Patterns of Children's Blood Lead Screening and Blood Lead Levels in North Carolina, 2011–2018—Who Is Tested, Who Is Missed?

Elizabeth M. Kamai,^{1,2} Julie L. Daniels,^{1,3} Paul L. Delamater,^{4,5} Bruce P. Lanphear,⁶ Jacqueline MacDonald Gibson,⁷ and David B. Richardson⁸

¹Department of Epidemiology, University of North Carolina at Chapel Hill (UNC-Chapel Hill), Chapel Hill, North Carolina, USA

²Department of Population and Public Health Sciences, Keck School of Medicine, University of Southern California, Los Angeles, California, USA

³Department of Maternal and Child Health, UNC-Chapel Hill, Chapel Hill, North Carolina, USA

⁴Department of Geography, UNC-Chapel Hill, Chapel Hill, North Carolina, USA

⁵Carolina Population Center, UNC-Chapel Hill, North Carolina, USA

⁶Faculty of Health Sciences, Simon Fraser University, Vancouver, British Columbia, Canada

⁷Department of Environmental and Occupational Health, Indiana University, Bloomington, Indiana, USA

⁸Department of Environmental and Occupational Health, University of California, Irvine, California, USA

BACKGROUND: No safe level of lead in blood has been identified. Blood lead testing is required for children on Medicaid, but it is at the discretion of providers and parents for others. Elevated blood lead levels (EBLLs) cannot be identified in children who are not tested.

OBJECTIVES: The aims of this research were to identify determinants of lead testing and EBLLs among North Carolina children and estimate the number of additional children with EBLLs among those not tested.

METHODS: We linked geocoded North Carolina birth certificates from 2011–2016 to 2010 U.S. Census data and North Carolina blood lead test results from 2011–2018. We estimated the probability of being screened for lead and created inverse probability (IP) of testing weights. We evaluated the risk of an EBLL of $\geq 3 \mu g/dL$ at <30 months of age, conditional on characteristics at birth, using generalized linear models and then applied IP weights to account for missing blood lead results among unscreened children. We estimated the number of additional children with EBLLs of all North Carolina children using the IP-weighted population and bootstrapping to produce 95% credible intervals (CrI).

RESULTS: Mothers of the 63.5% of children (402,002 of 633,159) linked to a blood lead test result were disproportionately young, Hispanic, Black, American Indian, or on Medicaid. In full models, maternal age ≤ 20 y [risk ratio (RR) = 1.10; 95% confidence interval (CI): 1.13, 1.20] or smoking (RR = 1.14; 95% CI: 1.12, 1.17); proximity to a major roadway (RR = 1.10; 95% CI: 1.05, 1.15); proximity to a lead-releasing Toxics Release Inventory site (RR = 1.08; 95% CI: 1.03, 1.14) or a National Emissions Inventory site (RR = 1.11; 95% CI: 1.07, 1.14); and living in neighborhoods with more housing built before 1950 (RR = 1.10; 95% CI: 1.05, 1.14) or before 1940 (RR = 1.18; 95% CI: 1.11, 1.25) or more vacant housing (RR = 1.14; 95% CI: 1.11, 1.17) were associated with an increased risk of EBLL, whereas overlap with a public water service system was associated with a decreased risk of EBLL (RR = 0.85; 95% CI: 0.83, 0.87). Children of Black mothers were no more likely than children of White mothers to have EBLLs (RR = 0.98; 95% CI: 0.96, 1.01). Complete blood lead screening in 2011–2018 may have identified an additional 17,543 (95% CI: 17,462, 17,650) children with EBLLs $\geq 3 \mu g/dL$.

DISCUSSION: Our results indicate that current North Carolina lead screening strategies fail to identify over 30% (17,543 of 57,398) of children with subclinical lead poisoning and that accounting for characteristics at birth alters the conclusions about racial disparities in children's EBLLs. https://doi.org/10.1289/EHP10335

Introduction

Lead is an established toxicant that adversely impacts human health.¹ Removing lead from gasoline, banning lead in consumer paint, and adding limits on lead in public drinking water in the United States in the 1970s, 1980s, and 1990s were accompanied by dramatic declines in children's lead exposure.^{2,3} Lead exposure remains a problem for U.S. children,⁴ and no safe level of lead has been identified for children.¹ Lead continues to be used in some industries⁵ and is extremely stable in soil, which serves as a sink for both historic and contemporary lead deposition.⁶

The key to protecting children from lead is identifying and mitigating lead hazards before children are exposed.⁷ State programs for protection from lead exposure, however, rely on identifying children with high concentrations of lead in their blood and then

Supplemental Material is available online (https://doi.org/10.1289/EHP10335).

conducting investigations to determine and remove lead exposure sources. Within these systems, two additional major barriers exist to protecting children. First, children must have their blood lead level (BLL) measured, ideally before 3 years of age.⁸ However, not every child receives a blood lead test, and those who do are not randomly sampled. Although some health services (e.g., immunizations, screening for autism spectrum disorder) are recommended for all children at regular intervals,9 lead exposure screening and blood lead testing are recommended only for children "at risk of lead exposure."8 This includes all children covered by Medicaid at 12 and 24 months of age; those between 24 and 72 months of age who have not previously been tested must receive a test as well. However, 35-60% of children on Medicaid are not tested for lead.^{11–13} Some clinicians use screening questionnaires to identify children at risk of lead exposure who should receive blood lead testing, but these are of limited predictive utility.¹⁴ A few stud-ies^{15–17} have found substantial variation in whether clinicians choose to test children's blood for lead and have shown that this decision is a function of physicians' knowledge, practice setting, patient demographics, and financial incentives. Second, investigations to identify possible sources of lead exposure are only triggered if a child's BLL is above an "action level," usually the U.S. Centers for Disease Control and Prevention (CDC) reference value.¹⁸ This reference level was recently lowered in October 2021 to 3.5 μ g/dL, down from the level of 5 μ g/dL, which was implemented in 2012.¹⁹ Estimates suggest that 500,000 children in the United States have a BLL >5 μ g/dL and that 12 million additional children have a BLL between 1 and 5 μ g/dL,^{8,20} levels at which

Address correspondence to Elizabeth M. Kamai, 1845 N. Soto St., Los Angeles, CA 90032 USA. Email: kamai@usc.edu

The authors declare they have no actual or potential competing financial interests.

Received 16 September 2021; Revised 2 May 2022; Accepted 4 May 2022; Published 1 June 2022.

Note to readers with disabilities: *EHP* strives to ensure that all journal content is accessible to all readers. However, some figures and Supplemental Material published in *EHP* articles may not conform to 508 standards due to the complexity of the information being presented. If you need assistance accessing journal content, please contact ehpsubmissions@niehs.nih.gov. Our staff will work with you to assess and meet your accessibility needs within 3 working days.

lead still can cause harm.^{8,20} These children may be chronically exposed to low levels of lead with no intervention, resulting in both irreversible and intergenerational health effects.^{21,22}

Studies of childhood lead exposure are frequently missing outcome data for children who were never tested. Analyses that condition on testing (i.e., analyses restricted to children who received a blood lead test) may suffer selection bias,²³ and correlations between explanatory variables and children's BLLs may be induced by restricting to the subsample of tested children. To our knowledge, no study has considered how restricting analyses to children tested for lead could bias estimates of association between sources of lead exposure and children's BLLs. Further, only a single study, conducted in Pennsylvania, has evaluated individual-level maternal and infant predictors of receiving a blood lead test.²⁴ Here, by leveraging the digitization of all birth certificates for the state of North Carolina in the period 2011-2016 and linking them to records of all available children's blood lead testing results for the period 2011-2018, we investigated the determinants of who gets lead tested, who gets lead tested early in life (i.e., <30 months of age), and who is found to have elevated BLLs (EBLLs) among all children born and residing in North Carolina in the years 2011–2018. Further, using inverse probability (IP) of testing weights to reduce the potential impact of selection bias into lead screening and testing programs, we estimated the number of children born in North Carolina in 2011–2016 who have EBLLs but currently remain untested.

Methods

Study Setting

North Carolina is the ninth most populous state in the United States, with 10.5 million residents,²⁵ 20% of whom live in rural areas.²⁶ North Carolina has a long history of occupational lead exposure incidents,²⁷ recent adult and children's lead exposure concerns,^{28–30} and a variety of documented environmental sources of lead.^{31–34}

Cohort Definition

We defined a birth cohort of children who were born and reside in North Carolina from birth certificate records from 1 January 2011 through 1 July 2016 provided by the North Carolina State Center for Health Statistics (NC SCHS) using the following procedure: Of the 662,234 live births that received a North Carolina birth certificate during this time period, 15,882 were excluded because the state of residence at birth reported was not North Carolina. We geocoded the remaining residential addresses provided on the birth certificate using the Geocode Addresses tool within ArcMap, using Esri Business Analyst 2017 Address Locator [StreetMap Premium for ArcGIS North America HERE 2016 (release 3; Esri)]. An additional 13,193 (2.0% of births to in-state residents) could not be geocoded to address points, street addresses, or street names, leaving a final cohort of 633,159 children.

Lead Testing

The primary outcomes for this study were the receipt of at least one blood lead test before 30 months of age and, among those tested, the highest BLL reported in childhood. The North Carolina Childhood Lead Poisoning Prevention Program (NCCLPPP) collects the results of all blood lead tests conducted in children in North Carolina.³⁵ We linked records of blood lead tests conducted in North Carolina from 2011 to 2018 for children born in 2011 or later (n = 958, 194 test result records), with the geocoded birth certificate cohort using deterministic matching of components included in both data sets: child's first, middle, and last names;

child's date of birth; and residential address information. Prior to linkage, we removed all spaces and special characters from name variables. We defined full name match as either: a) exact match on both i) first name or concatenated first and middle names and ii) last name or concatenated last name and suffix (birth certificate only), or b) exact match on the concatenated first, middle, and last names (or last name and suffix, for birth certificates only). We defined our fuzzy name match using the soundex, compged, and spedis functions in SAS (version 9.4; SAS Institute, Inc.) or by matching on at least one part of a multipart name (e.g., Martinez-Garcia matching to Martinez or Garcia). We defined a partial date of birth match as matching on at least two of day, month, or year. We conducted linkage in steps, the first of which required a full name match, full date of birth match, and match on all location information (approximate address, city, county, and ZIP code). At each step, we loosened one piece of the matching requirements. The final step required only a fuzzy name match and a partial date of birth match. At every step, we randomly selected 50 linked pairs to manually evaluate the quality of the linkage. If any pairs appeared to be erroneously linked, the linkage rule was refined (e.g., using more conservative values for compged and spedis functions, adding more location information) or discarded.

Each birth linked to at least one lead test was assigned a binary variable to indicate that the child had received a blood lead test. Results of blood lead tests are reported in micrograms per deciliter. Prior to 1 July 2017, all results reported to the NCCLPPP with decimal places were rounded. On or after 1 July 2017, all results reported with decimal places were truncated (rounded down) to the integer. NCCLPPP normalized results below the limit of detection (LOD) to 1 µg/dL. Because laboratory and point-of-care instruments used to measure children's BLLs vary in their limits of detection, at least one common instrument has an LOD of $3.3 \mu g/dL$,^{36,37} and the CDC has recently lowered its children's blood lead reference level to $3.5 \mu g/dL$,¹⁹ we primarily dichotomized BLLs as <3 µg/dL (reference) or $\geq 3 \mu g/dL$.

Study Covariates

We included the following maternal demographic and clinical characteristics reported on birth certificates as potential covariates: year of birth, sex assigned at birth, birth weight, preterm birth (<37 completed weeks gestation), plurality, method of delivery (vaginal or caesarean), parity, number of older children, maternal age at delivery, maternal highest education completed, maternal marital status, maternal race, maternal Hispanic ethnicity, maternal place of birth, maternal reported number of prenatal care visits, maternal weight gain during pregnancy, maternal self-reported smoking, maternal diabetes during and prior to pregnancy, primary form of payment (e.g., private insurance, Medicaid), and maternal receipt of Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) benefits during pregnancy. These characteristics are typically self-reported by the parent(s) and may result in WIC and Medicaid enrollment being underreported (V. DiBona, personal communication).

The 2011–2016 North Carolina birth certificate data provided by the State Center for Health Statistics classified maternal race using bridged-race methodology per the National Center for Health Statistics categories³⁸ into the following options: American Indian or Alaska Native (9,142, 1.4%), Black or African American (160,600, 24.3%), Chinese (3,110, 0.47%), Filipino (1,973, 0.30%), Japanese (542, 0.08%), Native Hawaiian (87, 0.01%), other Asian (21,137, 3.2%), other non-White (77,450, 11.7%), or White (388,193, 58.6%). This methodology assigns mixed-race individuals to a single racial category.³⁹ It is unknown whether maternal race on each birth certificate is self-reported or reported by a family member, clinician present at delivery, or someone else. We used race as a social construct in our models, not as a biologic or etiologic factor but, rather, as a proxy for differences between racial groups arising from historical and structural racism.^{40–42} We chose to collapse Asian Indian, Chinese, Japanese, Native Hawaiian, Filipino, and other Asian into a single Asian and Pacific Islander (AAPI) category given both the small numbers and expected greater similarities in lived experiences compared with mothers of other racial groups.

We linked publicly available data sources with geocoded residential addresses at birth [using ArcMap (version 10.7; Esri)] to describe the physical and social environment of the cohort. We primarily used data from 2010 to preserve temporality and ensure that predictive variables were assessed prior to the outcome of lead testing for all children. We assigned addresses with values of the 2010 U.S. Census and 2006-2010 5-y American Community Survey (ACS) block group and tract and included population-level measures of income, poverty, race, ethnicity, education, and housing characteristics (complete list in Table S1). We evaluated both tractand block group-level measures to identify those with the strongest predictive values. All census and ACS variables were categorized into quintiles after assignment to births. We also created a binary variable to indicate whether addresses were within a censusdesignated urbanized area, defined as densely populated areas of \geq 50,000 people, including dense suburban development.⁴³ Although newly built dense suburban areas do not have the same historic lead contamination as older cities⁴⁴—such as emissions from vehicles using leaded gasoline or paint in housing built before 1978—both are included in the definition of metropolitan statistical areas, resulting in systematically different eligibility for and allocation of federal funds to these areas than to less densely populated communities.⁴⁵ Moreover, we used additional covariates (see below) to account for many historic sources of environmental lead.

More than 2 million people in North Carolina rely on wells for drinking water,^{46,47} and there is recent evidence that BLLs of children who use private wells are 20% higher than those of children served by community water systems in Wake County, North Carolina.³¹ Drinking water from private wells is not regulated under the Safe Drinking Water Act, and in North Carolina, only newly permitted wells are required to be tested for contaminants; moreover, there is no comprehensive database or characterization of households in North Carolina that rely on private wells.⁴⁸ To address this potential source of lead exposure, we used a publicly available geographic information system data set of the boundaries of public water systems in North Carolina developed by the North Carolina Center for Geographic Information and Analysis.^{49,50} These data were mapped first using system boundaries in 2004–2006 and then projected for the year 2010 based on water system owners' expected future boundaries.⁵⁰ One polygon ("Nash County 1 & 2") from the 2004–2006 map was not included in the area of the projected map and was merged with the projected 2010 boundaries polygons. We categorized water system service area sizes using Safe Drinking Water Act⁵¹ cutoffs: large (>50,000), medium (10,001–50,000), small (3,301-10,000), very small ($\leq 3,300$) people. We spatially linked births to both sets of boundaries and assigned them the water system to which they were linked, using projected 2010 boundaries in our primary analyses given that we expected these areas to more closely reflect the water systems during the study period (2010-2018). However, we used the mapped 2004-2006 boundaries in our sensitivity analyses. We created a binary variable to describe whether a residential address at birth was within 100 m of a major roadway-defined as an interstate or principal arterial-using spatial data from the North Carolina Department of Transportation.⁵²

Finally, we linked births to Toxics Release Inventory (TRI) sites that released lead or lead compounds and National Emissions Inventory (NEI) stationary point sources that emitted lead. The TRI is a mandatory U.S. Environmental Protection Agency (EPA) program that requires industrial facilities employing ≥ 10 full time employees to report environmental releases of toxic chemicals.53 These reports have been released annually since 1988 (which reported on releases in 1987). The NEI is an estimate of emissions of air pollutants regulated by the U.S. EPA, released every 3 y beginning in 2008.⁵⁴ Stationary point sources in the NEI include airports, industrial facilities, and power plants. All TRI reports and NEI reports for lead or lead compound releases or emissions in North Carolina and neighboring states were downloaded from the Envirofacts database online⁵⁵ and the NEI website,⁵⁶ respectively. The coordinates of the facilities provided in the reports were geocoded in ArcMap, and we selected those within 2 km of the North Carolina border. Lead is extremely environmentally stable in soil, so releases from decades prior can still contribute to today's soil lead content.⁶ Because of the wide variability in reported levels of lead released or emitted since the inceptions of the TRI and NEI programs, we categorized reported TRI releases and NEI emissions below the median (<9.4 kg lead released for TRI reports in 1987–2008; <0.13 kg emitted for NEI reports in 2008–2017) as being low lead and those at or above the median as being high lead. For our main analyses, births were spatially linked to TRI and NEI sites within 2 km and reporting releases or emissions in the 5 y prior to and including the year of birth. For example, for births in 2014, the exposure time period of interest was 2009-2014, so births in 2014 were linked to all TRI and NEI sites within 2 km that reported releasing or emitting lead at any point in 2009–2014. We chose to use releases and emissions in the previous 5 y for a variety of reasons: because reporting did not appear consistent year to year (with facilities missing emissions reports for some years); because the NEI was released only every 3 y; and to balance both recent and legacy lead pollution. Proximity to these sites was further categorized as 0-1 or 1-2 km, and births >2 km from any sites were used as the referent. If there were multiple TRI or NEI reports filed at one or more sites within 2 km of a residence, we used the highest amount of lead reported under each program during the exposure time period of interest and within each category of proximity. Alternative exposure definitions were explored in our sensitivity analyses, described below.

Statistical Methods

We linked individual and neighborhood characteristics of children with birth certificates to blood lead test results and compared them with children without a corresponding blood lead test result to determine the probability of the following outcomes: a) receiving a blood lead test at any age, b) receiving at least one blood lead test by 30 months of age, or c) receiving a blood lead test at both 1 year of age (6–18 months) and 2 years of age (18–30 months).

We assessed the probability of receiving at least one blood lead test at <30 months of age (conditional on maternal, clinical, and neighborhood characteristics at birth) using logistic regression models. We examined potential covariates in bivariate models to estimate the association between each covariate and the probability of receiving a blood lead test at <30 months of age. We then added covariates significant in bivariate models at $\alpha < 0.20$ one at a time to a multipredictor logistic model in descending order of beta estimate. Because we had minimal missing data, we conducted complete case analyses. We retained variables if their addition improved the fit of the model [assessed using the c-index value (equivalent to the area under the receiver operator curve) and Akaike information criterion (AIC)] and did not result in model convergence problems. We further evaluated **Table 1.** Descriptive characteristics [n (%)] of geocoded North Carolina births 2011–2016, stratified by linkage to lead testing in North Carolina, 2011–2018.

Characteristics at birth	North Carolina	Tested for lead at 0–30 months of age	Cases: BLL $\geq 3 \ \mu g/dL$ at 0–30 months of age
Total live births	633,159	402,002 (64)	39,855 (10) ^a
Child sex assigned at birth ^b			
Female	309,818 (49)	196,947 (49)	18,613 (47)
Male	323,329 (51)	205,053 (51)	21,242 (53)
Missing Prenatal care visits ^{<i>b,c</i>}	12	2	—
	141,067 (22)	90,721 (23)	9,682 (24)
≥ 10	487,586 (78)	308,343 (77)	29,869 (75)
Missing	4,506	2,938	304
Payment (insurance) at delivery b,c			
Medicaid	277,906 (44)	212,319 (53)	25,531 (64)
Private	280,289 (44)	144,459 (36)	10,431 (26)
Self-pay	45,509 (7)	34,129 (9)	3,102 (8)
Other Missing	28,817 (5) 638	10,964 (3) 131	773 (2) 18
Older children ^{b,c}	050	151	10
None	258,252 (41)	164,768 (41)	16,622 (42)
1	203,753 (32)	125,746 (31)	11,789 (30)
≥2	171,027 (27)	111,424 (28)	11,438 (29)
Missing	127	64	6
Reported smoking before or during pregnancy ^{b,c}			0.050 (20)
Any	85,047 (13)	59,559 (15)	8,050 (20)
None Missing	547,981 (87) 131	342,391 (85) 52	31,802 (80) 3
Maternal age at delivery $(y)^{b,c}$	151	52	5
≤ 20	74,184 (12)	55,582 (14)	7,227 (18)
21–25	163,999 (26)	112,743 (28)	12,636 (32)
26–30	182,754 (29)	112,606 (28)	10,219 (26)
31–35	144,125 (23)	82,103 (20)	6,558 (17)
>35	68,086 (11)	38,961 (10)	3,213 (8)
Missing	11	7	2
Maternal marital status ^{b,c}	276 216 (50)	207 666 (52)	17 791 (45)
Married Unmarried	376,316 (59) 256,657 (41)	207,666 (52) 194,227 (48)	17,781 (45) 22,059 (55)
Missing	186	109	15
Maternal education at delivery b,c	100	109	15
Less than HS	105,525 (17)	81,354 (20)	9,924 (25)
HS graduate	141,022 (22)	101,262 (25)	11,686 (29)
Some college or associate's degree	199,684 (32)	128,985 (32)	12,028 (30)
Bachelor's degree or more	185,844 (29)	89,662 (22)	6,158 (16)
Missing WIC ^{b,c}	1,084	739	59
Reported on birth certificate	298,832 (47)	227,693 (57)	26,116 (66)
None	333,121 (53)	173,624 (43)	13,670 (34)
Missing	1,206	685	69
Maternal Hispanic ethnicity ^{b,c}	_,		
Hispanic	95,447 (15)	71,194 (18)	5,775 (15)
Non-Hispanic	537,410 (85)	330,675 (82)	34,061 (86)
Missing	302	133	19
Maternal race ^{b,c}	0.446(1)	(150 (0)	972 (2)
American Indian Asian and Pacific Islander	8,446 (1) 25,864 (4)	6,459 (2) 12,325 (3)	873 (2) 1,332 (3)
Black	154,067 (24)	12,323 (3)	1,923 (30)
White	369,536 (58)	216,196 (54)	20,926 (53)
Other non-White	75,246 (12)	59,114 (15)	4,801 (12)
Mother's state of birth ^{b,c}	· · · · ·	· · · · · ·	· · · · ·
North Carolina	286,676 (46)	205,358 (51)	23,108 (58)
Other U.S. states and territories	233,692 (37)	119,967 (30)	10,078 (25)
Remainder of world	109,087 (17)	74,834 (19)	6,537 (16)
Missing Year of birth ^{b,c}	3,704	1,843	132
2011	115,276 (18)	73,116 (18)	10,934 (27)
2011 2012	115,276 (18) 114,744 (18)	73,480 (18)	7,571 (19)
2012	114,154 (18)	71,347 (18)	6,281 (16)
2013	116,190 (18)	71,642 (18)	6,339 (16)
2015	116,197 (18)	74,878 (19)	6,184 (16)
2016	56,598 (9)	37,539 (9)	2,546 (6)

Characteristics at birth	North Carolina	Tested for lead at 0–30 months of age	Cases: BLL $\geq 3 \ \mu g/dL$ a 0–30 months of age
Environmental characteristics			
Residence in urbanized area			
Yes	387,548 (61)	227,216 (57)	19,714 (49)
No	245,611 (39)	174,786 (43)	20,141 (51)
Distance from major roadway ^{b,c}			
Within 100 m	23,316 (4)	16,029 (4)	1,799 (5)
>100 m	609,843 (96)	385,973 (96)	38,056 (95)
Estimated 2010 public water system service populati	on size ^{b,c}		
>50,000	321,736 (51)	190,524 (47)	16,839 (42)
10,001-50,000	130,574 (21)	87,093 (22)	8,800 (22)
3,301-10,000	52,069 (8)	37,316 (9)	3,911 (10)
≤3,300	26,247 (4)	19,339 (5)	2,398 (6)
Not on public water	102,533 (16)	67,730 (17)	7,907 (20)
Within 2 km of TRI or NEI site emitting/releasing le	ad in past 5 $y^{b,c}$		
TRI sites	68,291 (11)	45,346 (11)	4,959 (12)
Low lead (<median)< td=""><td>40,868 (6)</td><td>27,314 (7)</td><td>2,738 (7)</td></median)<>	40,868 (6)	27,314 (7)	2,738 (7)
High lead (≥median)	27,423 (4)	18,032 (4)	2,221 (6)
None	564,868 (89)	356,656 (89)	34,896 (88)
NEI sites	165,217 (26)	111,926 (28)	12,290 (31)
Low lead (<median)< td=""><td>89,692 (14)</td><td>60,556 (15)</td><td>6,163 (15)</td></median)<>	89,692 (14)	60,556 (15)	6,163 (15)
High lead (≥median)	75,525 (12)	51,370 (13)	6,127 (15)
None	467,942 (74)	290,076 (72)	27,565 (69)

Note: ---, not applicable; BLL, blood lead level; HS, high school; NEI, National Emissions Inventory; TRI, Toxics Release Inventory; WIC, Special Supplemental Nutrition Program for Women, Infants, and Children.

^aPercentage of children tested at <30 months of age.

^bStatistically significant difference in distribution between BLL $\geq 3 \mu g/dL$ and BLL $< 3 \mu g/dL$ at 0–30 months of age at p < 0.05 using chi-square testing.

 $^{\circ}$ Statistically significant difference in distribution between tested and not tested at 0–30 months of age at p < 0.05 using chi-square testing.

model prediction validity by estimating model parameters using births in even months and applying those parameters to births in odd months.

We used the predicted probability of testing produced from this model to create IP of testing weights to reduce potential for selection bias in lead testing. These weights were applied to the tested children to approximate the distribution of covariates in the total cohort and to derive the expected number and characteristics of children with elevated blood lead (defined as $\geq 3 \ \mu g/dL$) in the total cohort, accounting for the missing data on blood lead results for those children for whom test results are not available. We further calculated the number and characteristics of children who were missed by lead screening and did not receive a blood lead test result by subtracting these estimates from the number of actual children in the cohort with blood lead results at each covariate and test outcome level. We repeated this process in 500 bootstrapped samples (with replacement) to produce a 95% credible interval (CrI) for these estimates.

The estimated risk of receiving an elevated ($\geq 3 \ \mu g/dL$) blood lead test result at <30 months of age, conditional on maternal, clinical, and neighborhood characteristics at birth, was assessed using generalized linear models with robust error measurements to account for births with the same residential address at birth. Initially, all covariates were examined in bivariate regression models. As with the earlier models, covariates were added stepwise to a multipredictor model and retained if their addition improved the fit of the model (assessed using assessed using the c-index value and AIC). If the addition of a new predictor changed the statistical significance ($\alpha = 0.05$) of a previously added predictor, a step was added to determine whether removing the first predictor affected model fit. We also evaluated interactions between measures of housing age and housing value. Models were additionally adjusted for season, using the month the lead test was conducted [winter (referent): December, January, February; spring: March, April, May; summer: June, July, August; fall: September, October, November]. The type of blood used (venous vs. capillary), the location of testing (point of care vs. laboratory), year of test, and age at testing in months were explored as potential explanatory variables. The final models were selected to maximize the adjusted model cstatistic and minimize AIC for best model fit. We evaluated model prediction validity by estimating model parameters using births in even months and applying those parameters to births in odd months. Finally, we applied IP of testing weights to all models to account for the missingness of blood lead results among children never tested for lead.

Sensitivity Analyses

We conducted sensitivity analyses to evaluate the robustness of the results. To evaluate the sensitivity of the results to the exposure definition, the threshold distance to a TRI or NEI site was reduced to 1 km, the exposure time period was both restricted to 1 y prior to the year of birth and expanded to the entire reporting period, and indicators of proximity to TRI and NEI sites were combined into a single variable for point sources of environmental lead. To look at the tails of the lead release and emissions distributions, we additionally evaluated very high lead sites as those emitting or releasing above the 90th percentile of lead in North Carolina (>2,762 kg lead released for TRI reports, 53 >18.5 kg lead emitted for NEI reports⁵⁶) and very low lead sites as those in the 10th percentile of lead released or emitted in North Carolina (<0.0064 kg lead released for TRI reports,⁵³ <0.0036 kg lead emitted for NEI reports⁵⁶). In assessing which residences were close to a major roadway, we excluded roadways built in the year 2000, given that lead was fully removed from automotive gasoline by 1996,² making newer roadways a less important source of lead exposure. To account for residential and demographic characteristics changing between birth and lead testing, models of BLLs were restricted to children at the same address at both birth and testing, as well as restricted to children within the same ZIP code at both birth and testing. We additionally excluded children born in two counties that contain the two largest U.S. military bases in North Carolina⁵⁷ (Fort Bragg in Cumberland County and Camp Lejeune in Onslow County) due to the possibility that

Table 2. Selected U.S. Census characteristics [median (IQR)] of geocoded North Carolina births 2011–2016, stratified by linkage to lead testing in North
Carolina, 2011–2018.

Characteristics ^{<i>a,b</i>}	North Carolina	Tested for lead at 0–30 months of age	Cases: BLL $\geq 3 \ \mu g/dL$ at 0–30 months of age
Block group population size	1,809 (1,159)	1,739 (1,092)	1,663 (1,049)
Block group population density (per mile ²)	943 (2,152)	804 (2,108)	641 (1,984)
Tract population born in North Carolina (%)	58 (27)	62 (24)	67 (21)
Tract households with ≥ 1 child (%)	35 (12)	34 (11)	34 (10)
Block group population (%)			
Asian, non-Hispanic	1 (2)	1 (2)	1 (2)
Black, non-Hispanic	17 (29)	19 (31)	20 (35)
White, non-Hispanic	65 (41)	64 (45)	63 (47)
Hispanic or Latino, any race	7 (9)	7 (10)	6 (10)
Block group population ≥ 25 years of age with less than HS education (%)	15 (17)	17 (17)	19 (17)
Block group households earning <\$20,000 (%)	18 (18)	20 (19)	23 (20)
Block group housing units (%)			
Renter-occupied	30 (32)	30 (31)	31 (31)
Vacant	9 (6)	10 (6)	11 (7)
Built pre-1950	5 (12)	6 (13)	9 (16)
Built pre-1940	2 (7)	3 (8)	4 (11)
Tract median value of owner-occupied housing units	\$136,100 (\$75,300)	\$128,900 (\$68,100)	\$119,100 (\$62,800)

Note: BLL, blood lead level; HS, high school; IQR, interquartile range.

a Statistically significant difference in distribution between BLL $\geq 3 \mu g/dL$ and BLL $< 3 \mu g/dL$ at 0–30 months of age at p < 0.05.

^bStatistically significant difference in distribution between tested and not tested at 0–30 months of age at p < 0.05.

many children born on military bases may not continue to reside there later in childhood. Finally, analyses were repeated using the more conservative definition of elevated blood lead, $\geq 5 \ \mu g/dL$, and using blood lead test results at any age in childhood.

All models were run in SAS (version 9.4; SAS Institute Inc.). This study was approved by the institutional review board at the University of North Carolina at Chapel Hill. Informed consent was waived because no subjects were contacted and only the study team had access to the data.

Results

Blood Lead Testing

Overall, 419,001 (66.2%) of the geocoded 2011-2016 North Carolina birth certificates were linked to at least one of 861,870 (89.9%) of the children's lead tests conducted in North Carolina and reported to the NCCLPPP in 2011-2018, irrespective of age at time of testing. One third (33.8%) of children born in North Carolina in this period had no report of blood lead testing linked to their birth record. Ten percent of lead test reports could not be linked to a birth certificate because North Carolina's population has grown rapidly since 2010 as a result of in-migration,⁵⁸ we expect most of the unlinked reports belonged to children born out of state. Nearly all of the linked tests (402,002; 95.9%) were for children <30 months of age, making the probability of receiving a lead test by 30 months of age only slightly lower (64%) than the overall probability of testing at any age in this cohort. Less than one-third of children born in North Carolina were tested for lead at both 1 and 2 years of age (n = 199,707, 31.5%).

Birth certificates of children who were linked to a lead test result were more likely to report the following maternal characteristics at delivery: <25 years of age at delivery (42% vs. 38%); unmarried (48% vs. 41%); mother born in North Carolina (51% vs. 45%); covered by Medicaid (53% vs. 44%); receiving WIC benefits (57% vs. 47%); Hispanic ethnicity (18% vs. 15%), American Indian (2% vs. 1%), Black (27% vs. 24%), or other non-White race (15% vs. 12%); and less than college education (45% vs. 39%) (Table 1). Compared with the birth cohort, residential addresses at birth of the children tested for lead were more likely be in less-urban, sparsely populated neighborhoods, with higher proportions of people born in North Carolina and have more adults with less than a high school education, more children and families living in poverty, more vacant housing, more older housing stock (built before 1950 and 1940), lower median home value, lower proportions of Asian neighbors, and higher proportions of Black and African American neighbors (Table 2).

The final logistic regression model of the probability of receiving a blood lead screening test at <30 months of age included the following covariates: county of birth; year of birth; payment at delivery (Medicaid, private insurance, self-pay, other), maternal race (American Indian, AAPI, Black, White, other non-White); whether the mother had reported receiving WIC during pregnancy (yes/no), the number of previous live births still living (none, 1, ≥ 2); maternal marital status (married or unmarried); maternal location of birth (North Carolina, other U.S. states and territories, or international); maternal prenatal care (indicator for ≥ 10 prenatal care visits); percentage of tract population born in North Carolina (quintile); percentage of block group population <18 years of age (quintile); block group population density per square mile (quintile); percentage of block group population with less than high school education (quintile); percentage of block group housing that is renter-occupied (quintile); percentage of block group population White alone, not Hispanic or Latino (quintile); percentage of block group Hispanic or Latino (quintile); and 2010 estimated public water system service area size (>50,000 people, 10,001-50,000 people, 3,301-10,000 people, $\leq 3,300$ people, not on public water) (see Table S2 for beta estimates and standard errors). The c-statistic of the final model was 0.750, indicating moderately good predictive value. The IP of testing weights among those with lead test results ranged from 1.02 to 8.87 (mean = 1.56). Applying model parameters estimated using births from odd months to births in even months predicted lead testing with 72% accuracy, with a positive predictive value of 0.75 and a negative predictive value of 0.66.

BLLs

Among the 402,002 children that received a blood lead test at <30 months of age, 41,839 (10.4%) received a maximum test result of 3 μ g/dL or higher, while 10,677 (2.66%) received a maximum test result of 5 μ g/dL or higher, the CDC reference level at the time most of these tests were conducted. Just under 2,000 children (1,892, 0.47%) received a maximum test result of 10 μ g/dL or higher, the CDC reference level until 2012.

Risk of BLL \geq 3 \mu g/dL. In bivariate models, many predictors were strongly correlated with the risk of receiving a blood lead

Characteristics at birth	Bivariate, unweighted	Bivariate, IP-weighted to total population	Full model, IP weighted to total population ^a
Child female sex at birth (Ref = male)	0.91 (0.90, 0.93)	0.92 (0.90, 0.94)	0.93 (0.91, 0.95)
Payment (insurance) at delivery (Ref = Private or other)			
Medicaid	1.67 (1.63, 1.70)	1.74 (1.70, 1.78)	1.35 (1.32, 1.39)
Self-pay	1.26 (1.21, 1.31)	1.32 (1.27, 1.37)	1.30 (1.24, 1.36)
Reported smoking before or during pregnancy (Ref = none) Maternal age at delivery (y)	1.46 (1.42, 1.49)	1.50 (1.46, 1.54)	1.14 (1.12, 1.17)
≤ 20	1.43 (1.39, 1.47)	1.49 (1.45, 1.54)	1.16 (1.13, 1.20)
21–25	1.24 (1.20, 1.27)	1.26 (1.23, 1.30)	1.07 (1.05, 1.10)
26–30 (Ref)			
31–35	0.88 (0.85, 0.91)	0.87 (0.85, 0.90)	0.97 (0.93, 1.00)
>35 Matematika (Defense Riesenie)	0.91 (0.87, 0.94)	0.91 (0.87, 0.94)	1.01 (0.97, 1.05)
Maternal Hispanic ethnicity (Ref = non-Hispanic) Maternal race (Ref = White)	0.79 (0.77, 0.81)	0.83 (0.81, 0.86)	0.81 (0.76, 0.87)
American Indian	1.40 (1.31, 1.49)	1.49 (1.39, 1.59)	1.10 (1.03, 1.17)
Asian and Pacific Islander	1.12 (1.06, 1.18)	1.20 (1.13, 1.28)	1.33 (1.24, 1.42)
Black	1.14 (1.12, 1.17)	1.19 (1.16, 1.21)	0.98 (0.96, 1.01)
Other non-White	0.84 (0.81, 0.87)	0.90 (0.88, 0.93)	0.92 (0.86, 0.99)
Mother's state of birth (Ref = North Carolina)			
Other U.S. states and territories	0.75 (0.73, 0.76)	0.70 (0.69, 0.72)	0.92 (0.90, 0.95)
Remainder of world	0.78 (0.76, 0.80)	0.81 (0.78, 0.83)	1.10 (1.05, 1.15)
Year of birth	1.70(1.65, 1.75)	1.70(1.(5, 1.75))	1 25 (1 24 1 49)
2011 2012	1.70(1.65, 1.75)	1.70 (1.65, 1.75)	1.35 (1.24, 1.48)
2012 2013 (Ref)	1.17 (1.13, 1.21)	1.17 (1.13, 1.21)	1.10 (1.04, 1.16)
2013 (Ref)	1.01 (0.97, 1.04)	1.02 (0.98, 1.05)	0.89 (0.84, 0.94)
2015	0.94 (0.91, 0.97)	0.96 (0.93, 1.00)	0.89(0.84, 0.94) 0.89(0.81, 0.97)
2016	0.77 (0.74, 0.81)	0.79 (0.75, 0.83)	0.81 (0.71, 0.91)
Environmental characteristics	0177 (0171, 0101)	0177 (0172, 0102)	0101 (01/1, 01/1)
Residence in urbanized area ($Ref = no$)	0.75 (0.74, 0.77)	0.73 (0.72, 0.75)	0.94 (0.92, 0.97)
Within 100 m of major roadway (Ref $>$ 100 m)	1.14 (1.09, 1.19)	1.18 (1.12, 1.24)	1.10 (1.05, 1.15)
Public water system (Ref = not on public water) Within 2 km of TRI or NEI site emitting/releasing lead in past 5 y (Ref > 2 km)	0.81 (0.80, 0.83)	0.80 (0.78, 0.82)	0.85 (0.83, 0.87)
TRI sites			
Low lead (\leq median)	1.02 (0.99, 1.06)	1.06 (1.02, 1.10)	1.04 (1.00, 1.08)
High lead (>median)	1.26 (1.21, 1.32)	1.26 (1.21, 1.33)	1.08 (1.03, 1.14)
NEI sites			
Low lead (≤median)	1.07 (1.04, 1.10)	1.10 (1.07, 1.13)	1.09 (1.06, 1.12)
High lead (>median)	1.26 (1.22, 1.29)	1.27 (1.23, 1.31)	1.11 (1.07, 1.14)
Selected U.S. Census characteristics, categorized in quintiles			
Population born in state [Q (%)]			
1	0.74 (0.71, 0.77)	0.73 (0.70, 0.76)	0.89 (0.86, 0.94)
2	0.84 (0.82, 0.87)	0.83 (0.80, 0.86)	0.94 (0.90, 0.97)
3 (Ref)		1 21 (1 20 1 25)	
4	1.30 (1.26, 1.33)	1.31 (1.28, 1.35)	1.14 (1.10, 1.17)
5 Vecent housing $[O(\%)]$	1.39 (1.35, 1.43)	1.41 (1.37, 1.45)	1.15 (1.11, 1.18)
Vacant housing [Q (%)]	0.75 (0.73, 0.78)	0.74 (0.71, 0.77)	0.96 (0.92, 1.00)
$\frac{1}{2}$	0.94 (0.91, 0.97)	0.93 (0.89, 0.96)	1.01 (0.98, 1.04)
2 (Ref)	0.94 (0.91, 0.97)		
4	1.14 (1.10, 1.17)	1.15 (1.12, 1.19)	1.05 (1.02, 1.09)
5	1.31 (1.28, 1.35)	1.33 (1.29, 1.37)	1.14 (1.11, 1.17)
Housing built before 1940 [Q (%)]			
1	0.78 (0.76, 0.80)	0.77 (0.74, 0.79)	0.91 (0.86, 0.97)
2	0.90 (0.86, 0.93)	0.88 (0.85, 0.92)	0.98 (0.92, 1.04)
3 (Ref)	_	_	_
4	1.15 (1.12, 1.19)	1.17 (1.13, 1.21)	1.05 (0.99, 1.12)
5	1.36 (1.31, 1.40)	1.38 (1.33, 1.42)	1.18 (1.11, 1.25)
Housing built before 1950 [Q (%)]			
1	0.77 (0.74, 0.80)	0.77 (0.74, 0.80)	1.00 (0.95, 1.05)
2	0.87 (0.84, 0.90)	0.87 (0.84, 0.90)	1.02 (0.98, 1.06)
3 (Ref)			1.02 (1.00, 1.07)
4	1.18(1.14, 1.22) 1.40(1.26, 1.44)	1.21 (1.17, 1.25)	1.03 (1.00, 1.07) 1.10 (1.05, 1.14)
5 Selected interactions (Ref = 0.3 median home value, 0.3 housing built before 1040)	1.40 (1.36, 1.44)	1.42 (1.38, 1.47)	1.10 (1.05, 1.14)
Selected interactions (Ref = Q3 median home value, Q3 housing built before 1940) Q1 housing built before 1940 (least) Q1 median home value (lowest)			1 12 (1 04 1 20)
Q1 housing built before 1940 (least), Q1 median home value (lowest) Q1 housing built before 1940 (least), Q5 median home value (highest)	_		1.12(1.04, 1.20) 1.07(0.99, 1.14)
Q5 housing built before 1940 (nest), Q5 median home value (highest) Q5 housing built before 1940 (most), Q1 median home value (lowest)			$1.07 (0.99, 1.14) \\ 0.95 (0.90, 1.01)$
Q5 housing built before 1940 (most), Q5 median home value (lowest)			1.06 (0.97, 1.17)
Age at test (months, continuous)	1.08 (1.08, 1.08)	1.08 (1.08, 1.08)	1.08 (1.08, 1.08)
Laboratory test vs. point-of-care test	1.15 (1.12, 1.18)	1.14 (1.11, 1.17)	1.11 (1.08, 1.13)

Table 3. (Continued.)

Characteristics at birth	Bivariate, unweighted	Bivariate, IP-weighted to total population	Full model, IP weighted to total population ^a
Season (Ref = winter)			
Fall	1.25 (1.21, 1.28)	1.22 (1.19, 1.26)	1.12 (1.08, 1.15)
Spring	1.09 (1.06, 1.12)	1.08 (1.04, 1.11)	1.09 (1.06, 1.12)
Summer	1.27 (1.24, 1.30)	1.25 (1.21, 1.28)	1.19 (1.16, 1.23)
Year of test			
2011	1.38 (1.13, 1.69)	1.44 (1.16, 1.79)	2.12 (1.68, 2.68)
2012	1.31 (1.27, 1.36)	1.32 (1.28, 1.37)	1.41 (1.29, 1.54)
2013	1.08 (1.05, 1.12)	1.10 (1.06, 1.14)	1.03 (0.98, 1.09)
2014 (Ref)	_		
2015	0.86 (0.83, 0.89)	0.86 (0.83, 0.89)	1.00 (0.95, 1.06)
2016	0.95 (0.92, 0.98)	0.99 (0.96, 1.03)	1.19 (1.09, 1.31)
2017	0.82 (0.79, 0.86)	0.85 (0.81, 0.88)	0.98 (0.87, 1.11)
2018	1.77 (1.68, 1.88)	1.81 (1.70, 1.92)	1.18 (1.00, 1.39)

Note: CI calculated using robust standard errors to account for tested children living at the same address. —, not applicable; BLL, blood lead level; CI, confidence interval; IP, inverse probability; Q, quintile; NEI, National Emissions Inventory; Ref, reference; RR, risk ratio, TRI, Toxics Release Inventory. "Full model included all variables listed in this table.

test result $\geq 3 \,\mu g/dL$ (Table 3). Weighting the tested subpopulation so that it was comparable (with respect to covariates) to the overall cohort of live North Carolina births generally strengthened these correlations. The final multipredictor model of the risk of receiving a blood lead test result $\geq 3 \,\mu g/dL$ included birth certificate variables (year of birth, sex, insurance, maternal race, maternal Hispanic ethnicity, maternal age, maternal smoking, and maternal state of birth), environmental factors (residential proximity to TRI and NEI sites and major roadways, access to a public water system), neighborhood characteristics (proportion of the population born in North Carolina, proportion of housing built before 1950 and before 1940, proportion of vacant housing, whether the residence was in an urbanized area), information reported on test results (season of test, age at testing, specimen type), and an interaction term between housing built before 1940 and median home value (Table 3).

In covariate-adjusted models, a number of variables were positively associated with having a BLL $\geq 3 \mu g/dL$ (Table 3). Children born in 2011 [risk ratio (RR) = 1.35; 95% confidence interval (CI): 1.24, 1.48 compared with 2013], and whose birth certificates reported young maternal age (RR = 1.16; 95% CI: 1.13, 1.20 for those <20 years of age compared with those 26–30 years of age), maternal smoking (RR = 1.14; 95% CI: 1.12, 1.17), a mother born outside the United States (RR = 1.10; 95% CI: 1.05, 1.15 compared with born in North Carolina), AAPI or American Indian race (RR = 1.33; 95% CI: 1.24, 1.42 and RR = 1.10; 95% CI: 1.03, 1.17, respectively, compared with White race), or non-Hispanic ethnicity (RR = 0.81; 95% CI: 0.76, 0.87 for Hispanic compared with non-Hispanic ethnicity) had higher risk of elevated blood lead results. Although in IP-weighted bivariate models, children of Black mothers had 1.19 times the risk of receiving a blood lead test result $\geq 3 \,\mu g/dL$ compared with children of White mothers, in full models with all covariates there was no difference in risk between the two groups. Residential addresses that were within 2 km of a lead-releasing TRI or NEI facility, within 100 m of a major roadway, not on a public water service system, and in neighborhoods with older or vacant housing were also associated with higher odds of elevated blood lead results. We also found that addresses in census tracts with the fewest homes built before 1940 but the lowest home values were more likely to be linked to an elevated blood lead test result. The area under the receiver operator curve of the final predictive model was 0.698, indicating it had moderate predictive utility. Applying model parameters estimated using births from odd months to births in even months predicted lead testing with 90% accuracy, with a positive predictive value of 0.43 and a negative predictive value of 0.90.

Several sensitivity analyses were conducted to evaluate the robustness of these results to alternative specifications (Tables S3-S4). Restricting children to those residing at the same address or within the same city, ZIP code, or county at birth and at testing, and modeling the risk of BLLs $\geq 3 \,\mu g/dL$ at any age, did not alter our conclusions (Table S3). Similarly, excluding children born in counties with the two largest U.S. military bases in the state did not change the results (Table S3). Alternative definitions of residential proximity to lead-emitting or -releasing sites altered risk estimates (Table S4). Restricting sites to those within 1 km of residence at birth nullified the risk associated with proximity to TRI sites but did not substantially alter NEI estimates. Restricting sites to those releasing or emitting lead within 1 y of birth slightly strengthened results for TRI sites, whereas expanding the exposure definition to all sites ever reported releasing or emitting lead within 2 km of residential address at birth nullified associations between EBLLs and TRI sites but did not substantially change associations with NEI sites.

Risk of BLL $\geq 5 \mu g/dL$. Applying the same predictors to model the risk of a blood lead test $\geq 5 \mu g/dL$ altered the results (Table S5). Compared with children of White mothers (as reported on the birth certificate), children of AAPI mothers were 1.6 times more likely (95% CI: 1.42, 1.82) to be linked to a blood lead test result $\geq 5 \mu g/dL$, whereas children of Black mothers were less likely to be linked to a blood lead test result $\geq 5 \mu g/dL$. We observed stronger associations for maternal smoking and neighborhoods with high proportions of housing built before 1950, but reduced or null associations with residential proximity to TRI and NEI sites.

EBLLs missed by lead surveillance. When IP of testing weights were applied to the children tested for lead, the reweighted population included an estimated 57,398 children (9.2%) with a BLL $\geq 3 \mu g/dL$, 14,522 (2.3%) children with a BLL $\geq 5 \mu g/dL$, and 2,520 children (0.4%) with a BLL $\geq 10 \ \mu g/dL$. By subtracting the number of true elevated blood lead tests from those in the weighted population, we estimate that in our study population, between 2011-2018, 17,543 additional children had BLLs \geq 3 µg/dL who were never tested for lead (95% CrI: 17,462, 17,650; 7.9% of those not tested), including 4,457 with BLLs \geq 5 µg/dL (95% CrI: 4,435, 4,482; 2.0% of those not tested) (Table 4, Figure 1; Table S6). Equivalently, current lead testing strategies in North Carolina appear to miss $\sim 30\%$ of children with EBLLs (regardless of whether >3 or >5 μ g/DL is used as the threshold). Moreover, children with characteristics at birth associated with the highest relative risks for EBLLs in childhood frequently contribute the fewest cases. For example, selfreported Medicaid status during pregnancy was associated with

	All additional projected	Estimated number of additional children with EBLL [n (% of row)]	
Characteristics at birth	untested children [<i>n</i> (% of all untested children)]	$\geq 3 \ \mu g/dL$	≥5 μg/dL
Total	221,644 (100%)	17,543 (17,462, 17,650) ^a	4,457 (4,435, 4,482)
Child sex assigned at birth		(7.9%)	(2.0%)
Female	108,288 (49)	8,293 (7.7)	2,100 (1.9)
Male	113,427 (51)	9,250 (8.2)	2,357 (2.1)
Number of prenatal visits			
<10	50,769 (23)	4,360 (8.6)	1,123 (2.2)
≥ 10	169,340 (76)	13,053 (7.7)	3,297 (1.9)
Missing Insurance at delivery	1,605 (1)	130	36
Medicaid	67,531 (30)	7,515 (11)	1,866 (2.8)
Private	127,485 (57)	8,184 (6.4)	2,086 (1.6)
Self-pay	10,914 (4.9)	935 (8.6)	252 (2.3)
Other	15,914 (7.2)	926 (5.8)	259 (1.6)
Missing	-131 (0.06)		_
Older children			
None	88,376 (40)	7,024 (7.9)	1,821 (2.1)
1	74,719 (34)	5,650 (7.6)	1,419 (1.9)
≥2 	58,684 (26)	4,876 (8.3)	1,218 (2.1)
Missing	-64 (0.03)	-6	-2
Reported smoking before or during pregnancy None	197,150 (89)	14,722 (7.5)	3,728 (1.9)
Any	24,546 (11)	2,818 (11)	728 (3.0)
Missing	19 (0.01)	3	
Maternal age at delivery (y)		0	
≤20	17,658 (8)	2,020 (11)	466 (2.6)
21–25	48,421 (22)	4,610 (9.5)	1,152 (2.4)
26–30	67,987 (31)	5,069 (7.5)	1,321 (1.9)
31–35	59,144 (27)	3,887 (6.6)	1,023 (1.7)
>35	28,503 (13)	1,957 (6.9)	495 (1.7)
Missing	2 (0.00)	1	—
Maternal marital status Married	157,300 (71)	11,098 (7.1)	2,906 (1.8)
Unmarried	64,523 (29)	6,460 (10)	1,552 (2.4)
Missing	-109(0.05)	-15	-2
Maternal education at delivery		10	-
Less than HS	24,568 (11)	2,788 (11)	709 (2.9)
HS graduate	39,749 (18)	3,933 (9.9)	966 (2.4)
Some college or associate's degree	71,777 (32)	5,558 (7.7)	1,360 (1.9)
Bachelor's degree or higher	85,306 (38)	5,239 (6.1)	1,414 (1.7)
Missing	315 (0.14)	24	8
WIC Yes	60,822 (21)	7,279 (10)	1 790 (2 6)
No	69,833 (31) 151,493 (68)	10,230 (6.8)	1,789 (2.6) 2,665 (1.8)
Missing	389 (0.18)	34	3
Maternal Hispanic ethnicity			0
Hispanic	23,313 (11)	1,643 (7.0)	420 (1.8)
Non-Hispanic	198,283 (89)	15,886 (8.0)	4,031 (2.0)
Missing	119 (0.05)	15	5
Maternal race			
American Indian	1,950 (0.9)	224 (11)	47 (2.4)
Asian and Pacific Islander	11,971 (5.4)	1,235 (10)	394 (3.3)
Black White	46,539 (21) 146,066 (66)	4,142 (8.9) 10,854 (7.4)	922 (2.0)
Other non-White	15,189 (6.9)	1,088 (7.2)	2,815 (1.9) 277 (1.8)
Mother's state of birth	13,107 (0.7)	1,000 (7.2)	277 (1.0)
North Carolina	80,992 (37)	7,634 (9.4)	1,869 (2.3)
Other U.S. states and territories	110,180 (50)	7,294 (6.6)	1,868 (1.7)
Remainder of world	32,385 (15)	2,747 (8.5)	757 (2.3)
Missing	-1,843 (0.83)	-132	-37
Year of birth		,	
2011	39,961 (18)	4,629 (12)	1,065 (2.7)
2012	39,457 (18)	3,160 (8.0)	740 (1.9)
2013	40,863 (18)	2,815 (6.9)	709 (1.7)
2014 2015	42,755 (19) 39,895 (18)	3,090 (7.2) 2,788 (7.0)	887 (2.1) 768 (1.9)
2015	18,783 (8.5)	1,061 (5.6)	287 (1.5)

Table 4. (Continued.)

	All additional projected	Estimated number of additional children with EBLL [n (% of row)]	
Characteristics at birth	untested children $-$ [<i>n</i> (% of all untested children)]	≥3 µg/dL	≥5 µg/dL
Environmental characteristics			
Residence in urbanized area	150 115 ((0))	10 (00 (7 1)	2 700 (1 0)
Yes	150,115 (68)	10,600 (7.1)	2,700 (1.8)
No Distance from major roadway	71,599 (32)	6,943 (9.7)	1,757 (2.5)
Within 100 m	7,433 (3.5)	724 (9.7)	182 (2.4)
>100 m	214,282 (97)	16,819 (7.8)	4,275 (2.0)
Estimated 2010 public water system service population size	214,202 (57)	10,017 (7.0)	4,275 (2.0)
<3.300	7,088 (3.2)	725 (10)	183 (2.6)
3,301–10,000	14,624 (6.6)	1,301 (8.9)	314 (2.1)
10,001–50,000	42,647 (19)	3,618 (8.5)	988 (2.3)
>50,000	123,716 (56)	8,607 (7.0)	2,143 (1.7)
Not on public water	33,639 (15)	3,292 (9.8)	828 (2.5)
Within 2 km of TRI or NEI site emitting/releasing lead in past 5 y			
TRI sites	12 (2) ((2)	1 100 (0 7)	200 (2.2)
Low lead High lead	13,636 (6.2)	1,190 (8.7)	309 (2.3)
NEI sites	8,400 (3.8)	810 (9.6)	192 (2.3)
Low lead	28,872 (13)	2,501 (8.7)	653 (2.3)
High lead	23,780 (11)	2,268 (9.5)	544 (2.3)
elected U.S. Census characteristics, categorized in quintiles		_,	2.1 (2.3)
Block group population [Q (%)]			
1	38,041 (17)	3,776 (9.9)	984 (2.6)
2	40,458 (18)	3,504 (8.7)	904 (2.2)
3	43,444 (20)	3,503 (8.1)	882 (2.0)
4	46,959 (21)	3,435 (7.3)	838 (1.8)
5	52,812 (24)	3,324 (6.3)	849 (1.6)
Tract group population born in state [Q (%)]	(2,015,(21))	A = 1 (A = (C = 0))	1 177 (17)
1	68,915 (31) 49,781 (22)	4,164 (6.0) 3,282 (6.6)	1,177 (1.7) 825 (1.7)
2 3	39,148 (18)	3,093 (7.9)	755 (1.9)
4	34,216 (15)	3,629 (11)	855 (2.5)
5	29,659 (13)	3,375 (11)	847 (2.9)
Block group population Asian [Q (%)]		-,-,-()	0.17 (215)
1	31,543 (14)	3,586 (11)	878 (2.8)
2	38,576 (17)	3,553 (9.2)	882 (2.3)
3	45,929 (21)	3,519 (7.7)	911 (2.0)
4	51,771 (23)	3,302 (6.4)	804 (1.6)
	53,897 (24)	3,583 (6.6)	982 (1.8)
Block group population Black [Q (%)]	41,713 (19)	2 119 (9 2)	871 (2.1)
1	49,640 (22)	3,448 (8.3) 3,401 (6.9)	871 (2.1) 918 (1.8)
2 3	49,248 (22)	3,744 (7.6)	934 (1.9)
4	45,485 (22)	3,673 (8.1)	974 (2.1)
5	35,629 (16)	3,276 (9.2)	759 (2.1)
Block group population Hispanic or Latino [Q (%)]	· · · · ·		
1	38,290 (17)	3,505 (9.2)	847 (2.2)
2	47,737 (22)	3,698 (7.7)	988 (2.1)
2 3 4	49,806 (22)	3,732 (7.5)	984 (2.0)
	47,954 (22)	3,697 (7.7)	954 (2.0)
5 Plack group population White $[O(\%)]$	37,930 (17)	2,911 (7.7)	684 (1.8)
Block group population White [Q (%)]	35,415 (16)	3,133 (8.8)	736 (2.1)
2	44,309 (20)	3,719 (8.4)	998 (2.3)
3	49,825 (22)	3,656 (7.3)	941 (1.9)
4	49,842 (22)	3,578 (7.2)	932 (1.9)
5	42,325 (19)	3,458 (8.2)	850 (2.0)
Population >25 years of age who have not completed HS [Q (%)]			. ,
1	63,875 (29)	3,982 (6.2)	1,113 (1.7)
2 3	50,572 (23)	3,544 (7.0)	860 (1.7)
3	40,968 (18)	3,310 (8.1)	834 (2.0)
4	35,566 (16)	3,415 (9.6)	828 (2.3)
5 Block group housing units vacant [Q (%)]	30,740 (14)	3,292 (11)	822 (2.7)
1	57,353 (26)	3,454 (6.0)	878 (1.5)
2	47,191 (21)	3,445 (7.3)	878 (1.3) 832 (1.8)
2 3	42,765 (19)	3,369 (7.9)	858 (2.0)
4	38,256 (17)	3,496 (9.1)	893 (2.3)
5	36,164 (16)	3,780 (10)	996 (2.8)

Characteristics at birth	All additional projected untested children —	Estimated number of additional children with EBLL [n (% of row)]	
	[n (% of all untested children)]	\geq 3 µg/dL	$\geq 5 \ \mu g/dL$
Block group housing units built before 1940 [Q (%)]			
1 (0%)	97,310 (44)	6,316 (6.5)	1,591 (1.6)
2	36,281 (16)	2,639 (7.3)	675 (1.9)
3	31,548 (14)	2,596 (8.2)	613 (1.9)
4	28,607 (13)	2,784 (9.7)	693 (2.4)
5	27,976 (13)	3,208 (11)	885 (3.2)
Block group housing units built before 1950 [Q (%)]			
1	64,973 (29)	4,093 (6.3)	1,031 (1.6)
2	47,318 (21)	3,239 (6.8)	825 (1.7)
3 (Ref)	40,502 (18)	3,102 (7.7)	735 (1.8)
4	34,596 (16)	3,321 (9.6)	848 (2.5)
5	34,333 (15)	3,788 (11)	1,018 (3.0)

Note: —, not applicable; EBLL, elevated blood lead level; HS, high school; NEI, National Emissions Inventory; Q, quintile; Ref, reference; TRI, Toxics Release Inventory; WIC, Special Supplemental Nutrition Program for Women, Infants, and Children. "95% credible interval.

1.35 times the risk of receiving an elevated blood lead test at <30 months of age (95% CI: 1.32, 1.39) compared with children of mothers covered by private insurance at delivery. We estimate that there were 7,515 untested children with mothers on Medicaid with BLLs $\geq 3 \mu g/dL$, or 11% of untested children in this category. However, we estimate that more children (8,184) covered by private

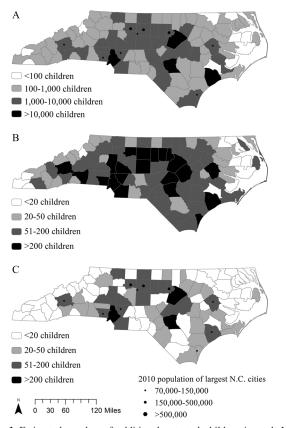


Figure 1. Estimated number of additional untested children in each North Carolina county 2011–2018 (A) total and with (B) blood lead $\geq 3 \ \mu g/dL$ and (C) blood lead $\geq 5 \ \mu g/dL$ at <30 months of age. The number of untested children is the number of children not linked to a blood lead test at <30 months of age. The estimated number of additional children with blood lead $\geq 3 \ \mu g/dL$ and $\geq 5 \ \mu g/dL$ at <30 months of age was calculated by subtracting the true number of children in the cohort with elevated blood lead levels from the IP weighted population (Table S6). This figure was generated in ArcMap (version 10.7; Esri). Note: IP, inverse probability.

insurance with a BLL $\geq 3 \ \mu g/dL$ were never tested. Children covered by private insurance were less likely to have elevated blood lead, but they constituted the majority of untested children.

Discussion

In this analysis, we linked birth certificate and blood lead testing data with publicly available neighborhood and environmental data to evaluate factors at birth related to blood lead testing and BLLs among North Carolina children in 2011–2018. We found that most children born in North Carolina between 2011-mid-2016 were screened for lead and received a blood lead test at least one time by 2.5 years of age. However, tested children did not reflect the demographics of North Carolina. After reweighting the tested children to represent the state as a whole, we identified several individualand neighborhood-level factors at birth that were predictive of elevated blood lead in toddlers, including being covered by Medicaid, smoking during pregnancy, young maternal age, maternal non-Hispanic ethnicity, maternal AAPI race, maternal international location of birth, living near a major roadway or industrial source of lead emissions or releases, lack of access to a public water system, and living in neighborhoods at the time of delivery with more older and vacant housing. As a result of the gaps in lead screening, we estimate that current estimates undercount the number of children in North Carolina with EBLLs by $\sim 30\%$.

Lead Testing

Prior studies of children in North Carolina reported that ~ 50% of children 1 and 2 years of age were tested for lead, an overall finding consistent with our results.²⁹ In the early 1990s, children tested for lead in North Carolina were disproportionately Black and residents of rural counties compared with the state population.⁵⁹

To our knowledge, only a single study has prospectively evaluated predictors of children's lead testing in a population-based cohort.²⁴ Chen et al. linked Pennsylvania birth certificates in 2015– 2016 to children's blood lead surveillance data from 2015–2018.²⁴ Consistent with our findings here, they reported that mothers of children reporting Medicaid at delivery or receipt of WIC benefits during pregnancy had higher odds of receiving a lead test by 12 or 24 months of age, whereas children of AAPI mothers were less likely to receive a lead test. They also reported that individual- and neighborhood-level markers of poverty were associated with increased odds of a BLL $\geq 5 \ \mu g/dL$. However, these analyses did not include potential environmental sources of lead exposure such as roadways, industrial sites, or drinking water source—and estimates of associations with EBLLs relied on complete case analysis and did not account for children who were not tested for lead.

A wealth of prior studies have evaluated associations between individual- or neighborhood-level variables and children's BLLs. However, nearly every study that attempts to predict or explain children's BLLs is restricted to a sample of children that have been tested for lead, with few exceptions.^{24,60,61} Children who are never tested for lead are missing the key outcome—BLL—and are therefore not selected into these studies. Because the predictors of lead testing are not well documented or understood, the structure of this missing data, and the potential for selection bias by conditioning on children with lead testing results, is not known. To our knowledge, no previous study has considered how restricting analyses to children tested for lead could bias associations between sources of lead exposure and children's BLLs and ultimately impact public health policy.

Risk of EBLL

Prior research in North Carolina found that Black children and (independent of race) children living in rural areas were more likely to have EBLLs than White children and children living in urban locations, respectively.⁵⁹ More recently, North Carolina children's BLLs were found to be positively correlated with living in a census block group that had a greater proportion of African American or Hispanic population or population receiving public assistance, warmer seasons of testing (particularly summer and fall), decreasing household median income, and increasing age of housing.^{62,63} Across the country, the year a child's home was built has been among the most consistent predictors of elevated blood lead.⁶⁴ The percentage of housing in a neighborhood built before 1950 has also been consistently and positively associated with both an individual child's BLL and the proportion of children in that neighborhood with elevated blood lead.^{65–71} Living in close proximity to industries with documented releases of lead into the environment, such as TRI sites or airports, has also been associated with elevated blood lead in children. 33,65,70,72

Contrary to findings in other parts of the country, but in accordance with research in North Carolina,⁵⁹ we found that children living in more rural areas in North Carolina are more likely to have EBLLs than those in urbanized parts of the state. However, most of North Carolina's population lives in urbanized areas, and children in these neighborhoods are also currently less likely to be tested for lead. Thus rural areas account for fewer absolute cases (both observed and missed) of children with EBLLs than urbanized areas. It is likely, however, that the risk of lead exposure is not equally distributed among urban residents; North Carolina's cities are among the most economically and racially segregated in the country.^{73–75}

In the United States, exposure to both household and environmental lead hazards is unevenly distributed; children who are Black or who live in a household with low income or low educational attainment are more likely to be exposed to lead.^{76–84} These patterns have been true at an ecologic level as well: Children living in census tracts and block groups with higher proportions of residents living in poverty, vacant homes, or residents who are African American, Latinx, or recently immigrated, have higher BLLs than those in neighborhoods with greater wealth, newer and owner-occupied housing, and more White and native-born residents.^{33,36,63,65–70,85–88} Given North Carolina's history of disproportionately siting toxic waste facilities in lower income communities of color,^{89,90} it is not improbable that, without properly accounting for these additional potential sources of lead exposure, the predictive value of the racial and socioeconomic makeup of a neighborhood may reflect that neighborhood's proximity to sources of environmental lead. When we account for the likelihood of receiving a blood lead test and for environmental sources of lead exposure, we find that children of Black mothers are at no higher risk than children of White mothers to have EBLLs, and for children of Hispanic women, the risk appears to be significantly lower. These findings implicate the important role of environmental injustice in persistent racial disparities in children's BLLs.

Although we evaluated an extensive array of individual- and neighborhood-level predictors of lead testing and BLLs in North Carolina children, there were a few potentially important factors we could not evaluate, including the age of each child's home. Although other analyses have used county tax records to determine the year a child's home was built,^{62,63} this information has not been compiled by the state for most of the property parcels in North Carolina.⁴⁹ Instead, we examined the census block group proportion of older housing in the child's neighborhood. This measure likely captures some of both the effect of a child's housing age on elevated blood lead risk, as well as the risk of living near older houses, which may have deteriorating external lead paint that could impact the exposure risk of both its occupants and their neighbors. We could not examine the presence of a parent or family member with an occupation or hobby with an elevated risk of lead exposure. Although take-home lead exposure from work is a concern for North Carolina children, documented instances of occupational lead exposure are largely concentrated in Forsyth County, North Carolina.³

We used public water system service area maps developed in 2004–2006 to approximate children's water source in our analyses. However, homes that overlap with public maps of water system service areas may not actually be served by these public utilities.⁹¹ Moreover, this misclassification is not random. In Wake County, which contains the second largest population of any North Carolina county, block groups with increasing proportions of Black residents have higher odds of being excluded from public water systems.⁹²

A key intent of this analysis was to identify factors at birth that could predict higher risk of EBLLs in early childhood, as early identification of risk factors could inform prevention efforts. However, there are limits to this approach. If a family moves, the neighborhood characteristics and proximity to sources of lead exposure of a child's address may change. To address this concern, we restricted the study to children living at the same address or in the same city, ZIP code, or county at testing as at birth in sensitivity analyses, with similar results. However, because we do not have information on parental occupation or time the child spent in neighborhoods other than that of their home address, we cannot account for exposures that occurred beyond their home neighborhood, for example, at a child care center, which may result in mismeasurement of lead exposure. We chose to use neighborhood-level measures from 2010 to preserve temporality of predictive variables prior to the outcome of lead testing for all children. However, these data may more accurately describe the neighborhoods of children born earlier in the cohort than those born more recently, whereas contemporary measures may better reflect families' environments and access to testing.

In addition, we noted that children born in 2011 were more likely to have EBLLs than children born in later years. It is not clear why this is the case. There were similar numbers and proportions of children tested in 2011 as in other years. However, children born in this year were among the first to be tested under the most recent CDC reference value for children's BLLs, lowered to $5 \ \mu g/dL$ in 2012.^{93,94} Lowering this threshold expanded the population of children who were required to receive follow up and lead exposure investigations after blood lead testing and could have resulted in more aggressive lead hazard removal and education efforts, improving environmental conditions not just for the children with elevated tests, but also for their communities.

Lead Surveillance Strategy

The CDC reference value for children's blood lead was established based on the estimated distribution of BLLs in U.S. children (~97.5th percentile), rather than a level of safety.⁹⁴ BLLs under the previous reference level of 5 μ g/dL (the level during this study period) are associated with behavioral problems, decreased cognition, and decreased kidney function.¹ Because there is no safe level of lead exposure, any measurable blood lead is too high; the level at which a child's blood lead is elevated enough to require an investigation is a somewhat arbitrary cutoff. However, as Geoffrey Rose wrote, "What is common is all right, we presume."⁹⁵

The current children's lead exposure reduction strategy in the United States follows a "high risk strategy,"95 relying on screening and targeted lead testing. If children who are at higher risk of being exposed to lead can be identified earlier-at birth-preventing such exposure may be more feasible, and public health systems could implement both individual- and population-level policies to reduce children's lead exposure. Screening questionnaires used by providers to decide whether or not to test a child's blood for lead do not reliably predict children's lead exposure.¹⁴ Public health researchers may be able to employ machine learning or complex statistical methods to develop algorithms to better identify the most children at the highest risk of an EBLL.96 However, the relatively small RRs and moderate predictive utility of our model demonstrate that targeting specific subpopulations for lead testing may not be a sufficient strategy for identifying children exposed to lead. Despite a testing approach meant to target the highest-risk children, our results indicate that the nearly two-thirds of children born in North Carolina who are tested for lead at <30 months of age are at only slightly higher risk of elevated blood lead compared with children never tested for lead. Moreover, the absolute number of children with the characteristics at birth associated with the highest relative risks for an EBLL in childhood is smaller than the number of children with relatively low risk characteristics. For example, our model indicates that children covered by Medicaid do have a significantly higher risk of elevated blood lead than children not covered by Medicaid, and targeted lead screening of these children⁹⁷ has been effective in identifying vulnerable children with EBLLs. However, only 42% of North Carolina's children are insured by Medicaid.⁹⁸ We estimate that the absolute number of children in North Carolina with elevated blood lead is greater among those not covered by Medicaid than among those covered by Medicaid. Similarly, we find that children born to mothers under 25 years of age at delivery are both more likely to receive a blood lead test and at increased risk of elevated blood lead at <30 months of age. Once again, however, we estimate that the absolute number of children with BLLs $\geq 3 \,\mu g/dL$, both tested and untested, is higher among children born to mothers >25 years of age, reflecting that targeted blood lead testing to children of younger mothers is insufficient. Moreover, the strata of characteristics at birth that include the greatest numbers of children with EBLLs missed by lead testing are those that are most common in our cohort. For example, we estimate that 14,722 (83%) of these children were born to mothers who did not report smoking during pregnancy. Children born to nonsmoking mothers are also the vast majority (547,981, or 87%) of all children in the cohort and 89% (197,150) of all untested children. Testing for blood lead in all children of nonsmoking mothers could identify the majority of these missed children, but it would no longer be a targeted approach and would fly in the face of evidence that these children are at significantly lower risk of elevated blood lead compared with children of mothers who reported smoking during pregnancy. Rather, our results suggest that encouraging clinicians to test the blood of all children <3 years of age for lead and providing the resources to do so, as well as adopting policies to expand blood lead testing as a universal requirement, rather than testing only high-risk populations, would benefit thousands—"a large number of people at a small risk may give rise to more cases of disease than the small number who are at a high risk."⁹⁵ Such an expansion would align with the North Carolina Division of Public Health's recent move to make free blood lead testing available to all pregnant women at local health departments.⁹⁹

The relatively small proportion of children covered by Medicaid missed by current blood lead surveillance speaks to some success of targeted testing in North Carolina. Universal testing must not come at the cost of reducing the effectiveness of lead testing for this vulnerable population. Children covered by Medicaid in North Carolina are disproportionately Black or Hispanic,¹⁰⁰ and because of historic and continued structural and environmental racism, Black families are more likely to live near industrial sources of lead pollution^{89,90,101} and less likely to have the generational wealth that provides access to high quality housing and private health insurance. Apparent differences in risk of EBLLs by maternal race are reduced or eliminated by accounting for Medicaid status and sources of environmental lead, providing contemporary evidence of these historic patterns. Moreover, lead exposure is intergenerational; lead stored in a mother's bones from her earlier life exposures is remobilized during pregnancy^{102,103} and can be transferred to her fetus in utero and to her child via breastfeeding,^{104,105} contributing to her child's lead exposure. Black children and women have consistently and historically had higher BLLs than their White contemporaries,^{81,106,107} but Black children are less likely to receive follow-up after an initial elevated blood lead test,¹⁰⁸ thus interventions that do not increase screening and reduce lead exposures for Black children and families will result in persistent disparities. If universal children's blood lead testing is not immediately politically or financially feasible, decisions regarding targeting children for testing must consider equity and history so that historic environmental injustices are not prolonged and scarce resources may be devoted to children who are multiply disadvantaged and burdened by lead exposure.

With lead, prevention of exposure is key. Reducing children's BLLs after lead exposure is not effective at improving neurological outcomes, and the neurotoxic effects of lead may be irreversible.²¹ Thus by far, the most effective method of reducing lead exposure has been to implement systemic population-level policies, which has resulted in dramatic declines in children's lead exposure.^{2,3} However, lead continues to be used in some industries,⁵ is extremely stable in soil (which serves as a sink for both historic and contemporary lead deposition),⁶ and lead can contaminate water when lead in service lines, solders, pipes, or fixtures corrodes.¹⁰⁹ Thus lead exposure remains a problem for U.S. children that requires population-level action.4,110,111 Public health workers may use neighborhood-level information to try to predict whether a child will be exposed to lead (e.g., the proportion of housing built before 1950), but the systems in place largely rely on individual-level actions to address lead exposure (e.g., removing chipping lead-based paint from a child's home). A true "population strategy"⁹⁵ to not only reduce, but prevent, children's lead exposure would require both widespread lead testing to identify exposed children and sources of exposure, but also follow-up regulatory action to remove those sources of exposure.

Acknowledgments

This research was supported by the National Institutes of Health/National Institute of Environmental Health Sciences (T32-ES007018 to E.M.K.).

The findings and conclusions in this publication are those of the authors and do not necessarily represent the views of the North Carolina Department of Health and Human Services, Division of Public Health.

References

- National Toxicology Program. 2012. NTP Monograph on Health Effects of Low-Level Lead. Research Triangle Park, NC: Department of Health and Human Services, National Institutes of Health, National Institute of Environmental Health Sciences, National Toxicology Program.
- 2. ATSDR (Agency for Toxic Substances and Disease Registry). 2020. *Toxicological Profile for Lead.* Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- Bellinger DC, Bellinger AM. 2006. Childhood lead poisoning: the torturous path from science to policy. J Clin Invest 116(4):853–857, PMID: 16585952, https://doi.org/10.1172/JCI28232.
- Raymond J, Brown MJ. 2017. Childhood blood lead levels in children aged <5 years—United States, 2009–2014. MMWR Surveill Summ 66(3):1–10, PMID: 28103215, https://doi.org/10.15585/mmwr.ss6603a1.
- Alarcon WA, State Adult Blood Lead Epidemiology and Surveillance Program Investigators. 2016. Elevated blood lead levels among employed adults— United States, 1994–2013. MMWR Morb Mortal Wkly Rep 63(55):59–65, PMID: 27736830, https://doi.org/10.15585/mmwr.mm6355a5.
- ATSDR. 2007. Toxicological Profile for Lead. Atlanta, GA: Agency for Toxic Substances and Disease Registry.
- Lanphear BP. 1998. The paradox of lead poisoning prevention. Science 281(5383):1617–1618, PMID: 9767027, https://doi.org/10.1126/science.281.5383.1617.
- Council on Environmental Health. 2016. Prevention of childhood lead toxicity. Pediatrics 138(1):e20161493, PMID: 27325637, https://doi.org/10.1542/peds. 2016-1493.
- Committee on Practice and Ambulatory Medicine, Bright Futures Perodicity Schedule Workgroup, Hackell JM, Abularrage JJ, Almendarez YM, Boudreau ADA, Berhane AB, et al. 2021. 2021 recommendations for preventive pediatric health care. Pediatrics 147(3):e2020049776, PMID: 33593848, https://doi.org/10. 1542/peds.2020-049776.
- Wachino V. 2016. Coverage of blood lead testing for children enrolled in Medicaid and the Children's Health Insurance Program. CMCS Informational Bulletin. 30 November 2016.Baltimore, MD: U.S. Department of Health and Human Services. https://www.medicaid.gov/federal-policy-guidance/downloads/cib113016.pdf [accessed 1 April 2019].
- Knighton AJ, Payne NR, Speedie S. 2016. Lead testing in a pediatric population: underscreening and problematic repeated tests. J Public Health Manag Pract 22(4):331–337, PMID: 26418307, https://doi.org/10.1097/PHH. 000000000000344.
- Levinson DR. 2010. Most Medicaid Children in Nine States Are Not Receiving All Required Preventive Screening Services. OEI-05-08-00520. May 2010. Washington, DC: U.S. Department of Health and Human Services. https://oig. hhs.gov/oei/reports/oei-05-08-00520.pdf [accessed 1 April 2019].
- Jones RL, Homa DM, Meyer PA, Brody DJ, Caldwell KL, Pirkle JL, et al. 2009. Trends in blood lead levels and blood lead testing among US children aged 1 to 5 years, 1988–2004. Pediatrics 123(3):e376–e385, PMID: 19254973, https://doi.org/ 10.1542/peds.2007-3608.
- Ossiander EM. 2013. A systematic review of screening questionnaires for childhood lead poisoning. J Public Health Manag Pract 19(1):E21–E29, PMID: 22668673, https://doi.org/10.1097/PHH.0b013e3182249523.
- Campbell JR, Schaffer SJ, Szilagyi PG, O'Connor KG, Briss P, Weitzman M. 1996. Blood lead screening practices among US pediatricians. Pediatrics 98(3 pt 1):372–377, PMID: 8784359, https://doi.org/10.1542/peds.98.3.372.
- Ferguson SC, Lieu TA. 1997. Blood lead testing by pediatricians: practice, attitudes, and demographics. Am J Public Health 87(8):1349–1351, PMID: 9279274, https://doi.org/10.2105/ajph.87.8.1349.
- Keeshan B, Avener C, Abramson A, Brennan J, Hill E, MacLean J, et al. 2010. Barriers to pediatric lead screening: implications from a web-based survey of Vermont pediatricians. Clin Pediatr (Phila) 49(7):656–663, PMID: 20150211, https://doi.org/10.1177/0009922809360926.
- CDC (Centers for Disease Control and Prevention). 2021. Recommended Actions Based on Blood Lead Levels. https://www.cdc.gov/nceh/lead/advisory/ acclpp/actions-blls.htm [accessed 1 February 2022].
- CDC. 2021. Blood Lead Reference Value. https://www.cdc.gov/nceh/lead/ data/blood-lead-reference-value.htm [accessed 1 February 2022].
- Bellinger DC. 2012. A strategy for comparing the contributions of environmental chemicals and other risk factors to neurodevelopment of children. Environ Health Perspect 120(4):501–507, PMID: 22182676, https://doi.org/10.1289/ehp. 1104170.
- Dietrich KN, Ware JH, Salganik M, Radcliffe J, Rogan WJ, Rhoads GG, et al. 2004. Effect of chelation therapy on the neuropsychological and behavioral development of lead-exposed children after school entry. Pediatrics 114(1):19–26, PMID: 15231903, https://doi.org/10.1542/peds.114.1.19.
- 22. Wang G, DiBari J, Bind E, Steffens AM, Mukherjee J, Azuine RE, et al. 2019. Association between maternal exposure to lead, maternal folate status, and intergenerational risk of childhood overweight and obesity. JAMA Netw Open

2(10):e1912343, PMID: 31577354, https://doi.org/10.1001/jamanetworkopen.2019. 12343.

- Cole SR, Platt RW, Schisterman EF, Chu H, Westreich D, Richardson D, et al. 2010. Illustrating bias due to conditioning on a collider. Int J Epidemiol 39(2):417–420, PMID: 19926667, https://doi.org/10.1093/ije/dyp334.
- Chen YH, Ma ZQ, Watkins SM. 2021. Effects of individual and neighborhood characteristics on childhood blood lead testing and elevated blood lead levels, a Pennsylvania birth cohort analysis. J Prim Care Community Health 12:21501327211017780, PMID: 34009062, https://doi.org/10.1177/ 21501327211017780.
- U.S. Census Bureau. 2022. U.S. and World Population Clock. https://www. census.gov/popclock/ [accessed 1 February 2022].
- USDA Economic Research Service. 2022. State Data. North Carolina. https:// data.ers.usda.gov/reports.aspx?StateFIPS=37&StateName=North%20Carolina& ID=17854 [accessed 4 February 2022].
- Dolcourt JL, Hamrick HJ, O'Tuama LA, Wooten J, Barker EL Jr. 1978. Increased lead burden in children of battery workers: asymptomatic exposure resulting from contaminated work clothing. Pediatrics 62(4):563–566, PMID: 714588, https://doi.org/10.1542/peds.62.4.563.
- Rinsky JL, Higgins S, Angelon-Gaetz K, Hogan D, Lauffer P, Davies M, et al. 2018. Occupational and take-home lead exposure among lead oxide manufacturing employees, North Carolina, 2016. Public Health Rep 133(6):700–706, PMID: 30231234, https://doi.org/10.1177/0033354918795442.
- Angelon-Gaetz K, Chelminski AN. 2018. Running the numbers: trends in lead poisoning prevention data for children aged < 6 years in North Carolina. N C Med J 79(5):339–342, PMID: 30228146, https://doi.org/10.18043/ncm.79.5.339.
- NCDHHS (North Carolina Department of Health and Human Services). 2018. North Carolina Adult Blood Lead Epidemiology Surveillance (ABLES) Program Summary of Findings for 2017. Raleigh, NC: North Carolina Department of Health and Human Services, Division of Public Health. https://epi.dph.ncdhhs. gov/oee/oii/docs/LeadSummary2017.pdf [accessed 1 April 2019].
- MacDonald Gibson J, Fisher M, Clonch A, MacDonald JM, Cook PJ. 2020. Children drinking private well water have higher blood lead than those with city water. Proc Natl Acad Sci USA 117(29):16898–16907, PMID: 32631989, https://doi.org/10.1073/pnas.2002729117.
- King KE, Darrah TH, Money E, Meentemeyer R, Maguire RL, Nye MD, et al. 2015. Geographic clustering of elevated blood heavy metal levels in pregnant women. BMC Public Health 15(1):1035, PMID: 26449855, https://doi.org/10. 1186/s12889-015-2379-9.
- Miranda ML, Anthopolos R, Hastings D. 2011. A geospatial analysis of the effects of aviation gasoline on childhood blood lead levels. Environ Health Perspect 119(10):1513–1516, PMID: 21749964, https://doi.org/10.1289/ehp.1003231.
- Pieper KJ, Nystrom VE, Parks J, Jennings K, Faircloth H, Morgan JB, et al. 2018. Elevated lead in water of private wells poses health risks: case study in Macon County, North Carolina. Environ Sci Technol 52(7):4350–4357, PMID: 29536726, https://doi.org/10.1021/acs.est.7b05812.
- NCDHHS. 2019. Childhood Lead Poisoning Prevention Program: Data. https:// ehs.ncpublichealth.com/hhccehb/cehu/lead/data.htm [accessed 1 April 2019].
- Moody HA, Darden JT, Pigozzi BW. 2016. The relationship of neighborhood socioeconomic differences and racial residential segregation to childhood blood lead levels in metropolitan Detroit. J Urban Health 93(5):820–839, PMID: 27538746, https://doi.org/10.1007/s11524-016-0071-8.
- CDC. 2013. Guidelines for Measuring Lead in Blood Using Point of Care Instruments. Atlanta, GA: Centers for Disease Control and Prevention, Advisory Committee on Childhood Lead Poisoning Prevention. https://www.cdc.gov/ nceh/lead/ACCLPP/20131024_POCguidelines_final.pdf [accessed 1 April 2019].
- Jones-Vessey KA. 2012. Revisions to the North Carolina birth certificate and their impact on tracking maternal and infant health data. Stat Primer 19:1–16.
- Ingram DD, Parker JD, Schenker, N, Weed JA, Hamilton B, Arias E, et al. 2003. United States Census 2000 Population With Bridged Race Categories. Vital Health Stat 2(135). Washington, DC: U.S. Department of Health and Human Services, National Center for Health Statistics. https://wonder.cdc.gov/ wonder/help/populations/bridged-race/VitalHealthStatistics-Series2No135.pdf [accessed 1 July 2021].
- VanderWeele TJ, Robinson WR. 2014. On the causal interpretation of race in regressions adjusting for confounding and mediating variables. Epidemiology 25(4):473–484, PMID: 24887159, https://doi.org/10.1097/EDE.000000000000105.
- Kaufman JS, Cooper RS. 1999. Seeking causal explanations in social epidemiology. Am J Epidemiol 150(2):113–120, PMID: 10412955, https://doi.org/10.1093/ oxfordjournals.aje.a009969.
- Ford CL, Airhihenbuwa CO. 2010. Critical race theory, race equity, and public health: toward antiracism praxis. Am J Public Health 100(suppl 1):S30–S35, PMID: 20147679, https://doi.org/10.2105/AJPH.2009.171058.
- U.S. Census Bureau. 2021. 2010 Census Urban and Rural Classification and Urban Area Criteria. https://www.census.gov/programs-surveys/geography/

guidance/geo-areas/urban-rural/2010-urban-rural.html [accessed 1 February 2022].

- Datko-Williams L, Wilkie A, Richmond-Bryant J. 2014. Analysis of U.S. soil lead (Pb) studies from 1970 to 2012. Sci Total Environ 468–469:854–863, PMID: 24076506, https://doi.org/10.1016/j.scitotenv.2013.08.089.
- Williams JD. 2014. Metropolitan Area Designations by OMB: History, 2010 Standards, and Uses. Washington, DC: Congressional Research Service. https://crsreports.congress.gov/product/pdf/R/R42005 [accessed 1 July 2021].
- Maupin MAK, Joan F, Hutson SS, Lovelace JK, Barber NL, Linsey KS. 2014. *Estimated Use of Water in the United States in 2010*. Circular 1405. Reston, VA: U.S. Geological Survey. https://pubs.usgs.gov/circ/1405/pdf/circ1405.pdf [accessed 1 July 2021].
- Dieter CA, Linsey KS, Caldwell RR, Harris MA, Ivahnenko TI, Lovelace JK, et al. 2018. Estimated Use of Water in the United States County-Level Data for 2015. Version 2.0, June 2018. Washington, DC: U.S. Geological Survey. https://doi.org/doi.org/10.5066/F7TB15V5 [accessed 1 July 2021].
- MacDonald Gibson J, Pieper KJ. 2017. Strategies to improve private-well water quality: a North Carolina perspective. Environ Health Perspect 125(7):076001, PMID: 28728142, https://doi.org/10.1289/EHP890.
- NC OneMap. 2021. NC OneMap: Geographic Data Serving a Statewide Community [Geospatial Portal]. https://www.nconemap.gov [accessed 1 April 2019].
- NC Center for Geographic Information & Analysis. 2007. Type A Future Public Water Systems. https://www.arcgis.com/sharing/rest/content/items/ 35d70d01c87842f7ba1ddfcb1ba384b0/info/metadata/metadata.xml?format= default&output=html [accessed 1 April 2019].
- U.S. Congress. 1974. Safe Drinking Water Act. Pub L 93-523. 93rd Congress, 3 December 1974.
- NC Department of Transportation. 2020. Connect NCDOT GIS Data Layers. https:// connect.ncdot.gov/resources/gis/Pages/GIS-Data-Layers.aspx [accessed 1 April 2019].
- U.S. EPA (U.S. Environmental Protection Agency). 2018. Toxics Release Inventory (TRI) Program. https://www.epa.gov/toxics-release-inventory-triprogram [accessed 1 April 2019].
- U.S. EPA. 2018. 2014 National Emissions Inventory (NEI) Data. https://www. epa.gov/air-emissions-inventories/2014-national-emissions-inventory-nei-data [accessed 1 February 2019].
- 55. U.S. EPA. 2020. Envirofacts. https://enviro.epa.gov/ [accessed 10/26/2020].
- U.S. EPA. 2020. Air Emissions Inventories, National Emissions Inventory (NEI). https://www.epa.gov/air-emissions-inventories/national-emissions-inventorynei [accessed 10/26/2020].
- Military Onesource. 2021. Military Installations. https://installations.militaryone source.mil/ [accessed 1 February 2022].
- Tippett R. 2019. NC is rapidly growing. Where are our new residents moving from? https://www.ncdemography.org/2019/10/03/nc-is-rapidly-growing-whereare-our-new-residents-moving-from/ [accessed 15 June 2021].
- Norman EH, Bordley WC, Hertz-Picciotto I, Newton DA. 1994. Rural-urban blood lead differences in North Carolina children. Pediatrics 94(1):59–64, PMID: 8008539, https://doi.org/10.1542/peds.94.1.59.
- Kaufmann RB, Clouse TL, Olson DR, Matte TD. 2000. Elevated blood lead levels and blood lead screening among US children aged one to five years: 1988–1994. Pediatrics 106(6):E79, PMID: 11099622, https://doi.org/10.1542/peds. 106.6.e79.
- Kemper AR, Cohn LM, Fant KE, Dombkowski KJ. 2005. Blood lead testing among Medicaid-enrolled children in Michigan. Arch Pediatr Adolesc Med 159(7):646–650, PMID: 15996998, https://doi.org/10.1001/archpedi.159.7.646.
- Kim D, Galeano MAO, Hull A, Miranda ML. 2008. A framework for widespread replication of a highly spatially resolved childhood lead exposure risk model. Environ Health Perspect 116(12):1735–1739, PMID: 19079729, https://doi.org/10. 1289/ehp.11540.
- Miranda ML, Dolinoy DC, Overstreet MA. 2002. Mapping for prevention: GIS models for directing childhood lead poisoning prevention programs. Environ Health Perspect 110(9):947–953, PMID: 12204831, https://doi.org/10.1289/ehp. 02110947.
- 64. Dixon SL, Gaitens JM, Jacobs DE, Strauss W, Nagaraja J, Pivetz T, et al. 2009. Exposure of U.S. children to residential dust lead, 1999–2004: II. The contribution of lead-contaminated dust to children's blood lead levels. Environ Health Perspect 117(3):468–474, PMID: 19337524, https://doi.org/10.1289/ehp. 11918.
- Zahran S, Iverson T, McElmurry SP, Weiler S. 2017. The effect of leaded aviation gasoline on blood lead in children. J Assoc Environ Resour Econ 4(2):575–610, https://doi.org/10.1086/691686.
- Lanphear BP, Byrd RS, Auinger P, Schaffer SJ. 1998. Community characteristics associated with elevated blood lead levels in children. Pediatrics 101(2):264–271, PMID: 9445502, https://doi.org/10.1542/peds.101.2.264.

- Brink LL, Talbott EO, Sharma RK, Marsh GM, Wu WC, Rager JR, et al. 2013. Do US ambient air lead levels have a significant impact on childhood blood lead levels: results of a national study. J Environ Public Health 2013:278042, PMID: 23983719, https://doi.org/10.1155/2013/278042.
- Sargent JD, Brown MJ, Freeman JL, Bailey A, Goodman D, Freeman DH Jr. 1995. Childhood lead poisoning in Massachusetts communities: its association with sociodemographic and housing characteristics. Am J Public Health 85(4):528–534, PMID: 7702117, https://doi.org/10.2105/ajph.85.4.528.
- Sargent JD, Bailey A, Simon P, Blake M, Dalton MA. 1997. Census tract analysis of lead exposure in Rhode Island children. Environ Res 74(2):159–168, PMID: 9339229, https://doi.org/10.1006/enrs.1997.3755.
- Brink LA, Talbott EO, Marsh GM, Sharma R, Benson S, Wu WC, et al. 2016. Revisiting nonresidential environmental exposures and childhood lead poisoning in the US: findings from Kansas, 2000–2005. J Environ Public Health 2016:8791686, PMID: 27042184, https://doi.org/10.1155/2016/8791686.
- Bailey AJ, Sargent JD, Blake MK. 1998. A tale of two counties: childhood lead poisoning, industrialization, and abatement in New England. Econ Geogr 74(suppl 1):96–111, https://doi.org/10.2307/144306.
- Guthe WG, Tucker RK, Murphy EA, England R, Stevenson E, Luckhardt JC. 1992. Reassessment of lead exposure in New Jersey using GIS technology. Environ Res 59(2):318–325, PMID: 1464285, https://doi.org/10.1016/s0013-9351 (05)80038-6.
- Chetty R, Hendren N. 2018. The impacts of neighborhoods on intergenerational mobility II: county-level estimates. Q J Econ 133(3):1163–1228, https://doi.org/10.1093/qje/qjy006.
- Kneebone E. 2014. The Growth and Spread of Concentrated Poverty, 2000 to 2008-2012. Washington, DC: Brookings Institution. https://www.brookings.edu/ interactives/the-growth-and-spread-of-concentratedpoverty-2000-to-2008-2012/#/M10420 [accessed 1 July 2021].
- Groeger LV, W A, Eads D. 2018. *Miseducation: Is There Racial Inequality at Your School*? New York, NY: ProPublica. https://projects.propublica.org/miseducation [accessed 1 July 2021].
- Jacobs DE, Clickner RP, Zhou JY, Viet S, Marker DA, Rogers JW. 2002. The prevalence of lead-based paint hazards in U.S. housing. Environ Health Perspect 110(10):A599–A606, PMID: 12361941, https://doi.org/10.1289/ehp.021100599.
- Dewalt FG, Cox DC, O'Haver R, Salatino B, Holmes D, Ashley PJ, et al. 2015. Prevalence of lead hazards and soil arsenic in U.S. housing. J Environ Health 78(5):22–29, PMID: 26738315.
- Moody H, Grady SC. 2017. Lead emissions and population vulnerability in the Detroit (Michigan, USA) Metropolitan Area, 2006–2013: a spatial and temporal analysis. Int J Environ Res Public Health 14(12):1445, PMID: 29168789, https://doi.org/10.3390/ijerph14121445.
- Apostolou A, Garcia-Esquinas E, Fadrowski JJ, McLain P, Weaver VM, Navas-Acien A. 2012. Secondhand tobacco smoke: a source of lead exposure in US children and adolescents. Am J Public Health 102(4):714–722, PMID: 21852639, https://doi.org/10.2105/AJPH.2011.300161.
- Gaitens JM, Dixon SL, Jacobs DE, Nagaraja J, Strauss W, Wilson JW, et al. 2009. Exposure of U.S. children to residential dust lead, 1999–2004: I. Housing and demographic factors. Environ Health Perspect 117(3):461–467, PMID: 19337523, https://doi.org/10.1289/ehp.11917.
- Lanphear BP, Weitzman M, Eberly S. 1996. Racial differences in urban children's environmental exposures to lead. Am J Public Health 86(10):1460–1463, PMID: 8876521, https://doi.org/10.2105/ajph.86.10.1460.
- Aelion CM, Davis HT, Lawson AB, Cai B, McDermott S. 2013. Associations between soil lead concentrations and populations by race/ethnicity and income-to-poverty ratio in urban and rural areas. Environ Geochem Health 35(1):1–12, PMID: 22752852, https://doi.org/10.1007/s10653-012-9472-0.
- Campanella R, Mielke HW. 2008. Human geography of New Orleans' highlead geochemical setting. Environ Geochem Health 30(6):531–540, PMID: 18563588, https://doi.org/10.1007/s10653-008-9190-9.
- Diawara MM, Litt JS, Unis D, Alfonso N, Martinez L, Crock JG, et al. 2006. Arsenic, cadmium, lead, and mercury in surface soils, Pueblo, Colorado: implications for population health risk. Environ Geochem Health 28(4):297–315, PMID: 16752202, https://doi.org/10.1007/s10653-005-9000-6.
- Haley VB, Talbot TO. 2004. Geographic analysis of blood lead levels in New York State children born 1994–1997. Environ Health Perspect 112(15):1577– 1582, PMID: 15531445, https://doi.org/10.1289/ehp.7053.
- Kaplowitz SA, Perlstadt H, Perlstadt H, Post LA. 2010. Comparing lead poisoning risk assessment methods: census block group characteristics vs. zip codes as predictors. Public Health Rep 125(2):234–245, PMID: 20297750, https://doi.org/10.1177/003335491012500212.
- Krieger N, Chen JT, Waterman PD, Soobader MJ, Subramanian SV, Carson R. 2003. Choosing area based socioeconomic measures to monitor social inequalities in low birth weight and childhood lead poisoning: the Public Health Disparities Geocoding Project (US). J Epidemiol Community Health 57(3):186–199, PMID: 12594195, https://doi.org/10.1136/jech.57.3.186.

- Zhen Z, Shao L, Zhang L. 2018. Spatial hurdle models for predicting the number of children with lead poisoning. Int J Environ Res Public Health 15(9):1792, PMID: 30134510, https://doi.org/10.3390/ijerph15091792.
- Norton JM, Wing S, Lipscomb HJ, Kaufman JS, Marshall SW, Cravey AJ. 2007. Race, wealth, and solid waste facilities in North Carolina. Environ Health Perspect 115(9):1344–1350, PMID: 17805426, https://doi.org/10.1289/ehp.10161.
- Mirabelli MC, Wing S, Marshall SW, Wilcosky TC. 2006. Race, poverty, and potential exposure of middle-school students to air emissions from confined swine feeding operations. Environ Health Perspect 114(4):591–596, PMID: 16581551, https://doi.org/10.1289/ehp.8586.
- Heaney CD, Wing S, Wilson SM, Campbell RL, Caldwell D, Hopkins B, et al. 2013. Public infrastructure disparities and the microbiological and chemical safety of drinking and surface water supplies in a community bordering a landfill. J Environ Health 75(10):24–36, PMID: 23858663.
- MacDonald Gibson J, DeFelice N, Sebastian D, Leker H. 2014. Racial disparities in access to community water supply service in Wake County, North Carolina. Front Public Health Serv Syst Res 3(3):6. https://doi.org/10.13023/ FPHSSR.0303.06.
- 93. Gilbert SG, Weiss B. 2006. A rationale for lowering the blood lead action level from 10 to 2 μ g/dL. Neurotoxicology 27(5):693–701, PMID: 16889836, https://doi.org/10.1016/j.neuro.2006.06.008.
- CDC. 2021. CDC's Childhood Lead Poisoning Prevention Program. https:// www.cdc.gov/nceh/lead/about/program.htm [accessed 1 June 2021].
- Rose G. 1985. Sick individuals and sick populations. Int J Epidemiol 14(1):32– 38, PMID: 3872850, https://doi.org/10.1093/ije/14.1.32.
- Liu X, Taylor MP, Aelion CM, Dong C. 2021. Novel application of machine learning algorithms and model-agnostic methods to identify factors influencing childhood blood lead levels. Environ Sci Technol 55(19):13387–13399, PMID: 34546733, https://doi.org/10.1021/acs.est.1c01097.
- Wachino V. 2016. Coverage of Blood Lead Testing for Children Enrolled in Medicaid and the Children's Health Insurance Program. https://www.medicaid.gov/federalpolicy-guidance/downloads/cib113016.pdf [accessed 1 February 2022].
- Center for Children & Families Children's Health Care Report Card. 2021. Children's Health Coverage in North Carolina. https://kidshealthcarereport. ccf.georgetown.edu/states/north-carolina [accessed 16 September 2021].
- North Carolina Childhood Lead Poisoning Prevention Program. 2019. NC Childhood Lead Testing and Follow-Up Manual. Raleigh, NC: North Carolina Childhood Lead Poisoning Prevention Program.
- 100. Miles DR, Sexton CM, Margolis LH, Sanderson M. 2010. Children's Health Care Coverage and Children's Health 2007–2009: A Report from the North

Carolina Child Health Assessment and Monitoring Program. Raleigh, NC: North Carolina Department of Health and Human Services. https://schs.dph.ncdhhs.gov/SCHS/pdf/CHAMP_Health_Care_Report_2007-09.pdf [accessed 1 April 2019].

- Banzhaf S, Ma L, Timmins C. 2019. Environmental justice: the economics of race, place, and pollution. J Econ Perspect 33(1):185–208, PMID: 30707005, https://doi.org/10.1257/jep.33.1.185.
- 102. Gulson BL, Mizon KJ, Korsch MJ, Palmer JM, Donnelly JB. 2003. Mobilization of lead from human bone tissue during pregnancy and lactation—a summary of long-term research. Sci Total Environ 303(1–2):79–104, PMID: 12568766, https://doi.org/10.1016/S0048-9697(02)00355-8.
- 103. Gulson B, Mizon K, Korsch M, Taylor A. 2016. Revisiting mobilisation of skeletal lead during pregnancy based on monthly sampling and cord/maternal blood lead relationships confirm placental transfer of lead. Arch Toxicol 90(4):805–816, PMID: 25877328, https://doi.org/10.1007/s00204-015-1515-8.
- 104. Ettinger AS, Téllez-Rojo MM, Amarasiriwardena C, Peterson KE, Schwartz J, Aro A, et al. 2006. Influence of maternal bone lead burden and calcium intake on levels of lead in breast milk over the course of lactation. Am J Epidemiol 163(1):48–56, PMID: 16282237, https://doi.org/10.1093/aje/kwj010.
- 105. Ettinger AS, Roy A, Amarasiriwardena CJ, Smith D, Lupoli N, Mercado-García A, et al. 2014. Maternal blood, plasma, and breast milk lead: lactational transfer and contribution to infant exposure. Environ Health Perspect 122(1):87–92, PMID: 24184948, https://doi.org/10.1289/ehp.1307187.
- Geronimus AT, Hillemeier MM. 1992. Patterns of blood lead levels in US black and white women of childbearing age. Ethn Dis 2(3):222–231, PMID: 1467759.
- Meyer PA, Pivetz T, Dignam TA, Homa DM, Schoonover J, Brody D. 2003. Surveillance for elevated blood lead levels among children—United States, 1997–2001. MMWR Surveill Summ 52(10):1–21, PMID: 14532866.
- Kemper AR, Cohn LM, Fant KE, Dombkowski KJ, Hudson SR. 2005. Follow-up testing among children with elevated screening blood lead levels. JAMA 293(18):2232–2237, PMID: 15886378, https://doi.org/10.1001/jama.293.18.2232.
- Miranda ML, Kim D, Hull AP, Paul CJ, Galeano MAO. 2007. Changes in blood lead levels associated with use of chloramines in water treatment systems. Environ Health Perspect 115(2):221–225, PMID: 17384768, https://doi.org/10. 1289/ehp.9432.
- 110. Lanphear B. 2017. Still treating lead poisoning after all these years. Pediatrics 140(2):e20171400, PMID: 28771415, https://doi.org/10.1542/peds.2017-1400.
- Lanphear BP. 2005. Childhood lead poisoning prevention: too little, too late. JAMA 293(18):2274–2276, PMID: 15886384, https://doi.org/10.1001/jama.293. 18.2274.