The estimated BLLs in are average adult BLLs given the corresponding estimated lead tap water concentrations resulting from LSL/GRR service line, CCT, POU, and pitcher filter status at steady-state, holding other exposures constant. In the SafeWater LCR model, water systems are tracked as they move from one LSL/GRR service line, CCT, pitcher filter or POU status to another as a result of rule implementation. The numbers of males and females in each age group served by those water systems are proportional to the age/sex makeup of the United States population as a whole. Age specific yearly BLLs are used in the benefit valuation modeling. shows the estimated change in average lifetime BLLs for adults who experience a change in water lead concentration as a result of pitcher filter use, LSL/GRR service line removal and/or installation of CCT or POU, rather than the set of initial LSL/GRR service line and CCT status combinations. Expected changes on average for all adults 40-80 due to changes in water concentrations due to the rule are displayed in Exhibit 5-23.

Exhibit 5-23: Estimated Lifetime Average Blood Lead Level Decrease for Adults Experiencing Alternate LSL/GRR, CCT, pitcher filter and POU Status Combinations

Pre-Rule Drinking Water			Post-Rule Drinking Water			Estimated Decrease in the Means of Blood Lead	
Lead Conc. (µg/L)	LSL Status	CCT Status	Lead Conc. (µg/L)	LSL Status	CCT Status	FEMALE: Ages 40-80 (µg/dL)	MALE: Ages 40-80 (µg/dL)
14.38	LSL	None	0.83	No LSL	None	1.36	1.05
14.38	LSL	None	4.27	LSL	Representative	1.01	0.78
14.38	LSL	None	0.83	No LSL	Representative	1.36	1.05
14.38	LSL	None	0.83	POU or pitcher filter		1.36	1.05
6.85	Partial/GRR	None	0.83	No LSL	None	0.6	0.47
6.85	Partial/GRR	None	2.14	Partial	Representative	0.47	0.37
6.85	Partial/GRR	None	0.83	No LSL	Representative	0.6	0.47
6.85	Partial/GRR	None	0.83	POU or pitcher filter		0.6	0.47
0.83	No LSL	None	0.83	No LSL	Representative	0	0
0.83	No LSL	None	0.83	POU or pitcher filter		0	0
7.93	LSL	Partial	0.83	No LSL	Partial	0.71	0.55
7.93	LSL	Partial	4.27	LSL	Representative	0.37	0.28
7.93	LSL	Partial	0.83	No LSL	Representative	0.71	0.55
7.93	LSL	Partial	0.83	POU or pitcher filter		0.71	0.55
3.84	Partial/GRR	Partial	0.83	No LSL	Partial	0.3	0.23
3.84	Partial/GRR	Partial	2.14	Partial	Representative	0.17	0.13
3.84	Partial/GRR	Partial	0.83	No LSL	Representative	0.3	0.23
3.84	Partial/GRR	Partial	0.83	POU or pitcher filter		0.3	0.23
0.83	No LSL	Partial	0.83	No LSL	Representative	0	0

Pre-Rule Drinking Water			Post-Rule Drinking Water			Estimated Decrease in the Means of Blood Lead	
Lead Conc. (µg/L)	LSL Status	CCT Status	Lead Conc. (µg/L)	LSL Status	CCT Status	FEMALE: Ages 40-80 (µg/dL)	MALE: Ages 40-80 (µg/dL)
0.83	No LSL	Partial	0.83	POU or pitcher filter		0	0
4.27	LSL	Representative	0.83	No LSL	Representative	0.35	0.27
4.27	LSL	Representative	0.83	POU or pitcher filter		0.35	0.27
2.14	Partial/GRR	Representative	0.83	No LSL	Representative	0.13	0.1
2.14	Partial/GRR	Representative	0.83	POU or pitcher filter		0.13	0.1
0.83	No LSL	Representative	0.83	POU or pitcher filter		0	0

Acronyms: LSL = lead service line; CCT = corrosion control treatment; POU = point-of-use; GRR = galvanized requiring replacement

5.5 Concentration Response Functions and Valuations used in the Estimation of Benefits to Children and Adults

The EPA undertook a rigorous process to identify concentration response functions to quantify benefits. This included reviewing all available studies which could be used to develop quantitative relationships between changes in lead exposure and/or changes in blood lead levels and changes in health endpoints. The EPA evaluated the studies for quality and potential biases. The EPA then developed a separate report for each health endpoint. In addition to the quality review findings, each report provides quantitative estimates, based on the identified functions, of potential changes in the health endpoint and was reviewed by EPA experts and/or externally peer reviewed. For the final LCRI the EPA has relied on concentration response functions for four quantified health endpoints that have been extensively reviewed by the agency and in the case of reductions in IQ losses, low birth weight and cardiovascular disease premature mortality, externally peer reviewed. Also, the approach used for IQ has been used in multiple prior rulemakings and undergone SAB review.

As with costs, the EPA estimated both high and low benefit scenarios for each health endpoint that is quantified. For lower birth weight, only one concentration response function was determined to be of high-quality, so this is used in both the high and low benefit scenario calculations. For IQ, ADHD, and CVD premature mortality, two or more functions were available, and the EPA selected the functions that gave the highest and lowest health benefit estimates across most blood lead levels.[173] For information on the uncertainties associated with the use of the selected concentration response functions see Section 5.7. The monetized benefit estimates provided in this chapter use the 2 percent discount rate as prescribed by the Office of Management and Budget's updated Circular A-4 (OMB Circular A-4, 2023).[174]

[173] As some of the functions are not linear, there are cases where these functions may not always give the highest or the lowest benefits.

[174] Because the EPA provided benefit estimates discounted at 3 and 7 percent for the proposed LCRI based on OMB guidance which was in effect at the time of the proposed rule analysis (OMB Circular A-4, 2003), the agency has also calculated the benefit impacts at both the 3 and 7 percent discount rates. See Appendix F for results.

5.5.1 *Concentration-Response Functions for Lead and IQ*

Previously, to estimate benefits supporting the 2021 LCRR, the EPA used a function based on Crump et al. (2013) in the main analysis and explored the choice of two additional IQ functions in the sensitivity analysis. Both functions in the sensitivity used the corrected Lanphear et al. (2005) function, as reported in Kirrane and Patel (2014): one with a low-dose linearization and the other without a low-dose linearization. To estimate avoided IQ loss in children for the final LCRI, the EPA selected two concentration-response functions. The low scenario benefits estimate is based on the study by Crump et al. (2013). The EPA chose the corrected Lanphear et al (2005, erratum 2019) function without low-dose extrapolation for the calculation of the high scenario benefit estimate for avoided IQ loss under the final LCRI. These studies were included in the EPA's SAB review of the 2021 LCRR (USEPA, 2020b).

This section provides an overview of these two key studies. Additional details of Crump et al. (2013) and Lanphear et al. erratum (2019) can be found in Appendix J of the Final 2021 LCRR EA (USEPA, 2020a), which provides more in-depth summaries of the key studies used in the concentration-response functions for the benefits analysis, as well as the Kirrane and Patel (2014) correction to the Lanphear et al. (2005) results, which was conducted prior to the publication of the Lanphear erratum.

Lanphear et al. erratum (2019) conducted a pooled analysis of seven international cohort studies that investigated the relationship between BLLs and full-scale IQ (the composite of verbal and performance IQ scores) in children 5–10 years old. The pooled study sample comprised 1,333 children, with a lifetime average BLL of 12.4 µg/dL. All the children underwent IQ testing with the Wechsler Intelligence Scale for Children. The mean IQ in the study sample was approximately 93 points. Associations between IQ and four different measures of BLLs in children were examined: concurrent (measurement obtained closest to the IQ test), maximum (peak value at any time before the IQ test), early (mean BLL from 6 to 24 months of age), and lifetime (mean BLL from 6 months of age to concurrent). For each of these measures, Lanphear et al. erratum (2019) estimated the relationship between BLLs and IQ by constructing an adjusted log-linear model.

Results of the Lanphear et al. erratum (2019) study showed that all blood lead measures were significantly associated with IQ loss, and were highly correlated with one another. Based on the R^2 values for each regression model (data not presented in the paper), Lanphear et al. erratum (2019) determined that concurrent BLLs were the strongest predictors of IQ, followed by lifetime average BLLs.

Exhibit 5-24 shows the beta estimates for the log-linear associations between each of the blood lead measures examined in Lanphear et al. erratum (2019). The estimated decreases in IQ associated with increases in concurrent BLLs from 2.4 to 10 µg/dL, 10 to 20 µg/dL, and 20 to 30 µg/dL were 3.8, 1.8, and 1.1 points, respectively. Consistent with the log-linear model, IQ deficits were greater at lower levels of lead exposures.

Changes in IQ associated with changes in BLLs for the high benefits scenario were estimated using Equation 9 below. Average BLLs for children age 0-7 (lifetime exposure) from the SHEDS-Pb modeling were used as inputs to the equation.

$$IQ\ Loss = \beta \times \ln\left(\frac{PbB_1}{PbB_2}\right) \quad \text{(Equation 9)}$$

Where:

β = Corrected lifetime beta estimate from Lanphear et al. (-3.25)

PbB_1 = Pre-rule BLL

PbB_2 = Post-rule BLL

In their 2013 paper, Crump et al. had two aims: 1) to perform a reanalysis of the methods in Lanphear et al. (2005), and 2) to conduct an independent analysis of the data from Lanphear et al. (2005). In the reanalysis, Crump et al. (2013) identified a few minor errors in the original Lanphear et al. (2005) paper. The correction of these minor errors resulted in slight changes to the regression coefficients but did not affect the main conclusions of the paper. These errors were confirmed by the EPA in a reanalysis by Kirrane and Patel (2014), which also reaffirmed that the main conclusions of Lanphear et al. (2005) remained unchanged, and Lanphear et al. erratum (2019) confirmed this in an *Erratum* of the original study. Kirrane and Patel (2014) additionally found that the early childhood blood lead measure had the highest R^2 value, though all R^2 values were similar.

In their independent analysis, Crump et al. (2013) made changes to the dataset used for final analysis (e.g., in selecting IQ measurements and defining blood lead measurements). Additionally, the authors opted to add 1 to the BLLs before log-transformation so that IQ loss was equal to 0 when BLL was 0, as shown in Equation 10.

$$IQ\ Loss = \beta \times \ln\left(\frac{PbB_1 + 1}{PbB_2 + 1}\right) \quad \text{(Equation 10)}$$

Where:

β = Lifetime beta estimate from Crump et al. (2013) independent analysis (-3.25)

PbB_1 = Pre-rule BLL

PbB_2 = Post-rule BLL

Changes in IQ associated with changes in BLLs for the low benefits scenario were estimated using Equation 10 based on the Crump independent analysis. As with the high benefit scenario, average BLLs for children ages 0-7 from the SHEDS-Pb model were used as inputs to the Equation 10.

For both equations, the SHEDS-Pb model estimated pre and post rule BLLs in children ages 0-7 are described in Section 5.4.

Exhibit 5-24: Comparison of Adjusted Coefficients from Lanphear et al. Erratum (2019) with Those Obtained in the Kirrane and Patel (2014), and the Reanalysis and Independent Analysis of Lanphear et al. (2005) by Crump et al. (2013)

BLL Variable	Kirrane and Patel (2014)		Lanphear et al. Erratum (2019)		Crump et al. (2013) Reanalysis ln(BLL)		Crump et al. (2013) Independent Analysis ln(BLL + 1)	
	β (95% CI)	R^2	β (95% CI)	R^{2a}	β (95% CI)	R^2	β (95% CI)	R^2
Early	-2.21 (-3.38, -1.04)	0.643	-2.21 (-3.38, -1.04)	n/a	-2.21 (-3.38, -1.03)	0.643	-2.46 (-3.82, -1.10)	0.659
Peak	-2.86 (-4.10, -1.61)	0.640	-2.86 (-4.10, -1.61)	n/a	-2.86 (-4.10, -1.61)	0.640	-2.48 (-3.83, -1.14)	0.656
Lifetime	-3.14 (-4.39, -1.88)	0.641	-3.25 (-4.51, -1.99)	n/a	-3.19 (-4.45, -1.94)	0.641	-3.25 (-4.66, -1.83)	0.659
Concurrent	-2.65 (-3.69, -1.61)	0.641	-2.65 (-3.69, -1.61)	n/a	-2.65 (-3.69, -1.61)	0.641	-3.32 (-4.55, -2.08)	0.658

Sources: Crump et al. (2013, Table 2 and Table 5), Kirrane and Patel (2014, Table 1), Lanphear et al. erratum (2019, Table 4).
[a] R^2 not reported in Lanphear et al. erratum (2019); however, the paper reported that the concurrent BLL was the largest R^2.
Notes: This table displays regression coefficients and R^2 values for the Lanphear et al. erratum (2019) analysis, the Crump et al. (2013) and Kirrane and Patel (2014) reanalysis of Lanphear et al. (2005), and the Crump et al. (2013) independent analysis of Lanphear et al. (2005). This table summarizes the relationship between BLL and IQ loss across various blood lead metrics.

As can be seen in Exhibit 5-24, the R^2 values are all similar: the strength of the relationship between BLLs and IQ loss appears to be similar regardless of the blood lead metric used. Because lifetime average BLLs are more reflective of the long-term changes in lead exposure anticipated under the final LCRI, the EPA chose to model the benefits under both the low and high benefit scenarios based on lifetime BLLs rather than concurrent BLLs.

No threshold has been identified for the neurological effects of lead (Schwartz and Otto, 1991; Budtz-Jørgensen et al., 2013; Crump et al., 2013; USEPA, 2024). Therefore, the EPA assumes that there is no threshold for this endpoint and quantified avoided IQ loss associated with all BLLs (Schwartz and Otto, 1991; Budtz-Jørgensen et al., 2013; Crump et al., 2013; USEPA, 2024). Budtz-Jørgensen et al. (2013), as well as the smaller cohort study of Min et al. (2009), used more recent BLLs than those used in the Crump and Lanphear analyses, and confirmed the results in Crump et al. (2013) and Lanphear et al. erratum (2019). Additionally, in Min et al. (2009), the steeper slopes at lower BLLs without log-transformation show increased IQ deficits, which provides additional evidence that reducing lead levels in the lower range of average BLLs has a significant impact on preventing IQ loss.

5.5.2 Valuation of Avoided IQ Loss

The economics literature provides a robust basis for estimating the relationship between IQ change and lifetime earnings. Because the literature relies on large datasets that are representative of the US population, it is appropriate to use the results to infer subpopulation-level impacts (though individual-level impacts) from changes in environmental policy, even when average impacts are very small in magnitude. The estimated effects of IQ on lifetime earnings are not predicated on a particular type or

pathway of pollutant exposure. Rather, they are broadly applicable to evaluating any type of policy intended to improve children's cognitive development (Lin et al. 2018).

The value of an IQ point used in the main analysis (both high and low scenarios) is derived from the EPA's (2019a) reanalysis of Salkever (1995), which estimates that a one-point change in IQ results in a mean 1.9 percent change in lifetime earnings for males and a mean 3.4 percent change in lifetime earnings for females. Lifetime earnings are estimated using the average of 10 American Community Survey (ACS) single-year samples (2008 to 2017) and projected cohort life tables from the Social Security Administration. Projected increases in lifetime earnings are then adjusted for direct costs of additional years of education and forgone earnings while in school. The USEPA (2019) reanalysis of Salkever (1995) estimates a mean change of 0.08 years of schooling per change in IQ point resulting from a reduction in lead exposure for males and a mean change of 0.09 years of schooling for females. This approach was reviewed by the EPA's SAB (USEPA, 2020b).

To estimate the uncertainty underlying the model parameters of the Salkever (1995) reanalysis, USEPA (2019a) used a bootstrap approach to estimate a distribution of model parameters over 10,000 replicates (using random sampling with replacement). For each replicate, the net monetized value of a one-point change in IQ is subsequently estimated as the gross value of an IQ point, less the value of additional education costs and lost earnings while in school.

Based on the mean value of the 10,000 sampling iterations, the USEPA (2019) estimated that the change in one IQ point discounted to age 7 is $42,226, in 2022 dollars, using a 2 percent discount rate. Note that the EPA's use of the term "2 percent discount rate" with regard to the calculation of the IQ point high and low values (which represent the present value of the change in lifetime earnings) is shorthand for a declining discount rate which begins with a 2 percent discount rate for the years 2024-2079, a 1.9 percent discount rate used for the years 2080-2096, and a 1.8 percent discount rate used in years 2095-2102. This declining rate structure was implemented to comply with updates to OMB Circular A-4 guidance which indicates that a declining discount rate may be used to capture the uncertainty in the appropriate discount rate over long time horizons like lifetime labor force participation.[175][176]

The Salkever IQ value is presented in 2022 dollars to be consistent with the cost estimates. As described in Section 5.6, benefits are further discounted back to year one of the analysis and annualized within the SafeWater LCR model. A summary of the Salkever component values, by sex, can be found in Exhibit 5-25.

[175] The revised Circular A-4 discusses discounting over long time horizons (OMB Circular A-4 2023). As noted by OMB in the updated Circular A-4, "[t]here are various reasonable approaches to long-term discounting that account for uncertainty and other relevant factors, and therefore lead to dynamic discount rates over time." When the time horizon of an analysis is sufficiently long (i.e., 2080 or beyond), use of a declining discount rate may be appropriate to capture uncertainty in the discount rate over long time horizons.

[176] Note that the declining discount rate structure was not used in the proposed rule calculation of IQ point values and the EPA has continued to use the constant discount rate IQ point values in the 3 and 7 percent benefit calculations found in Appendix F.

Exhibit 5-25 Updated Estimates for Lifetime Earnings, Additional Education Costs, and Lost Earnings from Additional Education (2022 USD), discounted at 2 percent to age 7

Estimate	Updated Salkever Estimates		
	Male	Female	Male and Female Combined
1. Lifetime Earnings	$2,174,849	$1,424,497	-
2. IQ Effect	1.87%	3.41%	-
3. IQ Effect*Lifetime Earnings	$40,700	$48,559	$44,551
4. Additional Education Costs	$1,702	$1,940	$1,819
5. Lost Earnings (from additional education)	$594	$415	$506
6. Value of an IQ Point (3 - (4+5))	**$38,404**	**$46,204**	**$42,226**

Note: The EPA uses of the term "2 percent discount rate" with regard to the calculation of the IQ point high and low estimates is shorthand for a declining discount rate which begins with a 2 percent discount rate for the years 2024-2079, a 1.9 percent discount rate used for the years 2080-2096, and a 1.8 percent discount rate used in years 2095-2102. This declining rate structure was implemented to comply with updates to OMB Circular A-4 guidance.

See Appendix F for a Sensitivity Analysis with an alternative value for IQ benefits based on Lin et. al. (2018). For additional discussion of the methods, also see Appendix J of the Final 2021 LCRR EA (USEPA, 2020a) and Appendix A of USEPA (2024c).

5.5.3 Concentration-Response Function for Lead and ADHD

This is the first regulation in which the EPA has estimated benefits of avoided cases of ADHD associated with reductions in lead exposure; as discussed below the approach for quantifying such benefits will continue to evolve as our understanding of the potential relationship improves. As described in Appendix D the USEPA (2024b) ISA strengthened the conclusions of the 2013 ISA and concluded that there was a causal relationship between lead exposure and inattention, impulsivity, and hyperactivity in children based on recent studies of children with group mean BLLs ≤5 µg/dL. The 2024 ISA states that "prospective studies of ADHD, including a study of clinical ADHD that controlled for parental education and SES [Socioeconomic status], although not quality of parental caregiving reported positive associations" (USEPA, 2024b. p. IS-30).The causes of ADHD are not fully understood, but research suggests a number of potential causes, including genetics, exposure to environmental toxins, prenatal cigarette smoking or alcohol intake, and brain changes (Tripp and Wickens, 2009; Pliszka et al., 2007). The EPA's 2013 lead ISA statedthat in children, "attention was associated with biomarkers of Pb exposure representing several different lifestages and time periods. Prospective studies did not examine a detailed Pb biomarker history, and results do not identify an individual critical lifestage, time period, or

duration of Pb exposure associated with attention decrements in children. Associations in prospective studies for attention decrements with tooth Pb level, early childhood average and lifetime average blood Pb levels point to an effect of cumulative Pb exposure." The 2024 ISA addresses the uncertainties presented in the 2013 ISA by stating that "The largest uncertainty addressed by the recent evidence base is the previous lack of prospective studies examining ADHD (Appendix 3.5.2.4–3.5.2.5). The bulk of the recent evidence comprises prospective studies that establish the temporality of the association between Pb [lead] exposure and parent or teacher ratings of ADHD symptoms and clinical ADHD. Across studies, associations were observed with tooth Pb concentrations, childhood BLLs (<6 µg/dL), and with maternal or cord BLLs (2–5 µg/dL)." The available studies relating blood lead to ADHD use one-time BLLs, while it is possible that cumulative exposure is also important. However, one-time and cumulative measures of BLLs in children are often correlated. Therefore, the EPA has chosen diagnosed cases of ADHD as an endpoint in this benefits analysis, because literature exists linking ADHD diagnosis to these monetizable outcomes. The larger body of literature on attention, impulsivity, and hyperactivity symptoms in children supports this association. The EPA chose a higher and lower concentration-response function for the estimates of avoided cases to partially address the uncertainty in the most appropriate function to use in estimating avoided cases due to the rule. Additional future research will help to further understand the critical exposure window (thus exposure metric), the mode of action of lead in the development of ADHD and/or related symptoms, and the interplay with genetic factors and exposures to other substances.

The approach used to quantify ADHD here is based on review and analysis that Abt Associates (Abt Associates, 2022a) conducted under contract to the EPA.

For the LCRI, the EPA estimates the benefits based on avoided cases of ADHD in children due to the rule. The EPA chose a higher and lower concentration-response function for the estimates of avoided cases to partially address the uncertainty in the most appropriate function to use in estimating avoided cases due to the final rule.

This section provides a brief overview of two studies that inform the high and low benefit estimates for ADHD. Froehlich et al. (2009) forms the basis of the high benefits estimates, and Ji et al. (2018) forms the basis of the low benefits estimates. The selection of these studies is summarized in a report prepared for the EPA (Abt Associates, 2022a) Additionally, see Section 5.7.5 for a discussion on the strengthened evidence addressing the uncertainty in the relationship between Pb and ADHD presented in the 2024 Pb ISA.

Froehlich et al. (2009) aimed to investigate the associations between ADHD and childhood lead exposures, both independently and in combination with prenatal tobacco exposures. The authors analyzed data from 2001-2004 NHANES on 2,588 children aged 8 to 15 years old with complete information on ADHD diagnosis, lead and tobacco exposures, and additional covariates. Children with high serum cotinine levels (>10 ng/mL), were excluded from the study to prevent confounding of the effects of secondhand tobacco exposure. In the main analyses, ADHD diagnosis in NHANES was based on completion of the Diagnostic Interview Schedule for Children (DISC) by caregivers. The DISC is a structured interview that contains questions on ADHD symptoms, onset, pervasiveness, and severity in the last 12 months and uses DSM-IV[177] criteria to diagnose ADHD. As a secondary outcome, the

[177] Diagnostic and Statistical Manual of Mental Disorders

definition of ADHD diagnosis was expanded to capture children with ADHD who did not meet full DSM-IV criteria due to appropriate medication treatment. In these secondary analyses, children that had a caregiver report both a history of ADHD diagnosis and ADHD medication use in the past year were additionally included in the analyses. The authors investigated variables that had previously been shown to be associated with ADHD as potential confounders. In the secondary analyses, health insurance status was also examined as a covariate. Logistic regression analyses were used to examine associations between lead exposures and ADHD, adjusted for confounders that were confirmed to be significantly associated with ADHD (χ^2 test, $p < 0.2$). The final logistic regression model was adjusted for sex, age, race/ethnicity, preschool attendance, birth weight[178], income/poverty ratio, maternal age at child's birth, and both current secondhand and prenatal tobacco exposures (operationalized by serum cotinine levels and via maternal report, respectively). Additional analyses were performed restricting the sample to children with blood lead < 5 µg/dL. Joint toxicant (i.e., both lead and tobacco exposure) effects were assessed by examining ADHD incidence at varying levels of co-exposures.

Froehlich et al. (2009) found that 8.7% of children studied met DSM-IV criteria for ADHD diagnosis. Children in the highest tertile of lead exposure (>1.3 µg/dL) were 2.3 times more likely to be diagnosed with ADHD (95% CI, 1.5-3.8) than children in the lowest tertile (0.2 to 0.8 µg/dL). The same adjusted odds ratio (OR) was observed when restricting the sample to children with blood lead < 5 µg/dL. When blood lead was logarithmically transformed and analyzed as a continuous variable, the adjusted OR for ADHD diagnosis was 1.8 (95% CI, 1.2-2.8) given a one-unit increase in natural log blood lead[179]. The significant association between lead exposures and ADHD remained when the definition of ADHD diagnosis was expanded in the secondary analyses: the adjusted OR was 2.0 (95% CI, 1.3-3.0). Childhood lead and prenatal tobacco exposures combined had a multiplicative effect on the risk of ADHD. Froehlich et al. (2009) estimated that 25% of ADHD cases among U.S. children with blood lead > 1.3 µg/dL are attributable to lead exposures, corresponding to approximately 598,000 cases.

Results of Froehlich et al. (2009) were consistent with prior studies that found a relationship between childhood lead exposures and DSM-IV ADHD diagnosis. The use of a national, population-based sample of children with low blood lead makes results generalizable to the U.S. population of children. The possibility of residual confounding from unmeasured genetic and environmental confounders (e.g., prenatal alcohol exposure) or parental characteristics remains. Because of small sample sizes for each subtype, the authors could not investigate associations between blood lead and specific ADHD subtypes.

Ji et al. (2018) investigated the relationship between early childhood exposure to lead (blood leads were measured prior to age 4) and the risk of being diagnosed with ADHD using a prospective cohort design, including effect modification by sex, maternal high density lipoprotein (HDL) levels, and stress during pregnancy. Data from the Boston Birth Cohort were utilized in this study. The Boston Birth Cohort includes mother-infant pairs enrolled at birth from the Boston Medical Center. Enrollment is on a rolling basis since 1998, and at the time of this study 3098 mother-infant pairs had enrolled in the post-natal follow-up study. After excluding mother-infant pairs due to missing data, lead measurements taken after an ADHD diagnosis, incorrect measurement dates, age over 4 years at measurement, and lead levels higher than 10 µg/dL, the final analysis including 1479 pairs.

[178] Birth weight could be one pathway through which Pb exposure affects ADHD.
[179] Per Joseph Braun, personal communication to Meghan Lynch

Data were collected using a questionnaire, electronic medical records, and maternal blood samples obtained 24 to 72 hours after delivery. A questionnaire was used to collect data from mothers on demographic characteristics, stress during pregnancy, and smoking status. Birthweight, gestational age, parity, intrauterine infections, complications, child lead levels, and ADHD diagnostic codes were obtained from electronic medical records. If a child had repeated lead measures, the earliest measurement taken was used for analysis. If a child's electronic medical record contained a diagnostic code for ADHD, the child was enrolled in the ADHD group. Children in the neurotypical group were not diagnosed with any of the ADHD codes, nor were they diagnosed with autism spectrum disorder, conduct disorders, developmental delays or intellectual disabilities, failure to thrive or congenital anomalies. HDL and lead levels were measured in maternal blood samples taken between 24 to 72 hours after delivery.

To examine the concentration-response relationship between lead and ADHD diagnosis, the authors used categorical and continuous multiple logistic regression, and adjusted for maternal age at delivery, mode of delivery, maternal race/ethnicity and education, smoking status during pregnancy, intrauterine infection, parity, child's sex, preterm birth, and birthweight in all models (except sex when it was included as joint or interaction term in the models). Additional analyses were conducted to investigate the effects of sex on the lead-ADHD relationship.

Ji et al. (2018) found elevated lead levels at 5-10 µg/dL were associated with a 66% increase in risk of an ADHD diagnosis, OR=1.66 (95% CI, 1.0-2.56), compared to children with lead levels less than 5 µg/dL. The natural log-transformed linear lead levels were associated with an increased risk of ADHD diagnosis (OR=1.25, 95% CI, 1.01-1.56). In joint association analyses, the effects of lead on the risk of ADHD diagnosis were attenuated in both stratified and joint effects models for females. For males, risk of ADHD diagnosis was 2.5 times higher when lead levels were 5-10 µg/dL compared to lead levels <5 µg/dL (OR=2.49, 95% CI, 1.46-4.26). Findings were similar in Cox proportional hazards models.

This main health impact function is applied to both the Froehlich et al. (2009) and Ji et al. (2018) studies[180]. Regression coefficients (βs) are summarized below the equation.

$$\Delta \boldsymbol{ADHD} = \left[\boldsymbol{p_0} - \frac{\boldsymbol{p_0}}{(\mathbf{1} - \boldsymbol{p_0}) \times \boldsymbol{e}^{-\beta_1 [\ln(\boldsymbol{Blood\ Pb_i}) - \ln(\boldsymbol{Blood\ Pb_f})]} + \boldsymbol{p_0}} \right] \times \boldsymbol{pop} \qquad \text{(Equation 11)}$$

Where:

p_0 = Baseline rate of ADHD in the population of interest

β_1 = Beta estimate from study: 0.223 using Ji et al. (2018) or 0.588 using Froelich et al. (2009)

$Blood\ Pb_i$= Initial blood lead (µg/dL)

$Blood\ Pb_f$= Final blood lead (µg/dL)

pop = Number of children in the population of interest

[180] A derivation of this function can be found in Abt Associates (2023).

Ji et al. (2018) measured early childhood BLLs, therefore, in the SafeWater LCR model analyses (see Section 5.6) the blood lead outputs from the SHEDS-Pb models were used, as these are more relevant to younger children. Benefits based on Ji et al. (2018) are captured at age 7, assuming all children over the analysis period are diagnosed with ADHD at age 7. This is the basis of the low benefits estimates for ADHD.

Froelich et al. (2009) measured BLLs in children ages 8-15. Therefore, output from the AALM model was used in the SafeWater LCR model analyses to estimate BLLs in that age group. Benefits using Froelich et al. (2009) are captured at age 11, assuming all children over the analysis period are diagnosed with ADHD at age 11.

For both the high and low benefit calculations, the baseline rate of ADHD is assumed to be 9.6 percent based on Danielson et al. (2018).[181]

5.5.4 Valuation of Avoided ADHD

This analysis applies a valuation for ADHD cases based on a study by Doshi et al. (2012) following a similar approach to that used in the EPA's (2023a) Economic Analysis of Updated Soil Lead Guidance for Sites and Facilities Being Addressed Under CERCLA and RCRA Authorities.

To value each case of ADHD avoided, the USEPA (2023a) applied the following values obtained from Doshi et al. (2012) for annual per-person incremental costs in 2023 dollars covering the following cost categories:

- Children/Adolescent costs
    - Health care (patient); ages 0-21: $2,348
    - Health care (family); ages 0-18: $1,930
    - Productivity losses (family); ages 0-18: $326
    - Education; ages 5-18: $4,680
    - Justice system; adolescents aged 13-17: $362

- Adult costs
    - Health care (patient); ages 18-64: $2,680
    - Health care (family); ages 19-44: $1,330
    - Justice system; ages 18-28: $2,405

As described in Section 5.5.3 two different concentration response functions are used for the high and low scenarios. Ji et al. (2018) measured early childhood BLLs. Benefits based on Ji et al. (2018) are captured at age 7, assuming all children over the analysis period are diagnosed with ADHD at age 7. This is the basis of the low benefits estimates for ADHD. Froelich et al. (2009) measured BLLs in children ages 8-15. Benefits using Froelich et al. (2009) are captured at age 11, assuming all children over the analysis period are diagnosed with ADHD at age 11. Therefore, for the valuation in the low scenario, costs for

[181] Note the EPA updated the baseline rate of ADHD based on Danielson et al. (2018). In the EPA assessment for the "Updated Residential Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities" the agency used a baseline rate for ADHD of 10.2 percent from Xu et al. (2018).

children 0-6 are not included in the estimate. For the high scenario, costs for children 0-10 are not included in the estimate.

There is uncertainty about what percent of ADHD cases persist into adulthood. Therefore, for the final LCRI rule analysis, the EPA uses a high and low estimate of the ADHD cost of illness, based on a high and low estimate of ADHD persistence into adulthood.

The high analysis assumes that 90 percent of childhood cases of ADHD persist into adulthood, based on Sibley et al. (2022) and as used in USEPA (2024c). This assumption is used to adjust the healthcare and justice system benefits realized at ages 18 and older for an avoided case of ADHD diagnosed in childhood. The assumption is derived from Sibley et al. (2022)'s finding that 9.1 percent of childhood cases (mean age 8 years) recovered from ADHD at the study's final 16-year follow up (mean age 25 years, sample size 558). Recovery was defined as a full remission of ADHD sustained for at least two consecutive study assessments (conducted approximately every two years). However, the authors find that most cases have ADHD symptoms and impairments that fluctuate over time, and only a small percentage are stable into adulthood, either as persistent case or full recovery status. For example, at the final 16-year follow-up, 39.7 percent of participants were categorized as having persistent ADHD (defined using DSM-5 symptom thresholds) and 45.7 percent were categorized with partial remission. These participants were comprised of a mix of those with stable persistence (10.8%) or partial remission over all study time periods (15.6%), and a majority with fluctuating occurrence of symptoms over time (63.8%).

In sum, while this analysis assumes that 90 percent of childhood ADHD diagnoses persist into adulthood, only a fraction of those cases are likely to meet the full DSM diagnostic criteria and/or present stable symptoms in each year of adulthood. Thus, the high analysis may potentially overestimate ADHD benefits resulting from the final rule to the extent that these variances are not captured in the cost-of-illness estimates for the value of an avoided case of ADHD.

The low estimate is based on Barbaresi et al. (2013) which reports a 29.3 percent persistence rate, where persistence is defined according to the number of ADHD symptoms in adulthood that exceed two standard deviations of the mean number of symptoms in non-ADHD controls. Barbaresi et al. (2019) is based on a small sample size (367) and the population is nearly all white, and focused on Rochester, Minnesota, which the authors describe as geographically isolated in southeastern Minnesota. The study categorizes itself as the only study to not look at cases referred to a specialty treatment program. It is possible this is an underestimate of persistence given that it excludes some cases of partial ADHD symptoms, which are likely to yield social costs. Given the range of persistence into adulthood, the EPA chose 29% as the lower bound.

The high and low net present value estimates of all avoided ADHD costs incurred through age 64 are presented in Exhibit 5-26 in 2022 dollars. The values have been discounted back to age 7 for use with Ji et al. and back to age 11 for use with Froelich et al. using a 2 percent discount rate. Once captured, SafeWater further discounts back to the first analysis year. [182]

[182] Because the EPA provided benefit estimates discounted at 3 and 7 percent for the proposed LCRI based on OMB guidance which was in effect at the time of the proposed rule analysis (OMB Circular A-4, 2003), the agency has also calculated the ADHD benefit impacts at both the 3 and 7 percent discount rates. In the calculation of these

Exhibit 5-26: Present Value of Avoided ADHD Cases 2022 USD, Per Case

Assumed Persistence of ADHD Into Adulthood	Age at ADHD Diagnosis	2% Discount Rate
90%	11 (High- Froelich)	$184,149
29.3%	7 (Low- Ji)	$128,559

Note: The EPA uses of the term "2 percent discount rate" with regard to the calculation of the ADHD high and low estimates is shorthand for a declining discount rate which begins with a 2 percent discount rate for the years 2024-2079, a 1.9 percent discount rate used for the years 2080-2085. This declining rate structure was implemented to comply with updates to OMB Circular A-4 guidance.

5.5.5 Concentration-Response Function for Lead and Birth Weight of Infants Born to Women of Child-Bearing Age

In this analysis, women of childbearing age are represented by the population of women between the ages of 17-45 years old. The EPA utilized the AALM to generate estimates of blood lead in women of childbearing age. Zhu et al. (2010) was used to develop a concentration-response function for the birth weight of children born to these women for both the high and low benefit scenarios as this was the only study of suitable quality for benefits analysis (see Abt Associates, 2022b).[183]

Zhu et al.'s study, *Maternal Low-Level Lead Exposure and Fetal Growth* (2010), examined the association between low-level (<10 µg/dL) lead exposure and decreased fetal growth, specifically measures of birth weight, pre-term birth, and small for gestational age. In their retrospective cohort study, Zhu et al. matched the blood lead records from New York State's Heavy Metals Registry (HMR)[184] to birth certificate data for singleton births in the state of New York for 43,288 mother–infant pairs from upstate New York (New York State excluding New York City). The mothers were 15–49 years of age in 2003–2005.[185] The study restricted the cohort to mothers with blood lead levels < 10 µg/dL. The mean and median blood lead levels for the cohort were 2.1 µg/dL and 2 µg/dL, respectively. The mean birth weight was 3,331 grams.

To assess the relationship between maternal blood lead and the continuous outcomes (e.g., birth weight in grams), Zhu et al. (2010) used a multiple linear regression with fractional polynomials (Royston et al.

benefits the EPA has used ADHD case values that are derived by discounting at the constant 3 and 7 percent rates. See Appendix F for ADHD case values and benefit results discounted at 3 and 7 percent.

[183] An earlier version of this report describing the choice of Zhu et al. was peer reviewed in 2015 as part of the External Peer Review of the *EPA's Approach for Estimating Exposures and Incremental Health Effects from Lead due to Renovation, Repair, and Painting Activities in Public and Commercial Buildings,*

[184] Starting in 1992, New York State began requiring that all lead test results be reported to the HMR. The authors pulled data on potential confounding factors from the birth certificate files.

[185] For any individuals who had more than one blood lead measurement, a single measurement was selected at random. Additionally, for any mothers who had more than one child between 2003 and 2005, only one birth was selected, also at random.

1999). They explored one or two terms of fractional polynomials in terms of x^p where the power of *p* was -2, -1, -0.5, 0.5, 1, 2, and 3, and also used a natural logarithmic transformation of lead.[186]

The authors state that the model that assumed a linear relationship between birth weight and the square root of blood lead fit the data better than models with all other combinations of fractional polynomials. The final model developed by Zhu et al. (2010) was adjusted for timing of the lead test, gestational age, maternal age, race, Hispanic ethnicity, education, smoking, alcohol drinking, drug abuse, in wedlock, participation in special financial assistance program, parity, and infant sex. The concentration-response relationship from Zhu et al. is:

$$BW = b_0 + \left(-\frac{27.4\text{g}}{\mu\frac{\text{g}}{\text{dL}}} \times PbB^{0.5} \right) \qquad \text{(Equation 12)}$$

Where:

BW = Birth weight in grams

b_0 = Birth weight when blood lead is equal to 0 µg/dL[187]

PbB = Blood lead in µg/dL

The results from the study are presented in Exhibit 5-27, which shows that changes in birth weight associated with a 1 µg/dL change in blood lead vary based on the starting blood lead concentration. For example, the reduction in birth weight from a change in blood lead from 0 to 2 µg/dL is approximately 40 grams and from 8 to 10 µg/dL is approximately 10 grams. As Zhu points out, "the model predicts the strongest estimated effects at the lowest levels of exposure, without a lower threshold of PbB [blood lead] below which there would be no predicted effect on birth weight" (p. 1473).

[186] While 0.5 is not listed in the methods of Zhu et al. (2010), this is stated to be the resulting best fit model; therefore, it is included our list.

[187] The birthweight when blood lead is equal to zero was not provided in the paper however from Figure 1 it appears to be approximately 3,310 g.

Exhibit 5-27: Association between a Change in Blood Lead Concentration and Birth Weight, Upstate New York, 2003–2005 from Zhu et al. (2010)

Change in Blood Pb Concentration (µg/dL)	Estimate (grams)	95% CI (grams)
0	Reference	-
1	-27.4	-17.1 to -37.8
2	-38.8	-24.1 to -53.4
3	-47.5	-29.6 to -65.4
4	-54.8	-34.2 to -75.5
5	-61.3	-38.2 to -84.4
6	-67.2	-41.8 to -92.5
7	-72.5	-45.2 to -99.9
8	-77.6	-48.3 to -106.8
9	-82.3	-51.2 to -113.3
10	-86.7	-54.0 to -119.4

Source: Table 3 from Zhu et al. (2010).
Notes: 1) The model was a linear regression with fractional polynomials after adjustment for timing of Pb test, gestational age, maternal age, race, Hispanic ethnicity, education, smoking, alcohol and drinking, drug abuse, in wedlock, participation in special financial assistance programs, parity, and infant sex. Blood Pb concentration was transformed using a square root. The coefficient was -27.4 with a standard error (SE) of 5.3.
2) In the LCRI analysis, modeled blood lead levels do not exceed 2.35 µg/dL.

5.5.6 Valuation of Avoided Reductions in Birth Weight

The valuation of changes in birth weight is based on an approach further described in Abt Associates (2022c) which was finalized after undergoing peer review coordinated by the EPA.[188]Their analysis of U.S. Department of Health and Human Services, Medical Expenditure Panel Survey (MEPS) data found that birth weight in the very low birth weight (VLBW)/ low birth weight (LBW) and normal ranges influences medical expenditures. The report provides simulated cost changes based on inpatient hospital stays. Since these models were non-linear, Abt Associates (2022c) conducted simulations to understand the magnitude of the overall effect of birth weight on expenditures.

Using birth weight spline specifications, the authors found the simulated cost changes for increases in birth weight are negative and significant in the VLBW, LBW, and normal birth weight ranges in models that do not also control for a preterm birth indicator[189] (see Exhibit 5-28). The effects are largest at lower starting birth weights. For an increase of 0.22 lb, expenditures for inpatient hospital stays

[188] Note this methodology was externally peer review, see MDB Inc. (2022).
[189] Due to strong negative correlation between birth weight and preterm birth, there are fewer significant results in the VLBW range when the preterm indicator is included.

decrease by $1,652[190] at the VLBW threshold of 3.3 lbs, and less than $100 at the normal birth weight threshold of 5.5 lbs.

Exhibit 5-28: Simulated Cost Changes (2010 USD) on Annual Medical Expenditures for Inpatient Hospital Stays, using Birth Weight Spline Specifications (N with Positive Expenditures = 450)

Birth Weight (lbs)	BW Splines (excluding Preterm)		
	+0.04 lb	+0.11 lb	+0.22 lb
2	-974.24 (573.13)*	-2,375.82 (1,395.14)*	-4,560.19 (2,669.45)*
2.5	-663.98 (376.82)*	-1,618.46 (915.69)*	-3,104.15 (1,747.07)*
3	-449.22 (240.68)*	-1,094.45 (583.64)*	-2,097.43 (1,109.73)*
3.3	-354.03 (180.93)*	-862.28 (438.13)*	-1,651.66 (831.06)**
4	-200.83 (87.76)**	-488.77 (211.65)**	-935.06 (398.76)**
4.5	-132.60 (49.29)***	-322.52 (118.43)***	-616.44 (221.78)***
5	-86.76 (26.01)***	-210.92 (62.23)***	-402.74 (115.69)***
5.5	-16.35 (6.85)**	-40.55 (16.91)**	-79.99 (33.09)**
6	-14.42 (5.61)**	-35.75 (13.83)**	-70.51 (27.05)**
7	-11.18 (3.66)***	-27.71 (9.02)***	-54.65 (17.61)***
8	-8.64 (2.29)***	-21.41 (5.64)***	-42.21 (10.99)***
9	10.63 (9.96)	26.93 (25.51)	55.03 (53.17)
10	15.47 (22.73)	39.14 (58.63)	79.86 (123.71)

Notes: 1) Results show mean and standard error of the difference between simulated cost for baseline birthweight (left) and each birth weight increase. Significance estimates for the difference are indicated at the 1% (***), 5% (**), and 10% (*) levels.
2) Results are based on the log-log model (probability) and a gamma distribution (expenditures), which appear to fit the data best (see Appendix D). Estimates are averaged over all infants/toddlers (including those with and without non-zero expenditures) up to age two years.

[190] In 2010 United States Dollars.

Exhibit 5-29: Simulated Cost Changes (2010 USD) on Annual Medical Expenditures for Inpatient Hospital Stays, for Birth Weight Indicator and a Pre-term Indicator Only Model (N with Positive Expenditures = 450)

Change in Indicator Value	Model with Indicators for LBW and Preterm Birth		Model with LBW Indicator (excluding Preterm)	Model with Preterm Indicator (Excluding BW)
	Simulated Change: LBW	Simulated Change: Preterm	Simulated Change: LBW	Simulated Change: Preterm
0 to 1	3,088.13 (1,154.62)***	949.29 (359.67)***	4,203.38 (1,278.27)***	2,316.15 (563.47)***

Notes: 1) Results show mean and standard error of the difference between simulated cost at each indicator variable value (0 to 1 for either LBW or Preterm indicator variables). Significance is indicated at the 1% (***), 5% (**), and 10% (*) levels.
2) Results are based on the log-log model (probability) and a gamma distribution (expenditures), which appear to fit the data best (see Appendix D). Estimates are averaged over all infants/toddlers (including those with and without non-zero expenditures) up to age two years.

In the SafeWater LCR model, costs are inflated to 2022 dollars in order to be consistent with the timeframe chosen for the regulatory analysis (using a multiplier based on GDP[191]).

Applying the cost of illness (COI) value in the benefits calculation is done by following the steps:

Step 1. Calculate the change in birth weight due to the rule. Outputs from Zhu et al. (2010) for each change in LSL/GRR service line, CCT, POU or pitcher filter use scenario provide this output.

Step 2. Calculate the valuation of the change in birth weight due to the rule based on the proportion of infants born at each birth weight. Because Abt Associates (2022) estimated COI values for three discrete changes in birth weight (0.04 lb, 0.11 lb, or 0.22 lb; or 20 grams, 50 grams, or 100 grams), this results in the assumption that changes in birth weight below 0.04 lb have no value[192], changes of 0.04 lb to below 0.11 lb have a value equal to the COI presented for 0.04 lb changes, changes of 0.11 lb to below 0.22 lb have a value equal to the COI presented for 0.11 lb changes, and changes of 0.22 lb and above have a value equal to the COI presented for 0.22 lb changes. We assume that any change in birth weight resulting from the rule impacts infants with baseline birth weights equal to the distribution of birth weights in the United States (see Exhibit 5-30. Using this distribution, the EPA calculates the valuation of the change in birth weight due to the rule using the following equation:

[191] The EPA used the U.S. Bureau of Economic Analysis Table 1.1.9 Implicit Price Deflators for Gross Domestic Product (the May 30, 2024 revision) to adjust dollar values to 2022. See: https://apps.bea.gov/iTable/?reqid=19&step=3&isuri=1&1921=survey&1903=13#eyJhcHBpZCI6MTksInN0ZXBzIjpbMSwyLDMsM10sImRhdGEiOltbIk5JUEFfVGFibGVfTGlzdCIsIjEzIl0sWyJDYXRlZ29yaWVzIiwiU3VydmV5Il0sWyJGaXJzdF9ZZWFyIiwiMjAxNiJdLFsiTGFzdF9ZZWFyIiwiMjAyMiJdLFsiU2NhbGUiLCIwIl0sWyJTZXJpZXMiLCJBIl1dfQ==

[192] In reality, there is likely value below this level and therefore this analysis results in an underestimate of benefits.

Value of Change in Birth Weight = $\sum_{bw2}^{bw10}(|\boldsymbol{VH_{bw,d} * P_{bw} * pop}| + |\boldsymbol{2 * VB_{bw,d} * P_{bw} * pop}|)$ (Equation 13**)**

where:
$\sum_{bw2}^{bw10} =$ Sum of "value" equation above for each birth weight listed in Exhibit 5-30 below;
$VH_{bw,d}$ = Savings in initial birth-related hospital stay expenditures for the applicable 0.04 lb, 0.11 lb, or 0.22 lb birth weight change (*d*) for the applicable baseline birth weight (*bw*);
$VB_{bw,d}$ = Savings in annual hospital stay expenditures in the first two years of life for the applicable 0.04 lb, 0.11 lb, or 0.22 lb birth weight change (*d)* for the applicable baseline birth weight (*bw*);
P_{bw} = Proportion of total births that belong to a particular baseline birth weight (*bw*); and
pop = Number of children born to number of women of childbearing age in each option scenario (the annual fertility rate is 62.5 births per 1,000 women aged 15–44 in 2015).

Exhibit 5-30: Distribution of Birth Weights in the United States

Birth Weight (lbs)	Proportion of Total Births
2	0.7%
2.5	0.3%
3	0.3%
3.3	0.5%
4	0.9%
4.5	1.3%
5	2.4%
5.5	4.1%
6	13.5%
7	33.2%
8	29.4%
9	11.1%
10	2.4%

Source: Distribution based on CDC WONDER data for 2014 (CDC. 2015).

5.5.7 Concentration-Response Function for Lead and Cardiovascular Disease Premature Mortality

In their review of the proposed LCRR, the EPA's SAB stated, "benefits associated with reduced lead exposure and associated reduction in hypertension/cardiovascular effects have been well documented

(Chowdhury et al. 2018) and should be monetized and included in the EA" (USEPA, 2020b, p.15). For the LCRI, the EPA uses a methodology to estimate avoided cases of CVD premature mortality[193] due to reductions in lead exposures developed in Brown et al. (2020) and Abt Associates (2023).[194] In order to quantify the benefits of avoided cases of CVD premature mortality, Brown et al. (2020) and Abt Associates (2023) identified four studies providing a total of five concentration-response functions relating adult BLLs to CVD premature mortality. Because, uncertainty exists regarding the lead exposure level, timing, frequency, and duration contributing to the associations observed between a single adult blood lead measurement and CVD premature mortality (see Section 5.7.7), the EPA selected the two concentration-response functions that produced the highest and lowest estimated reduction in mortality, or benefits, from the identified functions. Aoki et al. (2016) was used for the low benefits estimates, and Lanphear et al. (2018) was used in the high benefits estimates. The EPA will evaluate new and novel data as they become available, and will consider updating the methodology for estimating cardiovascular premature mortality effects of changes in adult lead exposure as appropriate.

The four evaluated studies – Menke et al. (2006), Aoki et al. (2016), Lanphear et al. (2018), and Ruiz-Hernandez et al. (2017) – all use regression models to relate log-transformed blood lead levels to CVD premature mortality. The concentration-response function associated with the relationship between blood lead and CVD premature mortality modeled in each study is:

$$\Delta CVD\ Premature\ Mortality = y_1\left(1 - e^{\beta \log_z\left(\frac{x_2}{x_1}\right)}\right) \qquad \text{(Equation 14)}$$

Thus, the function necessary to estimate the number of cases associated with a change in blood lead levels is:

$$Cases\ Avoided = y_1\left(1 - e^{\beta \log_z\left(\frac{x_2}{x_1}\right)}\right) * pop \qquad \text{(Equation 15)}$$

Where:

y_1 = Baseline hazard rate of CVD premature mortality in baseline scenario (i.e., without the rule)

β = Beta coefficient, which represents the change in CVD premature mortality per unit change in blood lead

$\log_z$ = Log transformation to the base z (e.g., $\log_{10}$)

x_2 = Blood lead level associated with the rule

x_1 = Blood lead level without the rule

[193]In 2020, the EPA's Science Advisory Board, in its review of the scientific and technical basis of the Lead and Copper Rule Revisions, recommended that the EPA quantify and monetize CVD premature mortality impacts in adults from reductions in lead in drinking water, citing "well documented" evidence of an association with cardiovascular impacts (EPA SAB, 2020).

[194] Note the Abt Associates (2023) methodology was externally peer reviewed. See the MDB, Inc. (2019) "Selection of Concentration-Response Functions between Lead Exposure and Adverse Health Outcomes for Use in Benefits Analysis: Cardiovascular-Disease Related Mortality" peer review documentation at https://cfpub.epa.gov/si/si_public_record_report.cfm?Lab=NCEE&dirEntryID=342855

pop = Population for whom the change in blood lead occurs

Equation 16 can be used to estimate the avoided CVD premature mortality from reductions in blood lead.

The beta coefficient, β, varies based on the study in question and is calculated by:

$$\beta = \frac{\ln(Hazard\ ratio)}{\log_z(Fold\ increase\ in\ blood\ lead\ for\ hazard\ ratio)} \qquad \text{(Equation 16)}$$

For example, the beta from Aoki et al. (2016) is based on a hazard ratio of 1.44, which was derived from a 10-fold increase in blood lead levels. Thus, the beta coefficient is equal to $\ln(1.44)/\log_{10}(10)$, which is 0.36. Exhibit 5-31 displays the study-specific inputs for Equation 16 associated with all five concentration-response functions presented in Brown et al. (2020) and Abt Associates (2023).

Exhibit 5-31: Inputs to the Health Impact Function Based on Selected Studies

Variable	Aoki et al. (2016)	Lanphear et al. (2018)
		Blood Pb <5 µg/dL
Log transformation ($\log_z$)	**Log_{10}**	**Log_{10}**
Central beta (β) estimate	**0.36**	**0.96**
Lower beta (β) estimate (based on lower bound of 95% CI for HR)	**0.05**	**0.54**
Upper beta (β) estimate (based on upper bound of 95% CI for HR)	**0.68**	**1.37**

Sources: Aoki et al. (2016) and Lanphear et al. (2018).
Note: Bolding identifies the parameters used in the LCRI analysis. For full descriptions of these and the functions not used to quantify CVD premature mortality, see Brown et al. (2020)

5.5.8 Valuation of Avoided Cardiovascular Disease Premature Mortality

In the scientific literature, estimates of willingness to pay for small reductions in mortality risks are often referred to as the "value of a statistical life." This is because these values are typically reported in units that match the aggregate dollar amount that a large group of people would be willing to pay for a reduction in their individual risks of dying in a year, such that the EPA would expect one fewer death among the group during that year on average. This is best explained by way of an example. Suppose each person in a sample of 100,000 people were asked how much they would be willing to pay for a reduction in their individual risk of dying of 1 in 100,000, or 0.001 percent, over the next year. Since this reduction in risk would mean that the EPA would expect one fewer death among the sample of 100,000 people over the next year on average, this is sometimes described as "one statistical life saved." Now suppose that the average response to this hypothetical question was $100. Then the total dollar amount that the group would be willing to pay to save one statistical life in a year would be $100 per person × 100,000 people, or $10 million. This is what is meant by the "value of a statistical life." Importantly, this is not an estimate of how much money any single individual or group would be willing to pay to prevent the certain death of any particular person.

The EPA uses a value of a statistical life (VSL) of $12.98 million in 2022 dollars, which is estimated using the EPA's (2014) recommended VSL of $4.8 million in 1990 dollars and the EPA's (2014) recommended method for adjusting the VSL for income growth and inflation. The $4.8 million value in 1990 dollars is updated to the $12.98 million in 2022 dollars by adjusting for inflation using the U.S. Bureau of Labor Statistics' (2019) Consumer Price Index and adjusting for income growth using real GDP per capita and an income elasticity of 0.4.

5.6 National Level Benefits Estimates

5.6.1 Implementation of Benefit Calculations in the SafeWater LCR model

Benefits are estimated based on LSL/GRR service line replacements, installation of POU devices, distribution of pitcher filters and installation and re-optimization of CCT that occur over the 35-year analysis period.

Benefits are captured in the analysis for each endpoint at a specific age, therefore it is necessary to estimate the number of people of each age who are served by each PWS receiving a benefit from a change in the lead concentration of their drinking water. This is handled by multiplying the number of people experience a drinking water change by the proportion of people that age in the U.S. population. For example, in order to estimate the number of 7-year-olds receiving a benefit in a given year, the SafeWater LCR model takes the total population experiencing each water lead change and multiplies that figure by the proportion of the United States population that is 7 years of age. A similar calculation is done for the applicable ages for the additional endpoints.

Because the SafeWater LCR model follows the population for a period of 35 years, all children who lived in areas experiencing the water lead concentration change who are younger than 7 years of age would also accrue benefits in future years of the 35-year period, as well as children born after the change in lead concentration as long as they reach the age of 7 during the course of the 35-year period. However, the proportion of the total PWS population experiencing a change in lead concentration that receives an IQ benefit in a given year remains the same: approximately 1.34 percent (the percentage of 7-year-olds in the total United States population according to the 2020 United States Census). This is because both the age distribution and the population served by each PWS are assumed to remain constant over the analysis period. Children who turn 7 a year after an LSLR will receive a comparatively smaller benefit than children who are born after the LSLR, due to living a larger proportion of their life without the higher contribution of lead in their drinking water, and the resulting difference in BLLs between the with- and without-rule scenarios (without considering discounting). The EPA refers to these comparatively smaller benefits as "partial benefits." This same procedure is used for cases of ADHD avoided, and for prevention of lower birthweight. ADHD benefits are captured at age 7 for the low benefits estimate and age 11 for the high benefits estimate. For birth weight, benefits are captured once yearly based on the birth rate in women ages 17-45. For CVD premature mortality, benefits are captured yearly from ages 40-79.

The EPA does not assume that all homes with replaced LSLs have members living in the home eligible to experience all four health endpoints. Rather, the EPA assumes that the proportion of each age and sex (for adults) living in homes that are undergoing an LSLR is equal to the proportion of the United States population that is that age and sex. This assumption takes care of the need to model the movement of

children and adults in and out of homes in the community, as the proportion of the population in these age groups is assumed to remain constant. For example, for IQ, if there are 1,000 households being served by a PWS that underwent a change in lead concentration, approximately 1.34 percent of the population (the percent of the U.S. population 7 years of age) in those households would accrue benefit annually, regardless of which specific home being served by the PWS they lived in. The accrued benefit for those children who are served by a PWS that has undergone a change is then a function of changes in the average lifetime BLL of the children due to the change in lead concentration, and the subsequent avoided IQ loss.

The modeling assumption that the percentage of children and adults are evenly distributed across LSL and non-LSL households is necessary to estimate the national level impacts of the final LCRI requirements. At the national level, total benefits calculated using these assumptions can be accurate, however, please note that the potential geographic variability in the impacted population of children or adults will not be represented in this national scale model. For example, some geographic areas of the country may have higher or lower percentages of young children, receiving greater or fewer benefits from implementing lead concentration reducing actions like CCT and LSL/GRR service line replacement. This national scale model does not capture the potential local variation in the estimated unit benefits for a given unit of cost at the local level.

5.6.2 Monetized National Annual Benefits

As described in Section 5.3, the EPA estimated benefits corresponding to the low and high scenarios used to characterize uncertainty in the estimates Benefits are discounted back to year one of the analysis and annualized within the SafeWater LCR model. The EPA summed benefits for all years and all PWSs, and then annualized benefits for both the baseline 2021 LCRR and LCRI using a 2 percent discount rate.

- As described in Section 5.5.1 and Section 5.5.2, the EPA applied both a high and a low concentration-response function in order to estimate the reductions in IQ loss expected under the rule, and a value of an IQ. Avoided IQ loss was captured at age 7, using a 2 percent discount rate, benefits are further discounted, at 2 percent, back to year one of the analysis and annualized within the SafeWater LCR model.

- As described in Section 5.5.3, the EPA estimated avoided cases of ADHD with high and low assumptions for the concentration response function. These avoided cases of ADHD were captured at age 11 for the high function, and at age 7 for the low function, the difference is due to the timing and methods in the source studies. The dose-response functions measure the change in probability that an individual develops ADHD in their lifetime. This is a lifetime change in risk rather than an annual change. In the case of Froehlich et al. (2009), this is because the study measured prevalence rather than incidence. In this analysis, the EPA uses prevalence as the baseline rate of ADHD in both concentration-response functions. As described in Section 5.5.4, high and low values, estimated using a 2 percent discount rate but assuming different rates of ADHD persistence into adulthood, were applied to each avoided case of ADHD for the high and low scenario respectively. Benefits are further discounted back to year one of the analysis and annualized within the SafeWater LCR model using the 2 percent rate.