HHS Public Access

Author manuscript

J Public Health Manag Pract. Author manuscript; available in PMC 2019 December 17.

Published in final edited form as:

J Public Health Manag Pract. 2019 ; 25(1): 53–61. doi:10.1097/PHH.0000000000000711.

Assessment of Child Lead Exposure in a Philadelphia Community, 2014

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Abstract

Introduction—Several urban neighborhoods in Philadelphia, Pennsylvania have a history of soil, household lead paint and potential lead-emitting industry contamination.

Objectives—1) Describe blood lead levels (BLLs) in target neighborhoods; 2) identify risk factors and sources of lead exposure; 3) describe household environmental lead levels; and 4) compare results with existing data.

Methods—A simple, random, cross-sectional sampling strategy was used to enroll children ≤ 8 years living in selected Philadelphia neighborhoods with a history of lead-emitting industry during July 2014. Geometric mean of child BLLs and prevalence of BLLs ≥ 5 μg/dL were calculated. Linear and logistic regression analyses were used to ascertain risk factors for elevated BLLs.

Results—Among 104 children tested for blood lead, 13 (12.4%, 95% CI, 7.5–20.2%) had BLLs ≥ 5 μg/dL. The geometric mean BLL was 2.0 μg/dL [95% CI, 1.7–2.3μg/dL]). Higher geometric mean BLLs were significantly associated with front door entryway dust lead content, residence built prior to 1900, and a child currently or ever receiving Medicaid. Seventy-one percent of households exceeded the screening level for soil, 25% had an elevated front door floor-dust lead level, 28% had an elevated child play area floor-dust lead level and 14% had an elevated interior window-dust lead level. Children in households with 2–3 elevated environmental lead samples were more likely to have BLLs ≥5 μg/dL. A spatial relationship between household proximity to historic lead-emitting facilities and child lead sampling results was not identified.

Conclusion—Entryway floor dust lead levels were strongly associated with blood lead levels in participants. Reduction of child lead exposure is crucial and continued blood lead surveillance,

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The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention/the Agency for Toxic Substances and Disease Registry or the Environmental Protection Agency.

testing and inspection of homes of children with BLLs ≥5 μg/dL to identify and control lead sources is recommended. Pediatric health care providers should be especially vigilant in screening Medicaid-eligible/enrolled children and children living in very old housing or near legacy lead sites.

Introduction

In children, lead decreases intelligence, growth and hearing; causes anemia; and can cause attention and behavior problems.[1,2] Young children are particularly susceptible to lead poisoning because they absorb more lead from their environments than adults and because their central nervous systems are still developing.[3] For children under 6 years of age, the Centers for Disease Control and Prevention (CDC) has defined an elevated BLL as ≥ 5 micrograms per deciliter (μg/dL), but there is evidence for subtle effects at even lower levels.[4] Sources of lead exposure include lead-based paint, soil, leaded gasoline, industrial emissions, cottage industries (e.g. informal battery recycling), lead soldered cans and water pipes, lead glazed ceramics, and traditional medicines.4

Several urban neighborhoods in Philadelphia, Pennsylvania, have a history of soil lead contamination due, in part, to lead-emitting industry, lead-based paint, and the legacy of leaded gasoline emissions. The point source of interest for this study is the John T. Lewis site (a.k.a., Anzon facility), which operated in the Kensington community of Philadelphia (Figure 1 supplemental). Lead paint was produced at the site from 1849 to 1996.

During July 2014, CDC, Agency for Toxic Substances and Disease Registry (ATSDR), U.S. Environmental Protection Agency (EPA) Region 3, and City of Philadelphia Department of Public Health (PDPH) conducted a study in Philadelphia. The target communities comprised ZIP code 19125 and portions of 19122, 19123, 19133, 19134 and 19148 (Figure 1 supplemental). Objectives of this representative population-based survey were: 1) describe child BLLs in the target communities; 2) identify risk factors and sources of lead exposure among children; 3) describe environmental lead levels among enrolled households; and 4) compare study findings with existing data.

Methods

Study Design

This 2014 study included a simple random sample used to select and enroll a target of 111 households with children ≤ 8 years of age, many living within a 0.8 km (0.5 miles) radius surrounding a legacy point source (the area most affected by historic emissions), personal and household risk factor questionnaires were administered to children's parent or guardian, and environmental sampling from enrolled households. The study population included children who lived at the same Philadelphia address on average, 2 days/ week for at least the prior 8 months. The study protocol received approval from CDC and PDPH institutional review boards. OMB Control no.: 0920–0008.

Using PDPH prepared tax assessor data, addresses were randomly selected from the full roster of residential addresses as starting points for each data collection team. Data

collection teams noted the outcome of each household visit (i.e., eligible, ineligible, refused, vacant). A household (i.e., an area with at least 1 bedroom, 1 bathroom, and a kitchen) with a specified address including apartment number, was defined as the sampling unit.

The sample size required for the blood lead survey was approximately 167 children or an estimated 111 households. Based on PDPH surveillance data and previous studies, we estimated 4% of children would have a BLL ≥ 5 μg/dL. Sample sizes were calculated to provide a margin of error around the geometric mean and prevalence estimate (95% CI ± 2.6%).

Blood Lead Survey

Trained study team members (i.e., pediatric phlebotomists) collected venous blood samples at home among children enrolled in the study. Venous blood specimens were analyzed for lead content 24–48 hours after collection by the Philadelphia Public Health Laboratory using Perkin Elmer's Atomic Absorption Analyzer (PerkinElmer Norwalk, Connecticut; www.perkinelmer.com). The lower limit of detection for blood lead was 0.1 μg/dL. One blood lead venous specimen was rejected because of insufficient quantity.

Health Education

An informational sheet was given to inform consenting parents/guardians who to contact if they have questions, concerns or problems. They also received a folder of healthy housing educational information provided by EPA and PDPH, including documents about lead poisoning prevention in the home and environment.

Environmental sampling and analyses

Enrolled households were offered environmental sampling (soil, water, and interior dust). Soil sampling consisted of collection of one composite exterior soil sample from five soil areas in the residential yard where resident children ≤ 8 years were said to play.[5] A soil lead hazard for play areas is defined as bare soil with lead ≥ 400 parts per million (ppm).[6] A 5-mL grab water sample was collected from the tap used for drinking/cooking. An unacceptable water lead level was defined as at or above the EPA action level, 0.015 ppm or 15 μg/L.[7] Two composite dust wipe samples were collected; one from the floor in the area where the resident child reportedly played; and one from the entryway of the house. An elevated dust lead floor measurement was defined as 40 μg/ft^2.[8] A third composite dust wipe sample was measured and collected from the bedroom interior window sill(s) of resident children. An elevated dust window measurement was defined as 250 μg/ft^2.[8]

The laboratory (Bureau Veritas North America, Novi, MI Laboratory) performed lead analysis of soil, dust wipes, and drinking water samples utilizing Inductively Coupled Plasma (ICP), ICP/Mass Spectrometry and atomic absorption/cold vapor instrumentation to determine contaminate concentrations in ppm. Methods used were SW-6010 (soil and dust),[9] OSHA ID 125-G (lead dust wipes),[10] and EPA 200.8 (lead in drinking water).[11] Bureau Veritas is accredited by the National Environmental Laboratory Accreditation Program.

Results to study participants

Child BLL results were provided to parent or legal guardian within 1 week from sample collection. Confirmatory venous testing was conducted for children with a BLL of 10 μg/dL or more based on CDC guidelines.[12] Environmental sampling results with an explanation of findings were malied to parents or legal guardians within 5 months from sample collection date.

Comparison with Existing Data

To determine percent of children in the study with a previous blood lead test, we matched enrolled child data to historic PDPH blood lead surveillance records. Surveillance records were identified by matching enrolled child's name, gender, birthdate and address to records in the PDPH blood lead surveillance database. For children enrolled in the survey but for whom a venous blood sample was not obtained, historical blood lead surveillance records were abstracted from PDPH for descriptive analysis, but not included in modeling analyses.

We compared BLLs of children in this study with existing PDPH BLL surveillance reports from the same neighborhoods and neighborhoods with similar socioeconomic characteristics (based on ZIP code). Similarly, we extracted historic PDPH home environmental inspection data from households having a child with a BLL ≥ 20 μg/dL (pre-2013 action level) and in households having a child with a BLL ≥ 10 μg/dL (2013-present action level) including lead dust wipes collected both pre- and post-lead abatement activities to compare with study interior dust results. We report highest result by sampling location (i.e., floor, windowsill and window well) by household.

Statistical Analysis

Data were entered into Epi Info (Epi Info version 7.2.0.1, CDC, Atlanta, Georgia), and 100% of records were reentered to confirm accuracy of data entry. Data were analyzed using SAS version 9.3 (SAS Institute Inc., Cary, North Carolina) and SUDAAN version 11.0.0 (Research Triangle Institute, Research Triangle Park, North Carolina) software.

Descriptive statistics were used to assess household and child characteristics. Linear regression techniques were used to examine risk factors for elevated BLLs among children with venous blood collection (n=104) obtained from household and child questionnaires and environmental sampling. BLL concentrations were natural log-transformed for linear regression analyses. Geometric mean and ratio of geometric mean estimates were later back-transformed. Risk factors are described in Tables 1–2 supplemental. The year each residence was built and structure type were abstracted from the Philadelphia Office of Property Assessment (http://property.phila.gov/). Based on a previous study, age of child was selected as a potential confounding variable.[13]

Bi-variable analyses were conducted to assess each risk factor's association with elevated BLLs. Risk factors significantly associated ($P < .05$) with elevated BLLs were evaluated in multivariable analyses. The first multivariable analysis assessed each risk factor along with selected confounding variable (age of child) and the assessment of potential interaction between age and the main effect. Statistically significant risk factors (at the $P < .10$ level)

identified in the first multivariable analysis were included in a second multivariable analysis. During the second multivariable analysis, we used a forward-selection strategy to add 1 risk factor variable at a time to the most predictive model, until all risk factors in the model were statistically significant ($P < .05$). Interactions between risk factors and the confounding variable were assessed. Variance inflation factors were used to assess collinearity between variables in predictive models.

Multivariable logistic regression techniques were employed to examine environmental lead sampling risk factors for BLLs ≥ 5 μg/dL among children with a venous blood collection (n=104). Risk factors considered were environmental sampling results and the child's age. Additionally, an indicator variable (range: 0–4) that counted number of environmental samples with lead levels equal to or above HUD and EPA standards in each household was used to predict BLLs ≥ 5 μg/dL among children.

Geographic analysis

Eckel and colleagues[14] identified 12-suspected lead-emitting facilities in the study area. We compared the spatial relationship among 12 point sources with data collected from enrolled children and their respective households. We used ArcGIS (ESRI 2011. ArcGIS Desktop: Release 10. Redlands, CA) for mapping and analysis. Global Moran's I was used to test for overall spatial clustering of values without locating any clusters themselves. For blood, soil and dust samples, SaTScan™ v8.0[15] was used to assess statistically significant ($P < .05$) geographic clusters of blood lead values ≥ 5 μg/dL and environmental sampling lead values above HUD and EPA standards.

Results

A total of 122 households and 163 children ≤ 8 years were enrolled. Of 5,111 households visited, 4,458 (87.2%) were ineligible for participation. Of the remaining 653 households, 593 were eligible for enrollment. The response rate was 20.6% (n=122). The refusal rate was 40.3% (n=239). Residents of the remaining 232 homes could not be reached during the study period after at least three visits. Three children from 3 households were excluded from the analyses because insufficient questionnaire data were obtained. The analytic dataset comprised 119 households and 160 children (Figure 2 supplemental).

Child characteristics

The average age of children was 3.6 years. Fifty-eight (36.2%) children were 12–35 months of age, and among those with available gender information, 79 (49.4%) were female; most children (n = 133; 83.1%) were born in Philadelphia (Table 1 supplemental). The most commonly reported racial groups were black or African American (n = 79; 49.4%) and white (n = 33; 20.6%). Twenty-two children (13.8%) were reported to have asthma (as told by a health care provider) and most (n = 140; 87.5%) reported up-to-date vaccinations.

Household Characteristics

The median occupancy was four persons per household (range, 2–18). The majority of homes were built before 1950 (n = 108, 90.8%). Among the 119 households, 69 (58.0%) of

families owned the residence and 111 (93.3%) households were a 2- or 3-story masonry row house. Most households used municipal water for drinking and cooking (n = 104; 87.4%). The mean distance the residence was to the point source of interest was 4,096 feet (0.8 miles or 1.3 kilometers) (Table 2 supplemental).

Blood lead results

Among the 160 children, 13 (8.1%) had no evidence of a previous blood lead test. Among the 104 children tested for blood lead in their household, their geometric mean BLL was 2.0 μg/dL [95% CI, 1.7–2.3]) and 13 (12.4%, 95% CI, 7.5–20.2%) had BLLs ≥ 5 μg/dL (2 who had BLLs ≥ 10 μg/dL). Ninety-one (87.5%) of these 104 children had a previous blood lead test on average 30.6 months prior to the study blood lead test. Among 42 children who did not have a venous blood lead sample collected as part of this study but whose BLL results were abstracted from historical surveillance data, none had BLLs ≥ 5 μg/dL.

Environmental characteristics

Complete (i.e., all five) environmental lead sampling results were collected for 58.8% of households. Among the 119 households, 116 (97.5%) had a tap water assessment, 98 (82.4%) had a dust front door floor assessment, 94 (79.0%) had a dust window assessment, 71 (59.7%) had a dust child play area floor assessment, and 70 (58.8%) had soil assessment (Table 3). No households had lead water levels above the EPA action level for drinking water, 50 (71.4%) households exceeded the screening level for soil, 24 (24.5%) households had an elevated front door floor-dust lead level (Figure 3 supplemental), 20 (28.2%) households had an elevated child play area floor-dust lead level and 13 (13.8%) households had an elevated window-dust lead level.

Risk Factors

Twenty-five households (21.0%) were remodeled during the previous 6 months; 19 (15.9%) had a resident who was a current smoker; 23 (19.3%) had a resident with an occupation involving lead; and 15 (12.6%) had a resident with a lead-related hobby (Table 2 supplemental).

In analyses adjusting for child's age, the log of the child's BLL was independently significantly associated with several variables (Table 3 supplemental). Higher geometric mean BLLs were found among children living in households with deteriorating interior and exterior paint and with recent home renovation. However, these results did not remain in multivariate analysis.

In the final model, the log of the child's BLL was significantly associated with dust front door lead content ≥ 40 μg/ft^2 (P = .0027), home built before 1900 (P = .0017), and child receipt of government medical insurance (Medicaid) (P = .0149) (Table 2). A collinearity assessment did not identify significant correlations between variables in any of the models.

The proportion of children with a BLL ≥5 μg/dL was significantly higher among those with elevated lead in interior floor dust by the entryway (Table 3). The age-adjusted odds of a BLL ≥5 μg/dL were 4.5 times higher (95% CI = 1.2, 16.6) for those with elevated floor dust

by the front door compared to those living in households without elevated lead in their environmental sample. Households with any two environmental samples with elevated lead content had a 4.1 times higher odds (95% CI = 1.2, 14.0, $P = .0256$) of having a BLL ≥5 μg/dL. Households with any 3 environmental samples with elevated lead content had 6.5 times higher odds (95% CI = 1.4, 29.5, $P = .0150$) of having a BLL ≥5 μg/dL (Table 4).

Spatial Analysis

Blood lead, soil and dust lead results were spatially distributed across the entire study area without a clear focus. Results of Moran's I for each of the sample types (blood, dust and soil) indicated only a slight clustering effect. Among blood, dust and soil samples collected during the survey, SaTScan results reported no statistically significant spatial clusters.

Comparison to existing data

During 2014, based on PDPH child blood lead surveillance data[16], the percent of children with BLLs ≥ 5 μg/dL was lower (range 4.3–12.8%) in all but one of the six study ZIP codes compared to children tested in our study (12.4% with BLLs ≥ 5 μg/dL). The current published U.S. geometric mean estimate from the National Health and Nutrition Examination Survey (NHANES) (among children 1–5 years of age) is 1.3 μg/dL (95% CI, 1.3–1.4).[17] During 2007–2014, the national estimate of percentage of children 1–5 years of age with BLLs ≥5 μg/dL is 1.9%.[18]

We extracted 3600 dust wipe results from 295 households in study ZIP codes. Environmental inspection records were collected between July 21, 2005 and March 4, 2014. Compared with PDPH environmental inspection sampling results we found higher lead exceedances on floor dust wipe (24.5% floor front door and 28.2% floor dust child play area vs 7.4% inspection data) and window sill (13.8% vs. 5.1%, $P = .0005$). Mean lead levels from the highest sample result by sample location within household were comparable to study floor results (66.2 μg/ft^2 floor front door and 49.3 μg/ft^2 floor dust child play area vs. 47.8 μg/ft^2) but differed by window sill results (396.4 μg/ft^2 vs. 215.7 μg/ft^2 $P = .0359$)

Discussion

In Philadelphia, all children should be tested for lead at ages 12 months and 24 months or at 36–72 months if there is not proof of prior screening.[19] In this population-based study, we found 90% of enrolled children were tested at least once and also had high (87.5%) self-reported immunization, indicating robust outreach by PDPH and pediatric health care providers serving these neighborhoods. Given the age of Philadelphia's housing stock, Philadelphia pediatric health care providers should continue routine testing of all children for blood lead particularly low income, Medicaid-eligible and Medicaid-enrolled children.

In the majority of study neighborhoods, a higher proportion of children with BLLs ≥5 μg/dL (12.4%) were observed compared to citywide Philadelphia child blood lead surveillance data (range: 4.3–12.8%) The geometric mean BLL among study children ≤ 8 years (2.0 μg/dL [95% CI, 1.7–2.3]) is higher than the most recent published U.S. estimate (among children < 6 years of age) (1.3 μg/dL [95% CI, 1.3–1.4]).

Three factors in age-adjusted multivariable analyses predicted an association with higher geometric mean BLLs among children: floor dust (front door entryway) lead content ≥ 40 μg/ft^2; residence built prior to 1900; and a child currently or ever receiving government medical insurance (i.e., Medicaid).

Lead from all environmental sources (e.g., dust, soil) contribute to a child's total lead exposure. Often, these exposures co-occur, making it difficult to identify and quantify the individual contribution of each lead source to a child's total lead burden. Our data suggest the most important indicator of lead exposure for these children was lead dust at the entryway. Interior dust lead is a well-documented predictor of elevated child BLLs. Paint chips[20] and deteriorated paint from inside the residence directly contaminate house dust. Entryway dust is an integrated measure of dust contributed from both interior and exterior lead sources. Child ingestion of lead contaminated dust occurs in several ways, including hand-to-mouth behavior, ingesting contaminated food, and mouthing objects contaminated with lead dust. Sixty-four percent of study children were observed by their parent/guardian to eat or mouth non-food items.

Living in a residence built prior to 1900 was associated with higher geometric mean BLLs among children. Age of housing as a predictor for child BLLs is well understood. The age of a home where a child resides is a risk factor for high BLLs used by child health care providers to target blood lead testing. Several studies have demonstrated that children residing in pre-1950 housing puts them at risk for having BLLs ≥ 10 μg/dL.[21,22,23] About 92 percent of all lead in paint is contained in housing built prior to 1950.[24] Homes built before 1940 typically have higher concentrations of lead in paint, ranging 10–50%.[25] The Federal government in 1978 banned residential lead-based paint.[26]

Children from households currently or ever receiving government medical insurance (i.e., Medicaid) had higher geometric mean BLLs than those who did not receive it. Current or previous receipt of Medicaid is a proxy for low-income households. These households are likely older, poorly maintained and frequently contain lead-based paint hazards.[27] Numerous reports have described the relationship between low-income housing and elevated BLLs.[28,29,30,31,]

Environmental lead levels were above current regulatory standards in a large proportion of survey households. Compared to children with 0–1 elevated environmental lead levels children in households with 2–3 elevated samples were more likely to have BLLs ≥5 μg/dL. This s underscores the importance of efforts to make housing lead safe by addressing all lead hazards in and around the home.

We were able to compare study interior dust lead results to historic PDPH inspection data back to 2005. We found that although highest dust lead levels were comparable, study floor dust lead levels were on average, more than 3 times higher and window dust lead levels were almost twice as high compared to historic investigations. This may be indicative of the higher concentration of old houses in our sample versus other Philadelphia neighborhoods, the contribution of legacy lead sites or it may be due to variations in environmental sampling and analytic techniques between study and PDPH inspections. Further study may elucidate

reasons for the discrepancy (e.g., comparison of similarly collected environmental lead samples from other neighborhoods with similar housing stock).

Our study had limitations. First, participation in our survey was 63% of the target. We had many refusals (40%) due to the transitional nature of several study neighborhoods (e.g., vacancy during renovation, gentrification, lack of parental interest in joining study, challenges with address identification). Second, we were not able to assess possible differences between children who did and did not participate in the study. Third, several of our reference databases were not directly comparable to study data. For example, city environmental inspection data included households of children with BLLs ≥ 10 or ≥ 20 (we had only 2 such children), city environmental inspection data included both pre- and post-clearance dust sample results, some of which may have been collected after cleaning resulting in bias. Fourth, we potentially observed an upward bias of child BLLs due to summertime sampling. Blood lead levels in children tend to be higher during summer months, a situation that may be related to differential seasonal distribution of household lead dust as well as higher child exposure to outdoor dust/soil associated with increased outdoor activity.[32,33] Fifth, in our multivariable predictive analyses, there were three factors that predicted higher child geometric mean BLLs. These results should not be interpreted as clinically significant because higher geometric mean BLLs do not coincide with recommended BLLs for the management of children with BLLs ≥ 5 μg/dL (http://www.phila.gov/health/pdfs/GuideforClinicians7_8_13.pdf). However, BLLs < 5 μg/dL are not without consequences for children and are an important part of children's medical information.

Nevertheless, this was a comprehensive, randomly sampled survey that included a face-to-face survey, venous BLL testing, environmental lead sampling, and visual housing inspection. We compared our findings to several other data sources, which strengthened assessment of community lead risk factors.

Implications for Policy & Practice

We are unable to quantify the contribution of any specific source of lead to a child's blood lead level. Nonetheless, we did find higher than anticipated soil and interior dust lead results. We are also found that in this community, where many sosurces including old housing, deteriorating lead paint, unsafe housing renovation and legacy lead sites combined with poverty, results inblood lead levels were significantly higher than those of the nation at large. We also found that within a house a higher number of lead hazards were strongly associated with the risk for BLLs ≥5 μg/dL. Both these findings support efforts to address lead hazards holistically both at the household and neighborhood level.

Our finding that all household water lead level results were below EPA action level should be intreted with caustion. Collecting standing water samples following the EpA protocol was beyond our capacity. Our water samples were collected during the study visit and provide a snapshot of the water lead levels during a child's waking hours.

Most children enrolled in our study were previously tested for blood lead, indicating robust outreach by PDPH and pediatric health care providers and widespread acceptance of blood

lead testing by parents and guardians. Continued child blood lead surveillance, blood lead testing and case investigations are recommended in the study neighborhoods. Although BLLs for low-income children have decreased substantially on a national level, in Philadelphia, pediatric health care providers should conitnue vigilant screening of low income and Medicaid-enrolled children, children living in very old housing. PDPH should ensure that health care providers and parents are aware of legagcy lead sites in Philadelphia neighborhoods in order that children living by these sites are screened appropriately and work with federal agencies and others to clean the contaminated sites.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

We would like to acknowledge the following persons for their contributions in planning and conducting this study. Without all of their effort, this endeavor would not have been possible.

Kevin Chatham Stevens, Rebecca D Merrill, Oluwatosin Olaiya, Palak Raval-Nelson, Paulette Smith, Jennifer Van Skiver, Martha Stefaniak, Kimberly Brinker, Natasha Bagwe, Jonathan Cruz, Kerry Buchs, and Weston Solution Inc. staff.

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Table 1:

Household environmental lead sampling results, Philadelphia, 2014 (N=119)

Environmental Sample Type	Number of HHs Sampled	Min/Max	Mean	Median	Number of HHs Exceeding Elevated Lead Level (%)	Elevated Lead Level Threshold
Soil Composite	70	40 – 7,700 ppm (or mcg/g)	760.6 ppm (or mcg/g)	595.0 ppm (or mcg/g)	50 (71.4%)	400 ppm (or µg/g)
Water	116	< 1.0 – 3.9 mcg/L	N/A	N/A	0	15 mcg/L
Dust Floor (Front Door)	98	5.2 – 2,322.6 µg/ft^2	66.2 µg/ft^2	17.7 µg/ft^2	24 (24.5%)	40 µg/ft^2
Dust Floor (Child Play Area)	71	5.0 – 631.7 µg/ft^2	49.3 µg/ft^2	13.9 µg/ft^2	20 (28.2%)	40 µg/ft^2
Dust Interior Window Sill (Child Room)	94	1.6 – 17,999.9 µg/ft^2	396.4 µg/ft^2	31.2 µg/ft^2	13 (13.8%)	250 µg/ft^2

HH = Household

Table 2:

Multi-variable linear regression, age-adjusted estimates of the association between log of blood lead level and other study variables, Philadelphia, 2014 (N=104)

Exposure variable	Geometric Mean BLL (95% CI)	Beta (SE)	Ratio of geometric means (95% CI)	*p* Value
Age, years (continuous)	1.95 (1.67, 2.32)	0.02 (0.05)	N/A	0.6162
Dust floor (front door) lead content		0.56 (0.18)	1.75 (1.22, 2.51)	0.0027
≥ 40 μg/ft^2	2.89 (1.16, 2.16)			
< 40 μg/ft^2	1.65 (1.11, 1.35)			
Year built		0.42 (0.16)	1.52 (1.09, 2.10)	0.0117
<1900	2.69 (2.10, 3.46)			
≥1900	1.79 (1.46, 2.18)			
Child currently or ever received government medical insurance (Medicaid)		0.39 (0.16)	1.48 (1.08, 2.20)	0.0149
Yes	2.41 (2.08, 2.83)			
No	1.63 (1.26, 2.14)			

*Least Squares Mean (Conditional Marginal)

Table 3:

Median, 25th and 75th percentile blood lead level and age-adjusted odds ratio of blood lead levels ≥ 5 μg/dL by environmental lead sampling result, Philadelphia, 2014 (N=104)

Environmental Sample Type	N (%)	Median BLL (μg/dL) *(IQR)*	BLL ≥ 5 μg/dL *Odds Ratio (95% CI)*
Dust floor (front door) lead content			
Elevated (>40 μg/ft^2)	25 (24.0)	3.1 (1.9, 5.3)	4.5 (1.2, 16.6)
Not Elevated (≤40 μg/ft^2)	63 (60.6)	1.7 (1.1, 2.9)	ref
Not collected	16 (15.4)	2.1 (1.1, 3.5)	NA
Yard soil lead content			
Elevated (>400 ppm)	38 (36.5)	2.9 (1.5, 4.4)	5.3 (0.6, 47.0)
Not Elevated (≤ 400 ppm)	22 (21.2)	1.8 (1.0, 2.4)	ref
Not collected	44 (42.3)	2.2 (1.2, 3.5)	NA
Dust in play area lead content			
Elevated (>40 μg/ft^2)	23 (22.1)	2.9 (1.6, 4.8)	1.4 (0.4, 5.3)
Not Elevated (≤40 μg/ft^2)	45 (43.3)	2.5 (1.5, 4.3)	ref
Not collected	36 (34.6)	1.5 (1.0, 2.4)	NA
Dust on window (child room) lead content			
Elevated (>250 μg/ft^2)	14 (13.5)	3.9 (2.3, 4.8)	1.7 (0.4, 7.7)
Not Elevated (≤250 μg/ft^2)	67 (64.4)	1.9 (1.1, 3.2)	ref
Not collected	23 (22.1)	1.9 (1.2, 2.9)	NA
Tap water			
Elevated (≥15 μg/L)	0 (0)	NA	
Not Elevated (<15 μg/L)	101 (97.1)	2.1 (1.2, 3.6)	NA
Not collected	3 (2.9)	1.8 (0.3, 4.7)	

Table 4:

Median, 25th and 75th percentile blood lead level and age-adjusted odds ratio of blood lead levels ≥ 5 μg/dL by number of elevated environmental lead sampling results, Philadelphia, 2014 (N=104)

Number of Environmental Sample Results	N (%)	Median BLL (μg/dL) *(IQR)*	BLL ≥ 5 μg/dL *Odds Ratio (95% CI)*	P value
One or more elevated lead sample types				
Zero	39	1.8 (1.0, 2.9)	ref	
Yes	61	2.4 (1.5, 4.3)	3.8 (0.8, 18.5)	0.0990
Not collected	4	2.5 (1.8, 3.5)	NA	
Two or more elevated lead sample types				
Zero or one	74	1.8 (1.1, 2.9)	ref	
Yes	26	3.3 (1.7, 5.3)	4.1 (1.2, 14.0)	0.0256
Not collected	4	2.5 (1.8, 3.5)		
Three or more elevated lead sample types				
Two or less	90	1.9 (1.1, 3.2)	ref	0.0150
Yes	10	4.4 (3.4, 6.2)	6.5 (1.4, 29.5)	
Not collected	4	2.5 (1.8, 3.5)		

One-sided Cochran-Armitage trend test P=0.007