# Risk for Elevated Blood Lead Levels in 3- and 4-Year-Old Children

Jaime S. Raymond · Roberta Anderson · Mark Feingold · David Homa · Mary Jean Brown

**Abstract** *Objective* We conducted a study to evaluate the relation between environmental, demographic, and medical risk factors and late-onset childhood lead poisoning, defined as children who were poisoned at age 3 or older. *Methods* We performed a retrospective case–control study of 262 children whose test results showed non-elevated (<10 µg/dl) blood lead levels (BLLs) before age 2 but levels ≥10 µg/dl after age 3 and of 300 control children who had non-elevated BLL test results before age 2 and also after age 3. The target population was children receiving care at MetroHealth Medical Center in Cleveland, OH. We modeled the association between demographic and clinical risk factors and BLLs ≥10 µg/dl after age 3. Covariates considered were race, gender, body mass index, immunization status, mean corpuscular volume, hematocrit, red cell volume distribution width (RDW), red blood count, hemoglobin, baseline BLL result, and time between the baseline and second BLL test. *Results* Case children were more likely to be male ($P < 0.0001$), black ($P = 0.0189$) and to have a high RDW defined as ≥14.5 % ($P = 0.0083$). On the basis of the final model, children with BLLs 7–9 µg/dl before age 2 and again after a follow-up BLL test (<21 months) were more likely to become lead poisoned than were control children. The risk of black children developing lead poisoning increased over time, regardless of the child's first BLL test result. *Conclusions* Although national recommendations are to test children's blood lead levels at ages 1 and 2, children living in high-risk areas with such risk factors should have a blood lead test at these older ages.

**Keywords** Childhood lead poisoning · Late onset lead poisoning · Case control study · Clinical risk factors

## Introduction

The neurotoxicity of lead in children is well recognized. Permanent neurological damage and behavioral disorders as a consequence of lead exposure have been observed in multiple studies, although a precise dose-response relation to children's age has not been established [1–4]. National recommendations call for high-risk children (e.g., Medicaid enrollees) to be tested for elevated blood lead levels (BLL) at ages 1 and 2 years, and if they were not tested at those ages, to be tested between ages 3 and 5 years [5–7]. Children's BLLs peak at age 18 months through 4 years, the age when hand to mouth activity is most common [8]. No national or other established recommendations recommend routinely retesting children older than age 2 years if they had a BLL <10 µg/dl on one or more earlier screenings.

The Centers for Disease Control and Prevention (CDC) guidelines for screening young children for lead poisoning call for targeted lead screening policies based on assessments of local data [6]. The national definition of a city at

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high-risk for lead poisoning is one in which ≥27% of housing was built before 1950 [6]. In Cleveland, Ohio, 85% of the housing stock was built before 1950. In 2000, approximately 40% of the children in Cleveland were living at or below the poverty level, and 56.5% were receiving Medicaid [7, 9]. High rates of children living in poverty or receiving Medicaid are also recognized risk indicators for lead poisoning [9–11]. Universal screening of 1- and 2-year-olds is recommended for areas where ≥12% of children have BLLs ≥10 μg/dl [7]. In Cleveland, an average of 11% of 1- through 5-year-olds tested had BLLs ≥10 μg/dl, and in some neighborhoods, more than 20% of the children tested had elevated BLLs in 2004 [12]. Thus, with its old housing stock, high rate of children living in poverty, and high prevalence of elevated BLLs, Cleveland can be defined as a city in which children are at higher than average risk of having elevated BLLs. According to the CDC, 12.8% of the children in the United States from birth to six years old were screened for lead and 1.76% had confirmed elevated BLLs [13]. Our data indicate that the risk of a child developing an elevated BLL in Cleveland extends beyond age 2 years and that 3- and 4-year-olds develop lead poisoning in substantial numbers. We undertook this study to describe factors that may put children ≥2 years of age at risk for lead poisoning. We hypothesized that children who develop elevated BLLs at 3 years of age and older have certain identifiable characteristics in common. Determining these risk factors may assist in reducing lead poisoning in this target population.

## Methods and Materials

### Subjects, Study Design, and Protocol

A retrospective case–control study was designed to examine the relation between environmental, demographic, and medical risk factors and late-onset childhood lead poisoning, defined as children who were poisoned at age 3 or older. Case subjects were 262 children ages 3–4 years living within the city of Cleveland, Ohio, who were diagnosed between 2001 and 2004 as having newly identified elevated BLLs. All case children were diagnosed or treated at the MetroHealth Medical Center, a county-owned hospital situated within Cleveland. A case was defined as a child having had a venous BLL < 10 μg/dl at age 1 or age 2 years followed by an elevated BLL (≥10 μg/dl) at age 3 years. Three hundred control children were randomly selected on the basis of their having being seen for routine ambulatory care at the same medical center and their living within the city of Cleveland. The control children had a non-elevated BLL by age two, same as the case children, and were tested again between their third and fourth

birthday resulting in another non-elevated BLL. All case children and control children had both their early and later blood lead tests performed between 2001 and 2004.

We obtained basic demographic information including gender, race, and time in months between the first and second BLL test and whether the child was living in a pre-1950 housing unit. Some children had multiple BLL tests before age 2 years and after age 3 years, and some had only one test during each age period. For both case children and control children, we chose the test closest to the child's second birthday as the baseline BLL test, and the test closest to the child's third birthday as the follow-up BLL test. All BLL samples were venous, and all were analyzed by atomic absorption spectroscopy graphite furnace (atomic absorption spectrophotometer; Varian Co., Varian Analytical Instruments, Walnut Creek, CA) with a coefficient of variation of less than 10% at the City of Cleveland Lead Laboratory. Additional information was available from either well-child clinic visits or lead clinic visits at or after age 3 years. The additional information included hemoglobin (HGB), hematocrit (HCT), mean corpuscular volume (MCV), red cell volume distribution width (RDW), and red blood count (RBC), all of which were determined on venous samples and were analyzed using a Sysmex XE-2100 hematology analyzer (TOA Medical Electronics, Kobe, Japan). For statistical analysis, these hematological variables were also coded as categorical variables: HGB ≤ 11.5 g/dl or >11.5 g/dl; HCT ≤ 34% or >34%; MCV ≤ 77 fL or >77 fL; RDW < 14.5% or ≥14.5%; and RBC ≤ 3.9 M/μl or >3.9 M/μl. These cut points are the clinically accepted values for testing 2-year-olds. The complete blood count (CBC) values were obtained as indicators for iron deficiency anemia which can cause an increased absorption of lead. Body mass index (BMI) was calculated from routine height and weight obtained at the time of outpatient visits. BMI was calculated as weight in pounds divided by height in inches squared and multiplied by 703 [14]. According to age standards established by CDC, BMI was categorized as low or within normal limits/high given a child's age and gender at the time of the measurement [15]. Immunization data were retrieved from the Epicare electronic medical record (Epic Systems Corporation, Madison, WI) for each child and were evaluated for completeness at age 3 years using the CASA software package [16]. To be considered immunization compliant, a child must have received three Hepatitis B, four diphtheria, tetanus and pertussis (DTaP), three *Haemophilus influenzae* type b (HIB), three Polio (IPV), one measles, mumps, and rubella (MMR), and one Varicella vaccine—a full 3:4:3:3:1:1 schedule as recommended by CDC [17]. Pneumococcal vaccination was not included because of a nationwide shortage of this vaccine during the study period. Elapsed time between the first and second BLL test

was measured in months. The baseline BLL and the time between the first and second BLL test were categorized by quartiles for all analyses and were analyzed for ordinal trend. The quartiles for the baseline BLL are 1–4, 5, 6, and 7–9 µg/dl. The categories by quartiles for the variable time between the first and second BLL test are 1–21, 21–27, 27–34, and 34–50 months.

The study also looked at test results from 2-year-olds with elevated BLLs to compare the information with that of the 3-year-olds with elevated BLLs. We performed the same analysis for the univariate analysis as well as for the multivariate analysis on the 2-year-olds including race, gender, BMI, immunization status, MCV, HCT, RDW, RBC, and HGB. We also determined whether the child lived in a pre-1950 housing unit.

## Statistical Analysis

Odds ratios (OR) and 95% confidence intervals (CI) were calculated by unconditional multivariate logistic regression [18]. Forward stepwise elimination was used to establish the final reduced logistic regression model. Variables were included into the final model only if their $P$-value was <0.05. The excluded variables did not meaningfully impact the magnitudes of the betas of variables retained in the final model. Specifically, BMI, MCV, HCT, HGB, RBC, residence in a pre-1950 housing unit, and baseline BLLs were each tested in the initial model. Results remained unaffected, however, with differences in the estimates of less than 5%, and these variables were dropped from the logistic regression. The final model included gender, race, immunization status, RDW, time between the first and second BLL tests, and one interaction term for immunization status with time between first and second BLL tests ($\ln\lambda = \alpha + \beta_1$Gender $+ \beta_2$Race $+ \beta_3$Immunization Status $+ \beta_4$RDW $+ \beta_5$Time between first and second BLL tests $+ \delta_1$Immunization Status $\times$ Time between first and second BLL tests). All calculations and analyses were performed with SAS software, version 9.1 [19].

## Results

### Study Subjects

Selected demographic and clinical characteristics for case children and control children are summarized in Table 1. Case children and control children were similar regarding BMI, HCT, MCV and RBC. However, a significantly higher proportion of case children were black ($P = 0.02$) and male ($P < 0.0001$) when compared with the control children. In addition, case children were more likely to

**Table 1** Distribution of variables associated with risk for lead poisoning for case children and control children

| Variable | Cases (262) | | Controls (300) | | Chi-square $p$-value |
|---|---|---|---|---|---|
| | $n$ | % | $n$ | % | |
| Race | | | | | |
| Black | 191 | 72.9 | 192 | 64.0 | 0.02 |
| Other | 71 | 27.1 | 108 | 36.0 | |
| Gender | | | | | |
| Male | 173 | 66.0 | 128 | 42.7 | <0.0001 |
| Female | 89 | 34.0 | 172 | 57.3 | |
| [a]BMI | | | | | |
| Low | 18 | 6.9 | 27 | 9.0 | 0.35 |
| Normal/High | 244 | 93.1 | 273 | 91.0 | |
| Immunization status | | | | | |
| Incomplete | 182 | 69.5 | 160 | 53.3 | <0.0001 |
| Complete | 80 | 30.5 | 140 | 46.7 | |
| [a]MCV test | | | | | |
| Low | 49 | 22.4 | 34 | 15.4 | 0.06 |
| Normal | 170 | 77.6 | 187 | 84.6 | |
| [a]HCT test | | | | | |
| Low | 57 | 25.7 | 56 | 23.1 | 0.53 |
| Normal | 165 | 74.3 | 186 | 76.9 | |
| [a]RDW test | | | | | |
| High | 36 | 16.3 | 15 | 6.8 | 0.0017 |
| Normal | 185 | 83.7 | 207 | 93.2 | |
| [a]RBC test | | | | | |
| Low | 7 | 3.2 | 11 | 4.9 | 0.36 |
| Normal | 211 | 96.8 | 212 | 95.1 | |
| [a]HGB test | | | | | |
| Low | 96 | 39.0 | 85 | 30.5 | 0.04 |
| Normal | 150 | 61.0 | 194 | 69.5 | |
| Pre-1950 housing | | | | | |
| Yes | 246 | 93.9 | 262 | 87.3 | 0.0085 |
| No | 16 | 6.1 | 38 | 12.7 | |

[a] BMI—body mass index; MCV—mean corpuscular volume; HCT—hematocrit; RDW—red cell volume distribution width; RBC—red blood count; HBG—hemoglobin

have incomplete immunizations for their age ($P < 0.0001$), high RDW ($P = 0.0017$), low HGB ($P$-value = 0.04), and to be living in a pre-1950 housing unit ($P = 0.0085$). Data were also analyzed comparing the case children with 2-year-olds who had elevated BLLs. The 3-year-olds were more likely to be male ($P = 0.0033$), black ($P = 0.003$), and have an incomplete immunization status ($P < 0.0001$) (data not shown).

Comparison of BLLs is presented in Table 2. Expressed as geometric means, the second BLL test varied greatly between the case children (13.8 µg/dl) and control children (3.9 µg/dl), $P < 0.0001$, whereas the mean BLL of the first test was statistically significant (5.1) but not clinically

**Table 2** Blood lead levels of the cases and controls

| | Cases | Range | Controls | Range | t-test P-value |
|---|---|---|---|---|---|
| Geometric mean blood lead level | | | | | |
| First test | 5.1 | (1, 9) | 4.6 | (1, 9) | 0.02 |
| Second test | 13.8 | (10, 114) | 3.9 | (1, 9) | <0.0001 |
| Differences in control blood lead levels | | | | | |
| First test | | | 4.6 | (1, 9) | <0.0001 |
| Second test | | | 3.9 | (1, 9) | |
| Time | | | | | |
| Mean follow-up time between first and second test (in months) | 25.8 | (5, 47) | 29.4 | (9, 50) | <0.0001 |
| Mean age at time of second test (in months) | 43.3 | (28, 59) | 44.8 | (25, 59) | 0.0527 |

significant (4.6), $P = 0.02$). Eliminating outliers ($n = 1$) in the second BLL for case children only reduced the geometric mean BLL level to 13.1 µg/dl and the t-test P-value remained the same ($P < 0.0001$). Therefore, all BLLs remained in the dataset. For control children, the geometric mean of the baseline BLLs was 4.6 µg/dl compared with 3.9 µg/dl for the second test. Although we used a paired t-test, this difference was statistically significant ($P < 0.0001$) but the 0.7 µg/dl difference would not be considered clinically meaningful. Case children showed a clinically significant increase in the geometric mean BLL between the first (5.1) and second (13.8) BLL test, $P < 0.0001$. Mean time interval between the two BLL tests was statistically different between case children (25.8 months) and control children (29.4 months), $P < 0.0001$. Mean age between the cases and controls was not statistically different, cases were 43.3 months old and

controls were 44.8 months old at the time of second blood lead test ($P = 0.0527$).

## Multivariate Analyses

Multivariate logistic regression results are shown in Table 3. Several variables showed a significant association with increased elevated BLLs at or after age 3 years: male gender (OR = 3.1, 95%, CI = 2.1,4.8; $P < 0.0001$); black race (OR = 1.5, 95% CI = 1.1,2.6; $P < 0.0189$; and high RDW (OR = 2.5, 95% CI = 1.3,5.0; $P = 0.0083$). In the multivariate analysis, one interaction term was found to be significant: immunization status and time interval between the first and second BLL test ($P = 0.0023$). In general, black male children with an incomplete immunization status should be tested for lead when living in a high risk

**Table 3** Multivariate logistic regression modeling results for elevated blood lead level at second test

| Variable | Model 1 | | | Model 2 | | |
|---|---|---|---|---|---|---|
| | Multivariate logistic regression model with interaction terms | | | Final reduced model | | |
| | Parameter estimate | OR | P-Value | Parameter estimate | OR | P-Value |
| Gender | 1.21 | 3.35 | <0.0001 | 1.14 | 3.1 | <0.0001 |
| Race | 0.60 | 1.82 | 0.0105 | 0.52 | 1.7 | 0.0189 |
| Immunization status | 1.53 | | <0.0001 | 1.57 | | <0.00001 |
| BMI | 0.05 | 1.05 | 0.9077 | | | |
| MCV | 0.15 | 1.16 | 0.6095 | | | |
| HCT | −0.43 | 0.65 | 0.1787 | | | |
| RBC | −0.38 | 0.68 | 0.5455 | | | |
| HGB | 0.14 | 1.15 | 0.6294 | | | |
| RDW | 0.96 | 2.61 | 0.0137 | 0.92 | 2.5 | 0.0083 |
| Pre-1950 housing | 0.61 | 1.84 | 0.1073 | | | |
| First blood lead level | 0.15 | 1.16 | 0.1045 | | | |
| Follow-up time between blood lead tests | 0.06 | | 0.6954 | 0.04 | | 0.8006 |
| *Immunization status × follow-up time between blood lead tests | −0.56 | | 0.0037 | 0.57 | | 0.0023 |

* Only interaction term included in Model 1 because of its significance ($P = 0.0037$)

area. Figure 1a and b illustrate the percent change in BLLs by race and time between tests. Figure 1a depicts the percent change in BLLs among the children whose baseline BLL was in the first quartile (≤4 µg/dl). The figure illustrates that as the time interval between tests increased, the BLLs for the black children in the study increased; whereas for the white children in the study, their BLLs decreased as the between-test time interval lengthened. A limitation of the study is the small numbers within some cells, particularly the cell with only six white children who had a baseline BLL ≤4 µg/dl and who had had a second blood lead test within 21 months of the first test. The small number might cause the percent change to appear inflated. Figure 1b is similar to Fig. 1a, but it presents data for children whose baseline BLL was in the highest quartile (between 7 and 9 µg/dl). Figure 1b indicates much smaller percent increases in BLL for the first three time quartiles and a decrease in percent change for children whose second blood lead test was obtained after the longest intervals (34–50 months). Children whose baseline BLL is in the highest quartile are at higher risk of having an elevated BLL when their follow-up time is short (less than 21 months) rather than long (more than 34 months). Eighteen white children had a baseline BLL between 7 and 9 µg/dl, and they had their second BLL test 27–34 months after their first test
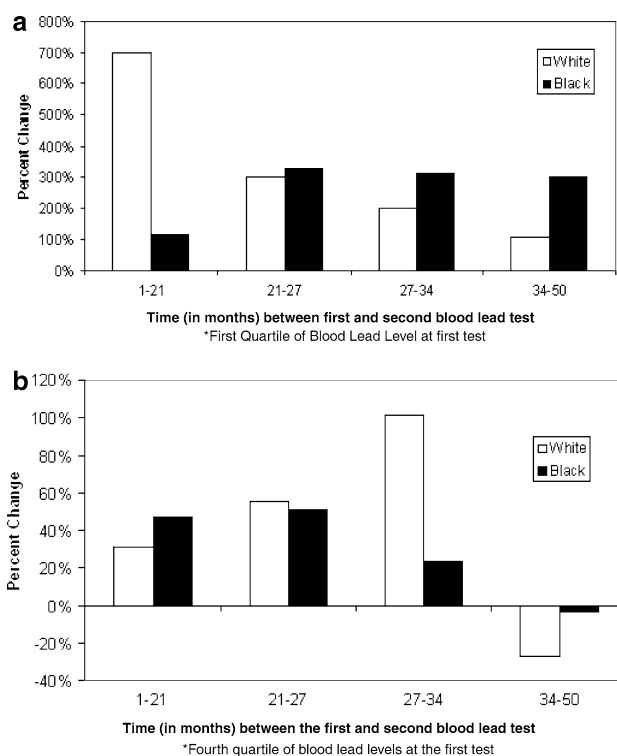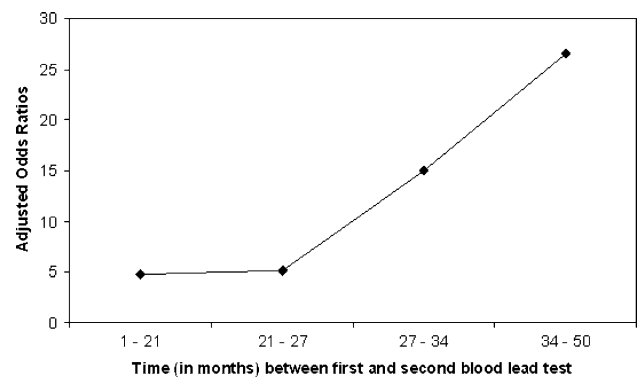


**Fig. 2** Risk for developing lead poisoning for children with incomplete immunization status

(nine case children and nine control children). The large percent change for this group when adjusted for a single outlier ($n = 1$, BLL = 114) would be 24.6%. Figure 2 shows the adjusted OR (aOR) by quartiles of time for children with incomplete immunization status. As this figure indicates, children with an incomplete immunization status are at increased risk for developing an elevated BLL as the time between the first and second blood lead test increases, particularly when the time interval is longer than 27 months. The aOR vary greatly, increasing from 4.8 to 26.6.

Of the 42 cases in which children that had high enough blood lead levels to warrant an environmental investigation by the local health department (16% of all cases), only 29 inspections were completed (69%); 13 families refused both interior and exterior environmental inspection (31%). Lead-based paint hazards (i.e., loose and peeling paint) were found at all 29 inspection sites. Windows and porches covered with non-intact or deteriorated lead-based paint were found in 28 of the 29 risk assessments (data not shown).

## Discussion

We conducted this study to determine whether particular, easily identified risk factors contribute to children having an elevated BLL at or after age 3 years. An aggressive clinical approach to lead screening in Cleveland, a community at high risk for lead poisoning, has already demonstrated that the onset of lead poisoning in children after age 2 years was an important health problem. Determining risk factors is particularly important because the current national recommendations call for high-risk children to be screened for lead poisoning at 1 and 2 years of age and if the children were not previously screened, to screen between 3 and 5 years of age [1–3]. Although



**Fig. 1** Percent change in blood lead levels for those children with first BLL (**a**) ≤4 µg/dl, (**b**) between 7 and 9 µg/dl* by race and follow-up time

primary prevention is the best approach to eliminating childhood lead poisoning, screening high-risk children older than the recommended screening age is advisable to identify and treat new cases of lead poisoning in a high-risk setting. Brody et al. [20] has reported in NHANES III data that children are at most risk for lead poisoning between ages 18 and 36 months. However, testing children after age 2 years is currently recommended only if a child has had an elevated BLL in a previous test. In high prevalence settings, testing older children, especially those with risk factors, might be expected to detect new cases. Considering the question of whether lead poisoning in older children has an impact on health, Kordos et al. [21] has established that school children with elevated concurrent BLLs demonstrate poorer cognitive performance on several tests. Chen et al. [22] illustrated that lead exposure continues to be toxic to children as they reach school age. Their study does not support the idea that all damage to children is done by the time a child is 2 years of age.

The study provided a unique opportunity to study children with blood lead tests performed after age two when they had never been elevated before. This was an important study showing specific predictors that might trigger a medical physician or nurse practitioner to test a child at age three and four living in a high risk area for lead poisoning. This study found that black race males, between the ages of three and four that were not up to date with their immunizations were more likely to be lead poisoned then their counterparts. If RDW data is available, this can be another predictor of the child's risk for becoming lead poisoned. These results may help inform health care professionals regarding these risk factors when determining if a child living in a high risk area should receive a blood lead test after age two.

With the continuing reduction of lead in the environment and the continuing decline of childhood lead poisoning over the past 20 years, we considered it important that all children within our study be close in age and have had their BLL tests at relatively the same time so that they all would have approximately similar exposures to lead [22]. We selected children who had been tested between 2001 and 2004. All were tested for lead before age 2 years and again after age 3 years. No children in this study had an elevated BLL before age 2 years. Our study identifies four independent risk factors (male gender, black race, and high RDW) that are associated with children becoming poisoned by lead after 2 years of age. The variable identifying HGB level, however, was eliminated from the multivariate model because of the explanatory power of the RDW level. Although the best way to diagnose iron deficiency remains unsettled, and an elevated RDW alone is probably an incomplete marker for that condition, RDW is included as one of the measures of iron status in the

National Health and Nutrition Examination Survey (NHANES) III [23–25]. The possibility exists that children with an elevated RDW are more susceptible to enhanced lead absorption due to their altered iron status [22]. Race is a well-known risk factor whose relation to elevated BLLs has been demonstrated [26]. Children who had incomplete immunization status were between 4.8 and 26.6 times more likely to develop an elevated BLL than were children with a complete immunization status, and the risk increased over time. Incomplete immunization status implies that a family, for various reasons (e.g., financial constraints, competing demands, lower educational status, lack of access to healthcare), gives less energy and attention to routine preventive health care and may be unable or unwilling to follow fully the recommendations of health providers.

The season could be confounding our findings. Most of our case children had had their second BLL tests during the summer months (62% for case children versus 42% for control children). Children can develop elevated BLLs during the summer months when they are exposed to more lead from windows being opened and closed, from greater access to porches that are likely to have deteriorating lead paint, and from playing in lead-contaminated soil [27–33]. In addition, the time interval between the first and second BLL test seems to affect the likelihood of children having elevated BLLs. Control children had on average a longer follow-up time than did case children; this difference raises the possibility that some of the control children might have developed elevated BLLs but "outgrew" their elevated levels as their rapid toddler growth lowered the concentration of lead in their bodies.

Although this study demonstrated that black males with a high RDW who are not up to date with their immunizations are at risk for lead poisoning beyond age 2 years, we also sought to determine whether these risk factors differed from those of children who had elevated BLLs at age 2. Three-year-old children with recently diagnosed elevated BLLs were more likely to be black, male, and have incomplete immunization status for their age. Among the 2-year-olds with elevated BLLs, 74.1% had complete immunization status, but among the 3-year-olds with recently diagnosed elevated BLLs, the percentage of complete immunization status was only 30.5%.

With the elimination of lead as a gasoline additive, childhood exposure to lead now occurs most commonly through ingestion of surface dust and soil. Most often, lead is inadvertently ingested by children putting contaminated hands, toys, and foods items into their mouths. The primary source of childhood lead exposure in the United States is lead paint in older, deteriorated housing [26, 31–33]. Lead-contaminated dust and soil may be brought into the house on shoes, clothing, or pets [34]. One study estimates that up

to 30% of household dust is derived from outdoor soil [35]. About 4.2 million housing units in the United States are occupied by families with young children, but approximately 40% of all U.S. housing units (24 million homes) have significant lead-based paint hazards [36–37].

The strengths of our study include the facts that case children and control children lived in the same zip codes within the city of Cleveland, case children and control children were identified using the same data sources, and many children were tested at ages 3 and 4, which provided a large comparison group for analysis. The use of electronic medical records provided accurate immunization data. All BLL tests were venous samples, thus minimizing environmental lead contamination that can artificially elevate capillary blood test results [38].

Our study has several limitations. Because we used a retrospective study, we were limited to available demographic, environmental, and laboratory data. Some of the results shown in Fig. 1a and b are affected by small cell size. Only 29 of the 262 case children had had documented home inspections performed. Environmental inspections during the study period were only being conducted for children with BLLs ≥ 20 µg/dl, and most of the BLLs of our case children were below this threshold. We had hoped to find pertinent information about the home environment for most of the case children. In the study, we did not identify children with developmental delays or cognitive impairments. We can reasonably expect that children with these conditions would have more persistent hand to mouth activity than other children. Finally, laboratory and clinical data suggest that adequate intake of iron, calcium, and vitamins can reduce the amount of lead absorbed. Thus, children with inadequate nutrition would be more likely to have higher BLLs. Our data set included several of the surrogates for iron deficiency, but we could not address the status of the other nutrients.

Although the percentage rate of children with elevated BLLs is declining across the United States, the rate remains high in many cities. Further reductions in the rate of childhood lead poisoning in a community will require a vigorous multi-disciplinary approach that should include adjusting lead screening policies to fit local conditions. Our study illustrates the need for selective BLL testing in 3- and 4-year-olds, and it identifies several risk factors for acquiring elevated BLLs at an age older than the age for routine lead screening. Children with later onset of lead poisoning are likely not being detected [39]. Children who live in high-risk environments need repeated blood testing for lead regardless of earlier test results. Efforts should be made to educate health care providers about the need to screen 3- and 4-year-olds living in high-risk environments because these children need repeated BLL testing regardless of earlier test results.

## References

1. Bellinger, D. C., & Needleman, H. L. (2003). Intellectual impairment and blood lead levels. *New England Journal of Medicine, 349*(5), 500–502.
2. Bellinger, D. C., Stiles, K. M., & Needleman, H. L. (1992). Low-level lead exposure, intelligence and academic achievement: A long- term follow-up study. *Pediatrics, 90*(6), 855–861.
3. Dietrich, K., Ris, M., Succop, P., Og, B., & Bornschein, R. (2001). Early exposure to lead and juvenile delinquency. *Neurotoxicology Teratology, 23*(6), 511–518.
4. Needleman, H., McFarland, C., Ness, R., Fineberg, S., & Tobin, M. (2002). Bone lead levels in adjudicated delinquents. A case control study. *Neurotoxicology Teratology, 24*(6), 711–717.
5. Sciarillo, W., Alexander, G., & Farrell, K. (1992). Lead exposure and child behavior. *American Journal of Public Health, 82*(10), 1356–1360.
6. American Academy of Pediatrics, Committee on Environmental Health. (2005). Lead exposure in children: Prevention, detection, and management. *Pediatrics, 116*(4), 1036–1046.
7. Centers for Disease Control and Prevention. (1997). Screening young children for lead poisoning. Guidance for state and local public health officials. Atlanta: US Department of Health and Human Services, Public Health Service.
8. Centers for Disease Control and Prevention. (2000). Recommendations for blood lead screening of young children enrolled in Medicaid: Targeting a group at risk. Atlanta: US Department of Health and Human Services.
9. Advisory Committee on Childhood Lead Poisoning Prevention, Centers for Disease Control and Prevention. (2000). Recommendations for blood lead screening of young children enrolled in Medicaid: Targeting a group at high risk. *MMWR, 49*(RR-14), 1–13.
10. Piomelli S. (1998). Lead poisoning. In: Nathan D., & Orkin S. (Eds.), Hematology of infancy and childhood (5th ed., p. 482). Philadelphia: WB Saunders.
11. CAN, D. O. Database [database on the Internet]. Cleveland (OH): Center on Urban Poverty and Social Change, Mandel School of Applied Social Sciences, Case Western Reserve University. [cited 2004 Dec 19]. Available from: http://povertycenter.cwru.edu/urban_poverty/dev/cando/overview.asp.
12. Lead poisoning. http://www.ehw.org Cleveland, (Ohio): Environmental Health Watch. cited 2005 Sep 2. Available from: http://www.ehw.org/Lead/LEAD_home3.htm.
13. Centers for Disease Control and Prevention (CDC), National Center for Environmental Health. [homepage on the Internet]. Atlanta: CDC. [cited 2006 Dec 1]. Available from: http://www.cdc.gov/nceh/lead/surv.
14. Centers for Disease Control and Prevention (CDC), National Center for Chronic Disease and Health Promotion. [homepage on the Internet]. Atlanta: CDC. [cited 2005 Dec 1]. Available from: http://www.cdc.gov/nccdphp/dnpa/bmi-adult-formula.htm.
15. Centers for Disease Control and Prevention, National Center for Chronic Disease and Health Promotion. [homepage on the Internet]. Atlanta: CDC. [cited 2005 Dec 1]. Available from: http://www.cdc.gov/nccdphp/dnpa/bmi-for-age.htm.
16. Centers for Disease Control and Prevention, National Immunization Program. [homepage on the Internet]. Atlanta: CDC. [cited 2005 Nov 14]. Available from: http://www.cdc.gov/nip/casa/Default.htm.
17. Centers for Disease Control and Prevention, National Immunization Program. [homepage on the Internet]. Atlanta: CDC. [cited 2005 Nov 14]. Available from: http://www.cdc.gov/nip/recs/child-schedule.htm.
18. Breslow, N. E., & Day, N. E. (1980). The analysis of case–control studies. Statistical methods in cancer research. Vol.1.

International Agency for Research on Cancer, IARC Scientific Publications No. 32.

19. SAS/STAT. (2007). User's guide. Version 8. Cary: SAS Institute Inc.

20. Brody, D. J., Pirkle, J. L., Gunter, E. W., et al. (1994). The decline in blood lead levels in the United States: the National Health and Nutrition Examination Surveys (NHANES). *JAMA, 272*, 284–291.

21. Kordas, K., Canfield, R. L., Lopez, P., Rosado, J. L., Vargas, G. G., Cebrian, M. E., et al. (2006). Deficits in cognitive function and achievement in Mexican first-graders with low blood lead concentrations. *Environmental Research, 100*, 371–386.

22. Chen, A., Dietrich, K. N., Ware, J. H., Radcliffe, J., & Rogan, W. J. (2005). IQ and blood lead from 2 to 7 years of age: Are the effects in older children the residual of high blood lead concentrations in 2-year-olds? *Environmental Health Perspectives, 113*(5), 597–601.

23. Looker, A. V., Gunter, E. W., Johnson, C. L. (1995). Methods to assess iron status in various NHANES surveys. *Nutrition Reviews, 53*(9), 246–254 (Review).

24. McClure, S., Custer, E., Bessman, J. D. (1985). Improved detection of early iron deficiency in nonanemic subjects. *JAMA, 253*(7), 1021–1023.

25. Uchida, T. (1989). Change in red blood cell distribution width with iron deficiency. *Clinical and Laboratory Haematology, 11*(2), 117–121.

26. Pirkle, J. L., Kaufmann, R. B., Brody, D. J., Hickman, T., Gunter, E. W., & Paschal, D. C. (1998). Exposure of the U.S. population to lead, 1991–1994. *Environmental Health Perspectives, 106*, 745–750.

27. Kwong, W. T., Friello, P., & Semba, R. D. (2004). Interactions between iron deficiency and lead poisoning: Epidemiology and pathogenesis. *The Science of the Total Environment, 330*(1–3), 21–37.

28. Hunter, J. M. (1977). The summer disease: An integrative model of the seasonality aspects of childhood lead poisoning. *Social Science & Medicine, 11*, 691–703.

29. Stark, A. D., Quah, R. F., Meig, J. W., & De Lousise, E. R. (1980). Season as a factor in variability of blood-lead levels in children. *Connecticut Medicine, 44*(7), 410–413.

30. Rabinowitz, M., Leviton, A., Needleman, H., Bellinger, D., & Waternaux, C. (1985). Environmental correlates of infant blood lead levels in Boston. *Environmental Research, 38*(1), 96–107.

31. Rothenberg, S. J., Williams, F. A., Delrahim, S., Khan, F., Kraft, M., Lu, M., et al. (1996). Blood lead levels in children in south central Los Angeles. *Archives of Environmental Health, 51*(5), 383–388.

32. Yiin, L. M., Rhoads, G. G., & Lioy, P. J. (2000). Seasonal influences on childhood lead exposure. *Environmental Health Perspectives, 108*(2), 177–82.

33. Centers for Disease Control and Prevention (CDC). Managing elevated blood lead levels among young children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention. Atlanta: US Department of Health and Human Services, CDC; 2002. [cited 2003 Nov 03]. Available from: http://www.cdc.gov/nceh/lead/CaseManagement/caseManage_main.htm.

34. Lanphear, B. P., Burgoon, D. A., Rust, S. W., Eberly, S., & Galke, W. (1998). Environmental exposures to lead and urban children's blood lead levels. *Environmental Research, 76*, 120–130.

35. Agency for Toxic Substances and Disease Registry (ATSDR). (1988) The nature and extent of lead poisoning in children in the United States: A Report to Congress. Atlanta: US Department of Health and Human Services.

36. Calabrese, E., & Stanke, E. (1992). What proportion of household dust is derived from outdoor soil? *Journal of Soil Contamination, 1*, 253–263.

37. Jacobs, D. E., Clickner, R. P., Zhou, J. Y., Viet, S. M., Marker, D. A., Rogers, J. W., Zeldin, D. C., Broene, P., & Friedman, W. (2002). The prevalence of lead-based paint hazards in U.S. Housing. *Environmental Health Perspectives, 110*, 599–606. [cited 2003 Nov 03]. Available from http://www.hud.gov/offices/lead/techstudies/HUD_NSLAH_Vol1.pdf.

38. Bernard, S. M., & McGeehin, M. A. (2003). Prevalence of blood lead levels ≥5 μg/dl among US children 1 to 5 years of age and socioeconomic and demographic factors associated with blood lead levels 5–10 μg/dl, Third National Health and Nutrition Examination Survey, 1988–1994. *Pediatrics, 112*, 1308–1313.

39. Schlenker, T. L., Fritz, C. J., Murphy, A., & Shepeard, S. (1994). Feasibility and effectiveness of screening for childhood lead poisoning in private medical practice. *Archives of Pediatrics & Adolescent Medicine, 148*(7), 761–764.