United States
Environmental Protection
Agency

Proposed Modeling Approaches for a Health-Based Benchmark for Lead in Drinking Water

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Abbreviations and Acronyms

AAP	American Academy of Pediatrics
ADHD	Attention deficit/hyperactivity disorder
AHHS	American Healthy Homes Survey
ATSDR	Agency for Toxic Substances and Disease Registry
BLL	Blood lead level
CCT	Corrosion control treatment
CDC	Centers for Disease Control and Prevention
CHAD	Consolidated Human Activity Database
CSFII	Continuing Survey of Food Intake for Individuals
EBLL	Elevated blood lead level
EFH	*Exposure Factors Handbook*
EPA	U.S. Environmental Protection Agency
FDA	U.S. Food and Drug Administration
FR	*Federal Register*
GSD	Geometric standard deviation
HUD	U.S. Department of Housing and Urban Development
IARC	International Agency for Research on Cancer
IEUBK	Integrated Exposure Uptake and Biokinetic model
IQ	Intelligence quotient
LCR	Lead and Copper Rule
LSL	Lead service line
MCLG	Maximum contaminant level goal
NAAQS	National Ambient Air Quality Standards
NCHS	National Center for Health Statistics
NDWAC	National Drinking Water Advisory Council
NHANES	National Health and Nutrition Examination Survey
NHEXAS	National Human Exposure Assessment Survey
NTP	National Toxicology Program
OEHHA	Office of Environmental Health Hazard Assessment
OGWDW	Office of Ground Water and Drinking Water
PAF	Passive Absorption Fraction
Pb	Lead
PCB	Public and commercial buildings
ppb	Parts per billion
PWS	Public water system
RLDWA	Reduction of Lead in Drinking Water Act
SDWA	Safe Drinking Water Act
SHEDS	Stochastic Human Exposure and Dose Simulation
SHEDS-Multimedia	Stochastic Human Exposure and Dose Simulation Model for Multipathway Chemicals
TDS	Total Diet Study
USDA	U.S. Department of Agriculture

Executive Summary

The Lead and Copper Rule (LCR) is a national primary drinking water regulation first promulgated in 1991 and revised in 2000 and 2007, which requires water systems to conduct tap sampling to determine the actions water systems must take to reduce exposure to lead and copper. Recognizing that there is no safe level of lead in drinking water, the LCR set a health-based maximum contaminant level goal of zero for lead in drinking water. Under the LCR, water systems must work with their customers to collect samples from locations with lead service lines or leaded plumbing materials. The LCR established an action level of 0.015 mg/L (15 ppb) for lead based upon an assessment that it was generally representative of what could be feasibly achieved at these taps with effective corrosion control treatment. The LCR action level is a screening tool for determining when certain treatment technique actions such as corrosion control, public education, and lead service line replacement are needed. If the lead action level is exceeded in more than 10 percent of tap water samples collected during any monitoring period (i.e., if the 90^{th} percentile level is greater than the action level), a water system must take actions to reduce exposure.

The U.S. Environmental Protection Agency (EPA) has engaged with stakeholder groups and the public on potential revisions to strengthen the LCR. As part of this work, the EPA's National Drinking Water Advisory Council's (NDWAC) Lead and Copper Rule Working Group was established to inform the Administrator on recommendations to strengthen public health protections of the LCR. In December 2015, the NDWAC provided specific recommendations to the EPA Administrator for LCR revisions related to lead service line replacement, public education, corrosion control treatment, copper, tap sampling, and the establishment of a *household action level*. The NDWAC recommended that this level be "based on the amount it would take an infant to have a blood lead level (BLL) greater than five micrograms per deciliter (µg/dL) based on consumption by an average, healthy infant of infant formula made with water" (Lead and Copper Rule Working Group, 2015, p. 37). The NDWAC recommended that water systems be required to notify the consumer, state drinking water program, and the local public health agency if this level were exceeded. The expectation is that individuals and local officials will use this information to take prompt actions at the household level to mitigate lead risks.

The EPA *Integrated Science Assessment for Lead* has concluded that lead exposure is causally or likely casually related to many neurodevelopmental endpoints in children including IQ loss; attention problems, impulsivity, and hyperactivity; antisocial behaviors (e.g., aggression, criminal activity); greater internalizing behaviors (e.g., withdrawn behavior, symptoms of depression, fearfulness, and anxiety); and auditory and motor function decrements (United States Environmental Protection Agency (U.S. EPA) 2013). Because of the well-documented functional relationship between increased BLLs and reduced IQ, EPA would be able to use the results of the lead modeling approaches relating drinking water lead concentrations to BLLs in children presented in this paper to assess the risk of reduced IQ.

While EPA has not yet determined the specific role of a household action level for lead in drinking water in the revised LCR, the Agency sees value in providing states, drinking water systems, and the public with a greater understanding of the potential health implications for vulnerable populations of specific levels of lead in drinking water. EPA has developed potential scientific modeling approaches to define the relationship between lead levels in drinking water and BLLs, particularly for sensitive life stages such as formula-fed infants and children up to age 7. EPA is using the terminology "health

based benchmark" to refer to this concept. EPA is conducting expert peer review of these alternative approaches to inform future consideration of a health-based benchmark for the LCR revisions. The purpose of this report is to obtain feedback on various modeling methods that can be used to assess the relationship between constant rate lead exposures of a representative child and a population of children across different age ranges and BLLs. The analysis also addresses different lead exposure scenarios including drinking water as the lone source of lead, and drinking water lead exposure in conjunction with other lead exposure pathways (air, food, soil and dust, and maternal contributions).

Approaches based on a modeled individual child include:

Approach 1. Estimate the concentration of lead in drinking water that would result in a 1 or 5 percent increase in the probability of a child having an elevated BLL (EBLL).

Approach 2. Estimate the concentration of lead in drinking water that would result in a 0.5 µg/dL or 1 µg/dL increase in a child's mean BLL.

Approaches based on a modeled population of children include:

Approach 3. Estimate the amount of lead in drinking water that would result in a target population of children's predicted distribution of BLLs having the 95^{th} or 97.5^{th} percentile BLL of 3.5 or 5 µg/dL.

In implementing these approaches, three age groups were evaluated due to their daily consumption patterns related to potential lead exposure and susceptibility to potential adverse health effects related to lead exposure:

- 0- to 6-month-old infants fed formula reconstituted with tap water, as this group is likely both susceptible to the effects of lead exposure due to their life stage and at risk of higher exposure if there is lead in their drinking water compared to other children, because of their higher water consumption rates.

- 1- to 2-year-olds, as this group is susceptible to the effects of lead exposure due to their life stage, and have the potential for higher exposures to lead from non-drinking water sources of lead, if present in their environment (i.e., soil and dust).

- 0- to 7-year-olds, as this group accounts for individuals potentially being exposed to lead in drinking water throughout their childhood up to age 7. Additionally, this age group represents cumulative exposure during the period over which lead exposure is estimated to have the greatest response in regard to changes in per unit IQ (Lanphear et al., 2005; Crump et al., 2013), one of several adverse health effects associated with lead exposure.

Approaches 1 and 2 are implemented using EPA's Integrated Exposure Uptake and Biokinetic Model (IEUBK), while approach 3 is implemented using EPA's Stochastic Human Exposure and Dose Simulation (SHEDS) model coupled with regression equations derived from IEUBK. In order to use updated information for current exposure profiles and human activity patterns, several of the default inputs to the IEUBK model were modified for this analysis. Additionally, it was necessary to input appropriate multimedia lead concentrations into the SHEDS model to conduct a multimedia exposure assessment.

1. Introduction

The U.S. Environmental Protection Agency (EPA) prepared this document to present and obtain feedback on potential modeling approaches that could inform the relationship between lead concentration in drinking water, potential exposure, and blood lead levels in children. Specifically, this report uses a number of methods to assess the relationship between constant rate lead exposures of a representative child and a population of children across different age ranges and blood lead levels (BLLs). The analysis also covers different lead exposure scenarios including drinking water as the lone source of lead, and drinking water lead exposures in conjunction with other lead exposure pathways (air, food, soil and dust, and maternal contributions).

1.1 Current Federal Regulatory Levels for Lead in Drinking Water

In 1991, EPA promulgated the Lead and Copper Rule (LCR) under the Safe Drinking Water Act (SDWA). The LCR establishes treatment technique protocols to reduce lead and copper in drinking water. It prescribes an action level for lead that is used as a screening tool for determining when certain treatment techniques are needed. Specifically, if lead concentrations exceed 15 µg/L (or 15 parts per billion (ppb)) in more than 10 percent of customers' tap samples, the system must undertake additional action to control corrosion and inform the public about steps they should take to protect their health. The current action level is not based on avoiding adverse health effects due to lead exposure but is instead based on "technical feasibility" based on EPA's evaluation of "available data on the ability of corrosion control to reduce lead levels at the tap" (56 FR 110 [7 June 1991], pp. 26490-26564).

In addition, SDWA requires EPA to set maximum contaminant level goals (MCLGs) for contaminants in drinking water. The MCLG is a non-enforceable standard that is defined under SDWA (Section 1412.b.4.B) as the concentration of a contaminant in drinking water at which "no known or anticipated adverse effect would occur, allowing for an adequate margin of safety" [42 U.S.C. § 300g–1]. Currently, the MCLG for lead in drinking water is 0 µg/L (U.S. EPA, 2016a). This is because the toxicity of lead is well characterized and lead is known to have adverse health effects even at low exposure levels. Regulatory agencies have stated that there is no threshold for adverse health effects due to lead exposure (U.S. EPA, 2016a).

1.2 Overview of the Health-Based Benchmark for Lead in Drinking Water

The current action level for lead in drinking water is based on the 90th percentile of all collected tap samples. The minimum number of samples collected is based on the population served by the public water system (PWS) (U.S. EPA, 2010a). The current action level is not based on health effects; rather, it is based on EPA's assessment on what was feasible using effective corrosion control treatment. When considering revisions to the LCR, EPA's National Drinking Water Advisory Council (NDWAC) recommended that EPA establish a level of lead in drinking water above which a PWS would be required to notify the consumer, local health department, and state drinking water authority of sample results. More specifically, the NDWAC recommended that EPA set what they called a *household action level*. The NDWAC recommended that this level be "based on the amount it would take an infant to have a blood lead level (BLL) greater than five micrograms per deciliter (µg/dL) based on consumption by an average, healthy infant of infant formula made with water" (Lead and Copper Rule Working Group, 2015, p. 37). In order to reduce confusion with the existing LCR system-wide "action level," this report will use the terminology "health-based benchmark" to refer to

this concept. EPA has not yet determined the specific role of a health-based benchmark such as the household action level in the revised LCR. However, the Agency sees value in providing states, drinking water systems, and the public with a greater understanding of the potential health implications for vulnerable populations of specific levels of lead in drinking water.

EPA has considered several potential modeling approaches to better define the potential relationship between lead in drinking water and BLL changes in children:

1. The level of lead in drinking water that results in an individual infant or child's probability of an elevated BLL (EBLL) being increased by 1 or 5 percent.
2. The level of lead in drinking water that results in an individual infant or child's BLL increasing by 0.5 or 1 µg/dL.
3. The level of lead in drinking water that results in the 95th or 97.5th percentile of predicted BLLs in the U.S. population of infants or children being equal to 3.5 or 5 µg/dL.

EPA has developed models to evaluate each of the above approaches, considering scenarios with and without non-drinking water sources of lead exposure.

1.3 Objective of the Report

The objective of this report is to obtain feedback on various modeling methods that can be used to assess the relationship between constant rate lead exposures to a representative child and a population of children across different age ranges and BLLs. To attain this objective, a brief overview is provided on lead exposure and health effects (Section 2). An overview of EPA's Integrated Exposure Uptake and Biokinetic (IEUBK) Model, which forms the basis of two of the three approaches for lead modeling, is presented in Section 3. The third approach uses EPA's Stochastic Human Exposure and Dose Simulation Model for Multipathway Chemicals (SHEDS-Multimedia), which is described in Section 4. Given that both models require inputs to specify an exposure profile of interest, the inputs used in the analysis are presented in Section 5. In Section 6, methods and results of the various analyses are presented, along with the strengths, weaknesses, and uncertainties of the approaches considered. Lastly, a comparison of results is presented in Section 7.

2. Background on Lead Exposure and Associated Adverse Health Effects

Lead is a naturally occurring, bluish-gray metal that is most commonly found in the environment in combination with other elements (Agency for Toxic Substances and Disease Registry (ATSDR) 2007). Lead in drinking water cannot be seen and does not have any taste or odor (U.S. EPA, 2016a). Lead enters drinking water mainly from corrosion of lead-containing plumbing materials. Larger lead particles quickly deposit in water. Once in the water, lead is highly persistent: it is relatively insoluble, and does not degrade (ATSDR, 2007).

2.1 Overview of Human Exposure to Lead

Individuals in the general population are primarily exposed to lead via ingestion, with inhalation as the second most common route of exposure (Thornton, Rautiu, & Brush, 2001). Ingestion exposures to lead can occur through a variety of pathways: directly via contaminated objects (e.g., when children place toys in their mouths), paint chips, food, or drinking water, or indirectly via hand-to-mouth contact after touching contaminated objects or surfaces with lead-containing paints (ATSDR, 2007; U.S. EPA, 2013).

Drinking water is unlikely to be contaminated with lead when it leaves water treatment plants and enters the distribution system; however, it may become contaminated due to the presence of lead pipes or leaded plumbing parts (Triantafyllidou & Edwards, 2012). Two types of lead can potentially contaminate drinking water: dissolved lead and particulate lead. Dissolved lead contamination can occur when water that has not been properly treated with optimized corrosion control treatment (CCT) stagnates in lead service lines (LSLs) (Clark, Masters, & Edwards, 2014). Particulate lead can produce spikes in lead concentrations when changes in water flow occur and pipe scales (i.e., deposits on the inner surfaces of pipes) are not durable or adherent (Clark et al., 2014). The contribution of particulate lead to total lead in drinking water is highly unpredictable because it depends on whether or not pipe scales are durable, which is difficult to determine (Triantafyllidou & Edwards, 2012). Thus, the cumulative level of lead in drinking water is variable and depends on numerous factors such as presence of LSLs, CCT, water flow, and pipe scale characteristics.

Although the use of LSLs in new construction was banned under the 1986 amendment to SDWA, LSLs may still be connected to or be present within older buildings and homes (Triantafyllidou & Edwards, 2012). In an attempt to decrease water lead concentrations where LSLs are present, partial LSL replacement has been implemented in many cities in the United States. This involves replacement of lead pipes owned by water utilities but not by property owners (Triantafyllidou & Edwards, 2012). However, partial LSL replacement was shown to be potentially harmful because it can increase water lead concentrations for at least several months after it is performed, due to disturbance of existing lead corrosion byproducts in scales and possible reactions between lead and pipes (Trueman, Camara, & Gagnon, 2016).

Additional sources of lead in drinking water are lead solder and brass plumbing components (Triantafyllidou & Edwards, 2012). Although plumbing parts sold for use in water systems intended for human consumption are required to be “lead free,” the definition of lead free varies by type of plumbing part and by year of manufacture (U.S. EPA, 2015b).

In 1986 Congress Amended the Safe Drinking Water Act, prohibiting the use of pipes, solder, or flux that was not "lead free" in public water systems or plumbing in facilities providing water for human consumption. At the time "lead free" was defined as solder and flux with no more than 0.2% lead and pipes with no more than 8%.

In 1996 Congress further amended the Safe Drinking Water Act, requiring plumbing fittings and fixtures (endpoint devices) sold for use in potable water sources to be in compliance with voluntary lead leaching standards. The amendments also prohibited the introduction into commerce of any pipe, pipe fitting, or plumbing fitting or fixture that is not lead free.

In 2011 Congress passed the Reduction of Lead in Drinking Water Act (RLDWA) revising the definition of lead free by lowering the maximum lead content of the wetted surfaces of plumbing products (such as pipes, pipe fittings, plumbing fittings, and fixtures) from 8% to a weighted average of 0.25%, establishing a statutory method for the calculation of lead content and eliminating the requirement that lead-free products be in compliance with voluntary standards established in accordance with SDWA Section 1417(e) for leaching of lead from new plumbing fittings and fixtures.

The 2011 RLDWA also created exemptions in SDWA Section 1417 from the prohibitions on the use or introduction into commerce of "pipes, pipe fittings, plumbing fittings or fixtures, including backflow preventers, that are used exclusively for non-potable services such as manufacturing, industrial processing, irrigation, outdoor watering, or any other uses where the water is not anticipated to be used for human consumption" (SDWA Section 1417(a)(4)(A)). Also exempt are "toilets, bidets, urinals, fill valves, flushometer valves, tub fillers, shower valves, service saddles, or water distribution main gate valves that are 2 inches in diameter or larger" (SDWA Section 1417(a)(4)(B)).

The Community Fire Safety Act of 2013 further amended SDWA Section 1417 to include fire hydrants in the list of exempted plumbing devices.

In some cases, lead contamination in drinking water has resulted from treatment changes that improve water quality but alter its chemistry, leading to corrosion of lead pipes, solder, and plumbing parts, and subsequent leaching into drinking water supplies (Renner, 2010).

2.2 Overview of Adverse Health Effects Associated with Lead Exposure

The human health effects of lead exposures are well documented and therefore only briefly summarized here. For a comprehensive review of the adverse health effects of lead exposure, the reader is directed to EPA's *Integrated Science Assessment for Lead* (U.S. EPA, 2013), the National Toxicology Program's *Monograph on Health Effects of Low-Level Lead* NTP, National Toxicology Program (NTP), 2012) (National Toxicology Program (NTP) 2012) and the *Toxicological Profile for Lead* of the Agency for Toxic Substances and Disease Registry (ATSDR, 2007).

Lead exposure is associated with a range of adverse health effects in adults and children, including neurological, cardiovascular, and developmental effects. An area of particular attention by public health agencies and organizations nationwide is neurological effects in children that have been associated with BLLs extending below 5 µg/dL; a threshold for cognitive function effects of lead is not discernable from the current evidence (American Academy of Pediatrics Council on Environmental Health, 2016; ATSDR, 2007; Centers for Disease Control and Prevention (CDC) 2016; U.S. EPA, 2013).

Children are especially vulnerable to the neurological effects of lead. EPA, in the *Integrated Science Assessment for Lead* (U.S. EPA, 2013), concluded that lead exposure is causally or likely casually related to many neurodevelopmental endpoints in children including IQ loss; attention problems, impulsivity, and hyperactivity; antisocial behaviors (e.g., aggression, criminal activity); greater internalizing behaviors (e.g., withdrawn behavior, symptoms of depression, fearfulness, and anxiety); and auditory and motor function decrements.

The adverse effects of lead exposures on intelligence have been observed early in development (during fetal, neonatal, and later postnatal periods) and in young children (7 years old or younger) with remarkable consistency across numerous studies involving varying study designs, different developmental assessment protocols, and diverse populations (U.S. EPA, 2013). In an international pooled analysis of seven cohort studies, Lanphear et al. (2005) found the relationship of IQ with children's BLL to be nonlinear, with greater change in IQ across lower as compared to higher BLLs. The log-linear model applied estimated an increase in a child's concurrent BLL from 2.4 µg/dL to 10 µg/dL to be associated with a decrease of approximately 3.8 IQ points.[1] For comparison, an increase in children's concurrent BLLs from 20 µg/dL to 30 µg/dL was only associated with an additional decrease in IQ of approximately 1.1 points (Lanphear et al., 2005).

In addition to deficits in intelligence, Nigg, Nikolus, Knatterus, Cavanaugh, and Friderici (2010) found that attention deficit/hyperactivity disorder (ADHD) diagnosis and symptom incidence were associated with BLLs in children even at levels less than half of the current reference value of 5 µg/dL established by the Centers for Disease Control and Prevention (CDC, 2016). Lead exposure can lead to delayed pubertal onset as well as atopic and inflammatory responses in children (U.S. EPA, 2013). Lead is also associated with adverse reproductive effects including low birth weight, which itself could be related to adverse neurodevelopmental outcomes (Shenkin, Starr, & Deary, 2004; U.S. EPA, 2013). Additional adverse effects on the reproductive system and development include reduced postnatal growth and decreased fertility in males (NTP, 2012; U.S. EPA, 2013).

Exposures to lead in adults have been associated with effects on the cardiovascular system, including increases in hypertension, cardiovascular disease, coronary heart disease, and cardiovascular disease-related mortality (NTP, 2012; U.S. EPA, 2013). In an evaluation of the epidemiological literature on the health effects of low-level lead exposures, NTP found sufficient evidence for an association between BLLs <10 µg/dL in adults and increases in blood pressure and hypertension (NTP, 2012). Evidence of associations between additional cardiovascular impacts and lower BLLs are mixed (NTP, 2012). EPA determined that there is a causal relationship between lead exposures and coronary heart disease endpoints (e.g., myocardial infarction, ischemic heart disease), although the level and timing of lead exposures contributing to these endpoints remains uncertain (U.S. EPA, 2013).

Studies have also suggested a relationship between lead exposures and impairments in kidney function (NTP, 2012; U.S. EPA, 2013). The International Agency for Research on Cancer (IARC) and EPA have classified inorganic lead compounds as probable human carcinogens (IARC, 2006; U.S. EPA, 1988). Based on animal studies of chronic exposures to high concentrations of lead, EPA concluded that there is evidence of a likely causal relationship between lead exposures and cancer

[1] The original Lanphear et al. (2005) publication states the increase to be approximately 3.9 IQ points. Corrections to that analysis were presented in Kirrane and Patel (2014), and the value was calculated to be 3.8 IQ points. These estimates are for BLLs measured at the same time (concurrent) with the IQ test.

(U.S. EPA, 2013). Epidemiological studies in humans have focused primarily on populations of highly exposed workers and have provided inconsistent evidence for the association between lead and cancer (U.S. EPA, 2013).

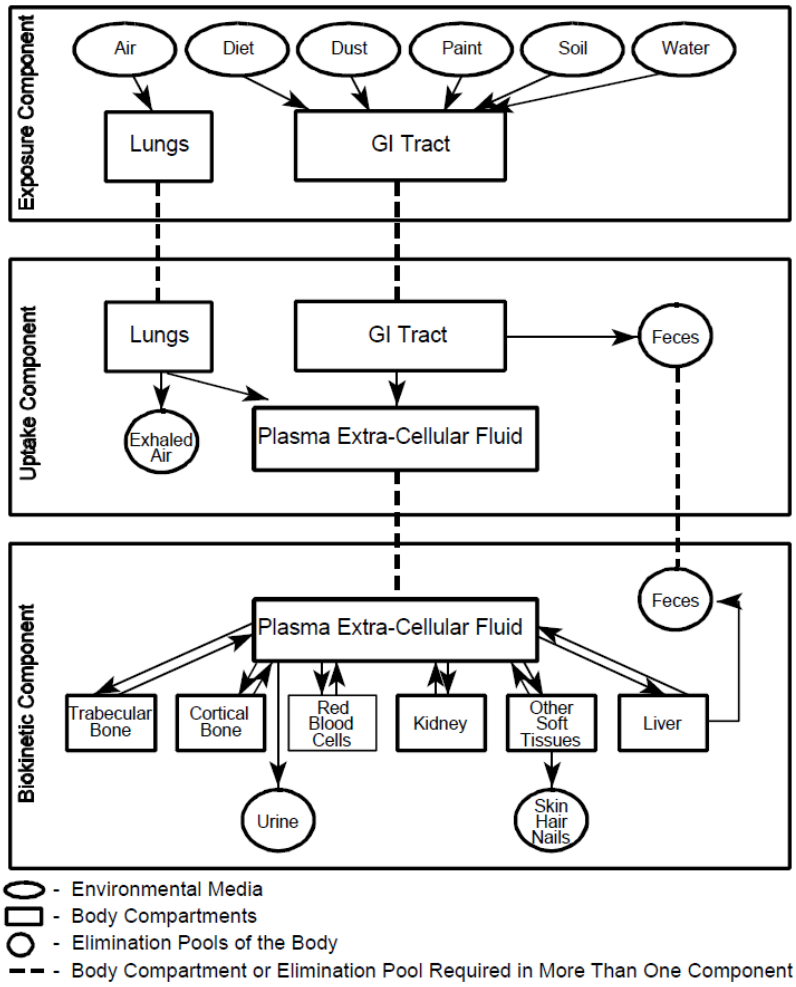
3. Overview of the Integrated Exposure Uptake and Biokinetic Model

The IEUBK model was developed as a simulation tool to predict BLLs in children from birth up to age 7 and to thereby assist in the risk assessment of contaminated sites (U.S. EPA, 1994). The model is intended to "enable rapid calculations and recalculations of an extremely complex set of equations that includes scores of exposure, uptake, and biokinetic parameters" (U.S. EPA, 1994, p. 1-1). An overview of the model is presented in Exhibit 1. The IEUBK model provides an estimate of the BLL for a population of similarly exposed children associated with specified concentrations of lead in media (e.g., water, soil) in the child's environment (U.S. EPA, 2007b). In addition, it estimates the probability that a population of similarly exposed children with a given exposure scenario will have a BLL greater than a specified level. Users can modify inputs and assumptions within the model (e.g., concentrations of lead in environmental media, intake rates for environmental media) to explore the effects on children's BLLs.

The IEUBK model was validated by using measured, residence-specific environmental lead concentrations as inputs and by comparing the predicted BLLs to those measured in children (Hogan, Marcus, Smith, & White, 1998). Hogan et al. (1998) found that predicted geometric mean BLLs were within 0.7 µg/dL of the observed values and that estimates of the proportion of BLLs above 10 µg/dL were within 4 percent of the measured values, indicating reasonable agreement of the model output with the observed data.

In addition to supporting response activities at Superfund sites, the IEUBK model has been used in risk or benefits assessments to support several EPA regulations, including the National Ambient Air Quality Standards for Lead (U.S. EPA, 2008a) and the Steam Electric Power Plant Effluent Guidelines (U.S. EPA, 2015a). Additionally, the model was applied in several peer-reviewed publications to predict site-specific changes in children's BLLs associated with changes in drinking water lead concentrations (Deshommes et al., 2016; Ngueta et al., 2014; Sathyanarayana, Beaudet, Omri, & Karr, 2006; Triantafyllidou, Gallagher, & Edwards, 2014; Triantafyllidou, Le, Gallagher, & Edwards, 2014).

Exhibit 1. Structure of the IEUBK Model for Lead in Children



Source: U.S. EPA (1994).

The IEUBK model uses four main components to mathematically and statistically link environmental lead exposure to children's BLLs: exposure, uptake, biokinetics, and variability (White et al., 1998). Exposures are quantified by combining information on the concentration of lead in environmental media, the amount of contact with the media (e.g., amount of drinking water ingested per day), and the duration of the contact (e.g., number of days) (White et al., 1998). The environmental media included in the IEUBK model are drinking water, soil, household dust, air, and food; exposure to leaded paint is assessed via its contribution to household dust and soil concentrations (White et al., 1998). The uptake component models the transfer of lead to the bloodstream (i.e., the absorption) after intake into the child's body via the inhalation or ingestion routes.

The biokinetic component consists of mathematical modeling of the movement of absorbed lead throughout the child's body, between the internal compartments and to the excretion pathways. This modeling is done with consideration of the volume and weight of specific compartments of a child's body, as a function of age. Lastly, variability in children's BLLs is characterized by use of the lognormal probability distribution in the IEUBK model (White et al., 1998). This accounts for the fact that children exposed to the same concentrations of lead in environmental media may have different

BLLs due to differences in individual behavior, biology, and household characteristics (e.g., frequency of cleaning). For further information on the development of and components within the IEUBK model see White et al.'s (1998) paper.

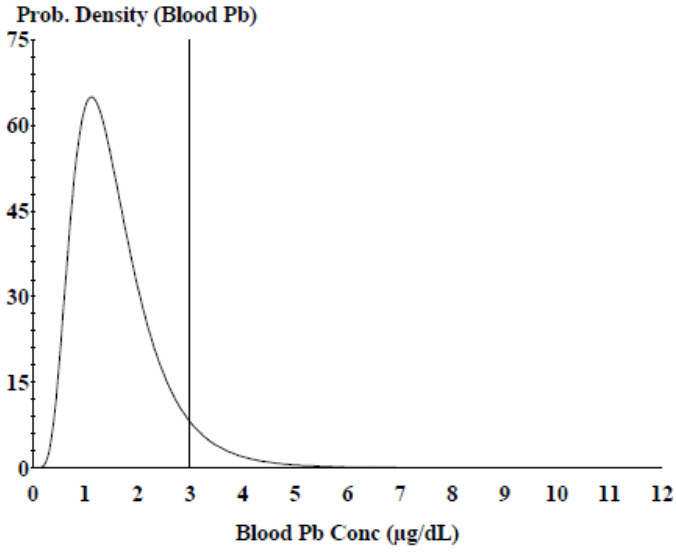
The IEUBK software provides default values for environmental (e.g., lead concentrations in drinking water) and biological parameters (e.g., gastrointestinal absorption coefficient) in the model. These defaults were developed to represent average lead exposures in U.S. children when the model was created during the 1980s and are based on empirical data in the literature (U.S. EPA, 1994). For example, ingestion parameters are based on data sources such as surveys of consumption patterns, laboratory testing of food products available on the U.S. market, and observational studies of soil and dust ingestion in U.S. children (U.S. EPA, 1994). Evidence from animal and human studies was used to derive the absorption and other biokinetic assumptions (U.S. EPA, 1994). However, some variables have been updated periodically since 1994 such as default values for dietary lead

Site- or child-specific information can be entered in place of the IEUBK defaults for air, diet, drinking water, soil/dust, maternal BLLs, alternative source data, contact rates, and gastrointestinal values/bioavailability inputs. For example, drinking water input data in IEUBK consist of mean lead concentration in drinking water (µg/L) and drinking water consumption rates (L/day), averaged per year of age. Each input in the IEUBK model is a point estimate of the parameter in question; variability within each parameter is not considered in the model. The way in which IEUBK accounts for variability in BLLs is through use of the geometric standard deviation (GSD) for BLLs. The default GSD value is based on empirical data and can be modified by the user. Additional information on the more than 100 inputs that may be modified in IEUBK can be found in U.S. EPA's 2007 report "User's Guide for the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK)."

The primary outputs in the IEUBK model are a geometric mean BLL (µg/dL) for a group of children aged 6 to 84 months, and the probability of a child or a population of children having a BLL in excess of a user-specified BLL. Geometric mean BLLs are displayed in tables summarizing mean lead exposures during each year of life. Percentages of children in a specified age range with BLLs in excess of a user-specified level of interest are presented in graphical form, as displayed in Exhibit 2. It is important to note that the probability distribution in Exhibit 2 is based only on the GSD input value, rather than on variability in the parameters used for model calculations. The geometric mean values that are the output of the IEUBK model can be interpreted as the "best estimate of a plausible range of blood lead concentrations for a hypothetical child with a specific lead exposure scenario" (U.S. EPA, 2007b, p. 27). Alternatively, the output can be considered the best estimate for a population of children who have identical exposure scenarios. However, results should not be interpreted as the geometric mean BLL for a community or for identifying the fraction of children in the community who will exceed a specified BLL.

IEUBK contains a batch run option that allows the user to run input data for many locations with different lead concentrations in a single model run, facilitating the estimation of BLLs for groups of children. The batch mode also allows input of child-specific ages and outputs age-specific BLL estimates for a child under each exposure scenario. To further assist in risk assessment of specific sites, the IEUBK model can also output average, media-specific, daily lead uptake rates (in µg/day) and media-specific remediation goals, which provide guidance in determining appropriate soil or dust lead cleanup levels.

Exhibit 2. Sample Graphical Output of the IEUBK Model for Children Aged 12 to 24 Months



Cutoff = 3.000 µg/dl
Geo Mean = 1.456
GSD = 1.600
% Above = 6.197
% Below = 93.803

Age Range = 12 to 24 months

Run Mode = Research

This probability density function is representative of the range of estimated blood lead levels, and shows that approximately 6.2 percent of the population are predicted to have BLLs above 3 µg/dL, given the input parameters for the particular modeling scenario.

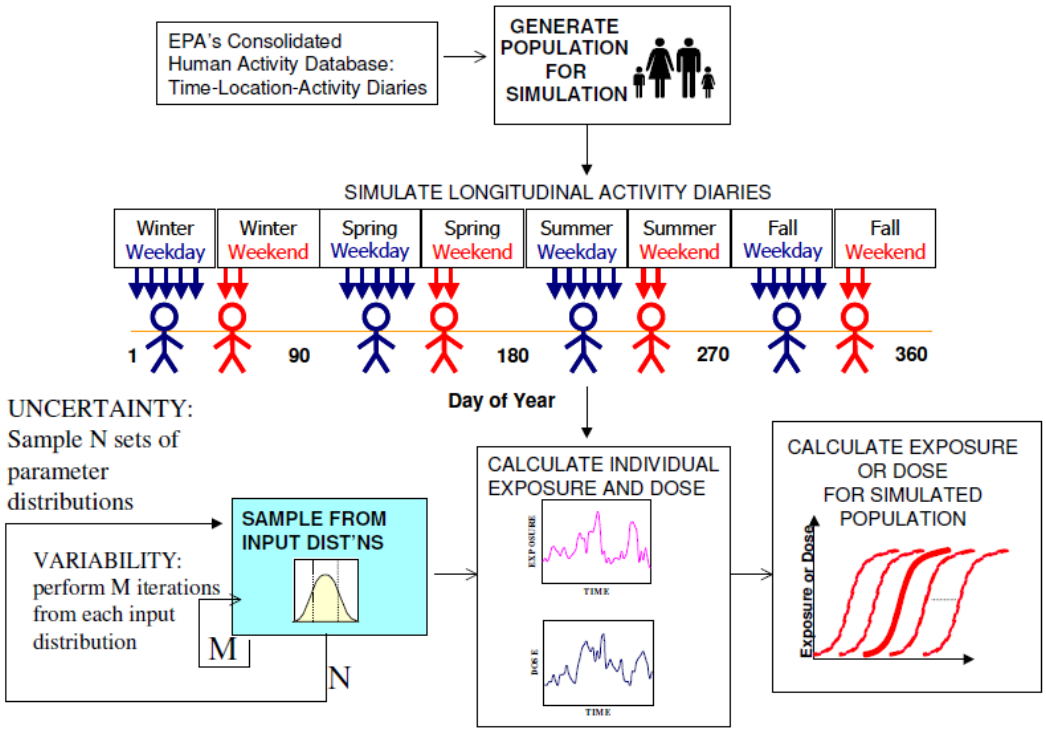
4. Overview of the Stochastic Human Exposure and Dose Simulation Model

One approach to support modeling lead in drinking water couples the IEUBK model with the SHEDS-Multimedia model. EPA's Office of Research and Development created SHEDS-Multimedia to meet the need for new methodologies to assess residential and dietary exposures to chemicals following the enactment of the Food Quality Protection Act in 1996 (Zartarian et al., 2012). The purpose of SHEDS is "to improve the understanding of aggregate and cumulative exposures over space and time for enhanced human health risk assessments involving chemicals" (Zartarian et al., 2012).

SHEDS is a probabilistic computer model that simulates aggregate (multimedia) and cumulative population exposures to chemicals over space and time based on realistic activity patterns, concentration distributions, and exposure factors (e.g., intakes) (Glen, Zartarian, Smith, & Xue, 2012; U.S. EPA, 2016b; Xue, Liu, Zartarian, Geller, & Schultz, 2014; Xue, Zartarian, Liu, & Geller, 2012; Xue, Zartarian, & Nako, 2012; Xue, Zartarian, Tornero-Velez, & Tulve, 2014; Xue, Zartarian, Wang, Liu, & Georgopoulos, 2010; Zartarian et al., 2006, 2012).

A schematic overview of the SHEDS model is provided in Exhibit 3.

Exhibit 3. Overview of SHEDS Methodology



Source: U.S. EPA (2008b).

The model provides exposure estimates as a result of both dietary and residential exposures. This includes assessing exposures due to ingestion, inhalation, and dermal[2] exposures. Model inputs include the concentration of the chemical(s) of interest in various media and human behavior data from EPA's Consolidated Human Activity Database (CHAD) and CDC's National Health and Nutrition Examination Survey (NHANES). It can complement IEUBK by considering human exposures probabilistically. EPA's Office of Research and Development's National Exposure Research Laboratory has put SHEDS-Multimedia through multiple external peer reviews, and published approximately 30 peer-reviewed journal articles on model inputs, approach, case studies, and evaluation. In this analysis, SHEDS-Multimedia is used to provide scientifically defensible values for lead in drinking water. SHEDS-Multimedia code, documentation, and Scientific Advisory Panel materials are available on EPA's public website.

For the current analysis, the inputs to SHEDS pertain to background lead concentrations and children's activity patterns. The output most useful to this report is a distribution of daily exposures to lead (µg/day). Additional information on inputs that may be modified in SHEDS can be found in Xue, Zartarian, & Nako's (2012) report *SHEDS-Dietary version 1 Technical Manual* and Glen et al.'s (2012) report *SHEDS-Residential version 4 Technical Manual.*

Several features of SHEDS contribute to making it a unique and powerful tool. First, since the model uses a time-series approach for simulating dietary and residential exposures, SHEDS accounts for variability that arises from separate activities or eating occasions. The model also uses two-stage Monte Carlo sampling, which allows variability in population exposure and dose estimates and uncertainty associated with different percentiles to be quantified. Another strength of the model is its diary-based approach. Recording sequential dermal hand and body exposures and linking hand-to-mouth ingestion time series with dermal hand exposures account for replenishment and removal processes, such as absorption into the skin or handwashing. SHEDS provides longitudinal diary assembly methods and single and multiple chemical algorithms for assessments, along with multiple methods for sensitivity and uncertainty analyses. In addition, SHEDS-Multimedia can account for correlated inputs. Finally, exposure profiles generated by SHEDS can be paired with physiologically based pharmacokinetic models to improve dose and risk quantification (Zartarian et al., 2012).

Although this is the first time SHEDS-Multimedia has been used specifically for lead exposure, EPA has used SHEDS-Multimedia to assess risks to children posed by wood playsets treated with chromated copper arsenate, estimate cumulative exposures to current-use pesticides, improve risk assessments for chemicals found in foods, and prioritize chemicals for further study on the basis of risk (Tulve et al., 2010; U.S. EPA, 2016b; Xue, Zartarian, Liu, et al., 2012; Xue et al., 2010; Zartarian et al., 2012). Additionally, Xue, Zartarian, et al. (2014) used SHEDS to assess the exposures and doses of 3- to 5-year-old children to seven pyrethroid pesticides in 1-year variability simulations. The results of the model were comparable to biomarker data collected in NHANES, affirming the usefulness of SHEDS in cumulative exposure assessments (Xue, Zartarian, et al., 2014).

[2] The SHEDS residential module includes dermal equations, but for the lead analysis the dermal pathway was not simulated specifically.

5. Inputs Used to Conduct Analyses

The analyses conducted to support modeling lead in drinking water use the IEUBK and SHEDS-Multimedia models. As described in Sections 3 and 4, the IEUBK model contains default exposure parameters that can be modified to reflect the exposure levels for the scenario of interest. Additionally, SHEDS-Multimedia uses individual time-activity diaries from the CDC's NHANES and EPA's CHAD, and related information including body weight, height, and activity level along with modifiable inputs to represent the background media concentrations of the contaminant of interest.

Due to the national scope of this exploratory lead modeling effort, and given the additional exposure modeling possibilities made available with the SHEDS tool, EPA modified several of the default inputs to the IEUBK model. The modified input values were developed from a number of national-scale data sources that were available to the Agency at the time this set of potential modeling methods were being developed. The selection and use of these input values are for illustrative purposes to allow for ease of comparison across model approaches. The purpose of this report is to obtain feedback on various lead modeling methods that can be used to characterize the relationship between lead in drinking water and children's BLLs. The input parameters used in this analysis do not represent high-end exposures.

As described in greater detail in Section 6, three different modeling approaches are outlined in this report to demonstrate potential approaches supporting modeling lead in drinking water. Two of these modeling approaches use IEUBK, and the third uses SHEDS-Multimedia combined with regression equations derived from IEUBK. To ensure consistency of inputs among the three modeling approaches, the same data sources are used for the IEUBK modified input parameters as are used in the SHEDS model. SHEDS inputs were used as the basis for all approaches because some of the exposure inputs are based on the default data underlying the SHEDS-Multimedia model and cannot be modified (e.g., the CHAD or NHANES). Additionally, SHEDS-IEUBK estimates performed well when compared with BLLs reported from NHANES (2013-2014) and from the National Human Exposure Assessment Survey (NHEXAS).[3] Details on the inputs for the SHEDS-IEUBK approach are provided below.

The following section describes the input values used that are different than the IEUBK or SHEDS-Multimedia default values. The sources of the input values for each modeling scenario are outlined in Sections 5.1 to 5.11. Section 5.12 summarizes the exposure inputs used for each model, and for comparison purposes also includes a list of the IEUBK default values that were subsequently modified. For more information on default values for the IEUBK and SHEDS-Multimedia models that were not updated for our analyses, please refer to the technical guidance document of the particular model (U.S. EPA, 1994, 2008b). Given that many of the input parameters are age dependent, a discussion is first presented on the populations evaluated in the modeling exercises before summaries of each of the inputs are presented.

[3] See additional discussion in Section 6.2.1 and Appendix B.

5.1 Population of Interest

Three age groups were selected for assessment in each of the approaches: infants with both average drinking water consumption rates (see Section 6) and those fed formula reconstituted with tap water since birth (see Appendix A), 1- to 2-year-olds with exposures similar to the national average, and 0- to 7-year-olds with exposures similar to the national average throughout their lifetime.[4]

Infants 0 to 6 months old with both average drinking water consumption rates and those fed formula reconstituted with tap water since birth were evaluated because members of this group are likely both susceptible to the effects of lead exposure due to their life stage and at risk of higher exposure if there is lead in their drinking water compared to other children, because of their higher water consumption rates. For infants who are fed with reconstituted infant formula mixed with tap water, tap water is expected to comprise approximately 90 percent of their diet by weight and over 50 percent of their total lead exposures (Triantafyllidou & Edwards, 2012).

Children aged 1-2 with average drinking water consumption rate since birth were also evaluated, based on the evidence of this being an age susceptible to the effects of lead exposure due to life stage, and the potential for higher exposures to lead from non-drinking water sources of lead, if present in these children's environment (i.e., soil and dust) (U.S. EPA, 2013, section 5.2.1.1).

Children aged 0-7 with average drinking water consumption rates were evaluated because this group accounts for individuals potentially being exposed to lead in drinking water throughout their childhood up to age 7. Additionally, this age group represents cumulative exposure during the period over which lead exposure is estimated to have the greatest response in regard to changes in per-unit IQ (Lanphear et al., 2005; Crump et al., 2013), one of several adverse health effects associated with lead exposure (Lanphear et al., 2005; U.S. EPA, 2013, section 3.2).

Given that modeling is conducted for individuals up to age 7, inputs are necessary for each year of life up to 7 years for each age-specific input parameter. Inputs specific to the 0- to 6-month-olds are also needed. Therefore, each age-specific input parameter will have a value for 0- to 6-month-olds, 0- to 1-year-olds and also each subsequent year of life up to age 7.[5]

5.2 Outdoor Air Lead Concentration

The outdoor air lead concentration was set to 0.01 $\mu g/m^3$ for all analyses. This value is based on data presented in a 2013 EPA memorandum that summarizes air lead concentration data from 50 urban sites with populations of more than 500,000 people (Cavender, 2013). Outdoor lead monitoring occurred in these sites on a continuous basis in 2013 to comply with an air monitoring requirement of the National Ambient Air Quality Standards (NAAQS). These non-source-oriented lead monitoring sites provide information about ambient air lead concentrations pertinent to large population

[4] These age groupings are not entirely consistent with EPA's *Guidance on Selecting Age Groups for Monitoring and Assessing Childhood Exposure to Environmental Contaminants* (2005) in order to concentrate on select age groups as described in this section.

[5] While the first population group, infants, is modeled for both average consumption rates (Section 6) and those consuming only formula (Appendix A) from birth through 6 months of age, the early exposure for the other two population groups is not limited to formula. Rather, national average estimates are used for drinking water intake from birth for the other two groups.

exposures in U.S. urban areas (U.S. EPA, 2014).[6] The average of the highest rolling 3-month averages for all 50 of the monitoring sites was 0.01 µg/m^3. This figure was therefore selected as the outdoor air lead concentration for all IEUBK and SHEDS modeling.

5.3 Time Spent Outdoors

Input figures for time spent outdoors were derived from information in the CHAD. The CHAD is a detailed collection of human activity data that is administered by EPA (U.S. EPA, 2009a). The data include exposure and time-use information from 22 different studies performed between 1982 and 2010, which altogether represent more than 54,000 individual study-days of human activity. A summary of the distribution used for time spent outdoors in the SHEDS analysis for each age group is summarized in Exhibit 4.[7] The central estimates for each age group used in the IEUBK analyses are bolded in Exhibit 4.

Exhibit 4. Summary of Time Spent Outdoors Input Data (hr/day)

Age	N[1]	Mean	Std	P50	GM	GSD	P75	P95	P99
0-<6[2] Months	587	0.542	1.193	0.000	0.048	9.705	0.583	2.750	5.333
0-<1 Years	587	0.542	1.193	0.000	0.048	9.705	0.583	2.750	5.333
1-<2 Years	721	1.026	1.552	0.433	0.158	11.906	1.500	4.000	6.583
2-<3 Years	1080	1.633	1.979	1.000	0.281	13.516	2.667	5.458	8.100
3-<4 Years	980	1.743	1.989	1.196	0.332	13.232	2.833	5.725	7.800
4-<5 Years	975	1.851	2.119	1.250	0.360	13.093	3.000	6.250	8.700
5-<6 Years	1012	1.807	2.167	1.183	0.339	13.225	2.750	6.300	8.700
6-<7 Years	1234	1.654	2.162	0.917	0.247	14.347	2.535	6.283	9.750

Source: CHAD.(U.S. EPA, 2009a)

Notes: Bold parameters represent the central estimates used as inputs in analyses that used IEUBK.

1. N represents person-days.

2. The IEUBK analyses includes children up to age 6 months (i.e., children within the first 6 months of life), while the coupled SHEDS-IEUBK analyses include 6-month-old children as well (i.e., children within the first 7 months of life).

[6] Data for source-oriented sites are also summarized in U.S. EPA (2014) and available at https://www.epa.gov/outdoor-air-quality-data.

[7] SHEDS simulates individuals using actual CHAD diaries reporting time spent outdoors to simulate individuals, not the distributions in this table directly.

These input values are slightly higher than EPA's proposed values for time spent outdoors that are presented in the 2011 *Exposure Factors Handbook* (EFH) (U.S. EPA, 2011).[8] Given the need to be consistent between modeling approaches and the fact that the CHAD is explicitly built into the SHEDS model as an underlying data source, it was necessary to use the CHAD for the SHEDS coupled modeling. Therefore, it was also necessary to use the CHAD for the IEUBK modeling to maintain consistency across approaches. The EFH mentions a number of limitations of the CHAD, which mostly arise from the fact that the CHAD combines multiple studies with differing study designs into one dataset. These limitations include differing coding systems that are not easily modified to match; start and end times from particular studies that need to be adjusted to fit a 24-hour time window; over- or under-sampling of cities or states due to entire studies being administered in those places; exclusions of large groups of people including smokers or non-English speakers; and occasional age restrictions of certain surveys, leading to an unequal distribution of ages. These limitations restrict the ability to generalize random individuals in the CHAD to the U.S. population since the sample represented in the CHAD cannot be considered random. In an attempt to overcome this shortcoming, EPA used U.S. Census data to simulate individuals by age and gender to represent the U.S. population, and then linked those individuals with CHAD diaries.

However, considering the low concentration of outdoor air lead being assumed in this analysis, air lead has a very small effect on overall blood lead values. The input for time spent outdoors affects only the inhalation pathway modeling in IEUBK (and not any ingestion pathways). Therefore, the inputs related to exposure to air lead, such as time spent outdoors, are not a significant contributor to the results of the IEUBK analysis. However, in SHEDS the input for time spent outdoors also impacts the soil and dust ingestion rate, as well as soil and dust exposure.

5.4 Inhalation Rate

Input figures for inhalation rate were obtained from the *Exposure Factors Handbook* (U.S. EPA, 2011). The EFH provides recommended long-term exposure values for inhalation rates of 0- to 6-month-olds based on two studies— Arcus-Arth & Blaisdell (2007) and Brochu, Ducré-Robitaille, & Brodeur (2006) —that used large data sets representative of the U.S. population. Both of these studies accounted for the relationship between body weight and inhalation rate. The recommended long-term exposure values for inhalation rates of 1- to 2-year-olds and for those in the 0- to 7-year-old analysis are based on the previously mentioned studies, as well as two additional studies, Stifelman (2007) and U.S. EPA (2009a).

Brochu et al. (2006) derived daily average inhalation rates for a sample population of 2,210 individuals aged 3 weeks to 96 years old based on disappearance rates of oral doses of doubly-labeled water (^2H_2O and $H_2{}^{18}O$) in urine measured with gas-isotope-radio mass spectrometry over an aggregate period of 30,000 days. Meanwhile, Arcus-Arth and Blaisdell (2007) used data adjusted to represent the U.S. population from the U.S. Department of Agriculture's (USDA's) Continuing Survey of Food Intake for Individuals (CSFII) 1994-1996, 1998. CSFII consisted of two non-consecutive 24-hour recall questionnaires administered within a 10-day time period. A combined total of 7,575 participants aged 0 to 7 years were included in the 1994-1996 and 1998 surveys. Daily

[8] All age groups in the analysis of the CHAD had higher values of time spent outdoors than in the EFH, with the exception of the 3- to 4-year-old age group, who had a slightly lower value in the EFH than in the CHAD (a difference of 0.04 hours).

energy intake values were calculated for participants on each of the 2 days they were surveyed. Study authors averaged these energy intake values and used a metabolic conversion method outlined in Layton (1993) to derive daily breathing rates for various age groups.

The two additional studies that were considered for the inhalation rates of 1- to 2-year-old children and the 0- to 7-year-old analysis strengthened the estimate for the inhalation rates. Stifelman (2007) estimated inhalation rates using a large international database of doubly-labeled water energy expenditure administered by the Institute of Medicine using the previously mentioned Layton (1993) method of deriving inhalation rates from energy expenditure. The U.S. EPA (2009b) study attempted to improve upon the Layton (1993) methodology by deriving the inhalation rate from an individual's assumed oxygen consumption rate. This study combined information from the CHAD on metabolic equivalents of work (an energy expenditure metric that represents activity levels) with body-weight, age, and sex data from the 1999-2002 NHANES. This information was used to estimate energy expenditure values for a given individual's likely activity pattern, which was simulated for NHANES participants from CHAD data. These energy expenditure values were then used to estimate oxygen consumption rates, which were inputted into an equation with body-weight, age, and sex data to estimate inhalation rates.

The daily average inhalation rates for long-term exposures for the age groups considered in this analysis are summarized in Exhibit 5. As displayed in this exhibit, the EFH uses more finely tuned age groups than those being considered for our analysis. Therefore, to generate an input specific to 0- to 6-month-olds we took a weighted average approach (with the weight being equal to the number of months in the age group). A rate specific to each age group was presented in the EFH. The values presented in Exhibit 5 were used in both the SHEDS and IEUBK modeling.

Exhibit 5. Summary of Inhalation Rate Input Data (m^3/day)

Age	Inhalation Rate (m^3/day)	Value Used in IEUBK and IEUBK/SHEDS Coupled Analysis (m^3/day)
0 to <1 Months	3.6	3.82 (Weighted Average Inhalation Rate)
1 to <3 Months	3.5	
3 to <6 Months	4.1	
0 to <1 Years	5.4	5.4
1 to <2 Years	8.0	8.0
2 to <3 Years	8.9	8.9
3 to <6 Years	10.1	10.1
6 to <7 Years	12.0	12.0
Source: U.S. EPA (2011, Table 6-1). Bold parameters represent the central estimates used as inputs in analyses that used IEUBK.		

5.5 Drinking Water Ingestion Rate

The mean drinking water ingestion rate was estimated using data from NHANES (CDC & NCHS, 2005). This was done to update the drinking water ingestion rate with more recent data in IEUBK, and also to ensure input data consistency between the IEUBK analyses and the SHEDS analysis.

NHANES is run by the CDC's National Center for Health Statistics (NCHS) and was designed to collect information on the health and nutritional status of the U.S. civilian, non-institutionalized population through in-home interviews and physical examinations. Each survey cycle is 2 years long. NHANES collects demographic (e.g., age, sex), dietary (e.g., 24-hour dietary recall), examination (e.g., body weight, height), laboratory (e.g., biomonitoring of chemicals of interest), questionnaire (e.g., smoking and tobacco use), and limited access data (e.g., geocoding).

NHANES data from the years 2005-2011 were used to estimate a drinking water ingestion rate for all ages up to 7 years. The analysis was based on recalled direct-intake 2-day average drinking water consumption. Distributional information from the NHANES data on daily water consumption rates by age is documented in Exhibit 6. The full empirical distribution is used in the SHEDS analysis (SHEDS simulates individuals using actual NHANES diaries), whereas the geometric mean values are used as the inputs into the analyses using IEUBK.

The selection of the geometric means for use in the two IEUBK-based modeling approaches is due to the lognormal distributions of the input data. Utilizing geometric mean input values differs from the use of arithmetic mean input values which were used in the evaluation of the IEUBK model. The IEUBK model results using geometric mean inputs are specifically intended for the purpose of comparing the utility of the three modeling approaches presented in this report and may not be generally applicable to other analyses. The selection of the geometric mean for exposure rate inputs (water ingestion, soil and dust ingestion, and dietary intake) in this draft report should not be construed as a recommendation for their usage as an input parameter in the IEUBK model.

Exhibit 6. Summary of Daily Water Consumption Rate Input Data (L/day)

Age	N	Mean	Std	P50	GM	GSD	P75	P95	P99
0-6 Months	1246	0.662	0.320	0.630	**0.526**	0.0025	0.854	1.216	1.481
0-1 Years	2618	0.581	0.349	0.532	**0.410**	0.0030	0.806	1.172	1.489
1-2 Years	1792	0.247	0.247	0.219	**0.151**	0.0033	0.306	0.690	1.148
2-3 Years	1948	0.300	0.312	0.251	**0.176**	0.0034	0.360	0.909	1.424
3-4 Years	1272	0.316	0.313	0.257	**0.193**	0.0031	0.398	0.917	1.640
4-5 Years	1358	0.320	0.333	0.261	**0.197**	0.0032	0.404	0.874	1.434
5-6 Years	1196	0.364	0.366	0.303	**0.213**	0.0035	0.447	1.037	1.802
6-7 Years	1306	0.377	0.353	0.332	**0.228**	0.0035	0.480	1.067	1.601

Source: 2005-2011 NHANES (CDC & NCHS, 2005, 2007, 2009, 2011)

Note: Bold parameters represent the central estimates used as inputs in analyses that used IEUBK.

As with the inputs for time spent outdoors, it is recognized that the inputs used in this analysis vary from EPA's proposed values in the 2011 *Exposure Factors Handbook* (U.S. EPA, 2011).[9] As with CHAD, NHANES is explicitly built into the SHEDS model as an underlying data source. Therefore, NHANES needed to be used in both the SHEDS analysis and the IEUBK analyses for this input in order to have consistency across approaches. The EFH recommended against using the information

[9] The inputs derived from NHANES for drinking water consumption rate are higher than those referenced in the EFH for children younger than 1, and are lower than the EFH figures for children age 1 and over.

presented in NHANES for children younger than 3 years of age because of sample size limitations. However, this recommendation was specific to an EPA analysis of daily average water ingestion rates using the 2003-2006 NHANES combined dataset (U.S. EPA, 2010c). The sample size of children younger than 3 years old in the 2005-2011 combined dataset that was used for this analysis is substantially larger than that referenced in the EFH (N=6,358 in this analysis versus N=2,123 in the EFH).

Using the NHANES values for estimating drinking water intake rates poses an additional analysis limitation specifically for formula-fed infants. The NHANES data represent drinking water intake rates for all individuals, not just those who are formula-fed. Given that children younger than 6 months of age drink little to no water directly, it is likely that most, if not all, of their water consumption comes from drinking formula reconstituted with water. The estimates of drinking water intake presented above in Exhibit 6 reflect water intake rates for all infants, including those who are breast-fed, those who are formula-fed, and also those who are both breast-fed and formula-fed. Therefore, these values may underestimate the drinking water intake rates for the potentially most exposed group, infants who are strictly fed formula reconstituted with tap water, due to the inclusion of breast-fed infants. For this reason, additional analysis is presented in Appendix A, which presents results of the IEUBK analyses using a drinking water consumption rate that is specific to formula-fed infants. Therefore, in the remainder of this report the infant population will be referred to as 0- to 6-month-olds. When a drinking water intake rate specific to formula-fed infants is used, the population will be referred to as formula-fed infants.

5.6 Soil Lead

Soil lead concentration values were estimated using data from the American Healthy Homes Survey (AHHS) (U.S. Department of Housing and Urban Development (HUD) 2011). AHHS is a nationally representative survey that was administered by HUD from June 2005 through March 2006 to measure levels of lead, lead hazards, allergens, arsenic, pesticides, and mold in homes. Demographic data were collected by a trained interviewer, and environmental samples were collected by a technician certified as a lead-based paint inspector/risk assessor from a probabilistic sample of permanently occupied, non-institutional housing units in the United States where children may have lived. In total, 942 housing units were sampled for soil lead. AHHS documentation mentions that soil samples were collected only for units where residents had use of a yard or patio that had soil.

In conducting the SHEDS-IEUBK analysis for soil lead concentration, an empirical distribution was fit to data provided by HUD from AHHS (HUD, 2011). The data were stratified and weighted by house age pre- and post-1950, and a correlation coefficient of 0.48 between dust and soil lead concentrations was assigned in SHEDS (see Exhibit 7). For the modeling approaches using IEUBK alone, all housing units were grouped together to calculate the geometric mean soil value, 37 µg/g. Additionally, given that soil lead is highly variable across the United States, the analysis is also conducted using the arithmetic mean estimate of soil lead of 160 µg/g.

Further, to specifically evaluate homes that may have soil and dust contamination due to lead paint, an analysis is presented in Appendix A that uses input parameters for soil and dust lead that are specific to homes built prior to 1950. Based on historical information on lead-based paint regulations, the year 1950 was selected as a cut point to identify homes with likely sources of lead-based paint. In 1951, the city of Baltimore, Maryland, became the first in the United States to ban the use of lead-based paint within homes (Steering Committee of the 2008 National Healthy Homes Conference,

2008). Although a nationwide ban on the use of lead-based paint in homes was not enacted until 1978, there was a decline in the use of interior lead-based paint throughout the United States during the 1950s and 1960s (CDC, 1991). Using data from homes constructed prior to 1950, rather than 1977, may thus better represent only homes with lead-based paint. Additionally, a recent large-scale analysis of BLLs in U.S. children by McClure, Niles, and Kaufman (2016) found that pre-1950 housing was a risk factor for high childhood BLLs. Children living in areas with a large proportion (≥ 51%) of homes built before 1950 had significantly higher proportions of BLLs above 5.0 µg/dL (OR: 5.86, 95% CI 5.71-6.01) or 10.0 µg/dL (OR: 6.34, 95% CI 5.97-6.74) than children living in areas with the lowest proportion (< 3.6%) of pre-1950s housing.

Because the soil lead concentration term is highly variable and an influential input variable in the IEUBK mode, the results obtained using 160 µg/g as the input would not necessarily be protective of children residing at properties with higher soil lead concentrations, such as those living in urban areas.

Exhibit 7. Summary of Soil Lead Input Data Used in SHEDS-IEUBK Analysis (µg/g)

Housing Age	N	Mean	Std	P5	P50	GM	GSD	P75	P95	P99
Data Used for IEUBK Alone Approaches										
All	942	160	488	6	27	37	5	87	810	2,130
Data Used for SHEDS-IEUBK Approach										
Before 1950	193	532.2	912.6	26.58	203.2	221.1	3.89	574.5	1841.3	5792.7
After 1950	749	63.7	202.0	4.9	19.2	23.0	3.37	39.9	207.7	933.3

Source: 2005-2006 AHHS (HUD, 2011)

Note: Bold parameters represent the central estimates used as inputs in analyses that used IEUBK.

5.7 Dust Lead

Dust lead concentration inputs were also estimated using data from AHHS, which was described in the preceding section (HUD, 2011). Dust lead information was obtained for AHHS by certified technicians taking floor dust wipe samples in housing units. Of the 5,612 floor dust wipe samples taken during the survey, only 404 were above the detection limit of 5 µg/ft^2. However, raw analytical data files were obtained by HUD from the laboratory processing the floor wipes, which included some of the samples below the level of detection, resulting in 1,131 dust wipe samples with data available to calculate dust lead levels. These additional data points were used by HUD in the calculation of the mean values for dust lead from floor wipes. According to HUD, "this procedure provides unbiased estimates of means, provided that measurements below the detection limit are normally distributed about the true value of the analyte, as is generally assumed in discussions of the detection limit" (HUD, 2011, p. 43). The remaining samples below the level of detection were not used in this analysis.

Dust wipe samples were measured in AHHS as dust lead loading levels (µg/ft^2), rather than dust lead concentrations (µg/g), which is the figure appropriate for input into the IEUBK model. In order to convert dust lead loading levels to dust lead concentrations, EPA used an empirical method that was introduced in EPA's 2007 *Lead: Human Exposure and Health Risk Assessments for Selected Case*

Studies (U.S. EPA, 2007a). This method was developed using indoor dust lead loading and concentration data that were collected as part of the HUD National Survey of Lead-Based Paint in Housing (U.S. EPA, 1995), and were subsequently put into a regression equation to understand the relationship. The equation estimated for the conversion of loading to concentration is as follows:

$$\ln(PbCONC) = 4.92 + 0.52 \times \ln(PbVAC)$$

where:

PbCONC = indoor dust concentration (µg/g)

PbVAC = vacuum indoor dust lead loading (µg/ft^2)

Vacuum indoor dust lead loading is estimated from indoor wipe lead loading levels using the following equation:

$$PbVAC = 0.185 \times PbWIPE$$

where:

PbVAC = vacuum indoor dust lead loading (µg/ft^2)

PbWIPE = indoor wipe lead loading (µg/ft^2)

Once a figure for indoor lead dust concentration was estimated from the reported indoor wipe lead loading level, EPA fit an empirical distribution to the data in order to estimate a central estimate of dust lead (see Exhibit 8). In the IEUBK-alone approaches, based on the shape of the distribution the geometric mean, 72 µg/g, is used as one of the central estimates for the dust lead input into the models. Given that dust lead concentration is assumed to be correlated with soil lead concentration and that both tend to be highly variable in different areas of the United States, the arithmetic mean dust lead estimate of 104 µg/g is used in analyses when the arithmetic mean soil lead estimate is used. Further, to specifically evaluate homes that may have soil and dust contamination due to lead paint, an analysis is presented in Appendix A, which uses soil and dust lead input parameters specific to homes built prior to 1950.

In conducting the SHEDS-IEUBK approach 3 analysis for soil lead concentration, an empirical distribution was fit to data provided by HUD from AHHS (HUD, 2011). The data were stratified and weighted by house age pre- and post-1950, and a correlation coefficient of 0.48 between dust and soil lead concentrations was assigned in SHEDS. A correlation coefficient of 0.2 was also applied to the water and dust concentration distributions, based on EPA's NHEXAS study Clayton, Pellizzari, Whitmore, Perritt, and Quackenboss (1999)

Exhibit 8. Summary of Dust Lead Input Data (µg/g)

Housing Age	N	Mean	Std	P5	P50	GM	GSD	P75	P95	P99
Data Used for IEUBK Alone Approaches										
All	1,131	104	136	19	70	72	2	104	287	825
Data Used for SHEDS-IEUBK Approach										
Before 1950	223	207.7	238.2	33.4	113.3	133.9	2.47	238.6	706.6	1108.9
After 1950	908	79.0	77.2	18.9	64.5	61.3	2.00	87.1	195.3	353.1

Source: 2005-2006 AHHS (HUD, 2011)

Note: Bold parameters represent the central estimates used as inputs in analyses that used IEUBK.

5.8 Soil and Dust Ingestion Rate

The soil/dust ingestion rate for children is a key input to which model results are highly sensitive, and for which data are limited and uncertain, especially for children <2 years; for older ages, values are similar between Ozkaynak, Xue, Zartarian, Glen, and Smith. (2011) and von Lindern, Spalinger, Stifelman, Stanek, and Bartrem (2016) developed using different methodologies. It is important to note that there are multiple approaches for estimating soil/dust ingestion rate as detailed in U.S. EPA (2011). To estimate the soil and dust ingestion rate distributions by age, the Ozkaynaket al. (2011) study approach was used. This was done to derive a distribution of soil and dust ingestion inputs since a distribution, as opposed to a point estimate, is needed for the SHEDS analysis for each age group considered. For additional information about the model methodology, please refer to the Ozkaynak et al. (2011) study. Results from the model are presented in Exhibit 9. Zero- to 6-month-olds and 0- to 1-year-olds were assumed to have the same soil and dust ingestion rate as 1-year-olds due to lack of data. In conducting the SHEDS analysis, the full distribution of soil and dust ingestion rates was used for each age group simulated.

The geometric mean of the Ozkaynak et al. (2011) total soil and dust ingestion data (listed in bold in Exhibit 9) was selected for use in the two IEUBK-based modeling approaches because the overall shape of the ingestion data distribution was lognormal. See the discussion of the use of geometric mean input values in Section 5.5. It is important to note that the selection of input values in the case of soil and dust ingestion rate can have a significant impact on IEUBK model results.

Exhibit 9. Summary of Soil and Dust Ingestion Rate Input Data (mg/day)

Age	Soil/Dust	Mean	Std	50th	GM	GSD	95th	97.5th	99th
0-6 Months	mg_total	43.9	54.8	27.8	26.6	2.8	135	188	262
0-1 Years	mg_total	43.9	54.8	27.8	26.6	2.8	135	188	262
1-2 Years	mg_total	43.9	54.8	27.8	26.6	2.8	135	188	262
2-3 Years	mg_total	45.2	58.8	25.8	25.9	3.0	146	201	276
3-4 Years	mg_total	51.7	64.2	31.1	28.9	3.2	168	220	304
4-5 Years	mg_total	57.8	75.5	34.0	31.6	3.2	197	268	364
5-6 Years	mg_total	62.6	79.8	37.9	34.4	3.2	204	270	380
6-7 Years	mg_total	54.3	76.1	30.4	29.2	3.2	183	252	357

Source: Ozkaynak et al. (2011).

Note: Bold parameters represent the central estimates used as inputs in analyses that used IEUBK.

The geometric mean of 26.6 mg/day used in approaches 1 and 2 is less than the recommended EPA *Exposure Factors Handbook* (2011) "central tendency"[10] values of 60 mg/day for individuals <1 year old and 100 mg/day for individuals between 1 and 21 years of age. A sensitivity analysis was conducted using the EFH central tendency value of 100 mg/day (by scaling the Ozkaynak et al. (2011) modeled distribution for the baseline runs). It was found in doing this that estimated BLLs were much higher than national averages, and therefore the input value of 100 mg/day was assumed to be too high for this analysis.

5.9 Dietary Lead

The dietary lead intake rates used were estimated based on lead in food data from the U.S. Food and Drug Administration (FDA) Total Diet Study (TDS) ,(FDA, 2007, 2008, 2009, 2010, 2011, 2012, 2013) and also from food ingestion data from NHANES. A detailed description of NHANES is provided in Section 5.5.

TDS is an ongoing FDA program that collects information on levels of various contaminants, including lead, that occur in food and beverages commonly consumed by the U.S. population. To estimate the levels of contaminants in these food products, the FDA buys these foods as a consumer would, prepares them as directed,[11] and then analyzes the prepared foods for levels of the contaminants of interest. This process yields nationally representative estimates of contaminant levels in more than 280 kinds of food and beverages.

To estimate the daily dietary lead intake for children and infants in SHEDS-IEUBK analyses, TDS data from 2007 to 2013 for lead levels in food and beverages were matched with 24-hour dietary consumption data collected in NHANES using FDA's file for mapping food items from TDS to NHANES in SHEDS-Multimedia (FDA, 2007, 2008, 2009, 2010, 2011, 2012, 2013).

[10] The EFH does not specify whether the reported central tendency estimate is the arithmetic mean or the geometric mean.

[11] FDA prepares its samples using deionized water.

The published Xue et al. (2010) method for modeling lead data under the limit of detection was used. According to the Xue et al. (2010) paper, "assignment of [lead] residues for non-detect values depended on the commodity: if there was at least one detection, half the limit of detection was assigned; if no [lead] values were detected, zero values were assigned." Higher limits of detection in particular commodities could cause bias in overall SHEDS-IEUBK results, especially at lower percentiles; however, we do not think this will impact results at higher percentiles. The distribution of daily dietary lead intake for each age group of interest is listed in Exhibit 10. There are several methods for developing lead in food estimates (Parsons, Munno, Buckamn, Tooze, & Dodd, 2009; Tooze et al., 2006; Xue et al., 2010). Therefore, there is uncertainty in the true distribution of the amount of lead consumed daily from food.

In conducting the SHEDS analysis, the full distribution of daily dietary lead intake rates was used. Based on the lognormal shape of the dietary intake rate distribution, the geometric mean of the distribution (listed in Exhibit 10 was used in the IEUBK approaches. See the discussion of the use of geometric mean input values in Section 5.5.

Exhibit 10. Summary of Daily Dietary Lead Intake Input Data (µg/day)

Age	N	Mean	Std	50th	GM	GSD	75th	95th	99th
0-6 Months	1072	0.7	0.98	0.3	**0.27**	4.75	0.91	2.71	3.47
0-1 Years	2674	1.27	1.31	0.87	**0.65**	4.12	1.87	3.88	5.59
1-2 Years	2226	2.58	1.84	2.17	**2.00**	2.16	3.41	5.83	7.63
2-3 Years	1788	3.44	2.03	3.06	**2.85**	1.94	4.49	7.23	8.46
3-4 Years	1160	3.54	2.06	3.18	**2.98**	1.89	4.63	7.26	8.43
4-5 Years	1240	3.57	2.16	3.18	**3.00**	1.87	4.55	7.25	8.63
5-6 Years	1066	3.85	2.18	3.43	**3.31**	1.77	4.83	7.86	9.52
6-7 Years	1086	3.80	2.02	3.51	**3.29**	1.76	4.84	7.55	8.30

Source: 2005-2012 NHANES (CDC & NCHS, 2005, 2007, 2009, 2011); 2007-2013 FDA TDS (FDA, 2007, 2008, 2009, 2010, 2011, 2012, 2013); Xue et al. (2010).

Note: Bold parameters represent the central estimates used as inputs in analyses that used IEUBK.

5.10 Maternal Blood Lead

The mean BLL for women of childbearing age was estimated using data from NHANES. A detailed description of NHANES is provided in Section 5.5. The two most recent NHANES survey cycles for which data are available were used (2011-2012 and 2013-2014). In the 2011-2012 data, all survey participants aged 1 year and older were eligible for the blood lead data file, while in 2013-2014, blood lead data were collected for all participants aged 1-11 years old, and for a one-half subsample of participants aged 12 years and older. Subjects' age and sex were also collected and were available in the NHANES demographic files. EPA pooled these data files across the two survey cycles to create a 4-year dataset. The NHANES Analytic and Reporting Guidelines (Johnson, Paulose-Ram, & Ogden, 2013) were followed to adjust the survey weights for each sample, and EPA generated estimates that are representative of the U.S. civilian, non-institutionalized population at the midpoint of those

4 years. For women of childbearing age (i.e., 18-45 years, N=2,003), a geometric mean BLL of 0.61 (SE = 1.02) µg/dL was estimated. This input was used in the analysis using IEUBK.[12]

5.11 Geometric Standard Deviation

Given that EPA generally encourages use of the model default GSD of 1.6 without modification for most environmental assessments (White, 2004) the default GSD of 1.6 was used when running IEUBK analyses on 1- to 2-year-olds and for all age groups in the 0- to 7-year-old analyses. According to White et al. "the recommended default GSD for the IEUBK model is 1.6, which is intended as a broadly applicable, not a conservative, value" (1998, p. 1525).

However, when considering formula-fed infants it is plausible that variability in dietary lead intake rates could be lower than for other pathways and infants may have fewer concurrent sources of lead exposure as compared with other age groups (White, 2004). Additionally, as there are empirical data available on the variability of BLLs for formula-fed infants, EPA previously made the decision to estimate an infant-specific GSD. The calculated GSD for 0- to 6-month-olds is 1.45, and this value was used in this analysis. The 1.45 value was originally presented in a 2004 EPA memorandum on the risks of elevated blood lead for infants drinking formula prepared with tap water (White, 2004) and has since been used in the published literature (Triantafyllidou, Gallagher, et al., 2014). This infant-specific GSD is based on data on the variance in BLLs of formula-fed infants in two studies, Ryu, Ziegler, Nelson, & Fomon (1983) and Lacey, Moore, & Richards (1985).

Ryu et al. (1983) reported group means and standard deviations of blood lead for infants drinking commercially marketed formulas. The study provided a group of newborns with ready-to-feed formula from glass bottles when the participants were 8 days old through age 111 days. From age 112 days to 195 days, half of the newborns were subsequently fed cow's milk from cartons, and the other half of the newborns were fed cow's milk or formula from cans known to contain lead.[13] The EPA memorandum used the group of infants who drank canned formula as the basis for calculating their GSDs from the Ryu et al. (1983) study, as this formula had higher lead content than the milk in cartons (70 µg/L versus 10 µg/L), and EPA determined that the group receiving formula[14] with 70 µg/L of lead was the best indicator of the variability of lead exposure for those infants with significant exposure to lead in formula. Exhibit 11 summarizes results from the group of infants who drank canned formula or milk in the Ryu et al. (1983) study, with the "Calculated CV" column representing the coefficient of variation calculated by EPA with an assumption of a lognormal distribution:

[12] The default value of 1 µg/dL blood lead was used when developing the regression equations used to convert SHEDS lead uptake values to BLLs. For more details, see Section 6.2.1.

[13] "Infants were fed milk or formula of three types as described in detail previously: (1) homogenized whole cow milk obtained in cartons from a local dairy, (2) a commercially available milk-based formula supplied in quart cans, and (3) homogenized whole cow milk supplied in quart cans and heat treated in the same manner as the commercially available formula." (Ryu et al., 1983, p.888)

[14] Seven children received formula from cans; 10 children received milk from cartons,

Exhibit 11. Results from Ryu et al. (1983)

Days of Age	Mean Blood Lead	SD	Calculated CV
140	9.3	4	0.43
168	12.1	4	0.333
196	14.4	4.4	0.306
Source: Ryu et al. (1983).			

The calculated GSDs of the three groups above, assuming a lognormal distribution, were 1.51, 1.38, and 1.35, which yielded a pooled estimate of the overall GSD of 1.41.

Lacey et al. (1985) reported information (in the form of a graph) on the variability of blood lead for infants drinking formula that had been reconstituted with lead-containing tap water. EPA analyzed the data from the graph by first dividing the data with lead concentrations less than 200 μg/L into eight concentration range groups with widths of 25 μg/L each. According to the White (2004) memorandum, "the range of data up to 200 μg/L was selected for analysis here as both more relevant to most concerns about drinking water contamination, and as that part of data set in which sampling points were more densely grouped to allow for stratification into concentration bins" (White, 2004, p. 10). Exhibit 12 comes from the EPA estimation of results from the Lacey et al. (1985) study.

Exhibit 12. Results from Lacey, Moore, & Richards (1985)

Water Lead Concentration Range (μg/L)	Blood Lead Data	
	Number of Points	Log Scale Variance
0-25	8	0.468
25-50	16	0.184
50-75	11	0.116
75-100	17	0.112
100-125	7	0.249
125-150	5	0.082
150-175	3	0.014
175-200	5	0.086
Source: Lacey, Moore, & Richards (1985).		

A pooled estimate of the GSD from these data was determined to be 1.52.

Based on the results from both the Ryu et al. (1983) and the Lacey et al. (1985) studies, EPA deemed the value of 1.45 to be a reasonably representative GSD estimate for BLL variability in infants consuming formula containing lead. Based on this prior EPA conclusion, a GSD of 1.45 was used in all IEUBK analyses for infants 0 to 6 months old.

The SHEDS-IEUBK coupled analysis does not apply a GSD. Additional discussion on this topic is presented in Section 0.

5.12 Summary of Inputs for Analyses

A summary of the inputs used in the analyses for modeling lead in drinking water that vary from IEUBK current defaults are presented in Exhibit 13 through Exhibit 20 for each age group of interest. Information on the default values that were not changed can be located in the IEUBK technical documentation (U.S. EPA, 1994). Also see Section 6, Exhibit 34, for IEUBK default absorption fractions by media.

Exhibit 13. Inputs Used for IEUBK for 0- to 6-Month-Olds

Variable	Input for IEUBK Analyses		Inputs for IEUBK-SHEDS Coupled Analysis	Data Source	Default IEUBK Input Value[1]
Time Spent Outdoors (h/day)	0.54		Empirical distribution – see Exhibit 4	CHAD (U.S. EPA, 2009a)	1
Inhalation Rate (m^3/day)	3.82		Empirical distribution – see Exhibit 5	EFH (U.S. EPA, 2011)	2
Soil/Dust Ingestion (g/day)	0.027		Empirical distribution – see Exhibit 9	Ozkaynak et al. (2011)	0.085
Mean Drinking Water Rate (L/day)	0.526		Empirical distribution – see Exhibit 6	2005-2011 NHANES; (CDC & NCHS, 2005, 2007, 2009, 2011)	0.2
Air Lead (µg/m^3)	0.01		0.01	Office of Air and Radiation recommendation based on NAAQS monitoring (Cavender, 2013)	0.1
Soil Lead (mg/kg)	37	160	Empirical distribution – see Exhibit 7	2005-2006 American Healthy Homes Survey (HUD, 2011)	200
Dust Lead (mg/kg)	72	104	Empirical distribution – see Exhibit 8	2005-2006 American Healthy Homes Survey (HUD, 2011)	150
Diet Lead (µg/day)	0.27		Empirical distribution – see Exhibit 10	2005-2012 NHANES (CDC & NCHS, 2005, 2007, 2009, 2011); 2007-2013 FDA TDS (FDA, 2007, 2008, 2009, 2010, 2011, 2012, 2013); Xue et al. (2010).	2.26
Maternal (µg/dl)	0.61		None	2011-2014 NHANES (CDC & NCHS, 2011, 2013)	1
Geometric Standard Deviation	1.45		N/A	Analysis of Ryu et al. (1983) and Lacey et al. (1985)	1.60

[1] IEUBK defaults are based on 1-year age ranges, and subsequently there are not default values for 0- to 6-month-olds. Therefore, the values for 0- to 1-year-olds are presented in this table for comparative purposes.

Exhibit 14. Inputs Used for IEUBK for Children 0 to 12 Months Old

Variable	Input for IEUBK Analyses		Inputs for IEUBK-SHEDS Coupled Analysis	Data Source	Default IEUBK Input Value
Time Spent Outdoors (h/day)	1.03		Empirical distribution – see Exhibit 4	CHAD (U.S. EPA, 2009a)	1
Inhalation Rate (m^3/day)	5.4		Empirical distribution – see Exhibit 5	EFH (U.S. EPA, 2011)	2
Soil/Dust Ingestion (g/day)	0.027		Empirical distribution – see Exhibit 9	Ozkaynak et al. (2011)	0.085
Mean Drinking Water Rate (L/day)	0.410		Empirical distribution – see Exhibit 6	2005-2011 NHANES; (CDC & NCHS, 2005, 2007, 2009, 2011)	0.2
Air Lead (μg/m^3)	0.01		0.01	Office of Air and Radiation recommendation based on NAAQS monitoring (Cavender, 2013)	0.1
Soil Lead (mg/kg)	37	160	Empirical distribution – see Exhibit 7	2005-2006 American Healthy Homes Survey (HUD, 2011)	200
Dust Lead (mg/kg)	72	104	Empirical distribution – see Exhibit 8	2005-2006 American Healthy Homes Survey (HUD, 2011)	150
Diet Lead (μg/day)	0.65		Empirical distribution – see Exhibit 10	2005-2012 NHANES (CDC & NCHS, 2005, 2007, 2009, 2011); 2007-2013 FDA TDS (FDA, 2007, 2008, 2009, 2010, 2011, 2012, 2013); Xue et al. (2010).	2.26
Maternal (μg/dl)	0.61		None	2011-2014 NHANES (CDC & NCHS, 2011, 2013)	1
Geometric Standard Deviation	1.60		N/A	IEUBK default	1.60

Exhibit 15. Inputs for IEUBK for Children 1 to 2 Years Old

Variable	Input for IEUBK Analyses		Inputs for IEUBK-SHEDS Coupled Analysis	Data Source	Default IEUBK Input Value
Time Spent Outdoors (h/day)	1.03		Empirical distribution – see Exhibit 4	CHAD (U.S. EPA, 2009a)	2
Inhalation Rate (m^3/day)	8.00		Empirical distribution – see Exhibit 5	EFH (U.S. EPA, 2011)	3
Soil/Dust Ingestion (g/day)	0.027		Empirical distribution – see Exhibit 9	Ozkaynak et al. (2011)	0.135
Mean Drinking Water Rate (L/day)	0.151		Empirical distribution – see Exhibit 6	2005-2011 NHANES; (CDC & NCHS, 2005, 2007, 2009, 2011)	0.5
Air Lead (μg/m^3)	0.01		0.01	Office of Air and Radiation recommendation based on NAAQS monitoring (Cavender, 2013)	0.1
Soil Lead (mg/kg)	37	160	Empirical distribution – see Exhibit 7	2005-2006 American Healthy Homes Survey (HUD, 2011)	200
Dust Lead (mg/kg)	72	104	Empirical distribution – see Exhibit 8	2005-2006 American Healthy Homes Survey (HUD, 2011)	150
Diet Lead (μg/day)	2.00		Empirical distribution – see Exhibit 10	2005-2012 NHANES (CDC & NCHS, 2005, 2007, 2009, 2011); 2007-2013 FDA TDS (FDA, 2007, 2008, 2009, 2010, 2011, 2012, 2013); Xue et al. (2010).	1.96
Maternal (μg/dl)	0.61		None	2011-2014 NHANES (CDC & NCHS, 2011, 2013)	1
Geometric Standard Deviation	1.60		N/A	IEUBK default	1.60

Exhibit 16. Inputs Used for IEUBK for Children 2 to 3 Years Old

Variable	Input for IEUBK Analyses		Inputs for IEUBK-SHEDS Coupled Analysis	Data Source	Default IEUBK Input Value
Time Spent Outdoors (h/day)	1.63		Empirical distribution – see Exhibit 4	CHAD (U.S. EPA, 2009a)	3
Inhalation Rate (m^3/day)	3.82		Empirical distribution – see Exhibit 5	EFH (U.S. EPA, 2011)	5
Soil/Dust Ingestion (g/day)	0.026		Empirical distribution – see Exhibit 9	Ozkaynak et al. (2011)	0.135
Mean Drinking Water Rate (L/day)	0.176		Empirical distribution – see Exhibit 6	2005-2011 NHANES; (CDC & NCHS, 2005, 2007, 2009, 2011)	0.52
Air Lead (µg/m^3)	0.01		0.01	Office of Air and Radiation recommendation based on NAAQS monitoring (Cavender, 2013)	0.1
Soil Lead (mg/kg)	37	160	Empirical distribution – see Exhibit 7	2005-2006 American Healthy Homes Survey (HUD, 2011)	200
Dust Lead (mg/kg)	72	104	Empirical distribution – see Exhibit 8	2005-2006 American Healthy Homes Survey (HUD, 2011)	150
Diet Lead (µg/day)	2.85		Empirical distribution – see Exhibit 10	2005-2012 NHANES (CDC & NCHS, 2005, 2007, 2009, 2011); 2007-2013 FDA TDS (FDA, 2007, 2008, 2009, 2010, 2011, 2012, 2013); Xue et al. (2010).	2.13
Maternal (µg/dl)	0.61		None	2011-2014 NHANES (CDC & NCHS, 2011, 2013)	1
Geometric Standard Deviation	1.60		N/A	IEUBK default	1.60

Exhibit 17. Inputs Used for IEUBK for Children 3 to 4 Years Old

Variable	Input for IEUBK Analyses		Inputs for IEUBK-SHEDS Coupled Analysis	Data Source	Default IEUBK Input Value
Time Spent Outdoors (h/day)	1.74		Empirical distribution – see Exhibit 4	CHAD (U.S. EPA, 2009a)	4
Inhalation Rate (m^3/day)	3.82		Empirical distribution – see Exhibit 5	EFH (U.S. EPA, 2011)	5
Soil/Dust Ingestion (g/day)	0.029		Empirical distribution – see Exhibit 9	Ozkaynak et al. (2011)	0.135
Mean Drinking Water Rate (L/day)	0.193		Empirical distribution – see Exhibit 6	2005-2011 NHANES; (CDC & NCHS, 2005, 2007, 2009, 2011)	0.53
Air Lead (µg/m^3)	0.01		0.01	Office of Air and Radiation recommendation based on NAAQS monitoring (Cavender, 2013)	0.1
Soil Lead (mg/kg)	37	160	Empirical distribution – see Exhibit 7	2005-2006 American Healthy Homes Survey (HUD, 2011)	200
Dust Lead (mg/kg)	72	104	Empirical distribution – see Exhibit 8	2005-2006 American Healthy Homes Survey (HUD, 2011)	150
Diet Lead (µg/day)	2.98		Empirical distribution – see Exhibit 10	2005-2012 NHANES (CDC & NCHS, 2005, 2007, 2009, 2011); 2007-2013 FDA TDS (FDA, 2007, 2008, 2009, 2010, 2011, 2012, 2013); Xue et al. (2010).	2.04
Maternal (µg/dl)	0.61		None	2005-2012 NHANES (CDC & NCHS, 2005, 2007, 2009, 2011); 2007-2013 FDA TDS (FDA, 2007, 2008, 2009, 2010, 2011, 2012, 2013); Xue et al. (2010).	1
Geometric Standard Deviation	1.60		N/A	IEUBK default	1.60

Exhibit 18. Inputs Used for IEUBK for Children 4 to 5 Years Old

Variable	Input for IEUBK Analyses		Inputs for IEUBK-SHEDS Coupled Analysis	Data Source	Default IEUBK Input Value
Time Spent Outdoors (h/day)	1.85		Empirical distribution – see Exhibit 4	CHAD (U.S. EPA, 2009a)	4
Inhalation Rate (m^3/day)	3.82		Empirical distribution – see Exhibit 5	EFH (U.S. EPA, 2011)	5
Soil/Dust Ingestion (g/day)	0.032		Empirical distribution – see Exhibit 9	Ozkaynak et al. (2011)	0.100
Mean Drinking Water Rate (L/day)	0.197		Empirical distribution – see Exhibit 6	2005-2011 NHANES; (CDC & NCHS, 2005, 2007, 2009, 2011)	0.55
Air Lead (μg/m^3)	0.01		0.01	Office of Air and Radiation recommendation based on NAAQS monitoring (Cavender, 2013)	0.1
Soil Lead (mg/kg)	37	160	Empirical distribution – see Exhibit 7	2005-2006 American Healthy Homes Survey (HUD, 2011)	200
Dust Lead (mg/kg)	72	104	Empirical distribution – see Exhibit 8	2005-2006 American Healthy Homes Survey (HUD, 2011)	150
Diet Lead (μg/day)	3.00		Empirical distribution – see Exhibit 10	2005-2012 NHANES (CDC & NCHS, 2005, 2007, 2009, 2011); 2007-2013 FDA TDS (FDA, 2007, 2008, 2009, 2010, 2011, 2012, 2013); Xue et al. (2010).	1.95
Maternal (μg/dl)	0.61		None	2005-2012 NHANES (CDC & NCHS, 2005, 2007, 2009, 2011); 2007-2013 FDA TDS (FDA, 2007, 2008, 2009, 2010, 2011, 2012, 2013); Xue et al. (2010).	1
Geometric Standard Deviation	1.60		N/A	IEUBK default	1.60

Exhibit 19. Inputs Used for IEUBK for Children 5 to 6 Years Old

Variable	Input for IEUBK Analyses		Inputs for IEUBK-SHEDS Coupled Analysis	Data Source	Default IEUBK Input Value
Time Spent Outdoors (h/day)	1.81		Empirical distribution – see Exhibit 4	CHAD (U.S. EPA, 2009a)	4
Inhalation Rate (m^3/day)	3.82		Empirical distribution – see Exhibit 5	EFH (U.S. EPA, 2011)	7
Soil/Dust Ingestion (g/day)	0.034		Empirical distribution – see Exhibit 9	Ozkaynak et al. (2011)	0.090
Mean Drinking Water Rate (L/day)	0.213		Empirical distribution – see Exhibit 6	2005-2011 NHANES; (CDC & NCHS, 2005, 2007, 2009, 2011)	0.58
Air Lead (μg/m^3)	0.01		0.01	Office of Air and Radiation recommendation based on NAAQS monitoring (Cavender, 2013)	0.1
Soil Lead (mg/kg)	37	160	Empirical distribution – see Exhibit 7	2005-2006 American Healthy Homes Survey (HUD, 2011)	200
Dust Lead (mg/kg)	72	104	Empirical distribution – see Exhibit 8	2005-2006 American Healthy Homes Survey (HUD, 2011)	150
Diet Lead (μg/day)	3.31		Empirical distribution – see Exhibit 10	2005-2012 NHANES (CDC & NCHS, 2005, 2007, 2009, 2011); 2007-2013 FDA TDS (FDA, 2007, 2008, 2009, 2010, 2011, 2012, 2013); Xue et al. (2010).	2.05
Maternal (μg/dl)	0.61		None	2005-2012 NHANES (CDC & NCHS, 2005, 2007, 2009, 2011); 2007-2013 FDA TDS (FDA, 2007, 2008, 2009, 2010, 2011, 2012, 2013); Xue et al. (2010).	1
Geometric Standard Deviation	1.60		N/A	IEUBK default	1.60

Exhibit 20. Inputs Used for IEUBK for Children 6 to 7 Years Old

Variable	Input for IEUBK Analyses		Inputs for IEUBK-SHEDS Coupled Analysis	Data Source	Default IEUBK Input Value
Time Spent Outdoors (h/day)	1.65		Empirical distribution – see Exhibit 4	CHAD (U.S. EPA, 2009a)	4
Inhalation Rate (m^3/day)	3.82		Empirical distribution – see Exhibit 5	EFH (U.S. EPA, 2011)	7
Soil/Dust Ingestion (g/day)	0.029		Empirical distribution – see Exhibit 9	Ozkaynak et al. (2011)	0.085
Mean Drinking Water Rate (L/day)	0.228		Empirical distribution – see Exhibit 6	2005-2011 NHANES; (CDC & NCHS, 2005, 2007, 2009, 2011)	0.59
Air Lead (μg/m^3)	0.01		0.01	Office of Air and Radiation recommendation based on NAAQS monitoring (Cavender, 2013)	0.1
Soil Lead (mg/kg)	37	160	Empirical distribution – see Exhibit 7	2005-2006 American Healthy Homes Survey (HUD, 2011)	200
Dust Lead (mg/kg)	72	104	Empirical distribution – see Exhibit 8	2005-2006 American Healthy Homes Survey (HUD, 2011)	150
Diet Lead (μg/day)	3.29		Empirical distribution – see Exhibit 10	2005-2012 NHANES (CDC & NCHS, 2005, 2007, 2009, 2011); 2007-2013 FDA TDS (FDA, 2007, 2008, 2009, 2010, 2011, 2012, 2013); Xue et al. (2010).	2.22
Maternal (μg/dl)	0.61		None	2005-2012 NHANES (CDC & NCHS, 2005, 2007, 2009, 2011); 2007-2013 FDA TDS (FDA, 2007, 2008, 2009, 2010, 2011, 2012, 2013); Xue et al. (2010).	1
Geometric Standard Deviation	1.60		N/A	IEUBK default	1.60

6. Potential Approaches for Modeling Lead in Drinking Water

EPA considered several potential modeling approaches for lead in drinking water. These can be grouped into two categories: approaches based on a modeled individual and approaches based on a modeled population. In total, three approaches were utilized. In two approaches, the IEUBK model was used (approaches 1 and 2). In the third approach, the SHEDS-Multimedia model was used with regression equations relating lead uptake to BLLs derived from IEUBK (approach 3). All three approaches use the same input data sources, but differ in how the model output is used.

6.1 Approaches Based on a Modeled Child

Two approaches focusing on a modeled individual were considered:

Approach 1. Estimate the concentration of lead in drinking water that would result in a 1 or 5 percent increase in the probability of a child having an EBLL.

Approach 2. Estimate the concentration of lead in drinking water that would result in a 0.5 µg/dL or 1 µg/dL increase in a child's geometric mean BLL.

6.1.1 Methods for Approaches Based on a Modeled Individual

In all exposure scenarios for the modeled individual, BLLs were modeled using the IEUBK model. Input values based on updated information were used in place of some IEUBK defaults to more accurately reflect the exposures of a child currently living in the United States, as discussed previously in Section 5.

Approaches 1 and 2 both assess the relationship between water lead concentration and changes in the probability distribution for an individual child's potential BLL, as estimated using IEUBK. Additionally, the two approaches use the same baseline distribution of BLLs (see Exhibit 21), derived using the inputs described in Section 5. However, the approaches evaluate different questions regarding how water lead may shift the baseline probability distribution of blood lead for an individual. That is, approach 1 evaluates the question of what concentration of lead in drinking water changes the probability of an EBLL by a defined percentage (i.e., concentrating on the tail of the probability distribution for blood lead levels). In approach 2, the question evaluated is the concentration of lead in drinking water that shifts the geometric mean blood lead level by a defined amount (e.g., 0.5 µg/dL or 1 µg/dL). For the purposes of this analysis, EBLLs were defined as either at or above 3.5 µg/dL,[15] or at or above 5 µg/dL.[16] These reference levels are not based on preventing adverse health outcomes, but rather based on a statistical approach considering BLLs at the national level.

In considering these "individual" approaches, it is important to recall that the output of the IEUBK model may be interpreted as being representative of an individual, or of a group of individuals with identical exposure profiles. The geometric mean BLL represents a singular estimate for the BLL of an

[15] This is the 97.5th percentile BLL based on 2011-2014 NHANES data for 1- to 5-year-olds (CDC & NCHS, 2011, 2013).

[16] This is the 97.5th percentile BLL based on 2007-2011 NHANES for 1- to 5-year-olds and is the current CDC reference level (CDC & NCHS, 2007, 2009, 2011).

individual or identical group, while the BLL distribution represents the range of plausible BLL values and provides the probability of a child or group of children having a BLL above a specified value. The BLL distribution accounts for differences in temporal variability in absorption and biokinetics, behavior changes, and inter-individual differences affecting contact rates with lead. Further information on interpreting IEUBK output is provided in Section 3 of this report.

In order to estimate the contribution of drinking water lead exposures to BLLs, an understanding of the baseline geometric mean BLL and probability of an EBLL from sources other than drinking water was first needed. Baseline lead exposure scenarios were constructed for infants aged 0 to 6 months, 1- to 2-year-olds, and 0- to 7-year-olds using background lead concentrations as described in Section 5 and assuming no drinking water lead exposures. The resulting geometric mean BLLs and probability of EBLL are presented in Exhibit 21. To explore the direct impact of drinking water alone, model runs were also conducted assuming no other sources of lead exposures.

Exhibit 21. Baseline (without Drinking Water Lead) Geometric Mean BLLs and Probability of EBLL with Various Soil/Dust Lead Concentrations: 0- to 6-Month-Olds, 1- to 2-Year-Olds, and 0- to 7-Year-Olds

Soil/Dust Concentration (µg/g)	Baseline Geometric Mean BLL (µg/dL)	Baseline EBLL Probability	
		EBLL = 3.5 µg/dL	EBLL = 5 µg/dL
0- to 6-Month-Olds			
0/0*	0	0.00%	0.00%
37/72**	0.42	0.00%	0.00%
160/104**	0.72	0.00%	0.00%
1- to 2-Year-Olds			
0/0*	0	0.00%	0.00%
37/72**	0.59	0.01%	0.00%
160/104**	0.84	0.12%	0.01%
0- to 7-Year-Olds			
0/0*	0	0.00%	0.00%
37/72**	0.622	0.01%	0.00%
160/104**	0.850	0.13%	0.01%

* Inputs for all non-drinking water exposures were set to 0.

** Inputs for non-drinking water exposures were as described in Section 5.

For approach 1, values of 1 percent or 5 percent were added to the baseline probabilities to determine the target probabilities of EBLLs. For approach 2, values of 0.5 µg/dL or 1 µg/dL were added to the baseline geometric mean BLLs to determine the target BLLs for model runs with increases in drinking water lead concentrations. Next, through an iterative process of trial and error, drinking water exposures were added into the IEUBK model until the target geometric mean BLL or probability of an EBLL was reached.

6.1.2 Results of Approach 1: Estimating the Concentration of Lead in Drinking Water That Increases the Probability of an EBLL by a Defined Percentage

The drinking water concentrations needed to result in a 1 percent increase in the probability of an EBLL are presented in Exhibit 22, Exhibit 23, and Exhibit 24 for 0- to 6-month-olds, 1- to 2-year-olds, and 0- to 7-year-olds, respectively. Results for a 5 percent increase in the probability of an EBLL are presented in Exhibit 25, Exhibit 26, and Exhibit 27.

Exhibit 22. Drinking Water Concentration (µg/L) Resulting in a 1 Percent Increase in the Probability of EBLLs for Various Soil Lead Levels: 0- to 6-Month-Olds

Soil/Dust Concentration (µg/g)	EBLL = 3.5 µg/dL		EBLL = 5 µg/dL	
	Drinking Water Concentration (µg/L) Resulting in a 1% Increase in the Probability of EBLLs	Resulting Geometric Mean BLL (µg/dL)	Drinking Water Concentration (µg/L) Resulting in a 1% Increase in the Probability of EBLLs	Resulting Geometric Mean BLL (µg/dL)
0/0*	11.3	1.5	16.6	2.1
37/72**	8.2	1.5	13.4	2.1
160/104**	5.9	1.5	11.2	2.1

* Inputs for all non-drinking water exposures were set to 0.

** Inputs for non-drinking water exposures were as described in Section 5.

Exhibit 23. Drinking Water Concentration (µg/L) Resulting in a 1 Percent Increase in the Probability of EBLLs for Various Soil Lead Levels: 1- to 2-Year-Olds

Soil/Dust Concentration (µg/g)	EBLL = 3.5 µg/dL		EBLL = 5 µg/dL	
	Drinking Water Concentration (µg/L) Resulting in a 1% Increase in the Probability of EBLLs	Resulting Geometric Mean BLL (µg/dL)	Drinking Water Concentration (µg/L) Resulting in a 1% Increase in the Probability of EBLLs	Resulting Geometric Mean BLL (µg/dL)
0/0*	26.5	1.2	38.5	1.7
37/72**	13.4	1.2	25.2	1.7
160/104**	8.1	1.2	19.4	1.7

* Inputs for all non-drinking water exposures were set to 0.

** Inputs for non-drinking water exposures were as described in Section 5.

Exhibit 24. Drinking Water Concentration (µg/L) Resulting in a 1 Percent Increase in the Probability of EBLLs for Various Soil Lead Levels: 0- to 7-Year-Olds

Soil/Dust Lead Concentration (µg/g)	EBLL = 3.5 µg/dL		EBLL = 5 µg/dL	
	Drinking Water Concentration (µg/L) Resulting in a 1% Increase in the Probability of EBLLs	Resulting Geometric Mean BLL (µg/dL)	Drinking Water Concentration (µg/L) Resulting in a 1% Increase in the Probability of EBLLs	Resulting Geometric Mean BLL (µg/dL)
0/0*	27.3	1.2	39.7	1.7
37/72**	12.9	1.2	25.1	1.7
160/104**	8.2	1.2	19.8	1.7

* Inputs for all non-drinking water exposures were set to 0.

** Inputs for non-drinking water exposures were as described in Section 5.

Exhibit 25. Drinking Water Concentration (µg/L) Resulting in a 5 Percent Increase in the Probability of EBLLs for Various Soil Lead Levels: 0- to 6-month-olds

Soil/Dust Lead Concentration (µg/g)	EBLL = 3.5 µg/dL		EBLL = 5 µg/dL	
	Drinking Water Concentration (µg/L) Resulting in a 5% Increase in the Probability of EBLLs	Resulting Geometric Mean BLL (µg/dL)	Drinking Water Concentration (µg/L) Resulting in a 5% Increase in the Probability of EBLLs	Resulting Geometric Mean BLL (µg/dL)
0/0*	14.9	1.9	21.9	2.7
37/72**	11.7	1.9	18.7	2.7
160/104**	9.4	1.9	16.4	2.7

* Inputs for all non-drinking water exposures were set to 0.

** Inputs for non-drinking water exposures were as described in Section 5.

Exhibit 26. Drinking Water Concentration (µg/L) and Resulting in a 5 Percent Increase in the Probability of EBLLs for Various Soil Lead Levels: 1- to 2-year-olds

Soil/Dust Lead Concentration (µg/g)	EBLL = 3.5 µg/dL		EBLL = 5 µg/dL	
	Drinking Water Concentration (µg/L) Resulting in a 5% Increase in the Probability of EBLLs	Resulting Geometric Mean BLL (µg/dL)	Drinking Water Concentration (µg/L) Resulting in a 5% Increase in the Probability of EBLLs	Resulting Geometric Mean BLL (µg/dL)
0/0*	37.1	1.6	54.1	2.3
37/72**	23.8	1.6	40.6	2.3
160/104**	18.2	1.6	34.7	2.3

* Inputs for all non-drinking water exposures were set to 0.

** Inputs for non-drinking water exposures were as described in Section 5.

Exhibit 27. Drinking Water Concentration (µg/L) Resulting in a 5 Percent Increase in the Probability of EBLLs for Various Soil Lead Levels: 0- to 7-Year-Olds

Soil/Dust Lead Concentration (µg/g)	EBLL = 3.5 µg/dL		EBLL = 5 µg/dL	
	Drinking Water Concentration (µg/L) Resulting in a 5% Increase in the Probability of EBLLs	Resulting Geometric Mean BLL (µg/dL)	Drinking Water Concentration (µg/L) Resulting in a 5% Increase in the Probability of EBLLs	Resulting Geometric Mean BLL (µg/dL)
0/0*	38.2	1.6	56.0	2.3
37/72**	23.6	1.6	41.1	2.3
160/104**	18.5	1.6	35.7	2.3

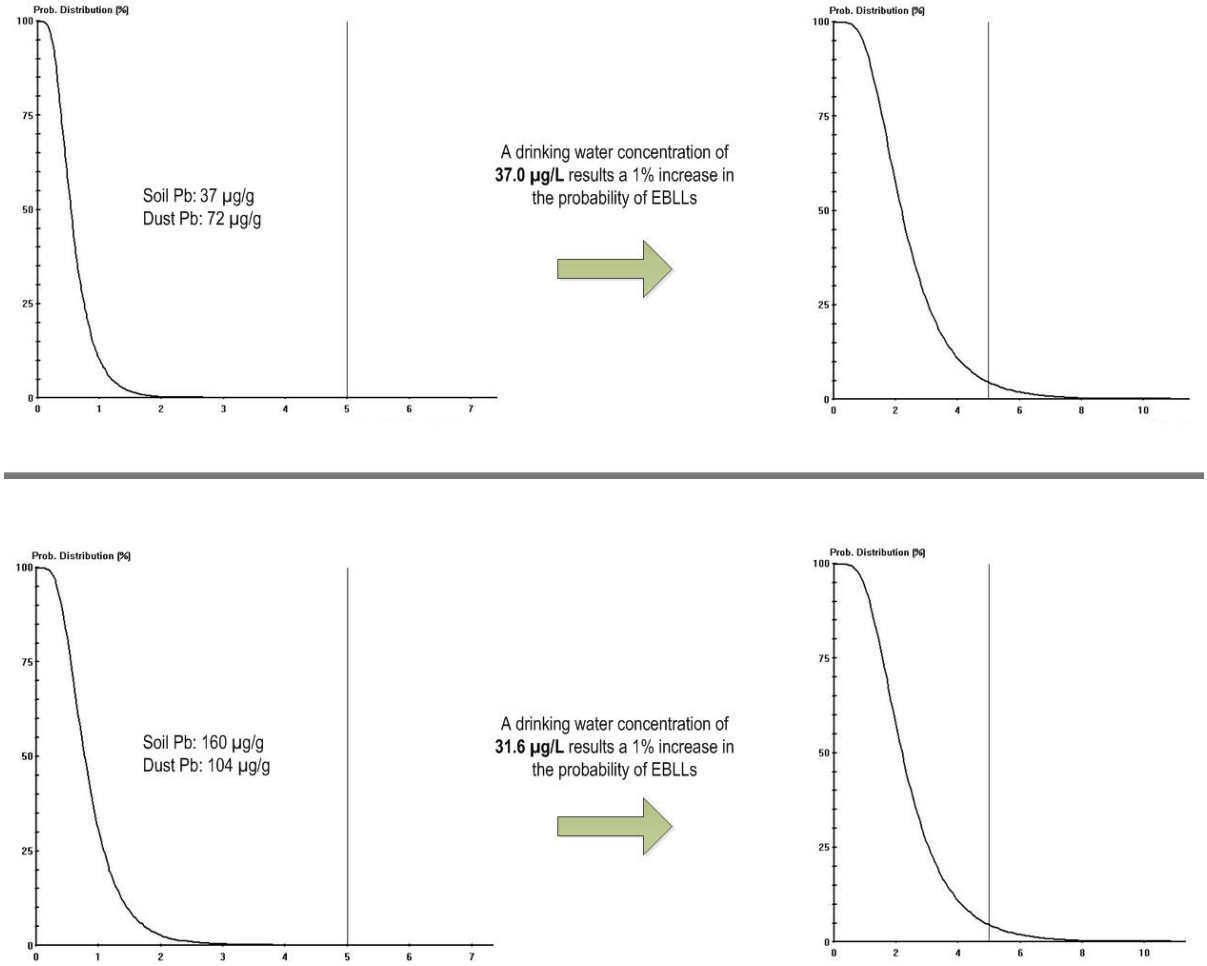
* Inputs for all non-drinking water exposures were set to 0.

** Inputs for non-drinking water exposures were as described in Section 5.

The above exhibits indicate that modeling results are lowest when considering 0- to 6-month-olds, which reflects the greater drinking water ingestion rates in this population relative to older children, given that at baseline (i.e., without drinking water) 0- to 6-month-olds are predicted to have the lowest BLLs compared to 1- to 2-year-olds and 0- to 7-year-olds (see Exhibit 21).

As background lead exposures increase, the concentration of lead in drinking water expected to increase the probability of an EBLL by a defined percentage decreases. Exhibit 28 demonstrates why the amount of drinking water lead expected to increase the probability of an EBLL decreases as soil lead concentration increases. In summary, to evaluate the amount of drinking water lead expected to increase the probability of an EBLL, the baseline BLL distribution is important because it defines the probability of a particular individual having a BLL at the higher end of the BLL spectrum. When considering individuals with the same non-soil lead exposures (and no drinking water lead exposures), an individual exposed to 160 µg/g of soil lead will have a baseline BLL distribution with the tail end of plausible BLLs closer to the specified EBLL value—or with a greater proportion of the tail end above the EBLL—than an individual exposed to 37 µg/g soil lead will. Therefore, the amount of water lead necessary to increase the probability of an EBLL is less for an individual with soil lead of 160 µg/g compared to an individual with soil lead of 37 µg/g. This is demonstrated in Exhibit 28. In evaluating this information it is important to keep in mind that this analysis is not specific to people with high-end exposures.

Exhibit 28. Visual Depiction of the Increase in the Probability Distribution of BLLs for a 1- to 2-year-olds with Increases in Water Lead Based on Different Background Lead Exposures



6.1.3 Results of Approach 2: Estimating the Concentration of Lead in Drinking Water That Increases a Geometric Mean BLL by a Defined Unit

The drinking water concentrations needed to result in a 0.5 or 1 µg/dL increase in the geometric mean BLLs of groups of 0- to 6-month-olds, 1- to 2-year-olds, and 0- to 7-year-olds are presented in Exhibit 29, Exhibit 30, and Exhibit 31, respectively.

Exhibit 29. Summary of Estimates Regarding the Amount of Lead in Drinking Water Resulting in a 0.5 or 1 µg/dL Increase in Geometric Mean BLL for 0- to 6-Month-Olds, With and Without Non-Drinking Water Lead Exposure

Soil/Dust Lead Concentration (µg/g)	Drinking Water Concentration (µg/L) Resulting in a 0.5 µg/dL Increase in the Geometric Mean BLL	Resulting Geometric Mean Blood Lead Level (µg/dL)	Drinking Water Concentration (µg/L) Resulting in a 1 µg/dL Increase in the Geometric Mean BLL	Resulting Geometric Mean Blood Lead Level (µg/dL)
0/0*	3.7	0.5	7.6	1
37/72**	3.8	0.92	7.7	1.42
160/104**	3.9	1.22	7.9	1.72
* Inputs for all non-drinking water exposures were set to 0. ** Inputs for non-drinking water exposures were as described in Section 5.				

Exhibit 30. Summary of Estimates Regarding the Amount of Lead in Drinking Water Resulting in a 0.5 or 1 µg/dL Increase in Geometric Mean BLL for 1- to 2-Year-Olds, With and Without Non-Drinking Water Lead Exposure

Soil/Dust Lead Concentration (µg/g)	Drinking Water Concentration (µg/L) Resulting in a 0.5 µg/dL Increase in the Geometric Mean BLL	Resulting Geometric Mean Blood Lead Level (µg/dL)	Drinking Water Concentration (µg/L) Resulting in a 1 µg/dL Increase in the Geometric Mean BLL	Resulting Geometric Mean Blood Lead Level (µg/dL)
0/0*	11.1	0.5	22.5	1
37/72**	11.4	1.09	23.1	1.59
160/104**	11.5	1.34	23.4	1.84
* Inputs for all non-drinking water exposures were set to 0. ** Inputs for non-drinking water exposures were as described in Section 5.				

Exhibit 31. Summary of Estimates Regarding the Amount of Lead in Drinking Water Resulting in a 0.5 or 1 µg/dL Increase in Geometric Mean BLL for 0- to 7-Year-Olds, With and Without Non-Drinking Water Lead Exposure

Soil/Dust Lead Concentration (µg/g)	Drinking Water Concentration (µg/L) Resulting in a 0.5 µg/dL Increase in the Geometric Mean BLL	Resulting Geometric Mean Blood Lead Level (µg/dL)	Drinking Water Concentration (µg/L) Resulting in a 1 µg/dL Increase in the Geometric Mean BLL	Resulting Geometric Mean Blood Lead Level (µg/dL)
0/0*	11.4	0.5	23.2	1
37/72**	11.7	1.12	23.8	1.62
160/104**	11.8	1.35	24.1	1.85
* Inputs for all non-drinking water exposures were set to 0. ** Inputs for non-drinking water exposures were as described in Section 5.				

These exhibits demonstrate that the concentration of lead in drinking water potentially leading to a 0.5 or 1 µg/dL increase in blood lead varies slightly based on the background concentration of lead (i.e., the soil/dust lead concentrations) in the exposure scenarios evaluated. To evaluate the sensitivity of this analysis to higher soil lead levels, we conducted an additional analysis assuming soil lead levels of 1,000 µg/g and dust lead levels of 72 µg/g (the geometric mean dust lead level; all other inputs were the same). In this scenario, the amount of lead in drinking water estimated to increase geometric mean BLLs by 0.5 or 1.0 µg/dL increased to 4.3 and 8.8 µg/L for 0- to 6-month-olds, to 12.2 and 24.8 µg/L for 1- to 2-year-olds, and to 12.5 and 25.5 µg/L for 6- to 7-year-olds.

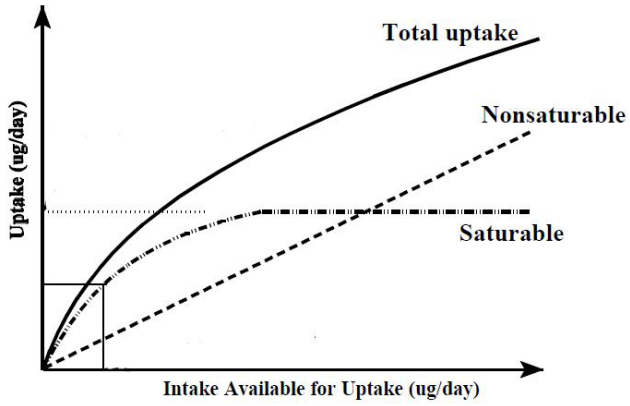
These results demonstrate that there is an increase in the amount of water lead necessary to increase blood lead by the same unit given increasing levels of background exposure to lead. This is due to the way lead uptake is modeled in IEUBK. That is, IEUBK models lead uptake from the gut to different body compartments (e.g., blood, bone, liver) using passive and active pathways. These pathways relate the amount of lead available for uptake (due to lead concentrations and intake rates) to the actual amount of lead uptake (U.S. EPA, 1994).[17] Briefly, the model allows for passive uptake pathways that are considered non-saturable. In these pathways, the amount of lead uptake will be linearly proportional to lead intake at all dose levels. IEUBK also allows for active uptake pathways, which may be saturable (i.e., at higher doses, only a fraction of available lead may be able to be absorbed). Collectively, the passive and active pathways define a saturable process: as the amount of lead available for uptake increases, the actual lead uptake as a fraction of total lead available for uptake decreases. Thus, the overall relationship between lead uptake and lead absorption is non-linear, as displayed in Exhibit 32.

[17] The uptake of lead from water is modeled in IEUBK using the following equation:

$$UpWater = InWater * ABSW * AVW * \left[PAF + \frac{1-PAF}{1+\frac{AVIntake}{SATIntake}}\right]$$

Where InWater = water intake; ABSW = total absorption for water at low saturation; AVW = the bioavailability of water; PAF = passive absorption at low dose (PAF, default = 0.2), AVIntake = the amount of lead available for intake from all sources, SATIntake = half the saturation point for absorbable lead intake.

Exhibit 32. Modeling of Lead Absorption in the IEUBK Model with Saturable and Non-Saturable Components



Source: U.S. EPA (1994, p. 26).

Given this relationship, smaller amounts of lead in drinking water will cause a defined increase in BLLs when modeling an individual with no other lead exposures. This is because, as the amount of total lead available for uptake decreases, the proportion of drinking water lead taken up in the gut increases. The maximum gut uptake of drinking water lead will thus occur when non-drinking water exposures are zero. In contrast, when the soil lead concentration is 1,000 µg/g, absorption in the gut is closer to the saturation point; the same increases in drinking water lead concentrations will result in lower proportions of uptake of drinking water lead into the gut. This is true for any source of lead exposure that approaches the uptake saturation point.

Results of approach 1 also show that when the same EBLL is used for all the age groups, modeled results are lower when considering 0- to 6-month-olds rather than 1- to 2-year-olds and 0- to 7-year-olds. This is because 0- to 6-month-olds generally have greater drinking water ingestion rates and lower non-drinking water lead exposures than older children. Thus, formula-fed 0- to 6-month-olds typically are exposed to a greater amount of lead from drinking water compared to older children.

6.1.4 Strengths, Limitations, and Uncertainties of the Approaches Based on a Modeled Individual

Approaches 1 and 2 provide context on the level of lead in drinking water that results in a shifted probability distribution of potential BLLs for a child with varying exposures to non-drinking water sources of lead. The two analyses in this section present results that depict how water lead concentration changes BLLs both at the geometric mean and at the tail of a probability distribution, while taking into account other exposures to lead. Given that lead is a multimedia pollutant it is important to understand how exposure to lead from a multitude of sources can cumulatively contribute to blood lead. The approaches presented in this section allow for this understanding.

Additionally, these approaches demonstrate several important concepts. One is that, for a child of a particular age group, the quantity of lead in drinking water associated with a defined unit change in BLL varies only slightly in each of the exposure scenarios evaluated. For example, when considering an infant across the first 6 months of life, the amount of lead in drinking water required to change the geometric mean blood lead level by 1 µg/dL ranges only from 7.6 to 7.9 µg/dL, as presented in

approach 2. In approach 1, the results are more dependent on the probability distribution of blood lead without drinking water exposure. For example, estimated results range from approximately 19 µg/dL to 38 µg/dL for a 1- to 2-year-old when considering a 1 percent increase in the probability of an EBLL, defined as 5 µg/dL.

Further, when considering lead as a multimedia contaminant, the analysis for approach 2 revealed that the amount of lead in drinking water expected to increase blood lead by a defined amount increases with increasing background exposures. That is, the lowest drinking water concentration that could be derived from this approach is based on a modeled individual with no other background exposures to lead. This is due to the slight saturation effect that takes place as individuals are exposed to more lead from non-drinking water sources. The opposite trend is seen in approach 1, when evaluating how much water lead increases an individual's probability of an EBLL. The amount of lead in drinking water required to increase an individual's probability of an EBLL decreases with increasing amount of lead exposure from non-drinking water sources. Both estimates demonstrate that the contribution from drinking water is less when there are multiple other exposure sources than when drinking water is the primary exposure source. Decreasing the lead concentrations in exposure media other than drinking water lowers their contribution to total exposure, and the drinking water source of exposure becomes more dominant. Because there is no known threshold for the developmental effects of lead in children, any increase in lead exposures can potentially place a child at risk for adverse health effects.

For the modeled individual, there is uncertainty inherent in potential approaches that are based on many different model inputs, each of which represents an estimate of central tendency and thus has its own associated uncertainty as touched on in Section 5. Beyond the inputs discussed in Section 5 there are additional uncertainties regarding the default parameters in IEUBK that were not changed, such as lead absorption fractions. These values are uncertain and likely vary with parameters such as age, time of last meal, diet, and others. While IEUBK does model uptake based on body weight scaled intake, the modeled absorption fractions are constant across ages, which may introduce uncertainty into our estimates. As noted in White et al., "there is evidence that the absorption of lead in food by infants is quite high—at least 40 to 50 [percent].... Although absorption probably decreases after infancy, we have no direct evidence on how to interpolate this range for children 2 to 6 years of age…. In view of the uncertainty about this, the IEUBK model uses the same default value of 50 [percent] for ages 1 to 6 years. This value will, at worst, slightly overestimate dietary lead uptake in older children" (1998, p. 1519-1520).

Additionally, it is important to acknowledge that the IEUBK model was developed during a time when lead exposures were higher than current levels. However, the empirical comparisons from Superfund sites do include exposures that resulted in observed blood lead concentrations below 2 µg/dL (Hogan et al., 1998, Figures 2-5). In the exposure scenarios, updated input values were used in place of IEUBK defaults in an attempt to reflect current exposure levels in U.S. children. Using the input values discussed in Section 5 and a drinking water lead concentration of 0.9 µg/L[18] in the IEUBK model, the predicted geometric mean BLL for children aged 1 to 2 years old ranges from approximately 0.6 to 0.9 µg/dL. The geometric mean BLL for 1- to 2-year-olds in the 2013-2014

[18] The value of 0.9 µg/L represents the population weighted average of drinking water lead concentration based on data from the 2009 EPA Office of Water Six Year Review dataset (U.S. EPA, 2010a).

NHANES cycle was 0.9 µg/dL. Thus, the maximum disparity between our modeled BLL estimates and NHANES BLL data is well within 1 µg/dL, which is the value that Hogan et al. (1998) noted as the range of difference between IEUBK predictions and empirical blood lead data from Superfund sites. However, since NHANES does not provide BLL data for infants less than 1 year old, we do not have a directly comparable dataset to evaluate the accuracy of our modeled BLL estimates based on updated input values for infants aged 0-6 months.

Further, a potential limitation in using the results from the applied approaches is that only individuals with average lead exposure levels are considered. Under approach 1, the resulting values may be underestimates of BLLs for children who are more highly exposed to lead than the average child because there tends to be a correlation between lead concentrations from different exposure pathways. For example, children living in older homes and multi-unit dwellings are likely exposed to lead in paint dust and chips as well as lead leaching from LSLs and lead-containing plumbing fixtures delivering drinking water to residences. In the model runs presented above, the presence of lead paint within a child's home is not explicitly considered, although the data used to derive the soil and dust lead input parameters likely include homes with lead paint present. To more specifically evaluate a higher exposure scenario with a more direct contribution from lead paint, an additional analysis was conducted using soil and dust lead inputs from homes built prior to 1950, which is presented in Appendix A. However, potential underestimates of the resulting BLLs using approach 1 only occur up until a certain level—the apex of the blood lead distribution curve. Beyond this point, some children in areas with high background exposure levels may already have EBLLs. As a result, larger drinking water lead concentrations would be needed to further shift the potential blood lead distribution, which is not useful in setting a protective value for lead in drinking water.[19]

The way in which the IEUBK model accounts for variability in BLLs attributable to inter-individual differences and other variations in behaviors and biokinetic parameters is through use of the geometric standard deviation of BLLs. That is, individuals who may have a greater susceptibility to high BLLs due to their physiological makeup would be at the high end of the BLL distribution curve based on the estimated geometric mean BLL and the GSD. An alternative method such as Monte Carlo simulations, which could assign variation to all parameters within the model while accounting for correlation between variables, would be able to better capture variability in BLLs for both

[19] Once the baseline BLLs are so high that there is more than a 50 percent probability of the BLL for an average child being above an EBLL, the amount of lead in drinking water estimated to increase the probability of an EBLL increases. Thus, in considering this approach, a particularly important feature of the probability density is the value of the geometric mean BLL at the peak of the distribution (i.e., at the highest probability) relative to the EBLL. If the defined EBLL is greater than the predicted geometric mean BLL, increases in drinking water lead shift the area under the density curve to higher values in a manner that captures increasing EBLL probability per unit shift in the BLL probability distribution. In other words, if the EBLL is greater than the geometric mean BLL for the age group, the amount of drinking water lead estimated to increase the probability of an EBLL decreases as the geometric mean approaches the EBLL value. In contrast, if the EBLL is lower than the geometric mean BLL, shifting the probability density to higher values will capture decreasing amounts of probability per unit shift in the BLL probability distribution. That is, it will take more drinking water lead to shift the distribution of BLL past a defined EBLL if the geometric mean BLL is greater than the EBLL cut point.

approaches. A distributional approach such as this, which more explicitly considers exposure variability, is presented in approach 3 (see Section 6.2.1).

Lastly, uncertainty remains as to how the drinking water concentration estimates generated using these approaches represents the real-world distribution of drinking water lead. While IEUBK models drinking water lead concentrations using an annual average, real-world drinking water lead concentrations can fluctuate widely depending on factors such as stagnation time, temperature, season, and water flow characteristics. Thus, the representativeness of model results for the current distribution of lead in drinking water lead is an additional source of uncertainty.

6.2 Approach Based on a Modeled Population of Children

Using the inputs described in Section 5, EPA evaluated one approach to investigate how drinking water lead might alter BLLs on a population basis:

Approach 3. Estimate the amount of lead in drinking water that would result in a population's predicted distribution of BLLs having a 95th or 97.5th percentile BLL at 3.5 or 5 µg/dL (Zartarian, Xue, Tornero-Velez, & Brown, 2016)

6.2.1 Approach 3: Estimate the Concentration of Lead in Drinking Water That Would Result in an EBLL Using SHEDS with IEUBK Derived Regression

The objective of approach 3 was to determine the level of lead in drinking water that could keep blood lead levels at specified percentiles of the simulated U.S. population distribution for children less than 7 years old below specified "targets." Approach 3 uses a probabilistic modeling methodology (SHEDS-Multimedia coupled with IEUBK) and multimedia lead exposure analysis that considers lead in water, soil, dust, food, and air. Another objective was to evaluate the coupled SHEDS-IEUBK modeled predictions using NHANES blood lead data, and identify key exposure pathways and model inputs (presented in Appendix B). Note that this current national population-based analysis is not designed to estimate a drinking water concentration for a target BLL in a specific at-risk population or specific households with high-risk attributes. The aim of this approach was to determine the level of lead in drinking water that would result in blood lead being equal to a specified value at a specified percentile. More specifically, in this analysis, the concentrations of lead in drinking water that would result in the following were evaluated:

- The 97.5th percentile blood lead being equal to 3.5 µg/dL
- The 97.5th percentile blood lead being equal to 5 µg/dL
- The 95th percentile blood lead being equal to 3.5 µg/dL
- The 95th percentile blood lead being equal to 5 µg/dL

This was done for 0- to 6-month-olds, 1- to 2-year-olds, and 0- to 7-year-olds for reasons described previously (see Section 5.1).

Method

The analyses conducted under this approach implemented a probabilistic multimedia lead exposure analysis using the EPA Office of Research and Development's SHEDS-Multimedia model coupled

with regression equations derived from IEUBK relating lead uptake to BLLs.[20] The probabilistic modeling allowed for an estimated distribution of lead intakes while accounting for variability in exposure due to differences in individual activity patterns and multimedia lead concentrations. Both IEUBK and SHEDS have exposure and dose components, but SHEDS-Multimedia simulates human exposures probabilistically.

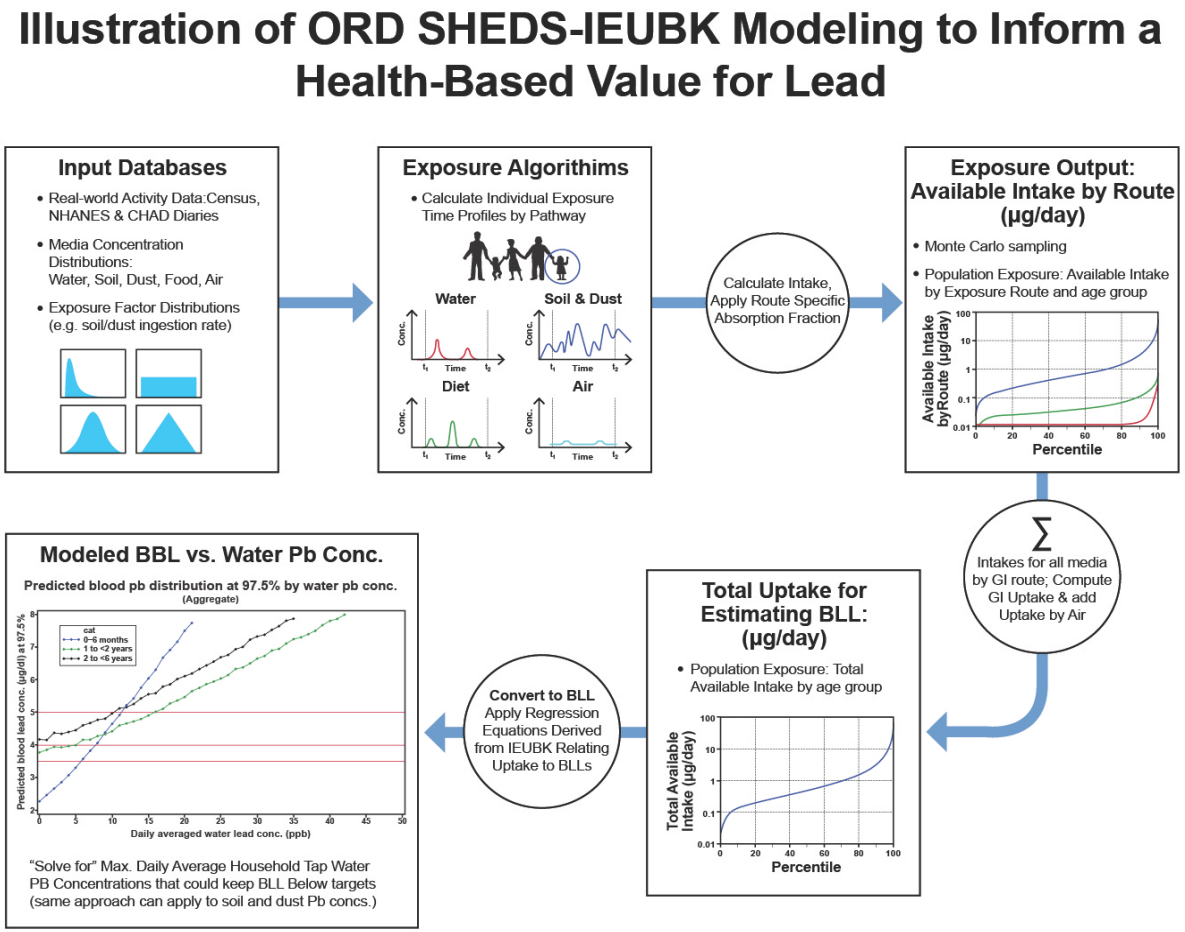
As previously summarized, IEUBK is comprised of four components: exposure, uptake, biokinetics, and variability. The aim of this work was to have SHEDS generate probabilistic exposures as inputs to IEUBK's exposure and uptake components and then use age-based relationships derived from IEUBK to relate lead uptake, estimated in SHEDS, to BLLs. An overview of SHEDS is provided in Section 4, and the inputs to the SHEDS model are described in Section 5. To account for the correlation between background soil and dust lead concentrations, a correlation coefficient of 0.48 was applied to the soil and dust distributions when conducting the Monte Carlo simulations. This coefficient is based on an analysis of data from the American Healthy Homes Survey (HUD, 2011). SHEDS simulated exposure for a 30-day time period.[21] A correlation coefficient of 0.2 was applied to dust and water concentrations based on data from EPA's NHEXAS Region 5 study Clayton et al. (1999).

An overview of the approach used for this analysis is presented in Exhibit 33.

[20] This approach was developed by EPA's Office of Research and Development and has been reviewed through a work-in-progress peer consultation.

[21] Additional analysis on other potentially correlated inputs is ongoing.

Exhibit 33. Overview of Approach 3 Using SHEDS with IEUBK Regression



Source: Zartarian et al. (2016)

Using input databases, such as the CHAD and NHANES, along with distributions of lead concentrations in various environmental media and exposure factors, SHEDS simulates exposure profiles for a population by age group (life stage). The first step in this exposure modeling results in estimates of the amount of lead intake per day by exposure route. These results take the form of a probability distribution of lead intakes (µg/day) accounting for variability. The lead intake rates are then converted to available intakes by multiplying each predicted intake distribution by the route-specific (e.g., inhalation, ingestion) absorption fractions. These are summarized in Exhibit 34 and are the default values in IEUBK.

Exhibit 34. Summary of Absorption Fractions by Media

Media	Absorption Fraction
Soil	30
Dust	30
Water	50
Diet	50
Air	32
Source: White et al. (1998).	

This results in distributions of available intake by exposure route, which can be summed to total available intake per day (µg/day).

Because lead intake is not linearly related to BLLs as shown in Exhibit 35 (and described in Section 6.1.1), EPA used the underlying equations in IEUBK that relate intake to lead uptake levels.[22]

In order to efficiently simulate a population's distribution of BLLs, regression equations relating lead uptake (µg/day) to blood lead (µg/dL) were developed using IEUBK. The regression equations were

[22] IEUBK methodology assumes a fraction of gastrointestinal (GI) intake (PAF) to be passively and completely absorbed. The PAF is pre-set at 20 percent. The complement, a fraction (1-PAF), 80 percent, is subject to a saturable process. The amount of lead that is absorbed by this saturable process is described in the IEUBK Technical Support Document (U.S. EPA, 1994):

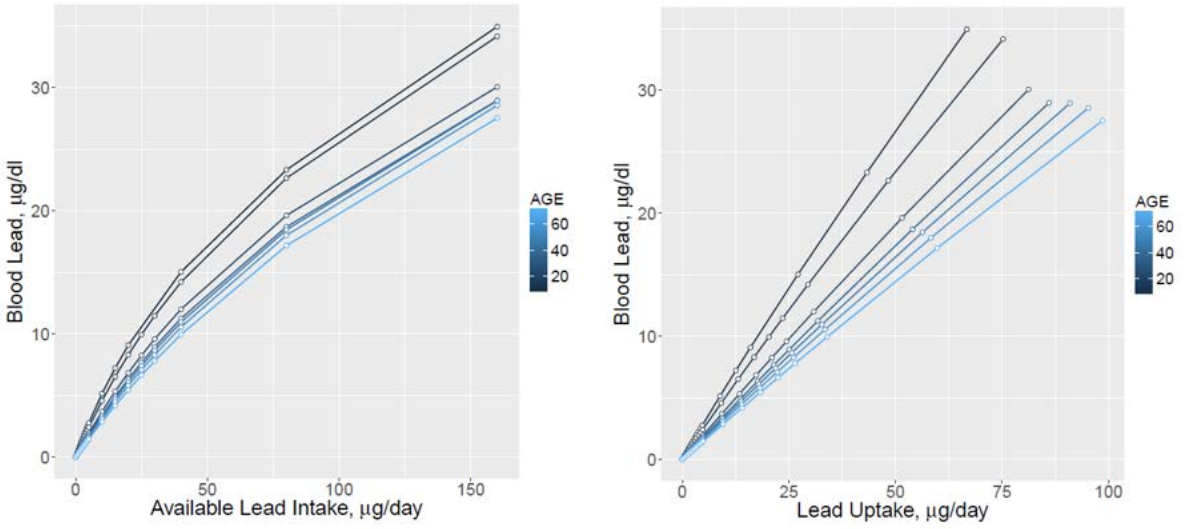
$$\frac{(1-PAF)\ x\ AVINTAKE(t)}{1+\frac{AVINTAKE(t)}{SATINTAKE(t)}}$$

Where the variable AVINTAKE(*t)* is the total available intake (µg/day) for a child of age *t* (months). SATINAKE(*t)* is constant for specific age (months), and given by Equation U-3 in the IEUBK Technical Support Document: $SATINTAKE(t) = SATINTAKE24\ x\ \frac{WTBODY(t)}{WTBODY(24)}$

Where SATINTAKE(24) is the value for a 24-month-old,100 µg/day. The equation for body weight, WTBODY(*t)* for a given month is given by Eqn B5-f in the IEUBK Technical Support Document. The weight for a 24-month-old, WTBODY(24), is 12.34 kg. Thus, the GI uptake is calculated as 20 percent available intake plus the amount of GI intake returned by Eqn A-1 in the IEUBK Technical Support Document. Available intake via the inhalation pathway is not subject to saturation and is added directly to the total uptake (µg/day).

developed by running IEUBK in batch mode with increasing amounts of lead intake as it relates to BLLs as predicted by IEUBK. This was done using the alternate source intake parameter (i.e., not the route-specific intake parameters) in IEUBK and stating 100 percent bioavailability, given that the absorption fractions are applied prior to applying the regression functions.

Exhibit 35. IEUBK Batch Model Output



Notes: Left Panel: For each age (months), there is a deterministic non-linear relationship between available intake (µg/day) and blood lead (µg/dL).
Right Panel: By accounting for saturable process in the GI, a linear relationship between uptake (mg/day) and blood lead (µg/dL) is observed.

The relationship between lead uptake and blood lead was shown to be linear (Exhibit 35, right panel), although not perfectly linear. Therefore, polynomial regression was used to address slight departures from linearity thought to arise from non-linear binding of lead to red blood cells. Additionally, there is a small intercept, because in running IEUBK to develop the regression equations, the default value of maternal blood lead of 1 µg/dL was not modified.

Exhibit 36 shows age-specific regressions used to describe an age-dependent relationship relating lead uptake to blood lead. The coefficients pertain to a third-order polynomial regression of the form:

$$\text{Blood Lead } (\mu g/dL) = \beta o + \beta_1 \text{ Uptake} + \beta_2 \text{ Uptake}^2 + \beta_3 \text{ Uptake}^3 + e$$

Coefficients for the month that represents the mid-point of the age range of interest were used in the analyses.

Exhibit 36. Polynomial Regressions Fit for Specific Months

IEUBK Age Interval (Year)	Age (Months)	β_0	β_1	β_2	β_3
0.5-1	9	0.00786	0.547	-0.00131	6.01E-6
1-2	18	-0.000311	0.447	-0.000637	1.53E-6
2-3	30	0.00123	0.379	-0.000429	8.45E-7
3-4	42	0.000658	0.355	-0.000371	6.24E-7
4-5	54	0.000636	0.336	-0.000338	5.44E-7
5-6	66	0.00165	0.313	-0.000278	3.57E-7
6-7	78	0.000132	0.288	-0.000230	3.08E-7
$R^2 > 0.995$					

With the lead uptake distribution calculated in SHEDS through probabilistic modeling of lead uptake and regression modeling relating lead uptake to BLLs, EPA was able to develop distributions of BLLs to determine the concentration of lead in drinking water that would result in a specified percentile of blood lead being equal to 3.5 or 5 µg/dL.

Results

Exhibit 37 and Exhibit 38 show the drinking water lead concentration results from the approach 3 analysis. These tables demonstrate that when considering aggregate exposures for individuals older than 6 months, modeled aggregate exposures from other sources already cause EBLLs even without drinking water lead (as indicated by the dashes in those columns). Therefore, in these age groups, it is not possible to keep the 97.5th percentile blood lead below 3.5 µg/dL if drinking water levels are included. This is because model results suggest that these individuals are already experiencing significant lead exposures from other sources including soil, dust, and diet. Accordingly, the drinking water concentration that results in the 97.5th percentile blood lead being equal to 3.5 µg/dL or 5 µg/dL ranges from 0 to 8 µg/L or 15 to 22 µg/L, respectively, when considering multimedia exposure to lead. The ranges reflect the differences in the age group of interest.

When considering aggregate exposure to lead, the concentration of lead in drinking water that results in blood lead remaining below 3.5 µg/dL or 5 µg/dL for the 95th percentile ranges from 9 to 13, or 17 to 30 µg/L, respectively. The ranges are reflective of the differences in the age groups being considered.

Exhibit 37. Drinking Water Concentration Resulting in the 97.5th Percentile Blood Lead Being Equal to 3.5 or 5 µg/dL

Age Group	Drinking Water Concentration (µg/L)	
	BLL = 3.5 µg/dL	BLL = 5 µg/dL
Aggregate Exposure Scenario		
0 to 6 months	8	15
1 to 2 years	-	22
0 to 7 years	-	17
Water Only Exposure Scenario		
0 to 6 months	13	19
1 to 2 years	25	37
0 to 7 years	20	29
Note: The values in this table for the 30-day analysis will be lower when biological variability is incorporated. Dashes in the table indicate where aggregate exposures from other sources already cause EBLLs even without drinking water lead.		

Exhibit 38. Drinking Water Concentration Resulting in the 95th Percentile Blood Lead Being Equal to 3.5 or 5 µg/dL

Age Group	Drinking Water Concentration (µg/L)	
	BLL = 3.5 µg/dL	BLL = 5 µg/dL
Aggregate Exposure Scenario		
0 to 6 months	10	17
1 to 2 years	13	30
0 to 7 years	9	23
Water Only Exposure Scenario		
0 to 6 months	14	20
1 to 2 years	31	46
0 to 7 years	27	41
Note: The values in this table for the 30-day analysis will be lower when biological variability is incorporated.		

6.2.2 Strengths and Limitations of the Approaches Based on Changes at the Population Level

Approach 3 is a probabilistic multimedia modeling approach that simulates real-world aggregate exposures, using well-reviewed, published, evaluated models that allow for contribution and sensitivity analyses, and identification of key factors and exposure pathways. SHEDS-IEUBK predictions compared well against real-world BLL data (small relative error modeled versus measured BLL) for several case studies (see Appendix B, Exhibit 54. It also uniquely allows relative contribution analyses for lead by media/pathway for all percentiles of the population (Appendix B, Exhibit 55). Population-based approaches allow a better characterization of variability in physiology and exposure than those based on a modeled individual. In addition, population-based approaches are consistent with previous EPA methods for assessing lead exposures. A potential limitation of

approach 3 is that IEUBK was only used as the basis for an analytical solution and was not used to allow its full capabilities of biokinetic modeling to estimate BLLs. In order to evaluate the reliability of using the regression equations, an approach was also developed that allowed for the SHEDS available intake distributions to be input into an IEUBK batch file through the alternate lead intake parameter field. Running IEUBK directly with the SHEDS inputs resulted in only a 0.5 percent difference in the estimates of BLLs.

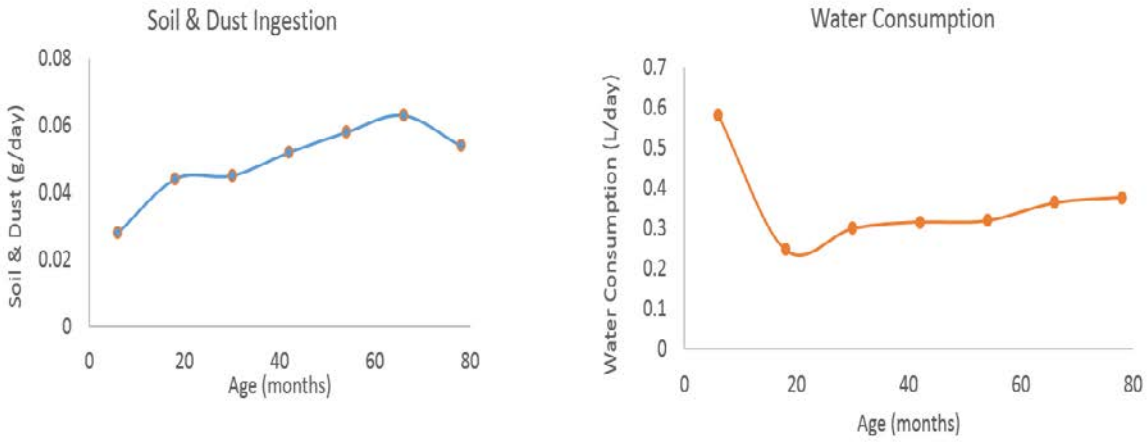
A critical assumption made when applying this approach using the batch mode in IEUBK through the alternate pathway parameter that an individual's lead intake (μg/day) is fixed over his/her lifetime from month zero to the month of interest. However, in standard operation of IEUBK, via the guided user interface windows, lead intake is modulated in accordance with the media intake profiles (e.g., soil ingestion in g/day) for the life of a child, considering different exposure factors at each age. Thus, IEUBK standard mode (via the guided user interface) and IEUBK batch mode (specifically, use of the alternate pathway) differ in this matter. The overall bias imposed on a population simulation is dependent on the media intake profile. To gauge the significance of this source of bias, the standard implementation of IEUBK was compared to batch implementation of IEUBK by media.

The results of this analysis are presented in Exhibit 39.

Exhibit 39. Results of Analysis Assessing Bias in SHEDS Analysis

Media	Lead Range	Bias[1]	6 mo	18 mo (N=896)	30 mo (N=974)	42 mo (N=636)	54 mo (N=679)	66 mo (N=588)	78 mo (N=653)
Soil	1–1,000 ppm	1.4%	0%	4%	4%	0.3%	2.5%	3.5%	-5.7%
Water	1–32 μg/L	-0.1%	0%	-9%	-9%	1.9%	0.4%	5.9%	1.9%
Diet	1.4–3.8 μg/day	1.9%	0%	3.6%	3.2%	0%	0%	3.4%	0%

Note:
1. Bias in this instance refers to relative error in IEUBK alternate input as compared with IEUBK standard input. Standard input means specifying media concentration, absorption fraction, and age-dependent media intake rates. As implemented, the alternate input held daily lead intake constant from birth to the age of the child.



The bottom right panel of Exhibit 39 shows consumption rates for water. Note that the water is consumed at a much greater rate (approximately 0.6 L/day) by 6-month-olds compared with 18- to 78-month-olds. Since lead intake for an 18-month-old is derived with the 18-month water consumption rate, assuming this lead intake is fixed over the entire exposure period (0-18 months) underestimated blood lead at 18 months because the water consumption was greater in the earlier months. Over time, however, impact of this bias might be reduced depending on the intake profile; note the bias is 1.9 percent at 24 months. Blood lead kinetics are most influenced by the exposures over the prior 3 months such that the overall impact of the bias on a population simulation is modest (0.1 percent for the water pathway for 1-to 6-year-olds, given this particular water consumption profile). By pathway, we do not observe an overall bias greater than 2 percent.

Additionally, as discussed in Section 5, there are limitations and uncertainties with some of the inputs associated with both the IEUBK and SHEDS model. In this approach, there is a limitation in using point estimates for the absorption fraction of lead from the different environmental media. However, the values used are consistent with what is currently used in the IEUBK model. Further, there is no drinking water intake value specific to formula-fed infants in the NHANES data that are used in SHEDS, and therefore the population that the NDWAC suggested considering is not explicitly considered in this approach. It is also unclear the extent to which the underlying distributions in the SHEDS model, as well as the NHEXAS-derived correlations between distributions in the SHEDS model, are accurate representations of those found in the U.S. population.

Other uncertainties in this analysis are the model averaging time, and how the coupled models capture biological and other sources of variability in the geometric standard deviation of BLLs. The latter issue relates to model coupling for this analysis. SHEDS-Multimedia estimates reflect exposure variability, but not biological variability or other sources of variability such as measurement or model error. Because IEUBK blood lead estimates do not reflect inter-individual behavioral and pharmacokinetic differences, a GSD of 1.6 is applied to outputs of IEUBK to account for biological variability and measurement error, but does not account for exposure variability. Outputs from the coupled SHEDS-IEUBK models, therefore, need a variability factor to account for the GSD difference between modeled and measured BLLs and reflect real-world BLLs that also account for biological variability.

This variability factor is affected by the exposure averaging time period. Initial analyses were conducted with 2-day exposure averaging times consistent with available activity diaries used in SHEDS-Multimedia. EPA subsequently focused on 30-day exposure averaging time simulations to be consistent with the minimum IEUBK output period of 1 month, per recommendations of an external peer consultation panel. The 30-day analysis results are provided in this document. The 30-day exposure period results under-predict the GSD and upper percentiles of BLLs in NHANES, as shown in Exhibit 40, and as such accordingly overestimate lead in water concentrations in Exhibit 37 and Exhibit 38. Using a 2-day model averaging time does not align with IEUBK, but compares more closely to NHANES BLL data and GSD. The 2-day results may approximate the distribution of BLL accounting for exposure, biological, and other sources of variability. Collecting more BLL data from states could help determine an appropriate variability factor and determine which averaging time is more appropriate to guide a health-based benchmark for lead. State-collected data will also supplement NHANES BLL data, which may not be fully representative of the true distribution of the U.S. population BLLs, particularly at the tails.

Exhibit 40. SHEDS-IEUBK BLL Evaluation with 2009-2014 NHANES Blood Data*

Age Group	Source	N	Mean	Std	50th	GM	GSD	95th	97.5th	99th	%>3 µg/dL
1 to 2 years old	Observed	475	1.47	1.30	1.12	1.16	1.92	3.60	5.54	7.90	6.95
	Predicted	3000	1.33	0.88	1.11	1.16	1.64**	2.95	3.75	4.88	4.87
	Relative Error		9%		1%	0%		18%	32%	38%	

N= sample size. GM = geometric mean. GSD = geometric standard deviation. Relative error is predicted minus observed, divided by observed.

*Longitudinal (30 days) with correlated key inputs.

**This GSD reflects the effect of exposure variability, but not biological variability on BLL.

7. Conclusions

This report presents several potential approaches to support modeling of lead in drinking water. The purpose of this review is to obtain feedback on the strengths and weaknesses of various modeling methods that can be used to assess the relationship between constant rate lead exposures of a representative child and a population of children across different age ranges and blood lead levels. Results are presented that reflect the concentrations of lead in drinking water that are predicted to result in specified unit changes in BLLs, increases in probability of EBLLs, and specific BLLs at given percentiles in a population distribution of blood lead. It is important to consider each of the potential approaches in the context of the extensive biomonitoring data that are currently available for BLLs in children in the United States. Over recent decades, BLLs in children in the United States have been dramatically reduced through a variety of regulatory actions and public health interventions, including implementation of the Lead and Copper Rule.

This analysis did not directly quantify potential resulting health impacts due to the BLLs predicted in the analyses presented. As discussed in Section 2, there are many adverse effects related to increases in BLLs including neurologic, cardiovascular, and adverse reproductive health outcomes. These adverse health effects have been observed even at low BLLs (i.e., less than 5 µg/dL). Results from each analysis are presented in Exhibit 41, Exhibit 42, and Exhibit 43.

Exhibit 41. Summary of Results 0- to 6-Month-Olds

Approach	Drinking Water Concentration (µg/L)	Additional Considerations
Approach 1: The concentration of lead in drinking water that would result in a 1 or 5 percent increase in the probability of a child having an EBLL.		
1% Increase in the Probability of an EBLL (EBLL = 3.5 µg/dL)	5.9 to 11.3	The lower value is based on a scenario with multimedia lead exposures, with arithmetic mean soil and dust lead concentrations. The upper value is based on a scenario only considering water lead exposure.
1% Increase in the Probability of an EBLL (EBLL = 5 µg/dL)	11.2 to 16.6	
5% Increase in the Probability of an EBLL (EBLL = 3.5 µg/dL)	9.4 to 14.9	
5% Increase in the Probability of an EBLL (EBLL = 5 µg/dL)	16.4 to 21.9	
Approach 2: The concentration of lead in drinking water that would result in a 0.5 µg/dL or 1 µg/dL increase in a child's geometric mean BLL.		
0.5 µg/dL Change in Geometric Mean BLL	3.7 to 3.9	The lower value is based on a scenario only considering water lead exposure. The upper value is based on a scenario with multimedia lead exposures, with arithmetic mean soil and dust lead concentrations.
1 µg/dL Change in Geometric Mean BLL	7.6 to 7.9	
Approach 3: The amount of lead in drinking water that would result in a population's predicted distribution of BLLs having a 95th or 97.5th percentile BLL at 3.5 or 5 µg/dL.		
95th Percentile BLL = 3.5 µg/dL	10 to 14	The lower value is based on a scenario with multimedia lead exposures. The upper value is based on a scenario only considering water lead exposures.
95th Percentile BLL = 5 µg/dL	17 to 20	
97.5th Percentile BLL = 3.5 µg/dL	8 to 13	
97.5th Percentile BLL = 5 µg/dL	15 to 19	

Exhibit 42. Summary of Results for 1- to 2-Year-Olds

Approach	Drinking Water Concentration (µg/L)	Additional Considerations
Approach 1: the concentration of lead in drinking water that would result in a 1 or 5 percent increase in the probability of a child having an EBLL.		
1% Increase in Probability of an EBLL (EBLL = 3.5 µg/dL)	8.1 to 26.5	The lower value is based on a scenario with multimedia lead exposures, with arithmetic mean soil and dust lead concentrations. The upper value is based on a scenario only considering water lead exposure.
1% Increase in Probability of an EBLL (EBLL = 5 µg/dL)	19.4 to 38.5	
5% Increase in the Probability of an EBLL (EBLL = 3.5 µg/dL)	18.2 to 37.1	
5% Increase in the Probability of an EBLL (EBLL = 5 µg/dL)	34.7 to 54.1	
Approach 2: The concentration of lead in drinking water that would result in a 0.5 µg/dL or 1 µg/dL increase in a child's geometric mean BLL.		
0.5 µg/dL Change in Geometric Mean BLL	11.1 to 11.5	The lower value is based on a scenario only considering water lead exposure. The upper value is based on a scenario with multimedia lead exposures, with arithmetic mean soil and dust lead concentrations.
1 µg/dL Change in Geometric Mean BLL	22.5 to 23.4	
Approach 3: The amount of lead in drinking water that would result in a population's predicted distribution of BLLs having a 95th or 97.5th percentile BLL at 3.5 or 5 µg/dL.		
95th Percentile BLL = 3.5 µg/dL	13 to 31	The lower value is based on a scenario with multimedia lead exposures. The upper value is based on a scenario only considering water lead exposures.
95th Percentile BLL = 5 µg/dL	30 to 46	
97.5th Percentile BLL = 3.5 µg/dL	- to 25	
97.5th Percentile BLL = 5 µg/dL	22 to 37	
Note: Dashes in the table indicate where aggregate exposures from other sources already cause EBLLs even without drinking water lead.		

Exhibit 43. Summary of Results for 0- to 7-Year-Olds

Approach	Drinking Water Concentration (µg/L)	Additional Considerations
Approach 1: the concentration of lead in drinking water that would result in a 1 or 5 percent increase in the probability of a child having an EBLL.		
1% Increase in Probability of an EBLL (EBLL = 3.5 µg/dL)	8.2 to 27.3	The lower value is based on a scenario with multimedia lead exposures, with arithmetic mean soil and dust lead concentrations. The upper value is based on a scenario only considering water lead exposure.
1% Increase in Probability of an EBLL (EBLL = 5 µg/dL)	18.5 to 38.2	
5% Increase in the Probability of an EBLL (EBLL = 3.5 µg/dL)	19.8 to 39.7	
5% Increase in the Probability of an EBLL (EBLL = 5 µg/dL)	35.7 to 56.0	
Approach 2: The concentration of lead in drinking water that would result in a 0.5 µg/dL or 1 µg/dL increase in a child's geometric mean BLL.		
0.5 µg/dL Change in Geometric Mean BLL	11.4 to 11.8	The lower value is based on a scenario only considering water lead exposure. The upper value is based on a scenario with multimedia lead exposures, with arithmetic mean soil and dust lead concentrations.
1 µg/dL Change in Geometric Mean BLL	23.2 to 24.1	
Approach 3: The amount of lead in drinking water that would result in a population's predicted distribution of BLLs having a 95th or 97.5th percentile BLL at 3.5 or 5 µg/dL.		
95th Percentile BLL = 3.5 µg/dL	9 to 27	The lower value is based on a scenario with multimedia lead exposures. The upper value is based on a scenario only considering water lead exposures.
95th Percentile BLL = 5 µg/dL	23 to 41	
97.5th Percentile BLL = 3.5 µg/dL	- to 20	
97.5th Percentile BLL = 5 µg/dL	17 to 29	
Note: Dashes in the table indicate where aggregate exposures from other sources already cause EBLLs even without drinking water lead.		

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Appendix A – Potential Values of Lead Modeling in Drinking Water Based on High Lead Exposure Scenarios

To explore the effects of modeling children with higher-than-average lead exposures on potential values, we assessed additional exposure scenarios for infants using drinking water intake rates specific to formula-fed infants and for infants and children living in homes with lead paint, as described in Sections A.1 and A.2, respectively. Approaches used to model lead in drinking water followed the same methods as described in Section 6. The analyses presented in this section are based on approaches 1 and 2.

A.1 Formula-Fed Infants

As mentioned previously in Section 5, the overall drinking water ingestion rate for 0- to 6-month-olds from NHANES was derived by considering all infants. This includes infants fed formula reconstituted with tap water, infants fed ready-to-drink formula, breast-fed infants, and infants who are breast-fed but receive supplemental formula. Since the latter three groups consume breastmilk or formula not mixed with tap water, they likely ingest smaller amounts of drinking water per day than infants fed formula reconstituted with tap water. Thus, the drinking water ingestion rate for 0- to 6-month-olds used in the main analyses is likely an underestimate of the true drinking water ingestion rate for infants fed formula reconstituted with tap water (from here on referred to simply as "formula-fed infants"). A drinking water ingestion rate specific to formula-fed infants of 0.64 L/day was derived using data from Kahn et al. (2013), as described in Section A.1.1. The methodology used to model lead in drinking water using this value is described in Section A.1.2, and results are presented in Sections A.1.3 and A.1.4.

A.1.1 Derivation of Drinking Water Ingestion Rate

A 2013 study by Kahn et al., "Estimates of Water Ingestion in Formula by Infants and Children Based on USDA's 1994-1996 and 1998 Continuing Survey of Food Intakes by Individuals," was used to derive a drinking water ingestion rate specific to formula-fed infants aged 0 to 6 months old. Kahn et al. (2013) estimated means and percentiles of community water (i.e., tap water) ingestion in U.S. infants and children using data from the Continuing Survey of Food Intake by Individuals (CSFII). In CSFII, drinking water data were collected over two non-consecutive days, and participants were given sample weights based on demographic factors. Kahn et al. (2013) estimated total average community water ingestion for all infants (n = 772), water consumers only, and formula consumers only. The formula consumers only group (i.e., formula-fed infants) was comprised of the subset of water consumers who were reported to have indirectly ingested community water as part of reconstituted or diluted infant formula. The mean total community water ingestion rates for formula-fed infants from Kahn et al. (2013) are displayed in Exhibit 44. Since results for formula-fed infants aged 0 to 6 months were presented in three age categories, the weighted average community water ingestion rate of 0.640 L/day was calculated for input into the IEUBK model. As expected, this value is larger than the drinking water ingestion rate of 0.526 L/day used for modeling exposures of 0- to 6-month-olds.

Exhibit 44. Summary of Drinking Water Intake Data for Formula-Fed Infants

Age Group (Months)	Sample Size	Mean Drinking Water Intake Rate (L/day)	90% Confidence Interval	Value Used in IEUBK (L/day)
0 to <1*	36	0.505	0.400-0.610	0.640
1 to <3	96	0.627	0.576-0.678	
3 to <6	214	0.699	0.652-0.746	

Source: Kahn et al. (2013, Table 2b).

*The sample size for 0- to <1-month-olds does not meet minimum reporting requirements as described in the *Third Report on Nutrition Monitoring in the United States, 1994-96* (Life Sciences Research Office, 1995); the mean drinking water intake for this age group is associated with a higher level of uncertainty.

A.1.2 Methodology Using a Drinking Water Ingestion Rate Specific to Formula-Fed Infants

Modeling of lead in drinking water based on consideration of formula-fed infants was generated using approaches 1 and 2.[23] For each approach, IEUBK model runs were performed using the drinking water ingestion rate of 0.64 L/day, along with the values for other inputs described previously in Section 5. Baseline geometric mean BLL and probability of EBLL for formula-fed infants under each soil/dust lead scenario assessed are displayed in Exhibit 45.

Exhibit 45. Baseline (without Drinking Water Lead) Geometric Mean BLLs and Probability of EBLL with Various Soil/Dust Lead Concentrations: Formula-Fed Infants

Soil/Dust Concentration (µg/g)	Geometric Mean BLL (µg/dL)	Baseline EBLL Probability	
		EBLL = 3.5 µg/dL	EBLL = 5 µg/dL
0/0*	0	0%	0%
37/72**	0.42	0%	0%
160/104**	0.72	0.001%	0%

* Inputs for all non-drinking water exposures were set to 0.

** Inputs for non-drinking water exposures were as described in Section 5.

A.1.3 Results of Approach 1: Estimating the Concentration of Lead in Drinking Water That Increases the Probability of an EBLL for a Formula-Fed Infant by a Defined Percentage

The drinking water concentrations needed to result in a given increase in the probability of an EBLL for formula-fed infants are presented in Exhibit 46 and Exhibit 47.

[23] Analogous analyses were not conducted for approach 3. This is because the methods for approach 3 require the use of SHEDS, which has drinking water intake rates programmed into the underlying model; therefore, the drinking water intake rate could not be changed.

Exhibit 46. Drinking Water Concentration (µg/L) Resulting in a *1 Percent* Increase in the Probability of EBLLs for Various Soil Lead Levels: Formula-Fed Infants

Soil/Dust Concentration (µg/g)	EBLL = 3.5 µg/dL		EBLL = 5 µg/dL	
	Drinking Water Concentration (µg/L) Resulting in a 1% Increase in the Probability of EBLLs	Resulting Geometric Mean Blood Lead Level (µg/dL)	Drinking Water Concentration (µg/L) Resulting in a 1% Increase in the Probability of EBLLs	Resulting Geometric Mean Blood Lead Level (µg/dL)
0/0*	9.3	1.47	13.7	2.11
37/72**	6.7	1.47	11.0	2.10
160/104**	4.9	1.48	9.2	2.11

* Inputs for all non-drinking water exposures were set to 0.

** Inputs for non-drinking water exposures were as described in Section 5.

Exhibit 47. Drinking Water Concentration (µg/L) Resulting in a *5 Percent* Increase in the Probability of EBLLs for Various Soil Lead Levels: Formula-Fed Infants

Soil/Dust Lead Concentration (µg/g)	EBLL = 3.5 µg/dL		EBLL = 5 µg/dL	
	Drinking Water Concentration (µg/L) Resulting in a 5% Increase in the Probability of EBLLs	Resulting Geometric Mean Blood Lead Level (µg/dL)	Drinking Water Concentration (µg/L) Resulting in a 5% Increase in the Probability of EBLLs	Resulting Geometric Mean Blood Lead Level (µg/dL)
0/0*	12.2	1.99	18.0	2.71
37/72**	9.6	1.90	15.4	2.72
160/104**	7.8	1.91	13.5	2.71

* Inputs for all non-drinking water exposures were set to 0.

** Inputs for non-drinking water exposures were as described in Section 5.

The amount of drinking water lead needed to increase the probability of an EBLL in formula-fed infants decreased as non-drinking water lead exposures increased, as discussed previously in Section 6.1.2. On average, modeling lead in drinking water by considering formula-fed infants was approximately 2-3 µg/L lower than that derived by using a drinking water intake rate for 0- to 6-month-olds that is not specific to formula-fed infants.

A.1.4 Results of Approach 2: Estimating the Concentration of Lead in Drinking Water That Increases the Geometric Mean BLL in Formula-Fed Infants by a Defined Unit

The drinking water concentrations needed to result in a given increase in the geometric mean BLL of formula-fed infants are presented in Exhibit 48.

Exhibit 48. Summary of Results Demonstrating the Amount of Lead in Drinking Water Resulting in a 0.5 or 1 µg/dL Increase in Geometric Mean BLL for Formula-Fed Infants, With and Without Non-Drinking Water Lead Exposure

Soil/Dust Lead Concentration (µg/g)	Drinking Water Concentration (µg/L) Resulting in a 0.5 µg/dL Increase in the Geometric Mean BLL	Resulting Geometric Mean Blood Lead Level (µg/dL)	Drinking Water Concentration (µg/L) Resulting in a 1 µg/dL Increase in the Geometric Mean BLL	Resulting Geometric Mean Blood Lead Level (µg/dL)
0/0*	3.0	0.5	6.2	1
37/72**	3.1	0.92	6.4	1.42
160/104**	3.2	1.22	6.5	1.72

* Inputs for all non-drinking water exposures were set to 0.

** Inputs for non-drinking water exposures were as described in Section 5.

The amount of drinking water lead needed to increase the BLL of formula-fed infants increased as non-drinking water lead exposures increased. Section 6.1.3 provides an explanation for this observed relationship. Results confirm that formula-fed infants are the most sensitive population (in regard to the amount of exposure to lead from drinking water) of those evaluated in this report. The potential concentrations of lead in drinking water are smaller than those derived from consideration of 0- to 6-month-olds, which ranged from 3.7 to 3.9 when evaluating a 0.5 µg/dL change and 7.6 to 7.9 when evaluating a 1 µg/dL change.

A.2 Infants and Children Living in Homes with Lead-Based Paint

The soil and dust lead concentration values presented in Section 5 were estimated using data from the American Healthy Homes Survey, which is a nationally representative survey of U.S. homes and thus includes homes with and without lead-based paint. Infants and children living in homes with lead-based paint are at greater risk for lead exposures as a result of higher soil and dust lead concentrations. To account for lead exposures associated with lead-based paint within a home, IEUBK runs were conducted using soil and dust lead levels of 221 µg/g and 134 µg/g, respectively. These concentrations are specific to homes built before 1950, as discussed in Section A.2.1. Methods used to model lead in drinking water based on infants and children living in homes with lead-based paint are presented in Section A.2.2, and results are displayed in Sections A.2.3 and A.2.4.

A.2.1 Derivation of Soil and Dust Lead Levels for Homes with Lead-Based Paint

The values of 221 µg/g and 134 µg/g for mean soil lead and dust lead concentrations, respectively, in homes with lead-based paint were derived from the American Healthy Homes Survey, which was described previously in Section 5.6. These values represent the geometric mean soil lead and dust lead concentrations observed in homes built prior to 1950. Based on historical information on lead-based paint regulations, the year 1950 was selected as a cut point to identify homes with likely sources of lead-based paint. In 1951, the city of Baltimore, Maryland, became the first in the United States to ban the use of lead-based paint within homes (Steering Committee of the 2008 National Healthy Homes Conference, 2008). Although a nationwide ban on the use of lead-based paint in homes was not enacted until 1978, there was a decline in the use of interior lead-based paint

throughout the United States during the 1950s and 1960s (CDC, 1991). Using data from homes constructed prior to 1950, rather than 1977, may thus better represent only homes with lead-based paint. Additionally, a recent large-scale analysis of BLLs in U.S. children by McClure et al. (2016) found that pre-1950 housing was a risk factor for high childhood BLLs. Children living in areas with a large proportion (≥ 51%) of homes built before 1950 had significantly higher proportions of BLLs above 5.0 µg/dL (OR: 5.86, 95% CI 5.71-6.01) or 10.0 µg/dL (OR: 6.34, 95% CI 5.97-6.74) than children living in areas with the lowest proportion (< 3.6%) of pre-1950s housing.

It is important to note that using pre-1950 mean values to represent homes with lead-based paint does not take into account the possibility of lead-based paint abatement. That is, a proportion of homes constructed prior to 1950 may have undergone full lead-based paint remediation and thus may no longer contain sources of lead-based paint. Thus, the selected soil and dust lead values may represent an underestimate of the true geometric mean of homes with lead-based paint.

A.2.2 Method Based on Infants and Children Living in Homes with Lead-Based Paint

Soil and dust lead concentrations of 221 µg/g and 134 µg/g, respectively, were used to account for the presence of lead-based paint within a home. Values for other inputs in the IEUBK model were the same as discussed previously in Section 5 and above. Using approaches 1 and 2 to model lead in drinking water, three populations living in homes with lead-based paint were assessed: 0- to 6-month-olds, 1- to 2-year-olds, and formula-fed infants. Exhibit 49 presents the baseline geometric mean BLLs and probability of EBLLs for these populations.

Exhibit 49. Baseline (without Drinking Water Lead) Geometric Mean BLLs and Probability of EBLLs for Infants and Children Living in Homes with Lead-Based Paint

Population Assessed	Geometric Mean BLL (µg/dL)	Baseline EBLL Probability	
		EBLL = 3.5 µg/dL	EBLL = 5 µg/dL
Formula-fed infants and 0- to 6-month-olds	0.89	0.01%	0%
1- to 2-year-olds	1.00	0.4%	0.03%
0- to 7-year-olds	0.99	0.4%	0.03%

A.2.3 Results for Approach 1: Estimating the Concentration of Lead in Drinking Water That Increases the Probability of an EBLL by a Defined Percentage

Exhibit 50 and Exhibit 51 show the drinking water concentrations needed to result in a 1 percent or 5 percent increase, respectively, in the probability of an EBLL for 0- to 6-month-olds, 1- to 2-year-olds, and formula-fed infants.

Exhibit 50. Drinking Water Concentration (µg/L) Resulting in a *1 Percent* Increase in the Probability of EBLLs in Infants and Children Living in Homes with Lead-Based Paint

Population Assessed	EBLL = 3.5 µg/dL		EBLL = 5 µg/dL	
	Drinking Water Concentration (µg/L) Resulting a 1% Increase in the Probability of EBLLs	Resulting Geometric Mean Blood Lead Level (µg/dL)	Drinking Water Concentration (µg/L) Resulting a 1% Increase in the Probability of EBLLs	Resulting Geometric Mean Blood Lead Level (µg/dL)
Formula-fed infants	3.8	1.48	8.1	2.11
0- to 6-month-olds	4.6	1.48	9.8	2.10
1- to 2-year-olds	5.7	1.24	16.1	1.69
0- to 7-year-olds	5.9	1.24	16.7	1.68

Exhibit 51. Drinking Water Concentration (µg/L) Resulting in a *5 Percent* Increase in the Probability of EBLLs in Infants and Children Living in Homes with Lead-Based Paint

Population Assessed	EBLL = 3.5 µg/dL		EBLL = 5 µg/dL	
	Drinking Water Concentration (µg/L) Resulting in a 5% Increase in the Probability of EBLLs	Resulting Geometric Mean Blood Lead Level (µg/dL)	Drinking Water Concentration (µg/L) Resulting in a 5% Increase in the Probability of EBLLs	Resulting Geometric Mean Blood Lead Level (µg/dL)
Formula-fed infants	6.6	1.89	12.4	2.71
0- to 6-month-olds	8.1	1.90	15.1	2.71
1- to 2-year-olds	15.1	1.64	31.3	2.31
0- to 7-year-olds	15.7	1.64	32.5	2.31

Under approach 1, results for infants and children living in homes with lead-based paint showed the opposite trend as seen in approach 2. The amount of drinking water lead needed to shift the probability of EBLLs in these populations is lower than that observed for the other soil/dust lead level scenarios assessed. Section 6.1.2 provides an explanation for this trend.

A.2.4 Results of Approach 2: Estimating the Concentration of Lead in Drinking Water That Increases a Geometric Mean BLL by a Defined Unit

The drinking water concentrations needed to result in a given increase in the geometric mean BLL of formula-fed infants, 0- to 6-month-olds, 1- to 2-year-olds, and 0- to 7-year-olds living in homes with lead-based paint are presented in Exhibit 52.

Exhibit 52. Summary of Results Demonstrating the Amount of Lead in Drinking Water Resulting in a 0.5 or 1 µg/dL Increase in Geometric Mean BLL for Infants and Children Living in Homes with Lead-Based Paint

Population Assessed	Drinking Water Concentration (µg/L) Resulting a 0.5 µg/dL Increase in the Geometric Mean BLL	Resulting Geometric Mean Blood Lead Level (µg/dL)	Drinking Water Concentration (µg/L) Resulting a 1 µg/dL Increase in the Geometric Mean BLL	Resulting Geometric Mean Blood Lead Level (µg/dL)
Formula-fed infants	3.2	1.39	6.6	1.89
0- to 6-month-olds	3.9	1.39	8.0	1.89
1- to 2-year-olds	11.6	1.50	23.5	2.00
0- to 7-year-olds	11.9	1.49	24.3	1.99

For each population living in homes with lead-based paint, the amount of drinking water needed to increase geometric mean BLL by a specified amount was greater than in the other soil/dust scenarios previously assessed (see Exhibit 29, Exhibit 30, Exhibit 31). This is because, as non-drinking water exposures increase, the amount of drinking water needed to produce a given increase in geometric mean BLL increases; this relationship is due to the way lead uptake is modeled in IEUBK and is discussed previously in Section 6.1.3.

Appendix B – Additional Information from SHEDS-IEUBK Analysis

Comparing SHEDS-IEUBK with Approach 1

Exhibit 53 summarizes SHEDS-IEUBK outputs using an "incremental risk approach," so that results can be compared to approach 1:

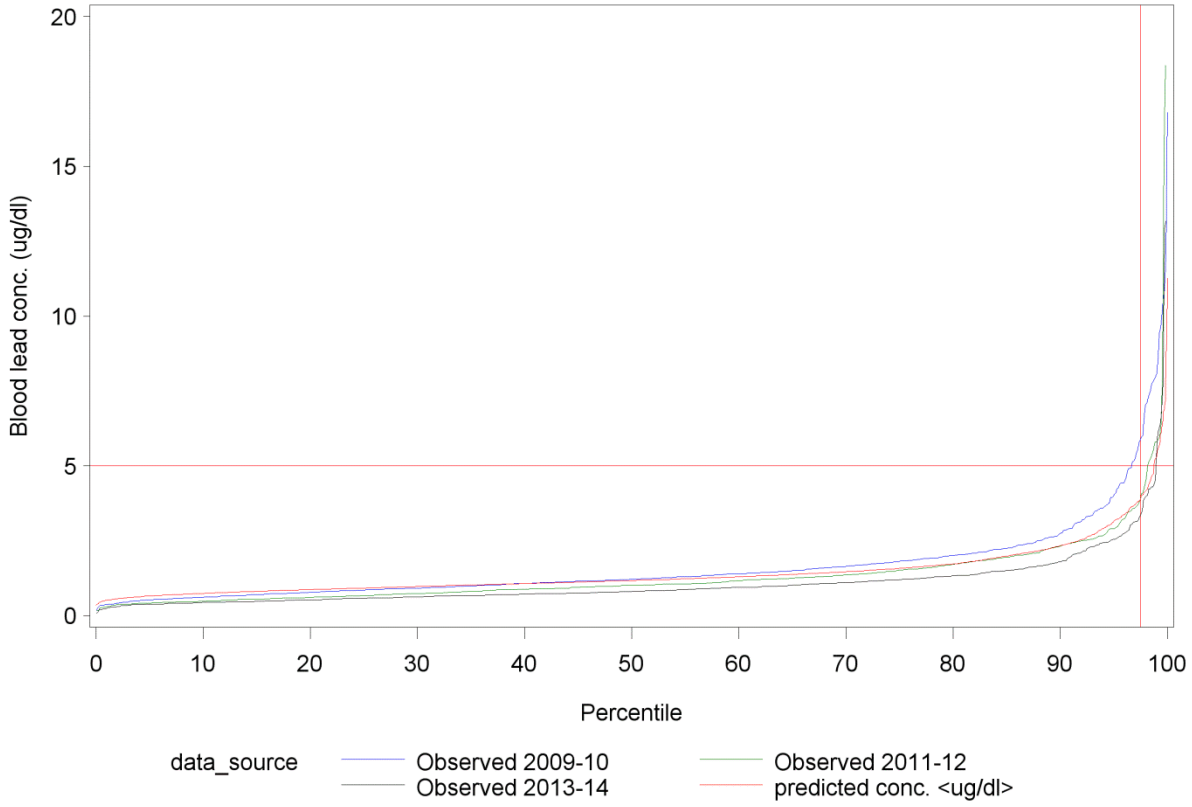
Exhibit 53. Water Lead Level (µg/L) Associated with a 1, 2.5, and 5% Increase in the Probability of EBLLs

Increase (%)	Age Group		
	0-6 Months	1 to 2 Years	0 to 7 Years
EBLL = 3.5 µg/dL			
1	3.5	6	4
2.5	5.2	10.5	8
5	6.8	14	11.5
EBLL = 5 µg/dL			
1	9.5	14	10.5
2.5	11.8	21	14.5
5	13	28	23

Evaluating SHEDS-IEUBK Results

A strength of approach 3 is that SHEDS-IEUBK predicted BLL could be evaluated against real-world NHANES BLL data for children of 1 to 7 years of age. For this evaluation, a population-weighted mean lead concentration of 0.89 µg/L was estimated from the 2009 EPA Office of Water Six-Year Review dataset (U.S. EPA, 2010a). NHANES data show a gradual reduction in children's BLLs from 2009-2010 to 2011-2012 and again from 2011-2012 to 2013-2014. As illustrated in Exhibit 54, the SHEDS-IEUBK predicted BLL distributions slightly underestimated 2009-2010 NHANES data, slightly overestimated 2013-2014 data, and agreed well with 2011-2012 data.

Exhibit 54. Evaluation of SHEDS-IEUBK Modeled BLL vs. 2009-2014 NHANES BLL



Another advantage of approach 3 is the ability to evaluate the contribution of all exposure pathways to BLL across the distribution of BLLs as illustrated in Exhibit 55. The use of central tendencies, such as by approaches 1 and 2, would only allow for the assessment of exposure pathway contributions at the median or geometric mean BLL.

Exhibit 55. Contribution of Different Exposure Pathways in SHEDS-IEUBK Modeled BLL