Lead: Human Exposure and Health Risk Assessments for Selected Case Studies

Volume I. Human Exposure and Health Risk Assessments - Full-scale

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

This document is the first volume of the report *Lead: Human Exposure and Health Risk Assessments for Selected Areas*. This volume describes the quantitative human exposure and health risk assessments[1] conducted to inform the U.S. Environmental Protection Agency's (EPA's) current review of the National Ambient Air Quality Standards (NAAQS) for lead (Pb).

As with the last review of the Pb NAAQS (see Section 1.2), the human exposure and health risk assessments (the risk assessment)[2] for this review reflect multimedia exposure pathways, and their influence on blood Pb levels as an internal index of exposure or dose (Section 1.1). The assessment for this review, as with that for the last review, utilizes a case study approach wherein a set of specific locations or case studies associated with policy-relevant Pb exposures are evaluated in detail. The case studies have been selected to provide a perspective on the nature and magnitude of air source Pb exposures and risk in the United States.

There are two phases to the risk assessment for the current review: pilot and full-scale. The first phase (the pilot assessment, described in Section 1.3) was presented in the first draft Staff Paper and accompanying technical report (USEPA, 2006a; ICF, 2006), and was the subject of a review by the Clean Air Scientific Advisory Committee (CASAC) on February 6 and 7, 2007 described in Section 1.4 (Henderson, 2007a). The initial full-scale analyses were presented in the July 2007 draft report (USEPA, 2007b) and were the subject of a CASAC review at a public meeting on August 28 and 29, 2007. In response to CASAC recommendations described in Section 1.4 (Henderson, 2007b), additional analyses, using a core modeling approach were conducted to complete the full-scale assessment.

All analyses for the full-scale assessment are described in this document, with the initial analyses being the focus in Chapters 3 and 4 and the additional analyses in Chapter 5. Further, given the significant time constraints of this review, risk results are provided in this document without substantial interpretation. Rather, interpretative discussion of these results is provided in the Staff Paper.

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[1] As described in the Preface to this document, the ecological risk analysis performed for this review, which will be considered in the policy assessment for the secondary standard, is presented in the draft technical report of the pilot phase risk assessments (ICF, 2006) and described in the Staff Paper (USEPA, 2007a).

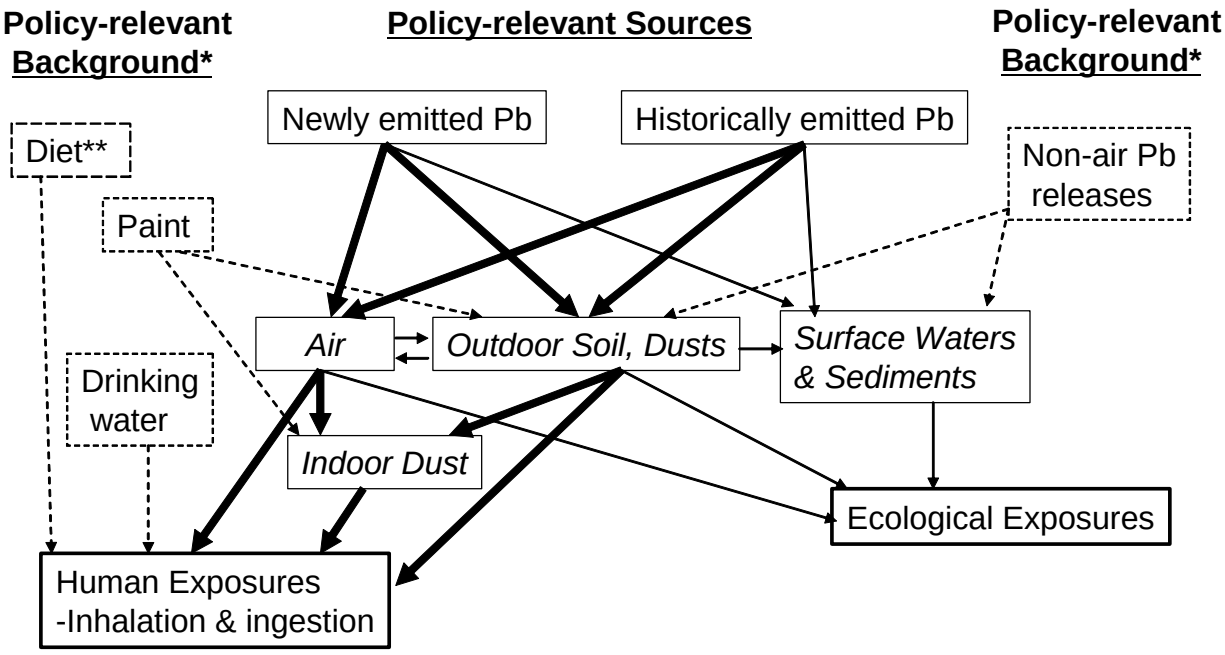
[2] Throughout the remainder of this document, the term "risk assessment" will be used to refer to both the human exposure and health risk assessments collectively, unless specific reference to either the human exposure or health risk assessment is required.

1.1 MULTIMEDIA ASPECT OF THE RISK ASSESSMENT

The focus for this Pb NAAQS risk assessment is on Pb derived from those sources emitting Pb to ambient air. In designing and implementing this assessment, we have been faced with significant limitations and complexity that go far beyond the situation for similar assessments typically performed for other criteria pollutants. In addition to the constraints of the timeframe allowed for this review, we are also constrained by significant limitations with regard to data and tools needed for the assessment. The multimedia and persistent nature of Pb and the role of multiple exposure pathways contribute significant additional complexity to the assessment as compared to other assessments that focus only on the inhalation pathway.

First, exposures to Pb emitted into the air occur via multiple pathways. As described in the *Air Quality Criteria for Lead* (USEPA, 2006b; henceforth referred to as the CD), "The multimedia aspects of Pb exposure can be seen in that Pb emissions to the air contribute to Pb concentrations in water, soil and dusts; Pb in soil and dust also can make important contributions to Pb concentrations in ambient air" (CD, p. 3-1).

Inhalation exposures can result from Pb emitted to the ambient air recently or from Pb emitted in the past that has deposited from air to soil or dust and then become resuspended in the ambient air. Further, Pb emitted into the ambient air can contribute to ingestion exposures (associated with indoor dust, outdoor soil/dust, agricultural products and surface water) of recently deposited Pb and of Pb that was deposited in the past. Consequently, this is a multipathway risk assessment in which we are considering both airborne Pb, as it contributes to human exposures through direct inhalation of particles containing Pb, and also Pb that has deposited from air to dusts, soil and other environmental media and that contributes to human exposures through ingestion. Further, we are considering that Pb, once deposited, may be resuspended in the air, contributing to human inhalation exposures or, upon redeposition, to human ingestion exposures. Thus, as illustrated in Figure 1-1, pathways that are directly relevant to a review of the NAAQS include both newly emitted Pb from currently operating sources, and Pb emitted in the past, either from currently operating sources or historic sources, which are collectively referred to as "policy-relevant sources".



*Policy-relevant background sources and pathways are indicated by dashed lines.
**Dietary exposure should not be considered to be limited to policy-relevant background, as it reflects a combination of Pb introduced into food items during processing (policy-relevant background), as well as Pb associated with atmospheric deposition (policy-relevant sources).

Figure 1-1. Principal pathways of human and ecological exposure to Pb. Among the policy-relevant pathways, heavy arrows indicate the predominant human exposures.

Due to limited data, models, and time available, however, we are not able to fully and completely characterize in our risk assessment all of the various complexities associated with Pb. Consequently, in our efforts to focus on and characterize risk associated with the ambient air-related[3] sources and exposures, we have made a number of simplifying assumptions in a number of areas. For example, Figure 1-1 illustrates that people are also exposed to Pb that originates from nonair sources, including leaded paint or drinking water distribution systems. For purposes of this assessment, the Pb from these nonair sources is collectively referred to as "policy-relevant background"[4]. Although Pb in diet and drinking water sources may derive from Pb emitted into

[3] Ambient air related sources are those emitting Pb into the ambient air (including resuspension of previously emitted Pb), and ambient air related exposures include inhalation of ambient air Pb as well as ingestion of Pb deposited out of the air (e.g., onto outdoor soil/dust or indoor dust).

[4] This categorization of policy-relevant sources and background exposures is not intended to convey any particular policy decision at this stage regarding the Pb standard. Rather, it is simply intended to convey an area of interest to this review.

the ambient air, the contribution from air pathways to these exposure pathways is not explicitly recognized, such that these exposures are treated as policy-relevant background.

1.2 RISK ASSESSMENT FROM LAST REVIEW

In the risk assessment conducted in support of the last review, air quality scenarios were compared in terms of their impact on the percentage of modeled populations that exceeded specific blood Pb levels chosen with consideration of the health effects evidence at that time (USEPA, 1986a, 1986b, 1990a). The 1990 analysis focused on both children (birth through 7 years of age) and middle-aged men residing in three case study locations, two near secondary Pb smelters and one near a primary Pb smelter (USEPA, 1990b). The analysis also introduced the use of pharmacokinetic blood Pb modeling for children, although it used empirically derived slope models for adult men to relate changes in air Pb to changes in blood Pb.

In the 1990 Staff Paper, staff concluded that at levels of 10-15 micrograms per deciliter (μg/dL) of blood Pb, there appeared to be "a convergence of evidence of lead-induced interference with diverse set of physiological functions and processes, particularly evident in several independent studies showing impaired neurobehavioral function and development" (USEPA, 1990).[5] Accordingly, the staff used blood Pb levels of 10 and 15 μg/dL to evaluate effects of alternate NAAQS on children in the 1990 analysis (USEPA, 1990). These values were chosen with consideration of the full body of health effects evidence at that time. Staff then used dispersion modeling (the Industrial Source Complex (ISC) model) combined with source characterization data to generate Pb air concentrations for each case study area. Statistically derived relationships based on data from other industrial locations, including Pb smelters, that linked concentrations of Pb in air to Pb in indoor dust and outdoor soil were then used to predict Pb in these media for the three case study locations, based on the modeled air Pb concentrations. An uptake/biokinetic model was also developed to predict child blood Pb levels. This model was used in place of a statistical regression slope model to allow consideration of the dynamic nature of Pb exposure in children. EPA combined model-derived central tendency blood Pb levels with an estimated geometric standard deviation (GSD) reflecting interindividual variability in blood Pb levels, to generate population distributions of blood Pb levels. These distributions were then used to estimate the percentage of children at each case study location that exceeded the blood Pb levels 10 and of 15 μg/dL, respectively.

For adult men, the 1990 assessment used blood Pb levels of 10 and 12 μg/dL to compare relative effects of alternate NAAQS (USEPA, 1990). The same approach was used for

[5] As a result of a parallel activity, the U.S. Centers for Disease Control and Prevention in 1991 reduced the children's blood Pb level warranting individual intervention to 15 μg/dL and identified a level of 10 μg/dL for implementing community-wide prevention activities (CDC, 1991; CDC, 2005).

generating media concentrations for the adult analysis as was used for the child assessment. However, rather than using a biokinetic model, as for the children's assessment, the 1990 analysis for adults used statistically derived slope models to relate air Pb to blood Pb levels with two versions of the slope models being employed: (a) the aggregate model which predicts blood Pb in adults based solely on air Pb levels (here a single slope factor captures both the direct inhalation pathway as well as the more complex pathway of Pb deposition to soil and dust followed by incidental ingestion) and (b) the disaggregate model which uses media-specific slopes to predict blood Pb based on Pb concentrations in soil, dust and air. Since the projected blood Pb levels were mean population levels, a GSD term was included to develop population-level blood Pb distributions. The GSD estimates for adults and children were derived from information on observed blood Pb levels in these subgroups. These population-level distributions were then queried to identify the percentage of adult men at each case study location with modeled blood Pb levels exceeding the levels of interest for adults (10 and 12 μg/dL).

The primary difference between the risk assessment approach used in the current pilot analysis and the assessment completed in 1990 involves the risk metric employed, which reflects the quantitative and qualitative health effects evidence available today that was not available in 1990 (CD). Rather than estimating the percentage of study populations with exposures above blood Pb levels of interest as was done in the last review (i.e., 10, 12 and 15 μg/dL), the current pilot analysis estimates the degree of health decrement in study populations exposed to Pb. Specifically, the pilot analysis estimates the distribution of IQ loss associated with Pb exposure for child populations at each of the case study locations with that IQ loss further differentiated between background Pb exposure and policy-relevant exposures.

1.3 PILOT PHASE ASSESSMENT FOR THE CURRENT REVIEW

The pilot phase of the risk assessment for the current review is described in the first draft Staff Paper and accompanying technical report (USEPA 2006a; ICF 2006). The pilot assessment was intended primarily as a demonstration of the risk assessment methodology being developed for the current review. Consequently, exposure and risk results from the pilot assessment are considered preliminary.

The pilot assessment included three case studies: (a) a primary Pb smelter (in Herculaneum, Missouri), (b) a secondary Pb smelter (in Troy, Alabama), and, (c) a near roadway (urban) location along a short road segment in Houston, Texas.[6] The case studies modeled for

[6] This case study was intended to provide perspective on the near roadway exposure scenario but was not intended to estimate total population risk for a full urban or metropolitan area.

the pilot were selected to provide a preliminary perspective on the nature and magnitude of air sourced Pb exposures and risk. In addition, they provided a range of exposure scenarios in which to test the risk assessment methodology developed for the current review. Because of differences in the exposure scenarios and available data at each of the case study locations, the approach used for modeling exposure and risk differed among the case studies. Results from the pilot assessment, as well as comments received from the public and CASAC (see Section 1.4) have informed decisions on the design for the full-scale assessment, including the types of case studies included.

1.4 CASAC ADVICE

The staff consulted with the CASAC on the draft analysis plan for the risk assessment (USEPA, 2006c) in June 2006 (Henderson, 2006), and subsequently developed the pilot assessment, summarized in Section 1.3, and described in the first draft Staff Paper and accompanying technical report (USEPA, 2006a; ICF 2006). On February 6-7, 2007, the CASAC Pb panel met to discuss these documents and CASAC's written comments and recommendations were provided in March 2007 (Henderson, 2007a).

Consistent with their mandate under the Clean Air Act, CASAC provided comments on both scientific aspects of the risk assessment and aspects related to the standards themselves (Henderson, 2007a).[7] With regard to the risk assessment, they recommended that the case study approach implemented for the pilot risk assessment be supplemented with a "population-based" analysis, and, in discussion at the public meeting, the panel raised the general occurrence of lower Pb levels in urban areas beyond more point source impacted areas as being an important focus for the risk assessment. As described below, consideration of comments in this area led to a significant difference in the design of the full-scale assessment as compared to the pilot assessment.

CASAC also recommended that uncertainty be characterized with regard to the relationship between a change in the NAAQS and the distribution of population blood Pb concentrations, and with regard to the relationship between blood Pb concentrations and the risk of adverse health effects (Henderson, 2007a). With respect to alternate NAAQS for consideration by EPA, CASAC recommended consideration of levels less than or about 0.2 $\mu g/m^3$ (micrograms per cubic meter) and of a monthly averaging time. Additionally, they indicated that they consider a population loss of 1-2 points in intelligence quotient (IQ) to be highly significant from a public health perspective and that the primary Pb standard should be set

[7] Consistent with the focus of this document on the human exposure and health risk assessment, CASAC comments regarding the ecological risk assessment and secondary standard considerations are not discussed here.

to protect 99.5 percent of the population from exceeding that loss (Henderson, 2007a, p. 6).[8] CASAC also recommended conducting future Pb monitoring with samplers for particulate matter less than ten microns in size (PM_{10}) rather than with samplers for total suspended particulate matter (TSP) (Henderson, 2007a, p. 7).

In consideration of CASAC's comments on the pilot phase assessment (Henderson, 2007a), we considered a number of alternate approaches for the full-scale assessment. As a result, several additions and modifications to the assessment design were implemented for the initial analyses of the full-scale assessment presented in the July 2007 draft risk assessment report (USEPA, 2007a) and chapters 1 through 4 of this document. The most significant of these modifications was the replacement of the near roadway case study with a general urban case study. This case study was designed to provide estimates of risk in urban areas associated with broad population level exposures to different ambient air levels of Pb. The general urban case study was assessed in addition to the two point source case studies, and differs from those case studies in basing the estimate of air quality on monitoring data (rather than on results from air quality modeling). The alternate NAAQS levels were selected to overlap with the range of levels suggested by CASAC. The target population and endpoint for the assessment remains young children and risk of IQ decrements associated with Pb exposure. To address CASAC comments on the cutpoint employed in the pilot assessment (Henderson, 2007a), the blood Pb concentration response function was re-examined, and three alternatives were included in the assessment. Additionally, in consideration of CASAC recommendations regarding the geometric standard deviation used in the blood Pb modeling, two values were included in the initial analyses for the general urban case study.

In their review of the July draft risk assessment report, in addition to reiterating their previous recommendations on aspects of the standards and concurring with several aspects of the draft assessment, the CASAC Pb Panel made several recommendations for additional exposure and health risk analyses (Henderson, 2007b), of which several notable ones are summarized here. First, they recommended that the general urban case study be augmented by the inclusion of risk analyses in specific urban areas of the U.S. Further, they recommended use of the hybrid dust Pb model in preference to the alternate that had additionally been employed in the initial analyses for the general urban case study. The Panel also recommended limiting presentation of population risk results to the 5^{th} through 95^{th} percentile of the distribution.[9] In response to

[8] CASAC's reference to 99.5 percent of the population in this statement is consistent with their recognition earlier in the same letter of the 99.5 percent figure being the EPA's risk management choice when they established the Pb NAAQS in 1978 (Henderson, 2007a, pp. 5 and 6).

[9] This recommendation was in recognition by Panel members of uncertainties in quantitative estimates from the assessment for this point on the distribution (Henderson, 2007b, Appendix D).

discussion by the CASAC Pb Panel associated with these recommendations at the August public meeting, staff has developed additional analyses that include three location-specific urban case studies, and focus on a core modeling approach (with regard to dust Pb model, blood metric and concentration-response functions). With regard to the blood Pb concentration-response function, CASAC recommended use of a two-piece or dual linear function that recognizes a change in slope (to a notably higher value) at blood Pb levels of 7.5 μg/dL and indicated less favor for the two-piece linear function with hinge at 10.82 μg/dL derived for the initial analyses of the full-scale assessment (see Section 4.1.1.3) (Henderson, 2007b). Accordingly, a different set of concentration-response functions was employed in the additional analyses (see Section 5.3.1.1), and risk estimates for the two-piece linear function with hinge at 10.82 μg/dL are not included in the range of risk estimates presented for the initial analyses in Chapter 4.

1.5 ORGANIZATION OF THE DOCUMENT

The remainder of this document is organized as follows. Chapter 2 describes the design of the exposure and risk assessments, covering such topics as the conceptual model used in designing the analysis (Section 2.1), the case studies included in the assessment (Section 2.2), the air quality scenarios simulated in the assessment (Section 2.3), and an overview of the analytical approach (Section 2.4). Chapter 3 describes the methods and results for the exposure assessment, as well as the performance evaluation. Chapter 4 describes the methods for deriving risk estimates, the resultant estimates, sensitivity analyses and a characterization of uncertainty. Chapter 5 presents additional analyses using a core modeling approach completed in response to the August 2007 CASAC review of initial full-scale analyses.

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2 DESIGN OF EXPOSURE AND RISK ASSESSMENTS

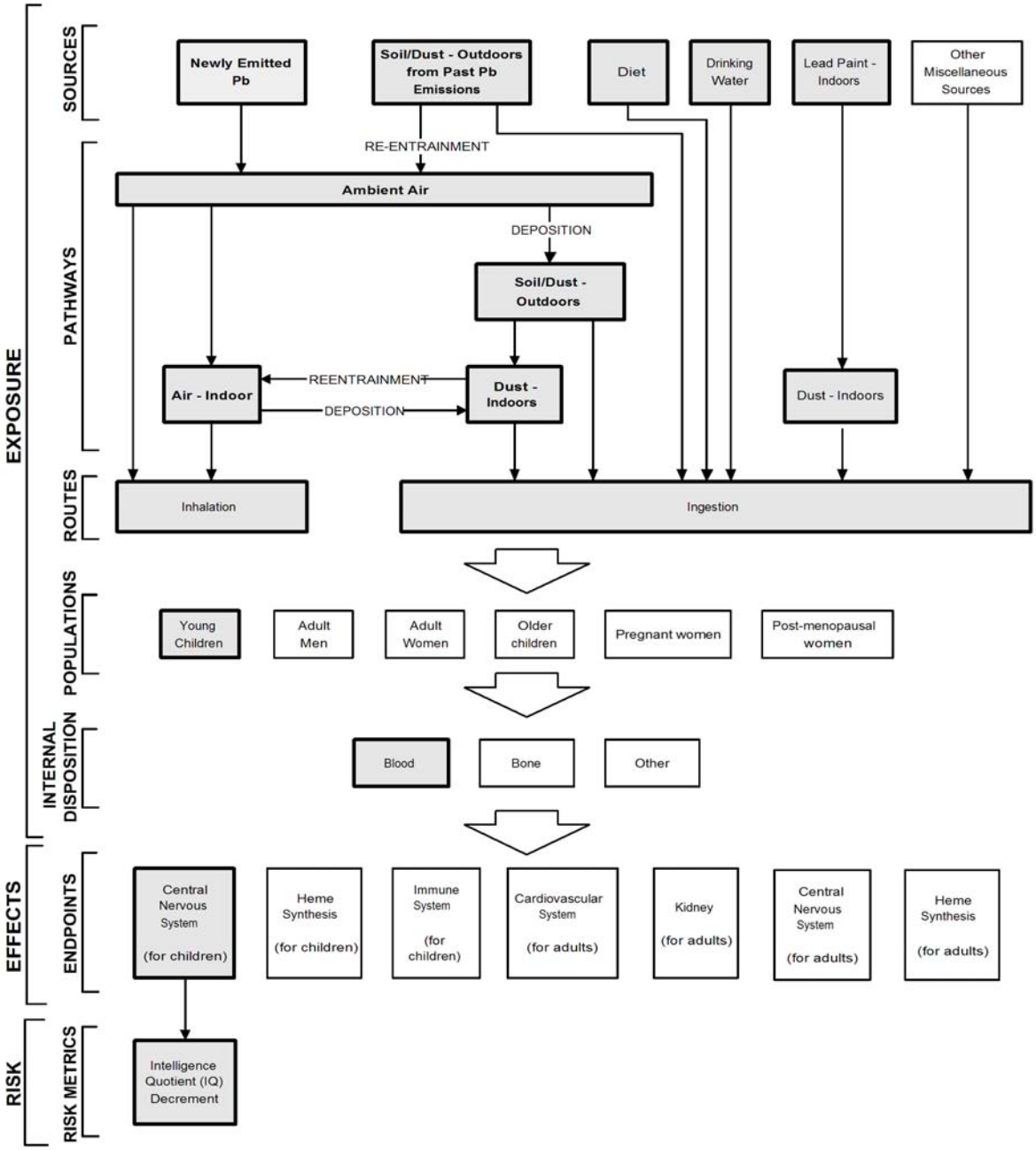
The risk assessment design relies on the use of case studies. The types of case studies included, as well as the analytical aspects of the assessment of each, reflect consideration of the evidence presented in the CD, air quality analyses, and findings of the pilot assessment (Section 1.3), as well as comments received from CASAC (Section 1.4) and the public.

Drawing primarily from the CD, Section 2.1 provides background for the risk assessment, with regard to key elements of Pb exposure and effects. The assessment scenarios evaluated in the assessment are described in Section 2.2. Background information on the three case studies is described in Section 2.3. Section 2.4 describes the analytical approach, with attention to key analytical steps, and discussion of temporal and spatial aspects of the assessment, as well as the categorization of policy-relevant exposure pathways, and the uncertainty characterization.

2.1 BACKGROUND INFORMATION ON LEAD EXPOSURE AND RISK

As recognized in Section 1.1, there are a variety of complexities associated with the assessment of air-related Pb exposure and risk. In this risk assessment, we have attempted to focus effort on those aspects that are most important and feasible to address within our scope and given the constraints of time, pertinent data, models, etc. With regard to some aspects, simplifying assumptions have been implemented. The following subsections describe elements of Pb exposure and effects pertinent to evaluating public health risks associated with Pb from ambient air, and specify those that are explicitly addressed in this quantitative risk assessment. 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 identified in bold text.

Figure 2-1. Conceptual model for Pb human health risk assessment.



Note: Boxes outlined in bold are included in the quantitative risk assessment. Sources and pathways for which ambient air has played a role are in bold text.

2.1.1 Sources, Pathways and Routes

As described in Section 1.1, for the purposes of this assessment, policy-relevant sources (in Figure 2-1 in bold type) include both sources of new Pb emissions (e.g., from active stationary and mobile sources) and re-emission or resuspension of historically deposited Pb (e.g., near roadways or associated with now inactive, or now lower emitting stationary sources, as discussed in Appendix A, Section A.1.1.3).

There are more than 13,000 individual sources in the U.S. for which we have estimated Pb emissions to the air (Appendix A, Section A.1.2). Cumulatively, those sources, in addition to mobile sources and other sources not individually quantified, emit some 1600 tons per year (tpy) of Pb in the U.S. (Appendix A). The largest categories (in terms of aggregate national emissions) include mobile sources (specifically combustion of leaded general aviation fuel), boilers and process heaters, and metals processes, such as primary and secondary Pb smelting. Of these, metals processing industries are among the largest emitters of Pb, in terms of emissions from individual facilities (Appendix A, Section A.1). Another potentially large category of Pb emissions, for which we do not have quantitative estimates in our national emissions inventory, is resuspension of recent and historically deposited Pb (Appendix A, Section A.1.1.3; CD, Section 2.3.3). Studies of emissions in southern California suggest that Pb in resuspended road dust may represent up to 40%-90% of Pb emissions in some areas (Appendix A, Section A.1.1.3). Resuspension is represented to differing degrees in the three case studies included in the risk assessment (Sections 2.2.1 and 2.2.2)

Lead in outdoor dust and soil may be derived from a range of sources including current and historical air emissions sources, as well as miscellaneous nonair sources (e.g., land disposal of wastes and subsequent weathering). Outdoor dust and soil may play a substantial role in human exposures, particularly for children (CD, Section 3.2). Additionally, Pb in house dust, which may be derived from Pb in outdoor dust and soil as well as from ambient air Pb (including previously deposited Pb resuspended into ambient air), is another source of children's exposure (CD, Sections 3.2 and 4.4). For example, blood Pb levels in children have been shown to be particularly influenced by exposures to Pb in dust (e.g., Lanphear and Roghmann 1997; Lanphear et al., 1998). Such findings and "other studies of populations near active sources of air emissions (e.g., smelters), substantiate the effect of airborne Pb and resuspended soil Pb on interior dust and blood Pb" (CD, p. 8-22).

In addition to airborne emissions (recent or those in the past), sources of Pb to the environment or to human exposure included old leaded paint, Pb in drinking water and Pb in the diet (Figure 1-1). As mentioned in Section 1.1, Pb in diet and drinking water may have air pathway related (i.e., policy-relevant) contributions as well as contributions from policy-relevant

background (e.g., Pb solder on water distribution pipes and Pb in materials used in food processing). Limitations in our data and modeling tools have handicapped our ability to separate these contributions in the risk assessment, such that we have labeled diet and drinking water policy-relevant background. Consequently, these sources of Pb exposure are depicted in Figure 2-1 as policy-relevant background (in nonbold type), although this is not intended to convey any particular policy decision at this stage regarding the Pb standard. Policy-relevant pathways (bold text in Figure 2-1) 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.

Human exposure to environmental Pb occurs predominantly via ingestion and inhalation routes, with ingestion (including incidental ingestion of dust and soil) recognized as generally playing a larger role for the general human population (CD, Section 4.5). The dermal route is relatively less well characterized but is not considered to play a large role in total Pb exposure (CD, Section 4.5), and is not included in this assessment (Figure 2-1).

2.1.2 At-risk Populations

In considering populations for inclusion in the risk assessment, we considered evidence regarding those with increased susceptibility (i.e., physiological factors contributing to a greater response for the same exposure), and those with increased exposure (including that resulting from behavior leading to increased contact with contaminated media). A behavioral factor of great impact on Pb exposure is the incidence of hand-to-mouth activity that is prevalent in very young children (CD, Section 4.4.3). Physiological factors include both conditions contributing to a subgroup's increased risk of effects at a given blood Pb level, and those that contribute to blood Pb levels higher than those otherwise associated with a given Pb exposure (CD, Section 8.5.3). An additional population characterization for which evidence was considered was vulnerability to pollution-related effects which additionally encompasses situations of elevated exposure, such as residing in old housing with Pb-containing paint or near sources of ambient Pb, as well as socioeconomic factors, such as reduced access to health care or low socioeconomic status (SES) (USEPA, 2003, 2005) that can contribute to increased risk of adverse health effects from Pb.

Three particular physiological factors contributing to increased risk of Pb effects at a given blood Pb level are recognized in the CD (e.g., CD, Section 8.5.3): age, health status, and genetic composition (or genotype). With regard to age, the susceptibility of young children to the neurodevelopmental effects of Pb is well recognized (e.g., CD, Sections 5.3, 6.2, 8.4, 8.5, 8.6.2), although the specific ages of vulnerability have not been established (CD, pp 6-60 to 6-64). Early childhood may also be a time of increased susceptibility for Pb immunotoxicity (CD,

Sections 5.9.10, 6.8.3 and 8.4.6), and childhood exposures have been associated with increased risk of cardiovascular and neurodegenerative effects in adulthood (CD, p. 8-74). Health status is another physiological factor in that subpopulations with pre-existing health conditions may be more susceptible (as compared to the general population) for particular Pb-associated effects, with this being most clear for renal and cardiovascular outcomes. For example, African Americans as a group, have a higher frequency of hypertension than the general population or other ethnic groups (NCHS, 2005), and as a result may face a greater risk of adverse health impact from Pb-associated cardiovascular effects. A third physiological factor relates to genetic polymorphisms. That is, subpopulations defined by particular genetic polymorphisms (e.g., presence of the δ-aminolevulinic acid dehydratase-2 [ALAD-2] allele) have also been recognized as sensitive to Pb toxicity, which may be due to increased susceptibility to the same internal dose and/or to increased internal dose associated with same exposure (CD, p. 8-71, Sections 6.3.5, 6.4.7.3 and 6.3.6).

Several physiological factors pertain to susceptibility by contributing to increased blood Pb levels (i.e., increased internal dose levels) over those otherwise associated with a given Pb exposure (CD, Section 8.5.3). These include nutritional status, which plays a role in Pb absorption from the GI tract (CD, Section 5.10.2.5); polymorphism for the vitamin D receptor, which studies suggest may contribute to increased Pb absorption from the GI tract (CD, Section 8.4.2.7); presence of the ALAD-2 allele, which studies suggest contribute to increased blood Pb levels (Section 8.5.3); and bone demineralization, such as occurs during pregnancy, lactation, and aging, which appears to influence Pb release from bone into the blood (CD, Section 4.3.2).

In summary, there are a variety of ways in which Pb exposed populations might be characterized and stratified for the purposes of health risk assessment. Age or lifestage is used to distinguish potential groups on which to focus the risk assessment (see Figure 2-1) in recognition of its influence on exposure and susceptibility. In consideration of the health effects evidence regarding endpoints of greatest public health concern and a recognition of effects on the developing nervous system as a sentinel endpoint for public health impacts of Pb (see Section 2.1.4), young children have been selected as the priority population for this risk assessment (see Figure 2-1). We recognize, however, other population subgroups as described above may also be at risk of Pb-related health effects of public health concern at similar or higher exposures. As currently available data do not generally support quantitative modeling that differentiates blood Pb levels and associated health risk within a particular population group such as young children on the basis of enhanced or reduced susceptibility to Pb effects (e.g., concentration response functions for IQ loss that differentiate between populations that are calcium deficient and those that are not), the assessment does not develop separate risk estimates for such subpopulations of

young children. In the risk assessment, however, interindividual variability in blood Pb is quantitatively considered (Section 3.2.3).

2.1.3 Internal Disposition

Once inhaled or ingested and absorbed into the blood stream, Pb is distributed throughout the body via the blood, with bone being the predominant site of Pb accumulation and storage in the body (CD, Sections 4.2 and 4.3). Additionally, the epidemiologic evidence indicates that Pb freely crosses the placenta resulting in continued fetal exposure throughout pregnancy, and that exposure increases during the later half of pregnancy (CD, Section 6.6.2).

During childhood development, bone represents approximately 70% of a child's body burden of Pb, and this accumulation continues through adulthood, when more than 90% of the total Pb body burden is stored in the bone (CD, Section 4.2.2). Accordingly, levels of Pb in bone are indicative of a person's long-term, cumulative exposure to Pb. In contrast, blood Pb levels are usually indicative of recent exposures. Depending on exposure dynamics, however, blood Pb may – through its interaction with bone - be indicative of past exposure or of cumulative body burden (CD, Section 4.3.1.5).

Throughout life, Pb in the body is exchanged between blood and bone, and between blood and soft tissues (CD, Section 4.3.2), with variation in these exchanges reflecting "duration and intensity of the exposure, age and various physiological variables" (CD, p. 4-1). For example, resorption of bone (e.g., in pregnant or nursing women, and associated with osteoporosis in postmenopausal women or, to a lesser magnitude, in older men) results in a mobilization of Pb from bone into circulation (CD, Sections 4.3.2.4 and 4.3.2.5). Past exposures that contribute Pb to the bone, consequently, may influence current levels of Pb in blood. Where past exposures were elevated in comparison to recent exposures, this influence may complicate interpretations with regard to recent exposure (CD, Sections 4.3.1.4 to 4.3.1.6). That is, higher blood Pb concentrations may be indicative of higher cumulative exposures or of a recent elevation in exposure (CD, pp. 4-34 and 4-133).

Bone measurements, as a result of the generally slower Pb turnover in bone, are recognized as providing a better measure of cumulative Pb exposure (CD, Section 8.3.2). The bone pool of Pb in children, however, is thought to be much more labile than that in adults due to the more rapid turnover of bone mineral as a result of growth (CD, p. 4-27). As a result, changes in blood Pb concentration in children more closely parallel changes in total body burden (CD, pp. 4-20 and 4-27). This is in contrast to adults, whose bone has accumulated decades of Pb exposures (with past exposures often greater than current ones), and for whom the bone may be a significant source long after exposure has ended (CD, Section 4.3.2.5).

Given the association with exposure, particularly recent exposure, and the relative ease of collection, blood Pb levels are extensively used as an index or biomarker of exposure by national and international health agencies (CD, Section 4.3.1.5). Although recent methods are making bone Pb measurements easier to collect (CD, Section 4.3.2.2) and consequently, their use more widespread, epidemiological and toxicological studies of Pb health effects and dose-response relationships tend to be dominated by blood Pb as the exposure metric (CD, Sections 4.3.1.3, 8.3.2 and Chapter 5).

Accordingly, blood Pb level is the index of exposure or exposure metric in this risk assessment. The use of concentration-response functions that rely on blood Pb (e.g., rather than ambient Pb concentration) as the exposure metric reduces uncertainty in the causality aspects of Pb risk estimates. The relationship between specific sources and pathways of exposure and blood Pb level is needed, however, in order to identify the specific risk contributions associated with those sources and pathways of greatest interest to this assessment (i.e., those related to Pb emitted into the air). For example, the blood Pb-response relationships developed in epidemiological studies of Pb-exposed populations do not distinguish among different sources or pathways of Pb exposure (e.g., inhalation, ingestion of indoor dust, ingestion of dust containing leaded paint). In the exposure assessment for this review, models that estimate blood Pb levels associated with Pb exposure (e.g., CD, Section 4.4) are used to inform estimates of contributions to blood Pb arising from ambient air related Pb *versus* contributions from other sources.

2.1.4 Health Endpoints

Lead has been demonstrated to exert "a broad array of deleterious effects on multiple organ systems via widely diverse mechanisms of action" (CD, p. 8-24 and Section 8.4.1). This array of health effects, the evidence for which is comprehensively described in the CD, includes

- Heme biosynthesis and related functions;
- Neurological development and function;
- Reproduction and physical development;
- Kidney function;
- Cardiovascular function; and,
- Immune function.

There is also some evidence of Pb carcinogenicity, primarily from animal studies, with limited human evidence of suggestive associations (CD, Sections 5.6.2, 6.7, and 8.4.10). [1]

[1] Lead has been classified as a probable human carcinogen by the International Agency for Research on Cancer, based mainly on sufficient animal evidence, and as reasonably anticipated to be a human carcinogen by the

This review is focused on those effects most pertinent to ambient exposures. Given the reductions in ambient Pb levels over the past 30 years, these effects are generally those associated with the lowest Pb levels of exposure. These are neurological, hematological and immune effects for children, and neurological, hematological, cardiovascular and renal effects for adults (CD, Tables 8-5 and 8-6), with neurological effects in children and cardiovascular effects in adults appearing to be of greatest public health concern (CD, p. 8-60). The toxicological and epidemiological information available since the time of the last review "includes assessment of new evidence substantiating risks of deleterious effects on certain health endpoints being induced by distinctly lower than previously demonstrated Pb exposures indexed by blood Pb levels extending well below 10 μg/dL in children and/or adults" (CD, p. 8-25). The CD indicates some health effects associated with blood Pb levels that extend below 5 μg/dL, with some studies observing these effects at the lowest blood levels considered (i.e., threshold levels for these effects cannot be discerned from the currently available studies).

The endpoints identified above are important considerations for this review and are described briefly in sections below, with detailed discussion of the evidence presented in the CD. Of these health endpoints, the focus of the quantitative health risk assessment is developmental neurotoxicity in children, with IQ decrement as the risk metric (Figure 2-1), described in Section 2.1.5

2.1.4.1 Developing Nervous System

The nervous system has long been recognized as a target of Pb toxicity, with the developing nervous system affected at lower exposures than the mature system (CD, Sections 5.3, 6.2.1, 6.2.2, and 8.4). While blood Pb levels in U.S. children ages one to five years have decreased notable since the late 1970s, newer studies have investigated and reported associations of effects on the neurodevelopment of children with these more recent blood Pb levels (CD, Chapter 6). Functional manifestations of Pb neurotoxicity during childhood include sensory, motor, cognitive and behavioral impacts. Numerous epidemiological studies have reported neurocognitive, neurobehavioral, sensory, and motor function effects in children at blood Pb levels below 10 μg/dL (CD, Section 6.2). Further, "extensive experimental laboratory animal evidence has been generated that (a) substantiates well the plausibility of the epidemiologic findings observed in human children and adults and (b) expands our understanding of likely mechanisms underlying the neurotoxic effects" (CD, p. 8-25; Section 5.3).

U.S. National Toxicology Program (CD, Section 6.7.2). U.S. EPA classified it in the past as a probable carcinogen (http://www.epa.gov/iris/subst/0277.htm).

Cognitive effects associated with Pb exposures that have been observed in epidemiological studies have included decrements in intelligence test results, such as the widely used IQ score, and in academic achievement as assessed by various standardized tests as well as by class ranking and graduation rates (CD, Section 6.2.16 and pp 8-29 to 8-30). As noted in the CD with regard to the latter, "Associations between Pb exposure and academic achievement observed in the above-noted studies were significant even after adjusting for IQ, suggesting that Pb-sensitive neuropsychological processing and learning factors not reflected by global intelligence indices might contribute to reduced performance on academic tasks" (CD, pp 8-29 to 8-30).

Other cognitive effects observed in studies of children have included effects on attention, executive functions, language, memory, learning and visuospatial processing (CD, Sections 5.3.5, 6.2.5 and 8.4.2.1), with attention and executive function effects associated with Pb exposures indexed by blood Pb levels below 10 μg/dL (CD, Section 6.2.5 and pp. 8-30 to 8-31). The evidence for the role of Pb in this suite of effects includes experimental animal findings (discussed in CD, Section 8.4.2.1; p. 8-31), which provide strong biological plausibility of Pb effects on learning ability, memory and attention (CD, Section 5.3.5), as well as associated mechanistic findings. Further, Pb-induced deficits observed in animal and epidemiological studies, for the most part, have been found to be persistent in the absence of markedly reduced environmental exposures (CD, Sections 5.3.5, 6.2.11, and 8.5.2). It is additionally important to note that there may be long-term consequences of such deficits over a lifetime. Poor academic skills and achievement can have "enduring and important effects on objective parameters of success" later in life, as well as increased risk of antisocial and delinquent behavior (CD, Section 6.2.16).

Other neurological effects associated with Pb exposures indexed by blood Pb levels near or below 10 μg/dL include behavioral effects, such as delinquent behavior (CD, Sections 6.2.6 and 8.4.2.2), sensory effects, such as those related to hearing and vision (CD, Sections 6.2.7, 7.4.2.3 and 8.4.2.3), and deficits in neuromotor function (CD, p. 8-36).

Neurocognitive impact, specifically decrement in IQ in young children, is a focus of the quantitative risk assessment due to the strength of evidence for association with blood Pb levels below 10 μg/dL, and the strength of the dose-response information at these exposure levels.

As discussed in the CD (Section 8.4.2) and by Rice (1996), while there is no direct animal test parallel to human IQ tests, "in animals a wide variety of tests that assess attention, learning, and memory suggest that Pb exposure {of animals} results in a global deficit in functioning, just as it is indicated by decrements in IQ scores in children" (CD, p. 8-27). The animal and epidemiological evidence for this endpoint are consistent and complementary (CD, p. 8-44). As stated in the CD (p. 8-44):

Findings from numerous experimental studies of rats and of nonhuman primates, as discussed in Chapter 5, parallel the observed human neurocognitive deficits and the processes responsible for them. Learning and other higher order cognitive processes show the greatest similarities in Pb-induced deficits between humans and experimental animals. Deficits in cognition are due to the combined and overlapping effects of Pb-induced perseveration, inability to inhibit responding, inability to adapt to changing behavioral requirements, aversion to delays, and distractibility. Higher level neurocognitive functions are affected in both animals and humans at very low exposure levels (≤10 μg/dL), more so than simple cognitive functions.

Further, "epidemiologic studies of Pb and child development have demonstrated inverse associations between blood Pb concentrations and children's IQ and other outcomes at successively lower Pb exposure levels" over the past 30 years (CD, p. 6-64). This is supported by multiple studies performed over the past 15 years (see CD, Section 6.2.13), with particularly compelling evidence for decrements in IQ at blood Pb levels below 10 μg/dL provided by a recent international pooled analysis of seven prospective studies (Lanphear et al., 2005; CD, Section 6.2.13). For example, this pooled analysis estimated a decline of 6.2 points (with a 95% confidence interval bounded by 3.8 and 8.6) in full scale IQ occurring between approximately 1 and 10 μg/dL blood Pb level, measured concurrent with the IQ test (CD, p. 6-76). This analysis (Lanphear et al., 2005) is relied upon in the quantitative risk assessment for this endpoint.

2.1.4.2 Adult Nervous System

The nervous system has long been recognized as a target of Pb toxicity (CD Sections 5.3.1, 8.4.2). For example, those chronically exposed in the workplace are at risk for various neurological effects including peripheral sensory nerve impairment, visuomotor and memory impairment, and postural sway abnormalities, with a blood Pb concentration >14 μg/dL being a possible threshold (CD, p. 6-87). Past occupational exposure also increases the risk of developing amyotrophic lateral sclerosis and motor neuron disease (CD, Section 6.3.5 and p. 6-87). Essential tremor is also associated with Pb exposures, particularly for those with genetic susceptibility (CD, Sections 6.3.5 and 6.3.6 and p. 6-86).

In elderly populations, significant associations have been reported between bone Pb levels and impaired cognitive performance or dysfunction (CD, Section 6.3.3 and 6.3.3.1), but not with blood Pb levels, perhaps indicating a role of cumulative and/or past Pb exposures (CD, p. 6-83). During demineralization of bone in the elderly, Pb may be released into the blood, thus augmenting blood Pb associated with current ambient exposures (CD, Section 4.3.2.4). An increased susceptibility among the elderly to Pb effects on cognitive function is supported by animal evidence (Section 5.3.7). With lifetime exposure, senescent animals have exhibited an

increased susceptibility to Pb, due to the increased exposure from bone resorption, and an apparently greater sensitivity to the biochemical effects of Pb (CD, Section 5.3.7). Laboratory animal research in rats and monkeys also indicates a potential for cognitive function effects in the elderly to be related to physiological effects (regulation of protein thought to play a role in Alzheimer's disease) of Pb exposures in early childhood (CD, p. 5-67; Basha et al., 2005). Thus, early life exposure to Pb may contribute to neurocognitive effects later in life due to the redistribution of Pb body burden from bone to brain and by enhanced susceptibility caused by age-related degenerative changes in various organs, including brain (CD, p. 8-40).

2.1.4.3 Cardiovascular System

Epidemiologic and experimental toxicology studies provide strong support for the relationship between Pb exposure and increased adverse cardiovascular outcomes, including increased blood pressure, increased incidence of hypertension, and cardiovascular morbidity and mortality (CD, Sections 5.5, 6.5 and 8.4.3). The cardiovascular effect most frequently examined in epidemiological studies is increased systolic blood pressure in adults, which has been repeatedly associated with Pb exposure (CD, Sections 8.4.3, 8.6.3, 6.5.2.3, and 6.5.7). The association has been observed with Pb levels in bone and also, in some cohorts, with Pb in blood (including blood Pb levels below 10 μg/dL). A recent meta-analysis by Nawrot and others (2005), that included a range of blood Pb levels from 2.3 to 63.8 μg/dL, reported an association of increased systolic blood pressure and decreased diastolic pressure with increased blood Pb level, including levels below 10 μg/dL. The magnitude of change observed has considerable significance at the population level (CD, p. 8-45, Section 8.6.3). The epidemiological evidence is supported by evidence in numerous animal studies of arterial hypertension with low Pb exposures, an effect that persists in animals long after cessation of exposure (CD, Sections 5.5 and 8.4.3).

Multiple studies reporting positive associations of blood pressure and hypertension with bone Pb levels highlight the important role of cumulative past Pb exposure in development of cardiovascular health effects (Sections 6.5.2.3 and 6.5.7). A study of young adults who lived as children in an area of high Pb exposures also indicates the potential role of childhood exposure. In this study, higher bone Pb levels were associated with higher systolic and diastolic blood pressure (CD, p. 6-138), while current blood Pb levels (mean of 2.2 μg/dL) were not (CD, p. 6-124).

Systolic blood pressure exerts a strong influence on more serious cardiovascular events by its role in hypertension and its adverse cardiovascular sequelae (CD, p. 8-83). Several analyses of National Health and Nutrition Examination Survey (NHANES) cohorts, including some recently released, have collectively suggested a "significant effect of Pb on cardiovascular

mortality in the general U.S. population" (CD, p. 8-88, Sections 6.5.3.2 and 8.6.3). For example recent analyses of NHANES blood Pb data from 1976 to 1980 and 1988 to 1994 provide supportive evidence for an increased risk of cardiovascular mortality, consistent with projected likely increases in serious cardiovascular events (stroke, heart attack) resulting from Pb-induced increases in blood pressure (CD, Section 8.6.3).

2.1.4.4 Renal System

Lead nephrotoxicity is mediated by alterations in the glomerular filtration rate (CD, Sections 5.7.3 and 8.4.5). The interaction of Pb with the kidney, including occurrences and mechanisms of Pb uptake by and accumulation in the kidney, and associated cellular alterations, is well described in animal research (CD, Section 5.7). A set of screening tests involving markers of nephrotoxic effects have been established for screening individuals exposed to Pb occupationally or environmentally (CD, Section 5.7.1). In the epidemiological literature, associations between blood Pb and indicators of renal function impairment (e.g., measures of glomerular integrity, such as creatinine levels in urine) have been found at blood Pb levels extending below 10 μg/dL, to as low as ~2 to 4 μg/dL (CD, Sections 6.4.4.1.5 and 8.4.5). Associations are also observed with cumulative Pb dose, assessed via bone Pb, and longitudinal renal function decline (CD, p. 6-94), indicating the potential role of earlier exposures.

The findings for non-occupational populations since the last review provide "strong evidence that renal effects occur at much lower blood Pb levels than previously recognized" (CD, p. 6-113). These findings of lower Pb renal effects thresholds in environmental compared to occupational research may be a result of potentially larger proportions of susceptible individuals in the general population as compared to occupational cohorts (CD, p. 6-107). The data available are not sufficient to determine whether these effects are related more to current blood-Pb levels, higher levels from past exposure, or both (CD, p. 8-49).

The findings regarding Pb exposures and renal effects are of particular concern with regard to certain susceptible subpopulations. At levels of exposure in the general U.S. population overall, Pb combined with other risk factors, such as diabetes, hypertension, or chronic renal insufficiency from causes unrelated to Pb, can result in clinically relevant effects. Notably, the size of such susceptible populations is increasing in the United States due to obesity (CD, p. 6-113). That is, Pb is recognized as acting cumulatively with other renal risk factors to cause early onset of renal insufficiency and/or a steeper rate of renal function decline in individuals already at risk for renal disease (CD, p. 6-107).

2.1.4.5 Heme Synthesis

It has long been recognized that Pb exposure is associated with disruption of heme synthesis in both children and adults. The evidence regarding effects on heme synthesis and

other hematological parameters in animal and humans is strong, and includes documented quantitative relationships between exposure and effects in children and adults. Interference with heme synthesis was identified as one of the targets of low-level Pb toxicity in children during the time of the last NAAQS review (USEPA, 1990), and was the primary focus for the initial setting of the Pb NAAQS in 1978 (USEPA, 1978).

Mechanisms associated with Pb interference with heme synthesis include inhibition of the enzymes ALAD and ferrochelatase (Table 3-1; CD Sections 5.2.1, 6.9.1, 6.9.2; USEPA 1986). Inhibition of ALAD has been associated with increased blood Pb concentrations at and somewhat below 10 μg/dL, in children and adults (Tables 3-1 and 3-2; CD, Table 6-7). Blood Pb concentrations at and above approximately 15 μg/dL, in children, and 15-30 μg/dL, in adults, are associated with elevation of erythrocyte protoporphyrin (EP), and notable reductions in hemoglobin synthesis (Tables 3-1 and 3-1; CD, p. 8-47; USEPA, 1986). In the setting of the Pb NAAQS in 1978, the Agency concluded that "the state of elevated EP must be regarded as potentially adverse to the health of young children" (USEPA, 1978). Blood Pb concentrations at and above 40 ug/dL are associated with frank anemia, a clinical sign of severe Pb poisoning (CD, p. 8-47). The evidence regarding Pb disruption of heme synthesis and associated mechanisms is presented in detail in past CDs (USEPA 1986, 1977), with more recent findings, including the role of genetic polymorphisms, discussed in the current CD (Sections 8.4.4, 5.2.1, 6.9.1 and 6.9.2).

2.1.4.6 Immune System

Since the time of the last review, there has been substantial research on the immunotoxicity of Pb. As summarized in the CD, "studies across humans and a variety of animal models are in general agreement concerning both the nature of the immunotoxicity induced by Pb as well as the exposure conditions that are required to produce immunomodulation" (CD, p. 5-244, Section 5.9). Lead is distinguished from other immunotoxicants, however, by the fact that the most sensitive biomarkers of its immunotoxicity are associated with specific functional capacities that influence risk of disease, as opposed to being associated with changes in immune cell numbers or pathological changes of lymphatic system organs (CD, Section 5.9.1). The main immune system targets of Pb are macrophages and T lymphocytes, leading to a potential for increased tissue inflammation, reduced cell-mediated immunity, and increased risk of autoimmunity (See CD, Figure 5-18, Section 5.9.11). Additionally, Pb exposures in both animal and human studies are associated with increased production of IgE, an immunoglobulin involved in allergic responses and asthma (CD, Section 5.9.3.2). These effects have been reported in epidemiologic studies of children, and supported

by evidence in neonatal and juvenile animals, at blood Pb levels extending below 10 μg/dL (CD, p. 6-197 and Sections 5.9.10 and 8.4.6).

2.1.5 Metric and Model for Risk Quantitation

The health endpoint on which we focused in the quantitative health risk assessment for this review is developmental neurotoxicity in children (Section 2.1.4.1), with IQ decrement as the risk metric. Among the wide variety of health endpoints associated with Pb exposures, there is general consensus that the developing nervous system in young children is the most sensitive and that neurobehavioral effects (specifically neurocognitive deficits), including IQ decrements, appear to occur at lower blood levels than previously believed (i.e., at levels <10 μg/dL). For example, the overall weight of the available evidence, described in the CD, provides clear substantiation of neurocognitive decrements being associated in young children with blood Pb levels in the range of 5 to 10 μg/dL, and some analyses indicate Pb effects on intellectual attainment of young children ranging from 2 to 8 μg/dL (CD, Sections 6.2, 8.4.2 and 8.4.2.6). That is, while blood Pb levels in U.S. children ages one to five years have decreased notably since the late 1970s, newer studies have investigated and reported associations of effects on the neurodevelopment of children with these more recent blood Pb levels (CD, Chapter 6).

The evidence for neurotoxic effects in children is a robust combination of epidemiological and toxicological evidence (CD, Sections 5.3, 6.2 and 8.5). The epidemiological evidence is strongly supported by animal studies that substantiate the biological plausibility of the associations, and provides an understanding of mechanisms of action for the effects (CD, Section 8.4.2). The selection of children's IQ for the quantitative risk assessment reflects consideration of the evidence presented in the CD as well as advice received from CASAC (Henderson, 2006, 2007a).

The epidemiological studies that have investigated blood Pb effects on IQ (see CD, Section 6.2.3) have considered a variety of specific blood Pb metrics, including: 1) blood concentration "concurrent" with the response assessment (e.g., at the time of IQ testing), 2) average blood concentration over the "lifetime" of the child at the time of response assessment (e.g., average of measurements taken over child's first 6 or 7 years), 3) peak blood concentration during a particular age range and 4) early childhood blood concentration (e.g., the mean of measurements between 6 and 24 months age). All four specific blood Pb metrics have been correlated with IQ (see CD, p. 6-62; Lanphear et al., 2005). In the international pooled analysis by Lanphear and others (2005), however, the concurrent and lifetime averaged measurements were considered "stronger predictors of lead-associated intellectual deficits than was maximal measured (peak) or early childhood blood lead concentrations," with the concurrent blood Pb level exhibiting the strongest relationship (CD, p. 6-29). It is not clear in this case or for similar

findings in other studies, whether the cognitive deficits observed were due to Pb exposure that occurred during early childhood or were a function of concurrent exposure. Nevertheless, concurrent blood Pb levels likely reflected both ongoing exposure and preexisting body burden (CD, p. 6-32).

Using concurrent blood Pb level as the exposure metric and IQ as the response from the pooled dataset of seven international studies, Lanphear and others (2005) employed mathematical models of various forms, including linear, cubic spline, log-linear, and piece-wise linear, in their investigation of the blood Pb concentration-response relationship (CD, p. 6-29; Lanphear et al., 2005). They observed that the shape of the concentration-response relationship is nonlinear and the log-linear model provides a better fit over the full range of blood Pb measurements than a linear one (CD, p. 6-29 and pp. 6-67 to 6-70; Lanphear et al., 2005). In addition, they found that no individual study among the seven was responsible for the estimated nonlinear relationship between Pb and deficits in IQ (CD p. 6-30). Others have also analyzed the same dataset and similarly concluded that, across the range of the dataset's blood Pb levels, a log-linear relationship was a significantly better fit than the linear relationship ($p=0.009$) with little evidence of residual confounding from included model variables (CD, Section 6.2.13; Rothenberg and Rothenberg, 2005).

A nonlinear blood Pb concentration-response relationship is also suggested by several other studies that have observed that each μg/dL increase in blood Pb may have a greater effect on IQ at blood Pb levels below 10 μg/dL than at higher levels (CD, pp. 8-63 to 8-64). While this may at first seem at odds with certain fundamental toxicological concepts, a number of examples of non- or supralinear dose-response relationships exist in toxicology (CD, pp. 6-76 and 8-83 to 8-89).[2] With regard to the effects of Pb on neurodevelopmental outcome such as IQ, the CD suggests that initial neurodevelopmental effects at lower Pb levels may be disrupting very different biological mechanisms (e.g., early developmental processes in the central nervous system) than more severe effects of high exposures that result in symptomatic Pb poisoning and frank mental retardation (CD, p. 6-76). In comparing across the individual studies and the pooled analysis, it is observed that at higher blood Pb levels, the slopes derived for log-linear and linear models are almost identical, and for studies with lower blood Pb levels, the slopes appear to be steeper than those observed in studies involving higher blood Pb levels (CD, p. 8-78, Figure 8-7).

Given the evidence summarized here and described in detail in the CD (Chapters 6 and 8), and in consideration of CASAC recommendations (Henderson, 2006, 2007a, 2007b), the risk

[2] Similarly, a nonlinear concentration-response relationship was observed for the relationship between blood Pb levels and blood pressure in adults (CD, pp. 8-83 to 8-89).

assessment for this review relies on the functions presented by Lanphear and others (2005) that relate absolute IQ as a function of concurrent blood Pb or of the log of concurrent blood Pb, and lifetime average blood Pb, respectively. As discussed above, the slope of the concentration-response relationship described by these functions is greater at the lower blood Pb levels (e.g., less than 10 μg/dL). The impact of the nonlinear slope is illustrated by the estimates of IQ decrements associated with increases in blood IQ for different ranges of blood Pb level reported for the log-linear model (Lanphear et al., 2005). These estimates were IQ decrements of 3.9 (with 95% confidence interval, CI, of 2.4-5.3), 1.9 (95% CI, 1.2-2.6) and 1.1 (95% CI, 0.7-1.5), for increases in concurrent blood Pb from 2.4 to 10 μg/dL, 10 to 20 μg/dL, and 20 to 30 μg/dL, respectively (Lanphear et al., 2005). For an increase in concurrent blood Pb levels from <1 to 10 ug/dL, the log-linear model estimates a decline of 6.2 points in full scale IQ which is comparable to the 7.4 point decrement in IQ for an increase in lifetime mean blood Pb levels up to 10 ug/dL observed in the Rochester study (CD, pp 6-30 to 6-31).

Several studies have examined the relationship of IQ decrement with blood Pb, quantified by a variety of metrics, at lower blood Pb levels. On a change in IQ per μg/dL basis, estimates of IQ decrement associated with blood Pb levels (concurrent, 24-month, peak, lifetime average or lifetime cumulative) below 10 μg/dL range from -0.4 to -1.8 (CD, Table 8.7). At the upper end of this range are the slopes derived for the subsets of children in the Rochester and Boston cohorts for which peak blood Pb levels were <10 μg/dL; these slopes are -1.8 (for concurrent blood Pb influence on IQ) and -1.6 (for 24-month blood Pb influence on IQ), respectively. The number of children in these low blood Pb subsets of the Rochester and Boston cohorts are 101 and 48, respectively. A similar stratification of the pooled dataset by Lanphear and others (2005) yielded a slope for the linear function of IQ change associated with concurrent blood Pb of -0.8 for the subset of the children in the pooled data set for which maximal or peak blood Pb levels were below 10 μg/dL. Of the 1333 children in the full pooled dataset, there were 244 in this subset. When the full dataset was restricted to a still smaller subset of 103 children for which peak blood Pb levels were below 7.5 μg/dL the slope of concurrent blood Pb and IQ was -2.94 (Lanphear et al., 2005). The analysis of this latter subset supported the authors' conclusions that "for a given increase in blood lead, the lead-associated intellectual decrement for children with a maximal blood lead level <7.5 μg/dL was significantly greater than that observed for those with a maximal blood lead level $\geq$ 7.5 μg/dL (p-0.015)" and that "environmental lead exposure in children who have maximal blood lead levels < 7.5 μg/dL is associated with intellectual deficits". This subset was composed primarily of children from the Rochester cohort (69 children), with smaller numbers of children from five of the other seven cohorts (Lanphear et al., 2005). The Rochester data included IQ test and concurrent blood Pb measurements taken at age six (Lanphear et al., 2005). The linear slope observed for this subset of the pooled dataset,

however, was notably greater than that previously reported for the low blood Pb subset of the Rochester cohort at age five described above, and greater than those slopes from other studies for blood Pb < 10 μg/dL summarized in the CD (e.g., CD, Table 8-7), providing some uncertainty with regard to the precise magnitude of slope for the full range of blood Pb below 7.5 μg/dL.

As discussed in the CD, threshold blood Pb levels for these effects cannot be discerned from the currently available epidemiological studies, and the evidence in the animal Pb neurotoxicity literature does not define a threshold for any of the toxic mechanisms of Pb (CD, Sections 5.3.7 and 6.2).

In applying relationships observed with the pooled analysis (Lanphear et al., 2005) to the risk assessment, which includes blood Pb levels below the range represented by the pooled analysis, several alternative blood Pb concentration-response models were considered in recognition of a reduced confidence in our ability to characterize the quantitative blood Pb concentration-response relationship at the lowest blood Pb levels represented in the recent epidemiological studies. The functions considered and employed in the initial risk analyses for this review include the following.

- Log-linear function with low-exposure linearization, for both concurrent and lifetime average blood metrics, applies the nonlinear relationship down to the blood Pb concentration representing the lower bound of blood Pb levels for that blood metric in the pooled analysis and applies the slope of the tangent at that point to blood Pb concentrations estimated in the risk assessment to fall below that level.
- Log-linear function with cutpoint, for both concurrent and lifetime average blood metrics, also applies the nonlinear relationship at blood Pb concentrations above the lower bound of blood Pb concentrations in the pooled analysis dataset for that blood metric, but then applies zero risk to all lower blood Pb concentrations estimated in the risk assessment.
- Two-piece linear function, for both concurrent and lifetime average blood metrics, applies a two-piece linear model derived from the log-linear function to all blood Pb concentrations estimated in the risk assessment.

In the additional risk analyses performed subsequent to the August 2007 CASAC public meeting (Section 1.4) using the core modeling approach, the first two functions listed above and the following two functions were employed (see Section 5.3.1 of the Risk Assessment Report for details on the forms of these functions as applied in this risk assessment).

- Population stratified dual linear function for concurrent blood Pb, derived from the pooled dataset stratified at peak blood Pb of 10 μg/dL and
- Population stratified dual linear function for concurrent blood Pb, derived from the pooled dataset stratified at 7.5 μg/dL peak blood Pb.

In interpreting risk estimates derived using the various functions, consideration should be given to the uncertainties with regard to the precision of the coefficients used for each analysis. The coefficients for the log-linear model from Lanphear et al. (2005) had undergone a careful development process, including sensitivity analyses, using all available data from 1,333 children. The shape of the exposure-response relationship was first assessed through tests of linearity, then by evaluating the restricted cubic spline model. After determining that the log-linear model provided a good fit to the data, covariates to adjust for potential confounding were included in the log-linear model with careful consideration of the stability of the parameter estimates. After the multiple regression models were developed, regression diagnostics were employed to ascertain whether the lead coefficients were affected by collinearity or influential observations. To further investigate the stability of the model, a random-effects model (with sites random) was applied to evaluate the results and also the effect of omitting one of the seven cohorts on the lead coefficient was assessed. In the various sensitivity analyses performed, the coefficient from the log-linear model was found to be robust and stable. The log-linear model, however, is not biologically plausible at very low blood Pb concentrations as they approach zero; therefore, in the first two functions the log-linear model is applied down to a cutpoint, selected based on the low end of the blood Pb levels in the pooled dataset, followed by a linearization or an assumption of zero risk at levels below that point.

In contrast, the coefficients from the two analyses using the population stratified dual linear function with cutpoints at 7.5 μg/dL and 10 μg/dL, peak blood Pb, have not undergone such careful development. These analyses were primarily done to compare the lead-associated decrement at lower blood lead concentrations and higher blood lead concentrations. For these analyses, the study population was stratified at the specified cutpoint and separate linear models were fitted to the data above and below the cutpoint. The fit of the model or sensitivity analyses were not conducted (or reported) on these coefficients. While these analyses are quite suitable for the purpose of investigating whether the slope at lower concentration levels are greater compared to higher concentration levels, use of such coefficients in a risk analysis to assess public health impact may be inappropriate. Further, only 103 children had maximal blood lead levels less than 7.5 μg/dL and 244 children had maximal blood lead levels less than 10 μg/dL. While these children may better represent current blood lead levels, not fitting a single model using all available data may lead to bias. Slob et al. (2005) noted that the usual argument for not considering data from the high dose range is that different biological mechanisms may play a role at higher doses compared to lower doses. However, this does not mean a single curve across the entire exposure range cannot describe the relationship. The fitted curve merely assumes that the underlying dose-response follows a smooth curve over the whole dose range. If biological mechanisms change when going from lower to higher doses, this change will result in a

gradually changing slope of the dose-response. The major strength of the Lanphear et al. (2005) study was the large sample size and the pooled analysis of data from seven different cohorts. In the case of the 7.5 μg/dL cutpoint, less than 10% of the available data is used in the analysis, with more than half of the data coming from one cohort (Rochester) and the six other cohorts contributing zero to 13 children to the analysis. Such an analysis dissipates the strength of the Lanphear et al. study.

In consideration of the preceding discussion, we place greater confidence in the log-linear model form compared to the dual-linear stratified models for our purposes in this risk assessment. Further, in considering risk estimates derived from the four core functions (log-linear function with low-exposure linearization, log-linear function with cutpoint, dual linear function, stratified at 7.5 μg/dL peak blood Pb, and dual linear function, stratified at 10 μg/dL peak blood Pb), we have assigned greatest confidence to risk estimates derived using the log-linear function with low-exposure linearization since this function (a) is a nonlinear function that describes greater response per unit blood Pb at lower blood Pb levels consistent with multiple studies identified in the discussion above, (b) is based on fitting a function to the entire pooled dataset (and hence uses all of the data in describing response across the range of exposures), (c) is supported by sensitivity analyses showing the model coefficients to be robust, and (d) provides an approach for predicting IQ loss at the lowest exposures simulated in the assessment (consistent with the lack of evidence for a threshold). Note, however, that risk estimates generated using the other three concentration-response functions are also presented to provide perspective on the impact of uncertainty in this key modeling step.

2.2 USE OF CASE STUDIES AND LOCATION SELECTIONS

Consistent with the risk assessment performed during the prior Pb NAAQS review and for the pilot phase assessment, the full-scale risk assessment for the current review relies on a case study approach. This approach is intended to provide a framework for considering the nature and magnitude of Pb exposures associated with air sources and associated risk to human health, and for comparing the impact of alternate NAAQS on those exposures and risks.

With consideration of CASAC comments on the pilot phase assessment (see Section 1.4), the design for the full-scale assessment expanded from the pilot assessment by replacing the urban near roadway case study with a general, non-location-specific, case study focused on population exposures in urban areas (Section 2.2.1), while retaining the two stationary source case studies (Sections 2.2.2.1 and 2.2.2.2). The point source case studies were intended to illustrate risks associated with Pb near point sources, with the primary Pb smelter case study representing one of significant impact on an individual basis, while the general urban case study is intended to assess more widespread Pb exposures, due to the large populations in urban areas.

Background information for all three case studies is presented in appendices A and B, and briefly summarized in Sections 2.2.1 and 2.2.2, below.

Consideration of CASAC comments on the draft full-scale assessment (see Section 1.4) led to the development of additional case studies which are presented in Chapter 5.

2.2.1 General Urban Case Study

In consideration of CASAC comments on the pilot assessment (Henderson, 2007a), a general urban case study is included in the assessment. This case study is designed to provide estimates of risk associated with the current and alternate NAAQS in urban areas, as well as with current conditions. This case study differs from the point source case studies in several ways. First it is not based on a specific location. Rather, it is designed to generally represent large urban areas in the U.S.[3] Second, as discussed further in subsequent sections (e.g., Sections 2.4.2 and 3.1.1), the media concentrations are assumed to be spatially uniform throughout the case study area (i.e., spatial variation within the area is not considered). Third, the ambient air quality for this case study is specified, based on analyses of Pb levels in large urban areas of the U.S. (Appendix A and Section 2.3.1), rather than derived from air quality modeling of particular air Pb sources. As a result of this third difference, this case study includes different types of uncertainties as compared to the case studies employing air quality models, and it does not distinguish among the different air Pb sources influencing the air concentrations, be they currently active stationary or mobile sources, or resuspension of previously deposited Pb. Fourth, the case study does not rely on any specific demographic values; that is, a theoretical population of unspecified size is assumed to be uniformly distributed across the study area. All of these distinctions of this case study from the others have produced a platform that is a simplified representation of urban areas intended to inform our assessment of the impact of changes in ambient Pb concentrations on risk.

2.2.2 Point Source Case Studies

Based on the Analysis Plan (USEPA, 2006a) and conclusions from the pilot phase risk assessment (USEPA, 2006b; ICF, 2006), the point source case studies for this full-scale assessment include a study area near a primary Pb smelter, and a second near another, smaller, stationary Pb source. The locations for these two case studies were chosen with consideration of

[3] While the air monitoring data used to characterize the current conditions scenario for this case study area are from large urban areas, other empirical datasets used in developing this case study, such as those for outdoor residential soil Pb and indoor dust Pb, are generally representative of U.S. residential properties (see Sections 3.1.1.1, 3.1.3.1 and 3.1.4.1). As most U.S. residential properties are in large urban areas because that is where a significant share of the U.S. population resides, these datasets will include greater representation by urban areas, particularly large ones, than non-urban areas. These datasets, however, are not limited to urban locations.

factors described in the Analysis Plan: (a) availability of site-specific monitoring data for ambient air Pb, (b) availability of measurement data for other environmental media (soil and indoor dust) and for Pb exposure (i.e., blood Pb levels), and (c) demographic and socioeconomic factors related to Pb exposure and risk (USEPA 2006a).

Both of these case studies rely on air quality modeling to estimate air Pb concentrations used in the risk assessment and as a result they include different types of uncertainties as compared to those associated with the air concentrations for the general urban case study. One of these is related to the representation of resuspension of previously deposited Pb as a source of Pb to ambient air. To the extent that emissions estimates are available for this source, this is represented in these case studies. However, as recognized in Section 2.1.1 and Appendix A, Section A.1.1.3, such emissions estimates are often uncertain or unavailable. Some resuspension sources are included in the primary Pb smelter case study, however no such sources are explicitly modeled in the secondary Pb smelter case study.

Background information for these case studies is briefly summarized below with regard to: (a) population characteristics, (b) reported emissions, (c) ambient air Pb levels, and (d) the availability of site-specific data characterizing levels of Pb in key media and Pb exposures (e.g., soil, blood Pb level data).

2.2.2.1 Primary Pb Smelter Case Study

The primary Pb smelter case study is focused on the only currently operating facility in the U.S., which is located in Herculaneum, Missouri. At primary Pb smelters, Pb-bearing ore concentrates are smelted to produce Pb metal. This smelter is one of the largest individual sources of Pb metal in the U.S., has been active for over a century and there exist a large amount of site-specific data in the surrounding area characterizing both media concentrations (soil, indoor dust, outdoor air) and population blood Pb levels (Appendix B, Section B.1).

The facility's century of operation has contributed to Pb contamination of the area surrounding the facility[4], which has triggered various remediation activities (e.g., removal of Pb-contaminated residential soil) as well as enlargement of the facility property to encompass many of the most heavily impacted private properties. The remediation activity introduces a complication to the risk modeling, especially aspects involving characterization of the relationship of ambient air Pb and residential soil Pb to indoor dust Pb (see Section 3.1.4).

[4] Portions of this study area comprise an active Superfund site and are subject to ongoing evaluation under the Superfund program administered by the Office of Solid Waste and Emergency Response. Methods used in conducting the human health exposure and risk assessment for the pilot analysis have been selected to address policy questions relevant to the Pb NAAQS review and consequently may differ from those used by the Superfund program.

Some key aspects of the background information for this case study (Appendix B) are summarized briefly in Table 2-1.

Table 2-1. Key aspects of primary Pb smelter case study.

Population	As of the 2000 U.S. Census, approximately 38,000 people lived within 10 kilometers (km) of the facility, 10% of which were children less than 8 years of age. Since 2000, actions associated with reducing facility-related Pb exposures have reduced the population size within 2 km of the facility such that six previously occupied census blocks near the facility were unoccupied in 2004. With those counts subtracted from the 2000 counts, the numbers of children (less than 8 years old) residing within 2 km, between 2 and 5 km and between 5 and 10 km were 171, 1,545 and 2,164, respectively (Appendix B).
Emissions	Lead is emitted from a wide variety of activities associated with the primary Pb smelter, including the transport of materials into and within the facility. The facility is estimated to be the largest Pb emitter in the U.S. (Appendix B, Section B.1 and Appendix A, Section A.1).
Air Quality	In 2005, annual average concentrations of Pb in total suspended particulate matter (Pb-TSP) at the nine monitors in this town, for which data are reported in the U.S. Air Quality System (AQS), ranged from 0.046 to1.56 $\mu g/m^3$. All of these nine monitors, fall within the top 30% of the 2005 annual average levels at AQS monitors nationally, with four of the nine monitors falling in the top 10% (Appendix B). Maximum quarterly average Pb-TSP concentrations at one of these monitors exceeded the current NAAQS in 2005, 2006 and 2007 (Appendix A; AQS).

The area within the city limits of Herculaneum is designated nonattainment for the Pb NAAQS and the existing State Implementation Plan (SIP) was approved in 2002 (67 FR 18497). EPA determined the existing SIP to be inadequate to attain and maintain the current NAAQS in 2006 (71 FR 19432), and consequently the state of Missouri developed a revised SIP for the area. U.S. EPA, Region 7 received Missouri's proposed SIP revision on May 31, 2007 (MDNR, 2007). The air dispersion modeling performed for the risk assessment described in this document built on the information and modeling developed for the revised SIP.[5]

The significant amount of site-specific data available for Herculaneum, paired with air dispersion modeling for the facility conducted in support of SIP development for Pb, provides a substantial data set for this study area which enhances the modeling of exposure and risk. For example, the Herculaneum facility has more site-specific monitoring data available to support risk assessment than the second point source case study location, including residential yard soil, indoor dust and road dust Pb measurements collected in areas potentially impacted by the facility. In addition, the Agency for Toxic Substances and Disease Registry (ATSDR) has conducted a number of health consultations that involved the collection of blood Pb measurements for children (Appendix B, Section B.1.3). Extensive air Pb monitoring data are also available and were considered in the performance evaluation of the modeling setup. The Herculaneum case study location also has a number of attributes that add complexity to the modeling of Pb exposure and risk including (a) complex terrain and meteorology which

[5] The 2007 draft SIP revision (MDNR, 2007), including the modeling, is currently under review by EPA.

complicate the modeling of Pb transport in ambient air, (b) a large and complex facility with a long history of operation and significant opportunity for fugitive emissions, making source characterization challenging, and (c) a history of remediation activities which has contributed to widely varying residential soil Pb concentrations across the town.

2.2.2.2 Secondary Pb Smelter Case Study

The secondary Pb smelter case study, in Troy, Alabama, involves a smaller point source than the primary Pb smelter case study, with relatively less site-specific data characterizing media concentrations and exposure levels. Secondary Pb smelters produce Pb from scrap and provide the primary means for recycling Pb acid automotive batteries, and are among the larger source categories of Pb emitters (see Appendix A, Section A.1). The Troy facility was one of 15 secondary Pb smelters operating within the U.S. in 2002 (see Appendix B, Section B.2). Some key aspects of the background information for this case study (Appendix B) are summarized briefly in Table 2-2.

Table 2-2. Key aspects of secondary Pb smelter case study.

Population	As of the 2000 U.S. Census, approximately 18,000 people lived within 10 km of the facility, 10 percent of which were children less than 8 years of age. Specifically, 187 children of that age group lived within 2 km of the facility, 896 lived between 2 and 5 km and 589 lived are between 5 and 10 km from the facility (Appendix B).
Emissions	Lead is emitted from the facility operations, from materials storage and handling, and from facility roadway dust. Similar to most secondary Pb smelters, emissions from this facility are estimated to fall between 1 and 5 tpy (see Appendix B, Section B.2.2 and Appendix A, Attachment A-1).
Air Quality	Annual average concentrations of Pb-TSP for 2005 at the two monitors located within 1 km of the Troy facility (300 and 800 m from the facility), for which data are reported in the U.S. Air Quality System, are approximately 0.4 and 0.1 $\mu g/m^3$, respectively. These values fall within the top 15% of Pb-TSP annual average values for 2005 (see Appendix B, Section B.2.5.1). Maximum quarterly average Pb-TSP concentrations at one of the monitors exceeded the current NAAQS in 2003 (Appendix A).

In contrast to the Herculaneum facility, we have not identified soil or indoor dust Pb measurements for this case study location. Additionally, although there are blood Pb measurements in children available at the county level, they are not available at a more refined scale that might relate more directly to this case study. The relative sparseness of site-specific Pb measurements means that the exposure assessment conducted for the secondary Pb smelter case study is more dependent on model projections, and consideration of measurements available for similar locations, and that there is less opportunity for rigorous performance evaluation of the modeling steps. The available air Pb monitoring data, however, are used in the performance evaluation of the air quality modeling.

2.3 ASSESSMENT SCENARIOS

The design of the scenarios assessed for each case study includes aspects particular to air concentrations of Pb (Section 2.3.1), surface soil/dust concentrations of Pb (Section 2.3.3) and background (Section 2.3.2). As the scenarios are primarily distinguished by the differences in air concentrations, we generally refer to the different assessment scenarios as air quality scenarios in this document (including the appendices). The different air quality scenarios include current conditions, meeting the current NAAQS of 1.5 $\mu g/m^3$ (maximum quarterly average) and meeting several alternate, lower NAAQS.

2.3.1 Air Concentrations

The air concentrations assessed in the different air quality scenarios include current conditions, meeting the current NAAQS of 1.5 $\mu g/m^3$ (maximum quarterly average) and meeting several alternate, lower NAAQS. In consideration of the range of levels suggested by CASAC (Henderson, 2007a), the alternate NAAQS scenarios included in the assessment are: 0.5 $\mu g/m^3$, 0.2 $\mu g/m^3$ and 0.05 $\mu g/m^3$ as maximum monthly averages, and 0.2 $\mu g/m^3$ as a maximum quarterly average. In response to discussion at the August 2007 meeting of the CASAC Pb Panel to extend lower the range of alternate levels considered, an alternate NAAQS scenario of 0.02 $\mu g/m^3$, as a maximum monthly average, is also included in the additional analyses presented in Chapter 5. While the current and alternate NAAQS scenarios are characterized by quarterly or monthly averaging times, it is the associated annual average ambient air concentrations that are then used in the risk assessment[6].

The current conditions scenario, performed for the general urban and secondary Pb smelter case studies, is intended to generally reflect recent conditions for these case studies based on data available for the characterization. For example, for the urban case study, air Pb levels for current conditions are based on 2003-2005 air quality data (Appendix A). For the secondary Pb smelter case study, for which we used air quality modeling, air Pb levels for current conditions are based on emissions characterizations drawn from currently available emissions information and recent meteorological data (see Appendix E).

The current NAAQS attainment air quality scenario was performed for the primary Pb smelter case study, for which current monitoring data indicate exceedance of the current Pb

[6] Use of the annual average concentration is consistent with the temporal period of this input to the primary blood Pb model (Appendix H) and also the generally longer term resolution of the blood Pb metrics associated with the concentration-response functions ("concurrent" and "lifetime average") (Lanphear et al., 2005).

NAAQS (Appendix B, Section B.1.5.1).[7] Additionally, although we consider it extremely unlikely that air concentrations in urban areas across the U.S. that are well below the current standard would increase to just meeting the standard, we recognize the potential for air Pb concentrations in some areas currently well below the standard to increase to just meet standard by way of, for example, expansion of existing sources (e.g., facilities operating as secondary smelters exercising previously used capabilities as primary smelters) or by the congregation of multiple Pb sources in adjacent locations. Accordingly, we have simulated this type of scenario (increased Pb concentrations to just meet the current standard) in the general urban case study and in the additional analyses for three location-specific urban case studies described in Chapter 5.

In developing the reduced air concentrations for the alternate NAAQS scenarios for the point source case studies (for which air quality models are employed), the maximum monthly or quarterly average (depending on averaging time for the alternate NAAQS) for each modeled receptor point is compared to the NAAQS level to identify the factor by which the highest average exceeds the NAAQS level. All monthly or quarterly averages are then reduced by this factor (i.e., a proportional roll back is implemented) and the associated annual average recalculated for each receptor point.

Two different current conditions scenarios are assessed for the general urban case study based on air Pb concentrations for the period 2003-2005 at monitors in U.S. urban areas with population greater than one million (Appendix A).[8] One of these two scenarios is based on the mean maximum calendar quarter average for these monitors, and the second is based on the 95th percentile of maximum calendar quarter averages for these monitors. Additionally, although the mean and 95th percentile maximum quarterly average of the large urban area monitors nationally do not exceed the current NAAQS level, an increased air concentration scenario (i.e., to the level of the current standard) has been included for this case study.

As the air Pb concentrations for this case study do not vary spatially (see Section 3.1.1.1), the air Pb concentration is simply set to the level specified for each scenario as either a monthly or quarterly average. The annual average air concentration (the metric used in the dust and blood

[7] Given the status of this area with regard to nonattainment and SIP revision, as well as the use of the modeling set-up developed for the SIP attainment demonstration, a current conditions scenario was not developed for the primary Pb smelter case study.

[8] In designing the general urban case study, we recognized CASAC's interest in our development of risk estimates that were more informative to the larger national population and exposures related to ambient air reflecting the aggregate contributions from multiple sources of Pb than might be discrete populations near particular point sources of Pb (Henderson, 2007a). Urban areas of greater than one million population were identified as informative for large populations in established cities (as compared to more newly-developed urban areas).

Pb modeling) is derived from the current conditions or NAAQS level (and averaging time) using relationships based on current Pb-TSP monitoring data for monitors in large U.S. urban areas (Appendix A). Lead in TSP monitoring data for the time period 2003-2005 from monitors in U.S. urban areas of population size greater than one million were analyzed to derive estimates of maximum quarterly average, maximum monthly average and annual average for each monitor. From these estimates, the ratios of the maximum quarterly and maximum monthly averages to the annual average were derived for each monitor and the arithmetic mean and 95^{th} percentile value of the monitor-specific ratios were derived. To derive the annual average air concentration used in the dust and blood Pb modeling for each scenario, one of these ratios was applied to the air quality scenario level. For example, the alternate NAAQS level of 0.5 (for a maximum monthly averaging time) was divided by the mean monitor ratio of maximum monthly average to annual average to derive the annual average air concentration estimate for that alternate NAAQS scenario. The air quality values associated with the different scenarios assessed for the urban case study are summarized in Table 2-3.

Table 2-3. Air quality scenarios assessed for the general urban case study.

Air Quality Scenario	Level ($\mu g/m^3$)	Averaging Time (Form)	Ratio	Associated Annual Average Concentration ($\mu g/m3$)
95th Percentile Current Conditions	0.87	calendar quarter (maximum)	7.6[a]	0.11
Mean Current Conditions	0.14	calendar quarter (maximum)	2.5[b]	0.056
Alternate NAAQS	0.5	Month (maximum)	4.0[c]	0.13
Alternate NAAQS	0.2	Month (maximum)	4.0[c]	0.05
Alternate NAAQS	0.2	calendar quarter (maximum)	2.5[b]	0.08
Alternate NAAQS	0.05	Month (maximum)	4.0[c]	0.013
Current NAAQS	1.5	calendar quarter (maximum)	2.5[b]	0.60
[a] This is the 95th percentile of the ratios of maximum quarterly average to annual average for monitors at sites in urban areas with population of one million or more people.				
[b] This is the mean of the ratios of maximum quarterly average to annual average for monitors at sites in urban areas with population of one million or more people.				
[c] This is the mean of the ratios of maximum monthly average to annual average for monitors at sites in urban areas with population of one million or more people.				

2.3.2 Policy-relevant Background

Given the multimedia, multipathway nature of Pb, levels of Pb in all exposure media (including those other than air) are essential aspects of the scenarios assessed for each case study. As discussed in Section 1.1, some of the Pb in other media may be derived from policy-relevant sources, while, for our purposes here, we have categorized others as policy-relevant background. Some amount of Pb in the air also derives from background sources, such as volcanoes, sea salt, and windborne soil particles from areas free of anthropogenic activity (CD, Section 2.2.1). The impact of these sources on current air concentrations is expected to be quite low and has been estimated to fall within the range from 0.00002 $\mu g/m^3$ and 0.00007 $\mu g/m^3$ based on mass balance calculations (CD, Section 3.1 and USEPA 1986, Section 7.2.1.1.3). The midpoint in this range, 0.00005 $\mu g/m^3$, has been used in the past to represent the contribution of naturally occurring air Pb to total human exposure (USEPA 1986, Section 7.2.1.1.3). It is noted that the data available to derive such an estimate are limited and that such a value might be expected to vary geographically with the natural distribution of Pb. Comparing this to reported air Pb measurements is complicated by limitations of the common analytical methods and by inconsistent reporting practices. This value is one half the lowest reported nonzero value in AQS. For the purposes of this assessment, however, the value of 0.00005 $\mu g/m^3$ was selected as representative of policy-relevant background Pb in air. Unlike for other criteria pollutants, the

role of this value for Pb is limited. In considering risk contributions from policy-relevant background, the contributions from exposures to nonair media are such that any credible estimate of policy-relevant background in air is likely insignificant in comparison. In developing the air Pb concentrations associated with the alternate NAAQS scenarios, the estimate of policy-relevant background in air was the floor below which concentrations were not lowered.

2.3.3 Outdoor Soil/Dust

With regard to surface soil[9] Pb concentrations for the alternate NAAQS scenarios, the presence of historically deposited Pb, associated with past periods of higher air concentrations and associated atmospheric deposition, affects the soil Pb dynamics. Under the alternate NAAQS scenarios, atmospheric deposition of Pb will continue to occur, albeit it will be reduced from the current rate which is reduced from historic rates. At any location, the type of response of the surface soil concentrations to the changed deposition rate will depend on the relationship of current surface soil concentrations to the surface soil concentrations associated with a steady state condition (i.e., when the rate of Pb addition to the surface soil equals the rate of Pb loss from the surface soil) at that location. If current surface soil concentrations are below their steady state levels for the current conditions (i.e., the rate of Pb addition is greater than the rate of Pb loss) and air concentrations are reduced (i.e., the rate of Pb addition via deposition is reduced), surface soil concentrations might be expected to continue an increasing trend, although at a reduced rate from the current rate. Alternatively, if current surface soil concentrations are above their steady state levels (i.e., the rate of Pb loss is greater than the rate of Pb addition) and air concentrations and associated deposition are reduced, surface soil concentrations might be expected to continue a decreasing trend and do so at a greater rate than the current one.

Information regarding the current dynamics of Pb concentrations in surface soils is limited with the predominant focus for such studies being somewhat remote forested areas (e.g., CD, Section 3.2 and 3.2.2 and pp. AX7-33 to AX7-34). Findings to date indicate that systems with little influence from local point sources are still responding to reduced Pb deposition rates associated with reduced atmospheric emissions of Pb, including those associated with the phase-out of leaded gasoline. For example, studies of forest soils have concluded that surface concentrations of Pb are decreasing in response to the reduced Pb deposition rates since the phase-out of leaded gasoline (Miller and Friedland, 1994; Kaste and Friedland, 2003). Studies in

[9] In the risk assessment, outdoor surface soil or dust is an important exposure pathway. Use of the term surface soil here is intended to include the terms outdoor soil and outdoor dust, with there being some overlap between those two terms in that the surface layer of outdoor soil might be referred to as outdoor dust. Specifically, the phrase "outdoor dust" refers to particles deposited on any outdoor surface, including, for example, soil, sidewalks, roadways, etc.

urban areas of southern California, where Pb has accumulated from past sources, suggest an environment in which Pb may remain at the soil surface (and other surfaces), contributing to air concentrations via resuspension in the near term (CD, pp. 2-65 to 2-67 and 3-18 to 3-19). Accordingly, the temporal trend in surface soil concentrations in this environment is considered to be influenced by the rate of resuspension, such that little to no reduction in soil Pb concentration in southern California is expected over the next few hundred years (CD, pp. 2-65 to 2-67 and 3-18 to 3-20; Harris and Davidson, 2005). Temporal trends in surface soils near established point sources are not well characterized. Available information for a few areas surrounding smelters after implementation of pollution controls shows a decline in Pb concentrations in outdoor dustfall, street dust and indoor dustfall, but has not indicated a noticeable decline in soil Pb concentrations (CD, pp. 3-23 to 3-24).

The above discussion suggests that a reduced air concentration in the three case studies would not be expected to yield a changed surface soil concentration over the near term, yet may yield a reduced surface concentration over a much longer term. An exception to this may be some areas of the primary Pb smelter case study where contaminated soil has been removed and replaced with "clean" soil. Measurements taken of Pb concentrations in such "clean" soil placed within ¾ mile of the facility exhibit small increasing temporal trends over a period of a few year[10] (USEPA, 2006c). In lieu of additional data or a multimedia modeling analysis, however, the surface soil concentrations for the current and alternate NAAQS scenarios in all case studies have been set equal to those used for the current conditions scenarios. This is generally believed to be a reasonable representation of soil Pb response to alternate NAAQS for at least six years, and likely much longer, after a new standard might be implemented.[11] A potential exception is the area of the primary Pb smelter case study within ¾ mile of the facility, where it may be that surface Pb concentrations in remediated soil may increase to higher levels under the current and some of the alternate NAAQS. This remains an area of uncertainty with potential implications for areas in which a Pb source or combination of Pb sources may locate where ones of comparable size had not been previously.

Additionally, we recognize that implementation of some alternate NAAQS could in some areas (e.g., areas of substantial past atmospheric deposition) involve control of surface soil/dust to reduce surface soil/dust Pb levels. That is, in places where surface soil/dust Pb concentrations contribute substantially to air concentrations, controls implemented to attain various alternate lower NAAQS might include reducing soil Pb concentrations. Such specific control actions have

[10] An increasing trend was not seen with soil at the two locations just beyond ¾ miles away.

[11] This was also the approach used in the risk assessment performed in the last Pb NAAQS review (USEPA, 1990).

not been addressed in this assessment, and as stated above, outdoor soil/dust concentrations in all air quality scenarios have been set equal to the values for the current conditions scenarios.

2.4 ANALYTICAL APPROACH

This section provides an overview of the analytical approach, describing key elements including: (a) temporal aspects, (b) spatial scale of the analysis and the type of spatial template used in modeling, (c) overview of the analytical steps of predicting media concentrations, modeling exposure, and modeling risk, (d) performance evaluation completed in support of the analysis and (e) the approach used to characterize uncertainty and evaluate model sensitivity.

The approach described here pertains to the initial analyses of the full-scale assessment for which results are described in Chapters 3 and 4, and to some aspects of the core modeling approach employed for the additional analyses described in Chapter 5.

2.4.1 Temporal Aspects

The risk assessment conducted for each case study uses a simulated child population for which exposure begins at birth and continues for 7 years. That is, the study population is assumed to be a single group, for which exposure begins at birth and continues until the group reaches 7 years of age.[12] Furthermore, it is assumed that no migration or immigration of these children occurs during this simulation period; that is, none of the children move out of the study area and no children move in.

For the point source case studies, the use of modeling (with a constant emissions rate and temporally varying meteorology) provides temporally varying air concentrations. However, the primary blood Pb model for this assessment is limited in the temporal resolution of its inputs (see Section 3.2), because the finest temporal resolution of inputs to the blood Pb model is a year. Consequently, in characterizing exposure media concentrations, annual averages are used.

With regard to temporal variation across the seven-year exposure period, several exposure factors and physiological parameters are varied on an annual basis within the blood Pb modeling step (see Section 3.2). Once set for the air quality scenario, however, the media concentrations of Pb are held constant throughout the seven year period (see Section 3.1).

2.4.2 Spatial Scale and Resolution

The size and resolution of the study area differed among the three case studies. The general urban case study is not set in a specific location, and involves non-spatially-varying media concentrations and population density. There is no spatially differentiated template *per se*

[12] Modeling of blood Pb levels for the child population includes contributions representative of prenatal Pb exposure.

and instead, a single generic urban study area is assumed with uniform population density and exposure concentrations. For the exposure modeling for the point source case studies, however, spatial templates were developed. The templates subdivide the study area into subunits, composed of U.S. Census blocks or block groups, across which media concentrations differ.[13] Media concentration estimates (e.g., for outdoor air, soil and indoor dust) are developed for each block or block group, and from these a central tendency estimate is developed of concurrent and lifetime average blood Pb levels for the resident children. Interindividual variability of blood Pb levels for children within a block or block group is considered through the use of a statistically derived GSD. The specific spatial templates used for each of the point source case studies are presented in Appendices D (Section D.1) and E (Section E.1).

For the two point source case studies, population risk estimates were derived separately for a portion of the full study area in addition to the estimates derived for the full study area. The subareas extended approximately 1.5 km out from the point source locations (see Appendix P).

2.4.3 Categorization of Policy-relevant Exposure Pathways

To inform policy aspects of the Pb NAAQS review, we have attempted to parse the assessment estimates for indoor dust Pb, blood Pb and IQ loss into the fraction associated with policy-relevant background (e.g., diet and drinking water) versus that associated with policy-relevant pathways, which include inhalation, outdoor soil/dust ingestion and indoor dust ingestion (Section 2.1.1). We have further categorized the policy-relevant pathways into one of two categories, “recent air” or “past air”. Conceptually, the recent air category includes those pathways involving Pb that is or has recently been in the air, whether or not it was also in the air in the past, and the past air category includes those pathways involving Pb that was in the air in the past and was not in the air recently. As discussed below, this conceptual distinction could not be entirely reflected in the risk assessment due to technical limitations. Thus, risk estimates associated with “past air” reflect some Pb that was recently in the air. Similarly, diet and drinking water are treated entirely as policy-relevant background, despite also reflecting contributions from policy-relevant pathways such as atmospheric deposition.

Conceptually, recent air refers to exposure contributions associated with inhalation of ambient air Pb and ingestion of the fraction of indoor dust Pb derived from recent ambient air Pb. To the extent that ambient air Pb includes contributions from resuspension of previously

[13] US Census block groups vary in size from several city blocks in densely populated urban areas to many square miles in less populated rural areas. Their population count varies from 600 to 3000 people per block group with the typical block group in the U.S. containing 1,500 people. US Census blocks are more refined than block groups and typically contain several hundred people or less. Their size can vary from a single city block in urban areas to multiple square miles in less populated rural locations.

deposited Pb, that source is represented in the recent air category. Thus, a "recent air" exposure may involve previously deposited Pb that is (1) resuspended into the air and inhaled or (2) resuspended into the air, transported into a building, deposited into the dust, contacted and ingested.

Conceptually, past air includes exposure contributions from ingestion of outdoor soil/dust that is contacted on surfaces outdoors, and ingestion of indoor dust Pb that is derived from past air sources. Although Pb that is currently in outdoor soil/dust may have been in the air recently or some time ago, we have assigned ingestion of outdoor soil/dust Pb contacted outdoors to the past air category in recognition of our inability to maintain a dynamically changing categorization of recent versus past air.[14] The past air category also includes the ingestion of indoor dust Pb that was in the ambient air in the past but not recently. These sources to indoor dust Pb include any residual legacy of historical air Pb in the indoor dust of older homes, as well as Pb occurring in indoor dust that is derived from outdoor soil/dust Pb that was not transported indoors by an air pathway. This latter pathway includes outdoor soil/dust Pb that is carried indoors by human contact (e.g., "tracking in").

To implement this categorization in the assessment, we developed estimates of the recent air portion of indoor dust Pb (i.e., contributions associated with recent ambient air Pb levels), and assigned the remainder of indoor dust Pb to other sources, which include those relevant to past air. That is, the "other sources" component of indoor dust Pb refers to contributions from indoor paint, outdoor soil/dust and additional sources. Among the additional sources is any residual legacy of historical Pb in the indoor dust of older homes.

The indoor dust Pb subdivision reflects and is limited by the models and inputs used to estimate indoor dust Pb levels for the different scenarios. All of them predict dust Pb concentration as a function of, among other factors, ambient air Pb concentration, and all of the models include a constant (e.g., the intercept in the regression-based models) that captures "other" sources, as well as uncertainty associated with the relationship with ambient air Pb.[15] One of the models (used in the secondary Pb smelter case study and in part of the primary Pb smelter case study) also includes a dependency on outdoor soil/dust Pb concentration. This difference among the models leads to an inconsistency across the case studies in the ability to separate the contribution to indoor dust Pb from outdoor soil/dust. Consequently, we have

[14] In concept, this assignment appears to inherently contribute to an underestimate of the recent air category. However, the reality of much higher air emissions in the past that have contributed to Pb concentrations in other media that are higher than those that would be associated with more recent lower emissions, complicates consideration of this assumption (see Section 2.3.3).

[15] The extent to which this intercept captures uncertainty about the relationship with ambient air will indicate presence of a recent air pathway in the past air category.

limited to two categories the subdivision of indoor dust contributions, with that from outdoor soil/dust included in "other" whether it is estimated by a model that includes a soil concentration coefficient or is accommodated by the constant or intercept term. Additionally, we have not subdivided indoor dust Pb estimates for the primary Pb smelter due to uncertainty regarding this categorization with the model used for that case study (Section 3.1.4.2). In presenting risk estimates associated with policy-relevant pathways (Chapter 4), we have included risk associated with the "other" component of indoor dust in the past air category. We recognize that the potential for this "other" component to include ambient Pb unrelated to air emissions contributes to a potential for the risk estimates associated with past air to be overestimates. Additionally, as the recent air portion of indoor dust Pb, which derives from transport of airborne Pb into a house, depends on the estimate of ambient air Pb concentration, selection of an ambient air Pb estimate that is not appropriate to this relationship (e.g., one that does not represent ambient air Pb levels immediately outside the house) may contribute to an under- or overestimate of recent air indoor dust Pb.

There is inherent uncertainty associated with the approaches used to divide indoor dust-related Pb exposures and risk into contributions from "recent ambient air" and from "other" sources. Further, the uncertainty may differ among the three case studies due to the different approaches used in modeling indoor dust Pb. For example, uncertainty associated with the hybrid mechanistic-empirical model used in the general urban case study includes that which arises from model inputs and model performance, while the empirical, regression-based, statistical models used in the point source case studies entail uncertainty regarding similarity of the conditions from which the model was derived to those to which it is applied, as well as uncertainty regarding variables that may be correlated with those explicitly represented in the model. Thus, given the various limitations of our modeling tools, blood Pb levels associated with air-related exposure pathways and current levels of Pb emitted to the air (including via resuspension) are likely to fall between the estimates derived for "recent air" pathways and those for "recent" plus "past air" pathways.

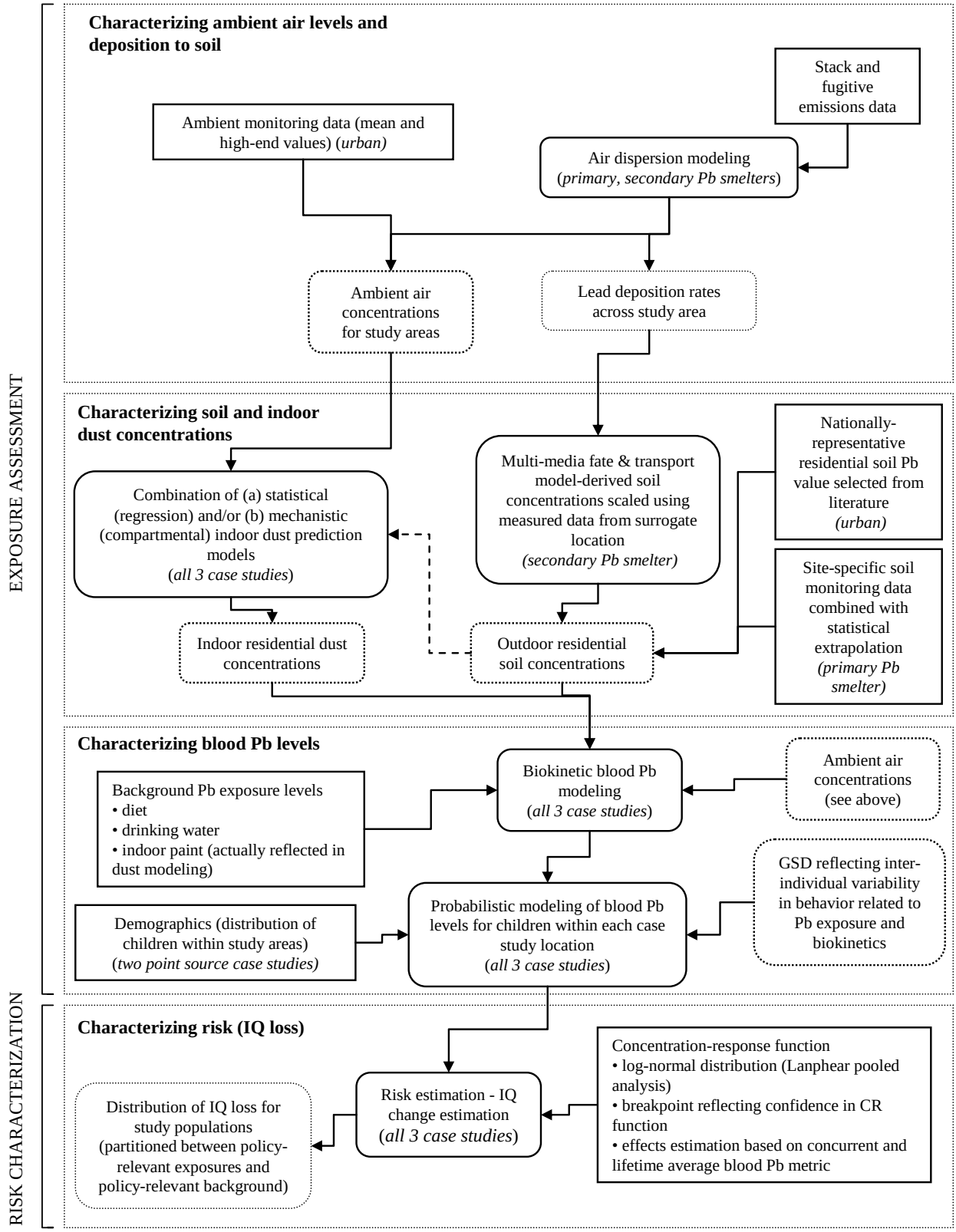
2.4.4 Overview of Analytical Steps

As illustrated in Figure 2-2, the risk assessment completed for the two point source case studies generally includes four analytical steps: (a) fate and transport of Pb released into outdoor air, including the dispersion of Pb away from the point of release and the deposition of Pb onto surfaces, (b) prediction of the resulting concentration of Pb in media of concern including outdoor air, outdoor surface soil/dust and indoor dust, (c) use of these Pb concentrations together with estimates of Pb in background exposure pathways, including diet, to estimate associated blood Pb levels in children using biokinetic modeling and (d) use of concentration-response

functions derived from epidemiology studies to estimate IQ loss associated with the estimated blood Pb levels. The modeling approach for the general urban case study is somewhat simpler, since it does not involve fate and transport modeling for air concentration estimates and instead, uses ambient monitor levels combined with an assumption of uniform ambient 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 studies, with the generation of population blood Pb 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 are employed in the exposure assessment (Section 2.4.4.1), while the fourth is the risk assessment step (Section 2.4.4.2).

Prior to focusing on a single modeling approach (see Chapter 5), and because of the quantitative influence of certain analytical steps on the results, we employed multiple approaches to perform some of the analytical steps. The multiple sets of results generated in this way for a case study and air quality scenario are intended to span a range indicative of the model and parameter uncertainty associated with those analytical steps of the risk analysis (see Section 2.4.6).

Figure 2-2. Overview of analysis approach.



2.4.4.1 Exposure Assessment

Concentrations of Pb are 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 recognized in Section 4.3. Specific approaches used at the three case study locations are briefly described below.

Characterization of Pb in ambient air relies on (a) dispersion modeling of facility-related (including fugitive) Pb emissions for the primary and secondary Pb smelter case studies and (b) the use of ambient monitor data for the general urban case study. For the general urban case study, monitoring data for U.S. urban areas of more than a million in population were used to identify two current conditions scenarios, one "typical", and one higher end, and to relate alternate NAAQS (of other forms) to annual average levels needed for blood Pb modeling. A key aspect of the general urban case study is that ambient air lead levels do not vary spatially within the study area. The approach is monitor-based rather than source-based as compared to two point source case studies. This means that we did not explicitly model specific source contributions for the general urban case study (e.g., resuspension of roadside dust, "fresh" industrial emissions) and instead, relied on empirical data to define ambient air Pb levels for this general case study, with these levels reflecting contributions from all contributing sources, be they currently active stationary or mobile sources, resuspension of previously deposited Pb or other.

Characterization of Pb concentrations in outdoor surface soil/dust, resulting from deposition of airborne Pb is based on the use of (a) existing site-specific measurements (primary Pb smelter case study), (b) nationally representative residential soil measurements obtained from the literature (general urban case)study and (c) fate and transport modeling (secondary Pb smelter case study). 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 have 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., exchange of outdoor air with indoor air, deposition rates of Pb to indoor surfaces, house cleaning rates). For both point source case studies, 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 urban case study. 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 Section 3.1.

Blood Pb levels are 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 (Section 3.2.1.1). A second biokinetic model, the International Commission for Radiation Protection model (hereafter referred to as the "Leggett model"), is included in the sensitivity analysis (Section 3.2.1.2).[16] The same fundamental approach was used to estimate population distributions of blood Pb levels for each of the two point source case studies, and a somewhat simpler approach was used for the general urban case study. The approach used for the two point source case studies involved two main steps:

1) Use biokinetic model to predict central tendency blood Pb levels for children within each exposure zone: The model outputs are then aggregated into the "concurrent" and "lifetime average" blood Pb metrics used in the concentration-response functions.
2) Implement probabilistic exposure model to generate a population distribution of blood Pb levels for children in each case study location: The probabilistic model generates a distribution of simulated blood Pb levels for the children in each study area based on consideration of three key factors: (a) the central tendency blood Pb levels generated for each exposure zone in the preceding step, (b) demographic data (distribution of children 0-7 years of age) across the zones comprising a given study area and (c) use of a GSD characterizing interindividual variability in blood Pb (e.g., reflecting differences in behavior and biokinetics related to Pb).

The step involving modeling population-level exposures for the general urban case study is somewhat simpler than that used for the two point source case studies in that demographic data for a specific location is not considered. As discussed in Section 3.2.2, this avoids the need for implementing a population-weighted probabilistic sampling procedure.

[16] The Leggett model was included along with IEUBK in the pilot analyses. Findings for the model in the pilot analyses and in subsequent performance analyses (Appendix J and Section 3.5) contributed to the decision to use the IEUBK model as the primary model in this full-scale assessment (Section 3.2), and the Leggett model in the sensitivity analysis (Section 3.5.2).

2.4.4.2 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 blood Pb levels (for the two blood Pb metrics) are combined with three blood Pb concentration-response functions for IQ loss (see Section 4.1). Three differing concentration-response functions (described in Section 4.1.1) have been selected to provide three different characterizations of behavior at low exposures. The decision to use three different functions is in recognition of uncertainty related to modeling this endpoint, particularly at lower exposure levels (e.g., blood Pb levels < 5 µg/dL). These three functions are all based on the lognormal concentration-response function described in the Lanphear et al, (2005) pooled analysis of epidemiology studies focusing on IQ loss in children. As these three functions were developed for each of the blood Pb metrics included in the analysis, concurrent and lifetime average, six separate functions were used in the analysis.

For each of the two point source case studies, we have produced two categories of risk metrics:

- Population risk percentiles: The IQ loss associated with policy-relevant exposure pathways for specific percentiles of the child population (e.g., the 50^{th}, 90^{th}, and 95^{th} 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 (50^{th} percentile, mean) to children experiencing higher exposures (90^{th}, 95^{th} 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 95^{th} percentile). This risk metrics 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, because it is not location-specific, 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.

Additional detail on the risk characterization is presented in Sections 4.1 and 4.2.

2.4.5 Variability Characterization

There are a variety of sources of variability associated with the results of this assessment which are presented in terms of risk estimates for specific population percentiles:

- variability in the concentration of lead in key media (e.g., diet, drinking water, ambient air, indoor dust),
- variability in behaviors which effect Pb exposure (e.g., dust ingestion, soil ingestion),
- variability in physiological response to Pb exposure leading to variations in blood Pb levels, and
- variability in the toxic response to Pb, resulting in differing degrees of IQ loss for the same degree of Pb exposure.

A variety of methods have been used to incorporate, to a limited extent, the above sources of variability in the risk assessment such that they are reflected in the results for the three case studies.

- For the two point source case studies, use of spatial templates developed using a geographic information system (GIS) to reflect the distribution of children across a study area (in relation to ambient air Pb and related media concentrations) in projecting population exposures.
- For the general urban case study, inclusion of two current conditions scenarios (mean and 95th percentile air Pb scenarios) which together, provide a degree of coverage for variation in ambient air Pb levels seen across urban areas in the U.S.
- Use of empirically derived GSDs reflecting interindividual variability in blood Pb levels, to provide coverage for multiple sources of variability associated with Pb exposure and biokinetics. Note, that the application of these GSDs provides the primary means of reflecting interindividual variability in blood Pb levels in this analysis. These GSDs also reflect uncertainty associated with measuring blood Pb levels and characterizing population-level distributions of those levels.

There is significant uncertainty associated with reflecting the sources of variability identified above in population-level exposure and risk. For example there is uncertainty associated with the GSD selected to reflect interindividual variability in blood Pb levels for a particular case study. This is considered in the uncertainty characterization (Section 2.4.6).

2.4.6 Uncertainty Characterization and Sensitivity Analysis

Several methods have been used to examine uncertainty in our modeling approach and its potential impact on exposure and risk estimates (Section 4.3). These include: (a) development of multiple sets of exposure and risk estimates for each case study and air quality scenario that illustrate the combined impact of different key models and parameters on risk results and the associated uncertainty, (b) evaluation of model performance (e.g., by comparison with empirical

data) to provide confidence in individual modeling steps and (c) qualitative discussion of key sources of uncertainty and their potential impact on exposure and risk estimates. Each of these elements of the uncertainty characterization is briefly summarized below.

In addition, we have completed a sensitivity analysis, intended to characterize the potential impact of individual modeling elements on risk results (see Section 2.4.6.3).

2.4.6.1 Performance Evaluations

Performance evaluation for the exposure assessment (Section 3.5) focused on evaluation of estimates of Pb in ambient air, outdoor soil, and indoor dust (discussed in Section 3.5.1) and estimates of Pb in blood (covered in Section 3.5.2). This evaluation focused on those estimates based on modeling.

2.4.6.2 Generating Multiple Sets of Results

There are multiple models or inputs that could be implemented for each of the analytical steps of the assessment. For those more highly influential analytical steps for which it is not clear which model or input would generate "best estimate" results, we have implemented multiple modeling approaches in the initial analyses presented in Chapters 3 and 4. Risk results considered across these multiple modeling approaches provide perspective on the range of potential risk, given key sources of uncertainty in the analysis. The multiple modeling approaches for each case study were developed by the following stepwise strategy:

- Identification of those modeling elements believed to contribute significant uncertainty to risk results,
- Identification of a set of plausible options for each of these key modeling elements (e.g., alternative models or input parameters), and
- Development of alternative modeling approaches by combining individual options from the previous step.

Identification of the modeling elements believed to contribute significant uncertainty (step 1) involved consideration of a number of factors including the results of the sensitivity analysis completed for the pilot analysis, and comments provided by CASAC and the public on the pilot analysis and analysis plan.

Because each of the case studies uses different modeling approaches for some of the analytical steps (e.g., different indoor dust models are used for each case study, and these are associated with differing uncertainty), the identify and size of the areas of uncertainty associated with each case study differs. The specific modeling approaches for each case study and their elements are presented in Figure 2-3. For the general urban case study, two different dust models and two GSDs were used, compared to one model and GSD for these analytical steps in

the two point source case studies. However, the same number of blood Pb metrics and IQ loss functions are used for all three case studies.

Figure 2-3. Modeling approaches for case study analyses presented in chapters 3 and 4.

Case Study	Elements of modeling approaches				Number of sets of results
	Indoor dust modeling	Blood Pb metric	GSD	Concentration-response function	
General Urban Case Study	2 models: (a) hybrid mechanistic-empirical (b) statistical (regression)	2 metrics: (a) concurrent (b) lifetime average	2 sets of GSDs, representing: (a) smaller scale (b) larger, regional scale	3 functions: (a) log-linear with linearization, (b) log-linear with cutpoint, and (c) two-piece linear	2 * 2 * 2 * 3 = 24
Each Point Source Case Study	1 model: statistical (regression) approach [b]		1 set of GSDs		1 * 2 * 1 * 3 =6

[b] Different models used for each point source case study.

The set of exposure and risk results generated for each case study and air quality scenario using the alternative modeling approaches indicates to some extent the magnitude of uncertainty surrounding the risk results. However, as discussed in Section 4.3, these sets of risk results do not represent an uncertainty distribution, since confidence levels are not specified for each modeling approach. In presenting the multiple sets of results generated for each case study, we have selected the highest and lowest sets of risk results from those generated, and used them to represent, respectively, upper and lower bounds on risk. The degree to which these actually represent upper and lower bounds depends on the whether the various modeling approaches evaluated in this analysis capture the largest sources of uncertainty. For example, if an important source of uncertainty was excluded in designing the alternative modeling approaches for a given case study, than the bounds represented by the set of risk results generated for that case study might not be wide enough.

2.4.6.3 Sensitivity Analysis

Sensitivity analysis techniques were used to examine the uncertainty for individual modeling elements and its impact on exposure and risk estimates. We used a "one element at a time elasticity analysis" approach, running the full risk model with one of the selected modeling elements adjusted to reflect an alternate input value or modeling choice. The results of that run with the modified modeling element would then be compared to those for the "baseline risk" run to determine the magnitude of the impact on risk results of selections for that one modeling element.

While the sensitivity analysis for the pilot was based on the primary Pb smelter case study, for the full-scale analysis we have focused the sensitivity analysis on the general urban case study. This reflects a desire to more fully understand the sensitivity of the modeling approach used for the general urban case study to key sources of uncertainty, recognizing that the results of the pilot sensitivity analysis are informative with regard to the point source case studies. The sensitivity analysis completed for the full-scale analysis focused on those modeling elements (including input datasets and modeling steps) believed to have a significant potential for impacting exposure and risk results. Those modeling elements include oral uptake factor, interindividual blood Pb variability GSD, biokinetic model, concentration-response function for IQ loss. This type of sensitivity analysis indicates which of the modeling elements included in the sensitivity analysis has the greatest impact on risk results, and can be used to guide future efforts to refine the overall risk model.

2.4.6.4 Qualitative Discussion of Sources of Uncertainty

In addition to the quantitative analyses described above, we have also included a qualitative discussion of key sources of uncertainty in the analysis. This includes sources not explicitly included in any of the above quantitative analyses due to a lack of necessary data (Section 4.3.1). This discussion also attempts to describe the nature of the impact of these sources of uncertainty on risk results, e.g., would a particular source of uncertainty likely result in an over- or underestimation of risk. To the extent possible, the likely magnitude in qualitative terms of a particular source of uncertainty is also discussed.

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3 EXPOSURE ASSESSMENT

This chapter describes the methods and results of the exposure assessment and performance evaluation for the initial analyses of the full-scale assessment. Additional analyses are described in Chapter 5.

3.1 METHODS FOR ESTIMATING MEDIA CONCENTRATIONS

To estimate media Pb concentrations for the three case studies, we used a combination of empirical data and fate and transport modeling, reflecting the different availability of Pb measurements for the two point source case studies and the non-location-specific nature of the general urban case study (Table 3-1). For all three case studies, media concentrations were estimated for multiple air quality scenarios including a range of alternative NAAQS (see Section 2.3). However, outdoor dust/soil concentrations for each of the three case studies were established for current conditions or the current NAAQS scenarios and those values were used for the current NAAQS and each of the alternate NAAQS scenarios evaluated (see Sections 2.3.3 and 3.1.3). Further, as described in Section 2.4.1, media concentrations, once defined, are held constant for the full exposure period simulated with the blood Pb modeling. For ambient air, outdoor soil/dust and indoor dust, estimates of annual average concentration were used for this purpose for all three case studies. Additionally, from the ambient air concentrations, annual average inhalation exposure concentrations were estimated with consideration for daily activity patterns by children and differences in outdoor (ambient) versus indoor air Pb levels (see Section 3.1.2).

Table 3-1. Case study approaches for estimating media Pb concentrations.

Modeling Step	General Urban Case Study	Primary Pb Smelter Case Study	Secondary Pb Smelter Case Study
Spatial template	Single generic study area (with spatially uniform media concentrations and population density)	Combination of U.S. Census blocks and block groups out to a 10 km radius around the facility (with media concentrations and demographics uniform within blocks/block groups)	U.S. Census blocks out to a 10km radius around the facility (with media concentrations and demographics uniform within blocks)
Ambient air concentrations for current conditions and/or current NAAQS	Monitor data (mean and high-end urban monitors selected to represent two current conditions scenarios)	Dispersion modeling	
Performance evaluation	[Monitoring data used as basis characterizing ambient air levels]	Comparison to Pb-TSP monitor data from study area	
Inhalation exposure concentrations	Estimates for all three case studies are based on ambient air concentrations and reflect the application of location-specific adjustment factors that account for (a) the time spent by children at different locations and at various activity levels and (b) differences between indoor and outdoor ambient air Pb levels		
Outdoor soil concentrations	Nationally representative residential soil value selected from literature (same value used for entire study area and for all air quality scenarios)	Near facility (remediation zone) relied on sampling data, and regression model used for outer portions of study area	Multiple Pathways of Exposure (MPE) model used to predict spatial distribution of surface soil Pb levels, then scaled using empirical data from surrogate location
Performance evaluation	[Empirical data used in characterizing soil levels]	[Estimates based on surrogate data]	Modeled estimates compared to surrogate data for other industrial (point source) locations
Indoor dust concentrations	Two approaches used: (a) hybrid mechanistic (compartmental) model augmented with empirical data developed specifically for this analysis and (b) empirical (air-only regression) model obtained form the literature	Near facility (remediation zone) relied on site-specific regression model (based on air) and pooled analysis regression model (based on air plus soil) for remainder of study area	Statistical (air-only regression) model obtained from the literature
Performance evaluation	Subcomponents of hybrid model evaluated, and case study estimates compared to literature estimates and national-scale survey	Site-specific sampling data used in deriving regression model for remediation zone; case study estimates compared to literature estimates and national-scale survey	Case study estimates compared to literature estimates and national-scale survey

3.1.1 Ambient Air Concentrations

Different methods were used for estimating annual average ambient air concentrations for the three case studies. For the primary and secondary Pb smelter case studies, air dispersion modeling of Pb emissions was performed, while Pb-TSP measurement data from the years 2003-2005 for urban areas with greater than one million residents were used for the general urban case study.

3.1.1.1 General Urban Case Study

The general urban case study is not site-specific and has been designed to represent general Pb exposures experienced by children residing in urban areas within the United States. The case study involves spatially uniform Pb concentrations in ambient air, outdoor soil/dust and indoor dust with a matching uniformly distributed child resident population. This is in contrast to the point source case studies which each have a relatively large number of exposure zones to track potentially significant spatial gradients in their concentrations of Pb in environmental media and the spatial distribution of children.

Two current conditions scenarios are included in the general urban case study based on Pb-TSP monitoring data from urban areas in the United States. Specifically, these two scenarios include: (a) a central tendency current conditions scenario based on the mean maximum quarterly average Pb measurement (0.14 $\mu g/m^3$) seen, in 2003-05 time period, at Pb-TSP monitors in urban areas with greater than a million people, and (b) a high end current conditions scenario based on the 95th percentile maximum quarterly average Pb measurement (0.87 $\mu g/m^3$) obtained from the same urban Pb-TSP dataset. The data analysis associated with these values is described in Appendix A, Section A.2.2.2.

In addition to these current conditions scenarios, the current NAAQS scenario (1.5 $\mu g/m^3$, as a maximum quarterly average) and four alternate NAAQS scenarios have also been evaluated (see Section 2.3 for additional details on the NAAQS scenarios). In each of these instances, the specific NAAQS of interest has been evaluated by assuming that air Pb concentrations in the entire study area are reduced to meet that specific ambient air Pb level and form.

Because the blood Pb modeling (Section 3.2) is based on annual average media concentrations, the maximum quarterly and maximum monthly average values used in defining the air quality scenarios were translated into equivalent annual average ambient air concentrations for the blood Pb modeling (Section 2.4.1). This is accomplished using ratios obtained from the 2003-2005 Pb-TSP monitoring dataset that relates maximum quarterly or maximum monthly averages to associated annual average values for each monitor located in an urban area of population more than a million (Appendix A, Section A.2.2). Ratios were selected

for each of the air quality scenarios according to the averaging time (calendar quarter versus month) and the percentile represented by the air quality scenario (e.g., mean *versus* high end for current conditions). Consequently, the annual average Pb concentration for the central tendency current conditions scenario was derived from the mean of monitor-specific maximum quarterly average concentrations using the mean of the monitor-specific *maximum quarterly-to-annual average* ratios, while the high end current conditions scenario was derived from the 95^{th} percentile of monitor-specific maximum quarterly average concentrations using the 95^{th} percentile of the monitor-specific *maximum quarterly-to-annual average* ratios. The mean of the *maximum quarterly-to-annual average* ratios was also used to derive the annual average concentration estimates for the alternative NAAQS air quality scenario with a quarterly averaging time. The mean of the *maximum monthly-to-annual average* ratios was used to derive the annual average concentration estimates for the alternative NAAQS scenarios with monthly averaging times. The derivation of these ratios is described in Appendix A, Section A.2.2.

3.1.1.2 Primary Pb Smelter Case Study

The air quality scenarios included for the primary Pb smelter case study were the current NAAQS and four alternate NAAQS (see Section 2.3.1). As the study area of the primary Pb smelter is currently in nonattainment for the current NAAQS and emissions profiles from the facility are being modified as additional controls are put in place, ambient air Pb concentrations for the current NAAQS scenario were estimated using the model, emissions and source parameters used in developing the 2007 proposed revision to the State Implementation Plan for the area (MDNR, 2007a, 2007b). Annual average Pb concentration estimates for the current NAAQS scenario were derived for each census block or block group from model outputs. Additionally, for the purposes of developing the alternative NAAQS scenarios, hourly estimates from the model were used to generate quarterly average and monthly average concentrations for each census block or block group. A proportional rollback procedure was then used to adjust the set of maximum monthly or quarterly averages to represent meeting a particular NAAQS scenario. That is, the block or block group with the greatest exceedance was reduced to meet the particular NAAQS and all locations were then reduced by that same fraction. After the proportional rollback procedure had been applied to the set of location-specific monthly or quarterly averages to meet a particular NAAQS, these adjusted quarterly or monthly average values were then used to derive annual averages which, in turn, were used in the exposure analysis.

The development of air Pb concentration estimates for this case study is described more fully in Appendix D, Section D.2.

3.1.1.3 Secondary Pb Smelter Case Study

Outdoor air concentration and deposition rates for Pb were estimated for the secondary Pb smelter case study current conditions scenario using the AERMOD dispersion model[1] and source characterization and emissions information for the facility (See Appendix E, Section E.2 for details). The Pb emissions modeled reflected processes at the facility (e.g., stack emissions) and fugitive dust emissions from materials storage and handling and roadway dust.

Annual average Pb concentrations for the current conditions scenario were derived for each census block from model outputs. As with the primary Pb smelter case study, alternative NAAQS scenarios were modeled using the proportional rollback procedure (see Section 3.1.1.2).

The development of air Pb concentration estimates for this case study is described more fully in Appendix E, Section E.2.

3.1.2 Inhalation Exposure Concentrations

Inhalation exposure concentrations for Pb were estimated for young children, the population of interest, from the estimated ambient air concentrations using age group- and location-specific relationships for Pb developed using the exposure modeling component of EPA's 1999 national-scale air toxics assessment (USEPA 2006a), one of the U.S. EPA's National Air Toxics Assessment (NATA) activities. These relationships account for air concentration differences indoors and outdoors and mobility or time spent in different locations (e.g., outdoors at home, inside at home etc.) for the population of interest.

The exposure modeling component of the NATA national-scale assessment generated inhalation exposure concentrations for sets of modeled children for each U.S. Census tract (USEPA, 2006a). For the two point source case studies, we used the ratio of these NATA national-scale assessment inhalation exposure concentrations to that assessment's corresponding estimates of ambient air Pb concentration matched by U.S. Census tract to develop adjustment factors that could be used to derive inhalation exposure concentrations from our estimates of ambient air Pb concentration (see Attachment D, Section D.2.3, and Attachment E, Section E.2.3 for additional detail on this procedure). Although analyses of the ambient air concentrations predicted in the NATA national-scale assessment indicate potential underpredictions, and there are particular uncertainties in the assessment predictions at small scales, the relationship between ambient air concentrations and exposure concentrations (i.e., the comparison used here) is not expected to be affected by these factors. For the general urban case study, we used the median adjustment factor from the full NATA national-scale assessment. The 0-4 years old age group is

[1] AERMOD is the current preferred Gaussian plume dispersion model for assessing stationary sources under the Clean Air Act (70FR(216): 68217-68261).

the closest age group to the age group of interest for this assessment for which outputs are available. For this age group, the adjustment factors or ratios between NATA national-scale assessment Pb inhalation exposure concentrations and ambient air concentrations ranged from 0.37 to 0.46 for the Census tracts within the two point source case study areas. A value of 0.43 was used for the general urban case study (see Appendix C, Section C.1.2). Use of these ratios for the 0 to 4 year old age group to represent the 0 to 7 year old age group modeled in this assessment contributes some uncertainty in the estimate of inhalation exposure concentrations.

3.1.3 Outdoor Surface Soil/Dust Concentrations

Pb concentrations in outdoor surface soil/dust were characterized for the current conditions or current NAAQS scenarios for the three case studies using a combination of modeling and empirical data. These estimates were also used for the alternate NAAQS scenarios. That is, it was assumed that reductions in ambient air concentrations associated with the alternate NAAQS scenarios did not have a significant impact on soil concentrations[2].

3.1.3.1 General Urban Case Study

The outdoor surface soil concentration for the general urban case study was derived after considering empirical data collected both at urban and other residential areas across the United States (Appendix C, Section C.2). A single outdoor soil Pb level representative of general residential yards in the U.S. was then selected for use with this case study. Specifically, a nationally representative arithmetic mean of soil Pb levels in residential yards (198 μg/g) was obtained from the National Study of Lead and Allergen in Housing (NSLAH) (USEPA, 2000). The NSLAH survey, which was conducted by the Department of Housing and Urban Development (HUD) between 1998 and 1999, was intended to generate a nationally representative sample of residential housing, including both private and public residences constructed between 1940 and 1998 (but excluding institutional and group housing).

3.1.3.2 Primary Pb Smelter Case Study

In the primary Pb smelter case study, a different approach was used to estimate outdoor surface soil/dust Pb concentrations near the facility than that used for more distant locations. This difference is in recognition of the remediation activities that have included the removal of contaminated soil at many of the residential yards closest to the facility, and replacement with "clean" soil. Consequently, soil Pb concentrations are estimated using a combination of

[2] As mentioned in Section 2.3.3, this also presumes that implementation methods for any of the alternate NAAQS do not involve taking action to separately change soil Pb concentrations.

measurement data for blocks within the remediation zone and statistically based predictions beyond the remediation zone (see Appendix D, Section D.3 for more detail).

Surface soil/dust Pb levels near the facility (within the remediation zone) are based on the most recent postremediation measurements available for a given block (obtained between 2001 and 2004 - see Appendix B Section, B.1.5.2). Preremediation soil levels are not used in estimating soil levels within the remediation zone. Although analyses of sequential (over time) postremediation measurements have indicated recontamination of remediated yards (USEPA, 2006b), the postremediation measurement based estimates were used for the current NAAQS scenario, and for the alternate NAAQS scenarios. It is recognized that this approach may produce an underestimate of Pb exposure for the ingestion of surface soil/dust for the current NAAQS scenario, and perhaps some alternate NAAQS scenarios. However, an analysis of the impact of a reduced NAAQS on the remediation zone temporal trend in surface soil/dust Pb levels was not available to inform an alternate approach. Further, the impact of such a potential bias is limited to the surface soil/dust pathway and does not affect the indoor dust pathway because the indoor dust Pb concentrations for this region of the primary Pb smelter case study were derived using an approach that did not rely on outdoor soil/dust Pb concentrations.

Characterization of soil levels for blocks and block groups beyond the remediation zone are based on a regression model predicting soil Pb as a function of distance from the facility, which was fitted to preremediation soil measurement data (available closer to the facility). The use of preremediation soil data in deriving the regression equation reflects the fact that little remediation has occurred in these more distant locations and consequently, spatial trends seen in the preremediation soil levels are more likely to be representative for these outer portions of the study area. The regression model used in these estimates has an r^2 of 0.92 which suggests a good fit and increases overall confidence in these statistical estimates. However, it should be noted that this increased confidence holds for areas of interpolation (areas with sampling data used to fit the model – out to about 2.3 km from the facility) more than for areas of extrapolation (areas without sampling data – beyond 2.3 km from the facility).

The development of surface soil/dust Pb concentration estimates for this case study is described more fully in Appendix D, Section D.3.

3.1.3.3 Secondary Pb Smelter Case Study

As noted in Section 2.2.2.2, soil sampling data for Pb were not identified for this case study. Consequently, a hybrid mechanistic-empirical modeling approach was used to characterize soil Pb levels for this case study, with fate and transport modeling employed to derive a soil Pb concentration surface for the study area and sampling data obtained from a surrogate secondary Pb smelter study area employed to adjust (calibrate) that surface.

In lieu of historical estimates of emissions, the fate and transport modeling was performed using current emissions estimates over a period consistent with the operating period of the facility. As the emissions estimates used did not reflect levels of historical emissions, generally believed to have been much higher than current estimates, the resultant Pb concentrations did not reflect current conditions for the location, and, as expected, were lower than concentrations reported for areas near other secondary smelters (see comparisons in Appendix E, Section E.3). Accordingly, the soil concentration surface was scaled up based on a set of factors (differing with distance from the facility) derived from empirical data for a surrogate secondary Pb smelter location. Specifically, Pb concentrations across the entire modeled surface were increased by a factor ranging from1 to 13 (depending on distance from the facility) to obtain surface soil/dust Pb concentrations consistent with those reported in the literature for areas near other secondary Pb smelters. These estimates for the current conditions scenario were also used for the alternate NAAQS scenarios. That is, it was assumed that reductions in ambient air concentrations associated with the alternate NAAQS scenarios did not have a significant impact on soil concentrations.

The development of surface soil/dust Pb concentration estimates for this case study is described more fully in Appendix E, Section E.3.

3.1.4 Indoor Dust Concentrations

Pb in indoor dust can originate from a variety of sources including (a) outdoor soil which is tracked into the house, (b) Pb in outdoor soil which is resuspended into the air and subsequently transported indoors (c) Pb released directly into outdoor air through ongoing anthropogenic activity (e.g., industrial point emissions) which is transported indoors and (d) interior sources of Pb (e.g., paint, hobbies) (Adgate et al., 1998, Von Lindern, 2003). In the exposure assessment conducted for the 1990 Staff Paper, indoor dust Pb concentrations were predicted based on Pb concentrations in outdoor soil and ambient air (USEPA, 1989). This is also the case for the default approach in the exposure component of the IEUBK model (USEPA, 1994a).

The importance of outdoor soil relative to outdoor air in influencing indoor dust Pb levels appears to depend on the nature of the Pb sources involved. Investigations in urban areas and near contaminated waste sites with elevated soil Pb levels without a currently active industrial point source emitter of Pb have indicated a greater association of measurements of dust Pb concentration with measurements of soil Pb concentration than with measurements of ambient air concentration (e.g., Adgate, 1998 and Von Lindern, 2003). By contrast, investigations in areas with currently operating large point sources of Pb (e.g., active Pb smelters) have implicated ambient air Pb as an important source of Pb to indoor dust (Hilts, 2003). Contributions of

ambient air Pb to indoor dust Pb levels have also been illustrated by a deposition study conducted in New York City (Caravanos et al., 2006). Caravanos and others described Pb deposition indoors resulting primarily from exterior environmental sources and not from interior Pb sources. For additional discussion of the relationship between indoor dust Pb, outdoor ambient air Pb and other related factors, refer to Appendix G, Section G.2

The prediction of indoor dust Pb based on Pb concentrations in outdoor ambient air and other media (e.g., outdoor soil/dust Pb, indoor paint) can be conducted using empirical models, mechanistic models, or by a combination of both techniques. Empirical models (typically implemented as regression models) have the advantage of being able to specify a relationship between indoor dust Pb and predictor variables (e.g., outdoor soil/dust, outdoor air) even when these relationships are complex and uncertain. However, statistical models have the disadvantage of requiring a significant amount of site-specific data for their derivation and not being well suited to extrapolation to scenarios with conditions different from those underlying their development. Mechanistic models, by contrast, can be developed in the absence of extensive site-specific data and are not as limited in the types of scenarios to which they can be applied as long as those scenarios are conceptually consistent with the scenario underlying their development. However, the development of mechanistic models can be quite challenging and subject to uncertainty if the system or process being predicted is complex and uncertain.

To date, efforts to predict indoor dust Pb have focused primarily on the development of empirically-based regression models (EPA, 1989). Furthermore, most of these regression models have been based on data associated with large industrial point sources (smelters) and their impacts on surrounding residential populations. Little progress has been made in developing comparable models for areas where ambient Pb levels are not so greatly influenced by a large industrial point source, such as in more general urban residential areas, including either regression models specific to these more general urban scenarios, or mechanistic models which could be applied more readily to different exposure scenarios including urban residential populations.

In this assessment, a combination of (a) statistical (regression) models obtained from the literature, (b) statistical regression model developed specifically for individual case studies and (c) mechanistic models (developed specifically for the general urban case study) was used in predicting indoor dust Pb. This reflects the fact that varying amounts of site-specific data were available across the three case studies for characterizing indoor dust Pb and related factors. In addition, the absence of statistical regression models in the literature specifically focused on urban residential locations necessitated the development of a hybrid mechanistic-empirical model for the general urban case study (see below). The approaches used to predict indoor dust

Pb for each of the case studies are presented below and described in greater detail in Appendix G.

3.1.4.1 General Urban Case Study

Two models are used to estimate indoor dust Pb in the general urban case study: a hybrid mechanistic-empirical model (the hybrid model), and a regression-based model. Application of these models with the ambient air Pb concentrations for this case study produced two sets of indoor dust Pb estimates for each air quality scenario (see Appendix C, Section C.3).

The hybrid model uses a mechanistic model to predict indoor dust Pb resulting from the infiltration of outdoor air containing Pb into indoor residential air with subsequent contribution to indoor dust Pb. This portion of indoor dust Pb derived with this model is subsequently referred to in this document as the recent air or recent ambient air related component or contribution to indoor dust Pb. To the extent that outdoor air Pb includes contributions from resuspension of historically deposited Pb, it is represented here. Other contributions to indoor dust Pb (e.g., tracking of outdoor soil/dust indoors, indoor paint flaking, etc.) are addressed using an empirically based estimate derived from a national-scale dataset characterizing indoor dust Pb loadings in U.S. residences (the U.S. Housing and Urban Development [HUD] National Survey of Lead-based Paint in Housing - USEPA, 1995). When combined, the recent air component (derived using the mechanistic model) and the other contributions component (derived using the HUD dataset) provide an estimate of total indoor dust Pb for the general urban case study (Appendix C, Exhibit C-7). Note that while the indoor dust Pb concentration contributed by the hybrid model's recent air Pb component will vary, depending on the ambient air Pb level, the concentration contributed from other sources will remain constant across the different air quality scenarios. The two components of the hybrid model (the mechanistic and empirical) are described in greater detail below.

The mechanistic model linking ambient air Pb to indoor dust Pb was developed by obtaining the steady state solution to a dynamic mass balance equation that predicts Pb in both indoor air and indoor floor dust as a function of outdoor air Pb. This dynamic mass balance equation was developed specifically for this assessment. In recognition of the complexity of the larger task of simulating contributions to indoor dust Pb from all sources, the mechanistic model development activity was limited to the area considered most essential to the needs of this assessment, i.e., the contribution to indoor dust Pb from recent ambient air. See Appendix G, Section G.3.2 for additional detail on the derivation of the mechanistic ambient air-to-indoor dust Pb model.

As noted above, contributions of other sources to Pb in indoor dust were addressed in the hybrid model using empirical data (rather than trying to model them mechanistically). The HUD

dataset, described above, that characterized the national distribution of residential indoor dust Pb (USEPA, 1995) was selected as the basis for this other sources component of indoor dust Pb. Specifically, a median value from the HUD dataset characterizing residential indoor dust Pb loadings (USEPA, 1995) was identified, and the ambient air related component of this indoor dust value estimated and subtracted from the HUD median value, leaving only the other sources component. This provided a central tendency estimate of the component of indoor dust Pb loadings associated with sources other than recent air Pb, for typical residences in the United States (see Appendix G, Section G.3.3 for additional detail). As noted earlier, this single estimate of other sources indoor dust Pb is used for modeling all air quality scenarios, with no change associated with the ambient air Pb level being evaluated.

The hybrid model generates estimates of indoor dust Pb in terms of loading, rather than concentration, while concentration is the form required by the blood Pb models used in this analysis. Conversion from loading to concentration was accomplished using a log-log regression equation derived from the HUD dataset described above. This dataset has matched data for sampled residences for both indoor dust loadings from vacuum samples, and indoor dust concentrations. Use of the HUD dataset based loading to concentration conversion required an additional conversion between loading estimates based on wipe samples (the form used by the hybrid model) and those based on vacuum samples (the form of the HUD data). This was accomplished using an equation developed by EPA (USEPA, 1997). Details on both conversions are provided in Appendix G, Sections G.3.4 and G.3.4.1.

The individual elements of the hybrid model, including both the mechanistic and empirical components as well as the loading-to-concentration conversion equations are presented in Table 3-2. The final hybrid equation (including all components) is presented last.

Table 3-2. Hybrid model for indoor dust Pb in general urban case study.

Model component	Formula
Hybrid mechanistic equation for "recent" air	FLOOR LOADING (PbWipe) = 104.2 * PbAIR Where, PbAIR is Pb in outdoor ambient air, and FLOOR LOADING is in terms of wipe sample loading.
Hybrid empirical component for other sources	1.15 $\mu g/m^2$
Converting wipe loadings to vacuum loadings	$PbVAC = 0.185 * PbWIPE^{0.921}$
Converting vacuum loadings to concentration	Ln(PbCONC) = 4.92 + 0.52 * ln(PbVAC)
Final combined hybrid equation	$PbDust = EXP [4.92 + 0.52 * Ln (0.185 * (104.2 * PbAir + 1.15)^{0.931})]$

Because the hybrid model has not been subject to extensive review and application outside of this analysis and given the influence of indoor dust Pb on Pb exposure and risk, dust Pb concentrations for the general urban case study are also estimated using an additional dust Pb model (see Appendix G, Section G.3.5). The use of two models is intended to inform our characterization of model uncertainty in this key portion of the analysis. Specifically, we have included the air-only regression model (EPA, 1989) as a second, parallel approach in predicting indoor dust Pb. That model estimates indoor dust Pb based on (a) outdoor ambient air Pb (multiplied by an air-related factor) and (b) an intercept which captures other impacts besides air (e.g., indoor paint). The air factor used in this equation is expected to capture longer-term impacts of outdoor air Pb on indoor dust, including the indirect effect of air Pb on outdoor soil/dust Pb with subsequent impacts of that outdoor soil/dust Pb on indoor dust Pb through other mechanisms (EPA, 1989). The air-only regression model is presented below:

$PbDUST(mg/kg \text{ or } ppm) = 60 + 844 * PbAIR(\mu g/m^3)$

3.1.4.2 Primary Pb Smelter Case Study

We used different regression models for predicting Pb concentrations in indoor dust in areas near the primary Pb smelter facility where soil has been remediated and more distant areas. For the remediation zone near the facility, a regression equation was developed using dust Pb measurements which had been collected from some of the houses within this area. These data, while adequate for development of a site-specific regression model, did not have sufficient spatial coverage to be used alone to represent indoor dust Pb levels for that portion of the study area. For the remainder of the study area, we employed a regression equation developed for the

last review (USEPA, 1989). Because of the presumed impact of the remediation activity on dust Pb, the site-specific dust Pb model developed for the remediation zone was not considered appropriate for use in areas beyond that area.

The dataset used to develop the model for the remediation zone was based on indoor dust samples collected in 17 houses within the remediation zone. Independent variables included in the analysis were: (a) estimated annual average Pb concentrations in air at census block centroids located within 200 meters of each of the 17 houses, (b) road dust Pb measurements for locations within 300 meters of each house and (c) postremediation residential soil Pb measurements for the yard of each house. Preremediation soil Pb concentrations were not included in the regression analysis since they were not expected to represent current conditions at the site. Multiple samples for each medium associated with a specific house within the dataset (e.g., reflecting multiple samples collected over time) were averaged to produce a "temporally averaged" value. A number of regression models were evaluated, (see Appendix G, Section G.4), and the “H5” model was ultimately selected based on goodness of fit and other considerations. This model relates the natural log of indoor house dust to the natural log of ambient air Pb (r^2=0.625):

ln(house dust, mg/kg or ppm) = 7.7892 + 0.7200*ln(air Pb, $\mu g/m^3$)

In applying the H5 model to the remediation zone portion of the study area, a constraint was applied in recognition of the our application of this regressions to scenarios with much lower air Pb than that for the dataset on which it is based, and findings for similar towns of residual levels of Pb in house dust that reflect historical contributions (e.g., von Lindern et al., 2003; Hilts, 2003). A floor value for the predicted dust Pb for alternative NAAQS scenarios was assigned to any model predictions falling below it. This value was 60 ppm, which is the intercept for the air only regression model derived at the time of the last review, see previous section. While this value may be an underestimate of the historical component of Pb in house dust of existing houses or houses existing prior to remediation, its use is intended to balance the need for a floor for such houses and the potential for the population to also have newer houses. The use of and need for this floor recognizes the uncertainty associated with this model. Additionally, the regression model used here does not lend itself to partitioning the recent air related Pb from other contributions (discussed in Section 2.4.3); accordingly, this partitioning is not done for the primary Pb smelter case study.

Several points regarding the other variables considered for the remediation zone regression are noted here. For example, road dust Pb concentration was not found to have significant predictive power for indoor dust Pb. This may reflect the fact that the road dust Pb dataset does not provide significant coverage for homes located near to the truck haul routes. Additionally, yard soil Pb concentration was found to be slightly, and statistically significantly, negatively correlated with indoor dust Pb levels. This counterintuitive finding may be a result of

the existence within the remediation zone of a patchwork of remediated yards, such that the remediation activity may have interfered with any correlation between yard soil Pb levels, ambient air Pb levels and indoor dust Pb levels that might have existed previously. The resulting slight negative correlation of dust Pb levels with soil Pb levels led us to exclude soil Pb in predicting indoor dust Pb, leading to the selection of the H5 model, which only considers ambient air Pb in predicting indoor dust Pb. The y-intercept for the selected model may reflect a number of factors not correlated with ambient air or distance from the facility, such as a general level of soil Pb contamination in the area and indoor Pb paint.

For areas beyond the remediation zone, we used a regression equation developed during the last review from data collected at a number of operational primary Pb smelters, including this case study location (USEPA, 1989, Appendix B). This model, the "AGG" or "aggregate" model, predicts indoor dust Pb concentration from both outdoor soil and ambient air Pb concentrations. We have selected the AGG model for the nonremediation portion of the primary Pb smelter case study area since this area has not been subjected to extensive remediation and is therefore likely to resemble the locations included in the pooled dataset used in deriving this model in terms of relationships among air, outdoor surface soil/dust and indoor dust. The AGG air and soil regression model (USEPA, 1989), selected for areas beyond the remediation zone is the following:

House dust (mg/kg or ppm) = 31.3 + 638*air Pb ($\mu g/m^3$) + 0.364*soil Pb (mg/kg)

3.1.4.3 Secondary Pb Smelter Case Study

A version of the empirical regression model used for the primary Pb smelter (USEPA, 1989) was also used for the secondary Pb smelter case study. In this case study, an air-only version of the model (USEPA, 1989) was employed reflecting the reduced overall confidence associated with soil characterization at this case study (see Section 3.1.3.3). The AGG model for estimating indoor dust (USEPA, 1989) was derived in two forms including an air-only model that based indoor dust concentrations on outdoor ambient air Pb (without explicitly considering outdoor soil Pb levels) and an air+soil model which based estimates on both outdoor soil and ambient air Pb data. It is important to note, however, that the air-only model implicitly reflects some consideration for the air-to-soil-to-indoor dust mechanism in the air signal. Specifically, the larger air factor for the air-only model relative to the air+plus dust model's air factor, reflects contribution of air Pb both directly to dust through penetration indoors and subsequent deposition to surfaces and indirectly to dust through deposition to outdoor soil which impacts indoor dust (USEPA, 1989). This air-only regression model (USEPA, 1989) is as follows:

House dust (mg/kg or ppm) = 60 + 844*air Pb ($\mu g/m^3$)

The model used for this case study was based on a number of studies focusing mainly on primary Pb smelters (a number of primary Pb smelters were operational at the time of model development). This may introduce uncertainty into indoor dust Pb predictions generated for this case study to the extent that factors related to the dependency of indoor dust Pb on ambient air Pb, such as particle size profiles and the nature of the airborne Pb compounds, differ for primary versus secondary Pb smelters.

3.2 METHODS FOR ESTIMATING BLOOD PB LEVELS

This section presents the methodology used to estimate blood Pb levels in the child study populations. The section begins with an overview of the primary biokinetic model used in this full-scale risk analysis, the Integrated Exposure and Uptake Biokinetic model (USEPA, 1994a). Findings associated with the use of both the IEUBK and Leggett models in the pilot analysis, in addition to subsequent performance evaluation analyses (see Appendix J) contributed to the decision to use IEUBK as the primary blood Pb model and reserve the Leggett model for use in the sensitivity analyses. In addition, we have considered an empirical slope model (the Lanphear model) in conducting our performance evaluation (Appendix J, Section J.2).

Following the overview of the IEUBK biokinetic model, the probabilistic approach used to generate population-level distributions of blood Pb levels for each study population is described. The section ends with a discussion of the GSDs used within each case study to reflect interindividual variability in behavior related to Pb exposure and Pb biokinetics (a key component in modeling population-level blood Pb).

3.2.1 Blood Pb Modeling

Blood Pb models are used in the assessment in order to (a) apportion exposure, the metric for which is blood Pb, between policy-relevant Pb exposures and policy-relevant background, and (b) estimate potential changes in blood Pb level distributions that would result from alternate ambient air Pb levels.

As discussed in Section 4.4.1 of the CD, there are two broad categories of blood Pb models available to support exposure and risk assessment:

- Statistical (regression) models, which attempt to apportion variance in measured blood Pb levels for a study population to a range of determinants or control variables (e.g., surface dust Pb concentrations, air Pb concentrations). The development of these models requires paired predictor-outcome data which restricts these empirical models to the domain of their observations (i.e., to applications involving the study population(s) and exposure scenarios used in their derivation or at least to scenarios very similar to the original study conditions).

- Mechanistic models, which attempt to model the process of transfer of Pb from the environment to human tissues. While these models are considerably more complex compared with the regression models (in terms of both the number of variables and their computational structure), by incorporating variables that vary temporally and spatially, or across individuals or populations, mechanistic models can be extrapolated to a wide range of scenarios, including those outside of the original populations and exposure scenarios used to develop/parameterize the models.

Given concerns over applying regression models to populations and exposure scenarios other than those used in their derivation, and consistent with recommendations from CASAC (see Section 1.4), we have relied primarily on mechanistic models in conducting the exposure analysis for this assessment. Additionally, a regression model developed by Lanphear et al. (1998) and described in Appendix H, Section H.2.3.was included in the blood Pb modeling performance evaluation (Appendix J, Section J.3.2). The CD (Section 4.4) describes three mechanistic (biokinetic) models developed over the past several decades including IEUBK for modeling child Pb exposure (CD, Section 4.4.5 and Appendix H, Section H.2.1) and two models designed to simulate Pb biokinetics and blood Pb levels from birth through adulthood, the Leggett model (CD, Section 4.4.6) and the model developed by O'Flaherty (CD, Sections 4.4.7). The Leggett and O'Flaherty models simulate smaller time steps and consequently demonstrate blood Pb responses on much shorter time scales than the IEUBK model, the outputs for which are described as indicative of quasi steady-state conditions (CD, Sections 4.4.5-4.4.7). All three models have the potential for application in Pb risk assessment and have been evaluated to varying degrees using empirical datasets (CD, Section 8.3.4). Based on results of the pilot analysis, subsequent performance evaluations, and advice from CASAC, we selected the IEUBK model as the primary blood Pb model for the full-scale analysis. The Leggett model has been included as part of the sensitivity analysis intended to assess the potential impact of uncertainty in blood Pb modeling on the overall analysis (See Section 4.3.2). A brief overview of the IEUBK and Leggett models is presented below.

3.2.1.1 Primary Analysis

The IEUBK model was selected for use in generating the primary set of exposure and risk results. IEUBK is a multicompartment pharmacokinetics model for children 0 through 84 months of age (the first 7 years of life), which predicts average quasi-steady state blood Pb concentrations corresponding to daily average exposures, averaged over periods of a year of more. The exposure module provides average daily intakes of Pb (averaged over a 1 year time increment) for inhalation (air, including consideration for both outdoor and indoor) and ingestion (soil, indoor dust, diet and water) (Section 4.4.5.1 of the CD). The model is intended to be applied to groups of children experiencing similar levels of Pb exposure and to generate a central

tendency blood Pb estimate for that group (USEPA, 1994a). In applications of IEUBK, interindividual variability in biokinetics and behavior (e.g., varying rates of dietary Pb ingestion) of the study population is typically characterized through the incorporation of a GSD which, together with the IEUBK-generated blood Pb level, provides a distribution of blood Pb levels for a group of modeled children.

For each exposure zone in each case study air quality scenario, estimates of the concurrent and lifetime average blood Pb level are derived from the outputs of the IEUBK model as described in Appendix H. Briefly, the concurrent metric is derived as the average over ages 73 to 84 months (approximately 6 to 7 years of age)[3], and the lifetime average metric is the average of blood Pb levels between ages 6 and 84 months.

Additional detail on the IEUBK model is described in Appendix H (Section H.2.1) and in Section 4.4.5 of the CD. Application of the IEUBK model is described in Appendix H, Section H.3.1, and input parameter values, and their basis, are described in Appendix H, Section H.4.

3.2.1.2 Sensitivity Analysis

The Leggett model has been included as part of the sensitivity analysis (Appendix L) and in model performance evaluations (Appendix J), but has not been used in generating primary exposure and risk results for this assessment. Originally developed from a model designed to simulate radiation doses for bone-seeking radionuclides, the Leggett biokinetic model has a temporal resolution of one day and can model exposure from infancy through adulthood (CD, Section 4.4.6). The daily resolution in Leggett allows more comprehensive treatment of the temporal pattern of exposure and its shorter-term impact on blood Pb levels than IEUBK, although for this assessment, which focuses on longer-term trends in Pb exposure, this functionality is less relevant. The Leggett model does not include a detailed pathway-level exposure module as does IEUBK. Rather the Leggett model takes total ingestion and inhalation exposure estimates as inputs. However, it is possible to link the Leggett model to a more detailed pathway-level exposure model, thereby allowing a more detailed treatment of Pb exposure pathways and their impact on blood Pb. The use of this type of external exposure model including pathway-specific modeling of exposure levels was implemented for the pilot analysis. As with IEUBK, Leggett can be used to derive central tendency blood Pb levels for groups of similarly exposed children. The same GSD used for IEUBK is then used to produce

[3] As described in Appendix I (Section I.2.1), the concurrent metric is calculated by averaging blood Pb estimates generated for ages 75 and 81 months with these estimates representing the first and second halves of the 7th year of life, respectively. Use of the 7th year of life is consistent with or similar to the average age of the IQ testing in the Lanphear et al. (2005) study on which the concentration-response function is based (Sections 2.1.5 and 4.1).

estimates of the distribution of blood Pb levels within study populations. For additional details on the Leggett model see Section 4.4.6 of the CD and Appendix H.

3.2.2 Exposure Pathway Apportionment and Probabilistic Population Modeling

This section describes the method used to estimate contributions to blood Pb and IQ from exposure pathways of interest (see Section 2.4.3), and the method used for probabilistic population modeling. Both are applied to the central tendency estimates of blood Pb developed for the two blood Pb metrics (concurrent and lifetime average) from the IEUBK model outputs.

To the extent feasible with the modeling tools and assessment design, estimates of the contribution to blood Pb (central tendency estimate) are developed for policy-relevant background versus policy-relevant exposures (Section 2.4.3). This is done by considering blood Pb model estimates for an exposure zone derived using only the pathways of interest (Appendix I, Section I.1). As discussed in Section 2.4.3, there are limitations on the resolution to which policy-relevant exposures can be distinguished which results in some simplifying assumptions. We developed estimates of contribution to blood Pb estimates (and IQ estimates) for the following pathways (or pathway combinations):

- Inhalation of ambient air Pb (i.e., "recent air" Pb): This is derived using the blood Pb estimate resulting from Pb exposure limited to the inhalation pathway.
- Ingestion of "recent air" indoor dust Pb: This is derived using the blood Pb estimate resulting from Pb exposure limited to ingestion of the Pb in indoor dust that is predicted to be associated with ambient air concentrations (i.e., via the air concentration coefficient in the regression-based dust models or via the mechanistic component of the hybrid blood Pb model (Section 3.1.4). For the primary Pb smelter case study, estimates for this pathway are not separated from estimates for the pathway described in the subsequent bullet due to uncertainty regarding this categorization with the model used for this case study (Section 3.1.4.2).
- Ingestion of "other" indoor dust Pb: This is derived using the blood Pb estimate resulting from Pb exposure limited to ingestion of the Pb in indoor dust that is <u>not</u> predicted to be associated with ambient air concentrations (i.e., that predicted by the intercept in the dust models plus that predicted by the outdoor soil concentration coefficient, for models that include one (Section 3.1.4)). This is interpreted to represent indoor paint, outdoor soil/dust, and additional sources of Pb to indoor dust including historical air (see Section 2.4.3). As the intercept in regression dust models will be inclusive of error associated with the model coefficients, this category also includes some representation of dust Pb associated with current ambient air concentrations (described in previous bullet). For the primary Pb smelter case study, estimates for this pathway are not separated from estimates for the pathway described above due to uncertainty regarding this categorization with the model used for this case study (Section 3.1.4.2).

- Ingestion of outdoor soil/dust Pb: This is derived using the blood Pb estimate resulting from Pb exposure limited to ingestion of outdoor soil/dust Pb.
- Ingestion of drinking water Pb: This is derived using the blood Pb estimate resulting from Pb exposure limited to ingestion of drinking water Pb.
- Ingestion of dietary Pb: This is derived using the blood Pb estimate resulting from Pb exposure limited to ingestion of dietary Pb.

The goal of this probabilistic exposure modeling is to generate population-level distributions of blood Pb levels that allow (a) specific percentiles of exposure (e.g., 50th, 90th, and 95th) within a study population to be identified. These are presented along with the differentiation by exposure pathway (e.g., policy-relevant background versus policy-relevant exposures, with the latter further differentiated as to ambient air inhalation, indoor dust ingestion and outdoor surface soil/dust ingestion, as indicated above). Therefore, for example, we may have an estimate of exposure for the 95^{th} percentile child in the primary Pb smelter case study, with that blood Pb level differentiated as to the fraction coming from (a) diet and drinking water, (b) recent air Pb, including ambient air inhalation and ingestion of the recent air component of indoor dust Pb, with this fraction potentially including resuspended, previously deposited Pb (see Section 2.4.3), and (c) other sources including outdoor soil/dust and historical air Pb, as well as indoor paint. The effort to differentiate the recent air and other sources of Pb to indoor dust is subject to different degrees of uncertainty for the various case studies, reflecting the different approaches used in modeling indoor dust (see Sections 2.4.3 and 3.1.4). Furthermore, it is noted that given the various limitations of our modeling tools (Sections 2.4.3), blood Pb levels associated with air-related exposure pathways and current levels of Pb emitted to the air (including via resuspension) are likely to fall between the estimates for "recent air" and those for "recent" plus "past air".

Probabilistic exposure modeling differs for the two point source case studies and the general urban case study. Because the two point source case studies are location-specific, demographic data for each study area are used to evaluate the interaction between child populations in those study areas and the distribution of Pb concentrations in each media (e.g., outdoor ambient air, outdoor soil/dust and indoor dust). By contrast, because the general urban case study is not location-specific, probabilistic exposure modeling does not involve location-specific demographic data and instead, is based on the assumption of a uniformly distributed child receptor population contacting media with uniformly distributed Pb.

3.2.2.1 General Urban Case Study

The approach used to generate exposure distributions for the general urban case study is simpler than that used for the two point source case studies (Section 3.2.2.2), due to the use of a uniformly distributed population and spatially uniform media concentrations of Pb in the design of this case study. This design negates the need for population-weighted sampling in generating the exposure distribution. Instead, the central tendency blood Pb level generated for a specific air quality scenario using IEUBK, is combined deterministically with the GSD reflecting interindividual variability in Pb exposure and biokinetics to produce a population-level distribution of blood Pb levels.[4] Specific percentile exposure levels are then identified from that distribution, with these levels interpreted as representing modeled individual children for the general urban case study (i.e., estimates conceptually equivalent to those generated for the two point source case studies). However, because the analysis is not location-specific, population incidence estimates are not generated.

The study area for the general urban case study can be considered a single exposure zone with a single IEUBK-derived central tendency blood Pb level that is combined with the GSD to produce the population-level exposure distribution for that study area. As is the case with individual exposure zones modeled for the point source case studies, the entire child population in the single exposure zone modeled here is given the same pathway apportionment. As with the point source case studies, this approach introduces uncertainty into the analysis, especially for simulated individuals with high-end blood Pb levels, since pathway apportionment would likely not be the same for all individuals in an exposure zone, even if all were exposed to the same Pb concentrations in each medium.

3.2.2.2 Point Source Case Studies

Probabilistic exposure modeling for the two point source case studies relied on information in three areas as summarized below:

- Central tendency blood Pb levels for each exposure zone: Outputs from the biokinetic blood Pb modeling (considered to be central tendency estimates) are used to produce central tendency estimates of concurrent and lifetime average blood Pb levels for each exposure zone (i.e., census block or block group) in each case study area.

[4] Note that as discussed in Section 3.2.3.1, the range of GSDs used in the general urban case study to reflect interindividual variability in lead exposure and biokinetics has been selected to include some degree of coverage for small-scale variation in media concentrations. Therefore, while the overall scenario modeled in the general urban case study is based on the assumption of generally uniform media concentrations (e.g., an ambient air concentration level which is generally constant across the study area), the use of larger GSDs provides some coverage for residence-to-residence variation in these media concentrations.

- Demographics (child distribution within study areas): The distribution of children (<7 yrs old) within each case study area is used to insure that the generation of population-level blood Pb level distributions for each case study reflects where children are located.

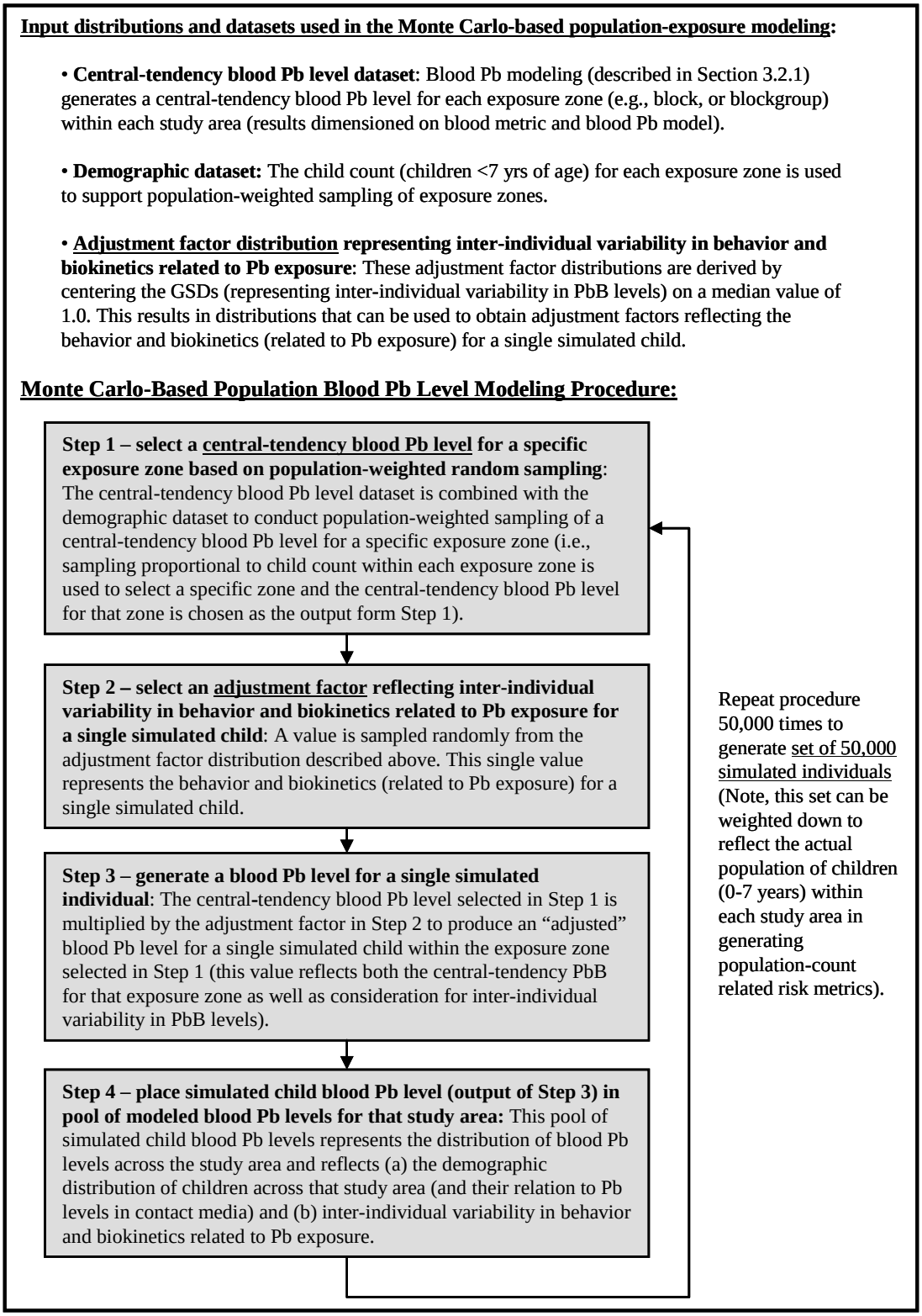
- GSD reflecting interindividual variability in blood Pb levels: A GSD is used to reflect interindividual variability for blood Pb levels in groups of similarly exposed children (i.e., within each exposure zone of a case study area). The GSD is combined with the central tendency blood Pb level estimates to generate a distribution of blood Pb levels for the group of children located in each exposure zone.

The stepwise procedure used to generate population-level blood Pb distributions for each of the point source case studies is illustrated in Figure 3-1, with the information described in the bullets above recognized as input data.

Several points related to implementation of this procedure are noted. For this assessment, 50,000 simulated individuals were generated for each point source case study in order to insure that the population-level blood Pb distributions generated met target stability goals (see Appendix M, Section M.2.2).[5] This simulation count represents a higher total child count than actually is associated with either study area. Using a higher number of simulated individuals was necessary, however, to generate blood Pb distributions with "stable" higher-end exposure estimates. If simulations matching the actual population count at each case study had been conducted, the distributions that would have resulted would have been "unstable" at higher percentiles. It is important to note, however, that child population count estimates for individual percentiles (Section 3.4) have been scaled to reflect the actual child count associated with each study area.

[5] The analysis of simulation stability focused on the general urban case study and demonstrated that even the highest population percentiles generated for this analysis (95th percentile) were relatively stable with coefficients of variation of less than 1% for total IQ loss estimates (see Appendix M, Section M.2.2).

Figure 3-1. Procedure for generating population blood Pb distributions for point source case studies.



An additional point of clarification relates to the process used in differentiating specific percentile total blood Pb levels into pathway-specific fractions for the two point source case studies. All simulated individuals associated with a given exposure zone (i.e., U.S. Census block or block group), were assigned the same pathway-specific apportionment, which was derived from the blood Pb modeling performed for the exposure zone. The set of simulated individuals with a range of total blood Pb levels, produced subsequently via application of the GSD, are assumed to all have the same pathway-specific apportionment of those blood Pb levels (i.e., the same apportionment as that generated for the central tendency blood Pb level modeled for that exposure zone). In reality, it is likely that pathway apportionment would vary across children with different blood Pb levels located in the same exposure zone (e.g., the contribution of indoor dust exposure to total blood Pb might differ for kids living near each other who demonstrate different total blood Pb levels). The modeling approach used, however, does not provide pathway apportionment within an exposure zone, only across exposure zones (i.e., each exposure zone has a different pattern of pathway apportionment for its simulated children).

The modeling approach presented in Figure 3-1 and described above, generates a population-level distribution of total blood Pb levels with pathway apportionment as described above. These distributions are used to generate several types of exposure metrics including:

- Population-weighted exposure percentiles: total blood Pb levels (with pathway apportionment) for simulated individuals representing specific points along the population blood Pb distribution (e.g., 50, 90, and 95th percentile).

- Incidence counts: number of children within a given study area projected to experience a specific degree of Pb exposure (total blood Pb level).

3.2.3 GSD for Population Blood Pb Modeling Procedure

A key aspect of the population-level blood Pb modeling for all three case studies is the application of the GSD reflecting interindividual variability in blood Pb levels. This GSD reflects a number of factors which operate together to produce interindividual variability in blood Pb levels, including: (a) biokinetic variability (differences in the uptake, distribution or clearance of Pb), (b) differences in behavior related to Pb exposure (e.g., varying hand-to-mouth activity, tap water ingestion rates, and time spent playing indoors) and (c) differences in environmental Pb exposure concentrations (e.g., spatial gradients in ambient Pb levels of a resolution beyond that simulated in each case study, differences in cleaning/vacuuming rates and air exchange rates).

GSDs will tend to be larger for more diverse populations and/or larger study areas, reflecting the potential for greater variability in the factors listed above. Specifically, more diverse populations will tend to demonstrate greater diversity in behavioral and biokinetic factors

related to Pb exposure, thereby producing greater variation in blood Pb levels. Larger study area will tend to produce greater GSDs due both to more diverse populations, with their greater behavioral and biokinetic variability, as well as greater variation in Pb media concentrations across the study area.

The dramatic reduction in blood Pb levels among children in the United States that was observed between the NHANES surveys of the late 1970s and those of the 1980s and later was accompanied by an increase in GSD (see Appendix H, Exhibit H-7). Possible reasons for this include the likelihood that, as blood Pb levels decrease, a wider variety of exposure pathways begin to play a role in determining overall blood Pb levels (at higher blood Pb levels, it likely that one, or a few related pathways dominate exposure). As more pathways come into play, the potential for interindividual variability in behavior and biokinetics related to these pathways increase, thereby producing greater variability in blood Pb levels (see Section 4.2.2 of EPA, 1994b). Another possible explanation for the increase in GSDs is that, while overall Pb exposure levels have decreased, some fraction of children nationwide continue to be exposed to Pb paint and Pb in drinking water (associated with Pb solder used in older plumbing). These higher nonair-related exposures can produce elevated blood Pb level, especially when compared to average blood Pb levels in the current general population. Therefore, while the geometric mean blood Pb level may have decreased, the tail of the distribution may have remained anchored (for these paint and drinking water exposed children) resulting in a larger GSD.

A number of studies have been conducted over the past three decades which provide insights into interindividual variability in Pb levels under various exposure conditions. Many of the studies from the 1970's and 1980's focused on populations living near smelters with fairly elevated blood Pb levels compared with levels modeled for our three case studies. For example, as seen in Appendix H, Exhibit H-7, geometric mean blood Pb levels for populations near active smelters in the past tended to exceed 8 µg/dL with GSDs on the order of 1.7. Note, that these earlier studies do not have readily available summaries of blood Pb levels for the metrics of interest in this analysis (concurrent and lifetime average metrics) and instead, provide more generalized summaries based on the individual measurements collected.

Beginning in the 1980's and extending through the 1990's, a number of studies were conducted focusing on urban populations in specific U.S. cities (e.g., Lanphear et al 2005). These studies, which were intended to examine the link between Pb exposure and neurological effects in children, have readily available blood Pb summary data for both concurrent and lifetime average blood metrics. As can be seen in Appendix H, Exhibit H-9, geometric mean blood Pb levels vary significantly across these studies, with concurrent values ranging from 5.5 µg/dL to 14.5 µg/dL and lifetime average values ranging from 4 µg/dL to 14.2 µg/dL. GSDs also vary

across the studies with GSDs of 1.4 to 1.7 reported for the lifetime average metric and 1.5 to 1.9 for the concurrent metric.

In addition to these smelter-related and city-specific studies, the CDC has also conducted several iterations of its NHANES national-scale survey over the past three decades which track changes in the national distribution of blood Pb levels among children in the U.S. (see CD, Section 4.3.1.3 and Appendix H). As mentioned above, between the earliest NHANES surveys and the later (post 1980) surveys average blood Pb levels in children in the U.S. decreased dramatically (following initiatives to remove Pb from gasoline and other products in the late 1970's and 1980's), and the GSD increased significantly. This is most pronounced in the geometric mean blood Pb levels and associated GSDs reported for the first NHANES survey (1967-1980) (CDC, 2005) and the second NHANES survey (1988-1991) (CDC, 2005) with blood Pb levels decreasing from 14.9 to 3.6 and GSDs increasing from 1.4 to 2.1.

GSDs were selected for each case study based on consideration for the study data summarized above. The selection of these GSDs reflected consideration for a number of factors including: (a) the type of study area and underlying population involved (e.g., point source versus more general urban area), (b) the fact that all three study areas use exposure zones with fairly uniform media concentrations and that the GSD selected will be used to represent blood Pb variability within each of those zones (c) age of the underlying survey population (the goal was to match the survey population to our study population to the extent possible), and (d) date of the survey (generally there is a desire, when possible, to use studies that are more contemporary to capture any underlying downward trends in blood Pb levels which have occurred). The GSDs for each of the case studies, as well as the rationale for their selection, is presented below.

3.2.3.1 General Urban Case Study

For this case study, two sets of population blood Pb estimates are developed for each blood Pb metric. This was done using GSDs intended to reflect: (a) a more uniform population living in a smaller urban area (represented by a smaller GSD for each metric) and (b) a more diverse urban population living in a larger urban area (represented by a larger GSD for each metric). Together, these two sets of estimates provide a range of results for each blood Pb metric intended to reflect uncertainty associated with this key component of the analysis (i.e., interindividual variability in blood Pb levels). The lower-bound GSDs (representing the more uniform, smaller urban population) were obtained from the Boston study (Bellinger, 1992) (i.e., concurrent GSD of 1.7 and a lifetime average value of 1.6). This study represents one of the more contemporary of the urban studies focusing on a smaller population. By contrast, the upper-bound GSDs (representing the larger, more diverse population) were obtained using NHANES IV (CDC, 2005). Here, the GSD from NHANES IV for 1-5 yr olds (2.1) is used for

the concurrent metric, and the GSD for the lifetime average metric (2.0) was derived by scaling the value for the concurrent metric using the relationship between concurrent and lifetime seen in the smaller urban-scale Boston study (Bellinger, 1992).

3.2.3.2 Point Source Case Studies

A critical consideration in identifying the GSD for use in the point source case studies, is the fact that each is modeled using a spatial template that divides the study area into exposure zones with relatively uniform media concentrations. As noted earlier, the GSDs are used to reflect interindividual variability in blood Pb levels for the group of modeled children located within each of those zones. Therefore, the GSD does not need to provide full coverage for media concentration variability and its impact on exposure, since this is covered to some extent by the spatial template. Therefore, a larger GSD such as that suggested by NHANES would likely overstate variability and a smaller GSD, possibly in line with values from smaller-scale studies such as the smelter or city-specific studies would seem to be more appropriate. Following this logic, a GSD of 1.7 was selected for the concurrent blood metric, based on consideration for the range of values from these smaller-scale studies. A matching lifetime average metric GSD value of 1.6 was selected, also based on the city-specific studies.

3.3 ESTIMATED MEDIA CONCENTRATIONS

This section summarizes the media concentration estimates for all air quality scenarios at all three case studies (Tables 3-3 to 3-6). The complete set of media concentration estimates for each air quality scenario is presented in Appendix C for the general urban case study, in Appendix D (Attachments D-7 through D-11) for the primary Pb smelter case study, and in Appendix E (Attachments E-3 through E-7) for the secondary Pb smelter case study. Estimates presented in this section are presented to three (for air) or zero (for dust and soil) decimal places, which results in various numbers of implied significant figures. This is not intended to convey greater precision for some estimates than others; it is simply an expedient and initial result of the software used for the calculation.

For each air quality scenario for the two point source case studies, a range of percentile estimates derived from a population-weighted distribution of these media concentrations are presented for each exposure medium. For the general urban case study, however, only a single value is presented for each exposure medium. This reflects the fact that, while the point source case studies are modeled using spatial templates that include a large number of US Census blocks and/or block groups (allowing percentile media concentrations to be identified), the general urban case study is modeled using a single study area with uniform media

concentrations. Consequently, there is only one value presented for the general urban case study for each medium in each air quality scenario.

As discussed in Section 2.3.3, Pb concentration in outdoor soil/dust is not changed with the alternate air quality scenarios. Rather, outdoor soil/dust concentration is held constant at the current conditions or current NAAQS level. This reflects the judgement that in most cases, a reduced air concentration would not yield a changed soil concentration over the near term (e.g., years to decades). In the case of an area such as the remediation zone of the primary Pb smelter case study, however, where soil dynamics have been changed by the substitution of contaminated soil with clean soil, or in areas where local sources may pose a more significant source to outdoor soil/dust than historic sources – and where there may be a currently increasing trend in surface Pb concentration - this may underestimate soil concentrations under some alternate NAAQS.

As expected, the highest media concentrations for each case study are associated with the current NAAQS scenario. The relatively lower media concentrations for the alternate NAAQS scenarios vary in the following order of decreasing estimates: 1) the 0.5 $\mu g/m^3$ maximum monthly average, 2) the 0.2 $\mu g/m^3$ maximum quarterly, 3) 0.2 $\mu g/m^3$ maximum monthly and 4) the 0.05 $\mu g/m^3$ maximum monthly average. In the case of the general urban case study, both of the current conditions scenarios (mean and high-end) generate media concentrations below the current NAAQS, since both monitor-based values are below the current NAAQS.

Table 3-3. Estimated annual ambient air concentrations.

	Average Annual Air Pb Concentration ($\mu g/m^3$)					
			Alternative NAAQS			
Statistic	**Current Conditions**	**Current NAAQS**	**0.2 $\mu g/m^3$, max quarterly**	**0.5 $\mu g/m^3$ max monthly**	**0.2 $\mu g/m^3$ max monthly**	**0.05 $\mu g/m^3$ max monthly**
General urban case study						
NA - single study area	High-end: 0.11 Mean: 0.056	0.600	0.080	0.130	0.050	0.013
Primary Pb smelter case study						
Maximum	NA	0.740	0.161	0.326	0.130	0.033
95th percentile		0.153	0.033	0.067	0.027	0.007
Median		0.042	0.009	0.019	0.007	0.002
5th percentile		0.015	0.003	0.007	0.003	0.001
Minimum		0.006	0.001	0.003	0.001	< 0.001
Secondary Pb smelter case study						
Maximum	0.126	NA[a]	0.034	0.071	0.028	0.007
95th percentile	0.015		0.004	0.008	0.003	0.001
Median	0.003		0.001	0.002	0.001	< 0.001
5th percentile	0.001		< 0.001	< 0.001	< 0.001	< 0.001
Minimum	< 0.001		< 0.001	< 0.001	< 0.001	< 0.001

[a]The current conditions scenario for secondary Pb smelter case study met the current NAAQS.

Table 3-4. Estimated inhalation exposure concentrations.

Statistic	Average Annual Inhalation Exposure Concentration of Pb ($\mu g/m^3$)					
	Current Conditions	Current NAAQS	Alternative NAAQS			
			0.2 $\mu g/m^3$ max quarterly	0.5 $\mu g/m^3$ max monthly	0.2 $\mu g/m^3$ max monthly	0.05 $\mu g/m^3$ max monthly
General urban case study						
NA - single study area	High-end: 0.049 Mean: 0.024	0.026	0.034	0.054	0.021	0.005
Primary Pb smelter case study						
Maximum	NA	0.310	0.067	0.136	0.055	0.014
95th percentile		0.064	0.014	0.028	0.011	0.003
Median		0.017	0.004	0.007	0.003	0.001
5th percentile		0.006	0.001	0.003	0.001	< 0.001
Minimum		0.002	< 0.001	0.001	< 0.001	< 0.001
Secondary Pb smelter case study						
Maximum	0.056	NA[a]	0.015	0.031	0.013	0.003
95th percentile	0.007		0.002	0.004	0.002	< 0.001
Median	0.001		< 0.001	0.001	< 0.001	< 0.001
5th percentile	< 0.001		< 0.001	< 0.001	< 0.001	< 0.001
Minimum	< 0.001		< 0.001	< 0.001	< 0.001	< 0.001

[a]The current conditions scenario for secondary Pb smelter case study met the current NAAQS.

Table 3-5. Estimated outdoor soil/dust concentrations.

Statistic	Projected Average Outdoor Soil/Dust Pb Concentration (mg/kg) (Same for all air quality scenarios)[a]
General urban case study	
NA - single study area	198
Primary Pb smelter case study	
Maximum	958
95th percentile	245
Median	85
5th percentile	30
Minimum	17
Secondary Pb smelter case study	
Maximum	315
95th percentile	66
Median	12
5th percentile	1
Minimum	<1

[a] Estimates developed for current conditions (or current NAAQS) scenario were used for all alternate NAAQS scenarios.

Table 3-6. Estimated indoor dust concentrations.

Statistic	Projected Average Indoor Dust Pb Concentration (mg/kg or ppm)					
	Current Conditions	Current NAAQS	Alternative NAAQS			
			0.2 (μg/m^3) max quarterly	0.5 (μg/m^3) max monthly	0.2 (μg/m^3) max monthly	0.05 (μg/m^3) max monthly
General urban case study						
NA - single study area	High-end: 157-198 Mean: 107-146	426-566	128-169	166-206	102-140	71-88
Primary Pb smelter case study						
Maximum	NA	1,944	648	1,077	557	383
95th percentile		219	152	172	149	138
Median		84	68	73	67	63
5th percentile		53	45	47	44	43
Minimum		41	38	39	38	38
Secondary Pb smelter case study						
Maximum	166	NA[a]	89	120	84	66
95th percentile	73		63	67	63	61
Median	63		61	61	61	60
5th percentile	60		60	60	60	60
Minimum	60		60	60	60	60

[a] The current conditions scenario for secondary Pb smelter case study met the current NAAQS.

3.4 ESTIMATED BLOOD PB LEVELS

Estimates of concurrent and average lifetime blood Pb level derived from outputs of the IEUBK model (Section 3.2) have been developed for each air quality scenario in each case study (see Appendix I). Further, multiple sets of blood Pb estimates were generated for each air quality scenario of each case study, reflecting an effort to consider key sources of uncertainty (e.g., indoor dust model, blood metric, GSD) and their impact on blood Pb estimates (see Section 2.4.6.2). That is, eight separate blood Pb distributions were generated for each air quality scenario of the general urban case study (four for each of the two blood Pb metrics) and two distributions were generated for each air quality scenario of the point source case studies (one for each of the two blood Pb metrics) (see Table 2-3). The greater number of blood Pb distributions for the general urban case study reflects the larger number of modeling approaches implemented for this more conceptual case study, which differ by indoor dust model and GSD.

Because general trends in blood Pb levels across both population percentiles and air quality scenarios (for a given case study) are similar for both the concurrent and lifetime average blood metrics, we have only presented estimates for the concurrent metric here. Full results are presented in Appendix I. Concurrent blood Pb estimates reflecting all pathways (total), the "recent air" pathways (see Section 3.2.2), and the recent plus past air pathways are presented in Table 3-7. The total estimates presented in Table 3-7 are those for the median in the distribution for each case study, and the estimates for the other two categories are the values for those

categories associated with the median for the total blood Pb estimate. The corresponding estimates for the 95th percentile in the distribution of total blood Pb estimates for each case study are presented in Table 3-8. In these tables, all values are rounded to one decimal place, and we have presented all values below 0.05 as <0.1.

It is noted that given the various limitations of our modeling tools (Sections 2.4.3 and 3.2.2), blood Pb levels associated with air-related exposure pathways and current levels of Pb emitted to the air (including via resuspension) are likely to fall between the estimates for "recent air" and those for "recent" plus "past air". Additionally, with regard to the urban case study "recent air" and "recent" plus "past air" categories, an artifact of the hybrid dust Pb model tends to mask trends in the two components of dust Pb (recent air and other), which contribute to "recent air" and "past air" estimates, respectively (see Section 4.3.1 discussion of this uncertainty). For the primary Pb smelter case study, uncertainty in parsing out the "recent air" and "other" components of indoor dust (specifically for the site-specific regression model used in the remediation zone) have lead us to conclude that only "recent plus past air" exposures should be presented and "recent air" should not be separately presented, as is done for the other case studies (see Section 3.1.4.2).

With regard to total blood Pb, estimates for the general urban and primary Pb smelter case studies indicate higher values for the current NAAQS scenario compared to any of the other scenarios. The difference is 1-2 µg/dL for the median estimates and up to 6 µg/dL difference for the 95th percentile estimates. Although no difference in median total blood Pb estimates (rounded to whole numbers) is observed between current conditions or alternative NAAQS scenarios for the general urban or primary Pb smelter case studies, differences among alternate NAAQS scenarios are observed for the primary Pb smelter case study subarea (Appendix P). Additionally, a 1 µg/dL difference in total blood Pb is observed at the 95th percentile between current conditions and lower alternative NAAQS scenarios for the general urban case study.

Table 3-7. Summary of blood Pb estimates for medians in total-exposure blood Pb distributions.

Case Study and Air Quality Scenario	Concurrent Blood Pb Level[a]					
	Recent air[b]		Recent plus past air[b]		Total Pb exposure	
General urban case study	**Low**	**High**	**Low**	**High**	**Low**	**High**
Current NAAQS (1.5 µg/m^3, max quarterly)	1.9	2.2	2.6	3.2	3.1	3.7
Current conditions - 95th percentile (0.87 µg/m^3, max quarterly)	0.4	0.8	1.4	1.6	2.0	2.1
Alternative NAAQS (0.5 µg/m^3, max monthly)	0.5	0.9	1.5	1.6	2.0	2.2
Alternative NAAQS (0.2 µg/m^3, max quarterly)	0.3	0.7	1.3	1.5	1.9	2.0
Current conditions - mean (0.14 µg/m^3, max quarterly)	0.2	0.6	1.2	1.4	1.8	1.9
Alternative NAAQS (0.2 µg/m^3, max monthly)	0.2	0.5	1.2	1.3	1.7	1.9
Alternative NAAQS (0.05 µg/m^3, max monthly)	0.1	0.2	1.1	1.1	1.6	1.7
Primary Pb smelter - *full study area*						
Current NAAQS (1.5 µg/m^3, max quarterly)	NA[c]		0.8		1.5	
Alternative NAAQS (0.5 µg/m^3, max monthly)			1.1		1.4	
Alternative NAAQS (0.2 µg/m^3, max quarterly)			0.7		1.4	
Alternative NAAQS (0.2 µg/m^3, max monthly)			0.5		1.4	
Alternative NAAQS (0.05 µg/m^3, max monthly)			0.7		1.4	
Primary Pb smelter - *1.5 km subarea*						
Current NAAQS (1.5 µg/m^3, max quarterly)	NA[c]		4.0		4.6	
Alternative NAAQS (0.5 µg/m^3, max monthly)			2.6		3.2	
Alternative NAAQS (0.2 µg/m^3, max quarterly)			1.8		2.5	
Alternative NAAQS (0.2 µg/m^3, max monthly)			1.8		2.3	
Alternative NAAQS (0.05 µg/m^3, max monthly)			1.1		1.7	
Secondary Pb smelter - *full study area*						
Current conditions	<0.1		0.4		1.0	
Alternative NAAQS (0.5 µg/m^3, max monthly)	<0.1		0.1		1.0	
Alternative NAAQS (0.2 µg/m^3, max quarterly)	<0.1		0.4		1.0	
Alternative NAAQS (0.2 µg/m^3, max monthly)	<0.1		0.4		1.0	
Alternative NAAQS (0.05 µg/m^3, max monthly)	<0.1		0.4		1.0	
Secondary Pb smelter - 1.5 km subarea						
Current conditions	0.1		0.7		1.3	
Alternative NAAQS (0.5 µg/m^3, max monthly)	<0.1		0.7		1.3	
Alternative NAAQS (0.2 µg/m^3, max quarterly)	0.1		0.8		1.3	
Alternative NAAQS (0.2 µg/m^3, max monthly)	0.1		0.8		1.3	
Alternative NAAQS (0.05 µg/m^3, max monthly)	<0.1		0.6		1.2	

a - Estimates are rounded to one decimal place.
b -The term "past air" includes contributions from the outdoor soil/dust contribution to indoor dust, historical air contribution to indoor dust, and outdoor soil/dust pathways, while "recent air" refers to contributions from inhalation of ambient air Pb or ingestion of indoor dust Pb predicted to be associated with outdoor ambient air Pb levels, with outdoor ambient air also potentially including resuspended, previously deposited Pb (see Section 2.4.3).
c – "Recent air" estimates were not developed for the primary Pb smelter case study (see Section 3.1.4.2).

Table 3-8. Summary of blood Pb estimates for 95th percentiles in total-exposure blood Pb distributions.

Air Quality Scenario (and case study)	Concurrent Blood Pb Level[a]					
	Recent air[b]		Recent plus past air[b]		Total Pb exposure	
General urban case study	**Low**	**High**	**Low**	**High**	**Low**	**High**
Current NAAQS (1.5 µg/m^3, max quarterly)	4.6	7.5	6.3	9.9	7.6	11.5
Current conditions - 95th% (0.87 µg/m^3, max quarterly)	1.1	2.8	3.4	5.4	5.1	7.2
Alternative NAAQS (0.5 µg/m^3, max monthly)	1.2	2.6	3.5	5.1	4.8	6.7
Alternative NAAQS (0.2 µg/m^3, max quarterly)	0.8	2.3	3.1	5.1	4.4	6.9
Current conditions - mean (0.14 µg/m^3, max quarterly)	0.5	1.9	2.9	4.7	4.2	6.5
Alternative NAAQS (0.2 µg/m^3, max monthly)	0.5	1.8	2.9	4.6	4.2	6.4
Alternative NAAQS (0.05 µg/m^3, max monthly)	0.1	0.7	2.6	3.9	3.9	5.7
Primary Pb smelter - *full study area*						
Current NAAQS (1.5 µg/m^3, max quarterly)	NA[c]		2.8		4.6	
Alternative NAAQS (0.5 µg/m^3, max monthly)			3.1		4.2	
Alternative NAAQS (0.2 µg/m^3, max quarterly)			2.4		4.0	
Alternative NAAQS (0.2 µg/m^3, max monthly)			2.5		4.0	
Alternative NAAQS (0.05 µg/m^3, max monthly)			1.9		3.8	
Primary Pb smelter - *1.5 km subarea*						
Current NAAQS (1.5 µg/m^3, max quarterly)	NA[c]		10.3		12.3	
Alternative NAAQS (0.5 µg/m^3, max monthly)			7.6		8.5	
Alternative NAAQS (0.2 µg/m^3, max quarterly)			5.9		6.6	
Alternative NAAQS (0.2 µg/m^3, max monthly)			4.9		6.1	
Alternative NAAQS (0.05 µg/m^3, max monthly)			3.5		4.5	
Secondary Pb smelter - *full study area*						
Current conditions	<0.1		0.8		2.4	
Alternative NAAQS (0.5 µg/m^3, max monthly)	0.1		1.1		2.4	
Alternative NAAQS (0.2 µg/m^3, max quarterly)	<0.1		0.8		2.3	
Alternative NAAQS (0.2 µg/m^3, max monthly)	<0.1		1.1		2.4	
Alternative NAAQS (0.05 µg/m^3, max monthly)	<0.1		0.8		2.4	
Secondary Pb smelter - 1.5 km subarea						
Current conditions	0.5		2.2		3.3	
Alternative NAAQS (0.5 µg/m^3, max monthly)	0.4		2.5		3.3	
Alternative NAAQS (0.2 µg/m^3, max quarterly)	0.2		2.3		3.1	
Alternative NAAQS (0.2 µg/m^3, max monthly)	<0.1		1.4		3.1	
Alternative NAAQS (0.05 µg/m^3, max monthly)	<0.1		1.6		3.1	

a - Estimates are rounded to one decimal place.
b -The term "past air" includes contributions from the outdoor soil/dust contribution to indoor dust, historical air contribution to indoor dust, and outdoor soil/dust pathways, while "recent air" refers to contributions from inhalation of ambient air Pb or ingestion of indoor dust Pb predicted to be associated with outdoor ambient air Pb levels, with outdoor ambient air also potentially including resuspended, previously deposited Pb (see Section 2.4.3).
c – "Recent air" estimates were not developed for the primary Pb smelter case study (see Section 3.1.4.2).

3.5 UNCERTAINTY CHARACTERIZATION AND SENSITIVITY ANALYSIS

The characterization of uncertainty associated with exposure assessment included performance evaluation which is discussed in this section. In addition, uncertainty in exposure assessment is also considered as part of characterizing uncertainty in the risk estimates and is reflected in inclusions of multiple modeling approaches and the qualitative discussion of key sources of uncertainty which are discussed in Section 4.3. Elements of exposure assessment are also included in the sensitivity analysis described in Section 4.3.2.

Performance evaluation for the exposure assessment focused on evaluation of projections of Pb in exposure media (i.e., ambient air, outdoor soil, and indoor dust) (discussed in Section 3.5.1) and projections of Pb in blood (covered in Section 3.5.2). Those case studies (or case study elements) for which media concentrations were estimated using empirical data as the basis are not considered here; only those estimates based directly on modeling were included.

Comparing model output to monitoring data is often considered the most desirable form of performance evaluation. In addition to monitoring data, or in the absence of such data, outputs from other models and expert opinion about how outputs should look can be used as comparison benchmarks in performance evaluation. The performance evaluation activities reported here have focused on the comparison of model outputs to measurements. Favorable comparisons, including a lack of systematic trends in either over- or underestimation of modeled results relative to empirical data, provide confidence in the modeling estimates.

3.5.1 Performance Evaluation Related to Exposure Media Modeling

This section discusses performance evaluation conducted for modeling of Pb concentrations in exposure media including ambient air, outdoor soil and indoor dust.

3.5.1.1 Evaluation of Modeled Ambient Air Pb Concentrations

Performance evaluation for ambient air Pb predictions focused on the two point source case studies, in which air dispersion modeling was used.

For the primary Pb smelter case study current NAAQS scenario, the ISCST3-Prime model was used with input files (e.g., source characterization, meteorological data) used in developing the 2007 proposed revision to the SIP for that location (Appendix D). The submission to EPA for the proposed SIP revision included a model performance evaluation, focused on the "actual value" modeling scenario (MDNR 2007a). The actual value modeling included three separate comparisons based on relating model predictions to measured Pb concentrations at five monitor sites in the study area. These comparisons included:

- Day-to-day evaluation of modeling output compared to monitor values: The review of the model performance evaluation conducted by the state of Missouri concluded that all sites demonstrated a pattern of overall accuracy for directional prediction (i.e., high modeled days being high monitored days and low modeled days being low monitored days), suggesting that the model was performing well in relating wind direction to Pb transport (MDNR, 2007a).

- Comparison of source contribution analysis using chemical mass balance (CMB) with dispersion model predicted relative contributions: Source contribution analysis using CMB of monitor filter residue to identify significant sources of Pb for each monitor (e.g., roads inside facility boundaries and yard dust, blast furnace) were compared with relative contributions predicted by the dispersion model for individual modeled sources. The review of the model performance evaluation concluded that there was generally good agreement between the CMB results and the air dispersion results in terms of major sources contributing Pb at each monitor (MDNR, 2007a).

- Comparison of overall average modeled results with monitored levels: This performance evaluation involved comparing modeled results (for 247 days simulated for 2005) at six monitor locations with actual measured values for that same period at those locations. Results of this evaluation suggested a slight overprediction bias (<10%) for those sites likely to have the greatest impacts from the facility.

This evaluation of model performance for the actual value modeling scenario increases confidence in estimates developed for the current NAAQS scenario with this modeling setup and inputs particular to the 2007 proposed SIP revision.

Performance evaluation of air dispersion modeling for the secondary Pb smelter is discussed in Appendix E (Section E.2.4), and involved comparing modeled ambient air Pb levels to measured levels at two monitoring locations within the study area. Results of that evaluation suggest that the model might be slightly underpredicting levels at the closest monitor, and slightly more underpredictive of levels at the more distant monitors. The use of meteorological data that is not site-specific and may not be fully representative of actual wind patterns in the area may be contributing to this. When modeled concentrations at distances matching those of the monitors, and for all directional points around the facility, are compared with monitor values, modeled values are identified which match or exceed the measured values.

3.5.1.2 Evaluation of Modeled Outdoor Soil/Dust Pb Concentrations

Modeling was used in estimating the spatial pattern of outdoor soil/dust Pb concentrations for the secondary Pb smelter case study (see Section 3.1.3.3). As the modeling did not use estimates of historical Pb emissions (presumed to be higher than current emissions), absolute values of the model predictions were not expected to be representative of current Pb concentrations in soil/dust in this case study. Although measurements were not available for the

case study location, comparison of model predicted soil Pb levels with Pb concentrations reported for a surrogate secondary Pb smelter location confirmed this expectation (see Appendix E, Section E.3). Model predictions were - on average - a factor of 3 lower than Pb concentrations at the surrogate location. Accordingly, we used a hybrid approach for characterizing outdoor soil Pb levels for this case study, scaling the model estimates of soil Pb concentration across the study area by factors derived from studies at other secondary Pb smelter locations (see Appendix E, Section E.3).

3.5.1.3 Evaluation of Modeled Indoor Dust Pb Concentrations

Performance evaluation completed in support of indoor dust Pb prediction involved two components. First, the hybrid model developed for the general urban case study was evaluated using available data from the literature (see Appendix G, Section G.3.6). Results of this evaluation help us assess the overall reasonableness of this model in supporting indoor dust prediction for the general urban case study in particular. Additionally, predicted indoor dust Pb levels for all three case studies were compared with data identified in the literature, including measurements from individual studies focusing on smaller areas and data from national-scale surveys. The general urban case study model and model projections are discussed here first

The hybrid indoor dust model developed for the general urban case study (Section 3.1.4.1 and Appendix G) is a combination of mechanistic model (to relate outdoor ambient air Pb to indoor dust Pb) and empirical data (to characterize the nonair related fraction of indoor dust Pb in the residential urban setting). Components of the mechanistic portion of the model were subjected to a range of evaluations based on available data in the literature. In addition, the conversion of model-generated indoor dust Pb loadings to concentrations (a key step required prior to blood Pb modeling) was evaluated. Finally, model predictions of indoor dust Pb levels were evaluated using data from several studies for specific cities.

Evaluation of the mechanistic component of the hybrid dust model focused on (a) predicted deposition fluxes of Pb to indoor surfaces and (b) prediction of relationship between indoor air Pb and ambient outdoor air Pb (Appendix G, Section G.3.6). Generally, modeled indoor deposition fluxes for Pb appear to be in line with values reported for a location in Manhattan with closed windows (Caravanos et al., 2006). The model predicted ratio of indoor air Pb to outdoor ambient air Pb are lower (~50%) than ratios based on data collected from residences in the Midwest (Roy et al., 2003) but similar to ratios generated by a different hybrid model which combines empirical data with a mass balance modeling approach in predicting indoor ambient air concentrations (Riley et al., 2002). The underestimate of this ratio, when compared with the empirical data from Roy et al. (2003), may result from the fact that the hybrid model does not consider resuspension of indoor dust Pb. Generally, these findings with regard to

substeps of the mechanistic (and air-focused) component of the hybrid model (Appendix G, Section G.3.6) suggest a potential to underpredict the influence of outdoor ambient air Pb on indoor dust Pb. The conversion for predicted indoor dust loadings to concentrations, however, may overpredict concentrations based on a comparison data collected in residences in the Midwest (Roy et al., 2003; Appendix G, Section G.3.6). This finding may counterbalance any potential for the mechanistic component of the model to underestimate the influence of ambient air Pb on indoor dust Pb.

Indoor dust Pb concentrations (in terms of mass per mass) were identified from a few studies in the literature (e.g., CD, Table 3-8; Tang et al., 2004), as well as the HUD National Survey of Lead-based Paint in Housing (USEPA, 1995) described in Section 3.1.4.1. These studies are identified in Table 3-9. Although the HUD survey was used for the empirical component of the hybrid model, it is considered here with regard to the total dust Pb concentrations predicted by both the hybrid and air-only models used in the general urban case study (use of these models is described in Section 3.1.4.1).[6] Comparison of the model-predicted indoor dust concentrations for the current conditions scenarios (146 and 198 ppm for the hybrid model and 107-157 ppm for the air-only model) to empirical data collected in Jersey City, NJ, and Ottawa Canada (Table 3-9) suggest that the model may be underpredicting indoor dust Pb, although the housing stock in these studies was much older than housing generally in U.S., which may mean that the measurements are impacted to a greater extent by indoor Pb paint than would be the general case in the U.S. Thus, it would be expected that those reported values would be higher than the model predictions. Additionally, the model-predicted values fall between the medians for the youngest and oldest houses sampled in the HUD national survey (Table 3-9). Given that indoor dust Pb modeling completed for the general urban case study was aimed at capturing central tendency indoor dust Pb levels (and is not expected to, for example, capture variability related to cleaning rates or indoor paint Pb levels), this finding provides confidence in the estimates.

[6] The use of HUD indoor dust Pb data for performance evaluation is considered reasonable, even in light of its use in deriving the air-related portion of the hybrid model, since total indoor dust Pb levels generated by the hybrid model (reflecting both the air-related and non air-related components of the model) are being examined in the performance evaluation. In this context, nationally representative indoor dust Pb concentrations (obtained from the HUD dataset) are considered a useful empirical dataset for the evaluation.

Table 3-9. Evaluation of model-predicted indoor dust Pb levels against empirical data obtained from the literature.

Case study	Modeled indoor dust Pb levels (ppm)			Indoor Dust Pb Observations reported in the literature
	Air quality scenario	Median	5th to 95th Percentile (min-max)	
General urban case study	Current conditions (air-only regression and hybrid)	Mean current conditions: (107 to 146) 95th percentile current conditions: (157-198)[a]		- Residences near smelters: 1283-4140 ppm (CD, Table 3-8) - Jersey City, NJ housing (floor): 857 ppm (CD, Table 3-8) - Residences in the Midwest (windowsill): 954ppm (CD, Table 3-8) - Ottawa Canada housing (floor): 222 ppm (median), 406 ppm (mean) (Tang et al., 2004) - HUD survey of US housing: 87 ppm (median for newest houses, built 1960-1979), 406 ppm (median for oldest housing, build <1940) (USEPA, 1995).
Primary Pb smelter case study	Current NAAQS	84	53 - 219 (41 - 1,944)	
Secondary Pb smelter case study	Current conditions	63	60 – 73 (60 – 166)	

a - because a single study area is used for the general urban case study, a single dust estimate was generated for each combination of current conditions air quality scenario and indoor dust model (i.e., four separate estimates were generated for the general urban case study).

For the two point source case studies, the model-predicted indoor dust Pb concentrations were compared to observations from the literature (see Table 3-9). An important factor to keep in mind when reviewing the modeled results presented in Table 3-10 is that the current NAAQS scenario modeled for the primary Pb smelter involved regions of the study area with ambient air Pb levels significantly higher than either of the other case studies.

In consideration of indoor dust Pb levels predicted for the primary Pb smelter, we have focused more on the central tendency values. This reflects the fact that the higher-end model-generated values for this case study likely reflect significant ambient air impacts which are not captured in any of the empirical data identified in the literature, thereby reducing the utility of performance evaluations for these higher-end predictions. The median indoor dust Pb concentration generated for the primary Pb smelter case study (84 ppm) falls near the lower end of the range in the HUD dataset (i.e., near the median of 87 ppm reported for newer housing in that study). This seems reasonable since a significant fraction of the study area for this case study has ambient air Pb levels not significantly different from ambient air Pb levels seen across the U.S. (see Table 3-4).

Regarding the secondary Pb smelter case study, the median value for this case study (63 ppm) is below the range of values reported in the past for smelters (CD, Table 3-8), and also just

below median value reported for the youngest housing in the HUD national data set (USEPA, 1995), suggesting that indoor dust Pb levels may be underpredicted for this case study.

3.5.2 Performance Evaluation Related to Blood Pb Modeling

Performance evaluation completed in support of blood Pb modeling involved three steps: (a) evaluation of candidate blood Pb models for the analysis, (b) comparison of ambient *outdoor air Pb-to-blood Pb* ratios generated for the three case studies against ratios obtained from the literature and (c) comparison of modeled blood Pb levels for these three case studies against NHANES IV data. Each of these evaluation steps is discussed below.

3.5.2.1 Evaluation of Candidate Blood Pb Models

Evaluation of candidate blood Pb models (IEUBK and Leggett) involved three separate stages: (a) application of the candidate models to three hypothetical individual child exposure scenarios used previously by EPA and others in evaluations of blood Pb models, (b) comparison of candidate model predictions for a general U.S. childhood exposure scenario (using typical Pb exposures for key pathways) to NHANES IV empirical data, and (c) evaluation of candidate model performance in replicating measurements of urban child blood Pb levels obtained in Rochester. Detailed results of the evaluation of candidate blood Pb models are presented in Appendix J (and summarized in Section J.4).

The first stage (focusing on reproducing results of previous performance evaluations) demonstrated that we were applying the candidate models correctly. Tests of the models against specific individual exposure scenarios (Section J.1.3) reproduced, to a high degree, the results of previous model comparison.

The second stage of the model evaluation (focusing on reproducing general US child blood Pb levels presented in NHANES IV) demonstrated that, depending on assumptions regarding typical outdoor soil/dust and indoor dust Pb concentrations, the IEUBK model either moderately overpredicted GM blood Pb levels (by two-fold or less) or generated predictions close to NHANES summary statistics (see Section J.3.1). By contrast, predictions from the Leggett model were more than three to six times higher than the age-specific NHANES IV GM values.

The third stage of the model evaluation focused on evaluating the candidate models in predicting blood Pb levels for an urban child cohort (Appendix J, Section J.3.2). The dataset, which included matched Pb media concentrations (outdoor soil/dust and indoor dust levels) and blood Pb levels, was collected as part of an epidemiological study focusing on the effects of Pb exposure in children living in Rochester, NY (Lanphear et al., 1995; Lanphear and Roghmann, 1997). Blood Pb levels for each child sampled in the study were predicted using IEUBK and Leggett, with the measured media Pb levels collected as part of the study as inputs. These

predicted blood Pb levels were then compared to the measured blood Pb levels for each child. The results of this stage of the model evaluation were similar to those from the NHANES evaluation in that IEUBK results suggested a moderate overprediction (~70%) while Leggett results indicated a much greater overprediction (a factor of 2 to 6).

Results of the model evaluation for IEUBK and Leggett suggest that IEUBK generates more representative blood Pb estimates relative to Leggett in the context of the evaluations conducted here. Based on the results of the model evaluation, we decided to use IEUBK in generating the primary set of exposure and risk results for this analysis, and include Leggett as part of the sensitivity analysis but not in the primary analysis (see Section 4.3.2).

3.5.2.2 Evaluation of model-derived outdoor air Pb-to-blood Pb ratios

In deriving the current NAAQS in 1978 (43 FR 46246), USEPA used an estimate of the relationship between ambient air Pb concentration and associated blood Pb concentration (i.e., 1:2, $\mu g/m^3$ to $\mu g/dL$). In this assessment, we rely on several distinct modeling steps which, when taken together, translate ambient air Pb into blood Pb. As part of the blood Pb model performance evaluation we have extracted air-to-blood Pb ratios from the modeling completed for the three case studies for comparison to estimates reported in the literature.

Ratios were developed that relate ambient air Pb to blood Pb contributed from the following different exposure pathways or pathway combinations: (a) inhalation of ambient air, (b) inhalation of ambient air plus ingestion of the Pb in indoor dust that is predicted to be associated with ambient air Pb levels (i.e., "recent air" per Section 2.4.3), and (c) inhalation of ambient air plus ingestion of indoor dust plus ingestion of outdoor soil/dust (i.e., not including the diet and drinking water ingestion pathways). The limitations of our modeling tools precluded us from parsing air-related blood contributions any more finely. The ratios (actually, the blood Pb side of the ratio) will be larger as you move from (a) to (c) because there is a progressively larger fraction of overall blood Pb (exposure) being associated with air. With regard to the potential impact of ambient air Pb on blood Pb, the first ratio (inhalation pathway) is an underestimate because it excludes the important ingestion pathways to which ambient air Pb can contribute. Conversely, the third ratio, although not including any impact of air Pb on diet (and blood), potentially includes some contributions to blood Pb that are not influenced by air (e.g., indoor paint). For the purposes of this model performance discussion, we have focused on the second type of ratio (those for "recent air") derived using the concurrent blood Pb metric. The full set of ratios are presented for each case study in Appendix I

In this evaluation, we have considered the ratios derived from the blood Pb estimate prior to application of the GSD reflecting interindividual variability in Blood Pb levels (i.e., the central tendency estimate of blood Pb derived for each US Census block or blockgroup for the point

source case studies and for the entire study area for the general urban case study). Although air-to-blood Pb ratios can be derived for individual simulated children after application of the GSD, these would be less relevant to empirical ratios reported in the literature, which tend to capture central tendency or typical ratios for a study population (through statistical regression modeling, for example). While this interindividual variability in exposure parameters is not reflected in the ratios evaluated here, variation in exposure concentration is reflected by the range of ratios derived for the different exposure zones of the two point source case studies. For the general urban case study, ratios are presented for both current conditions scenarios (reflecting a mean and high-end estimate of air Pb concentration).

Table 3-10. Air-to-blood Pb ratios for "recent air" contribution to concurrent blood Pb level.

Case study	Annual average ambient air Pb concentrations ($\mu g/m^3$)	Air-to-Blood Pb Ratio	Air-to-Blood Pb Ratios Identified from the Literature
General urban – current conditions			- Review of studies published before 1984 reports air-to-blood Pb ratios for children generally ranging from 1:3 to 1:5 (Brunekreef, 1984). - Pooled analysis of air-to-blood Pb relationship (log-log regression) based on above studies yielded ratios of 1:3 to 1:6 (Brunekreef, 1984). - An air-to-blood ratio developed based on more recent study data (Hilts, 2003) is 1:7 (for children in the vicinity of an operating smelter which experienced a modification in facility operations, leading to a marked decrease in air Pb emissions).
Current conditions (mean)	0.056	1:4 & 1:10[a]	
Current conditions (95th percentile)	0.114	1:4 & 1:7[a]	
Primary Pb smelter – current NAAQS			
Median air concentration	0.093	1:3	
95th percentile air concentration	0.458	1:7	
Secondary Pb smelter- current conditions			
Median air concentration	0.005	1:5	
95th percentile air concentration	0.011	1:4	

[a] The two ratios for the general urban case study correspond to results obtained from the two indoor dust models.

Several observations can be made regarding higher ratios generated for two of the case studies presented in Table 3-10:

- For the general urban case study, the higher ratios (i.e., 1:10 and 1:7) result from application of the hybrid dust model which produces a higher indoor dust

concentration per unit ambient outdoor air Pb relative to the air-only regression model.

- For the primary Pb smelter case study, the higher ratio (1:7) is also the result of a more potent dust model (the site-specific regression model developed for the remediation zone of this study area). That indoor dust model was used for the higher-impact (95^{th} percentile) block, while the regression (air plus soil) model was used for the majority of the study area, including the median block (producing the 1:3 ratio).

The air-to-blood Pb ratios from the literature presented in Table 3-10 all come from a combination of older studies summarized in a single review from 1984 (Brunekreef, 1984) and more recent data (Hilts, 2003). The Brunekreef, 1984 review presents both (a) the ratios identified from the reviewed studies, which included surveys focused on smelter and urban study areas (generally range from 1:3 to 1:5) and (b) the results of a pooled analysis where a log-log regression model was developed relating ambient air Pb directly to blood Pb levels based on the underlying study data (this yields the range of 1:3 to 1:6 presented in Table 3-10).

Data presented in Hilts, 2003, track Pb exposure levels (e.g., outdoor ambient air Pb, outdoor soil Pb and indoor dust Pb) and measured blood Pb levels (annual geometric mean blood Pb levels for children 6 to 60 months of age), for a population of children living in the vicinity of smelting operations in Trail, British Columbia for the period 1996 to 2001. The facility experienced a change in operations in 1997 which resulted in a significant decrease in Pb emissions from the facility and a resulting decrease in ambient air Pb levels in the vicinity of the facility. In deriving an air-to-blood Pb ratio from these data, we compared ambient air Pb levels for 1996 and 1999 to geometric mean blood Pb levels for the same years (all data were obtained from Table 3 in Hilts, 2003). An air-to-blood Pb ratio of 1:7 was generated based on this comparison. Note, also, that a second air-to-blood Pb ratio could have been generated by comparing data for 1996 and 2001 (which would have tracked the continued decrease in both ambient air Pb levels and geometric blood Pb levels). However, blood Pb measurements in 2001 focused on children 6-36 months of age, while blood Pb measurements for 1996 and 1999, involved children 6-60 months of age. This disconnect in the age groups would have prevented a direct comparison of the ratios generated using the two pairings of years. Therefore, we focused on the ratio developed by comparing 1996 and 1999 since the blood Pb measurements for these two years focused on an age group (6-60 month old children) closer to that used in our modeling of risks for the case studies (children <7 years of age).

The air-to-blood Pb ratio of 1:7 generated using the Hilts, 2003 data is slightly higher than the range presented in Brunekreef, 1984 (1:3 to 1:6). This might reflect the fact that overall exposures associated with the Hilts, 2003 study are lower than those reflected in the Brunekreef,

1984 study and, as noted in Brunekreef, 1984, there appears to be greater efficiency in translating air Pb to blood Pb at lower exposure levels, compared with higher levels.

Although the blood Pb levels associated with Hilts, 2003 (geometric mean blood Pb levels of 4 to >10 µg/dL) are closer to those associated with our modeled case studies than are the levels in Brunekreef (1984), the levels in Hilts are still higher. In the case of the blood Pb levels reflected in the studies summarized in Brunekreef (1984), these measured blood Pb levels (typically > 10ug/dL) are significantly higher than those modeled for our case studies. This disconnect between the studies in the literature and the modeled case studies (regarding blood Pb ranges) introduces uncertainty into performance evaluation of air-to-blood Pb ratios completed here. That is, it is possible that ratios seen at blood Pb levels significantly above 5 to 10 µg/dL (levels seen in the literature) are different from those below 5 µg/dL (those associated with the modeled case studies), although this can not be verified without updated characterizations of air-to-blood Pb ratios based on contemporary trends in Pb exposure and blood Pb levels. Despite this uncertainty, however, comparison of the air-to-blood Pb ratios developed for the three case studies against those found in the literature is considered useful.

The lower air-to-blood ratios generated for the three case studies (1:4 for the general urban case study, 1:3 for the primary Pb smelter case study and 1:4 to 1:5 for the secondary Pb smelter case study) fall within the range of ratios identified in the literature (1:3 to 1:7). However, the higher ratios, ranging from 1:7 to 1:10, are higher than the bulk of Brunekreff (1984) reported ratios and just include the 1:7 ratio developed from the Hilts (2003) dataset, although a subset of the individual studies reviewed by Brunekreef (1984) presented air-to-blood Pb ratios that were 8.5 and higher. In several instances, such higher ratios are associated with lower blood Pb levels and lower ambient air Pb levels (both factors that would seem to be more relevant to exposure conditions found in the three case studies modeled for this analysis). However, the studies reporting these higher ratios are complicated by a number of factors (e.g., involving older children, use of ambient air measurement techniques which may underestimate results, thereby inflating ratios, relatively small sample sizes, etc.).

3.5.2.3 Comparison of modeled blood Pb levels to nationally representative data

Evaluation of modeled blood Pb levels using empirical data depends on the availability of empirical datasets for populations with exposure similar to those in the case studies. While there are many blood Pb studies reported in the literature, they are largely composed of populations experiencing exposures from all pathways that are higher than exposures reflected in our case studies. No datasets were identified for specific subpopulations with exposures corresponding to current exposures that would be appropriate for performance evaluation here.

In the case of the general urban case study, while it is not location-specific, we have not identified any contemporary datasets for urban locations with ambient air Pb levels matching those modeled in the current scenario that are not heavily influenced by Pb-based paint. For the primary Pb smelter case study location, the most generally available blood Pb monitoring study was completed in 2001 and 2002, and given the changes in exposures since that time and the use of a future (current NAAQS) scenario these older blood Pb surveys do not correspond to the exposures modeled for this case study. In the case of the secondary Pb smelter, the blood Pb data available for the county containing the secondary Pb smelter does not indicate residence location handicapping efforts to consider blood Pb levels for children in the secondary Pb smelter case study location.

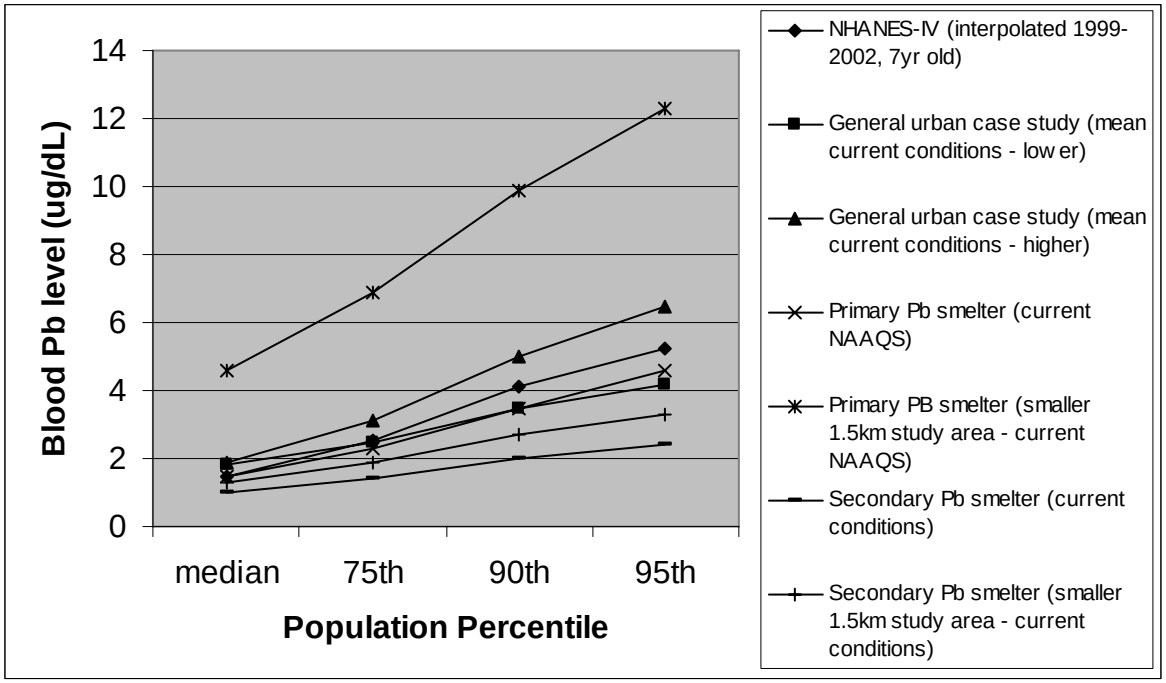
While blood Pb datasets were not identified for populations representative of those modeled for the three case studies, we have compared the modeled blood Pb levels against general national-scale blood Pb levels (for a child age group matching that modeled for the case studies). While each of the case studies involves a population that is different than the national population (reflecting the nature of the exposure scenario being considered), the background exposures for the case studies and the national population are similar. Accordingly, the relationship between the modeled blood Pb levels for each study area and the national distribution are evaluated. For this evaluation, we have used measured values interpolated from the NHANES IV dataset. Specifically, we have interpolated a series of percentile estimates for 7 yr olds based on summary data presented for NHANES IV (CDC, 2005). The process of interpolating these values involved the following steps: (a) use summary statistics (GM and associated confidence intervals) for 1-5 yr olds and 6-19 yr olds (for years 1999-2002) to establish lognormal distributions for each age range, (b) identify population percentiles of interest (e.g., median, 90^{th}, 95^{th} percentiles) for each age group using these lognormal distributions, and (c) interpolate a series of percentiles for a 7yr age cohort using the percentiles for the two age ranges (the 1-5 yr old and 6-19 yr old). This interpolation procedure resulted in the population percentile estimates for a 7 yr old cohort (for the years 1999-2002) presented in Table 3-11.

Table 3-11. Blood Pb levels for 7 year olds in the U.S. (interpolated from NHANES IV, 1999-2002).

Population percentile	Blood Pb level (µg/dL)
median	1.5
75th	2.55
90th	4.1
95th	5.25

Figure 3-2 compares modeled blood Pb levels for the three case studies with the interpolated NHANES IV blood Pb levels presented in Table 3-11. Modeled blood Pb levels presented in Figure 3-2 include (a) the (mean) current conditions scenario for the general urban case study, including both low-end and high-end estimates reflecting application of different indoor dust models, and GSDs for this case study, and (b) the current NAAQS scenarios for both the primary and secondary Pb smelter case studies.

Figure 3-2. Comparison of NHANES IV blood Pb levels with modeled estimates.



The following observations are made regarding the blood Pb modeling results for the three case studies (Figure 3-2):

- *General urban case study:* The low- and high-end estimates for the mean current conditions scenario bracket the NHANES IV data. Of the three case studies, it is reasonable to assume that results for the current conditions (mean) scenario for this

case study might be the closest to matching the NHANES. Interestingly, of the results for the low- and high-end modeling approaches for this scenario, the high-end results appear to be closer to the NHANES data, which would indicate that modeling for this case study may not be biased high with regard to total blood Pb levels. Additionally, unlike the point source case studies, the general urban case study included consideration of variability in background exposure pathways through application of the larger GSD in the high-end modeling approach for that case study.

- *Primary Pb smelter case study:* It would be expected that higher percentile exposure levels for this case study might exceed the NHANES data, however, that is not seen either at the 90th or 95th percentiles in Figure 3-2. There are three factors which may account for this observation. First, we have not generated higher population percentile estimates (i.e., greater than 95th percentile) and it is possible that exposures for those higher percentiles at the primary Pb smelter might begin to exceed levels seen with the NHANES-interpolated data. Second, it is important to reiterate that modeling for this case study did not account fully for exposure variability in background Pb exposures. It is likely that if modeling for this case study had included consideration of background exposure variability (e.g., relatively high paint Pb and drinking water exposures), there would be a greater difference between high-end blood Pb levels modeled for this case study and those interpolated form NHANES IV, with the potential for the primary Pb smelter percentiles to exceed those derived from the NHANES IV data. Note, however, this lack of full accounting for blood Pb variability from background sources is not considered a major limitation in this analysis because the focus is on ambient air-related exposures. Finally, as discussed in Section 4.3.1, modeling for the point source case studies did not consider other sources of ambient air Pb besides the smelter. This could produce low bias in predictions of exposure, especially for portions of the study area further from the facility.

- *Secondary Pb smelter case study:* Modeled blood Pb levels for this case study are significantly lower than NHANES IV levels, especially for higher population percentiles. As with the primary Pb smelter case study, this likely reflects three factors: (a) absence of predictions (due to technical limitations) of higher population percentiles above the 95th percentile, where exposure related to facility emissions is likely more dominant, (b) exclusion of consideration for higher background exposures to Pb in paint and drinking water, and (c) failure to consider ambient air Pb sources impacting the study area, other than the smelter facility. Note, also that, for the secondary Pb smelter case study, overall uncertainty associated with modeling of exposure and risk is considered sufficiently high to significantly reduce overall confidence in results for this case study (see Section 4.3.1).

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4 RISK ASSESSMENT

This chapter describes the approach used to characterize risk for the initial analyses of the full-scale assessment, including discussion of the methodology (Section 4.1), presentation of risk estimates (Section 4.2), and uncertainty characterization (Section 4.3). Additional analyses completed after the August 2007 CASAC meeting (Henderson, 2007a), involving a core modeling approach, are presented in Chapter 5.

4.1 METHODS FOR DERIVING RISK ESTIMATES

Risk characterization for this assessment focuses on IQ loss in children. IQ loss is derived using a set of concentration-response functions developed based on results from a pooled analysis of epidemiology studies (Lanphear et al., 2005). These concentration-response functions are combined with the population-level blood Pb distributions generated for the case studies to produce distributions of IQ loss estimates for each study population. IQ loss is also apportioned among different exposure pathways using the pathway apportionment information generated as part of the exposure analysis.

Two key elements of the risk methodology are described in greater detail below: (a) the concentration-response functions used in the analysis (Section 4.1.1) and (b) the stepwise analytical procedure used to generate the IQ loss (risk) distributions (Section 4.1.2).

4.1.1 Concentration-Response Functions

As discussed in Section 2.1.5, log-linear concentration-response functions for IQ loss for the concurrent and lifetime average blood Pb metrics were obtained from a large pooled study (Lanphear et al., 2005) and used as the basis for estimating IQ changes in children in this analysis. Three types of concentration-response functions were drawn from this study, and two equations of each type are used in this assessment: one for the concurrent blood Pb metric, and a second for the lifetime average blood Pb metric. The three types include: (a) a log-linear function with cutpoint (Section 4.1.1.1), (b) a log-linear function with low-exposure linearization (Section 4.1.1.2) and (c) a two-piece linear function (Section 4.1.1.3). Plots of the three types of IQ loss functions developed for this assessment are presented in Figure 4-1 for the concurrent blood Pb metric. Table 4-1 presents a comparison of total IQ loss and incremental IQ loss (IQ loss/µg/dL) for the three functions across a range of concurrent blood Pb levels. As can be seen by comparing the plots of the three functions together with the results presented in Table 4-1, the log-linear function with low-exposure linearization will generate the greatest IQ change across the exposure range 0 to 10 µg/dL followed by the log-linear function with cutpoint and then the two-piece linear function.

Inclusion of these three types of concentration-response functions in this assessment produces a range of risk estimates that is indicative of the model uncertainty related to predicting IQ loss based on modeled blood Pb levels.

Figure 4-1. Comparison of three concentration-response functions for concurrent blood Pb levels < 10 µg/dL.

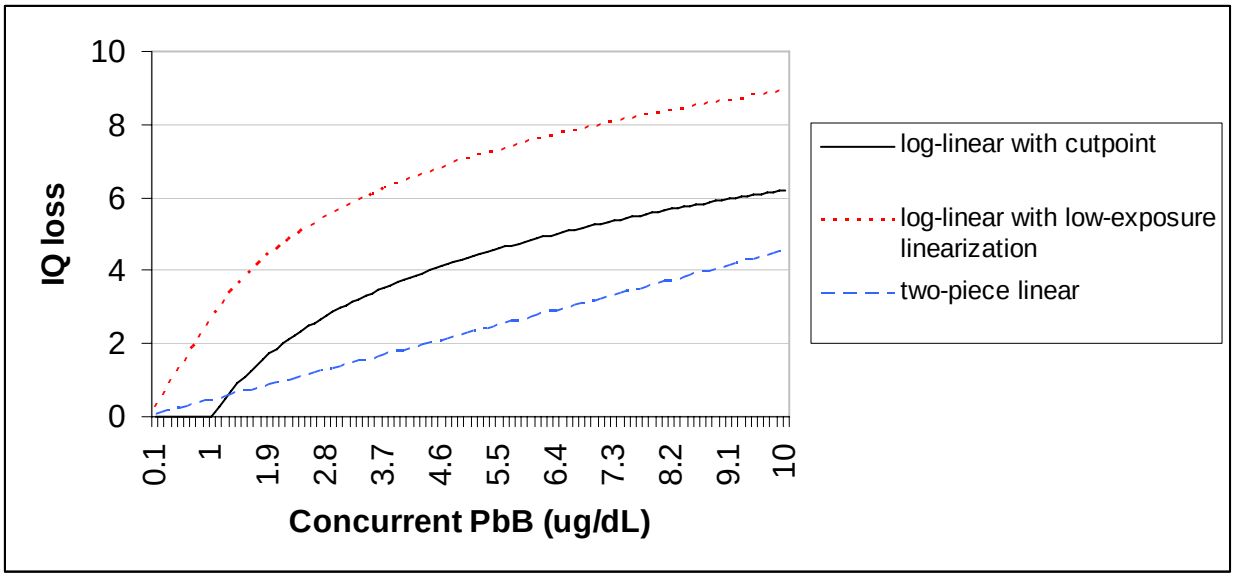

Table 4-1. Comparison of total and incremental IQ loss estimates below 10 µg/dL for the three concentration-response functions.

Performance Metric		Concentration-Response Function		
		Log-linear with cutpoint	Log-linear with low-exposure linearization	Two-piece linear
		Points, IQ loss		
Total IQ loss	at 2 µg/dL	1.9	4.6	0.9
	at 5 µg/dL	4.3	7.0	2.3
	at 7.5 µg/dL	5.4	8.1	3.4
	at 10 µg/dL	6.2	8.9	4.5
Incremental IQ loss (points per µg/dL)	<2 µg/dL	0.94	2.29	0.45
	<5 µg/dL	0.87	1.41	0.45
	<7.5 µg/dL	0.73	1.09	0.45
	<10 µg/dL	0.62	0.89	0.45

4.1.1.1 Log-Linear Function with Cutpoint

This concentration-response function is the log-linear function for IQ change from Lanphear et al. (2005), with incorporation of a cutpoint at the blood Pb level corresponding to the lowest levels represented by measurements in the underlying pooled analysis. The values used for cutpoints, for both the concurrent and lifetime average blood Pb metrics, are based on the corresponding low end of the range of values for these two indices in the pooled analysis (Hornung, 2007a, b). The values are 1.0 μg/dL and 1.47 μg/dL, for the concurrent and lifetime average blood Pb metrics, respectively. Slopes for the two log-linear functions were obtained directly from the study (Lanphear et al., 2005). Parameterization for these two log-linear functions with cutpoints is presented below, along with the mathematical form of the function:

- Form of function: IQ loss = beta * ln (concurrent blood Pb/cutpoint)
- Log-linear function with cutpoint (concurrent metric):
  - beta (slope): -2.70
  - cutpoint: 1.0 μg/dL
- Log-linear function with cutpoint (lifetime average metric):
  - beta (slope): -3.04
  - cutpoint: 1.47 μg/dL

4.1.1.2 Log-Linear Function with Low-Exposure Linearization

We also developed risk estimates that reflect the possibility that IQ loss is associated with the entire range of Pb exposure all the way down to "zero" exposure, as recommended by CASAC (Henderson, 2007b). The risk assessment included IQ loss prediction based on a log-linear IQ change function with a linearization of the log-linear function taking over at lower exposure levels. The transition point from the log-linear function to the linearized slope was selected with the same basis as the cutpoint described in Section 4.1.1.1, the lower end of the range of the pertinent blood Pb index values in the pooled analysis (Lanphear et al., 2005). The linearized slope is obtained by taking the tangent to the log-linear function at the point of departure, with different slopes being identified for the concurrent and lifetime average blood Pb metrics (per Lanphear et al, 2005). Parameterization for the two log-linear functions with low-exposure linearization is presented below, with the mathematical form of the function:

- Form of function:
  - For blood Pb level $\geq$ cutpoint:

    IQ loss = beta * ln (concurrent blood Pb/cutpoint) + linear slope * cutpoint
  - For blood Pb level < cutpoint:

    IQ loss = linear slope * concurrent blood Pb

- Log-linear function with low-exposure linearization (concurrent metric):
  - beta (slope): -2.70
  - linear slope to be applied below 1.0 μg/dL: -2.70
- Log-linear function with low-exposure linearization (lifetime average metric):
  - beta (slope): -3.04
  - linear slope to be applied below 1.47 μg/dL: -2.1

4.1.1.3 Two-piece Linear Function

This category of concentration-response function was developed by fitting a two-piece linear model to the log-linear IQ loss function obtained from the pooled analysis (Lanphear et al., 2005). Inclusion of this function in the risk assessment is intended to address the practical problem of the shape associated with application of the log-linear model at the low end of the blood Pb value range. In this case, we consider an alternate functional form that allows ready prediction of IQ loss across the full range of modeled Pb exposure. The procedure involved first generating blood Pb values for each of the two blood Pb metrics, concurrent and lifetime average, for a set of N = 1,333 simulated children representative of those included in the pooled analysis (Lanphear et al., 2005). This was accomplished by sampling from a blood Pb distribution constructed from the median and 95th percentile of the concurrent and lifetime average blood Pb indices, respectively, reported in Lanphear et al. (2005). IQ values for each of the 1,333 simulated children were then estimated using the reported log-linear models that relate blood Pb to absolute IQ (Lanphear, et al., 2005). Nonlinear regression techniques were then used to fit two piece linear models to these two sets of simulated children with their matched pairs of blood Pb and IQ values. The regressions provided parameter estimates (slopes and "hinge" point) of the best fitting two piece linear segment function for each blood Pb metric, concurrent and lifetime average. As discussed in Section 4.3.1, the use of a constructed dataset introduces uncertainty related to fitting of a model to a model. Parameterization for the two two-piece linear functions is presented below, with the mathematical form of the function:

- Form of function:
  - For blood Pb level > hinge:

    IQ loss = beta 2 * concurrent blood Pb
  - For blood Pb level ≤ hinge:

    IQ loss = beta 1 * concurrent blood Pb
- Two-piece linear function (concurrent metric):
  - "hinge" linking two segments: 10.82 μg/dL
  - beta 1 (slope at ≤ 10.82 μg/dL): -0.4539
  - beta 2 (slope at >10.82 μg/dL: -0.1130

- Two-piece linear function (lifetime average metric):
  - "hinge" linking two segments: 13.39 μg/dL
  - beta 1 (slope at ≤ 13.39 μg/dL): -0.3790
  - beta 2 (slope at >13.39 μg/dL: -0.1187

4.1.2 Projection of Population Risk

Risk characterization completed for this assessment involved converting the population-level blood Pb distributions generated for the three case studies into population-level distributions of IQ loss using the three types of concentration-response functions described in the last section. This procedure is described below for each of the functions.

- *Log-linear function with cutpoint:* Each modeled blood Pb level is compared against the cutpoint. If the blood Pb level is lower than the cutpoint, then no IQ loss is estimated because the simulated individual's blood Pb level is below the level for predicting IQ loss with this function. If the blood Pb level is greater than the cutpoint, then the log-linear function is used to predict IQ loss for the portion of the estimated blood Pb level extending above the cutpoint.

- *Log-linear function with low-exposure linearization:* Each modeled blood Pb level is compared against the point of linearization. If the blood Pb level is below the point where the function becomes linear, than the linear slope is used to predict IQ loss. If the modeled Pb level is above the point where the function becomes linear, than IQ loss is calculated as the sum of IQ loss across the linear portion of the curve plus the additional contribution from the log-linear portion of the function extending up to the total blood Pb level.

- *Two-piece linear function:* Similar to the last function, the modeled blood Pb level is compared against the blood Pb level of the "hinge". If the blood Pb level falls below the hinge, as is the case for most simulated individuals at the three case studies, then the steeper, low-exposure slope is used to estimate IQ loss. If the simulated blood Pb level falls above the hinge, then IQ loss associated with the low-exposure (steeper slope) piece of the function (for the portion of the blood Pb level up to the hinge) is combined with IQ loss estimated using the shallower and higher exposure slope, for that portion of the blood Pb level extending above the hinge.

The IQ loss estimates generated using this approach are pooled to form a population-level distribution of IQ loss for a given study area. Each of these IQ loss estimates are pathway apportioned among policy-relevant pathways and policy-relevant background based on pathway contribution to the underlying blood Pb levels (see Sections 2.4.3 and 3.2.2). As with pathway apportionment for blood Pb levels (Section 3.2.2), pathway apportionment of risk estimates is also at the level of exposure zone. All simulated individuals from a given zone are assigned the

same pathway apportionment. As noted with regard to blood Pb levels (Section 3.2.2), there is increased uncertainty associated with pathway apportioned IQ loss estimates for higher-end risk percentiles, since these reflect an assumption that relative pathway contributions at central tendency exposure levels hold at the high-end percentiles of the blood Pb level distribution.

Just as with the population-level exposure estimates discussed in Section 3.2.2, risk estimates generated using the approach outlined above are used to generate several types of risk metrics, depending on the case study. For the two point source case studies, because they are location-specific and include a defined and enumerated receptor population, two categories of risk metrics are generated:

- *Population-weighted risk (IQ loss) percentiles:* IQ loss (with pathway apportionment) for simulated individuals representing specific points along the population risk distribution (e.g., 50, 90, 95 percentile simulated individuals).

- *Incidence counts:* Number of children within a given study area projected to experience a magnitude of IQ loss.

As for the exposure estimates, the general urban case study risk metrics are restricted to population-weighted risk (IQ loss) percentiles.

4.2 RISK ESTIMATES

Estimates of IQ loss resulting from Pb exposure have been developed for each air quality scenario in each case study (see Appendix K). In addition, IQ loss results have been developed for subareas associated with each of the point-source case studies (see Appendix P). Further, multiple sets of risk results were generated for each combination of case study and air quality scenario, in an effort to consider key sources of uncertainty and their impact on blood Pb estimates (see Section 2.4.6.2). That is, twenty four separate risk distributions were generated for each air quality scenario of the general urban case study and six distributions were generated for each air quality scenario of the point source case studies (see Table 2-3).

As discussed in Section 2.4.6.2, generating multiple sets of risk results for each combination of case study and air quality scenario provides a range of results reflecting the impact of key sources of uncertainty on risk results. However, because we could not assign specific confidence levels to each modeling approach, these multiple sets of results are not translated into single uncertainty distributions of risk for each air quality scenario in each case study. Therefore, we consider the multiple sets of risk results to span the best estimate risk distribution. In response to CASAC comments on the July 2007 draft risk assessment report (Henderson, 2007a), however, we have omitted results from the two-piece linear (with hinge at

10.82 µg/dL for concurrent blood metric) from the summary of results presented in this chapter (these results are available in Appendix K).

Three types of risk estimates are summarized in Tables 4-2 and 4-3:

- *Risk estimates for recent air:* This estimate is the portion of total IQ loss derived from inhalation of ambient air Pb and ingestion of Pb in indoor dust that is predicted to be associated with ambient air Pb concentrations based on the indoor dust model (i.e., "recent air" risk per Sections 2.4.3 and 3.2.2) Given the modeling approach used in this analysis and its limitations, this set of risk estimates is expected to be most responsive to alternative NAAQS.

- *Risk estimates for recent air plus past air (other indoor dust and outdoor soil contribution):* This estimate is the portion of total IQ loss derived from inhalation of ambient air Pb, ingestion of indoor dust and ingestion of outdoor soil/dust (Sections 2.4.3 and 3.2.2). That is, this estimate includes the recent air pathways in addition to other indoor dust and outdoor soil pathways. This estimate include some contribution from indoor paint because the indoor dust models handicapped our ability to parse out this contribution. Otherwise, these estimates reflect contributions from all except the policy-relevant background pathways of diet and drinking water.

- *Risk estimates for all pathway contributions*: This estimate is the IQ loss associated with the total blood Pb concentration.

As noted previously (Sections 2.4.3 and 3.2.2), policy-relevant pathways are represented in the first two bullets above, with the true values for the policy-relevant pathways considered to fall between the estimates for "recent air" and those for "recent" plus "past air". Additionally, with regard to the urban case study "recent air" and "recent" plus "past air" categories, an artifact of the hybrid dust Pb model tends to mask trends in the two components of dust Pb (recent air and other), which contribute to "recent air" and "past air" estimates, respectively (see Section 4.3.1 discussion of this uncertainty). For the primary Pb smelter case study, uncertainty in parsing out the "recent air" and "other" components of indoor dust (specifically for the site-specific regression model used in the remediation zone) have led us to conclude that only "recent plus past air" IQ loss estimates should be presented and "recent air" results should not be separately presented for the primary Pb smelter, as is done for the other case studies (see Section 3.1.4.2).

IQ loss estimates reflecting all exposure pathways (total), the "recent air" pathways (see Section 3.2.2), and the recent plus past air pathways are presented in Tables 4-2 and 4-3. The total estimates presented in Table 4-2 are those for the median in the distribution of total risk, and the estimates for the other two categories in that table (i.e., recent air and recent plus past air) are the values for those categories associated with the median for the total exposure pathway

estimate. The corresponding estimates for the 95th percentile in the distribution of IQ loss estimates for total blood Pb for each case study are presented in Table 4-3. This presentation may lead to some seeming inconsistencies in trends for recent air and recent air plus past air risk estimates across air quality scenarios. This is because the recent air or recent plus past air estimates associated with the median and 95th percentile total blood Pb estimate may not necessarily be the median and 95th percentiles of the distribution of estimates for those specific categories. This is because the blood Pb level (and associated total IQ loss) for a simulated child reflects not only the total Pb uptake (from both background and ambient air-related pathways), but also the GSD-based adjustment factor. This means that two simulated children could have identical blood Pb levels (and IQ loss estimates), but one child could have a higher recent air exposure, with the other child having their lower recent air exposure compensated for by a higher GSD-based adjustment factor, thereby resulting in both children having the same total blood Pb level.

In presenting IQ loss estimates, all values are rounded to one decimal place. The complete set of risk estimates are presented in Appendix K. The risk estimates in Tables 4-2 and 4-3 indicate the following:

- IQ loss estimates were uniformly higher for the current NAAQS air quality scenario compared with any of the alternative NAAQS or current conditions air quality scenarios for the primary Pb smelter and general urban case studies. This trend held for both the median and 95th percentile risk results. Risk estimates for the secondary Pb smelter case study (both the full study area and sub-area) differed little across any of the air quality scenarios.
- IQ loss estimates decreased with increasingly lower alternative NAAQS, as a general observation across case studies. However, this trend varied in consistency and magnitude. For example, the trend was more pronounced with the recent air estimates (both median and 95th percentile) for the general urban case study, and was less obvious with the risk estimates for total blood Pb (in this case, it is likely that background exposures overwhelmed the recent air related exposures). The trend was also stronger for the subarea of the primary Pb smelter, compared with the full study area, reflecting the fact that the subarea has higher ambient air Pb impacts and therefore, is likely to demonstrate greater variation in IQ impacts across alternative NAAQS.
- As expected, risks associated with recent plus past air contributions are larger, across all population percentiles for all case studies and air quality scenarios evaluated, compared with the recent air risk estimates. This reflects that the “past air” category includes contributions from the outdoor soil/dust contribution to indoor dust, historical air contribution to indoor dust, and outdoor soil/dust pathways, while “recent air” refers to contributions associated with outdoor ambient air Pb levels, either by inhalation of ambient air Pb or ingestion of indoor dust Pb predicted to be associated with outdoor ambient air Pb levels, including resuspended, previously deposited Pb

(see Section 2.4.3). As noted previously (Sections 2.4.3 and 3.2.2), policy-relevant pathways are represented in both of these categories, with the true values for the policy-relevant pathways considered to fall between the estimates for "recent air" and those for "recent" plus "past air".

- Recent air and recent plus past air IQ loss estimates were higher for the subareas of the point source case studies. This reflects the fact, mentioned above, that these subareas experience significantly greater ambient air-related impacts compared with the full study areas. While the full study areas include a large number of children in areas with ambient air Pb levels well below the alternative NAAQS considered, whose exposures consequently are less affected under alternative NAAQS, these areas are omitted from the subarea, thus increasing the percentage of simulated children whose exposures and associated risks are notably reduced under alternate NAAQS.

- Risk estimates generated for both of the point source case studies (full study areas) are lower across all population percentiles (for the same air quality scenario) compared with the general urban case study. This reflects two factors. First, the point source case studies include spatial gradients, such that areas near the facility may be at or near a given ambient air Pb level (associated with an air quality scenario), while the majority of the study area (and consequently study population) experiences ambient air Pb levels considerably lower than the level associated with the air quality scenario. By contrast, the general urban case study assumed a uniform ambient air Pb concentration at the level of the air quality scenario being evaluated. Consequently, the entire hypothetical study population would experience that uniform air concentration. The second reason for the point source case studies having risk estimates lower than those for the general urban case study, is that they only consider Pb emitted from the industrial facility of interest and do not consider emissions from any other sources within the study area, or near the study area that could impact populations within the study area. By contrast, current conditions for the general urban case study is based on monitoring data for urban locations, which will by default reflect the actual mix of Pb sources influencing air Pb concentrations. Consequently, the general urban case study reflects the combined impact of different Pb sources, while the point source case studies only consider emissions from the smelter under consideration and the influence of these emissions lessens (and consequently air Pb concentrations decline) with distance from the facilities.

- For the general urban case study, the large difference seen in the recent air risk estimates between the three higher and the lowest alternate NAAQS scenario for the 95^{th} percentile is not seen for the risk estimates for recent plus past air contributions. This reflects an artifact of the approach used in the hybrid indoor dust model to characterize "other" (non-"recent air"-associated) contributions to indoor dust (see Appendix G). Because of the method used to convert dust loadings to dust concentrations within the hybrid dust model and the approach used to apportion total dust concentrations between other and recent air components, the "other" indoor dust concentration predicted by this model varies with air quality scenario, with that value increasing as the ambient air Pb level decreases (see Appendix C). This means that, as the recent air contribution to exposure through indoor dust ingestion decreases (as the lower alternative NAAQS levels are considered), the estimate of contribution of

"other" indoor dust actually increases. This partially accounts for the trend noted above. That is, even though recent air risk estimates drop, they are partially offset by risk resulting from an increase in exposure to the "other" Pb contributions to indoor dust. The impact of this aspect of the hybrid dust model is identified in Section 4.3.1 and evaluated in a sensitivity analysis presented in Chapter 5.

- Risk estimates for the secondary Pb smelter case study are considerably lower than risk estimates for the other case studies (for all air quality scenarios considered). Limitations in the design for the secondary Pb smelter case study (described in Section 4.3.1) contribute significant uncertainty to these estimates. Accordingly these results should not be used to draw conclusion regarding similar facilities risks experienced by populations living in the vicinity of other similar point sources.

Table 4-2. Summary of risk estimates for medians of total-exposure risk distributions.

Case Study and Air Quality Scenario	Points IQ loss[a]					
	Recent air[b]		Recent plus past air[b]		Total Pb exposure	
	Low	High	Low	High	Low	High
General urban case study						
Current NAAQS (1.5 µg/m^3, max quarterly)	2.1	4.3	2.9	6.0	3.5	7.0
Current conditions - 95th percentile (0.87 µg/m^3, max quarterly)	0.4	2.0	1.3	4.0	1.8	5.4
Alternative NAAQS (0.5 µg/m^3, max monthly)	0.5	2.1	1.4	4.1	1.9	5.4
Alternative NAAQS (0.2 µg/m^3, max quarterly)	0.3	1.7	1.2	3.8	1.7	5.2
Current conditions – mean (0.14 µg/m^3, max quarterly)	0.2	1.4	1.1	3.6	1.5	5.0
Alternative NAAQS (0.2 µg/m^3, max monthly)	0.2	1.4	1.0	3.6	1.5	5.0
Alternative NAAQS (0.05 µg/m^3, max monthly)	<0.1	0.6	0.9	3.1	1.3	4.6
Primary Pb smelter - *full study area*						
Current NAAQS (1.5 µg/m^3, max quarterly)	NA[c]		0.6	2.5	1.1	3.8
Alternative NAAQS (0.5 µg/m^3, max monthly)			0.8	2.2	1.0	3.7
Alternative NAAQS (0.2 µg/m^3, max quarterly)			0.6	2.7	0.9	3.6
Alternative NAAQS (0.2 µg/m^3, max monthly)			0.4	2.1	0.9	3.6
Alternative NAAQS (0.05 µg/m^3, max monthly)			0.4	2.4	0.9	3.5
Primary Pb smelter - *1.5 km subarea*						
Current NAAQS (1.5 µg/m^3, max quarterly)	NA[c]		3.2	6.3	3.7	6.8
Alternative NAAQS (0.5 µg/m^3, max monthly)			2.1	4.9	2.6	5.8
Alternative NAAQS (0.2 µg/m^3, max quarterly)			1.5	4.3	2.0	5.2
Alternative NAAQS (0.2 µg/m^3, max monthly)			1.2	4.0	1.9	2.0
Alternative NAAQS (0.05 µg/m^3, max monthly)			0.9	2.9	1.4	4.6
Secondary Pb smelter - *full study area*						
Current conditions	0[d]	<0.1	0[d]	1.0	0[d]	2.8
Alternative NAAQS (0.5 µg/m^3, max monthly)	0	<0.1	0	1.0	0	2.7
Alternative NAAQS (0.2 µg/m^3, max quarterly)	0	<0.1	0	1.0	0	2.7
Alternative NAAQS (0.2 µg/m^3, max monthly)	0	<0.1	0	0.9	0	2.7
Alternative NAAQS (0.05 µg/m^3, max monthly)	0	<0.1	0	1.0	0	2.7
Secondary Pb smelter - *1.5 km subarea*						
Current conditions	<0.1	0.4	0.4	2.7	0.8	3.8
Alternative NAAQS (0.5 µg/m^3, max monthly)	<0.1	0.1	0.4	1.9	0.7	3.7
Alternative NAAQS (0.2 µg/m^3, max quarterly)	<0.1	<0.1	0.3	1.7	0.6	3.6
Alternative NAAQS (0.2 µg/m^3, max monthly)	<0.1	0.1	0.4	1.9	0.6	3.6
Alternative NAAQS (0.05 µg/m^3, max monthly)	<0.1	<0.1	0.3	2.1	0.6	3.6

a - With the exception of "true zero values" (see note d below), all estimates are rounded to one decimal place.
b -The term “past air” includes contributions from the outdoor soil/dust contribution to indoor dust, historical air contribution to indoor dust, and outdoor soil/dust pathways, while “recent air” refers to contributions from inhalation of ambient air Pb or ingestion of indoor dust Pb predicted to be associated with outdoor ambient air Pb levels, with outdoor ambient air also potentially including resuspended, previously deposited Pb (see Section 2.4.3).
c – “Recent air” estimates were not developed for the primary Pb smelter case study (see Section 3.1.4.2).
d – Table entries of “0” (in the “low” column) are truly zero values and reflect application of log-linear concentration-response function with cutpoint to blood Pb estimates below 1 µg/dL.

Table 4-3. Summary of risk estimates for 95th percentiles of total exposure risk distributions.

Case Study and Air Quality Scenario	Points IQ loss[a]					
	Recent air[b]		Recent plus past air[b]		Total Pb exposure	
	Low	High	Low	High	Low	High
General urban case study						
Current NAAQS (1.5 µg/m^3, max quarterly)	3.3	6.0	4.6	8.3	5.5	10.5
Current conditions - 95th percentile (0.87 µg/m^3, max quarterly)	1.0	3.4	3.1	6.6	4.2	8.8
Alternative NAAQS (0.5 µg/m^3, max monthly)	1.0	3.6	3.1	6.9	4.2	9.1
Alternative NAAQS (0.2 µg/m^3, max quarterly)	0.7	2.9	2.8	6.4	4.0	8.7
Current conditions – mean (0.14 µg/m^3, max quarterly)	0.5	2.5	2.7	6.1	3.9	8.5
Alternative NAAQS (0.2 µg/m^3, max monthly)	0.4	2.3	2.7	6.0	3.9	8.4
Alternative NAAQS (0.05 µg/m^3, max monthly)	0.1	1.0	2.4	5.5	3.7	8.1
Primary Pb smelter - *full study area*						
Current NAAQS (1.5 µg/m^3, max quarterly)	NA[c]		2.3	4.2	3.7	6.8
Alternative NAAQS (0.5 µg/m^3, max monthly)			2.5	4.5	3.4	6.6
Alternative NAAQS (0.2 µg/m^3, max quarterly)			1.9	5.2	3.2	6.5
Alternative NAAQS (0.2 µg/m^3, max monthly)			2.0	4.0	3.2	6.4
Alternative NAAQS (0.05 µg/m^3, max monthly)			1.5	4.9	3.1	6.3
Primary Pb smelter - *1.5 km subarea*						
Current NAAQS (1.5 µg/m^3, max quarterly)	NA[c]		5.7	8.8	6.8	9.5
Alternative NAAQS (0.5 µg/m^3, max monthly)			5.1	7.5	5.8	8.5
Alternative NAAQS (0.2 µg/m^3, max quarterly)			4.7	6.9	5.3	7.8
Alternative NAAQS (0.2 µg/m^3, max monthly)			3.9	6.7	4.9	7.6
Alternative NAAQS (0.05 µg/m^3, max monthly)			2.8	4.7	3.6	6.5
Secondary Pb smelter - *full study area*						
Current conditions	<0.1	<0.1	0.8	1.8	2.4	5.2
Alternative NAAQS (0.5 µg/m^3, max monthly)	0.1	0.1	1.0	2.0	2.3	5.1
Alternative NAAQS (0.2 µg/m^3, max quarterly)	<0.1	<0.1	0.8	2.1	2.3	5.1
Alternative NAAQS (0.2 µg/m^3, max monthly)	<0.1	<0.1	1.1	1.8	2.3	5.1
Alternative NAAQS (0.05 µg/m^3, max monthly)	<0.1	<0.1	0.8	1.7	2.3	5.1
Secondary Pb smelter - *1.5 km subarea*						
Current conditions	0.5	1.4	2.1	5.0	3.2	6.3
Alternative NAAQS (0.5 µg/m^3, max monthly)	0.4	0.5	2.4	3.9	3.2	6.3
Alternative NAAQS (0.2 µg/m^3, max quarterly)	0.2	0.1	2.3	3.2	3.1	6.1
Alternative NAAQS (0.2 µg/m^3, max monthly)	<0.1	0.2	1.4	4.3	3.1	6.1
Alternative NAAQS (0.05 µg/m^3, max monthly)	<0.1	<0.1	1.6	4.2	3.0	6.1

a - Estimates are rounded to one decimal place.
b -The term "past air" includes contributions from the outdoor soil/dust contribution to indoor dust, historical air contribution to indoor dust, and outdoor soil/dust pathways, while "recent air" refers to contributions from inhalation of ambient air Pb or ingestion of indoor dust Pb predicted to be associated with outdoor ambient air Pb levels, with outdoor ambient air also potentially including resuspended, previously deposited Pb (see Section 2.4.3).
c – "Recent air" estimates were not developed for the primary Pb smelter case study (see Section 3.1.4.2).

4.3 UNCERTAINTY CHARACTERIZATION AND SENSITIVITY ANALYSIS

This section discusses uncertainty related to exposure and risk estimates generated for this analysis. Several methods have been used to examine uncertainty in our modeling approach and its potential impact on exposure and risk estimates. These include:

- qualitative discussion of key sources of uncertainty and their potential impact on exposure and risk estimates (Section 4.3.1),
- evaluation of model performance, including comparison with empirical data (Section 4.3.3), and,
- development of multiple sets of exposure and risk estimates for each assessment scenario that illustrate the combined impact of different models and input data on risk results and the associated uncertainty (Section 4.3.4).

In addition to these methods for considering uncertainty, a sensitivity analysis was also conducted to characterize the potential impact of individual modeling elements on risk results (see Section 4.3.2). Each of these elements of the uncertainty characterization, along with the sensitivity analysis, is briefly summarized below.

4.3.1 Qualitative Discussion of Key Sources of Uncertainty

Given the complexity of this assessment and the range of models and input data used in completing it, there is a wide variety of sources of uncertainty potentially impacting the exposure and risk results generated (Appendix M, Table M-1). This section identifies those sources of uncertainty with the potential to have a significant impact on risk results (see Appendix M, Table M-1 bold text). When it was feasible with the available methods and data, these key sources of uncertainty have been quantitatively assessed for their potential impact on risk results either as part of the multiple modeling approaches implemented (see Section 4.3.4). In addition, some of these modeling elements have been included in the sensitivity analysis (see Section 4.3.2). Key sources of uncertainty include:

- *Temporal aspects*: As described in Section 2.4.1, exposure for the simulated child population begins at birth and continues for 7 years, with Pb concentrations in all exposure media remaining constant throughout the period, and children residing in the same exposure zone throughout the period. In characterizing exposure media concentrations, annual averages are derived and held constant through the seven year period. Exposure factors and physiological parameters vary with age of the cohort through the seven year exposure period, several exposure factors and physiological parameters are varied on an annual basis within the blood Pb modeling step (see Section 3.2). These aspects are a simplification of population exposures that contributes uncertainty to our exposure and risk estimates.

- *General urban case study*: This case study differs from the others in several ways (described in more detail in Section 2.2.1). It is by definition a general case study and not based on a specific location. There is a single exposure zone for the case study within which all media concentrations of Pb are assumed to be spatially uniform; that is, no spatial variation within the area is simulated (see Sections 2.4.2, and 3.1.1). Additionally, the case study does not rely on any specific demographic values. Within the single exposure zone a theoretical population of unspecified size is assumed to be uniformly distributed. Thus this case study is a simplified representation of urban areas intended to inform our assessment of the impact of changes in ambient Pb concentrations on risk, but which carries with it attendant uncertainties in our interpretation of the associated exposure and risk estimates. For example, the risk estimates for this case study, while generally representative of an urban residential population exposed to the specified ambient air Pb levels, cannot be readily related to a specific urban population. Specific urban populations are spatially distributed in a nonuniform pattern and experience ambient air Pb levels that vary through time and space. Consequently, interpretations of the associated blood Pb and risk estimates with regard to their relevance to specific urban residential exposures carry uncertainty and presumably an upward bias in risk, particularly for large areas, across which air concentrations may vary substantially.

- *Point source case studies:* Dispersion modeling was used to characterize ambient air Pb levels in the point source case studies. This approach simulates spatial gradients related to dispersion and deposition of Pb from emitting sources. In the case of the point sources modeled, sources were limited to those associated with the smelter operations, and did not include other sources such as resuspension of roadside Pb not immediately related to facility operations, and other stationary sources of Pb within or near the study area. This means that, with distance from the facility, there is likely underestimation of ambient air-related Pb exposure because with increased distance from the facility there would be increasing influence of other sources relative to that of the facility. We believe this limitation to have more significant impact on risk estimates associated with the full study than on those for the subareas, and to perhaps have a more significant impact on risk estimates associated with the smaller secondary Pb smelter (see below).

- *Secondary Pb smelter case study:* Air Pb concentration estimates derived from the air dispersion modeling completed for the secondary Pb smelter case study are subject to appreciably greater uncertainty than that for those for the primary Pb smelter case study due to a number of factors, including: (a) a more limited and less detailed accounting of emissions and emissions sources associated with the facility (particularly fugitives), (b) a lack of prior air quality modeling analyses and performance analyses, and (c) a substantially smaller number of Pb-TSP monitors in the area that could be used to evaluate and provide confidence in model performance[1]. Further, as mentioned in the previous bullet, no air sources of Pb other than those associated with the facility were accounted for in the modeling. Given the relatively smaller magnitude of

[1] The information supporting the air dispersion modeling for the primary Pb smelter case study (see Section 3.5.1.1) provides substantially greater confidence in estimates for that case study.

emissions from the secondary Pb smelter, the underestimating potential of this limitation with regard to air concentrations with distance from the facility has a greater relative impact on risk estimates for this case study than for the primary Pb smelter case study. The aggregate uncertainty of all of these factors has left us with low confidence in estimates for this case study. We note that exposure and risk estimates are lower than those for the other case studies. Although we had initially intended to use this case study as an example of areas near stationary sources of intermediate size (smaller than the primary Pb smelter), our experience with this analysis indicates that substantially more data and multiple case studies differing in several aspects would be needed to broadly characterize risks for such a category of Pb exposure scenarios.

- *Indoor dust Pb modeling for the general urban case study:* The hybrid indoor dust Pb model was developed for the general urban case study due to a lack of an existing urban-focused dust model, and this hybrid model is subject to particular uncertainties. Key among these uncertainties is failure to consider house-to-house variability in factors related to the infiltration of ambient air Pb indoors and subsequent buildup of Pb on indoor surfaces. This handicaps our ability to predict variation in indoor dust Pb levels for non-typical residential conditions. In addition, a lack of comprehensive data in the literature on rates and efficiency of indoor cleaning, introduces uncertainty into the model. The method used to convert Pb loadings generated by the hybrid model to Pb concentrations is also subject to uncertainty, partially due to the age of the underlying dataset. Because the underlying dataset is an older dataset (see Section 3.1.4.1), there is potential for bias toward greater representation of housing with Pb-based paint. Additionally, the component of the hybrid model that converts dust Pb loadings to concentrations (Appendix G, Section G.3.4) introduces an uncertainty into the estimates of percent contribution from recent air compared to other pathways. This is related to a nonlinearity in this conversion and results in the "other" indoor dust concentration predicted by this model varying with air quality scenario, with that value increasing as the ambient air Pb level decreases (see Appendix C). This means that, as the recent air contribution to exposure through indoor dust ingestion decreases for the lower alternative NAAQS levels, the estimate of contribution of "other" indoor dust actually increases. This issue of the uncertainty in estimating the "other" component of indoor dust (and relating it to the "recent air" component) has been examined further as a sensitivity analysis (see Section 5.3.3.4).

- *Estimates of indoor dust Pb concentrations for the primary Pb smelter case study (application of the site-specific regression model):* There is uncertainty associated with the site-specific regression model applied in the remediation zone (see Section 3.1.4.2), and relatively greater uncertainty associated with its application to air quality scenarios that simulate notably lower air Pb levels. Limitations in the dataset from which the model was derived limited its form to that of a simple regression that predicts dust Pb concentration as a function of air Pb concentration plus a constant (intercept). We recognize, however, that there may be variables in addition to air that influence dust Pb concentrations and their absence in the regression contributes uncertainty to the resulting estimates. To the extent that these unaccounted for variables are spatially related to the smelter facility Pb sources, our estimates could be biased, not with regard to the absolute dust Pb concentration, but with regard to

differences in dust Pb concentration estimate between different air quality scenarios. Those differences may be overestimated because of potential overestimation of the air coefficient and underestimation of the intercept in the regression model. Examples of such not-accounted-for variables are roadside dust Pb and historically contributions to current levels of indoor dust Pb (e.g., dust Pb contributed to a house in the past that continues to contribute to current dust Pb levels). See Sections 3.1.4.2 and 3.5.1.3 for additional discussion.

- *Estimates of outdoor soil/dust Pb concentrations*: Outdoor soil/dust Pb concentrations in all air quality scenarios have been set equal to the values for the current conditions scenarios. That is, we are not simulating an impact of changes in air Pb concentrations on soil concentrations, or the associated impact on dust concentrations, blood Pb and risk estimates. In areas where air concentrations have been greater in the past, however, implementation of a reduced NAAQS might be expected to yield reduced soil Pb levels over the long term. As described in Section 2.3.3, however, there is potentially uncertainty associated with this specification, particularly with regard to implications for areas in which a Pb source may locate where one of comparable size had not been previously. Additionally, we note that control measures implemented to meet alternative NAAQS may result in changes to soil Pb concentrations; these are not reflected in the assessment

- *Recontamination of residential yard soil near the primary Pb smelter*: Although data collected in residential yards within ¾ mile of the primary Pb smelter indicate a trend of increasing surface soil Pb concentrations, soil Pb estimates in this area of the primary Pb smelter case study were not adjusted using this information. Therefore, these estimates likely were underestimates of soil Pb levels associated with the current ambient air Pb levels. Because the indoor dust Pb model used in this part of this case study did not rely on outdoor soil/dust concentrations this did not affect indoor dust Pb estimates, although as described in a previous bullet the lack of soil data to consider in developing the indoor dust Pb model increased uncertainty in that model's results. Additionally, the outdoor soil/dust ingestion pathway is a small contributor to blood Pb levels, which also reduced the impact of this factor on blood Pb estimates .

- *Interindividual variability GSD*: There is uncertainty associated with the GSD specified for each case study. In the case of the general urban case study, the use of a uniform ambient air Pb concentration across the hypothetical study area complicates selection of a GSD. This is because any urban area for which GSDs can be developed would be subject to spatial variation in ambient air Pb levels. To the extent that these ambient air Pb levels influence children's blood Pb levels, this influence would be reflected in the associated GSD. In the case of the two point source case studies, the use of the spatial templates complicates selection of a GSD for a different reason. Because the spatial templates are intended to contribute variability in risk results related to spatial gradients in air-related Pb media concentrations, the GSDs used for these case studies need to reflect the remaining sources of variability in blood Pb levels (e.g., interindividual variability in behavior and biokinetics related to Pb exposure as well as variability in non-air related Pb exposures). The extent to which the specified GSDs reflect the sources of variability at play in each of these case studies is unknown.

- *Exposure pathway apportionment for higher percentile blood Pb level and IQ loss estimates:* As discussed in Section 3.2.2, pathway apportionment of blood Pb levels for higher population percentiles is specified to be the same as that estimated using the central tendency estimate of blood Pb in an exposure zone. This introduces uncertainty into projections of pathway apportionment for higher population percentiles of blood Pb and IQ loss. In reality, pathway apportionment may shift as you consider higher exposure percentiles. For example, paint and/or drinking water exposures may increase in importance, with air-related contributions decreasing as an overall percentage of blood Pb levels and associated risk.
- *Projection of IQ loss at lower exposure levels*: Because available epidemiological data are limited in their description of the relationship of IQ decrement to blood Pb at lower blood Pb levels (e.g., < 5 µg/dL), there is uncertainty associated with projecting IQ loss at these blood Pb levels which are particularly relevant to the current population of children in the U.S. (see discussion in Section 2.1.5). For additional discussion of uncertainty associated with predicting IQ loss resulting from Pb exposure, see Sections 5.3.3.1.

4.3.2 Sensitivity Analysis

Sensitivity analysis techniques were used to evaluate the impact of individual modeling elements on exposure and risk estimates. Specifically, we used a "one element at a time elasticity analysis" approach, in which the full risk model was run with one of the selected modeling elements adjusted to reflect an alternate input value or modeling choice. The results of that run with the modified modeling element was then compared to those for the "baseline risk" run to determine the magnitude of the impact on risk results of selections for that one modeling element.

The sensitivity analysis described in Appendix L focused on the general urban case study, reflecting the fact that this case study has potential relevance for a larger number of Pb-exposed children compared with the two point source case studies. Additionally, we recognized that exposure patterns for urban children can be highly variable compared with exposures near Pb smelters and the availability of data sets for specific urban areas that reflect current blood Pb levels is limited. The modeling elements examined in the sensitivity analysis included those inputs and modeling steps believed to have a significant potential for impacting exposure and risk results (e.g., oral uptake factor, interindividual blood Pb variability GSD, biokinetic model, concentration-response function for IQ loss). The one-element-at-a-time sensitivity analysis indicates which of the modeling elements included in the sensitivity analysis has the greatest impact on risk results, which can be used to guide future efforts to refine the overall risk model. Note, that an additional sensitivity analysis involving the hybrid indoor dust model and specifically the derivation of the "other" component of indoor dust was conducted as part of the core analysis (see Section 5.3.3.4).

The results of the sensitivity analysis (Appendix L) can be considered both in terms of the impact of individual modeling element choices on (a) overall risk results and (b) recent air risk results, i.e., IQ loss estimates for inhalation plus ingestion of indoor dust Pb predicted to be associated with ambient outdoor air Pb levels, where ambient outdoor air potentially includes resuspended, previously deposited Pb (see Section 2.4.3). Given the relevance of the recent air exposure pathways to the NAAQS review, the results of the sensitivity analysis are summarized here in terms of their impact on recent air risk results, rather than total risk results. Results of the sensitivity analysis for total risk estimates, however, are similar (Appendix L).

Results of the sensitivity analysis showed the following modeling elements to have the greatest impact on recent air risk estimates for the general urban case study (i.e., $\geq$40% impact on the 95th percentile recent air risk estimates and an even greater impact, in terms of percent change, on median estimates). These elements are presented in order of decreasing magnitude of impact on risk estimates (Note, results presented here reflect the impact on the 95th percentile risk results - see Appendix L for sensitivity analysis results reflecting impact on the median population percentile):

- *IQ loss function:* Use of the log-linear with linearization IQ loss model (the model producing the greatest IQ loss) resulted in 226% increase in recent air risk results over results for baseline which used the 2-piece linear model. These results suggest that characterization of the relationship between Pb exposure and IQ loss has the greatest impact on high-end risk results.
- *Indoor dust Pb modeling:* Use of a combination of high-end modeling parameters for the hybrid dust model results in a 139% increase in recent air risk estimates over results using the hybrid model with those input values used in the primary analysis. In addition, use of the air-only regression model in place of the hybrid model results in a 60% decrease in the recent air risk results. These results together suggest that predicting the relationship between outdoor ambient air Pb and indoor dust Pb is an important factor impacting risk results.
- *Blood Pb modeling:* Use of the Leggett model as compared to the IEUBK model, results in a 170% increase in recent air risk estimates. This indicates the importance of the blood Pb modeling step in the analysis. However, these results need to be considered in the context of the performance evaluation for blood Pb which suggested that the Leggett model may be significantly overpredicting blood Pb levels, and the fact that the Legget model is designed to provide results over shorter time frames (e.g., with daily time step), while the IEUBK model provides longer-term quasi steady-state estimates.
- *Monitor-based ratio of maximum quarterly to annual average Pb-TSP concentrations:* A ratio of monitored maximum quarterly average to annual average air Pb concentrations is used in the general urban case study to translate maximum quarterly average air concentrations into the annual average values used in the risk assessment.

Use of the 95th percentile of urban Pb-TSP monitor ratios as compared to the mean value, results in a 50% reduction in recent air risk estimates.

- *GSD reflecting interindividual variability in blood Pb levels:* Use of the larger GSD (2.1) in place of the smaller value (1.7) resulted in a 40% increase in the recent air risk estimates.

Alternate selections for the other modeling elements included in the sensitivity analysis yielded changes to the 95th percentile risk results of less than 40% from baseline.

4.3.3 Performance Analyses

Performance evaluation for the exposure assessment (Section 3.5) focused on evaluation of estimates of Pb in ambient air, outdoor soil, and indoor dust (discussed in Section 3.5.1) and estimates of Pb in blood (covered in Section 3.5.2). Consideration of the results of performance evaluation can provide insights into potential sources of uncertainty in an analysis, by identifying those elements of the analysis that appear inconsistent with available empirical data. This can, in turn, point to underlying bias or other errors associated with that particular modeling step, reflecting either parameter or model uncertainty. This section identifies key findings of the performance analysis describing the nature of associated uncertainty, including results which either supported modeling elements or suggested increased uncertainty.

- *Modeled ambient air Pb levels*: The evaluation of the air model performance for the primary Pb smelter case study (Sect 3.5.1.1) indicated performance generally consistent with empirical data, increasing our confidence in air-related results generated for this case study. Evaluation of air dispersion model performance for the secondary Pb smelter suggested the potential for low bias in predictions of ambient air Pb concentrations.
- *Estimates of outdoor soil/dust Pb concentration for secondary Pb smelter case study*: The use of a combination of dispersion and soil mixing models to generate a spatial pattern of concentrations combined with a scaling factor based on a surrogate location contributes significant uncertainty to the soil Pb characterization for this case study. Specifically, while the approach used is believed unlikely to significantly underestimate soil Pb levels at this type of facility, the exact extent to which it is representative of conditions at this specific location is not known.
- *Modeled indoor dust Pb concentrations:* Evaluation of the hybrid indoor dust model used in the general urban case study suggested the potential for both under- and overestimation. The mechanistic ambient air-related portion of the model may underestimate that component of dust Pb, while the indoor dust loading-to-concentration conversion algorithm may contribute to overestimate of dust Pb. It is not know to what extent these two biases cancel out each other. Overall comparison of indoor dust Pb concentrations generated for the three case studies against available empirical data suggest that: (a) for the general urban case study, estimates fall within the range of measured values from a national-scale study, adding confidence to the estimates, (b) central tendency estimates for the primary Pb smelter are close to the

lower end of the range for the national-scale dataset referenced above for the general urban case study and high-end estimates seem to fit with available data near smelters, and (c) comparison of estimates for the secondary Pb smelter against empirical data suggest that these estimates may be biased low.

- *Evaluation of candidate blood Pb models*: A number of performance evaluations were completed on the two candidate blood Pb models considered for this analysis (IEUBK and Leggett), as well as the Lanphear empirical model. The results of these performance evaluations, which included application of both models in replicating national-scale child blood Pb levels (NHANES IV results) and blood Pb levels for an urban child cohort, suggested that the Leggett model consistently overpredicted blood Pb levels by a factor of 3 to 6, while IEUBK estimates were usually within a factor of 2. These findings, in addition to CASAC recommendations, resulted in our selecting the IEUBK model as primary blood Pb model for this assessment, with the Leggett model being reserved for application in the sensitivity analysis.

- *Outdoor air Pb-to-blood Pb ratios*: Three sets of outdoor air Pb-to-blood Pb ratios were derived. These related outdoor ambient air Pb to blood Pb resulting from: (1) the inhalation pathway only; (2) all recent air pathways (inhalation plus ingestion of indoor dust Pb predicted to be associated with ambient air Pb levels, with ambient air potentially including resuspended, previously deposited Pb); and (3) all recent and past air pathways (see Section 2.4.3). All ratios were derived prior to application of the GSD reflecting interindividual variability in blood Pb levels and therefore reflect central tendency blood Pb levels and not high-end population percentiles. The modeled ratios were compared to both empirical data and statistically derived ratios based on a pooled analysis (Section 3.5.2.2). With the exception of the primary Pb smelter case study recent air ratio using 95^{th} percentile air concentration and the general urban case study recent air ratios for the hybrid dust model, the ratios for recent air contribution to concurrent blood Pb level (Table 3-10) generated for the three case studies were reasonable and supported by available empirical data. As described in Section 3.5.2.2, the exceptions relate to use in those case studies of the dust models predicting the greatest influence of air concentration on indoor dust Pb among the set of dust models used. These were the general urban case study hybrid dust model and the site-specific regression dust model used in the remediation zone for the primary Pb smelter case study. In both cases, the ratios based on these models were higher than empirically derived ratios obtained from the literature. However, the literature indicated a potential for higher ratios for locations with either lower ambient air Pb levels or lower blood Pb levels, both conditions of which are present at the two case studies. Therefore, the higher modeled ratios obtained using the site-specific indoor dust models for the general urban and primary Pb smelter case studies do not necessarily point to potential high bias in predicting blood Pb levels for the study populations.

- *Comparison of modeled blood Pb levels to nationally representative data*: Our ability to compare modeled blood Pb levels to empirical data was handicapped by a lack of studies for populations of children directly comparable to those in the three case studies (see Section 3.5.2.3). Therefore, we focused most of our evaluation of modeled blood Pb levels on consideration of the national-level data obtained from NHANES IV. Note

that while higher-end population percentiles for the three case studies might not be reflected in a national-level dataset, blood Pb levels associated with background exposures for all three case studies are similar to blood Pb levels in the national dataset. Thus, we considered a comparison of modeled blood Pb levels for the three case studies against a national-scale dataset for central tendency population percentiles appropriate. In this comparison, we included the highest and lowest of the multiple sets of blood Pb estimates for the general urban case study generated using the multiple modeling approaches employed used for this case study. These two blood Pb distributions bracketed the NHANES IV based distribution, which adds confidence to blood Pb modeling conducted for this case study. Results for the primary Pb smelter (full study area) were similar to the NHANES IV distribution across population percentiles up to the 95th. This result is not surprising given the fact that exposure modeling for the primary Pb smelter did not fully consider variability in background Pb exposure (e.g., paint, diet, drinking water). Had background exposures been fully considered, the degree of divergence between the case study and national-level percentiles would likely have been much greater (with the primary Pb smelter distribution exceeding the NHANES IV distribution at higher population percentiles). Results for the subarea of the primary Pb smelter case study were consistently higher than the NHANES IV distribution across all population percentiles, as is expected given that the entire study area experiences elevated facility-related ambient air Pb levels. Blood Pb levels for all population percentiles for the secondary Pb smelter are lower than those for the national-scale dataset. As with the primary Pb smelter case study, this likely reflects the fact that variability in background Pb exposures was not fully considered in modeling this case study.

4.3.4 Uncertainty in Modeling Approaches – Multiple Sets of Results

For those more highly influential analytical steps for which it is not clear which model or input would generate "best estimate" results, we have implemented multiple modeling approaches (see Section 2.4.6.2). Because each of the case studies uses different modeling approaches for some of the analytical steps such as the indoor dust modeling step, and these approaches are associated with differing uncertainty, the identity and size of the areas of uncertainty associated with each case study differs. The specific modeling approaches for each case study and their elements are presented in Figure 2-3. For the general urban case study, two different dust models and two GSDs were used as compared to one model and GSD for these analytical steps in the two point source case studies. However, the same number of blood Pb metrics and IQ loss functions are used for all three case studies.

Consideration of the range of risk results generated using the multiple modeling approaches for each case study provides perspective on the combined effect of key sources of uncertainty on risk results. The median and 95th percentile estimates associated with the modeling approaches yielding the highest and lowest risk results for specific scenarios of the three case studies are presented in Table 4-4.

Table 4-4. Impact of multiple sources of uncertainty on risk results.

Case study	Modeling approach	IQ loss (population percentile)		Percent difference between highest and lowest approaches	
		Median	95th	Median	95th
General urban ***(mean current conditions)***	**Lowest risk:** Dust Model (Air-only Regression-based), GSD (1.7), blood Pb Metric (Concurrent), IQ Function (Two-piece Linear)	2	4	230%	120%
	Highest risk: Dust Model (Hybrid), GSD (2.0), blood Pb Metric (Lifetime), IQ Function (Log-linear with Linearization)	5	9		
Primary Pb smelter ***(current NAAQS)***	**Lowest risk:** blood Pb Metric (Concurrent), IQ Function (Two-piece Linear)	1	4	280%	80%
	Highest risk: blood Pb Metric (Lifetime), IQ Function (Log-linear with Linearization)	4	7		
Secondary Pb smelter ***(current conditions)***	**Lowest risk:** blood Pb Metric (Concurrent), IQ Function (low-linear with cutpoint)	0	2	>300%	120%
	Highest risk: blood Pb Metric (Lifetime), IQ Function (Log-linear with Linearization)	3	5		

The lack of a consistent pattern regarding the magnitude of differences between the modeling approaches generating the lowest and highest risk results either across case studies, or across population percentiles (Table 4-4), is due to different influences of various factors specific to the modeling approaches for each case study. For example, the finding with regard to the 95th percentile results that the greatest difference across approaches occurs for the secondary Pb smelter case study (>300%) reflects the fact that the lowest predicted median blood Pb level for this case study is below the cutpoint associated with one of the three concentration-response functions (thereby resulting in zero predicted IQ loss). The finding for the 95th percentile results that there is a greater difference for the general urban case study than the primary Pb smelter case study is because of the larger range of GSDs applied in this case study (2.1 and 1.6).

In summary, results presented in Table 4-4 suggest that these key sources of uncertainty, when acting in concert, can produce an impact on overall risk results on the order of a factor of 2 to 3.

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5 ADDITIONAL ANALYSES

This chapter presents the additional analyses performed for the Pb NAAQS risk assessment subsequent to the public meeting of the CASAC Pb Panel on August 28-29, 2007 (Henderson, 2007b). These analyses include reanalysis of the general urban and primary Pb smelter case studies and new analyses for three location-specific urban case studies located in Cleveland, Chicago and Los Angeles.[1] The methods used in conducting these additional analyses, as well as the resultant exposure and risk estimates, sensitivity analyses and uncertainty characterization are presented in this chapter.

5.1 DESIGN OF EXPOSURE AND RISK ASSESSMENTS

This section presents an overview of the design for the additional analyses. Additional details regarding the methods used in estimating exposure for both analyses can be found in Section 5.2. Details on the concentration-response functions for IQ loss and methods used to generate population risk estimates for these additional analyses can be found in Section 5.3.

5.1.1 Assessment Scenarios

The design of the assessment scenarios for the additional analyses is generally the same as that for the initial analyses (see Section 2.3 and subsections). The main difference is the addition of an alternate NAAQS scenario of 0.02 $\mu g/m^3$, as a maximum monthly average; otherwise, the air quality scenarios for the additional analyses are the same as those included in the initial analyses (Section 2.3.1). Specification of the air concentrations for the current conditions location-specific urban case studies is summarized in Section 5.1.3 and described more fully in Section 5.2.2.1 and Appendix O.

5.1.2 Analytical Approach

The general analytical approach employed for the initial analyses, described in Section 2.4, was also employed for the additional analyses, including the temporal aspects (Section 2.4.1), categorization of policy-relevant exposure pathways (Section 2.4.3), and the analytical steps (Section 2.4.4). Additionally, the spatial scale and resolution aspects of the reanalyses for the general urban and primary Pb smelter case studies were the same as for the initial analyses (see Section 2.4.2). The spatial scale and resolution aspects of the location-specific urban case studies are summarized in Section 5.1.3.

[1] Due to limitations with the design of the secondary Pb smelter case study and the associated high level of uncertainty noted in Sections 4.2 and 4.3.1,this case study was not part of the additional analyses described in this chapter.

A notable difference in the approach between the initial analyses and the additional analyses is that the multiple modeling approaches described in Section 2.4.6.2 and implemented for the initial analyses were narrowed down to a core modeling approach for the additional analyses. This was done in consideration of CASAC comments on the initial analyses (Henderson, 2007). Consistent with CASAC comments, the core modeling approach includes a single indoor dust model for the general and location-specific urban case studies, focuses on the concurrent blood Pb metric, and applies a single blood Pb GSD for the general urban case study. Additionally, CASAC recommended use of a linear concentration-response function derived from a subpopulation with peak blood Pb levels below 7.5 µg/dL, and indicated less favor for the two-piece linear function employed in the initial analyses of the full-scale assessment (Section 4.1.1.3). Accordingly, a different set of concentration-response functions was employed in the additional analyses (see Section 5.3.1.1). Figure 5-1 identifies the core modeling approach for each case study.

Figure 5-1. Core modeling approach for each case study.

Case Study	Elements of modeling approaches				Number of sets of results
	Indoor dust modeling	Blood Pb metric	GSD	Concentration-response function	
General Urban Case Study And Location-specific Urban Case Studies	1 model: hybrid mechanistic-empirical	1 metric: (a) concurrent	1 GSD, representing larger, regional scale	4 functions: (a) log-linear with low-exposure linearization, (b) log-linear with cutpoint, and (c) dual linear, stratified at 7.5 µg/dL (d) dual linear, stratified at 10 µg/dL	1 * 1 * 1 * 4 = 4
Primary Pb Smelter Case Study	1 model: statistical (regression) approach		1 GSD		1 * 1 * 1 * 4 =4

5.1.3 Location-Specific Urban Case Studies

The primary design difference between the location-specific case studies and the general urban case study, is that, unlike the spatially uniform general urban case study in which ambient air Pb levels are uniform and fixed at the air concentration considered (see Section 2.2.1) both the number of children and the ambient air Pb levels vary within the location-specific urban study areas. As a result, although the data on which these location-specific urban case studies are based are quite limited, exposure and risk estimates for these location-specific urban case studies provide a better representation of some urban areas in the U.S. than do those for the general urban case study.

Locations were selected for these study areas based on consideration of the number and spatial distribution of Pb-TSP monitors, the relative representation of source-oriented versus other monitors and the relative Pb concentrations reported for those monitors (Appendix A, Attachment A-2). Due to time constraints, the 2003-2005 dataset of Pb-TSP monitors analyzed in Appendix A (Attachment A-2) was the focus for this selection process and for subsequent consideration of air quality for the selected areas. Among all metropolitan areas represented in this dataset, preference was given to urban areas with relatively more monitors having a more uniform distribution and relatively higher Pb concentrations, and monitors that were not considered source oriented (see Appendix A, Section A.2.2.2.3). This led to selection of areas in Cleveland (6 monitors), Chicago (11 monitors) and Los Angeles (7 monitors).

The three study areas differ in several respects. First, they differ in Pb air levels, with the highest reported for Cleveland and the lowest for Los Angeles. Further, while monitors in Cleveland and Chicago included a subset classified as source oriented, this was not the case in Los Angeles. Last, the source oriented monitors were segregated from the other monitors in Chicago, while they were relatively more dispersed among the other monitors in Chicago.

The size of each study area is based on a polygon encompassing the area of each city in which the monitors occur. That polygon is defined initially using those monitors as apices and then a one mile-wide buffer is included beyond them in defining the final study area polygon (Appendix O, Exhibits O-2 through O-4). The U.S. Census blocks whose centroids fall within this polygon comprise the study area for each location-specific case study (Appendix O). Using centroids as the basis for determining whether a Census block is inside or outside of the study area provides a simple way for determining membership for those blocks that straddle the study area boundary. The target population for each study area is those children less than 7 years of age located within the study area blocks. The three study areas range in size from 67 to 1091 square km, and in target population size from 13,990 to 396,511 children (less than 7 years of age).

Exposure zones within each study area were defined based on assignment of census blocks to monitor sites, such that the number of exposure zones for a study area was equal to the number of monitors in a study area. The total child count for each exposure zone reflects the sum of children less than seven years old in the U.S. census blocks assigned to it, and the ambient air Pb concentrations for that zone reflect characteristics of data for that monitor. Consistent with the approach for all case studies, Pb concentrations in air (as well as other media) are homogenous within each exposure zone.

Within each study area, Census blocks were assigned to exposure zones differently depending on whether or not they were near a source oriented monitor. All blocks whose centroids fall within a mile of a source oriented TSP monitor were assigned to a source oriented

exposure zone defined by that monitor. All other blocks (i.e., those for which the centroid was farther than a mile from a source oriented monitor) were assigned to the nearest non source oriented monitor. This approach reflects the recognition that air Pb concentrations associated with sizeable stationary sources exhibit sharper gradients with distance than air Pb concentrations less influenced by such sources. Accordingly, source influenced Pb concentrations are constrained to smaller exposure zones than non source influenced concentrations.

Lead concentrations in indoor dust and outdoor soil/dust were estimated for the location-specific urban case studies using specific elements of the core modeling approach applied in the general urban case study (e.g., the indoor dust Pb model and Pb level in outdoor dust/soil).

Once the media concentrations are assigned to the exposure zones, the design of the exposure and risk analyses for each of these location-specific urban case studies is similar to that employed in the initial analyses to model the point source case studies (see Section 2.4.4.1 and 2.4.4.2), with a population-weighted simulation of exposure and risk conducted at the U.S. Census block level.

5.2 EXPOSURE ASSESSMENT

5.2.1 Methods for General Urban and Primary Pb Smelter Case Studies

For estimating ambient air concentration, inhalation exposure concentration and Pb concentration in outdoor soil/dust, the methods described in Section 3.1.1, 3.1.2 and 3.1.3 for the general urban and primary Pb smelter case studies also apply to the additional analyses for those case studies.

For the general urban case study, indoor dust Pb concentrations were estimated using the hybrid model. The primary rationale for deemphasizing the air only regression model and favoring the hybrid model in the additional analyses is that there is reduced confidence in using the air only regression model in the urban residential context, since this statistical model was developed with datasets that favored areas near Pb smelter sites (see Section 3.1.4.1 and Appendix G.1.1). By contrast, the hybrid indoor dust model was developed specifically for application in the urban residential context.

For the primary Pb smelter case study, indoor dust Pb concentrations are estimated as described in Section 3.1.4.2.

Blood Pb concentrations for the additional analyses for the general urban and primary Pb smelter case studies were estimated using methods previously described in Section 3.2, with two distinctions. First, for these additional analyses, the core modeling approach employed included only the concurrent blood Pb metric, consistent with CASAC recommendations (Henderson, 2007b). Secondly, for the general urban case study core approach we used the GSD of 2.1 which

represents the GSD for the concurrent blood Pb metric identified from NHANES IV (Section 3.2.3.1). This GSD is consistent with the CASAC recommendation to use a GSD more descriptive of the variability in blood Pb levels reported in NHANES-IV, as compared with the smaller GSDs observed for more localized populations near specific Pb emitting industries (e.g., see Section 3.2.3). The rationale for selecting the larger GSD, is that larger urban areas are likely to include more heterogeneous populations with regard to Pb exposure and blood Pb levels that more closely resemble the national NHANES population than a smaller more homogeneous population. For the primary Pb smelter case study, which includes a more localized study population, we have retained the smaller GSD of 1.7 used in the initial analyses (see Section 3.2.3.2).

The remaining elements of the core analysis, related to blood Pb modeling, including the blood Pb model, characterization of background Pb exposures, apportionment of exposure pathway contributions and probabilistic population modeling are those described in Section 3.2.

5.2.2 Methods for Location-specific Urban Case Studies

The methods used to estimate media and blood Pb concentrations in the location-specific urban case studies are described in the following subsections. The primary difference in methods between the location-specific case studies and the general urban case study is the inclusion of multiple exposure zones per study area across which numbers of children and ambient air Pb levels vary.

5.2.2.1 Ambient Air and Inhalation Exposure Concentrations

Air quality scenarios evaluated for these case studies included: (a) a current conditions scenario based on monitor-specific Pb-TSP statistics for the 2003-2005 dataset (Appendix A, Attachment A-2), (b) a current NAAQS scenario based on a roll-up of Pb-TSP values to the current NAAQS level, and (c) alternative NAAQS scenarios for levels falling below the current conditions levels for each case study.

For each scenario, the annual average ambient air Pb concentration estimate for each exposure zone that is the input to the blood Pb model for that exposure zone is based on the statistics for the monitor associated with that exposure zone, using the monitor-specific statistics presented in Appendix A, Attachment A-2. How this is derived varies with the air quality scenario as described below.

- *Current conditions scenario*: Each exposure zone is assigned the Pb-TSP values derived from the analysis of the 2003-2005 dataset in Appendix A (Attachment A-2) for the monitor assigned to that zone. Specifically, the annual average ambient Pb concentration estimate that is the input to the blood Pb model for a given exposure

zone is the three-year annual average concentration associated with the Pb-TSP monitor assigned to that zone.

- *Current NAAQS scenario:* A proportional roll-up procedure is used to derive air concentrations for all exposure zones such that the highest maximum quarterly average concentration among all of the study area monitors just meets the current NAAQS (maximum quarterly average of 1.5 μg/m3).

  — For the Cleveland and Los Angeles study areas, this procedure involves adjusting up the maximum quarterly average estimates for all monitors by the same factor as that used to set the highest monitor equal to the current NAAQS (max quarterly value of 1.5 $\mu g/m^3$). Then those adjusted maximum quarterly values are converted into annual averages using the ratio of annual average to maximum quarterly average specific to each monitor (as presented in Appendix A, Attachment A-2).

  — For Chicago, given the spacing of the monitors, we have defined two independent roll-up subareas, each represented by a separate set of monitors. For this scenario, the highest maximum quarterly average value in each of the two subareas is rolled up to equal the current NAAQS and the other monitors in each subarea are increased by the same proportion. Then the annual average values are calculated using monitor-specific ratios as for the other study areas.

- *Alternative NAAQS scenarios*: The procedure for rolling back ambient air Pb levels to reach alternative NAAQS is similar to the roll-up procedure used for the current NAAQS scenario, except the values are reduced rather than increased. Only those alternative NAAQS that are exceeded by the current conditions for a given case study were evaluated for that case study. Consequently, several of the higher alternative NAAQS were not evaluated at all for the location-specific urban case studies. As for the roll-up for the current NAAQS scenario, roll-backs for the Chicago case study are implemented separately for each of the two independent subareas, based on the highest Pb-TSP monitor in each subarea.

Inhalation exposure air concentrations were defined for both source oriented and non source oriented exposure zones using the same procedure employed for the general urban case study (see Section 3.1.2).

5.2.2.2 Other media Concentrations

Characterization of Pb exposure from incidental outdoor soil/dust ingestion used the values used for the general urban case study (Section 3.1.3.1), with the same values being used to characterize conditions in all exposure zones (source oriented or not) in each of the three case studies.

Indoor dust Pb concentrations are estimated for the location-specific urban case studies using the hybrid indoor dust model (see Section 3.1.4.1). Specifically, the hybrid indoor dust model is combined with the annual average ambient air Pb level for a particular exposure zone and air quality scenario to generate an indoor dust Pb level for that zone. Therefore, while background Pb exposure levels and outdoor soil/dust Pb levels are fixed across each of the urban

case studies, both ambient air Pb levels and indoor dust Pb levels are allowed to vary across source and non source exposure zones.

5.2.2.3 Blood Pb levels

Estimates of concurrent blood Pb are derived from application of the IEUBK model as described in Appendix H, with characterization of Pb exposure from background sources (i.e., drinking water and diet) relying on the same values as for all case studies (Appendix H). The apportionment of the blood Pb levels among the various exposure pathways is described in Section 3.2.2.

Population-level exposure modeling for the location-specific urban case studies utilizes essentially the same population-weighted sampling procedure as was used for the point source case studies (Section 3.2.2.2, including Figure 3-1). Use of this more sophisticated, population-weighted, multizone approach is required for the location-specific urban case studies since they utilize multiple exposure zones with differing demographic data and ambient air Pb levels, negating the use of the simpler, single zone approach used in the general urban case study. The remaining elements of the blood Pb modeling approach used for the location-specific urban case studies are identical to those used for the core approach for the general urban case study (Section 5.2.1). These include: (a) use of the concurrent blood Pb metric, (b) use of the IEUBK blood Pb model and (c) use of the GSD of 2.1 to represent interindividual variability in blood Pb levels.

5.2.3 Media concentrations

The following subsections summarize the media concentration estimate for all case studies and air quality scenarios. Estimates presented in these subsections are presented to three (for air) or zero (for dust and soil) decimal places, which results in various numbers of implied significant figures. This is not intended to convey greater precision for some estimates than others; it is simply an expedient and initial result of the software used for the calculation.

5.2.3.1 Location-specific Urban Case Studies

This section summarizes the media concentration estimates for the core modeling approach for each air quality scenario for the location-specific urban case studies. The complete set of media concentration estimates for these case studies is presented in Appendix O.

As discussed in Section 2.3.3, Pb concentration in outdoor soil/dust is not changed with the alternate air quality scenarios. Rather, outdoor soil/dust concentration is held constant at the current conditions or current NAAQS level.

Media concentrations generated for the three location-specific urban case studies are summarized in Tables 5-1 through 5-4, for annual ambient air Pb concentrations, inhalation exposure concentrations, outdoor soil/dust Pb and indoor dust Pb concentrations, respectively.

5.2.3.2 General Urban and Primary Pb Smelter Case Studies

This section summarizes the media concentration estimates for the core modeling approach for each air quality scenario for the general urban and primary Pb smelter case studies. The complete set of media concentration estimates for these case studies are presented in Appendix N.

For each air quality scenario for the primary Pb smelter case study, a range of percentile estimates are presented for each exposure medium. For the general urban case study, however, only a single value is presented for each exposure medium. This reflects the fact that, while the primary Pb smelter case study is modeled using spatial templates that include a large number of U.S. Census blocks and/or block groups (allowing percentile media concentrations to be identified), the general urban case study is modeled using a single study area with uniform media concentrations. Consequently, there is only one value presented for the general urban case study for each medium in each air quality scenario.

As mentioned above, it is assumed that outdoor soil/dust Pb concentrations are not effected by changes in ambient air Pb levels. In the case of an area such as the remediation zone of the primary Pb smelter case study, however, where soil dynamics have been changed by the substitution of contaminated soil with clean soil, or in areas where local sources may pose a more significant source to outdoor soil/dust than historic sources – and where there may be a currently increasing trend in surface Pb concentration - this may underestimate soil concentrations under some alternate NAAQS.

Media concentrations generated for the primary Pb smelter and general urban case studies using the core modeling approach are summarized in Tables 5-1 through 5-4, for annual ambient air Pb concentrations, inhalation exposure concentrations, outdoor soil/dust Pb and indoor dust Pb concentrations, respectively. As with similar presentations in Chapter 3, the percentiles presented here are from population weighted distributions of the media concentrations (see description in Appendices N, O and P).

Table 5-1. Estimated annual average ambient air concentrations.

Statistic*	Average Annual Air Pb Concentration (µg/m^3)						
			Alternative NAAQS				
	Current Conditions	Current NAAQS	1 0.2 µg/m^3, Max Quarterly	2 0.5 µg/m^3, Max Monthly	3 0.2 µg/m^3, Max Monthly	4 0.05 µg/m^3, Max Monthly	5 0.02 µg/m^3, Max Monthly
Location Specific Case Study(Chicago)							
Maximum	0.040	0.809	0.040	0.040	0.040	0.010	0.004
95th percentile	0.040	0.809	0.040	0.040	0.040	0.010	0.004
Median	0.027	0.428	0.027	0.027	0.021	0.005	0.002
5th percentile	0.014	0.286	0.014	0.014	0.014	0.004	0.001
Minimum	0.011	0.225	0.011	0.011	0.011	0.003	0.001
Location Specific Case Study(Cleveland)							
Maximum	0.121	0.506	0.067	0.108	0.043	0.011	0.004
95th percentile	0.121	0.506	0.067	0.108	0.043	0.011	0.004
Median	0.021	0.085	0.011	0.018	0.007	0.002	0.001
5th percentile	0.017	0.071	0.009	0.015	0.006	0.002	0.001
Minimum	0.017	0.071	0.009	0.015	0.006	0.002	0.001
Location Specific Case Study(Los Angeles)							
Maximum	0.022	0.360	0.022	0.022	0.022	0.009	0.003
95th percentile	0.022	0.360	0.022	0.022	0.022	0.009	0.003
Median	0.019	0.300	0.019	0.019	0.019	0.007	0.002
5th percentile	0.015	0.239	0.015	0.015	0.015	0.006	0.002
Minimum	0.006	0.091	0.006	0.006	0.006	0.002	0.001
General Urban Case Study							
NA – Single Study Area	High-end: 0.114 Mean: 0.056	0.600	0.080	0.125	0.050	0.013	0.005
Primary Pb SmelterCase Study- full study area							
Maximum	NA	0.740	0.161	0.326	0.130	0.033	0.013
95th percentile		0.153	0.033	0.067	0.027	0.007	0.003
Median		0.042	0.009	0.019	0.007	0.002	0.001
5th percentile		0.015	0.003	0.007	0.003	0.001	< 0.001
Minimum		0.006	0.001	0.003	0.001	< 0.001	< 0.001
Primary Pb SmelterCase Study- 1.5 km sub area							
Maximum	NA	0.740	0.161	0.326	0.130	0.033	0.013
95th percentile		0.675	0.147	0.297	0.119	0.030	0.012
Median		0.238	0.052	0.105	0.042	0.011	0.004
5th percentile		0.137	0.030	0.060	0.024	0.006	0.002
Minimum		0.098	0.021	0.043	0.017	0.004	0.002

*- The percentiles presented here are from population weighted distributions of the media concentrations.

Table 5-2. Estimated inhalation exposure concentrations.

Statistic*	Average Annual Inhalation Exposure Concentration of Pb (g/m^3)						
			Alternative NAAQS				
	Current Conditions	Current NAAQS	1 0.2 $\mu g/m^3$, Max Quarterly	2 0.5 $\mu g/m^3$, Max Monthly	3 0.2 $\mu g/m^3$, Max Monthly	4 0.05 $\mu g/m^3$, Max Monthly	5 0.02 $\mu g/m^3$, Max Monthly
Location Specific (Chicago)							
Maximum	0.017	0.347	0.017	0.017	0.017	0.004	0.002
95th percentile	0.017	0.347	0.017	0.017	0.017	0.004	0.002
Median	0.012	0.184	0.012	0.012	0.009	0.002	0.001
5th percentile	0.006	0.123	0.006	0.006	0.006	0.002	0.001
Minimum	0.005	0.097	0.005	0.005	0.005	0.001	< 0.001
Location Specific (Cleveland)							
Maximum	0.052	0.217	0.029	0.047	0.019	0.005	0.002
95th percentile	0.052	0.217	0.029	0.047	0.019	0.005	0.002
Median	0.009	0.037	0.005	0.008	0.003	0.001	< 0.001
5th percentile	0.007	0.030	0.004	0.006	0.003	0.001	< 0.001
Minimum	0.007	0.030	0.004	0.006	0.003	0.001	< 0.001
Location Specific (Los Angeles)							
Maximum	0.010	0.154	0.010	0.010	0.010	0.004	0.001
95th percentile	0.010	0.154	0.010	0.010	0.010	0.004	0.001
Median	0.008	0.129	0.008	0.008	0.008	0.003	0.001
5th percentile	0.006	0.103	0.006	0.006	0.006	0.002	0.001
Minimum	0.002	0.039	0.002	0.002	0.002	0.001	< 0.001
General Urban							
NA – Single Study Area	High-end: 0.049 Mean: 0.024	0.258	0.034	0.054	0.021	0.005	0.002
Primary Pb Smelter- full study area							
Maximum	NA	0.310	0.067	0.136	0.055	0.014	0.005
95th percentile		0.064	0.014	0.028	0.011	0.003	0.001
Median		0.017	0.004	0.007	0.003	0.001	< 0.001
5th percentile		0.006	0.001	0.003	0.001	< 0.001	< 0.001
Minimum		0.002	< 0.001	0.001	< 0.001	< 0.001	< 0.001
Primary Pb Smelter- 1.5 km subarea							
Maximum	NA	0.310	0.067	0.136	0.055	0.014	0.005
95th percentile		0.282	0.061	0.124	0.050	0.012	0.005
Median		0.100	0.022	0.044	0.018	0.004	0.002
5th percentile		0.057	0.012	0.025	0.010	0.003	0.001
Minimum		0.041	0.009	0.018	0.007	0.002	0.001

*- The percentiles presented here are from population weighted distributions of the media concentrations.

Table 5-3. Estimated outdoor soil/dust concentrations.

Statistic*	Projected Average Outdoor Soil/Dust Pb Concentration (mg/kg) (Same for all air quality scenarios)
Location Specific (Chicago)	
NA - Full Study Area	198
Location Specific (Cleveland)	
NA - Full Study Area	198
Location Specific (Los Angeles)	
NA - Full Study Area	198
General Urban	
NA – Single Study Area	198
Primary Pb Smelter- full study area	
Maximum	958
95th percentile	245
Median	85
5th percentile	30
Minimum	17
Primary Pb Smelter- 1.5km sub area	
Maximum	294
95th percentile	223
Median	150
5th percentile	42
Minimum	42

*-The percentiles presented here are from population weighted distributions of the media concentrations.

Table 5-4. Estimated indoor dust concentrations.

Statistic*	Projected Average Indoor Dust Pb Concentration (mg/kg or ppm)						
			Alternative NAAQS				
	Current Conditions	Current NAAQS	1 0.2 µg/m^3, Max Quarterly	2 0.5 µg/m^3, Max Monthly	3 0.2 µg/m^3, Max Monthly	4 0.05 µg/m^3, Max Monthly	5 0.02 µg/m^3, Max Monthly
Location Specific (Chicago)							
Maximum	128	491	128	128	128	84	71
95th percentile	128	491	128	128	128	84	71
Median	111	363	111	111	103	74	67
5th percentile	91	300	91	91	91	70	65
Minimum	86	269	86	86	86	68	64
Location Specific (Cleveland)							
Maximum	203	392	158	193	132	85	72
95th percentile	203	392	158	193	132	85	72
Median	101	174	86	98	78	66	63
5th percentile	96	161	82	93	75	65	63
Minimum	96	161	82	93	75	65	63
Location Specific (Los Angeles)							
Maximum	104	334	104	104	104	81	68
95th percentile	104	334	104	104	104	81	68
Median	99	307	99	99	99	78	67
5th percentile	92	276	92	92	92	75	65
Minimum	75	179	75	75	75	67	63
General Urban							
NA – Single Study Area	High-end: 198 Mean: 146	426	169	206	140	88	73
Primary Pb Smelter- full study area							
Maximum	NA	1944	648	1077	557	383	381
95th percentile		219	152	172	149	138	120
Median		84	68	73	67	63	62
5th percentile		53	45	47	44	43	42
Minimum		41	38	39	38	38	38
Primary Pb Smelter- 1.5km subarea							
Maximum	NA	1944	648	1077	557	205	106
95th percentile		1819	606	1008	521	192	99
Median		860	287	477	246	91	60
5th percentile		578	193	320	166	61	60
Minimum		453	151	251	130	60	60

*- The percentiles presented here are from population weighted distributions of the media concentrations.

5.2.4 Blood Pb levels

Estimates of concurrent blood Pb level derived from outputs of the IEUBK model (Section 3.2) have been developed for each air quality scenario in each case study (see Appendices N and O). Table 5-5 presents median concurrent blood Pb estimates for the location-specific urban case studies (Chicago, Cleveland and Los Angeles) and the core analysis of the primary Pb smelter (and sub-area) and the general urban case study. Table 5-6 presents 95th percentile concurrent blood Pb estimates for the same set of case studies. As with blood Pb level results presented in Section 3.4, these tables present the median and 95th percentile total Pb levels (i.e., simulated children for each case study area ranked based on total Pb level in order to identify population percentiles), along with the blood Pb contributions to those total blood estimates from policy-relevant pathways.[2] While some of the blood Pb levels presented in Section 3.4 (those for the primary Pb smelter) included a range reflecting multiple modeling options related to blood Pb estimation, the results presented in Tables 5-5 and 5-6 are single values, reflecting the fact that the core analysis, while including multiple concentration-response functions for IQ loss, did not include multiple modeling options related to blood Pb estimation (see Section 5.1.2).

It is noted that given the various limitations of our modeling tools (Sections 2.4.3 and 3.2.2), blood Pb levels associated with air-related exposure pathways and current levels of Pb emitted to the air (including via resuspension) are likely to fall between the estimates for "recent air" and those for "recent" plus "past air". Additionally, with regard to the location-specific urban case studies "recent air" and "recent" plus "past air" categories, an artifact of the hybrid dust Pb model tends to mask trends in the two components of dust Pb (recent air and other), which contribute to "recent air" and "past air" estimates, respectively (see Section 4.3.1 discussion of this uncertainty). For the primary Pb smelter case study, uncertainty in parsing out the "recent air" and "other" components of indoor dust (specifically for the site-specific regression model used in the remediation zone) have led us to conclude that only "recent plus past air" exposures should be presented and "recent air" results should not be separately presented for the primary Pb smelter, as is done for the other case studies (see Section 3.1.4.2).

[2] Seemingly inconsistent trends across air quality scenarios seen in these two categories (recent air and recent plus past air) may be a result of presenting results for these policy-relevant categories based on population-weighted ranking of total blood Pb.

Table 5-5. Summary of blood Pb level estimates for median total blood Pb.

Air Quality Scenario (and case study)	Concurrent Blood Pb Level[a]		
	Recent[b]	Recent plus Past[b]	Total Pb Exposure
Location-specific (Chicago)			
Current NAAQS (1.5 µg/m^3, max quarterly)	1.9	2.5	3.0
Current conditions (0.14 µg/m^3 max quarterly; 0.31 µg/m^3 max monthly)	0.4	1.2	1.8
Alternative NAAQS (0.2 µg/m^3, max monthly)	0.3	1.2	1.8
Alternative NAAQS (0.05 µg/m^3, max monthly)	0.1	1.1	1.6
Alternative NAAQS (0.02 µg/m^3, max monthly)	<0.1	1.0	1.6
Location-specific (Cleveland)			
Current NAAQS (1.5 µg/m^3, max quarterly)	1.2	1.8	2.1
Current conditions (0.36 µg/m^3 max quarterly; 0.56 µg/m^3 max monthly)	0.3	1.2	1.8
Alternative NAAQS (0.5 µg/m^3, max monthly)	0.7	1.3	1.8
Alternative NAAQS (0.2 µg/m^3, max quarterly)	0.2	1.1	1.7
Alternative NAAQS (0.2 µg/m^3, max monthly)	0.1	1.1	1.7
Alternative NAAQS (0.05 µg/m^3, max monthly)	<0.1	1.0	1.6
Alternative NAAQS (0.02 µg/m^3, max monthly)	<0.1	1.0	1.6
Location-specific (Los Angeles)			
Current NAAQS (1.5 µg/m^3, max quarterly)	1.3	2.1	2.6
Current conditions (0.09 µg/m^3 max quarterly; 0.17 µg/m^3 max monthly)	0.3	1.2	1.7
Alternative NAAQS (0.05 µg/m^3, max monthly)	0.2	1.1	1.6
Alternative NAAQS (0.02 µg/m^3, max monthly)	0.1	1.0	1.6
General urban			
Current NAAQS (1.5 µg/m^3, max quarterly)	1.9	2.6	3.1
Alternative NAAQS (0.5 µg/m^3, max monthly)	0.9	1.6	2.2
Current conditions –high-end (0.87 µg/m^3, max quarterly)	0.8	1.6	2.1
Alternative NAAQS (0.2 µg/m^3, max quarterly)	0.7	1.5	2.0
Current conditions - mean (0.14 µg/m^3, max quarterly)	0.6	1.4	1.9
Alternative NAAQS (0.2 µg/m^3, max monthly)	0.5	1.4	1.9
Alternative NAAQS (0.05 µg/m^3, max monthly)	0.2	1.1	1.7
Alternative NAAQS (0.02 µg/m^3, max monthly)	0.1	1.1	1.6
Primary Pb smelter - *full study area*			
Current NAAQS (1.5 µg/m^3, max quarterly)	NA[c]	0.8	1.5
Alternative NAAQS (0.5 µg/m^3, max monthly)		1.1	1.4
Alternative NAAQS (0.2 µg/m^3, max quarterly)		0.7	1.4
Alternative NAAQS (0.2 µg/m^3, max monthly)		0.5	1.4
Alternative NAAQS (0.05 µg/m^3, max monthly)		0.7	1.4
Alternative NAAQS (0.02 µg/m^3, max monthly)		0.9	1.4
Primary Pb smelter - *1.5km study area*			
Current NAAQS (1.5 µg/m^3, max quarterly)	NA[c]	4.0	4.6
Alternative NAAQS (0.5 µg/m^3, max monthly)		2.6	3.2
Alternative NAAQS (0.2 µg/m^3, max quarterly)		1.8	2.5
Alternative NAAQS (0.2 µg/m^3, max monthly)		1.8	2.3
Alternative NAAQS (0.05 µg/m^3, max monthly)		1.1	1.7
Alternative NAAQS (0.02 µg/m^3, max monthly)		1.1	1.6

a - Estimates are rounded to one decimal place.
b -The term "past air" includes contributions from the outdoor soil/dust contribution to indoor dust, historical air contribution to indoor dust, and outdoor soil/dust pathways, while "recent air" refers to contributions from inhalation of ambient air Pb or ingestion of indoor dust Pb predicted to be associated with outdoor ambient air Pb levels, with outdoor ambient air also potentially including resuspended, previously deposited Pb (see Section 2.4.3).
c – "Recent air" estimates were not developed for the primary Pb smelter case study (see Section 3.1.4.2).

Table 5-6. Summary of blood Pb level estimates for high-end total blood Pb.

Air Quality Scenario (and case study)	Concurrent Blood Pb Level[a]		
	Recent[b]	Recent plus Past[b]	Total Pb Exposure
Location-specific (Chicago)			
Current NAAQS (1.5 µg/m^3, max quarterly)	6.5	8.7	10.2
Current conditions (0.14 µg/m^3 max quarterly; 0.31 µg/m^3 max monthly)	1.1	4.1	6.0
Alternative NAAQS (0.2 µg/m^3, max monthly)	1.5	4.2	6.0
Alternative NAAQS (0.05 µg/m^3, max monthly)	0.3	3.6	5.5
Alternative NAAQS (0.02 µg/m^3, max monthly)	0.2	3.6	5.4
Location-specific (Cleveland)			
Current NAAQS (1.5 µg/m^3, max quarterly)	2.3	5.4	7.4
Current conditions (0.36 µg/m^3 max quarterly; 0.56 µg/m^3 max monthly)	0.9	4.1	6.1
Alternative NAAQS (0.5 µg/m^3, max monthly)	1.5	4.2	6.0
Alternative NAAQS (0.2 µg/m^3, max quarterly)	0.7	3.9	5.8
Alternative NAAQS (0.2 µg/m^3, max monthly)	0.4	3.8	5.7
Alternative NAAQS (0.05 µg/m^3, max monthly)	0.1	3.5	5.4
Alternative NAAQS (0.02 µg/m^3, max monthly)	0.1	3.5	5.3
Location-specific (Los Angeles)			
Current NAAQS (1.5 µg/m^3, max quarterly)	4.6	7.1	8.9
Current conditions (0.09 µg/m^3 max quarterly; 0.17 µg/m^3 max monthly)	1.1	4.0	5.9
Alternative NAAQS (0.05 µg/m^3, max monthly)	0.5	3.7	5.5
Alternative NAAQS (0.02 µg/m^3, max monthly)	0.2	3.5	5.4
General urban			
Current NAAQS (1.5 µg/m^3, max quarterly)	6.4	8.8	10.6
Alternative NAAQS (0.5 µg/m^3, max monthly)	2.9	5.6	7.4
Current conditions –high-end (0.87 µg/m^3, max quarterly)	2.7	5.4	7.2
Alternative NAAQS (0.2 µg/m^3, max quarterly)	2.3	5.0	6.8
Current conditions - mean (0.14 µg/m^3, max quarterly)	1.9	4.7	6.5
Alternative NAAQS (0.2 µg/m^3, max monthly)	1.7	4.6	6.4
Alternative NAAQS (0.05 µg/m^3, max monthly)	0.7	3.9	5.7
Alternative NAAQS (0.02 µg/m^3, max monthly)	0.4	3.7	5.5
Primary Pb smelter - *full study area*			
Current NAAQS (1.5 µg/m^3, max quarterly)	NA[c]	2.8	4.6
Alternative NAAQS (0.5 µg/m^3, max monthly)		3.1	4.2
Alternative NAAQS (0.2 µg/m^3, max quarterly)		2.4	4.0
Alternative NAAQS (0.2 µg/m^3, max monthly)		2.5	4.0
Alternative NAAQS (0.05 µg/m^3, max monthly)		1.9	3.8
Alternative NAAQS (0.02 µg/m^3, max monthly)		3.2	3.8
Primary Pb smelter - *1.5km study area*			
Current NAAQS (1.5 µg/m^3, max quarterly)	NA[c]	10.3	12.3
Alternative NAAQS (0.5 µg/m^3, max monthly)		7.6	8.5
Alternative NAAQS (0.2 µg/m^3, max quarterly)		5.9	6.6
Alternative NAAQS (0.2 µg/m^3, max monthly)		4.9	6.1
Alternative NAAQS (0.05 µg/m^3, max monthly)		3.5	4.5
Alternative NAAQS (0.02 µg/m^3, max monthly)		3.0	4.2

a - Estimates are rounded to one decimal place.
b -The term "past air" includes contributions from the outdoor soil/dust contribution to indoor dust, historical air contribution to indoor dust, and outdoor soil/dust pathways, while "recent air" refers to contributions from inhalation of ambient air Pb or ingestion of indoor dust Pb predicted to be associated with outdoor ambient air Pb levels, with outdoor ambient air also potentially including resuspended, previously deposited Pb (see Section 2.4.3).
c – "Recent air" estimates were not developed for the primary Pb smelter case study (see Section 3.1.4.2).

5.2.5 Uncertainty Characterization

As was done for the initial analyses (see Section 3.5), characterization of uncertainty related to the exposure assessment for the additional analyses focused on performance evaluation of both modeled media concentrations and modeled blood Pb levels. The sensitivity analysis described in Section 4.3.2 included several modeling elements related to exposure assessment which have bearing on blood Pb levels generated for the analyses described in this chapter for the primary Pb smelter and general urban case study and the location-specific urban case studies (e.g., interindividual variability GSD, blood lead model selection). Additional key sources of uncertainty related to exposure assessment specifically for the location-specific urban case studies (e.g., relating child study populations within the study areas to Pb-TSP monitor values in order to assign ambient air Pb levels) have not been investigated quantitatively, but are addressed as part of the qualitative discussion of uncertainty for the overall risk assessment (Section 5.3.3.1).

Performance evaluation completed for both the modeled media concentrations and modeled blood Pb levels is discussed below.

5.2.5.1 Performance Evaluation of Modeled Media Concentrations

As discussed in Section 3.5.1 for the previous analyses, performance evaluation for modeled media concentrations focuses on those estimates that were generated primarily through modeling. Media concentrations characterized using empirical data, such as air concentrations estimated using monitor measurements, are not subjected to performance evaluation. The following observations can be made regarding performance evaluation of model-estimate media concentrations for the analysis using the core modeling approach of the primary Pb smelter, general urban and location-specific urban case studies.

- All media concentrations for the primary Pb smelter and general urban case study are presented in Chapter 3, and the reader is referred back to Section 3.5.1 for performance evaluation of those estimates.
- Ambient air Pb levels (and, indirectly, inhalation exposure concentrations) for the location-specific urban case studies are based on Pb-TSP monitoring data and therefore will not be addressed quantitatively in this performance evaluation. Note, however, that the method for assigning Pb-TSP monitors to subsets of the child study populations in each study area is a key step in this analysis and one which is subject to uncertainty. However, characterizing ambient air Pb levels for the study populations at the location-specific urban case studies is addressed as part of the qualitative discussion of key sources of uncertainty (see Section 5.3.3.1).
- Outdoor soil/dust Pb levels for the location-specific urban case studies utilize the same uniform value as was used for the general urban case study (see Section 3.5.1.2).

- Indoor dust Pb levels for the location-specific urban case studies are generally similar to those generated for the general urban case study. This is expected since the location-specific urban case studies utilize the same hybrid indoor dust Pb model as was used for the general urban case study. Because of the similarity of the indoor dust Pb levels generated for the location-specific urban case studies and the general urban case study, the reader is referred to the previous performance evaluation discussion for indoor dust Pb addressing the general urban case study (see Section 3.5.1.3).

5.2.5.2 Performance Evaluation of Modeled Blood Pb Levels

In this section, we discuss air-to-blood ratios derived by comparing changes (deltas) in median total blood Pb level (concurrent) to associated changes in annual average air Pb levels as you step to the next lowest air quality scenario (Table 5-7). This analysis is only available for the general urban and primary Pb smelter case studies (including both the full study area and 1.5 km subarea for the latter). Note that these air-to-blood ratios are different than those presented in Section 3.5.2.2 for the general urban, primary Pb smelter and secondary Pb smelter case studies. The ratios derived in Section 3.5.2.2 are based on comparing annual average ambient air Pb levels (used in modeling these case studies) to the recent air portions of blood Pb levels. Therefore, these ratios reflect a generalization of the relationship between air Pb and blood Pb across the entire exposure spectrum, reflecting that relationship as an average of the two absolute values. By contrast, the air-to-blood ratios presented in this section look at the incremental change in blood Pb as ambient air Pb changes incrementally with lower alternate NAAQS levels (Table 5-7).

The model-derived air-to-blood ratios presented in Table 5-7 can be compared to ratios identified from the literature (see last column of Table 3-24). In fact, conceptually, it may be more appropriate to compare the air-to-blood ratios presented in Table 5-7 (than those in Section 3.5.2.2) to ratios obtained from the review by Brunekreef (1984), since the individual ratios summarized in that analysis included a large number of studies that compared blood Pb levels of populations living in different locations with that associated different ambient air Pb levels to derive air-to-blood ratios. Conceptually, this is fairly similar to the approach used in deriving the air-to-blood ratios presented in this section. Similarly, Hilts (2003) considered exposure levels (ambient air Pb) and associated blood Pb levels for the same population at two different points in time, straddling a change in operation of a nearby smelter. This is conceptually similar to the way the air-to-blood ratios are calculated here. Comparison of the air-to-blood ratios presented in Table 5-7 with the ratios identified in Brunekreef (1984) and calculated based on data presented in Hilts (2003), as summarized in Table 3-24, results in the following observations:

- Air-to-blood ratios for the general urban case study for air quality scenarios ranging from current NAAQS down to the alternative NAAQS of 0.2 $\mu g/m^3$, maximum monthly average, are similar to ratios reported in the literature (i.e., between 1:3 and

1:5). Ratios for the two lowest alternative NAAQS are larger, ranging from 1:6 to 1:9. However, even these larger ratios are within the range of some of the studies reported in Brunekreef (1984) and within the range of ratios calculated based on data presented in Hilts (2003). As noted in Section 3.5.2.2, there are a number of factors which may produce higher air-to-blood ratios, which may pertain to urban locations, such as lower blood Pb levels relative to those reflected in the meta analysis by Brunekreef (1984).

- Air-to-blood ratios for the primary Pb smelter case study (full study area) are similar in magnitude to the general urban case study and the points made in the previous bullet apply to this case study.
- Air-to-blood ratios for the 1.5 km subarea of the primary Pb smelter are significantly higher than ratios for the other two study areas noted above, with values ranging from 1:10 to 1:19. These ratios, and particularly the higher values associated with the lower NAAQS levels, are significantly higher than values reported in Brunekreef (1984) and those based on data in Hilts (2003). Many of the studies cited in Brunekreef (1984) use differences in average ambient air Pb levels and blood Pb levels between clusters of individuals living in different areas to derive their ratios. It is possible that the indoor dust Pb model developed for the remediation zone of the primary Pb smelter case study captures a relatively stronger relationship between ambient air Pb and indoor dust Pb (and hence blood Pb) which exists in the vicinity of industrial facilities. It is also possible that reservoirs of Pb associated with past airborne Pb are still within these houses near the facility and correlated with current ambient air Pb levels are reflected in the site-specific regression model established between ambient air Pb levels and indoor dust Pb. Thus, a portion of indoor dust Pb that results from contributions from indoor reservoirs may be being attributed through our application of the model to current ambient air Pb.

The air-to-blood ratios presented earlier in Section 3.5.2.2 for the general urban case study and primary Pb smelter case study are also pertinent to the core modeling approach results for these two case studies presented in this chapter and the reader is referenced to that earlier section for discussion of these alternative ratios and implications in the context of performance evaluation for blood Pb modeling. Other elements of performance evaluation for blood Pb modeling presented earlier in Section 3.5.2 (e.g., evaluation of candidate blood Pb models - see Section 3.5.2.1) also pertain to the analyses described in this section.

Table 5-7. Air-to-blood ratios derived by comparing air quality scenario air and blood Pb estimates.

Air Scenario	Median Total Blood Pb (µg/dL)	Annual Average Ambient Air Concentration ($\mu g/m^3$)[a]	Ratio[b]
General urban case study			
Current NAAQS (1.5 µg/m3, max quarterly average)	3.12	0.600	
Alternative NAAQS 2 (0.5 µg/m3, max monthly average)	2.16	0.125	1:2
Current Conditions (95th percentile)	2.13	0.114	1:3
Alternative NAAQS 1 (0.2 µg/m3, max quarterly average)	2.01	0.080	1:4
Current Conditions (mean)	1.91	0.056	1:4
Alternative NAAQS 3 (0.2 µg/m3, max monthly average)	1.88	0.050	1:5
Alternative NAAQS 4 (0.05 µg/m3, max monthly average)	1.67	0.013	1:6
Alternative NAAQS 5 (0.02 µg/m3, max monthly average)	1.61	0.005	1:9
Primary Pb smelter case study (full study area)			
Current NAAQS (1.5 µg/m3, max quarterly average)	1.50	0.042	
Alternative NAAQS 2 (0.5 µg/m3, max monthly average)	1.44	0.019	1:3
Alternative NAAQS 1 (0.2 µg/m3, max quarterly average)	1.40	0.009	1:3
Alternative NAAQS 3 (0.2 µg/m3, max monthly average)	1.40	0.007	1:4
Alternative NAAQS 4 (0.05 µg/m3, max monthly average)	1.38	0.002	1:4
Alternative NAAQS 5 (0.02 µg/m3, max monthly average)	1.37	0.001	1:7
Primary Pb smelter case study (1.5 km subarea)			
Current NAAQS (1.5 µg/m3, max quarterly average)	4.58	0.238	
Alternative NAAQS 2 (0.5 µg/m3, max monthly average)	3.20	0.105	1:10
Alternative NAAQS 1 (0.2 µg/m3, max quarterly average)	2.50	0.052	1:13
Alternative NAAQS 3 (0.2 µg/m3, max monthly average)	2.35	0.042	1:15
Alternative NAAQS 4 (0.05 µg/m3, max monthly average)	1.75	0.011	1:19
Alternative NAAQS 5 (0.02 µg/m3, max monthly average)	1.63	0.004	1:19

[a] For the primary Pb smelter case study entries, these are the median air concentrations in a population weighted distribution of air concentration estimates for each scenario.

[b] These are air-to-blood ratios derived by comparing changes (deltas) in median total blood Pb levels (concurrent) to associated changes in annual average air Pb levels as you step to the next lowest air quality scenario. Accordingly, a ratio is not presented adjacent to the current NAAQS air quality scenario (for any of the case studies). In this case, the first ratio presented for any of the case studies is generated by comparing median blood Pb levels at the current NAAQS level to the median blood Pb level at the highest of the alternative NAAQS levels (the 0.05 ug/m^3 maximum monthly value).

5.3 RISK ASSESSMENT

This chapter describes the approach used to characterize risk using the core modeling approach, including discussion of the methodology (Section 5.3.1), presentation of risk estimates (Section 5.3.2), and uncertainty characterization (Section 5.3.3).

5.3.1 Methods for Deriving Risk Estimates

Risk characterization for this assessment focuses on IQ loss in children. IQ loss is derived using a set of concentration-response functions developed based on results from a pooled analysis of epidemiology studies (Lanphear et al., 2005). These concentration-response functions are combined with the population-level blood Pb distributions to produce distributions of IQ loss estimates for each case study population. IQ loss is also apportioned among different exposure pathways using the pathway apportionment information generated as part of the exposure analysis.

Two key elements of the risk methodology are described in greater detail below: (a) the concentration-response functions used in the analysis (Section 5.3.1.1) and (b) the stepwise analytical procedure used to generate the IQ loss (risk) distributions (Section 5.3.1.2).

5.3.1.1 Concentration-response Functions

For the risk analyses described in this chapter, performed subsequent to the August 2007 public meeting of the CASAC Pb Panel, four types of concentration-response functions for IQ loss were used. Some of the functions in this set differ from those used in the initial analyses (Section 4.1.1), reflecting CASAC recommendations to use a two-piece or dual linear function that recognizes a change in slope (to a notably higher value) at blood Pb levels of 7.5 μg/dL and give less prominence to the two-piece linear function with hinge at 10.82 μg/dL derived for the initial analyses of the full-scale assessment (Henderson, 2007b). The set of four concentration-response functions used in the analyses presented in this chapter include the following:

- *The log-linear function with a cutpoint at 1.0* μg/*dL* (for the concurrent blood Pb metric): This function is described in Section 4.1.1.1.
- *The log-linear function with low-exposure linearization* (for the concurrent blood Pb metric): This function is described in Section 4.1.1.2.
- *Dual linear function, stratified at 10* μg/*dL peak*: This represents two linear functions reported in Lanphear et al., 2005. To derive these, the full study population was stratified into two groups - children with peak blood Pb levels above and below 10 μg/dL - and separate linear functions were fit to the IQ and concurrent blood Pb values for these two groups (Lanphear et al., 2005). In order to utilize this model, we considered the relationship between peak and concurrent blood Pb levels in the pooled dataset, as well as the relationship in the cohort comprising the bulk of the low blood Pb subsets. For the full pooled dataset, the average peak blood Pb is 18.0 μg/dL, while

the average concurrent blood Pb is 9.7 µg/dL, approximately a factor of two difference (Lanphear et al., 2005). For the Rochester cohort, which comprised the majority of the subset of children with peak blood Pb values below 10 µg/dL, the average peak blood Pb is 9.0 µg/dL, while the average concurrent blood Pb is 4.0 µg/dL, approximately a factor of two difference (Lanphear et al., 2005). Accordingly, in specifying the concurrent blood Pb level at which this function would change slope, we assumed that the peak blood Pb of 10 µg/dL corresponded to a concurrent blood Pb of 5 µg/dL. The form of the function as applied in this risk assessment is:

- For concurrent blood Pb level ≤ 5 µg/dL:

  IQ loss = beta 1 * concurrent blood Pb

- For concurrent blood Pb level > 5 µg/dL:

  IQ loss = beta 2 * concurrent blood Pb

  Where:

  beta 1 = -0.80

  beta 2 = -0.13

- *Dual Linear function, stratified at 7.5* µg/*dL peak*: This represents two linear functions, and like the previous ones, these were separately derived from subgroups of the full study population (Lanphear et al., 2005). In this case, the population was stratified into groups of children with peak blood Pb levels above and below 7.5 µg/dL. For the same considerations of the relationship between peak and concurrent blood Pb levels stated for previously described function in specifying the concurrent blood Pb level at which this function would change slope, we assumed that the peak blood Pb of 7.5 µg/dL corresponded to a concurrent blood Pb of 3.75 µg/dL. The form of this function as applied in this risk assessment is:

  - For concurrent blood Pb level ≤ 3.75 µg/dL:

    IQ loss = beta 1 * concurrent blood Pb

  - For concurrent blood Pb level > 3.75 µg/dL:

    IQ loss = beta 2 * concurrent blood Pb

    Where:

    beta 1 = -2.94

    beta 2 = -0.16

The four functions are presented in Figure 5-2 and compared in Table 5-8 with regard to total IQ loss and incremental IQ loss (IQ loss per µg/dL) across a range of concurrent blood Pb levels. A number of observations can be made by considering the plots of the four functions together with the results presented in Table 5-8. This discussion presents IQ loss estimates rounded to the nearest integer, although they are presented with greater precision in Table 5-8. At blood Pb levels less than or equal to about 2 µg/dL, with 2 µg/dL being near the average blood Pb level for young children in the U.S., based on NHANES-IV data and near the average

blood Pb estimated in the risk assessment for the three location-specific urban case studies, the *log-linear with cutpoint* model and the *dual linear - stratified at 10 µg/dL peak* generate similar IQ loss estimates. Both models predict a total of 2 points IQ loss associated with a blood Pb level of 2 µg/dL. By contrast, the remaining two models (*log-linear with low-exposure linearization* and *dual linear - stratified at 7.5 µg/dL peak*) generate substantially larger IQ loss estimates, ranging from 5 to 6 points IQ loss.

At blood Pb levels above 2 µg/dL, the pattern of predicted IQ loss shifts substantially across the four models, with the *dual linear - stratified at 7.5 µg/dL peak* maintaining a constant slope (up to 3.75 µg/dL) which is significantly higher than the other models, all of which either have slopes which decrease (for the log-linear models) or have a lower slope to begin with (the *dual linear - stratified at 10 µg/dL peak*). At higher exposure levels ranging from 2 and 10 µg/dL, the *dual linear - stratified at 10 µg/dL peak* and the *log-linear with cutpoint* perform similarly, with the latter predicting somewhat higher total IQ loss at 10 µg/dL (6 points, versus 5 for the dual linear-stratified at 10 µg/dL). Within this higher exposure range, the *log-linear with linearization* model generates IQ loss estimates which are higher than the two models just discussed, but lower than the *dual linear - stratified at 7.5 peak*. Specifically, at 10 µg/dL, the *log-linear with linearization* model predicts 9 points IQ loss, while the *dual linear - stratified at 7.5 peak* model predicts 12 points IQ loss. As discussed in Section 2.1.5, the dataset from which the lower blood Pb portion of the *dual linear – stratified at 7.5 peak* function is derived is much smaller than those from which the other functions are derived and includes variable representation of six of the seven cohorts in the pooled analysis with a dominance (~65% of total) of one cohort.

Both of the dual linear models with stratification have lower slopes above 5 µg/dL than either of the log-linear models. This means that reductions in blood Pb levels for modeled children with relatively high exposures (> 5 µg/dL total blood Pb) will result in relatively lower IQ benefits using the two dual-linear models, compared with the two log-linear models (even if, in the case of the dual linear - stratified at 7.5 µg/dL model, the overall IQ loss is higher for that simulated child than with either of the log-linear models).

Figure 5-2. Comparison of four concentration-response functions for concurrent blood Pb levels < 10 µg/dL.

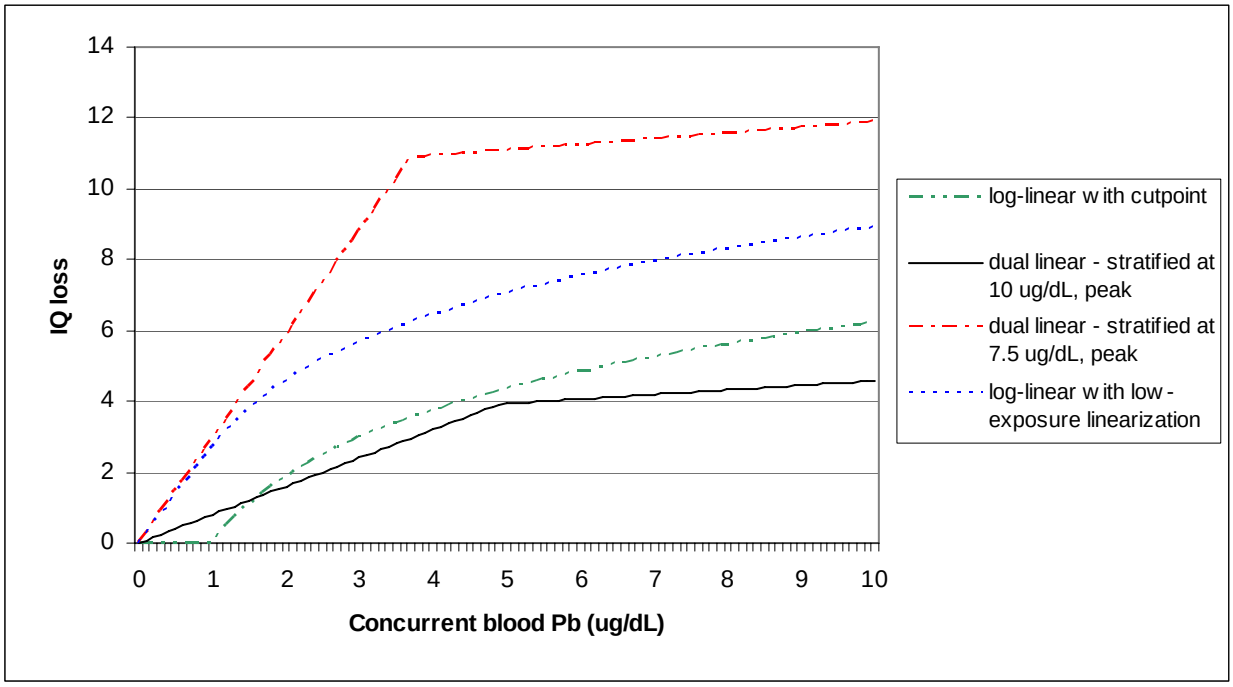

Table 5-8. Comparison of total and incremental IQ loss estimates below 10 µg/dL for the four concentration-response functions.

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

5.3.1.2 Projection of Population Risk

The methods used to project population-level risk for the additional analyses are those described in Section 4.1.2.

With regard to population-level risk metrics, risk estimates for different population percentiles are generated for all case studies. For the location-specific urban case studies, an additional type of population risk metric is also presented. Specifically, we have estimated the number of children associated with different magnitudes of IQ loss resulting from total Pb exposure under different ambient air quality scenarios (i.e., IQ loss incidence estimates). This metric illustrates (a) the overall number of children within a given urban case study location projected to experience various levels of IQ loss due to Pb exposure and (b) how that distribution of incidence changes with alternate NAAQS levels.

5.3.2 Risk Estimates

Estimates of IQ loss resulting from Pb exposure have been developed for each air quality scenario in each case study (see Appendices N and O). Further, multiple sets of risk results were generated for each combination of case study and air quality scenario, in an effort to consider uncertainty specifically related to specifying the relationship between Pb exposure and IQ loss (see Section 5.1.2). That is, four separate risk distributions were generated for each air quality scenario for each of the case studies presented here (see Table 5-1). These risk estimates include (a) population-risk distribution estimates (in the form of estimated IQ loss for specific population percentiles) and (b) IQ loss incidence estimates, as described below.

As noted earlier in presenting exposure results (see Section 5.2.4), IQ loss estimates associated with air pathways and current levels of Pb emitted to the air (including via resuspension) are likely to fall between the estimates for “recent air” and those for “recent” plus “past air”. Additionally, with regard to the location-specific urban case studies “recent air” and “recent” plus “past air” categories, an artifact of the hybrid dust Pb model tends to mask trends in the two components of dust Pb (recent air and other), which contribute to “recent air” and “past air” estimates, respectively (see Section 4.3.1 discussion of this uncertainty). For the primary Pb smelter case study, uncertainty in parsing out the "recent air" and "other" components of indoor dust (specifically for the site-specific regression model used in the remediation zone) have led us to conclude that only "recent plus past air" IQ loss estimates should be presented and "recent air" results should not be separately presented for the primary Pb smelter, as is done for the other case studies (see Section 3.1.4.2).

5.3.2.1 Population Risk Distribution Estimates

Risk estimates for specific population percentiles of the risk distribution associated with total blood Pb levels are presented in Tables 5-9 and 5-10. These include the median (Table 5-9) and 95th percentile (Table 5-10) IQ loss estimates based on total blood Pb. Two additional types of risk estimates are summarized in Tables 5-9 and 5-10 in addition to IQ loss based on total blood Pb. Also presented are the contributions to IQ loss from the recent air and recent plus past air exposure pathways (see Section 3.2.2), which as noted previously (Sections 2.4.3 and 3.2.2) represent the policy-relevant pathways, with the true values for the policy-relevant pathways considered to fall between the estimates for "recent air" and those for "recent" plus "past air". More detailed pathway apportionment estimates (including percentile contributions to total IQ loss from individual exposure pathways) are presented in the detailed risk results tables in Appendices N, O and P. These more detailed risk results are, however, subject to the same limitations related to presentation of pathway apportionment, as noted above for individual case studies (e.g., reduced confidence in parsing out recent air-related risk from recent plus past air risk for the primary Pb smelter case study).

The total Pb exposure risk estimates presented in Table 5-9 are those for the median in the distribution of total risk, and the estimates for the other two categories in that table (i.e., recent air and recent plus past air) are the values for those categories associated with the median for the total exposure pathway estimate. The corresponding estimates for the 95th percentile in the distribution of IQ loss estimates for total blood Pb for each case study are presented in Table 5-10. This presentation may lead to some seeming inconsistencies in trends for recent air and recent air plus past air risk estimates across air quality scenarios. This is because the recent air or recent plus past air estimates associated with the median and 95th percentile total blood Pb estimate may not necessarily be the median and 95th percentiles of the distribution of estimates for those specific categories. This is because the blood Pb level (and associated total IQ loss) for a simulated child reflects not only the total Pb uptake (from both background and ambient air-related pathways), but also the GSD-based adjustment factor. This means that two simulated children could have identical blood Pb levels (and IQ loss estimates), but one child could have a higher recent air exposure, with the other child having their lower recent air exposure compensated for by a higher GSD-based adjustment factor, thereby resulting in both children having the same total blood Pb level. In presenting IQ loss estimates in tables 5-9 and 5-10, all values are rounded to one decimal place.

As mentioned in Chapter 1, risk results are provided here without substantial interpretation. Rather, interpretative discussion of these results is provided in the Staff Paper.

Table 5-9. Summary of risk estimates for medians of total-exposure risk distributions.

Case Study and Air Quality Scenario	Points IQ Loss[a]								
	Recent Air[b] (low, LLL and high C-R function estimates)			Recent plus Past Air[b] (low, LLL and high C-R function estimates)			Total Pb Exposure (low, LLL and high C-R function estimates)		
	Low	LLL[c]	High	Low	LLL	High	Low	LLL	High
Location-specific (Chicago)									
Current NAAQS (1.5 μg/m³, max quarterly)	1.4	3.4	5.6	2.0	4.7	7.4	2.4	5.6	8.8
Current conditions (0.14 μg/m³ max quarterly; 0.31 μg/m³ max monthly)	0.3	0.6	0.7	1.0	2.9	3.5	1.4	4.2	5.2
Alternative NAAQS (0.2 μg/m³, max monthly)	0.3	0.6	0.9	1.0	2.9	3.6	1.4	4.2	5.2
Alternative NAAQS (0.05 μg/m³, max monthly)	0.1	0.2	0.2	0.9	2.6	3.2	1.3	4.0	4.8
Alternative NAAQS (0.02 μg/m³, max monthly)	<0.1	0.1	0.1	0.8	2.6	3.1	1.3	4.0	4.7
Location-specific (Cleveland)									
Current NAAQS (1.5 μg/m³, max quarterly)	0.6	2.8	2.1	1.3	3.9	4.6	1.7	4.7	6.3
Current conditions (0.36 μg/m³ max quarterly; 0.56 μg/m³ max monthly)	0.2	0.7	0.9	1.0	2.9	3.6	1.4	4.2	5.2
Alternative NAAQS (0.5 μg/m³, max monthly)	0.2	0.6	1.9	1.0	2.9	3.9	1.4	4.2	5.2
Alternative NAAQS (0.2 μg/m³, max quarterly)	0.1	0.4	0.5	0.9	2.8	3.3	1.4	4.1	5.0
Alternative NAAQS (0.2 μg/m³, max monthly)	0.1	0.6	0.4	0.9	2.8	3.2	1.3	4.1	4.9
Alternative NAAQS (0.05 μg/m³, max monthly)	<0.1	0.1	0.1	0.8	2.6	3.1	1.3	4.0	4.7
Alternative NAAQS (0.02 μg/m³, max monthly)	<0.1	<0.1	0.1	0.8	2.6	3.0	1.2	3.9	4.6
Location-specific (Los Angeles)									
Current NAAQS (1.5 μg/m³, max quarterly)	1.1	2.7	4.0	1.7	4.2	6.2	2.1	5.3	7.7
Current conditions (0.09 μg/m³ max quarterly; 0.17 μg/m³ max monthly)	0.2	0.7	0.9	0.9	2.9	3.5	1.4	4.2	5.1
Alternative NAAQS (0.05 μg/m³, max monthly)	0.1	0.3	0.4	0.9	2.7	3.2	1.3	4.0	4.8
Alternative NAAQS (0.02 μg/m³, max monthly)	<0.1	0.1	0.2	0.8	2.6	3.1	1.3	4.0	4.7
General urban									
Current NAAQS (1.5 μg/m³, max quarterly)	1.5	3.5	5.6	2.1	4.8	7.7	2.5	5.8	9.2
Alternative NAAQS (0.5 μg/m³, max monthly)	0.7	1.9	2.5	1.3	3.6	4.8	1.7	4.8	6.4
Current conditions (0.87 μg/m³ max quarterly)	0.6	1.8	2.4	1.3	3.6	4.7	1.7	4.7	6.3
Alternative NAAQS (0.2 μg/m³, max quarterly)	0.5	1.5	2.0	1.2	3.4	4.3	1.6	4.6	5.9
Current conditions (0.14 μg/m³ max quarterly)	0.4	1.3	1.6	1.1	3.2	4.1	1.5	4.5	5.6
Alternative NAAQS (0.2 μg/m³, max monthly)	0.4	1.2	1.5	1.1	3.2	4.0	1.5	4.4	5.6
Alternative NAAQS (0.05 μg/m³, max monthly)	0.2	0.5	0.6	0.9	2.8	3.3	1.3	4.1	5.0
Alternative NAAQS (0.02 μg/m³, max monthly)	0.1	0.3	0.3	0.9	2.6	3.1	1.3	4.0	4.8
Primary Pb smelter - full study area									
Current NAAQS (1.5 μg/m³, max quarterly)	NA[d]			0.6	1.9	2.3	1.2	3.8	4.4
Alternative NAAQS (0.5 μg/m³, max monthly)				0.8	2.9	2.7	1.0	3.7	4.2
Alternative NAAQS (0.2 μg/m³, max quarterly)				0.6	2.3	2.6	0.9	3.6	4.2
Alternative NAAQS (0.2 μg/m³, max monthly)				0.4	2.7	3.0	0.9	3.6	4.1
Alternative NAAQS (0.05 μg/m³, max monthly)				0.4	1.7	1.9	0.9	3.6	4.0
Alternative NAAQS (0.02 μg/m³, max monthly)				0.6	2.6	3.0	0.9	3.6	4.1
Primary Pb smelter - 1.5 km subarea									
Current NAAQS (1.5 μg/m³, max quarterly)	NA[d]			3.2	6.0	9.4	3.7	6.8	11.2
Alternative NAAQS (0.5 μg/m³, max monthly)				2.1	4.5	7.7	2.6	5.8	9.4
Alternative NAAQS (0.2 μg/m³, max quarterly)				1.5	3.8	5.6	2.0	5.2	7.4
Alternative NAAQS (0.2 μg/m³, max monthly)				1.2	3.7	5.1	1.9	5.0	6.9
Alternative NAAQS (0.05 μg/m³, max monthly)				0.9	2.8	3.4	1.4	4.2	5.1
Alternative NAAQS (0.02 μg/m³, max monthly)				0.9	2.9	3.3	1.3	4.0	4.8

a - Estimates are rounded to one decimal place.
b -The term "past air" includes contributions from the outdoor soil/dust contribution to indoor dust, historical air contribution to indoor dust, and outdoor soil/dust pathways, while "recent air" refers to contributions from inhalation of ambient air Pb or ingestion of indoor dust Pb predicted to be associated with outdoor ambient air Pb levels, with outdoor ambient air also potentially including resuspended, previously deposited Pb (see Section 2.4.3).
c - Log-linear with low-exposure linearization concentration-response function.
d – "Recent air" estimates were not developed for the primary Pb smelter case study (see Section 3.1.4.2).

Table 5-10. Summary of risk estimates for 95th percentiles of total exposure risk distributions.

Case Study and Air Quality Scenario	Points IQ Loss[a]								
	Recent Air[b] (low, LLL and high C-R function estimates)			Recent plus Past Air[b] (low, LLL and high C-R function estimates)			Total Pb Exposure (low, LLL and high C-R function estimates)		
	Low	LLL[c]	High	Low	LLL	High	Low	LLL	High
Location-specific (Chicago)									
Current NAAQS (1.5 μg/m³, max quarterly)	3.0	5.8	7.7	4.0	7.6	10.3	4.7	9.0	12.1
Current conditions (0.14 μg/m³ max quarterly; 0.31 μg/m³ max monthly)	0.7	1.3	2.0	2.8	5.2	7.8	4.1	7.5	11.4
Alternative NAAQS (0.2 μg/m³, max monthly)	1.0	1.9	2.8	2.9	5.3	8.1	4.1	7.5	11.4
Alternative NAAQS (0.05 μg/m³, max monthly)	0.2	0.4	0.7	2.7	4.8	7.5	4.1	7.3	11.3
Alternative NAAQS (0.02 μg/m³, max monthly)	0.2	0.3	0.5	2.7	4.8	7.4	4.1	7.3	11.3
Location-specific (Cleveland)									
Current NAAQS (1.5 μg/m³, max quarterly)	1.4	2.6	3.7	3.1	5.9	8.5	4.3	8.1	11.6
Current conditions (0.36 μg/m³ max quarterly; 0.56 μg/m³ max monthly)	0.6	1.2	1.8	2.8	5.2	7.8	4.1	7.6	11.4
Alternative NAAQS (0.5 μg/m³, max monthly)	1.1	1.9	2.9	2.9	5.3	8.1	4.1	7.5	11.4
Alternative NAAQS (0.2 μg/m³, max quarterly)	0.5	0.9	1.4	2.8	5.0	7.6	4.1	7.4	11.3
Alternative NAAQS (0.2 μg/m³, max monthly)	0.3	0.6	0.9	2.7	4.9	7.5	4.1	7.4	11.3
Alternative NAAQS (0.05 μg/m³, max monthly)	0.1	0.2	0.3	2.6	4.7	7.4	4.0	7.2	11.3
Alternative NAAQS (0.02 μg/m³, max monthly)	<0.1	0.1	0.1	2.6	4.7	7.2	4.0	7.2	11.3
Location-specific (Los Angeles)									
Current NAAQS (1.5 μg/m³, max quarterly)	2.3	4.4	6.1	3.6	6.9	9.5	4.5	8.6	11.8
Current conditions (0.09 μg/m³ max quarterly; 0.17 μg/m³ max monthly)	0.8	1.4	2.1	2.8	5.2	7.8	4.1	7.5	11.4
Alternative NAAQS (0.05 μg/m³, max monthly)	0.4	0.7	1.1	2.7	4.9	7.6	4.1	7.3	11.3
Alternative NAAQS (0.02 μg/m³, max monthly)	0.1	0.2	0.4	2.7	4.7	7.4	4.0	7.2	11.3
General urban									
Current NAAQS (1.5 μg/m³, max quarterly)	2.9	5.5	7.3	3.9	7.6	10.1	4.7	9.1	12.1
Alternative NAAQS (0.5 μg/m³, max monthly)	1.7	3.2	4.6	3.3	6.1	8.8	4.3	8.1	11.6
Current conditions – high-end (0.87 μg/m³ max quarterly)	1.6	3.1	4.4	3.2	6.0	8.7	4.3	8.0	11.6
Alternative NAAQS (0.2 μg/m³, max quarterly)	1.4	2.6	3.8	3.1	5.8	8.5	4.2	7.9	11.5
Current conditions - mean (0.14 μg/m³ max quarterly)	1.2	2.2	3.3	3.0	5.6	8.3	4.2	7.7	11.5
Alternative NAAQS (0.2 μg/m³, max monthly)	1.1	2.1	3.1	3.0	5.6	8.2	4.2	7.7	11.4
Alternative NAAQS (0.05 μg/m³, max monthly)	0.5	0.9	1.4	2.8	5.0	7.7	4.1	7.4	11.3
Alternative NAAQS (0.02 μg/m³, max monthly)	0.3	0.5	0.7	2.7	4.8	7.5	4.1	7.3	11.3
Primary Pb smelter - full study area									
Current NAAQS (1.5 μg/m³, max quarterly)	NA[d]			2.3	4.2	6.8	3.7	6.8	11.2
Alternative NAAQS (0.5 μg/m³, max monthly)				2.5	4.9	8.3	3.4	6.6	11.1
Alternative NAAQS (0.2 μg/m³, max quarterly)				1.9	3.9	6.6	3.2	6.5	11.1
Alternative NAAQS (0.2 μg/m³, max monthly)				2.0	4.0	7.0	3.2	6.4	11.1
Alternative NAAQS (0.05 μg/m³, max monthly)				1.5	3.1	5.4	3.1	6.3	11.0
Alternative NAAQS (0.02 μg/m³, max monthly)				2.5	5.3	9.2	3.1	6.3	11.0
Primary Pb smelter - 1.5 km subarea									
Current NAAQS (1.5 μg/m³, max quarterly)	NA[d]			4.2	8.0	10.4	5.0	9.5	12.4
Alternative NAAQS (0.5 μg/m³, max monthly)				4.0	7.5	10.5	4.5	8.5	11.8
Alternative NAAQS (0.2 μg/m³, max quarterly)				3.7	6.9	10.2	4.2	7.8	11.5
Alternative NAAQS (0.2 μg/m³, max monthly)				3.4	6.1	9.2	4.1	7.6	11.4
Alternative NAAQS (0.05 μg/m³, max monthly)				2.8	5.3	8.7	3.6	6.8	11.1
Alternative NAAQS (0.02 μg/m³, max monthly)				2.4	4.7	7.9	3.3	6.5	11.1

a - Estimates are rounded to one decimal place.
b -The term "past air" includes contributions from the outdoor soil/dust contribution to indoor dust, historical air contribution to indoor dust, and outdoor soil/dust pathways, while "recent air" refers to contributions from inhalation of ambient air Pb or ingestion of indoor dust Pb predicted to be associated with outdoor ambient air Pb levels, with outdoor ambient air also potentially including resuspended, previously deposited Pb (see Section 2.4.3).
c - Log-linear with low-exposure linearization concentration-response function.
d – "Recent air" estimates were not developed for the primary Pb smelter case study (see Section 3.1.4.2).

5.3.2.2 IQ Loss Incidence Estimates

Incidence of different levels of IQ loss has been estimated for the three location-specific urban case studies (Appendix O). These estimates are summarized in Tables 5-11 and 5-12. Specifically, Table 5-11 shows the number of children projected to have IQ loss greater than one point under the current conditions scenario and the change in that metric under the current NAAQS and the alternate NAAQS considered for each location-specific urban case study. The value of one IQ point was selected as consistent with CASAC recommendations as to the magnitude of IQ loss they considered to be significant from a public health perspective (Henderson, 2007b). Similarly, Table 5-12 presents incidence estimates associated with a greater magnitude of cognitive impact, the number of children exceeding 7 points IQ loss. The value of 7 IQ points is the magnitude of IQ loss projected for the 95^{th} population percentile (using the log-linear with low-exposure linearization function) under current conditions at the case studies evaluated in this analysis (See Table 5-10). As mentioned in Chapter 1, risk results are provided here without substantial interpretation. Rather, interpretative discussion of these results is provided in the Staff Paper.

Table 5-11. Incidence of children with >1 point IQ loss.

Air Quality Scenario (for location-specific urban case studies)	dual linear - stratified at 7.5 µg/dL peak		log-linear with linearization		dual linear - stratified at 10 µg/dL peak		log-linear with cutpoint	
	Incidence of >1 point IQ loss	Delta (change in incidence compared to current conditions)	Incidence of >1 point, IQ loss	Delta (change in incidence compared to current conditions)	Inci-dence of >1 point, IQ loss	Delta (change in incidence compared to current conditions)	Incidence of >1 point, IQ loss	Delta (change in incidence compared to current conditions)
Chicago (total modeled population: 396,511)								
Chicago Current Conditions	391,602		389,754		271,031		236,257	
Current NAAQS (1.5 µg/m^3 Maximum Quarterly)	395,797	4,195	395,528	5,773	347,415	76,384	314,053	77,795
Alternative NAAQS (0.2 µg/m^3 Maximum Monthly)	391,158	-444	389,461	-293	271,444	412	235,559	-698
Alternative NAAQS (0.05 µg/m^3 Maximum Monthly)	389,572	-2,030	387,407	-2,347	253,775	-17,256	224,394	-11,864
Alternative NAAQS (0.02 µg/m^3 Maximum Monthly)	389,176	-2,427	386,630	-3,125	249,865	-21,166	219,294	-16,963
Cleveland (total modeled population: 13,990)								
Cleveland Current Conditions	13,809		13,745		9,526		8,515	
Current NAAQS (1.5 µg/m^3 Maximum Quarterly)	13,893	84	13,857	112	10,664	1,137	9,769	1,254
Alternative NAAQS (0.2 µg/m^3 Maximum Quarterly)	13,770	-38	13,703	-42	9,221	-305	8,160	-354
Alternative NAAQS (0.5 µg/m^3 Maximum Monthly)	13,789	-20	13,720	-25	9,497	-29	8,464	-51
Alternative NAAQS (0.2 µg/m^3 Maximum Monthly)	13,759	-50	13,694	-51	9,083	-443	8,010	-505
Alternative NAAQS (0.05 µg/m^3 Maximum Monthly)	13,729	-80	13,642	-103	8,785	-741	7,720	-795
Alternative NAAQS (0.02 µg/m^3 Maximum Monthly)	13,720	-88	13,628	-117	8,736	-790	7,668	-846
Los Angeles (total modeled population: 372,252)								
Los Angeles Current Conditions	282,216		280,711		191,675		170,474	
Current NAAQS (1.5 µg/m^3 Maximum Quarterly)	285,272	3,056	284,945	4,234	240,988	49,313	226,608	56,134
Alternative NAAQS (0.05 µg/m^3 Maximum Monthly)	281,112	-1,104	279,658	-1,053	183,395	-8,280	161,914	-8,560
Alternative NAAQS (0.02 µg/m^3 Maximum Monthly)	280,740	-1,476	279,057	-1,654	180,745	-10,929	158,234	-12,240

Table 5-12. Incidence of children with >7 points IQ loss.

Air Quality Scenario (location-specific urban case studies)	dual linear - stratified at 7.5 µg/dL peak		log-linear with linearization		dual linear - stratified at 10 µg/dL peak		log-linear with cutpoint	
	Incidence of > 7 points IQ loss	**Delta** (change in incidence compared to current conditions)	**Incidence of > 7 points IQ loss**	**Delta** (change in incidence compared to current conditions)	**Inci-dence of > 7 points IQ loss**	**Delta** (change in incidence compared to current conditions)	**Incidence of > 7 points IQ loss**	**Delta** (change in incidence compared to current conditions)
Chicago (total modeled population: 396,511)								
Chicago Current Conditions	136,709		33,664		63		1,015	
Current NAAQS (1.5 µg/m^3 Maximum Quarterly)	244,401	107,692	100,159	66,495	555	492	5,226	4,211
Alternative NAAQS (0.2 µg/m^3 Maximum Monthly)	136,067	-642	32,546	-1,118	48	-16	1,007	-8
Alternative NAAQS (0.05 µg/m^3 Maximum Monthly)	120,706	-16,003	27,367	-6,297	16	-48	864	-151
Alternative NAAQS (0.02 µg/m^3 Maximum Monthly)	117,819	-18,890	26,027	-7,637	8	-56	690	-325
Cleveland (total modeled population: 13,990)								
Cleveland Current Conditions	4,834		1,212		3		46	
Current NAAQS (1.5 µg/m^3 Maximum Quarterly)	6,139	1,305	1,858	647	4	2	105	59
Alternative NAAQS (0.2 µg/m^3 Maximum Quarterly)	4,525	-309	1,073	-139	1	-2	40	-6
Alternative NAAQS (0.5 µg/m^3 Maximum Monthly)	4,806	-28	1,180	-31	1	-2	43	-3
Alternative NAAQS (0.2 µg/m^3 Maximum Monthly)	4,424	-410	1,026	-186	1	-2	43	-3
Alternative NAAQS (0.05 µg/m^3 Maximum Monthly)	4,106	-728	886	-326	0	-3	24	-22
Alternative NAAQS (0.02 µg/m^3 Maximum Monthly)	4,051	-783	866	-345	0	-3	27	-18
Los Angeles (total modeled population: 372,252)								
Los Angeles Current Conditions	94,684		22,665		23		732	
Current NAAQS (1.5 µg/m^3 Maximum Quarterly)	158,171	63,487	57,834	35,168	183	160	3,771	3,038
Alternative NAAQS (0.05 µg/m^3 Maximum Monthly)	87,303	-7,382	19,781	-2884	11	-11	624	-109
Alternative NAAQS (0.02 µg/m^3 Maximum Monthly)	83,909	-10,775	17,939	-4726	17	-6	498	-235

5.3.3 Uncertainty Characterization and Sensitivity Analysis

This section discusses uncertainty related to exposure and risk estimates generated for the results for the core modeling approach applied to the primary Pb smelter, general urban and location-specific urban case studies. It also discusses sensitivity analyses completed to inform application of the core modeling approach to these case studies. Several methods have been used to examine uncertainty in our modeling approach and its potential impact on exposure risk estimates. These methods for uncertainty evaluation include the following:

- Qualitative discussion of key sources of uncertainty and their potential impact on exposure and risk estimates (Section 5.3.3.1). This qualitative discussion focuses on factors particular to the new analyses presented in this section - those sources of uncertainty that these new analyses share with the analyses presented in Chapters 3 and 4 are not repeated here (see Section 4.3.1 for that discussion).
- Evaluation of model performance, including comparison with empirical data (Section 5.3.3.2). As with the qualitative discussion of uncertainty, only those performance evaluation elements particular to the analyses in this chapter (i.e., performance evaluation of modeled blood Pb levels - see Section 5.2.5.2) are discussed here. The reader is referred back to Section 4.3.3 for a more complete discussion of performance evaluation related to modeled media concentrations and blood Pb modeling for this risk assessment.
- Development of multiple sets of risk estimates for each assessment scenario that illustrate the impact of different concentration-response models relating Pb exposure to IQ loss and the associated uncertainty (see Section 5.3.3.3).

In addition to these methods for evaluating uncertainty in the modeling approach, an additional sensitivity analysis has been completed for this portion of the analysis, specifically focused on the hybrid indoor dust Pb model used in the general urban case study and the location-specific urban case studies. This sensitivity analysis is discussed in Section 5.3.3.4. The results of the sensitivity analysis described in Section 4.3.2 for the general urban case study are broadly applicable to that case study, as well as the location-specific urban case studies.

5.3.3.1 Qualitative Discussion of Key Sources of Uncertainty

This section provides qualitative discussion of key sources of uncertainty related to the analyses presented in this section. Specifically, it addresses sources of uncertainty specific to the location-specific urban case studies; sources of uncertainty related to the primary Pb smelter and general urban case studies have already been discussed (see Section 4.3.1). In addition, many of the sources of uncertainty that impact the general urban case study also impact the location-specific urban case studies, since both share similar modeling elements (e.g., hybrid indoor dust Pb model, IQ loss functions, blood Pb GSD). The reader is referred back to Section 4.3.1 for

discussion of those sources of uncertainty. Sources of uncertainty particular to the location-specific urban case studies include:

- *Location-specific urban case studies*: As recognized in Appendix A, the Pb-TSP monitoring network is currently quite limited. The number of monitors available to represent air concentrations in these case studies ranged from six for Cleveland to 11 for Chicago. Accordingly, our estimates of the magnitude of and spatial variation of air Pb concentrations are subject to uncertainty associated with the limited data. In applying the available data to each of these case studies, exposure zones, one corresponding to each monitor, were created and each U.S. Census block (and the children within that demographic unit) were distributed among the exposure zones. The details of the approach used are described in Section 5.1.3. Although this approach provides a spatial gradient across the study area due to differences in monitor values for each exposure zone, this approach assumes a constant concentration within each exposure zone (i.e., no spatial gradient within a zone). Additionally, the nearest neighbor approach to assign blocks to exposure zones assumes that a monitor pertains to all locations that are closer to that monitor than to any of the others in the study area. In reality, there may be different and more variable spatial gradients in a study area than those reflected in the approach used here. This introduces uncertainty into the characterization of risk for the urban case studies.
- *Current NAAQS air quality scenarios*: For the location-specific urban case studies, proportional roll-up procedures were used to adjust ambient air Pb concentrations up to just meet the current NAAQS (see Sections 2.3.1 and 5.2.2.1 for detailed discussion). Staff recognizes that it is extremely unlikely that Pb concentrations in urban areas would rise to meet the current NAAQS and that there is uncertainty with our simulation of such conditions. In these case studies we have simulated a proportional roll-up, such that it is assumed that the current spatial distribution of air concentrations (as characterized by the current data) is maintained and increased Pb emissions contribute to increased Pb concentrations, the highest of which just meets the current standard. There are many other types of changes within a study area that could result in a similar result such as increases in emissions from just one specific industrial operation that led to air concentrations in a part of the study area that just meet the current NAAQS, while the remainder of the study area remained largely unchanged (at current conditions). For the primary Pb smelter case study, where current conditions exceed the current NAAQS, reaching the current NAAQS was simulated using air quality modeling, emissions and source parameters used in developing the 2007 proposed revision to the State Implementation Plan for the area (see Section 3.1.1.2 for details).
- *Alternative NAAQS air quality scenarios*: In all case studies, proportional roll-down procedures were used to adjust ambient air Pb concentrations downward to reach alternative NAAQS (see Sections 2.3.1 and 5.2.2.1). We recognize that there is uncertainty in simulating conditions associated with the implementation of emissions reduction actions to meet a lower standard. There are a variety of changes other than that represented by a proportional roll-down that could result in air concentrations

that just meet lower alternative standards. For example, control measures might be targeted only at the specific area exceeding standard, resulting in a reduction of air Pb concentrations to the alternate standard while concentrations in the rest of the study area remain unchanged (at current conditions). Consequently, there is uncertainty associated with estimates for the alternate NAAQS scenarios.

- *Relating blood Pb levels to IQ loss*: Specification of the quantitative relationship between blood Pb level (exposure) and IQ loss is subject to uncertainty, especially in the projection of IQ loss at lower blood Pb levels (below 5 µg/dL concurrent blood Pb). As discussed in Section 2.1.5, this reflects limitations in the data available for characterizing the concentration-response relationship. For example, the pooled international dataset analyzed by Lanphear and others (2005) includes relatively few children with blood Pb levels below 5 µg/dL and no children with levels below 1 µg/dL (see Section 2.1.5). As presented in Section 5.2.4, blood Pb levels in this region are a particular focus in this review. For example, as is the case for mean blood Pb levels nationally in the U.S. (CD, Section 4.3.1.3), concurrent blood Pb estimates for the median of the populations simulated in this assessment fall below 5 µg/dL. In recognition of the uncertainty in specifying a quantitative concentration-response relationship at such levels, our core modeling approach involves the application of four different functions to generate a range of risk estimates (see Sections 4.2.6 and 5.3.1). The range of absolute IQ loss seen for a given case study/air quality scenario combination when modeled using the four concentration-response functions is typically close to a factor of 3. However, it is important to point out that the relative (proportional) change in IQ loss across air quality scenarios (i.e., the pattern of risk reduction across air quality scenarios) is fairly consistent across all four models. Note, however, that the function producing higher overall risk estimates (the dual linear function, stratified at 7.5 µg/dL, peak) will also produce larger absolute reductions in IQ loss compared with the other three functions.

5.3.3.2 Performance Analyses

This section discusses the performance evaluation completed for blood Pb modeling for the core analysis of the general urban and primary Pb smelter case studies. As mentioned above, the performance evaluation of media concentrations for these case studies is discussed in Section 4.3.3.

The additional performance evaluation discussed in this chapter focused on a set of air-to-blood ratios developed using a different approach than that used in Chapter 3– (see Section 5.2.5.2). The results of this analysis suggested that ratios generated for the general urban case study and for the primary Pb smelter (full study area) were generally similar to those identified in the literature, with the exception of ratios associated with the lowest alternative NAAQS, which were larger than the typical range of ratios seen cited in the literature. By contrast, the set of ratios generated for all air quality scenarios for the 1.5 km subarea of the primary Pb smelter were noticeably larger than the values cited in the literature. A number of plausible explanations for this discrepancy are presented in Section 5.2.5.2.

5.3.3.3 Uncertainty in Modeling Approaches - Multiples Sets of Results

For the case studies included in the additional analyses presented in this chapter, four sets of risk estimates were generated for each air quality scenario, reflecting consideration of four concentration-response functions relating Pb exposure to IQ loss. The resulting median and 95th percentile risk estimates for each case study and air quality scenario combination are presented in Tables 5-9 and 5-10. As can be seen from these tables, estimates based on these four functions result in a risk range spanning a factor of 4 for a particular case study and air quality scenario, although the range among results for three of the four functions is much tighter.

5.3.3.4 Sensitivity Analysis – Indoor Dust Pb Modeling

One additional sensitivity analysis was completed as part of the analyses based on the core modeling approach. Specifically, an alternate version of the hybrid indoor dust Pb model used for the general urban and location-specific urban case studies was considered. This alternate form of the model used a different approach for partitioning total dust Pb estimates between recent air and "other" categories (see Section 3.1.4.1). Note, that consideration for alternative concentration-response functions for IQ loss is not discussed here as part of the sensitivity analysis, since all four concentration-response functions were used to evaluate the full set of case studies and those results can be reviewed to identify differences in the performance of these alternate models. The sensitivity analysis focusing on the hybrid indoor dust Pb model is presented here.

Comments received from a member of the public regarding the full-scale analysis methodology suggested a modification to the hybrid indoor dust Pb model used in evaluating the general urban case study (see Section 3.1.4.1 for details on the hybrid indoor dust Pb model). Specifically, rather than establishing the nonair fraction of indoor dust Pb as part of overall Pb loading, as is currently done in the hybrid model, the recommendation was made that EPA take the final version of the hybrid model, which predicts indoor dust Pb concentration, set the air term to zero and solve for nonair indoor dust Pb (this providing an estimate of nonair indoor dust Pb). This recommendation was made, in part, to address concerns raised by the commenter that the nonair indoor dust Pb concentrations generated by the hybrid model, were not constant across air quality scenarios. EPA recognizes that the hybrid indoor dust Pb model, as used in the full-scale analysis, does produce nonair indoor dust Pb concentrations which vary by air quality scenarios. This results from nonlinearity in the loading to concentration conversion algorithm

within the model, which generates a greater per unit concentration at lower overall loading levels.

To inform our understanding of model uncertainty related to predicting indoor dust Pb levels for the urban scenarios, we have included this alternative hybrid model in this sensitivity analysis. However, it is important to point out a key limitation in this formulation of the hybrid dust model. By setting ambient air Pb levels to zero and then solving for indoor dust Pb, one is using the steepest part of the loading-to-concentration curve to conduct the key step of translating indoor dust Pb loading to equivalent concentration. In reality, we would expect to always have a mixture of indoor dust Pb loadings from nonair and air sources, thereby resulting in a larger total loading value, which would in turn be translated into an indoor dust Pb concentration at a flatter portion of the curve.

Solving of the hybrid indoor dust Pb model for an ambient air Pb level of zero, yields the fixed nonair indoor dust Pb level of 61 ppm. For the alternative hybrid model, this value has been used as the nonair indoor dust Pb level. The ambient air-related portion of indoor dust Pb concentration was then generated for each simulated individual by subtracting this value of 61 ppm from the total indoor dust Pb level generated by the hybrid model. If the total indoor dust Pb concentration was lower than 61 ppm, then we fixed the concentration at 61 ppm and assigned all of it to nonair sources.

Table 5-13 presents the median and 95th percentile IQ estimates associated with total blood Pb estimates associated with applying the two versions of the hybrid indoor dust Pb model to the general urban case study. The table also includes the IQ loss apportioned to recent air (i.e., inhalation plus ingestion of indoor dust loaded from air) for the median and 95th percentile IQ estimates associated with total blood Pb estimates. All IQ loss estimates presented in Table 5-13 were generated using the log-linear with low-exposure linearization concentration-response model.

Table 5-13. Comparison of hybrid indoor dust model with a modified form of the model.

Air quality scenario (General urban case study)	Points, IQ loss							
	Hybrid indoor dust model (from full-scale analysis)				Alternate hybrid indoor dust model (with fixed "other" dust value)			
	Median		95th percentile		Median		95th percentile	
	Total	Recent air	Total	Recent air	Total	Recent air	Total	Recent air
Current NAAQS (1.5 µg/m^3, max quarterly)	5.8	3.5	9.1	5.5	5.8	3.1	9.1	4.8
Current conditions–high-end (0.87 µg/m^3, max quarterly)	4.7	1.8	8.0	3.1	4.7	1.4	8.0	2.3
Current conditions – mean (0.14 µg/m^3, max quarterly)	4.5	1.3	7.7	2.2	4.4	0.9	7.7	1.6
Alternative NAAQS (0.5 µg/m^3, max monthly)	4.8	1.9	8.1	3.2	4.8	1.5	8.1	2.4
Alternative NAAQS (0.2 µg/m^3, max quarterly)	4.6	1.5	7.9	2.6	4.6	1.1	7.9	1.9
Alternative NAAQS (0.2 µg/m^3, max monthly)	4.4	1.2	7.7	2.1	4.4	0.8	7.7	1.5
Alternative NAAQS (0.05 µg/m^3, max monthly)	4.1	0.5	7.4	0.9	4.1	0.3	7.4	0.5
Alternative NAAQS (0.02 µg/m^3, max monthly)	4.0	0.3	7.3	0.5	4.0	0.1	7.3	0.3

Results presented in Table 5-13 suggest that the alternate hybrid indoor dust model (with the fixed "other indoor dust value") does produce indoor dust Pb concentrations for recent air that are noticeably lower than values generated using the original hybrid indoor dust model used in the full-scale analysis. However, it is important to point out that because both versions of the hybrid model generate the same total indoor dust Pb value (and only differ in the way that value is apportioned between other and recent air), the trend in total IQ loss due to Pb exposure across the air quality scenarios (moving from current NAAQS to the lowest alternative NAAQS evaluated) is the same for both versions of the hybrid model.

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