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# Estimated IQ points and lifetime earnings lost to early childhood blood lead levels in the United States



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## HIGHLIGHTS

# GRAPHICAL ABSTRACT

- Despite decreasing lead levels in the USA, there is no safe level of lead in blood.
- We simulated a nationwide sample of infants using cross-sectional survey data.
- We estimated IQ points and lifetime earnings lost based on lead levels.
- Black infants experienced higher IQ point and earning loss due to blood lead.
- Low levels of blood lead explain the majority of estimated lifetime earning loss.



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### ABSTRACT

There is no safe detectable level of lead (Pb) in the blood of children. Blood lead levels (BLLs) at ages 6-24 months  $\geq 2 \mu g/dL$  result in lost grade school intelligence quotient (IQ) points at ages 5–10 years. Black children continue to have the highest BLLs in the United States. Therefore, we examined currently undetermined racial/ethnic disparities in anticipated IQ points and associated lifetime earnings lost to early childhood blood lead. We conducted secondary analysis of infants with blood lead (in µg/dL) measured at ages 12–24 months by the cross-sectional National Health and Nutrition Examination Survey (NHANES) during 1999 to 2010. Nationally-representative estimates were produced using weighted simulation model. A total of 1241 infants were included from the NHANES sample (52% male; mean [SD] age, 18.5 [3.5] months; 25% Black [non-Hispanic], 42% Hispanic [any race], 5% Other/Multiracial, and 29% White [non-Hispanic]) after excluding 811 without BLL determinations. For national outcomes, Black infants experienced approximately 46-55% greater average estimated loss of grade school IQ points from blood lead than Hispanic or White infants (-1.78 IQ points vs. -1.15 and -1.21 respectively) with similar disparities in costs to expected lifetime earnings (-\$47,116 USD vs. -\$30,393 and -\$32,356 respectively). Our estimated nationwide costs of IQ points lost to BLLs during this 12-year period totaled \$554 billion (\$46.2 billion/year), in which blood lead <5 µg/dL accounted for 74% of this total burden. We report two aspects of the substantial national costs attributable to lead exposure in just the second year of life alone, which disproportionately impact predominately African-American Black infants from continuing legacies of environmental racism in lead exposure. Our findings underscore the remarkably high costs from recognized hazards of blood lead even at the lowest levels and the importance of primary prevention regarding childhood lead exposure.

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#### 1. Introduction

Lead (Pb) is a soft gray-colored heavy metal that bioaccumulates in the human body where it has no known necessary or beneficial physiological role. Lead presents with various systemic and neurotoxic properties (Mayans, 2019), while lead toxicity is significantly worse for children and especially younger children at critical stages of early childhood development. The neurotoxicity of lead in young children impacts the cerebellum, hippocampus, and prefrontal cerebral cortex (Sanders et al., 2009). Early childhood lead exposure can result in neuropsychological outcomes that negatively impact attention, behavior, cognition, decision-making, intellect, memory, and mental health (Dietrich et al., 1991; Banks et al., 1997; Lanphear et al., 2000; Lanphear et al., 2005; Chiodo et al., 2007; Bellinger, 2008; Bao et al., 2009; Senut et al., 2012; Searle et al., 2014; Shah-Kulkarni et al., 2016; Vorvolakos et al., 2016; Arnold and Liu, 2020). Greater childhood lead exposure is associated with decreased brain volume and lower structural brain integrity observed later in adulthood (Cecil et al., 2008; Reuben et al., 2020). Developing infants are also more vulnerable to lead and suffer more exposure in part from their comparatively greater body surface area, increased heart and respiratory rates, ingestion and inhalation of contaminated dust or soil from greater hand-to-mouth activity, pica, sitting and crawling, and low stature to the ground where lead dust settles (Mielke and Reagan, 1998).

The Centers for Disease Control (CDC) (Burns and Gerstenberger, 2014), in addition to the American Academy of Pediatrics (AAP) (Prevention of childhood lead toxicity, 2016), recognize there is no safe amount of lead in the blood at any detectable level. CDC guidelines designate BLLs  $\geq$ 5 µg/dL as an elevated BLL (EBLL), which reflects the nationwide 97.5th percentile of BLLs among children ages 1-5 years during 2007 to 2010 (CDC, 2013). In the United States, the hazards of early childhood lead exposure were first recognized with gradual reductions of lead additives and overall concentrations in gasoline, paints, and installed plumbing delivering drinking water over two decades beginning in the early 1970s (Dignam et al., 2019). Substantial reductions in environmental lead concentrations resulted in dramatic decreases in average BLLs among children 1-5 years of age that have fallen from an average 16 µg/dL during 1976 to 1980 to a historic low of 2 µg/dL during 2007 to 2010 (CDC, 2013; Jones et al., 2009; Meyer et al., 2003; Pirkle et al., 1994; Mahaffey et al., 1982). However, even the lowest detectable levels of blood lead present with deleterious population-wide effects.

The effects of early childhood BLLs previously considered low-level at <10 µg/dL were reviewed in 1997 (Banks et al., 1997), while BLLs  $\geq 2 \mu g/dL$  during early childhood (ages 6–24 months) result in the loss of intelligence quotient (IQ) points that only first appear later in grade school (ages 5–10 years) (Lanphear et al., 2005; Crump et al., 2013). This observable effect is particularly concerning as the steepest loss of IQ points is seen with the very first few point increases in blood lead between 2 and 4 µg/dL. (Lanphear et al., 2005; Mahaffey et al., 1982) These findings have led to calls for lowering the current elevated or actionable BLL from 5 and 10 µg/dL respectively to a newly revised actionable EBLL threshold of 2 µg/dL (Gilbert and Weiss, 2006). Similarly, blood lead  $>1 \mu g/dL$  during early childhood (ages 0–5 years) results in lower math and reading test scores that remain stable during grade school and middle school, while the most pronounced effects are observed with the first few point increases in BLLs  $<5 \mu g/dL$  (Shadbegian et al., 2019). Furthermore, increasing blood lead  $<3 \mu g/dL$  is associated with greater risk for attention-deficit/hyperactivity disorder (ADHD)-like symptoms in children ages 4-17 years (He et al., 2019), while BLLs >1.6 µg/dL are associated with anemia and decreasing iron status in children ages 0-5 years (Guo et al., 2020).

In a previous 2009 study by Gould (2009), the total social costs attributable to early childhood BLLs in the United States were reported, in addition to the substantial economic benefits of eliminating of early childhood lead exposure by remediating or abating sources of exposure. These estimates were produced via modeling the log-linear effect of early childhood blood lead in relation to IQ point losses originally reported from international pooled analysis (Lanphear et al., 2005; Crump et al., 2013). In the Gould study examining nationwide BLLs during 2003 to 2006 (Gould, 2009), it was observed that for each \$1 USD spent just on the elimination of leaded household paint hazards alone – a total savings of \$17 would be generated by lowering early childhood BLLs to prevent the later loss of grade school IQ points amounting to \$181 billion USD in total net savings. Similar methods have been utilized to estimate costs associated with early childhood BLLs and savings generated from subsequent prevention of lead-associated IQ point losses in Belgium (Remy et al., 2019), France (Pichery et al., 2011), and middle-to low-income nations in Africa, Asia, the Caribbean, and Central or South America (Attina and Trasande, 2013).

Significant racial disparities in BLLs for Black children have been documented in the United States since 1976 (CDC, 2013; Jones et al., 2009; Meyer et al., 2003; Pirkle et al., 1994; Mahaffey et al., 1982). Racial disparity in early childhood blood lead continues to persist for Black children at the national level (CDC, 2013; White et al., 2016), which both ourselves and many others have reported for the states of Illinois (Winter and Sampson, 2017), Indiana (Morrison et al., 2013), Louisiana (Mielke et al., 2007), Maryland (Wheeler et al., 2019a), Massachusetts (Sargent et al., 1995), Michigan (Moody et al., 2016; Bezold et al., 2020), Minnesota (Wheeler et al., 2019b), Missouri (Rabito et al., 2007), New York (Haley and Talbot TO, 2004), North Carolina (Kim et al., 2008), Ohio (Raymond et al., 2009), South Carolina (Aelion and Davis, 2019), and Tennessee (Ford et al., 2016). Recently, we found that racial/ethnic disparity persists for risk factors of lead exposure among children ages 1-5 years with significantly worse outcomes of continuous BLLs or rates of EBLLs  $\geq 5 \,\mu g/dL$  observed for Black children even after controlling for other risk factors and confounding variables (Yeter et al., 2020). This national condition results from significantly greater amounts of environmental lead exposure still suffered by Black children and predominately Black residential areas. Black racial disparity has been demonstrated for early childhood lead exposure from household dust and paint hazards (Lanphear et al., 1996; Jacobs et al., 2002; Raymond et al., 2011), contaminated soils (Mielke et al., 2007; Dietrich, 2020; Campanella and Mielke, 2008; Aelion et al., 2013; Ha et al., 2016), industrial emissions and the ambient air (Moody and Grady, 2017; Benson et al., 2017), demolition activity (Bezold et al., 2020; Rabito et al., 2007; Farfel et al., 2003), and both pre- or postnatal exposure from their Black birthing parent (Cassidy-Bushrow et al., 2017).

Racial/ethnic disparities have yet to be estimated for losses of grade school IQ points and associated costs to expected lifetime earnings attributable to early childhood blood lead. Therefore, we examined these negative outcomes using blood lead measurements from a nationally-representative sample of infants in the United States during their second year of life.

#### 2. Methods

The NHANES (National Health and Nutrition Examination Survey) is a cross-sectional study to assess the current health and nutritional status of individuals residing in the United States, which conducts a nationally-representative sample of the non-institutionalized population that is de-identified and then made freely accessible online (www.cdc.gov/nchs/nhanes.htm) (http://www.cdc.gov/nchs/nhanes. htm, n.d.). For this study, publicly-available data were retrieved from the NHANES and thus did not require additional institutional review board (IRB) approval. Data were retrieved for infants examined at ages 12–24 months by the NHANES during the reporting years of 1999 to 2010. Obtained covariate data included two-year survey cycles, blood lead (measured in µg/dL), binary gender, exam age (months), race/ethnicity, and NHANES sample weights for interviews and examinations. After excluding 811 cases for lacking BLL determination, a total of 1241 children were included in our analyses. Lead

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concentrations were initially determined by the NHANES in whole blood using validated analytical procedures that included employing a multi-element electrothermal atomic absorption spectrometer (ETAAS) before 2003, while inductively coupled plasma-mass spectrometry (ICP-MS) has been used thereafter. We rounded continuous BLLs to the first decimal place to maintain significant figures, which had a lower limit of detection (LLOD) of 0.3 µg/dL for both forms of spectroscopic analyses. Children younger than 12 months of age are not included in venous blood sample draws by the NHANES.

In order to account for uncertainty in the relationships between blood lead and IQ loss, and its associated costs to lifetime earnings, we constructed a population of infants aged 12-24 months using the NHANES statistical weights (the inverse of the sampling weights at the exam stage). As such, our population includes the number of children that the NHANES sampled children were calculated to represent, a total of 16,177,181 children aged 12-24 months. We then applied a two-step process to estimate the lifetime earning loss for each infant in the simulated population taking into account uncertainty resulting from variability in the exposure effects and sampling error. We first sampled an IQ point loss for each infant based on their BLL using the exposure effect from Gould (2009), which we used to estimate an IQ loss attributable to blood lead for a linear relationship between BLLs and IQ point loss over three ranges: 2–10 µg/dL, 10–20 µg/dL, and 20–30 µg/dL. These three effect estimates were determined using the IQ measurements and BLLs of an international pooled analysis (Lanphear et al., 2005; Crump et al., 2013; Gould, 2009; Remy et al., 2019), which assumes a uniform distribution of BLLs within each range.

Thereafter, to account for uncertainty in the effect estimates and using different samples and populations from Gould (2009), we used the effect estimates and associated standard errors as the basis for distributions representing the effect of blood lead on IQ points in the three ranges. For these three ranges, we sampled the effect of lead exposure on IQ loss on outcomes from a Gaussian distribution of [Normal  $(\mu = 0.54, \sigma = 0.1020)$ , Normal  $(\mu = 0.19, \sigma = 0.0357)$ , Normal  $(\mu =$ 0.11,  $\sigma = 0.0204$ )] respectively, which was then multiplied by the BLL of each child to produce an estimated IQ point loss. Lastly, we considered two different approaches to account for BLLs <2 µg/dL that have no reported exposure effect estimates (Lanphear et al., 2005). The first approach extended the effect distribution for BLLs at 2-10 µg/dL to assume that this effect held at BLLs  $<2 \mu g/dL$ . The second approach assumed no effect on IQ was present for BLLs  $<2 \mu g/dL$ . In addition, four infants had lead levels  $>30 \,\mu\text{g/dL}$ . In both analyses, we extended the effect distribution of blood  $20-30 \mu g/dL$  for higher BLLs.

After estimating IQ loss outcomes for each child in relation to their BLLs in the simulated population, we estimated the associated losses of expected lifetime earnings in United States dollars (USD) per IQ

# Table 1

Characteristics for the analytic sample of infants ages 12-24 months - U.S. NHANES, 1999 to 2010.

point lost. To do so, we incorporated respective lifetime earnings estimates for boys (\$1,413,313) and girls (\$1,156,157) from Attina and Trasande (2013), which also represent the same time period as our analytic sample, in addition to percentages of lifetime earnings lost per IQ point decrement (1.76 to 2.37%) (Attina and Trasande, 2013; Salkever, 1995; Schwartz et al., 1985). Owing to the relative uncertainty in these effects, we simulated a percentage of costs to lifetime earnings associated with IQ point loss from a uniform distribution using the aforementioned percentages as endpoints. We then used the simulated percent of lost lifetime earnings to obtain an estimate of losses each child would incur over their lifetime, which was done using the following equation:

Estimated lifetime earnings loss

- = L [lead, blood ( $\mu g/dL$ )] · I [IQ points lost] · P
  - [percent lifetime earnings lost] · E [expected lifetime earnings]

Lastly, we calculated summary measures of lifetime earning loss by race/ethnicity, as well as total lifetime earning loss by race/ethnicity for the entire simulated population all analyses were conducted in R (version 3.6.1) with a 0.05 level of significance used for tests of homogeneity in age, continuous blood lead, or binary gender by race/ethnicity.

#### 3. Results

A total of 1241 infants were included (52% male; mean [SD] age, 18.5 [3.5] months; 42% Hispanic, 25% non-Hispanic Black, 29% non-Hispanic White, and 5% Other/Multiracial) after excluding 811 cases without BLL determinations. Age in months at exam and gender did not significantly differ by race/ethnicity (Table 1). Significant differences were observed for blood lead by race/ethnicity (Kruskal-Wallis  $\chi^2 = 93$ , df = 3, p < 0.001) with Black infants having the highest mean BLLs. Hispanic infants had the lowest mean BLLs with White infants had similar yet slightly higher BLLs. No infants presented with undetectable blood lead in the analytic sample.

For Black infants in the first simulation model (Table 2), the average estimated loss of IQ points attributable to blood lead was 1.78 points, which is 0.57–0.63 more IQ points than the losses for White or Hispanic infants respectively. On average, the estimated associated costs to expected lifetime earnings was \$47,116 for Black infants, which is approximately \$15,000–17,000 higher in costs than White or Hispanic infants. For these two outcomes, average national losses were 46–55% greater for Black infants than their White or Hispanic peers. Considerable variability is evident in these estimated losses, owing to the variability present in the distribution of BLLs as well as in the effects of BLLs on outcomes. These effects persisted yet were somewhat attenuated in

| Variable                   | Total      | Black (non-Hispanic) | Hispanic (any race) | Other/Multiracial | White (non-Hispanic) | P-Value |
|----------------------------|------------|----------------------|---------------------|-------------------|----------------------|---------|
| Sample size <sup>a</sup>   | 1241       | 311 (25.1)           | 515 (41.5)          | 56 (4.5)          | 359 (28.9)           | -       |
| Age at exam <sup>b</sup>   |            |                      |                     |                   |                      | 0.50    |
| In months                  | 18.5 (3.5) | 18.7 (3.4)           | 18.3 (3.5)          | 18.3 (3.5)        | 18.5 (3.6)           |         |
| Binary gender <sup>a</sup> |            |                      |                     |                   |                      | 0.48    |
| Female                     | 597 (48.1) | 156 (50.1)           | 249 (48.3)          | 30 (53.6)         | 162 (45.1)           |         |
| Male                       | 644 (51.9) | 155 (49.9)           | 266 (51.7)          | 26 (46.4)         | 197 (54.9)           |         |
| Blood lead <sup>c</sup>    |            |                      |                     |                   |                      | < 0.001 |
| In µg/dL                   | 1.9 (1.7)  | 2.6 (2.3)            | 1.7 (1.5)           | 2.3 (2.0)         | 1.7 (1.4)            |         |
| Blood lead <sup>a</sup>    |            |                      |                     |                   |                      | < 0.001 |
| <2 µg/dL                   | 632 (50.9) | 96 (30.9)            | 304 (59.0)          | 23 (41.1)         | 209 (58.2)           |         |
| 2-5 µg/dL                  | 488 (39.3) | 160 (51.4)           | 180 (35.0)          | 28 (50.0)         | 120 (33.4)           |         |
| 5–10 µg/dL                 | 94 (7.6)   | 42 (13.5)            | 27 (5.2)            | 3 (5.4)           | 22 (6.1)             |         |
| 10–20 µg/dL                | 21 (1.7)   | 10 (3.2)             | 2 (0.4)             | 2 (3.6)           | 7 (1.9)              |         |
| 20–30 µg/dL                | 2 (0.2)    | 0                    | 1 (0.2)             | 0                 | 1 (0.3)              |         |
| 30–48 µg/dL                | 4 (0.3)    | 3 (1.0)              | 1 (0.2)             | 0                 | 0                    |         |

<sup>a</sup> Summarized with count (proportion) and tested using chi-squared test

<sup>b</sup> Summarized with mean (standard deviation [SD]) and tested using one-way analysis of variance.

<sup>c</sup> Summarized with median (interquartile range) and tested using Kruskal-Wallis test.

#### Table 2

Mean national losses of IQ and earnings attributable to early childhood blood lead - U.S. NHANES, 1999 to 2010.

| Variable                          | Grade school IQ points l | ost          | Expected lifetime earnings lost |                     |  |
|-----------------------------------|--------------------------|--------------|---------------------------------|---------------------|--|
| Simulated population <sup>a</sup> | Model 1                  | Model 2      | Model 1                         | Model 2             |  |
|                                   | Mean (SD)                |              | Mean (SD)                       |                     |  |
| Total                             | -1.29 (1.13)             | -0.92 (1.34) | -\$34,241 (30,470)              | -\$24,480 (36,010)  |  |
| Blood lead                        |                          |              |                                 |                     |  |
| $< 2 \mu g/dL$                    | -0.67(0.26)              | 0            | -\$17,878 (7361)                | 0                   |  |
| 2–5 µg/dL                         | -1.56 (0.52)             | -1.56 (0.52) | -\$41,502 (14,869)              | -\$41,509 (14,880)  |  |
| 5–10 μg/dL                        | -3.58 (0.91)             | -3.58 (0.91) | -\$94,510 (27,627)              | -\$94,455 (27,654)  |  |
| 10–20 μg/dL                       | -5.94 (1.10)             | -5.94 (1.10) | -\$153,335 (35,260)             | -\$153,345 (35,242) |  |
| 20–30 µg/dL                       | -7.69 (1.12)             | -7.68 (1.13) | -\$211,779 (36,998)             | -\$211,176 (36,903) |  |
| 30–48 µg/dL                       | -9.28 (1.37)             | -9.28 (1.36) | -\$261,734 (51,866)             | -\$261,440 (51,257) |  |
| Race/ethnicity                    |                          |              |                                 |                     |  |
| Black (non-Hispanic)              | -1.78 (1.49)             | -1.53 (1.68) | -\$47,116 (40,431)              | -\$40,612 (45,359)  |  |
| Hispanic (any race)               | -1.15 (0.95)             | -0.76 (1.16) | -\$30,393 (25,642)              | -\$20,051 (31,066)  |  |
| Other/multiracial                 | -1.42 (1.15)             | -1.12 (1.36) | -\$37,717 (32,541)              | -\$29,959 (37,770)  |  |
| White (non-Hispanic)              | -1.21 (1.06)             | -0.82 (1.27) | -\$32,356 (28,350)              | -\$21,824 (33,975)  |  |

<sup>a</sup> Tested using data simulation assuming either (Model 1) the same effect remains for IQ point losses and blood lead <2 µg/dL as for blood lead 2–10 µg/dL or (Model 2) no effect for blood lead <2 µg/dL.

the second model that treated blood lead  $<2 \ \mu g/dL$  as having no effect on IQ point loss. Furthermore, racial/ethnic disparities persisted and even worsened for Black infants in the second model.

Using the first model that estimated effects for blood lead  $<2 \mu g/dL$  (Table 3), the total estimated national costs during this 12-year period (1999 to 2010) amounted to 20.9 million lost IQ points with an associated \$554 billion USD in costs to expected lifetime income for infants during their second year of life. Black infants only composed 14% of the simulated national population while disproportionately accounting for 20% of the total estimated nationwide losses in IQ points and earnings from blood lead. The distributions of estimated lifetime earnings lost to blood lead demonstrate a greater number of Black infants suffer the highest losses (Fig. 1). Lastly, 74% of the total national burdens in these outcomes from blood lead result from blood lead <5  $\mu g/dL$ , while the largest proportion of IQ point losses were from BLLs 2–5 (Fig. 2).

#### 4. Discussion

In the present study, we used childhood blood lead levels from a nationally-representative sample for the United States to estimate IQ points lost and lifetime earnings lost due to early life lead exposure during 1999 to 2010. We estimated more than half a trillion \$USD in total costs to expected lifetime earnings just from the loss of grade school IQ points attributable to blood lead among infants during their second year of life (Table 3) – which primarily resulted from low-level blood lead  $<5 \ \mu g/dL$ . This represents an astounding figure of nearly \$50 billion per year in total expected costs to infants across their lifetime once they reach employment age and involves only one effect of lead exposure in early childhood. Most importantly, predominately African-American Black infants shoulder disproportionately higher burdens for these negative outcomes from blood lead both on average and in total (Fig. 1).

Our present findings of racial disparities are striking and alarming. However, these negative outcomes should be anticipated given that increasingly higher early childhood blood lead is significantly associated with Black race or residing in areas with higher percent Black populations. In our previous study (Yeter et al., 2020), we speculated that even slight increases in low-level blood lead - including the roughly 1 µg/dL average higher BLLs we previously found among young Black children - could result in individual costs amounting to billions in USD from associated IQ point losses alone. We further reviewed various issues of environmental racism regarding Black racial disparities in safe housing, risk screening, and other injustices that help explain the greater amounts of lead that Black children are exposed to (Bezold et al., 2020; Rabito et al., 2007; Lanphear et al., 1996; Jacobs et al., 2002; Raymond et al., 2011; Dietrich, 2020; Campanella and Mielke, 2008; Aelion et al., 2013; Ha et al., 2016; Moody and Grady, 2017; Benson et al., 2017; Farfel et al., 2003; Cassidy-Bushrow et al., 2017),

Table 3

National burdens of lost IQ and earnings attributable to early childhood blood lead - U.S. NHANES, 1999 to 2010.

| Variable             | Simulated population <sup>a</sup> |         | Grade school IQ points lost |         | Expected lifetime earnings lost |         |
|----------------------|-----------------------------------|---------|-----------------------------|---------|---------------------------------|---------|
|                      | Model 1                           |         |                             |         |                                 |         |
|                      | Sum                               | Percent | Sum                         | Percent | Sum                             | Percent |
| Total                | 16,177,181                        | -       | -20,860,907                 | -       | -\$553,925,157,953              | -       |
| Blood lead           |                                   |         |                             |         |                                 |         |
| $< 2 \mu g/dL$       | 8,830,554                         | 54.6    | -5,916,093                  | 28.4    | -\$157,874,450,264              | 28.5    |
| 2–5 µg/dL            | 6,040,235                         | 37.3    | -9,430,737                  | 45.2    | -\$250,683,652,827              | 45.3    |
| 5–10 µg/dL           | 1,006,161                         | 6.2     | -3,601,645                  | 17.3    | -\$95,092,387,944               | 17.2    |
| 10-20 µg/dL          | 253,780                           | 1.6     | -1,506,549                  | 7.2     | -\$38,913,339,786               | 7.0     |
| 20-30 μg/dL          | 15,944                            | 0.1     | -122,632                    | 0.6     | -\$3,376,600,730                | 0.6     |
| 30–48 µg/dL          | 30,507                            | 0.2     | -283,249                    | 1.4     | -\$7,984,726,402                | 1.4     |
| Race/ethnicity       |                                   |         |                             |         |                                 |         |
| Black (non-Hispanic) | 2,334,629                         | 14.4    | -4,159,583                  | 19.9    | -\$109,999,489,058              | 19.9    |
| Hispanic (any race)  | 4,254,807                         | 26.3    | -4,895,809                  | 23.5    | -\$129,315,164,452              | 23.3    |
| Other/Multiracial    | 818,003                           | 5.1     | -1,165,146                  | 5.6     | -\$30,852,962,516               | 5.6     |
| White (non-Hispanic) | 8,769,742                         | 54.2    | -10,640,368                 | 51.0    | -\$283,757,541,927              | 51.2    |

<sup>a</sup> Assumes the same effect remains for IQ point losses and blood lead  $<2 \mu g/dL$  as for blood lead  $2-10 \mu g/dL$  (Model 1).



Fig. 1. Density plot of estimated lifetime earnings lost for Black and White infants. Outcomes produced using model 1 assuming the effect of IQ point losses from blood lead 2–10 µg/dL extends to blood lead <2 µg/dL – U.S. NHANES, 1999 to 2010.

in addition to having the highest BLLs (White et al., 2016; Winter and Sampson, 2017; Morrison et al., 2013; Wheeler et al., 2019a; Sargent et al., 1995; Moody et al., 2016; Bezold et al., 2020; Wheeler et al., 2019b; Rabito et al., 2007; Haley and Talbot TO, 2004; Kim et al., 2008; Raymond et al., 2009; Aelion and Davis, 2019; Ford et al., 2016). Black racial disparities in the United States are evocative of the total international burdens for blood lead primarily impacting Black and Indigenous children of color throughout the Global South (Burki, 2020).

In the United States, the primary source of early childhood lead exposure continues to be the legacy of historical leaded paint hazards in older housing built before 1978. To date, the occurrence of household leaded paint hazards that are highly cost-prohibitive to abate disproportionately involve Black households, in addition to these households suffering from higher levels of lead dust hazards (Lanphear et al., 1996; Jacobs et al., 2002; Raymond et al., 2011). Predominately Black neighborhoods suffer from greater amounts of environmental lead exposure from contaminated soils that are another major source of childhood lead exposure (Mielke et al., 2007; Dietrich, 2020; Campanella and Mielke, 2008; Aelion et al., 2013; Ha et al., 2016), along with greater industrial lead emissions and demolition activity that increase lead in the

ambient air and can also contribute to household lead dust and contaminated soils (Bezold et al., 2020; Rabito et al., 2007; Moody and Grady, 2017; Benson et al., 2017; Farfel et al., 2003). There are also concerns of intergenerational transmission of lead exposure from Black mothers to their children, which results from Black mothers having higher lead burdens than their racial/ethnic peers (Lee et al., 2005; Schell et al., 2003). The disparate lead burden suffered by Black children is observed to begin in utero and then persists into early childhood, which results from transmission by their Black mothers (Cassidy-Bushrow et al., 2017). Lastly, in the Global South, other significant sources of lead exposure include mining operations, lead battery production, electronic waste, traditional medicines, and glazed ceramics among others (Obeng-Gyasi, 2019).

Our identified racial/ethnic disparities for these study outcomes likely demonstrate variations in the strength among differing locales. In our previous study of Maryland (Wheeler et al., 2019a), the highest rates of early childhood EBLLs  $\geq 5 \mu g/dL$  were largely clustered in the city of Baltimore, which is predominately Black, leading us to suggest targeted prioritization for areas with a higher percent Black population or pre-1940 housing. Despite Black children suffering greater amounts



Fig. 2. Cumulative proportion of estimated IQ points lost attributable to early childhood blood lead level. Outcomes produced using model 1 assuming the effect of IQ point losses from blood lead 2–10 µg/dL extends to blood lead <2 µg/dL, while red dashed lines indicate that children with BLLs <2 µg/dL and <5 µg/dL shoulder 28% and 74% of total IQ points losses respectively – U.S. NHANES, 1999 to 2010.

of lead exposure and the highest BLLs – Black race is not currently used as an indicator in risk screenings tools for early childhood lead exposure from the CDC (Centers for Disease Control, 1991), the AAP (Cantor et al., 2019), or most targeted screenings differing by individual states (Ossiander, 2013). Risk screening for early childhood blood lead only performs slightly better than if left to pure chance alone (Cantor et al., 2019; Ossiander, 2013), while incorporating Black race yields more favorable performance (Kaplowitz et al., 2012). Nationally, BLL testing coverage varies greatly and remains low, while targeted screening efforts fail to identify 1 out of 3 children ages 0–5 years with an EBLL  $\geq 5 \mu g/dL$  (Roberts et al., 2017). Universal BLL testing would identify all at-risk children (Madrigal and Roberts, 2018; Kemper et al., 1998).

Our study measures involved effects of blood lead on IQ point loss observed from a separate sample population (Lanphear et al., 2005), to which we employed two simulation models to help account for uncertainty in our outcomes. These reported effects were not observed for blood lead  $<2 \mu g/dL$ , which accounted for 55% of our simulated population and were treated as having an effect on IQ point loss. Our negative outcomes were somewhat blunted when assuming no effects for blood lead  $<2 \mu g/dL$  (Model 2), which accounted for 28–29% of the total IQ and earnings losses (Model 1), while racial/ethnic disparities persisted and even worsened for Black children when no effects were assumed. NHANES data were not available for infants ages 6-11 months, which limited our investigation. Lastly, the NHANES data do not reveal the highest BLL that infants experienced during the survey years or early childhood. As peak BLLs are a stronger predictor of IQ point loss (Lanphear et al., 2005), our current national estimates are more conservative.

The present study expands upon a previous examination of the United States during 2003 to 2006 and includes findings for an additional eight survey years. However, more prospective study is needed to account for the full costs of lead exposure during early childhood and across the lifespan. Our measured outcomes did not include other neurological or systemic effects from blood lead exposure in children or its associated costs, in addition to outcomes resulting from prenatal exposure (Taylor et al., 2015; Rodosthenous et al., 2017; Goto et al., 2020). Further data of early childhood BLLs are available from the NHANES II (1976 to 1980), NHANES III (1988 to 1994), and continuous NHANES (1999 to present) that can be used to examine historical trends in racial/ethnic disparities from costs attributable to blood lead. Lastly, Black racial disparities in negative outcomes from having higher BLLs should be examined at state and local levels. This would demonstrate costs inherited by infants, its disparities, needs for improved targeting, and the importance of primary prevention for childhood lead exposure and the consequences of even the lowest levels of blood lead.

#### 5. Conclusions

The costs attributable to even at the lowest levels of blood lead during early childhood are astonishingly high. We report alarming losses of grade school IQ points and associated lifetime earnings attributable to early childhood blood lead. Outcomes were substantially worse for predominately African-American Black infants who suffer the highest BLLs, which still mostly involve BLLs  $<5 \mu g/dL$ . Black disparity in early childhood blood lead and exposures persist despite being documented for over four decades. Harm has been done to Black children and continues to transpire, which demands equity in environmental justice to empower disproportionately affected Black individuals and communities. This requires addressing issues of safe housing, the built environment, and industrial emissions that result in greater amounts of lead exposure to Black children, in addition to increasing screening of at-risk Black children who present with the highest BLLs compared to their racial/ ethnic peers. Lastly, our findings underscore the remarkably high costs from recognized hazards of blood lead even at the lowest levels and the importance of primary prevention regarding childhood lead exposure for all children in the United States and abroad.

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#### **CRediT authorship contribution statement**

**Joseph Boyle:** Methodology, Software, Formal analysis, Writing – original draft, Writing – review & editing, Visualization. **Deniz Yeter:** Conceptualization, Data curation, Writing – original draft, Writing – review & editing, Supervision. **Michael Aschner:** Conceptualization. **David C. Wheeler:** Methodology, Software, Writing – original draft, Writing – review & editing, Visualization, Supervision, Project administration.

#### **Declaration of competing interest**

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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