Environmental lead exposure during early childhood

Bruce P. Lanphear, MD, MPH, Richard Hornung, PhD, Mona Ho, MS, Cynthia R. Howard, MD, MPH, Shirley Eberle, MS, and Karen Knauf, BS

Objective: To determine the relative contribution of residential lead hazards to children's blood lead concentrations during early childhood.

Methods: We enrolled children 6 months of age and followed them until 24 months of age. Blood and samples of dust, soil, water and paint were analyzed for lead at 6-month intervals, and interviews were conducted to estimate nutritional, behavioral, and demographic factors linked with lead exposure.

Results: Of the 276 children enrolled, 249 (90%) were followed until 24 months of age. The geometric mean blood lead concentration of children at 6 months of age was 2.9 μg/dL (95% CI, 2.7-3.1). At 24 months of age, children's mean blood lead was 7.5 μg/dL; 82 (33%) had a blood lead level of ≥10 μg/dL. In adjusted analyses, lead-contaminated floor dust, soil, and water contributed to children's lead intake throughout the first 2 years of life ($P < .05$). Lead-contaminated dust from window troughs was a source of lead exposure, especially in the second year of life. Dietary iron intake, but not calcium intake, was inversely associated with blood lead levels ($P < .05$). Blood lead concentration was over 50% higher in black than in white children ($P = .0001$).

Conclusion: Lead-contaminated house dust is the major source of lead intake during early childhood. Black children remain at increased risk for higher blood lead concentration after adjusting for environmental lead exposures and dietary intake. (J Pediatr 2002;140:40-7)

Despite a dramatic decline in blood lead concentration, many children have blood lead levels consistent with subclinical lead toxicity.[1] Moreover, blood lead concentrations below 10 μg/dL, the current value considered acceptable, have been linked with cognitive deficits in children and the adverse consequences of lead exposure persist into early adulthood.[2-6] Thus, although the prevalence of children with undue lead exposure is decreasing, the importance of prevention is increasing as the consequences of lower blood lead concentrations are recognized.

The major environmental sources of children's lead exposure have been identified, but questions remain. Ingestion of lead-contaminated dust and soil through normal mouthing behaviors is the predominant mechanism of exposure in early childhood.[7-11] Children living in older, deteriorated housing or housing undergoing renovation are at risk for undue lead exposure.[10] The relative contribution of these sources, however, is poorly defined.[11] Because dietary calcium and iron intake have been inversely associated with lead absorption in observational studies or experimental models,[12-22] some scientists have recommended increasing dietary calcium and iron intake to reduce lead absorption in children[23-25]; however, there is no evidence from randomized, controlled trials that higher dietary intake of calcium and iron reduces blood lead in children.[26] Furthermore, there are no human data simultaneously measuring environmental lead exposure and nutritional intake. Finally, the racial disparity in children's blood lead levels, recognized over 3 decades ago, are poorly understood.[1,27-28]

The purpose of this study was to estimate the contribution of residential lead hazards to children's blood lead concentration, adjusted for dietary iron and calcium intake, and to examine whether racial differences in blood lead concentration persist after adjusting for dietary intake of iron and calcium and for environmental exposures.

Methods

Children in this study were participants in a randomized, controlled trial of dust control reported elsewhere.[29] Children were eligible for the study if they lived in the city of Rochester, New York, denied plans to relocate within

From the General and Community Pediatrics Department, Children's Hospital Medical Center, Cincinnati, Institute for Health Policy and Health Services Research, Cincinnati, Ohio, and the Departments of Pediatrics and Biostatistics, University of Rochester School of Medicine and Dentistry, New York.

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Reprint requests: Bruce P. Lanphear, MD, MPH, Children's Hospital Medical Center, 3333 Burnet Avenue, Cincinnati, OH 45229.

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the next 3 months, and were 5 to 7 months of age at the baseline visit. At least 6 attempts to were made to contact each potential patient's parents by telephone. Once a family was deemed eligible and agreed to participate, a study team visited the home, obtained informed consent and a blood sample from the infant, conducted an interview, and collected environmental samples. The Internal Review Board of the University of Rochester School of Medicine approved the study.

Home visits were made to families at baseline (6 months) and at 12, 18, and 24 months. During each of the 4 home visits, a trained interviewer conducted a face–to–face survey with the primary caretaker to assess risk factors for lead exposure, including mouthing behaviors (eg, soil ingestion, paint chip ingestion), time spent outdoors, and attainment of developmental milestones. Blood was taken at each visit by a certified phlebotomist and measured for lead by using Electrothermal Atomization Atomic Absorption Spectrometry (Wadsworth Laboratories, Albany, NY). All results are the means of 6 separate analyses (3 aliquots/day measured on 2 consecutive days) performed on each blood sample. The detection limit for lead in blood was 1.0 μg/dL.

An environmental technician systematically conducted dust sampling at each visit to characterize children's exposure to lead-contaminated dust.[30] Three to 4 composite interior dust wipe samples were taken from surfaces that were accessible to a child (ie, carpeted floors, noncarpeted floors, and window sills) or known to be heavily contaminated with lead (window troughs) in the child's bedroom, the kitchen, and the living room. A composite dust sample consisted of a maximum of 3 wipe samples collected from the same surface (ie, carpeted floor, noncarpeted floor, interior window sill, or window troughs).

The condition and lead content of paint and lead content of soil and water samples were measured by an environmental technician at baseline and when a child moved to a new residence. Lead content of paint was measured by using a portable radiographic fluorescence analyzer (Microlead I, Warrington, Austin, Tex). The condition of painted surfaces was evaluated by visual inspection.[31] Three soil samples were taken on each side of the house near the perimeter of the foundation where bare soil was present. These samples were combined for a single composite foundation sample. Parents collected a water sample (250 mL) in the morning from the kitchen tap after the water flowed for 1 minute.

Dust samples were analyzed first by flame atomic absorption, followed by graphite furnace if levels were below 5.0 μg per sample. The detection limit of graphite furnace for the dust wipe was 0.5 μg per sample. Soil was analyzed separately with flame atomic absorption spectroscopy; the detection limit for lead in soil was 25 μg/g. Water was analyzed by using atomic absorption, with a detection limit of 5 μg/L.

A trained interviewer assessed children's nutritional intake at each visit. Respondents were questioned about the content of the child's diet using a food frequency checklist[32] that was modified to reflect the dietary content of children in the study population. Nutritional contributions to infant's diets from breastmilk were calculated based on published data.[33] The breastmilk intake of 6-month-old infants who were not exclusively breast-fed was estimated from the literature to be 769 mL per day.[34] To verify the proportion of the diet composed by breastmilk in nonexclusively breast-fed infants, surveys were individually reviewed.[35] Contributions from formula were calculated by ounces of formula (brand and type) consumed daily at 6 and 12 months.[36]

The distributions of continuous variables were examined to determine whether particular variables should be log-transformed. For statistical analyses, blood lead concentration was log-transformed. Floor dust lead values were

Table I. Baseline characteristics of children and their families in the Rochester Longitudinal Lead Cohort

Characteristic	n (%)
Race	
Black	166 (60)
White	54 (20)
Latino	29 (10.5)
Asian	4 (1.4)
Native American	1 (0.4)
Other or unknown	22 (8.0)
Housing condition	
Poor	50 (19)
Good	220 (81)
Household income	
<$15,500	194 (71)
≥$15,500	78 (29)
Rental housing	235 (86)
Marital status	
Single	161 (58)
Married	71 (26)
Single, living together	26 (9)
Divorced, separated or widowed	18 (7)

Table II. Blood lead concentration, dietary intake (95% CI), environmental lead (95% CI) and behaviors by age for children enrolled in the Rochester Longitudinal Lead Study

	Age of patients			
Variable	6 mo	12 mo	18 mo	24 mo
Geometric mean blood lead (μg/dL)*	2.9 (2.7, 3.1)	5.7 (5.3, 6.2)	6.1 (5.6, 6.6)	7.5 (7.0, 8.2)
Blood lead ≥ 10 μg/dL (%)	1.4	16.9	22.7	33.3
Iron intake (mg/d)	20.9(19.7-22.1)	13.4(12.4-14.4)	10.1(9.5-10.7)	9.9(9.3-10.5)
Calcium intake (mg/d)	679 (646-712)	987 (940-1034)	968 (913-1023)	836 (788-884)
Vitamin D intake (IU/d)	374 (353-395)	355 (336-374)	283 (263-303)	250 (228-272)
Vitamin C intake (mg/d)	94 (88-100)	94 (87-101)	97 (88-106)	95 (88-102)
Floor lead (μg/ft^2)	12.6 (10.9-14.3)	7.6 (6.5-8.7)	7.8 (6.5-9.0)	8.4 (7.0-9.7)
Sill lead (μg/ft^2)	2422 (873-3971)	936 (427-1444)	633 (405-861)	733 (294-1172)
Trough lead (μg/ft^2)	70,662 (58,581 -82,743)	14,477 (11,002-17,952)	13,356 (10,425-16,287)	14,473 (10,752-18,194)
Soil lead (μg/g)	1712 (1470-1954)	1536 (1295-1777)	1583 (1340-1826)	1635 (1379-1890)
Behaviors, n (%)				
Formula use	253 (92)	114 (43)	8 (3)	2 (0.8)
Finger-sucking	266 (97)	190 (72)	159 (63)	95 (38)
Sitting	202 (73)	263 (99)	254 (100)	246 (99)
Crawling	103 (37)	261 (99)	253 (99)	246 (99)
Standing	110 (40)	259 (98)	253 (99)	246 (99)
Walking	1 (0.4)	206 (78)	247 (97)	246 (99)
Soil ingestion	9 (3)	79 (30)	79 (31)	53 (21)
Time spent outdoors†, n (SD)	2.4 (1.9)	1.8 (1.7)	1.9 (1.9)	1.8 (1.6)
Paint chip ingestion	1 (0.4)	27 (10)	23 (9)	12 (5)
Mouthing window sill	8 (3)	84 (32)	89 (35)	71 (28)
Breast-feeding	49 (18)	23 (9)	12 (5)	1 (0.4)

*Blood lead is presented as geometric mean. All other values are arithmetic mean.
†Time spent outdoors is hours per day.

highly skewed. Because the most extreme floor dust values exerted a high degree of influence on regression coefficients, we chose to truncate extreme floor dust lead values (>98.5th percentile) rather than log transform data. Although both carpeted and noncarpeted floor dust lead loading values were significant predictors of children's blood lead concentrations, we combined the floor samples to form a single floor dust lead variable for the purpose of statistical analysis. A paint lead index variable was created by multiplying the paint condition (good, 1; average, 2; poor, 3) by the paint lead measurement of all measurements taken in the home. Because only a small proportion of water samples had lead concentration above the detection limit, water lead was dichotomized as above or below the detection limit.

For the unadjusted analysis, we defined high exposure as ≥3 of 4 visits with exposure in the highest tercile exposure and low exposure as ≥3 of 4 visits with exposure in the lowest tercile for exposure (Table III). All other exposures were defined as moderate. For the adjusted analyses, the increase in children's blood lead concentration for continuous variables were presented by estimating the increase in blood lead levels from the 5th to the 95th percentile in exposure. For dichotomous variables, the data were presented as the increase in blood lead for children with exposures in the lower versus the higher exposure category.

Multiple regression models, using repeated measures analysis were developed to predict geometric mean blood lead concentration. Repeated measures analysis is a mixed-model regression method that accounts for the correlation among outcomes measured on the same child over time. The participants in this study were considered to be random effects, whereas the intervention, environmental lead exposure variables, nutritional intake, and mouthing behaviors were considered as fixed effects. The PROC MIXED procedure in SAS (SAS Institute, Cary, NC) was used to fit the repeated measures analysis. Numerous interactions were examined, including age of child and dietary intake of calcium and iron with environmental

lead exposures and race. This cohort was established to test the effect of dust control on children's blood lead concentrations.[29] The intervention group variable was forced into the model during the selection process.

RESULTS

Of 276 children enrolled at 6 months of age, 249 children (90%) completed the study (Table I). The geometric mean blood lead of children at 6 months was 2.9 μg/dL (95% CI, 2.7-3.1) (Table II). By 24 months, the mean blood lead level was 7.5 μg/dL (95% CI, 7.0-8.2); 82 (33%) had a blood lead level of ≥ 10 μg/dL, 32 (13%) had a blood lead level ≥15 μg/dL, and 14 (6%) had a blood lead level ≥20 μg/dL.

The proportion of children who were reported to stand upright, walk, mouth interior windowsills, or put paint chips or soil into their mouths increased with age (Table II). In contrast, the proportion of children who were reported to suck their thumb or fingers, or who ingested formula, declined. Of mothers who reported feeding their children formula, 89% reported using tap water to prepare formula at 6 and 12 months. As described earlier,[25] the decline in dust lead loading observed for window sills and window troughs was probably an artifact of sampling (ie, the act of sampling lead-contaminated house dust was equivalent to cleaning).

Lead-contaminated floor dust was a major source of lead exposure (Table III). Of children who were consistently exposed to the highest tercile of lead-contaminated floor dust, 65.3% had blood lead concentrations that exceeded 10 μg/dL compared with 5.3% of children who were exposed to the lowest tercile ($P < .01$). Of children who were exposed to the highest tercile of lead-contaminated soil, 53% had blood lead levels that exceeded 10 μg/dL compared with 8% of children in the lowest tercile ($P < .01$).

Table III. Unadjusted contribution of environmental lead exposure, behaviors, and race to blood lead concentration at 24 months of age

Variable	n	Geometric mean blood lead (μg/dL)	% Blood lead ≥ μg/dL
Race			
Black	150	9.6	47.3
Other	47	5.5	17.0
White	49	4.8	6.1
Floor lead			
High*	49	11.7	65.3
Moderate	178	7.3	27.5
Low	19	3.3	5.3
Interior sill lead			
High*	53	9.2	49.1
Moderate	165	7.7	30.9
Low	28	4.5	17.9
Window trough lead			
High*	50	9.1	48.0
Moderate	169	7.6	31.4
Low	27	5.2	18.5
Soil lead			
High*	64	9.8	53.1
Moderate	144	7.5	31.2
Low	38	4.9	7.9
Water lead†			
> 5 ppb‡	65	8.4	36.9
Undetectable	181	7.2	32.0
Rental housing			
Always	196	8.1	35.7
Sometimes	39	8.8	53.8
Never	40	5.0	15.4
Paint chips in mouth			
Yes	46	9.1	43.5
No	200	7.2	31.0
Soil ingestion			
Yes	133	7.9	35.3
No	113	7.1	31.0

ppb, Parts per billion.
*High exposure was defined as ≥ 3 of 4 visits with exposure in the highest tercile and low exposure was defined as ≥ 3 of 4 visits with exposure in the lowest tercile. All other exposures were considered moderate. For rental housing we defined exposure as "Always" (for all 4 home visits), "Sometimes" (1, 2, or 3 home visits), or "Never".
†Not significant in univariate comparisons.
‡>5 ppb of lead in water or "yes" response for paint chip or soil ingestion indicates positive response for >1 of 4 visits.

Multivariable Analysis

Lead-contaminated floor dust was associated with blood lead concentration throughout the first 2 years of life ($P < .001$) (Table IV). An increase from the 5th to 95th percentile in dust lead levels was associated with a 36% (1.8 μg/dL) rise in blood lead concentration. Lead-contaminated soil also was associated with blood lead concentration

Table IV. Change in blood lead concentration (μg/dL) for sources of environmental lead, behaviors, dietary iron intake, and demographic characteristics from 6 to 24 months of age

		Change in blood lead concentration				
Variables	Estimate (SE)	5th percentile	95th percentile	Percent change	Absolute change (μg/dL)	*P* value
Floor lead (μg/ft^2)	0.0147 (0.0034)	0.8	30.7	36.0	1.8	< .001
Soil lead (ppm)	0.0022 (0.001)	0.18	46.6	10.8	0.54	.040
Water lead (>5 ppb)	0.185 (0.052)			20.4	1.02	< .001
Spent time outdoors (Y vs N)	0.76 (0.033)			7.9	0.40	.021
Soil ingestion (Y vs N)	0.146 (0.036)			15.7	0.79	< .001
Intervention (Y vs N)	–0.073 (0.049)			–7.0	–0.35	.14
Variables modified by age						
Iron intake (mg/d)	0.012 (0.0039)					.003
6 mo		5.6	39.4	–22.2	–1.11	
12 mo		4.9	31.0	–0.8	–0.40	
18 mo		4.4	18.1	2.3	0.12	
24 mo		5.0	20.0	8.0	0.40	
Rental housing	–0.318 (0.089)					.002
6 mo				–7.0	–0.35	
12 mo				15.4	0.77	
18 mo				31.8	1.59	
24 mo				44.4	2.22	
Ingest paint chips	-0.560 (0.107)					.024
6 mo				40.2	2.01	
12 mo				17.1	0.86	
18 mo				5.4	0.27	
24 mo				–2.2	–0.11	
Trough lead (μg/ft^2)	0.018 (0.0073)					.009
6 mo		0.034	23.20	–12.5	–0.63	
12 mo		0.005	4.69	3.2	0.16	
18 mo		0.005	4.41	6.4	0.32	
24 mo		0.002	6.52	13.3	0.67	
Black race	0.1842 (0.067)					< .001
6 mo				26.0	1.30	
12 mo				43.1	2.16	
18 mo				54.3	2.72	
24 mo				62.6	3.13	

Change in blood lead concentration for an increase in continuous exposures from 5th to 95th percentile was based on a blood lead concentration of 5 μg/dL.

(*P* = .04). An increase from the 5th to 95th percentile in soil lead levels was associated with a 10.8% (0.54 μg/dL) rise in blood lead concentration. Children who were reported to ingest soil had blood lead levels that were 15.7% (0.8 μg/dL) higher than children who were reported not to ingest soil (*P* < .001). Children who were reported to spend time outdoors had blood lead concentrations that were 7.9% (0.4 μg/dL) higher than children who did not spend time outdoors. Water lead concentration also was directly associated with blood lead concentration (*P* < .001). Children who lived in housing with water lead concentration >5 parts per billion had blood lead concentrations that were 20.4% (1.0 μg/dL) higher than children who had water lead levels below 5 parts per billion.

Several risk factors showed a significant interaction with age, indicating that the effect on blood lead concentration was modified by a child's age (Table IV). Lead-contaminated window trough dust was associated with blood lead concentration, but only during the second year of life. An increase from the 5th to the 95th percentile in dust lead levels in window troughs was associated with a 13.3% (0.67 μg/dL) rise in blood lead concentration at 24 months, but a lower rise for younger children.

Children who lived in rental housing were at increased risk for higher blood lead concentration. At 6 months, children who resided in rental housing had blood lead concentration that was 7.0% (0.4 μg/dL) lower than those living in owner-occupied housing. By 24 months, however, children who lived in rental housing had blood lead concentrations that were 44.4% (2.2 μg/dL) higher than children who lived in owner-occupied housing (Table IV). Thirty three percent of rental housing was in poor condition compared with 13% of owner-occupied housing (P = .01).

The mean blood lead concentrations of 6-month-old infants who were reported to ingest paint chips was 40.2% (2.0 μg/dL) higher than infants who did not put paint chips in their mouths. By 24 months, the effect of paint chip ingestion on children's blood lead concentration diminished.

Black race remained a predictor of blood lead concentration after adjusting for behaviors, demographic variables, environmental exposures, and dietary intake (Figure). At 6 months, black children's blood lead concentration was 26% (1.3 μg/dL) higher than white children's, but the difference was modified by age. By 24 months, black children's blood lead concentration was 62.6% (3.1 μg/dL) higher than white children's blood lead concentration after adjustment for other risk factors.

Iron intake was inversely associated with blood lead concentration (P = .003). The effect of iron intake was stronger in early infancy. For 6-month-old infants, an increase from the 5th to 95th percentile in iron intake was associated with a 22% (1.11 μg/dL) decrease in blood lead concentration. Calcium intake was not associated with blood lead concentration. Other interactions, including calcium and iron intake with lead-contaminated dust, race, and soil ingestion were not statistically significant. Calcium intake was only marginally significant when we lagged calcium intake by one visit with lead-contaminated floor dust (P = .061).

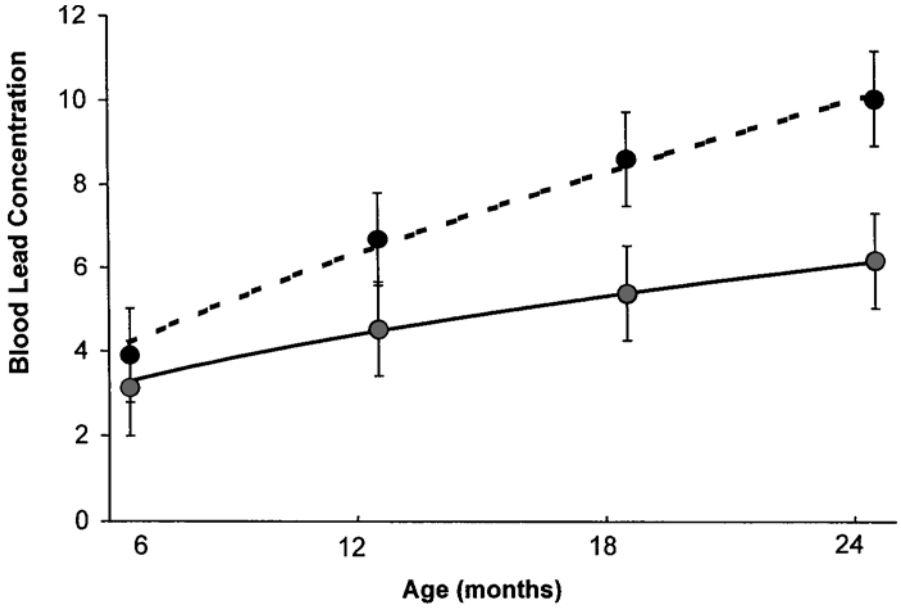

Figure. Geometric mean blood lead concentration (μg/dL) from 6 to 24 months of age, by children's race. Black children are identified by the *hatched lines* and *dark circles*; white children are identified by the *straight line* and *gray circles*.

DISCUSSION

Children's blood lead concentrations are tied to specific environmental sources and behaviors that change during early childhood. In this study, lead-contaminated floor dust was a major pathway or source of lead intake that persisted throughout early childhood. In contrast, exposure to lead-contaminated window troughs became an increasingly important source of lead as children became mobile and stood upright. Iron intake was associated with lead intake during infancy, defined as the first year of life, when dietary intake was the predominant exposure to the environment.

Lead-contaminated dust from floors and window troughs is a recognized source of lead intake in childhood.[8-12,37,38] We confirmed that lead-contaminated floor dust contributes to children's lead intake at levels considerably lower than EPA's recently promulgated standard of 40 μg/ft^2.[8,10,11] We also found that lead-contaminated dust in window troughs is a source of lead intake, casting doubt on the United States Environmental Protection Agency's decision to omit window troughs sampling from their residential lead standards.

Children who lived in rental housing were also at risk for higher blood lead concentration. Rental housing is often in poor condition. Screening rental housing for lead hazards may be a cost-effective strategy for the primary prevention of lead exposure.

Lead absorption mimics calcium and iron absorption in the gastrointestinal tract,[12-22] leading to recommendations to increase dietary calcium intake to reduce lead absorption.[23-25] However, we did not find an inverse relation of dietary calcium intake and blood lead levels, and calcium supplementation has not been proven to reduce children's blood lead levels.[26] The protective effect of calcium may be limited to the simultaneous ingestion of calcium- and lead-containing formula or to children with inadequate dietary calcium intake.

Dietary iron intake was inversely associated with blood lead concentrations, especially during the first year of life. Data indicate that iron deficiency and

low dietary iron intake are both associated with increased lead absorption.[20,22] The proportion of children in this study with insufficient dietary iron intake, as estimated from semiquantitative food frequency, was high. Still, it is unclear whether iron supplementation will result in lower absorption of lead.

We showed previously that environmental exposures contribute to the racial disparity in blood lead levels.[28] In the current study, black children were at higher risk for elevated blood lead levels even after adjusting for environmental exposures, behaviors, socioeconomic status, and dietary intake. Racial disparity in blood lead concentration may be due to more efficient calcium absorption and, by mimicry, lead absorption among black children.[39]

There are limitations of this study. Nutritional surveys in children are susceptible to day–to–day variability.[32] Dietary intake in this study was similar for children of comparable ages surveyed in National Health and Nutrition Examination Survey III, but iron and calcium intake measured with the semiquantitative food frequency survey was not correlated with a 3-day diary for a random sample of 26 18-month-old children. Thus, the results of our nutritional survey should be interpreted cautiously. Other behavioral measures, such as mouthing behaviors, were also based on parental report.

Early childhood is a susceptible age for environmental lead exposure; blood lead concentration typically peaks between 18 to 30 months.[8,10,29] We found that specific behaviors and exposures during early childhood were associated with an increased risk of elevated blood lead concentrations. These findings should guide the development of standards to protect children from residential lead hazards. To protect children from the subclinical lead toxicity, research is needed to prove that existing environmental and nutritional interventions are both safe and effective, especially for black children who live in older, rental housing.[40]

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