, selection of this population (or lifestage) reflected consideration of the evidence that the developing nervous system in children is among, if not, the most sensitive of the endpoints associated with Pb exposure. Further, the blood Pb model available for the review (summarized in section 2.1.3 below) focused on simulating PbB concentrations through the first 7 years of childhood.

In terms of internal disposition and the biometrics used to assess Pb exposure, PbB levels continue to be extensively used as an index or biomarker of exposure by national and international health agencies. This reflects the association of PbB with exposure, particularly recent exposure in young children, and the relative ease of collecting PbB measurement. 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 PbB as the exposure metric. Therefore, we focused on modeling PbB in young

[3] We did not explicitly consider Pb exposure related to consumer products (e.g., toys, cosmetics, dishes).

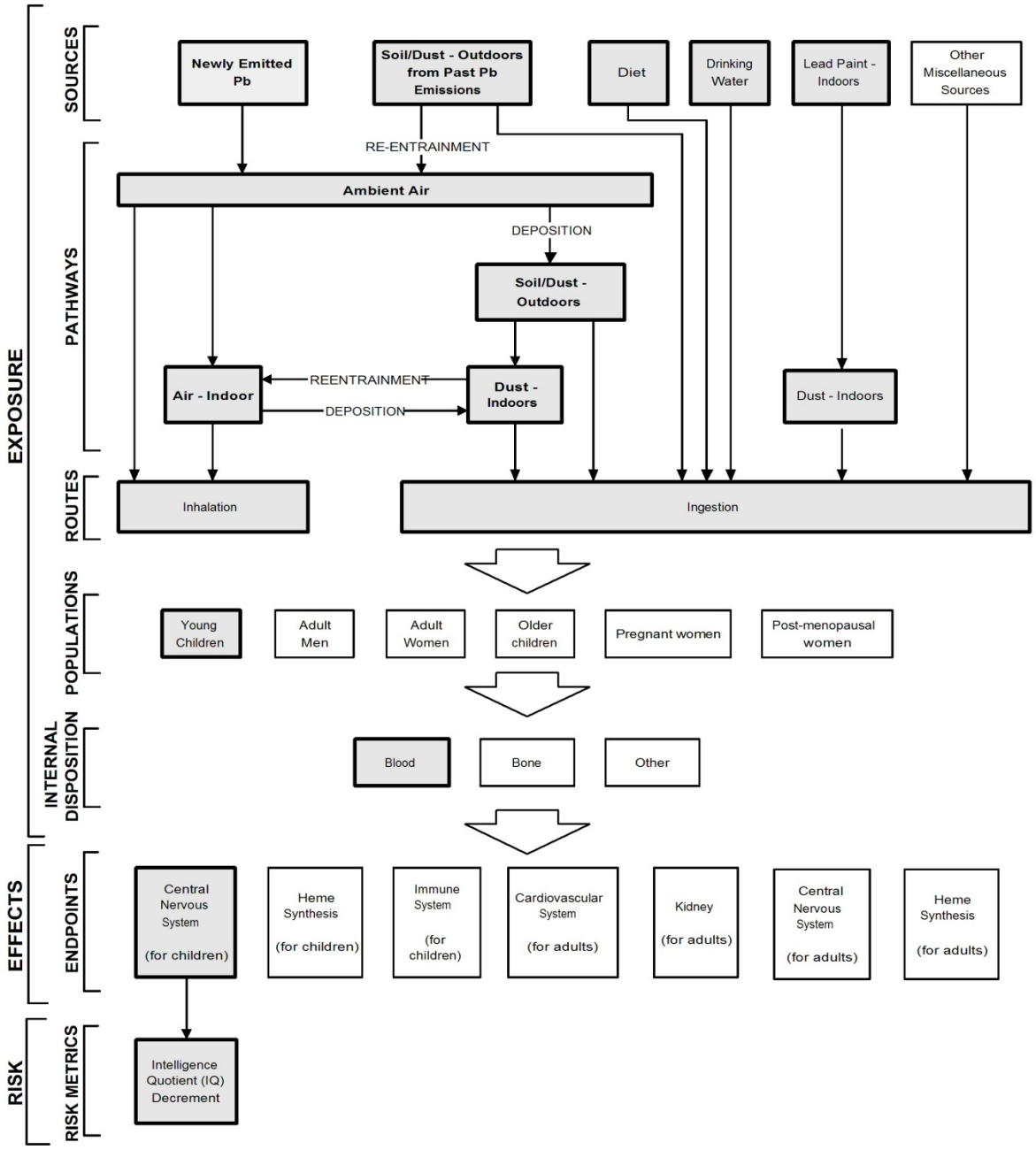
[4] The assessment grouped the exposure and risk estimates for Pb in diet and drinking water together in a "background" category which was combined with the other pathways in estimates presented for "total Pb exposure". Characterization of the risk assessment results in the rulemaking recognized the contribution, albeit unquantified, from air-related pathways within this category.

children, developing estimates for two PbB metrics: "concurrent" and "lifetime average". For the former we estimated PbB at age 7 years, while lifetime average was estimated as the average of PbB levels across the 7 year period.

At the time of the last review, we noted that limitations precluded prediction of changes in adult PbB 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 PbB 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 presented in the 2006 AQCD (USEPA, 2006a), the assessment focused on risk to the central nervous system in childhood as the most sensitive effect that could be quantitatively assessed, with decrement in Intelligence Quotient (IQ) used as the risk metric.

Figure 2-1. Conceptual Model for Previous Exposure and Risk Assessment



Note: Boxes outlined in bold and shaded are included in the quantitative risk assessment.

2.1.2 Case Studies and Air Quality Scenarios

The risk assessment estimated risk for five case studies[5,6] that generally represent two types of residential population exposures: (1) more highly air-pathway exposed young children (as described below) residing in small neighborhoods or localized residential areas with air concentrations somewhat near the standard being evaluated, and (2) location-specific urban populations with a broader range of air-related exposures.

The case studies representing the children most highly exposed via air-related pathways were the general urban case study and the primary Pb smelter case study. The general 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. Additionally, the method for simulating temporal variability in air Pb concentrations in this case study relied on national average estimates of the relationships between air concentrations in terms of the statistics considered for different forms of the standard being assessed and the annual ambient air concentrations required for input to the PbB model.[7] Thus, while this case study provided characterization of risk to children that are relatively more highly air pathway exposed (as compared to the location-specific case studies), it was not considered to represent a high-end scenario with regard to the characterization of ambient air Pb levels and associated risk. The primary Pb smelter case study provides risk estimates for children living in a specific area that is currently not in attainment with the current NAAQS. We focused particularly on a subarea within 1.5 km of the facility, where airborne Pb concentrations were closest to the current standard and where children's air-related exposures are most impacted by emissions associated with the Pb smelter from which air Pb concentrations were estimated.

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[5] A sixth case study (the secondary Pb smelter case study) is also described in the Risk Assessment Report. However, as discussed in Section 4.3.1 of that document (USEPA, 2007a), significant limitations associated with predicting ambient air Pb levels in the vicinity of the facility using dispersion modeling contributed to large uncertainties in the corresponding risk estimates.

[6] In addition to the six case studies included in the Risk Assessment Report, the pilot phase of the risk assessment from the last review also included a near-roadway case study (ICF International, 2006). Based on the pilot results and advice from CASAC, this case study was not carried into the full-scale analysis. Rather, we substituted the general urban case study for the near-roadway case study since the near-roadway case study focused on a small subset of the urban area (populations exposed immediately near roadways), while for purposes of the risk assessment, we wanted a case study that provided broader coverage for residents potentially exposed at the standard level being assessed, which was better provided by the general urban case study (USEPA, 2007a).

[7] As the PbB model used in the risk assessment was limited in that it did not accept inputs of a temporal time step shorter than annual average, ratios of relationships in the available air monitoring data between different statistical forms being considered for the standard and an annual average were employed for the urban case studies (that did not rely on dispersion modeling) as a method of simulating the temporal variability in air Pb concentrations that occurs as a result of meteorology, source and emissions characteristics.

The three location-specific urban case studies focused on specific residential areas within Cleveland, Chicago, and Los Angeles to provide representations of urban populations with a broader 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.

Based on the nature of the population exposures represented by the two categories of case study, the first category (the general urban and primary Pb smelter case studies) includes populations that are relatively more highly exposed by way of air pathways to air Pb concentrations somewhat near the standard level evaluated, compared with the populations in the three cities.

The air concentrations in the different air quality scenarios included those representing current conditions for the different case studies, conditions meeting the then-current (at the time of the last review) NAAQS of 1.5 $\mu g/m^3$, maximum calendar quarter average, and conditions meeting several alternate, lower standards. The set of air quality scenarios assessed in the risk assessment is listed in Table 2-1.

Table 2-1 Air Quality Scenarios Assessed in the Risk Assessment for Previous Review.

Air Quality Scenario	Averaging Time (Form)	Level ($\mu g/m^3$)	Case Study
Then-current Standard	Calendar Quarter (maximum)	1.5	All
Current Conditions		0.87 (95^{th} percentile) 0.14 (mean)	General Urban
		0.14	Chicago
		0.36	Cleveland
		0.09	Los Angeles
Alternate Standard		0.2	General Urban Primary Smelter
Alternate Standards	Month (maximum)	0.5	General Urban Primary Smelter Cleveland
		0.2	All except Los Angeles
		0.05	All
		0.02	All

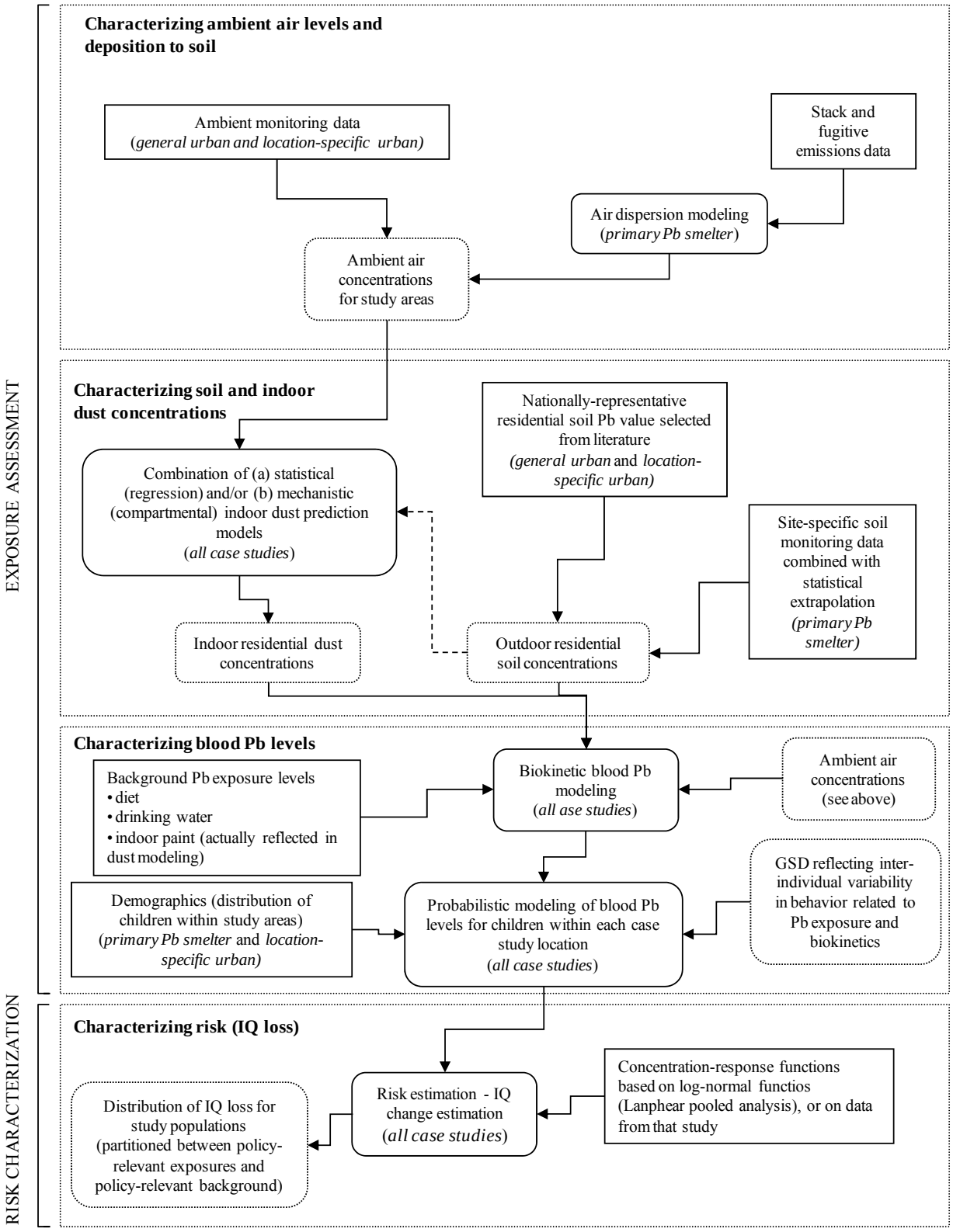
The concentrations of Pb used for these scenarios for other media and exposure pathways varied depending on the type of case study (as summarized below in section 2.1.3) and, in some

cases (as summarized below in section 2.1.4), limitations in the available data and modeling tools precluded the assessment from varying concentrations of some nonair media across the air quality scenarios. For example, dietary and drinking water Pb concentrations, as well as soil Pb concentrations, were not varied in any case studies across the air quality scenarios. The modeling simulations for all cases studies did, however, include changes in indoor dust Pb concentrations, for different air quality scenarios, although as summarized in section 2.1.3, the methods used differed among the different types of case studies.

2.1.3 Analysis Approach and Modeling Elements

This section describes the risk assessment approach (including discussion of key analysis steps), used in assessing risk for the two categories of case studies. As illustrated in Figure 2-2, the risk assessment completed for the primary Pb smelter case study included four analytical steps: (a) fate and transport of Pb released into outdoor air, including the dispersion of Pb away from the point of release, (b) prediction of the resulting concentration of Pb in media of concern including outdoor air and indoor dust, (c) use of these Pb concentrations together with estimates of Pb in background exposure pathways, including diet, to estimate associated PbB levels in children using biokinetic modeling, and (d) use of concentration-response functions derived from epidemiology studies to estimate IQ loss associated with the estimated PbB levels. The modeling approach for the general urban case study and location-specific urban case studies is somewhat simpler, since it does not involve fate and transport modeling for air concentration estimates and, instead, uses ambient monitor levels to characterize air Pb levels across the study area. Subsequent steps in the general urban case study analysis are fairly similar to what is described above for the point source case study, with the generation of population PbB levels being somewhat simplified for the general urban case study. Figure 2-2 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, while the fourth is the risk assessment step (exposure assessment and risk assessment steps are discussed separately below).

Figure 2-2. Overview of Analysis Approach for Exposure and Risk Assessment.



Exposure Assessment

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 background 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). Both of these uncertainties are discussed below in Section 2.1.5. Specific approaches used at the three case study locations are briefly described below.

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 general 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 in exposure for three urban areas in the U.S. By contrast, the general urban study area 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 (see Section 5.2.2 of US EPA, 2007a for additional detail).

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 the available empirical data was used to predict soil levels for portions of the study area beyond this zone.

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, a combination of regression-based models obtained from the literature and developed based on site-specific data were used, and a customized hybrid empirical-mechanistic model was developed 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 general urban case study. Additional detail on methods used to characterize media Pb concentrations for each case study can be found in Sections 3.1 and 5.2.3 of US EPA, 2007a). Blood Pb levels were predicted from estimates of Pb contained in various media (e.g., ambient air, diet, water, indoor dust) using the Integrated Exposure and Uptake Biokinetic (IEUBK) model (Sections 3.2.1.1 and 5.2.4 of US EPA, 2007a).[8] However, rather than completing a fully-probabilistic simulation of PbB levels for a set of simulated children (where we would first simulate variability in Pb intake and then estimate PbB levels for each child separately in IEUBK), we used IEUBK to generate a central-tendency estimate of PbB levels for a group of children within a given study area. We then combined this central-tendency estimate with a geometric standard deviation (GSD) reflecting variability in PbB levels for groups of children to generate a distribution of PbB levels for a study area.[9] Note, that for all of the study areas, we assumed that pathway apportionment of PbB levels based on the modeling of the central-tendency PbB level (using IEUBK) holds for all percentiles of PbB levels derived by combining that central-tendency with the GSD. PbB modeling completed for all case studies included estimates of both concurrent and lifetime-average PbB metrics, although ultimately, we focused on the concurrent PbB metric in estimating risk.[10]

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

[9] The procedure for combining the IEUBK-based central tendency blood Pb estimate with the GSD to generate a population distribution of PbB 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 central-tendency estimate of PbB levels and this is in turn, combined with the GSD to produce an population-distribution of PbB levels. However, for both the primary Pb smelter and the location-specific urban study areas, smaller polygons within the larger study area (e.g., US Census blocks for the location specific urban study areas) are used as the basis for generating distributions of PbB levels and these are then population weighted prior to aggregation to form an overall PbB distribution for each study area (see sections 3.2.2 and 5.2.2.3 of US EPA, 2007a).

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

Risk Characterization

The risk characterization step involves generating a distribution of IQ loss estimates for the set of children simulated in the exposure assessment. Specifically, estimated PbB levels (for the concurrent PbB metric)[11] were combined with four PbB concentration-response functions for IQ loss (see Section 5.3.1.1 of US EPA 2007a). Four different concentration-response functions were selected to provide different characterizations of behavior at low exposures. The decision to use four different functions is in recognition of uncertainty related to modeling this endpoint, particularly at lower PbB levels for which there is limited representation in the Lanphear et al (2005) pooled dataset. For example, the 5th percentile for the concurrent PbB measurements in that dataset was 2.5 µg/dL (73 FR 66978). The four different functions are either based directly on the lognormal concentration-response function described in the Lanphear et al, (2005) pooled analysis of epidemiology studies focusing on IQ loss in children, or they are derived from data presented in that study. The four functions are presented in Figure 2-3 and compared in Table 2-2 with regard to total IQ loss and incremental IQ loss (IQ loss per µg/dL) across a range of concurrent PbB levels. A brief description of each of the functions is also provided below:

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

[11] 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 were given primary emphasis, however, due to the slightly more significant association found for concurrent PbB with IQ by Lanphear et al (2005) in addition to advice from CASAC.

Figure 2-3. Comparison of Four Concentration-response Functions Used in the Previous Risk Assessment.

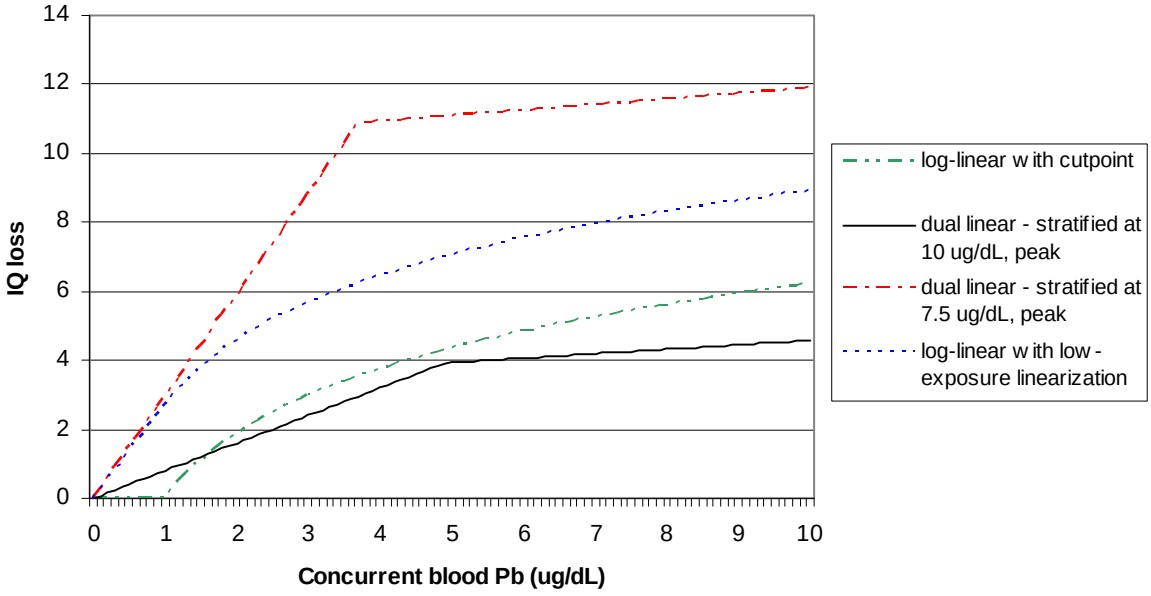


Table 2-2. Comparison of Total and Incremental IQ Loss Estimates Below 10 µg/dL for the Four Concentration-response Functions.

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

Two categories of risk metrics were generated for each of the 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).

- *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 general urban case study, only the first type of risk metric, population risk percentiles, was developed because this case study is not location-specific. Child frequency counts are not applicable, since a specific location with associated demographic data was not modeled.

2.1.4 Challenges in Characterizing Air-related Exposure and Risk

In the risk assessment, we attempted to separate estimates of total (all-pathway) PbB and IQ loss into a policy-relevant background category and two air-related (policy relevant) categories, referred to as "recent air" and "past air". However, significant limitations in our modeling tools and data resulted in an inability to parse specific risk estimates into specific pathways, such that we had approximated estimates for the air-related and background categories.

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.

Among the limitations affecting our estimates for the air-related and background categories is the apportionment of background (nonair) pathways. For example, while conceptually indoor Pb paint contributions to indoor dust Pb would be considered background and included in the "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, conceptually, exposure to indoor paint Pb is considered part of background exposure. At the same time, Pb in ambient air does contribute to the exposure pathways included in the "background" category (drinking water and diet), and is likely a substantial contribution to diet. We could not separate the air contribution from the nonair contributions, and the total

contribution from both the drinking water and diet pathways is categorized as “background” in this assessment. As a result, our “background” risk estimate 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 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.

In summary, because of limitations in the assessment design, data and modeling tools, our risk estimates for the “past air” category include both risks that are truly air-related and potentially, some background risk. Because we could not sharply separate Pb linked to ambient air from Pb that is background, 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.

2.1.5 Key Uncertainties and Limitations

Although the risk assessment completed for the last review utilized a number of innovative modeling elements in order to generate representative estimates of risk for our study populations, like all risk models there was uncertainty associated with the model and its output. For example, because of the evidence for a nonlinear response of PbB to exposure and also the nonlinearity reflected in the C–R functions for estimation of IQ loss, the assessment first estimated total PbB levels and associated risk (i.e., for air- and nonair-related exposure pathways), and then separated out those estimates of PbB and associated risk for pathways of interest in this review. We separated out the estimates of total (all-pathway) PbB and IQ loss into a background category and two air-related categories (“past” and “recent”). However, significant limitations in our modeling tools and data resulted in an inability to parse specific risk estimates into specific pathways, such that we approximated estimates for the air-related and

background categories. As discussed 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 past-air and recent-air categories.

Additional limitations, assumptions and uncertainties, which were recognized in various ways in the assessment and presentation of results, are listed below, beginning with those related to design of the assessment or 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 remains at the same residence (while exposure factors and physiological parameters are adjusted to match the age of the child).
- *General Urban Case Study:* The design for this case study employs assumptions regarding uniformity that are reasonable in the context 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 general urban 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.
- *Location-specific Urban Case Studies:* Limitations in the ambient air monitoring network limit our characterization of spatial gradients of ambient air Pb levels in these case studies.
- *Air Quality Simulation:* The proportional roll-up and roll-down procedures used in some case studies to simulate the then-current NAAQS and alternate NAAQS levels, respectively, assume proportional changes in air concentrations across the study area in those scenarios for those case studies. EPA recognizes the uncertainty with our simulation of higher air Pb concentrations that just meet the then-current 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.
- *Outdoor Soil/Dust Pb Concentrations:* Uncertainty regarding soil/dust Pb levels and the inability to simulate the influence of changing air Pb levels related to lowering the NAAQS contributes uncertainty to air-related risk estimates.
- *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.
- *Interindividual Variability in PbB Levels:* Uncertainty related to population variability in PbB levels and limitations in modeling of this introduces significant uncertainty into PbB and IQ loss estimates for the 95th percentile of the population.

- *Pathway Apportionment for Higher Percentile PbB and IQ Loss:* Limitations primarily in data prevented us from characterizing the degree of correlation between high-end Pb exposures across pathways (e.g., the degree to which an individual experiencing high drinking water Pb exposure would also potentially 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 PbB level and IQ loss is subject to greater uncertainty at lower PbB levels (e.g., particularly below 2.5 µg/dL concurrent PbB).

2.2 CONSIDERATION OF NEWLY AVAILABLE EVIDENCE

This section evaluates the information, methods and models newly available since the last review (as summarized in the first draft Pb ISA) to assess the extent to which it has the potential to address these limitations and uncertainties. Table 2-3 (a) identifies limitations and sources of uncertainty in the risk assessment model developed in the last Pb NAAQS review, (b) identifies the evidence newly available since the last review that may address these uncertainties or limitations (in those instances where new information is available) and (c) assesses the degree to which the information (e.g., technical insights, data, modeling approaches etc.) resulting from that research may address the source of uncertainty or limitation. Based on the information provided in Table 2-3, EPA staff conclusions regarding the potential role of the risk assessment model and associated risk estimates generated in the last review in the current Pb NAAQS review are presented below in section 2.3.

Table 2-3. Assessment of Information (including methods, models, etc.) Newly Available in this Review.

Limitation/Uncertainty in Risk Model	Information Newly Available in this Review	Consideration of Potential Utility and Impact on Quantitative Exposure/Risk Assessment
Characterizing Exposure Pathways		
A) Characterizing spatial gradients in ambient air Pb levels in the urban context. Limitations in our monitoring network and in studies of smaller scale spatial gradients in ambient Pb levels in the urban setting introduced uncertainty into our characterization of exposure levels and risk for residential populations modeled for the location-specific urban study areas. Similarly, limitations in our characterization of ambient air Pb levels prevented us from characterizing areas across the U.S. with the attributes associated with the general urban study area.	Section 3.5.1.2 (Intra-urban Variability) of the draft ISA observes that evidence from a number of studies suggests that there is substantial intra-urban variability in ambient air Pb levels with elevated levels being associated with proximity to specific sources (and often captured by source-oriented monitors), while lower levels are often associated with more generalized urban areas. However, the monitoring network in place is generally not refined enough to provide comprehensive coverage for an urban area such that the nature of the spatial gradient can be well-characterized on a more localized (neighborhood) level. The draft ISA also highlights the fact that monitors from different networks can capture different size gradients for particles containing Pb, which can make it hard to compare monitored levels across monitors (i.e., if they are from different networks).	In the last risk assessment, we used monitoring data to try and identify the fraction of monitor locations with ambient air Pb levels near the current standard level (or alternative lower standard levels) which, therefore, could have potential risks at or near those estimated for the general urban study area. However, without having more information on the gradient in air Pb levels near those higher source-oriented monitors, any assessment of the size of populations experiencing those elevated air Pb levels is highly uncertain. The newly available information does not appear to provide substantially improved data for characterizing gradients at this more refined spatial scale. We also note, that even if we did have highly-refined information on ambient air Pb gradients in urban areas, in order to complete more detailed residential Pb-risk modeling, we would also need more detailed information on other aspects of Pb exposure specific to those residential areas (e.g., indoor dust Pb concentrations, soil concentrations, and drinking water Pb concentrations). Availability of this level of refined exposure information for the urban areas that might be of interest is likely limited or lacking. In addition, we would still be hampered by limitations in our ability to apportion total risk between air and nonair pathways especially for individuals exposed to higher pathway-specific Pb levels, as would be associated with this type of specific residential location (i.e., we do not have information on the degree of correlation among Pb exposure pathways).

Limitation/Uncertainty in Risk Model	Information Newly Available in this Review	Consideration of Potential Utility and Impact on Quantitative Exposure/Risk Assessment
B) Limitations in our ability to simulate alternative (lower) standard levels (implementing rollback). Significant uncertainty is associated with the prediction of reductions in ambient air Pb levels, reflecting potential actions by urban areas to attain alternative (lower) standards.	Section 2.1.1 (Sources, Fate and Transport of Ambient Lead) and section 3.3 (Fate and Transport of Lead) of the draft ISA note that Pb associated with coarse PM will tend to deposit near sources, while Pb associated with finer PM will tend to be transported further. The draft ISA also notes that the cycle of deposition and resuspension for even coarse-phase Pb can be substantial leading to diffusion in the urban context. This more generalized information on the relationship between Pb particle size and deposition helps us to better understand and frame conceptually the issue of spatial gradients in reduction around sources. However, there does not appear to be new research or information on how ambient air Pb levels might decrease given reductions in Pb emissions from specific sources, or categories of sources.	It does not appear that we have substantially improved information to support design of our rollback strategies for specific case studies. However, it is also important to note that the general urban study area does not require this type of representative characterization of simulated reduction in ambient air Pb levels, since that case study assumes that ambient air Pb levels are at the standard level being assessed (i.e., it provides an estimate of risk for children living in an area at or near the standard level). While it is difficult to characterize areas across the U.S. that reflect key aspects of the general urban study area (e.g., to estimates how many children it may represent), this case study provides an important assessment of risk for children living in areas that just meet the standard assessed (i.e., a reasonable higher-end risk associated with a particular standard).
C) Characterizing Pb levels in other exposure media, including soil, drinking water and dietary items (indoor dust is discussed separately below – see entry "D"). Limitations in our ability to characterize Pb levels in other media (besides ambient air) and differentiate those levels for different subsets of urban populations in the location-specific urban study areas evaluated, introduces uncertainty into the analysis.	Section 3.6.1 (Soils) of the draft ISA provides a table (3-9) identifying soil Pb levels in urban areas from the literature, including a number of values from studies published since the previous review yet conducted across a broad range of years back to 1976. Therefore, depending on the availability of information to clarify the context for these measurements particularly with regard to air-related and other sources, these may include new data to inform the characterization of urban soil Pb levels for the risk assessment. Section 4.1.1.3 of the draft ISA describes	It is important to note that while improved soil Pb data (and data for Pb in other exposure media including drinking water and dietary items) would provide us with refined central-tendency estimates of Pb exposure and risk for the study areas, the key factor that would need to be addressed to substantially improve our estimates of exposure and risk is the degree of correlation between Pb exposure pathways, particularly for individuals experiencing elevated exposure (e.g., to what extent is higher drinking water Pb exposure correlated with higher soil Pb exposure and with areas of elevated ambient air Pb exposure). This type of correlational information for more highly Pb exposed

Limitation/Uncertainty in Risk Model	Information Newly Available in this Review	Consideration of Potential Utility and Impact on Quantitative Exposure/Risk Assessment
	information on Pb in drinking water (including studies on the role of different drinking water systems in releasing Pb from pipes) and Pb in dietary items (Pb in crops, fish and game).	children will only be available if we have matched sets of measured Pb levels in exposure media for a moderate to larger set of children (as noted below, ideally, this would include PbB measurements so we can look in detail at the air-blood ratios). In the absence of this improved information on the nature of correlations between Pb exposure pathways, we are not in a position to substantially improve our estimates of exposure and risk (including the ambient air-related fraction of risk), particularly for higher PbB children, including those with elevated ambient air-sourced Pb exposures.
D) Modeling the relationship between ambient air Pb (outdoors) and indoor dust Pb (focusing on uncertainties related to application of the hybrid indoor dust model). Elements of the hybrid (mechanistic-empirical) indoor dust model are subject to uncertainty including: (a) the simulation of the interplay between air exchange rates (ambient outdoor and indoor), loading to indoor surfaces from indoor air and removal through cleaning, (b) estimating the fraction of indoor dust Pb that originates from old paint versus the fraction originating from ambient outdoor air Pb (combines mechanistic modeling for ambient air-related indoor dust with empirical data on total dust Pb, including the fraction from indoor paint), and (c) converting estimates of indoor dust Pb loading to	Section 4.1.1.2. (Exposure to Lead in Soil and Dust) of the draft ISA discusses research published since the last review focused on assessing risk reductions associated with vacuuming of surfaces with Pb dust (Hunt et al., 2008). This study suggests that, while vacuuming may clean much of the soil mass deposited onto hard surface flooring, Pb dust in a relatively fine form (1-3 μm particles, which may be more accessible through incidental hand-to-mouth activity by children) remains. While this study provides information on cleaning efficiency, it is important to note that the soil Pb used in the analysis was sourced from the Herculaneum location and the study focused exclusively on hard-surface flooring, providing no new information regarding cleaning efficiencies for indoor environments with additional types of surfaces (e.g., fabrics).	While the Hunt et al. (2008) study provides some information on cleaning efficiency and highlights the potential for vacuuming to leave fine-particle Pb on floors, the utility of the study in providing inputs to the hybrid dust model (e.g. additional data on cleaning efficiency) is limited by the fact that the study used soil Pb samples from Herculaneum and the fact that it focused on hard floors (and did not evaluate carpeted flooring or any other textiles common in indoor environments). Given the site-specific nature of this material (i.e., associated with primary Pb smelting activity that includes a significant historical signal combined with repeated remediation activity), data originating from the study, while potentially useful in guiding remediation efforts and house cleaning strategies for residences near Herculaneum, cannot be readily extrapolated to other urban scenarios involving Pb exposure. Note, however, that information obtained from the study may be used qualitatively to further help interpret risk estimates generated in the previous review. Specifically, the Hunt et al., 2008 study

Limitation/Uncertainty in Risk Model	Information Newly Available in this Review	Consideration of Potential Utility and Impact on Quantitative Exposure/Risk Assessment
dust Pb concentrations, which are needed for PbB simulation in IEUBK.		identifies the potential for low-bias in our risk estimates, if dust containing Pb that is not cleaned is ultimately in a finer form that is more accessible to incidental ingestion by children. Conversely, the Hunt study also identifies the potential for high-bias in residences that lack carpets and other fabrics, if their relative cleaning efficiency rates hold in a more generalized urban setting (their rates are generally higher than ours). Note, however that the Hunt et al., (2008) study exclusively focused on hard floor surfaces and not carpeting, and we would expect carpeting to have substantially lower cleaning efficiency.
E) Modeling the relationship between ambient air Pb (outdoors) and indoor dust Pb and then relating these to PbB levels (focusing here more broadly on the relationship between ambient air Pb and PbB, with indoor dust Pb as the primary linkage). There is uncertainty in relating changes in ambient air Pb to changes in indoor dust Pb and PbB. Any data characterizing this relationship between ambient air Pb and PbB (including matched data sets for a group of children) could be used in performance assessing and possibly calibrating, the exposure component of the risk assessment.	Section 4.5.2. (Environmental Lead-Blood Lead Relationships) discusses studies based on the CDC's National Health and Nutrition Examination Survey (NHANES) data from 1999-2004. These studies assess the relationship between residential dust Pb and PbB levels in children (e.g., Gaitens et al., 2009 and Dixon et al., 2009). However, these studies do not account for ambient air Pb (and its potential relationship with indoor dust Pb and consequently, total Pb exposure).	While NHANES-based studies and other studies of specific locations provide insights into which factors drive indoor dust Pb levels and PbB levels, an important limitation of these studies is that they do include measurements of ambient air Pb. Because these studies do not provide explicit coverage for urban areas with elevated ambient air Pb levels (where this Pb source can be of greater importance), their utility in performance assessing elements of the risk assessment is limited. Had these studies included consideration for ambient air Pb (including areas with elevated air Pb levels), then the utility of the studies in the context of the risk assessment would be greater. Note, that as part of the risk assessment completed for the last review, we considered studies based on the EPA-sponsored National Human Exposure Assessment Survey (NHEXAS) in performance evaluating simulation of indoor dust Pb levels (see Appendix G, Exhibit G-6 of the Pb HEHR, USEPA, 2007a).

Limitation/Uncertainty in Risk Model	Information Newly Available in this Review	Consideration of Potential Utility and Impact on Quantitative Exposure/Risk Assessment
Estimating Exposure and PbB for Young Children		
F) Uncertainty in modeling multiple-pathway exposure to Pb in young children, with consideration for the degree of correlation between pathways. If we had detailed survey data on pathway exposures for a set of children (including ambient air Pb, indoor dust, soil Pb and dietary Pb levels for each child), we could then use those data to enhance our simulation of total Pb exposure for the urban case studies. This kind of matched pathway-specific data for a set of children would allow us, in particular, to define potential correlations between Pb exposure pathways (e.g., the extent to which children with high drinking water Pb are also exposed to high indoor dust Pb and ambient air Pb). This, in turn, would potentially improve our ability to simulate risk for children with elevated Pb exposures and parse out the fraction associated with ambient air Pb.	Section 4.1.1 (Pathways for Lead Exposure) in the draft ISA discusses research based on the EPA-sponsored National Human Exposure Assessment Survey (NHEXAS) dataset. For example, Egeghy et al., 2005, used a dataset for Maryland location to examine determinants of variability in PbB levels (including ambient air Pb, dust Pb and dietary Pb levels). While this analysis did use a matched dataset for a group of 80 individuals, an important attribute of the study is that all individuals are >6 yrs of age (i.e., ranging older than the age group modeled in our risk assessment). Additionally, the air Pb measurements were restricted to particles smaller than 10 microns potentially affecting conclusions regarding pathways involving deposition of larger particles. The short sampling period (8 days) also may preclude a representative sample.	While the NHEXAS-Maryland dataset used in the Egeghy et al., (2005) study includes data for a variety of exposure pathways (including ambient air Pb, dietary pathways, soil and drinking water), because the analysis includes predominantly individuals outside of our child age range of interest, it is of reduced utility in informing our consideration of pathways of exposure and potential correlation between those pathways. In addition, the relatively small sample size (80 individuals), could potentially limit any more detailed assessment of the degree of correlation between pathways for different percentiles of the study population with regard to PbB level. It is interesting to note, that in the Egeghy et al., 2005 study, outdoor air Pb, while statistically associated with indoor air Pb, was not associated directly with indoor dust Pb levels, although this may reflect the fact that their dataset reflected residential areas with relatively low ambient air Pb, such that other factors (e.g., home age, condition of indoor paint and construction material) drove loadings of indoor dust Pb.
G) Modeling PbB levels in young children given pathway-specific intake estimates (use of IEUBK). There is uncertainty both in the	Section 4.6 (Biokinetic Models of Lead Exposure-Blood Lead Relationships) of the draft ISA observes, that while modeling of human Pb exposures and biokinetics has	It does not appear that there have been significant developments in the modeling of PbB levels in children since the last review. While there are data that could be used to update the GSDs used in the risk assessment to

Limitation/Uncertainty in Risk Model	Information Newly Available in this Review	Consideration of Potential Utility and Impact on Quantitative Exposure/Risk Assessment
prediction of central-tendency PbB levels as well as the application of geometric standard deviations (GSDs) for characterizing population variability in PbB levels around those central-tendency estimates. As noted in the risk assessment, we did not complete a probabilistic simulation of PbB variability based on modeling PbB levels for each simulated individual using IEUBK and, instead, relied on GSDs to characterize variability around the IEUBK-modeled central tendency PbB level for a particular study population.	advanced considerably during the past several decades, there have been relatively few developments since the 2006 Pb AQCD (USEPA, 2006a) was published in the last review (draft ISA, p. 4-93). Regarding newer data characterizing population variability in PbB levels (i.e., sources for updating the GSDs used to characterize population variability in PbB levels in the risk assessment), there are updated characterizations of PbB variability in children for the U.S. based on the 2005-2008 NHANES data (see section 4.4.1 - Lead in Blood – in the draft ISA). In addition, recent studies of Pb effects in children could provide characterization of variability in PbB levels for smaller (more localized) groups of children (for example, see section 5.3.3.1. Epidemiologic Studies of Behavioral Effects in Children - in the draft ISA).	characterize population variability in PbB levels (for children), without improved characterization of the potential correlations between pathways, updated estimates of higher percentile PbB levels (and IQ loss), will still be subject to substantial uncertainty due to an inability to reliably apportion total risk between the contributing pathways (at those higher-end total Pb exposure levels).
Estimating Exposure for Other Populations/Lifestages		
H) Estimating exposure for other populations/lifestages. Limitations in our ability to relate changes in recent air Pb-related exposure to PbB levels in adults was one of the reasons that adult health endpoints were not part of the assessment for the last review. A	As noted above, section 4.6 (Biokinetic Models of Lead Exposure-Blood Lead Relationships) of the draft ISA does not identify substantial improvements in PbB prediction models, including models for simulating changes in PbB (or other biometrics) for adults and for children older	It does not appear that there have been significant improvements in our ability to model exposure (including PbB levels) for adult or older child populations. Furthermore, predicting changes in PbB levels for older children and adults resulting from recent changes in ambient air Pb-related exposures is complicated significantly by the presence of

Limitation/Uncertainty in Risk Model	Information Newly Available in this Review	Consideration of Potential Utility and Impact on Quantitative Exposure/Risk Assessment
critical source of uncertainty in modeling adult PbB is the complex role played by the storage of Pb in various compartments (mainly in bone) over a person's lifetime, as well as the mobilization of this Pb during certain physiological conditions. This accumulated Pb in adults derives from earlier exposures when both Pb intake and environmental concentrations, may have been substantially higher. The models available for children older than 7 yrs (the age limit of IEUBK) and into adulthood (e.g., Leggett, 1993) have not been routinely used by the EPA in supporting regulatory risk assessment or subjected to the extensive and rigorous performance evaluation which has been so thoroughly reported for IEUBK (AQCD, section 4.4.10). In addition, some of the same uncertainties identified above for adults also pertain to some extent in modeling older children/teenagers.	than 7 years (the IEUBK limit), including teenagers.[12] Regarding the potential for empirical air-to-PbB models, section 4.5.1.2 of the Pb ISA, which addresses the availability of recent studies in this area, describes an occupational exposure study. The study size was relatively small with occupational exposure concentrations well above those common in ambient air (the geometric mean Pb concentration over the two week period was 58 μg/m^3).	accumulated Pb in bone which can contribute to PbB levels and can itself reflect Pb exposure earlier in life.
Estimating Risk for Young Children		
I) Modeling IQ loss in young children. Uncertainty associated with	The ISA (Section 2.8.2) summarizes the current evidence that informs characterization	The varied studies on associations between IQ and PbB now available do not provide a strong foundation for

[12] As noted in the draft ISA, EPA's All Ages Lead Model is still in development,

Limitation/Uncertainty in Risk Model	Information Newly Available in this Review	Consideration of Potential Utility and Impact on Quantitative Exposure/Risk Assessment
the magnitude of IQ loss predicted for simulated children, particularly at lower total PbB levels (e.g., < 2.5 µg/dL) reflects the appreciably less extensive information available on which to base our characterization of the concentration-response function at lower PbB levels levels.	of concentration-response relationships for those health endpoints for which such evidence is most well established. These relationships are for PbB levels in children with cognitive deficits and PbB levels in adults with increased blood pressure, mortality, and indicators of nephrotoxicity. With regard to studies published since the last review that might inform our understanding of the concentration-response relationship for Pb associated IQ loss, three studies (Chiodo et al., 2007; Jusko et al., 2008; Kim et al., 2009) report effect estimates for IQ loss in study populations with mean PbB levels at or below 5 µg/dL.[13] However, there is limited representation of PbB levels below 3 µg/dL in Chiodo et al. (2007) and Jusko et al. (2008). In comparison to these two studies, Kim et al. (2008), which involved South Korean children aged 8-11 years of age, includes a lower distribution of PbB levels.	development of a new or adjusted concentration-response function. As noted in section 2.1.3 above, the risk assessment for the last review included four different quantitative functions (based on or extending from the nonlinear function from Lanphear et al 2005) for the concentration-response relationship in the lowest PbB level region (see table 2-2 and figure 2-3). The new studies do not provide support for development of a quantitative function for this PbB level region that falls outside of the range of C-R functions represented by these approaches. Of the three studies (Chiodo et al., 2007; Jusko et al., 2008; Kim et al., 2009) identified since the last review, only Kim et al. (2009) provides information on the blood-Pb IQ concentration-response relationship for PbB levels below 3 µg/dL. Further, Kim et al. (2009) utilizes a Korean population and an age group older than the group modeled in the last risk assessment. With this older population, there is greater uncertainty regarding the exposure conditions associated with the observed response. Further, the Agency's well established PbB model, the IEUBK model, which was used in the last assessment, does not provide estimates for ages beyond 7 years.
J) Modeling other endpoints in young children. The assessment for the last review did not quantitatively assess risk of health endpoints other than IQ loss primarily due to	The ISA (Section 2.8.2) describes the current evidence that informs characterization of concentration-response relationships for those health endpoints for which such evidence is most well established. These relationships are	The extent to which the evidence provides strong support for a quantitative characterization of the concentration –response relationship for non-IQ behavioral endpoints is unclear. Further, concentration-response functions utilized in

[13] Chiodo et al.(2007) and Jusko et al. (2008) will be included in the second draft ISA.

Limitation/Uncertainty in Risk Model	Information Newly Available in this Review	Consideration of Potential Utility and Impact on Quantitative Exposure/Risk Assessment
limitations in the available information or an absence of relevant information for developing informative concentration-response functions.	for PbB levels in children with cognitive deficits and PbB levels in adults with increased blood pressure, mortality, and indicators of nephrotoxicity. With regard to the evidence for concentration-response relationships of neurobehavioral outcomes (other than IQ), a number of recent studies have investigated impacts on cognitive domains at lower PbB levels including those less than 5 µg/dL (e.g., Chiodo et al., 2008; Jedrychowski et al., 2008; Krieg et al., 2010; Miranda et al., 2009) and behavior (e.g., Braun et al. 2006, 2008; Nigg et al., 2008; Wang et al., 2008; Nicolescu et al,, 2010) in children. Among these studies, some involve child populations ranging in age up to 15 or 16 years of age. Additionally they variously provide continuous and/or noncontinuous effect estimates and some analyze associations of prenatal exposure (based on cord blood measurements).[14] The ISA also recognizes evidence for associations of renal, immune system, hematological, and reproductive effects with PbB levels below 5 µg/dL in child populations (draft ISA, Tables 2-2 and 2-3). However, the evidence for these effects has limited applicability for a quantitative assessment of the risk of specific health	quantitative risk assessment are typically developed from epidemiological analyses that report continuous functions and not typically developed from non-continuous epidemiological analyses (e.g., quartile regression analyses). There is reduced resolution associated with the information from the latter which contributes additional uncertainty to concentration-response functions developed from these analyses. Since most of the recent studies of non-IQ neurobehavioral endpoints do not include continuous effect estimates (e.g., quartile regression analysis), they do not provide strong support for a concentration-response function for use in quantitative risk assessment. Additionally, some of these studies focus on older (teenage) children, and as noted in the discussion (see prior row) of the Kim et al. (2009) study, EPA's established childhood PbB model, the IEUBK model, does not provide estimates for older children. Regarding the other non-neurological endpoints, we recognize that the evidence is unclear regarding the role of lead-induced changes in individual immune system, red blood cell function indicators and delayed onset to puberty in eliciting or contributing to immunological, hematological or reproductive health outcomes. An improved understanding of these linkages is needed to support quantitative risk assessment for the impact of ambient air Pb exposure in contributing to risk of outcomes related to these endpoints.

[14] Jedrychowski et al. (2008), Nigg et al. (2008), and Wang et al. (2008) will be included in the second draft ISA.

Limitation/Uncertainty in Risk Model	Information Newly Available in this Review	Consideration of Potential Utility and Impact on Quantitative Exposure/Risk Assessment
	outcomes. The recent studies include: an NHANES analysis (Fadrowski et al., 2010), which reports associations between PbB and a kidney function indicator in adolescents aged 12-20 years; some studies reporting Pb-associated changes in specific immune system indicators in child populations ranging in age from 6 months through mid teenage years (e.g, Sarasua et al., 2000, Karmaus et al., 2005) or associations of prenatal exposure (based on cord blood measurements) with allergic sensitization in young children (Jedrychowski et al., 2011); a study reporting an association of low cord PbB levels with decreased calcium-magnesium ATPase activity in newborn red blood cells (Huel et al., 2008); and, two studies reporting an association of childhood PbB with delayed onset of puberty (e.g., Hauser et al., 2008; Williams et al., 2010).	More detailed limitations associated with applicability of recent non-neurological endpoint studies for quantitative risk assessment variously include: focus on older children, for which there is increased uncertainty associated with interpretations regarding the exposure conditions contributing to the measured PbB levels and to the associated effects; focus on associations with measures of prenatal exposures (e.g., cord blood) for which we are lacking in established PbB modeling tools; and, focus on non-U.S. populations for which characteristics influencing Pb uptake (e.g., diet) may differ from U.S. populations. Additionally, quantitative risk assessment for some of the identified effects (e.g., kidney function) would require baseline incidence data the availability of which may be limited for the age groups studied.
Estimating Risk for Other Populations/Lifestages		
K) Modeling endpoints for adult cohorts. The assessment for the last review did not quantitatively assess the risk of health endpoints that have been associated with PbB levels in adults in epidemiological studies primarily due to uncertainties regarding the role of	The draft ISA (Section 2.8.2) describes the current evidence that informs characterization of concentration-response relationships for those health endpoints for which such evidence is most well established. Among adults, these relationships with blood lead levels are increased blood pressure, mortality,	Lack of information regarding the specific Pb exposures during a lifetime (time periods and magnitudes) which contribute to the observed health outcomes continues to introduce substantial uncertainty to the simulation of adult health outcomes resulting from changes in air Pb. This prevents us from developing concentration-response functions for

Limitation/Uncertainty in Risk Model	Information Newly Available in this Review	Consideration of Potential Utility and Impact on Quantitative Exposure/Risk Assessment
historical exposures in eliciting these health outcomes. Additionally, we are limited in our capabilities for simulating the impact of changes in ambient air Pb levels on adult PbB levels (or bone Pb) (see entry "H" above).	and indicators of nephrotoxicity. With regard to the evidence for concentration-response relationships, a number of studies have investigated impacts at lower blood lead levels including those less than 5 μg/dL with neurobehavioral outcomes, (e.g., Krieg et al., 2009), cardiovascular effects (e.g., Sun et al., 2008), renal effects (e.g., Akesson et al., 2005), and immune system indicators (e.g., Kim et al., 2007). However, substantial uncertainty continues to be associated with the interpretation of adult epidemiology studies as to the role of historical exposures in eliciting the health outcomes observed and with regard to the role of endogenous Pb in influencing concurrent blood or bone Pb measurements. Furthermore, ongoing research has not substantially reduced uncertainty related to predicting changes in PbB or bone Pb associated with reductions in ambient air Pb-related exposures.	modeling potential adult health endpoints. The absence of biokinetic models specifically covering adults also limits our ability to model adult health endpoints as a component in a risk assessment evaluating ambient air Pb. Our consideration of any effect estimates for adult health endpoints occurring at non-occupational levels of Pb exposure is limited by our lack of a reliable means for estimating relationships between air-Pb levels (e.g., associated with alternate potential standards) and PbB levels in adulthood. Further, the contribution to blood lead from endogenous Pb (e.g., stored in bone) that can reflect historical Pb exposures complicates the utilization of blood lead as a biomarker of current exposures in adults, and consequently contributes uncertainty in interpreting epidemiological studies with regard to the Pb exposures eliciting observed health outcomes.

2.3 KEY OBSERVATIONS AND CONCLUSIONS

Information presented in Table 2-3 reflects EPA staff assessment of the degree to which research published since the last Pb NAAQS review (as summarized in the draft ISA) can address specific uncertainties associated with the Pb NAAQS risk assessment. In those instances where there is new information in the areas of an identified source of uncertainty in the previous assessment, the critical consideration by EPA staff is with regard to the extent to which use of that new information in a quantitative assessment would provide risk estimates for exposure to air-related Pb that are appreciably different or with which the uncertainty is appreciably lower than the estimates generated for the previous review. In considering this point, staff recognizes that this and all newly available evidence will be considered in the Policy Assessment in terms of both evidence-based considerations and risk/exposure-based considerations. With regard to the latter, to the extent to which a decision is made not to develop a new version of the risk model to generate new exposure and risk estimates, staff intends to consider any newly available information to help to qualitatively interpret the risk estimates generated for the last review.

The decision whether to develop a comprehensive new assessment of exposure and risk associated with air-related Pb for this review focuses on consideration of the extent to which the newly available information, if used to update the risk model, has the potential to result in new exposure and risk estimates that are substantially different from estimates generated for the previous review. In this context, "substantially different" would 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. As noted above, in the event that a new exposure and risk assessment is not warrant, newly available evidence may still be used to qualitatively or semi-quantitatively interpret exposure and risk estimates generated for the last review, in order to enhance their potential utility in informing the current review.

Key observations regarding the potential impact of newly available information in a revised risk model are presented below, in the general order in which sources of uncertainty are considered in Table 2-3.

- **Ambient air Pb levels (focus on urban study areas):** We are not in a position to substantially improve our characterization of ambient air Pb levels in the context of the urban study areas, including our simulation of current and alternative standard levels using proportional adjustment. Furthermore, we do not have additional information that would help us further interpret risk estimates generated for the urban study areas in the last review with regard to this element of the analysis.
- **Urban soil Pb and drinking water Pb levels:** While there is updated information on Pb levels in urban soil (and likely drinking water), we do not have updated data for improving our characterization of the degree of correlation between pathways in

modeling total Pb exposure and risk. This means that, even if we were to incorporate improved data on urban soil Pb and drinking water Pb, estimates of higher-end exposures (which are of particular interest in the risk assessment) would still be subject to uncertainty reflecting our inability to representatively characterize the degree to which pathway exposures are correlated (particularly at higher-percentiles of exposure).

- **Simulation of urban dust Pb levels using the hybrid model:** New information characterizing the cleaning efficiency of vacuuming on hard surface floors (and associated information on the size of Pb particles left behind) may be informative to our interpretation of the dust modeling and associated urban residential risk estimates developed in the last review. However, the new data are not directly applicable in the context of updating or even quantitatively performance-evaluating the hybrid indoor dust model.

- **Assessing the pathway-specific nature of child Pb exposure (including ambient air-sourced Pb) in the urban residential context:** New studies based on analysis of NHANES and NHEXAS datasets and other studies in specific locations provide insights on factors related to PbB levels in children, including detailed assessment of housing characteristics linked to indoor dust Pb levels and ultimately PbB and/or consideration for dietary and drinking-water exposure in addition to indoor dust Pb. However, a number of factors preclude the direct use of these studies to either update elements of the risk assessment, or quantitatively evaluate the performance of models (e.g., the studies exclude consideration of ambient air-sourced Pb, while focusing on other sources such as indoor paint Pb, or the studies may focus on an older study population that is not directly comparable to the child population reflected in the risk assessment). For these reasons, while information from these studies is not conducive to updating the risk model, in some instances, the information may be useful in further interpreting risk estimates generated in the previous analysis.

- **Predicting PbB levels for residential child populations:** There have not been significant refinements to the IEUBK model (or further development of alternative models) for simulating child PbB levels. By contrast, there are newer data available for characterizing the variability in child PbB levels (i.e., the GSDs used in defining population variability in PbB levels in the risk assessment). However, our ability to refine estimates of higher-end PbB levels through the use of enhanced GSDs is compromised significantly by ongoing uncertainty (referenced above) in characterizing pathway contributions to total exposure and risk for these higher percentiles of the simulated population. Therefore, while the newer data on PbB variability could be useful in further interpreting risk estimates generated for the previous review, we believe there is little utility in using these updated GSDs to generate new risk estimates.

- **Modeling IQ loss in young children:** Studies published since the last review continue to support the conclusions from the last review regarding the role of environmental levels of Pb exposure in contributing to IQ loss and the evidence continues to support the association of this endpoint with the lowest blood levels studied. These findings reaffirm our emphasis on this endpoint as risk metric for quantifying the impact of Pb

exposure on neurocognitive function in young children (up to 7yrs of age) in the last review. The newly available studies of PbB associations with IQ loss do not provide strong support for development of a new or revised concentration-response function for blood lead-IQ loss.

- **Modeling other endpoints in children or other populations/lifestages:** The newly available studies for additional endpoints in children that are recognized in the draft ISA (e.g., Table 2-2) inform our understanding of Pb-associated effects as discussed in the draft ISA, and will be additionally considered in the subsequent Policy Assessment for this review. However, these studies do not appear to provide support for quantitative risk assessment for these endpoints. For example, as noted in Table 2-3 above, there is uncertainty in interpreting the relationship of some of the immune system and red blood cell function indicators with health outcomes. Additionally, our capability for quantitatively assessing endpoints for older cohorts (e.g., older than 7 years of age) is limited by the fact that EPA's well established IEUBK model that we used in the last risk assessment does not cover these older cohorts. Our ability to model risk of lead-induced health outcomes in older children and adults in epidemiological studies is limited by uncertainties regarding the role of past exposures in eliciting the health outcomes associated with current PbB levels (as well as influencing current PbB levels). In the case of adult studies, the role of past exposures, which are likely higher, contributes particular uncertainty regarding the specific Pb exposures eliciting the observed outcomes. There is also uncertainty associated with simulation of changes in adult PbB levels (or bone Pb) resulting from reductions in exposure to Pb originally sourced from ambient air. These uncertainties affect our ability to disentangle the effects of past exposures from exposures to the lower air concentrations associated with the current standards.

In conclusion, we note the availability of new information on Pb exposure and risk published since the last review that may be useful in further interpreting risk estimates generated for the previous review, thereby enhancing their utility in informing the current review. However, we do not believe that the information newly available in this review provides 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. Specifically, we do not believe that any of the primary sources of uncertainty identified to have the greatest impact on risk estimates would be substantially reduced by using this new information to update the risk model, including inputs to that model. Notwithstanding our consideration here of the use of newly available information in a new or updated risk assessment, we note the need to carefully consider the newly available information (as characterized in the final ISA) with regard to any appropriate further interpretation of the risk assessment findings from the last review in our risk and exposure based considerations in the Policy Assessment for this review.

3 ECOLOGICAL RISK ASSESSMENT

The evaluation of information newly available in this review is structured around consideration of two key questions:

- Is there newly available information relevant to critical uncertainties or limitations associated with ecological risk assessment in the last review?
- To what extent does the currently available information support the development of an updated or new quantitative risk assessment that would generate results providing more specific or more certain estimates of ecological risk associated with the current Pb secondary standard?

Overall, we consider the extent to which the available information supports a new quantitative risk assessment likely to contribute to substantive new conclusions regarding the risk to welfare associated with Pb under current air quality conditions that will better inform the Administrator's judgment of the adequacy of protection against adverse environmental effects afforded by the current NAAQS.

As noted in chapter 1 above, the CAA § 302(h) defines "Effects on welfare" to include a wide range of effects including effects on soil, water, crops, vegetation, and manmade materials, "whether caused by transformation, conversion, or combination with other pollutants." Because of the broad range of effects that must be considered in the design and implementation of an assessment of exposure and risk associated with Pb, we are faced with a level of complexity that is substantially greater than for similar assessments typically performed for other criteria pollutants. For example, unlike most other criteria pollutants, risk associated with exposure to Pb originally released to ambient air is multimedia in nature, with exposure and risk associated with a range of pathways associated with deposition of ambient air Pb. Additionally, the persistent nature of Pb means that exposure and risk can be associated with Pb originally emitted into the ambient air recently or at some point in the past, from current or historic sources, under conditions associated with previous Pb NAAQS or under those prior to the existence of any Pb NAAQS. Furthermore, ecological exposures and risk also result from uses of Pb that contribute Pb to the environment without passing through ambient air, such as land and water disposal of wastes, leaching of solder used in water distribution systems into water that flows through wastewater treatment facilities and land uses such as mining.

The screening-level ecological risk assessment for aquatic and terrestrial case studies completed for the last review, while limited and accompanied by various uncertainties, suggested occurrences of environmental Pb concentrations with the potential for adverse environmental effects to exist under the then-current standards. These findings supported similar, largely

qualitative, conclusions drawn from consideration of the evidence. Given the limited quantitative understanding at that time of the impacts of air-related Pb in ecosystems under conditions meeting the NAAQS, evidence of Pb effects on organisms was generally extrapolated to ecosystem effects. Taken together, the Agency concluded that the available data and evidence, primarily qualitative, suggested the potential for adverse environmental impacts under the then-current standard. While lacking data to provide a quantitative basis for setting a secondary standard different from the primary, the Administrator concurred with CASAC's conclusion that the level of the secondary standard should be reduced to at least as low as the level of the revised primary standard. Accordingly, the secondary standard was revised to be identical to the revised primary standard (USEPA, 2007c; 73 FR 66964).

In the previous review, the scientific evidence of direct effects of Pb from ambient air under conditions meeting the then-current standard was limited or lacking for specific ecosystems, ecosystem services, or organisms. In considering the extent to which the now available information warrants development of a quantitative ecological risk assessment in this review, we consider the availability of evidence to support a more refined understanding of the direct and indirect effects of deposited ambient Pb on ecosystems and organisms and of the long-term behavior of deposited Pb. We focus most specifically on the ability of current data sets to characterize exposure of ecosystems in the U.S. to ambient Pb being deposited under the current standard. Critical to this focus is consideration of the extent to which the available information improves our understanding of ecological effects attributable to Pb deposited from ambient air under conditions associated with the current standard in light of other sources of current and historic Pb in the environment. We also look to any new scientific evidence that might be available to provide additional insight into the responsiveness of ecosystems to changes in Pb deposition. As part of this evaluation we consider the adequacy of any new scientific evidence on critical loads that might be used in assessing ecosystems potentially vulnerable to Pb on a scale that is large enough to provide information which could inform the Administrator regarding the adequacy of the current standard.

While focusing here on evaluation of the newly available evidence with regard to a role in quantitative risk assessment, we will be further and more comprehensively evaluating the evidence, as well as past quantitative analyses in the context of the Policy Assessment to be developed for this review. As described in the integrated review plan, the Policy Assessment, a draft of which will be completed subsequent to completion of the ISA, will consider conclusions that can be drawn from the currently available evidence as well as any available quantitative analysis, including that from the previous review, in light of that evidence, as to the adequacy of the current standard (USEPA, 2011b).

This chapter first provides an overview of the quantitative exposure and welfare risk assessment performed in the last review (section 3.1) and then considers the extent to which information and conclusions presented in the ISA provide support for the development of a new quantitative assessment of welfare effects (section 3.2), particularly with regard to the extent which such an assessment could be expected to inform our consideration of the adequacy of the current standard. Key observations and conclusions drawn from this evaluation are presented in section 3.3.

3.1 OVERVIEW OF THE PREVIOUS ASSESSMENT

3.1.1 Conceptual Model

In planning for the quantitative ecological risk assessment in the last review, we developed and considered a conceptual model of environmental pathways of Pb distribution, associated exposures and associated endpoints and risk metrics (Figure 3-1). This model provided a framework in which to consider the evidence for Pb in designing the approach for the assessment. Central to this model is the assessment of terrestrial and aquatic exposures and, specifically within the context of the NAAQS review process, the portion of these exposures that are associated with ambient air concentrations allowed under the current standard.

The focus in the review of the Pb NAAQS, and consequently for an informative assessment, is on Pb emitted to ambient air. As recognized in Figure 3-1, however, other (nonair) sources of environmental Pb (including mining activities, contaminated landfills, etc.) also contribute to Pb concentrations in environmental media. Of most interest in the quantitative analysis for the review were present and past emissions to air and the associated deposition to sensitive ecosystems.

Ecologically significant pathways of exposure to air-related Pb are predominantly those involving deposition of Pb from air to other media (soils, surface waters, and sediments). Exposure to Pb in air has been considered a relatively less significant ecological exposure/risk pathway. Accordingly, the analysis developed for the last review focused primarily on deposition and resulting concentrations in environmental media.

Those organisms in contact with Pb-contaminated media, whether directly or by ingestion of prey species that have accumulated Pb due to direct contact with contaminated media, are likely to be the most highly exposed organisms in the environment. There is limited evidence for biomagnification of Pb in food chains, but sensitivities to Pb vary widely within and among groups of organisms with similar exposures.

As recognized in the 2006 AQCD and the Staff Paper developed for the review, sufficiently elevated environmental exposure to Pb can cause a range of effects at the species and

population levels thereby altering ecosystem processes. Known effects of elevated Pb exposure include changes in growth, development and reproduction, hematological effects, neurobehavioral effects, and increased mortality rates among some organisms. These effects, in turn, can adversely impact community structure, biodiversity and ecosystem functions. In a general sense, changes in the functioning of ecosystems can impact services provided by an ecosystem to human populations.[15]

In planning the quantitative analysis for the last review, staff considered the potential for developing risk metrics descriptive of individual, population, and ecosystem effects estimated to result from Pb exposures. Given data and other limitations, the focus was on organism and population-level metrics. Individual-level toxicity data considered informative to thresholds for population-level effects in sensitive species were used to develop the screening levels used in the assessment performed during the previous review.

On an ecosystem scale, an approach for integrating the consideration of exposure pathways and risk for multimedia pollutants such as Pb is represented by critical loads analyses.[16] Application of this type of approach requires a wide array of data to support the quantitative characterization of the disposition and impact of the pollutant in specific ecosystems.[17] During the last review, the 2006 AQCD assessed the available information on critical loads for Pb which was drawn primarily from then somewhat recent work in Europe. Analyses were not available for U.S. locations, and the European critical load values for Pb that had been developed were highly specific to the bedrock geology, soil types, vegetation, and historical deposition trends in each European country (AQCD, p. E-24). As a result, the 2006 AQCD concluded that "[a]t this time, the methods and models commonly used for the calculation of critical loads have not been validated for Pb" and that "[m]any of the methods neglect the speciation of Pb when estimating critical limits, the uptake of Pb into plants, and

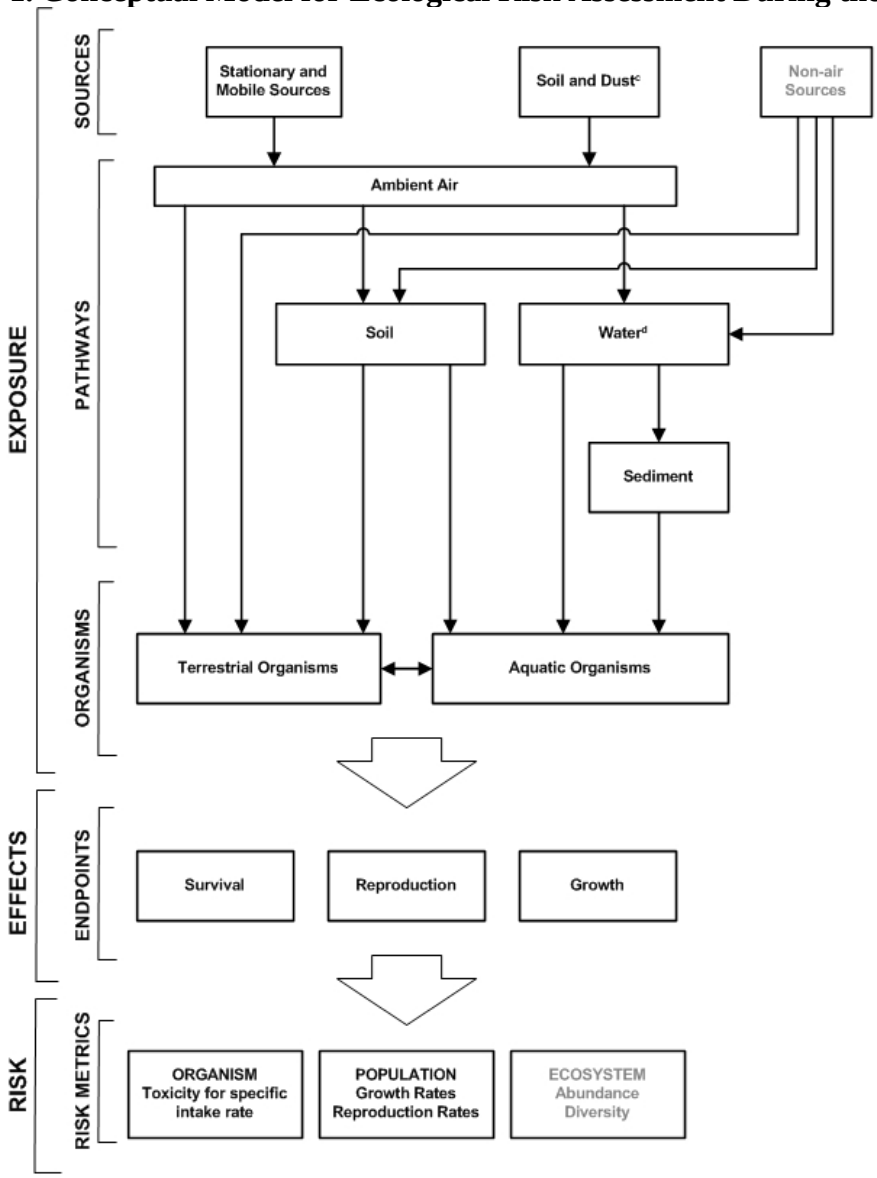
[15] In the current review of the secondary standards for nitrogen and sulfur oxides, for which there was sufficient evidence to provide for such a consideration, ecological services has been recognized as a useful tool for assessing adverse impacts to public welfare (USEPA, 2011).

[16] In the 2006 AQCD, critical loads were defined as threshold deposition rates of air pollutants that current knowledge indicates will not cause long-term adverse effects to ecosystem structure and function (AQCD, p. 7-33). Conceptually, the critical load approach provides a means of gauging whether a particular ecosystem in a given area receives deposition that results in a level of biological harm that is defined by a critical limit in the sentinel parameter for the targeted pollutant and type of ecosystem harm. The critical load estimate for an ecosystem is analogous to a quantitative estimate of that ecosystem's "susceptibility" to the type of ecosystem harm being assessed. The greater the critical load value, the greater the ability of the ecosystem to accommodate the pollutant loading without harm.

[17] The critical load concept was recently used in the quantitative assessment developed for consideration in the currently ongoing review of the secondary NAAQS for oxides of nitrogen and sulfur (USEPA, 2009). That assessment was based on the well-established state-of-knowledge and location-specific data, developed over the past four decades, on nitrogen and sulfur deposition and their role on ecosystem acidification in the U.S., with resulting impacts to various ecosystem services.

outflux of Pb in drainage water, limiting the utility of current models" (AQCD, p. 7-46). Further, the AQCD noted that future efforts were needed that fully incorporated the role of Pb speciation into critical load models, and include validations of the assumptions used by the models (AQCD, p. 7-46). Accordingly, the quantitative assessment for the last Pb NAAQS review did not involve critical load analyses.

Figure 3-1. Conceptual Model for Ecological Risk Assessment During the Last Review.



[a] Many of the processes and pathways above are circular in nature. For the clarity of the schematic, they are shown as bidirectional.
[b] Components with gray text were not addressed in the quantitative assessment due to uncertainty regarding available data and modeling tools.
[c] Includes contributions of historical sources, including (but not limited to) emissions from the use of leaded gasoline, historical emissions from stationary sources, and exterior leaded paints.
[d] Water in this schematic represents all surface water bodies; however, only freshwater was addressed in this assessment.

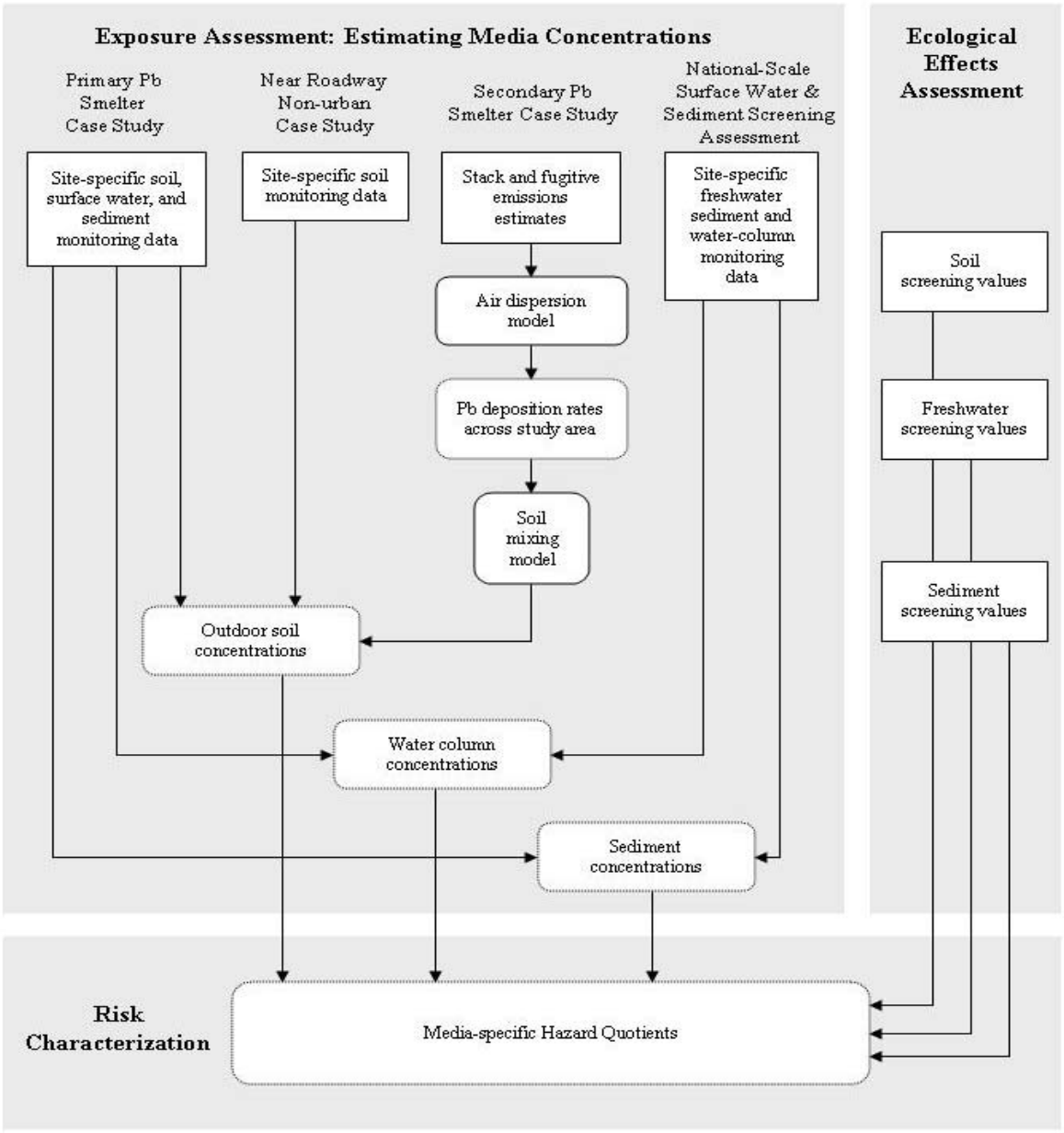
3.1.2 Overview of Analytical Approach

The screening-level assessment in the last NAAQS review involved several case studies and a national-scale screening assessment (Figure 3-2). Three quantitative case studies were designed to estimate the potential for ecological risks associated with exposures to Pb emitted into ambient air in three situations: areas surrounding a primary Pb smelter, areas surrounding a secondary Pb smelter, and a near-roadway, nonurban location. Activities for a fourth case study (ecologically vulnerable locations) focused on identification and description of the location as well as consideration of literature findings regarding the role of atmospheric Pb and the movement of Pb within this ecosystem, although new quantitative analyses were not performed.[18]

Exposure concentrations in soil, surface water, and/or sediment were estimated for the three case studies from available monitoring data or modeling analysis and then compared to ecological screening benchmarks (e.g. Ecological Soil Screening Levels (Eco-SSLs) and Ambient Water Quality Criteria (AWQC)) to assess the potential for ecological impacts from Pb that was emitted into the air. A national-scale screening assessment was also used to evaluate surface water and sediment monitoring locations across the United States for the potential for ecological impacts associated with atmospheric deposition of Pb.

[18] 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: (1) it is in an acidified watershed and therefore was expected to have higher bioavailability of Pb, (2) there were no identified point sources of Pb in the surrounding area, which might allow for an evaluation of impacts of regional background Pb concentrations; (3) it is in an elevated area subject to comparatively higher deposition of Pb due to wind speed and precipitation; and (4) there are available data on concentration trends 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 (USEPA, 2006, Appendix E). Studies of the three media variously reported the following: (1) atmospheric Pb inputs do not directly affect Pb levels in streams at HBEF because deposited Pb is almost entirely retained in the soil profile; (2) results of the soil horizon analysis show that Pb has become more concentrated at lower depths over time and that the soil profile serves as a Pb sink, drastically reducing dissolved Pb levels 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, leading authors to conclude that the contribution of dissolved Pb from soils to streams is insignificant (less than 0.2 $g \cdot ha^{-1} \cdot yr^{-1}$).

Figure 3-2. Analytical Approach for Screening-level Assessment in the Last Review.



The measures of exposure in the screening assessment for the last review were total Pb concentrations in soil, dissolved Pb concentrations in fresh surface waters (water column), and total Pb concentrations in freshwater sediments. Exposure concentrations were estimated for the three case studies and the national-scale screening assessment as described below.

- For the primary Pb smelter case study, which involved a smelter that had been operation for more than 100 years, measured concentrations of total Pb in soil, dissolved Pb in surface waters, and total Pb in sediment were used to develop point estimates for locations with Pb thought to be associated with atmospheric Pb deposition, rather than with nonair sources, such as runoff from waste storage piles.
- For the secondary Pb smelter case study, soil concentrations of Pb were estimated using fate and transport modeling based on EPA's Multiple Pathways of Exposure (MPE) methodology (USEPA 1998).
- For the near-roadway non-urban case study, soil concentration measurements collected for two locations adjacent to interstate highways, one in an area of fairly high-density development (Corpus Christi, Texas) and another in an area of medium-density development (Atlee, Virginia), were used to develop point estimates of Pb associated with historical deposition.
- In the national-scale surface water and sediment screening assessment, measurements of dissolved Pb concentrations in surface water and total Pb concentrations in sediments for locations across the United States were used. Air emissions, water discharge, and land use data for the areas surrounding these locations were assessed to identify locations where atmospheric Pb deposition may be expected to contribute to potential ecological impacts. The exposure assessment focused on these locations.

The tools used for assessing the potential for effects included ecological screening values, derived from the Ecological Soil Screening Levels (Eco-SSLs) developed by the EPA's Superfund program (USEPA 2003, 2005c), EPA's recommended ambient water quality criteria (AWQC), and sediment screening values developed by MacDonald and others (2000, 2003). A hazard quotient (HQ) was calculated for various receptors to determine the potential for risk to that receptor. The HQ was calculated as:

$$HQ = \text{(estimated media concentration)} / \text{(ecotoxicity screening value)}$$

For 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). HQ values less than 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.

3.1.3 Key Limitations and Uncertainties

While the screening-level analyses performed in the previous review provided examples of the distribution of atmospheric Pb into other media where it can contribute to ecological risks, they were severely limited in the extent to which they could inform an understanding of the role of atmospheric deposition under conditions associated with the NAAQS.[19]

- The ecological risk screen was limited to specific case study locations and other locations for which Pb data were available. The limited availability of U.S. locations for which recent measurements of Pb concentrations were available even with the reliance of national databases to identify ecosystems and datasets constrained our ability to characterize what we knew nationally regarding current conditions, including the influence of current atmospheric Pb conditions, or those associated with the then-current standard.
- Further, while efforts were made to identify situations in which the Pb exposures would have primary contributions from airborne Pb and not be dominated by nonair sources, there was uncertainty as to whether other sources might have actually contributed to the Pb exposure estimates, and the extent to which air-attributable contributions represented conditions when the then-current standard was not met.
- Additionally, the screening-level tools used for effects assessment (e.g., AWQC, Eco-SSLs, sediment criteria) and/or the media-specific parameters available for the media concentration estimates in the assessment did not accommodate a more rigorous or detailed consideration of bioavailability characteristics influential to Pb toxicity in these media (USEPA, 2007c).

The screening assessment results included several locations where concentrations of Pb in soil, surface water and sediments exceeded screening values for these media, indicating the potential for adverse effects associated with Pb. However, the contribution of air emissions to

[19] Limitations in the assessment generally reflected those recognized by the AQCD assessment of the evidence: *In summary, due to the deposition of Pb from past practices (e.g., leaded gasoline, ore smelting) and the long residence time of Pb in many aquatic and terrestrial ecosystems, a legacy of environmental Pb burden exists, over which is superimposed much lower contemporary Pb loadings. The potential for ecological effects of the combined legacy and contemporary Pb burden to occur is a function of the bioavailability or bioaccessibility of the Pb, which, in turn, is highly dependent upon numerous site factors (e.g., soil organic carbon content, pH, water hardness). Moreover, while the more localized ecosystem impacts observed around smelters are often striking, these perturbations cannot be attributed solely to Pb. Many other stressors (e.g., other heavy metals, oxides of sulfur and nitrogen) can also act singly or in concert with Pb to cause such notable environmental impacts.* [AQCD, p. E-24]

concentrations, and consequently to potential risk, in all of these cases is unknown. While it was not possible to dissect the contributions of air Pb emissions from other sources, and it is likely that, at least for the long-operating primary smelter, the air contribution is significant, the Pb source at that location had been in operation well before the establishment of the Pb NAAQS. Thus, the assessment results were consistent with evidence-based observations of the influence of airborne Pb and changes in airborne Pb on Pb in aquatic systems, such as those drawn from historical patterns observed in sediment cores from lakes and from other Pb measurements. However, the quantitative analyses, as well as the available evidence, were severely limited in their ability to inform quantitative conclusions regarding the secondary standard.

3.2 CONSIDERATION OF NEWLY AVAILABLE EVIDENCE

In considering the evidence newly available in this review, we focus here on the extent to which it addresses information gaps or areas of uncertainty associated with the information available in the last review. Such gaps limited the quantitative assessment that could be developed in that review as well as conclusions that could be drawn from it. As discussed in the sections above, key areas included limitations and gaps in the information needed to support a critical loads analysis, or other analyses that would quantitatively inform consideration of the adequacy of the then-current secondary standard to provide the requisite protection against welfare effects associated with Pb in ambient air.

In the subsections below, four areas of limited information in the last review are considered with regard to the availability of new information in this review as described in the first draft ISA. Our focus in these sections is on the extent to which the now available information addresses key limitations in developing quantitative analyses that would substantively and quantitatively inform consideration of the adequacy of the current secondary NAAQS. Further, beyond these four areas, we note that with regard to ecosystem services, the draft ISA concludes that "[a]lthough evidence is available to support Pb impacts to supporting, provisioning, regulating and cultural ecosystem services, there [are] insufficient data available to adequately quantify these adverse effects" (draft ISA, p. 7-112).

3.2.1 Factors Affecting Lead Bioavailability

As recognized in the last review, a wide range of environmental factors affect the distribution of Pb in the environment and Pb bioavailability and, accordingly, Pb-induced toxicity and associated ecological risk (2006 AQCD, summarized in Section 8.7; draft ISA, section 2.6.1. The first draft ISA discusses the current evidence regarding various aspects of Pb bioavailability and toxicity. For example, the draft ISA concludes that the evidence in this review further supports the findings of the previous Pb AQCDs that biological effects of Pb on terrestrial organisms vary with species and life stage, duration of exposure, form of Pb, and soil

characteristics (draft ISA, p. 2-29). The evidence reviewed in sections 7.2.3 and 7.2.4 of the draft ISA used as the basis for the conclusion demonstrates that "many factors, including species and various soil physiochemical properties, interact strongly with Pb concentration to modify those effects" (ISA, p. 2-30).

Newly available evidence on Pb bioavailability from sediments and soils to aquatic and terrestrial vegetation, respectively, indicates Pb to be relatively more bioavailable in sediment than in soil (draft ISA, 7.3.10.2). Currently available models for predicting bioavailability in aquatic systems focus on acute toxicity and do not consider all possible routes of uptake, making them of limited applicability, especially when considering species-dependent differences in uptake and bioaccumulation of Pb (draft ISA, 7.3.10.1). With regard to this area, the draft ISA concludes "there are large differences in species sensitivity to Pb, and many environmental variables (e.g., pH, organic matter) determine the bioavailability and toxicity of Pb" (draft ISA, section 2.6.11).

3.2.2 Transport of Lead between Ecosystem Compartments

At the time of the last review, information supporting quantitative descriptions of Pb transport within and among different ecosystem compartments was limited.

With regard to terrestrial systems, the currently available information supports and expands on the evidence regarding some of the complexities associated with the movement of Pb in those systems, particularly vertical transport across soil horizons. For example, location-specific differences in soil horizons have been observed to affect Pb movement across those horizons (draft ISA, section 3.3.3.1). Recent research provides insights into the details of Pb sequestration processes (draft ISA, section 3.3.3.2) and the roles of iron, manganese and calcium in soils (draft ISA, section 3.3.3.2).

Recent research on Pb transport in aquatic systems confirms the dominant role of iron and organic rich colloids in Pb transport and provides additional information on Pb residence times in some rivers and lakes (draft ISA, section 3.3.2.1). Newly available studies provide additional detail on resuspension processes of Pb from sediments in natural waters, including the influence of organic material, iron and manganese, as well as the role of sediment anoxic or depleted oxygen conditions in Pb cycling (draft ISA, section 3.3.2.3). This newly available research confirms the important influence of resuspension on the lifetime of Pb in aquatic systems (draft ISA, section 3.3.2.3). Additionally, newly available information expands the evidence base describing the movement of Pb in surface runoff, including greater detail on amounts, particle size distributions and composition (draft ISA, 3.3.2.4).

In considering the available information on the movement of Pb among ecosystem compartments, the draft ISA concludes that despite our increasing knowledge there is a lack of

information not only on bioavailability, as affected by the specific characteristics of the receiving ecosystem, but also on the kinetics of Pb distribution in ecosystems in long-term exposure scenarios (draft ISA, section 2.6.11). This lack of information limits our ability to assess the proportion of observed effects that are attributable to atmospheric sources.

3.2.3 Relative Roles of Atmospheric Deposition and Other Pb Sources to Ecosystems

The role of current atmospheric deposition on Pb-associated risk to ecosystems was a large source of uncertainty that limited the design of and conclusions drawn from the quantitative assessment in the last review. As a general matter, recent studies reviewed in the draft ISA report deposition data consistent with fluxes reported in the 2006 AQCD and consistently demonstrate reductions in Pb deposition to soils since the phase-out of leaded on-road gasoline (draft ISA, section 3.3.3.1).

Chapter 3 of the 1st draft ISA (2011a) reviews the sources of ambient Pb and deposition into ecological systems. This included studies on ambient deposition, runoff and re-suspension. Atmospheric deposition is thought to be the largest source of Pb in surface waters (AQCD, 2006; ISA, Section 3.3.2), however runoff is also identified as a major source.

As summarized in the draft ISA, the recently completed Western Airborne Contaminants Assessment Project (WACAP) of the U.S. National Parks Service is the most comprehensive database, to date, on contaminant transport and depositional effects on sensitive ecosystems in the U.S. In this study, contaminants were shown to accumulate geographically based on proximity to individual sources or source areas, primarily agriculture and industry. A clear decline in Pb concentrations in sediments after the discontinued use of leaded on-road gasoline was observed in sediment cores at almost all WACAP locations. Although, Pb was measured in snow, water, sediment, lichen and fish during the multiyear project, this metal was not quantified in air samples (draft ISA, section 3.6.2).

In its assessment of this area of the current evidence, the draft ISA concludes that there is "limited evidence to relate ambient air concentrations of Pb to levels of deposition onto terrestrial and aquatic ecosystems and subsequent movement of atmospherically-deposited Pb though environmental compartments (e.g., soil, sediment, water, biota)," and the relative contribution from atmospheric versus other sources in studies reporting on Pb accumulation in biota is usually not known (draft ISA, section 2.6.11).

3.2.4 Critical Loads (CL) Models

As noted in section 3.1.1. above, the critical load (CL) models for both terrestrial and aquatic systems available at the time of the last review (AQCD, 2006) did not account for Pb bioavailability within the system, fluxes within systems, or focused solely on atmospheric

deposition, ignoring other contributions. This was considered a main limitation to the use of CL models in the risk assessment. The draft ISA reviews several new studies utilizing CL models in terrestrial systems, however, as summarized in the draft ISA, "since the 2006 Pb AQCD there is no new significant information on critical loads of Pb in aquatic systems" (draft ISA, p. 7-112).

Since the last review our understanding of how CL can be used in the context of the NAAQS review process has improved. A CL model was utilized in the recently completed review of the Secondary National Ambient Air Quality Standards for Oxides of Nitrogen and Oxides of Sulfur (US EPA 2008, 2009, 2011c) and the overall approach was reviewed favorably by the CASAC committee. As noted above, that assessment was based on the well-established state-of-knowledge and location-specific data, developed over the past four decades, on nitrogen and sulfur deposition and their role on ecosystem acidification in the U.S., with resulting impacts to various ecosystem services. Thus, while since the last review we have gained experience with critical loads modeling, and in the case of Pb our understanding of the various factors that influence its environmental movement and toxicity is improving, we are still strongly limited with regard to data to quantify these processes and influences adequately in CL modeling of U.S. ecosystems.

As reviewed in the previous sections, there is new research available for the areas which were considered limitations during the previous review; however, the data remain difficult to incorporate into a larger-scale CL model. The CL study by De Vries and Groenenberg (2009), reviewed in the draft ISA (Section 7.2.7), did include fluxes of Pb within a system, one area previously considered a limitation in the model, however, application of this methodology at a national scale, requires localized data across a wide range of ecosystems, which are currently unavailable or inadequate. It is this lack of data regarding bioavailability, speciation, sources and fluxes at a local scale, which could be applied in a large-scale assessment that remains the primary limitation in using a CL model approach.

3.3 KEY OBSERVATIONS AND CONCLUSIONS

As described in section 3.2 above, the draft ISA describes evidence that expands our knowledge of the movement and potential adverse effects of Pb in ecosystems.

- **Bioavailability, speciation, fluxes and sources of Pb.** New research on bioavailability and speciation of Pb continues to describe the complexity of Pb bioavailability in ecosystems and the associated challenges to describing toxicity. The evidence currently available does not support the development of exposure-response functions. If such exposure-response functions were developed, for example for adverse impacts on fish populations, they could be directly linked to ecosystem services…
- **Contribution of atmospheric Pb to ecosystem loading.** The concept of critical loads has been applied in the literature to assess the risk to ecosystems from atmospheric deposition of pollutants. However, in the case of Pb, there are substantial limitations in the data needed to incorporate the necessary components (flux, bioavailability, speciation, and source) at ecosystem levels in a way that could support a large-scale application of a CL model to inform the NAAQS review.

In summary, while there are a number of new studies that improve 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. 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.

The new evidence reviewed in the draft ISA generally supports conclusions drawn in the previous review regarding the potential for Pb to impact ecosystems and also adds to our understanding of some aspects of the effects of Pb in ecosystems. However, gaps, limitations and uncertainties remain in the information available regarding areas that are critical to developing quantitative estimates of ecosystem risk associated with Pb in ambient air. In light of these critical limitations and uncertainties, staff concludes that the currently available information does not provide the means 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.

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