

Screening Housing to Prevent Lead Toxicity in Children

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SYNOPSIS

Objective. Screening children to identify those with blood lead levels ≥ 10 $\mu\text{g}/\text{dl}$ fails to protect children from lead-associated cognitive deficits and behavioral problems. To broaden our efforts at primary prevention, screening criteria are needed to identify lead-contaminated housing before children are unduly exposed. The purpose of this study was to identify and validate housing characteristics associated with children having elevated blood lead levels (≥ 10 $\mu\text{g}/\text{dl}$).

Methods. Two existing studies were used to examine housing characteristics linked with undue lead exposure: a cross-sectional study of 205 children aged 12 to 31 months, and a random sample from a longitudinal study of 276 children followed from 6 to 24 months of age. Logistic regression analysis was conducted to examine the association of children's blood lead levels ≥ 10 $\mu\text{g}/\text{dl}$.

Results. The mean age of the 481 children was 17.8 months; 99 (20.6%) had a blood lead concentration of 10 $\mu\text{g}/\text{dl}$ or higher. The following characteristics were associated with blood lead concentration ≥ 10 $\mu\text{g}/\text{dl}$: floor lead loading >15 $\mu\text{g}/\text{ft}^2$ (odds ratio [OR]=2.2; 95% confidence interval [CI] 1.3, 3.8); rental housing (OR=3.2; 95% CI 1.3, 7.6); poor housing condition (OR=2.1; CI 1.2, 3.6); African American race (OR=3.3; CI 1.9, 6.1); paint chip ingestion (OR=5.8; CI 1.3, 26.5); and soil ingestion (OR=2.2; CI 1.1, 4.2). Housing characteristics including rental status, lead-contaminated floor dust, and housing condition had a range of sensitivity from 47% to 92%; specificity from 28% to 76%; a positive predictive value from 25% to 34%; and a negative predictive value of 85% to 93%.

Conclusions. Housing characteristics and floor dust lead levels can be used to screen housing to identify lead hazards prior to occupancy, before purchasing a home, or after renovation to prevent children's exposure to lead hazards.

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Despite a dramatic decline in lead exposures, many children in the United States have blood lead levels consistent with lead toxicity.¹ The adverse consequences of low-level lead exposure, including intellectual impairments, behavioral problems, and delinquency persist into adolescence or early adulthood.²⁻⁶ Moreover, there is increasing evidence that blood lead concentrations below 10 µg/dl, the level set by the Centers for Disease Control and Prevention and the World Health Organization to indicate sufficient harm to justify action, are linked with intellectual impairments and reading deficits in children.⁷⁻⁹

The current strategy to “prevent” lead toxicity is to screen children and identify those who have blood lead of 10 µg/dl or higher. Unfortunately, this strategy fails to prevent the adverse consequences of lead exposure because the child with an elevated blood lead concentration is used as a trigger to control lead hazards. In contrast, screening housing to identify those that contain lead hazards should focus our efforts on the prevention of lead toxicity.¹⁰ A wipe test (first used 100 years ago by Lockhart Gibson¹¹) along with an assessment of other housing characteristics, offers considerable promise to identify housing that contains lead hazards. Housing characteristics that increase a child’s risk for having an elevated blood lead level are well documented, but the screening characteristics of various dust lead levels or specific housing characteristics are poorly defined.

The purpose of this analysis was to identify and validate housing characteristics that are associated with children having an elevated blood lead level (i.e., ≥ 10 µg/dl).

METHODS

Children in these analyses were participants from two studies conducted in Rochester, New York. The first study, a randomly sampled cross-sectional study conducted in 1993, involved 205 children who were 12 to 31 months of age and resided in the same house since at least 6 months of age.¹² The second study, a randomized, controlled trial of dust control that began in 1995, involved 276 children followed from 6 to 24 months of age.¹³ Once a family was deemed eligible and agreed to participate, a study team visited their home, obtained informed consent and a blood sample, conducted an interview, and collected environmental samples. The IRB of the University of Rochester School of Medicine approved both studies.

During each of the 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 (e.g., soil ingestion, paint chip ingestion) and time spent outdoors. Blood was taken at each visit by a certified phlebotomist and measured for lead using electrothermal Atomization Atomic Absorption spectrometry at Wadsworth Laboratories, Albany NY. All results are the means of six or more separate analyses (three aliquots/day measured on two consecutive days) performed on each blood sample. Measured levels of lead in whole blood < 1 µg/dl were reported as less than the detection limit.

An environmental technician systematically conducted dust sampling to characterize children’s exposure to lead-contaminated dust.¹¹⁻¹⁴ Three to four interior dust wipe samples were taken from surfaces that were accessible to a

child (i.e., carpeted floors, non-carpeted 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. An environmental technician measured paint lead content, visually assessed paint condition, and took soil and water samples. Lead content of paint was measured by using a portable x-ray fluorescence analyzer (XRF) (Microlead I, Warrington). The condition of painted surfaces was done by visual inspection.¹⁵ Three soil samples were combined for a composite foundation sample. Parents collected a water sample (250 cc) in the morning from the kitchen tap after the water flowed for one minute.

Dust samples were analyzed first by flame atomic absorption, which was followed by graphite furnace if levels were below 5 µg/sample. The detection limit of graphite furnace for the dust wipe was 0.5 µg/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.

Statistical methods

Although carpeted and non-carpeted floor dust lead loading values were both predictors of 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 or poor = 3) by the paint lead concentration as measured using the XRF for 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.

We conducted the analyses in two steps. First, the two data sets were combined; next, a random sample representing 80% of the children in the combined data sets was selected. A logistic regression model was then developed with the outcome variable coded as 1 if the child’s blood lead level was ≥ 10 µg/dl and 0 if < 10 µg/dl. The model was developed in a modified stepwise procedure. Variables of interest (e.g. lead-contaminated floor dust) were forced in the model and additional variables were added one at a time. We then determined whether each additional variable had a statistically significant relationship to blood lead concentration or if inclusion of that term substantially changed the coefficients of the lead exposure terms. Variables were included if they were significant in a two-sided test ($p < 0.05$) or marginally significant ($p < 0.10$) if their inclusion changed the lead exposure coefficients by more than approximately 25%.

We validated the initial model by comparing its predictions with the remaining 20% of the data not used to develop the model. We did this by computing the probability of being over 10 µg/dl for each child in the 80% group and then selecting the value of the probability (p) as a cut-off for predicting blood lead concentration ≥ 10 µg/dl that maximized the sensitivity and specificity. This value of p was used to predict which children in the 20% sample had blood lead concentrations ≥ 10 µg/dl. The predictions were assessed for their agreement with the actual measured blood lead levels using a kappa statistic. The agreement was highly significant (kappa=0.51, $p < .001$). The area under the re-

ceiver operating characteristics (ROC) curve for the initial model was 0.81. We concluded that the model was acceptable, and subsequently combined the development and validation data sets and re-estimated the model coefficients.

We created ROC curves for the logistic regression models and residential characteristics associated with children having blood lead levels of 10 µg/dl or higher. The ROC curves were constructed by varying the cut-off point for each variable and plotting the true positive rate (sensitivity) by the false-positive rate (1-specificity) at each point. These curves illustrate the trade-off between the true-positive rate and a low false-positive rate. We used SAS for all statistical analyses.¹⁶

RESULTS

The mean age of 481 children tested was 17.8 months (standard deviation [SD]=6.7 months). The arithmetic mean blood lead level was 7.2 µg/dl (95% CI 6.7, 7.6 µg/dl); 99 (20.6%) had a blood lead concentration of 10 µg/dl or higher (Table 1). The majority of families (76%) lived in rental housing; 238 (50%) were single-parent households. Mouthing behaviors previously shown to be risk factors for exposure to environmental sources of lead were common. Soil ingestion was reported by a parent for 82 (17%) of children, mouthing the windowsill was reported for 94

(19.5%), and paint chip ingestion was reported for 12 (2.5%) of children (Table 1).

The arithmetic mean floor dust lead levels, 16.8 µg/ft² (95% CI 13.5, 20.1), were considerably lower than EPA standards of 40 µg/ft². The arithmetic mean windowsill dust lead levels were 1,352 µg/ft² (95% CI 434, 2,270) and the arithmetic mean window trough dust lead levels were 33,871 µg/ft² (95% CI 26,510, 41,233). The arithmetic mean soil lead concentration was 1,880 µg/g (95% CI 1,627, 2,133).

There was a large increase in the proportion of children with a blood lead concentration ≥10 µg/dl at residential floor lead levels considerably lower than the U.S. EPA standard.¹⁶ Compared with children who were exposed to floor lead levels below 2.5 µg/ft² (referent group), children who were exposed to floor lead levels 5 µg/ft² to 10 µg/ft² were at 3.5-fold greater risk for having a blood lead concentration ≥10 µg/dl, a 4.1-fold greater risk at floor dust lead levels between 15 to 25 µg/ft², and a 8.7-fold greater risk for exposures >25 µg/ft² (Figure).

In logistic regression analysis, the following housing characteristics were associated with blood lead concentration ≥10 µg/dl: floor lead loading >15 µg/ft² (OR=2.2; 95% CI 1.3, 3.8); rental housing (OR=3.2; 95% CI 1.3, 7.6); and poor housing condition (OR=2.1; 95% CI 1.2, 3.6) (Table 3). Individual risk characteristics for having a blood lead level ≥10 µg/dl were African American race (OR=3.3; 95% CI 1.9, 6.1); paint chip ingestion (OR=5.8, CI=1.3, 26.5); and soil ingestion (OR=2.2; 95% CI 1.1, 4.2) (Table 2).

Rental status, as an indicator of residential lead hazard, identified over 90% of all children who had blood lead level ≥10 µg/dl, but only 1 in 4 children had a blood lead ≥10 µg/dl (Table 3). We also present screening characteristics for two indices of housing characteristics. In the first index, rental housing with dust lead levels >15 µg/ft² identified about 50% of housing units with a lead hazard, with a specificity of about 80% (Table 3). By adding poor housing condition to the index, the sensitivity fell to 33%, while the specificity increased to 92%.

Floor dust lead levels set at 5 µg/ft² identified 87% of children with blood lead level ≥10 µg/dl, but only about one-third of children living in housing units that exceeded 5 µg/ft² had a blood lead blood lead level ≥10 µg/dl. In contrast, a dust lead level >15 µg/ft² identified 54% of children with a blood lead level ≥10 µg/dl; 72% of children living in housing units that exceeded 15 µg/ft² had a blood lead level ≥10 µg/dl. The screening characteristics for lead-contaminated house dust were comparable for rental units and owner-occupied housing (data not shown separately) (Table 4). The current U.S. EPA residential floor standard (40 µg/ft²) failed to identify 85% of housing units of children who had a blood lead concentration of 10 µg/dl. By itself, poor housing condition identified 47% of children with a blood lead level ≥10 µg/dl.

DISCUSSION

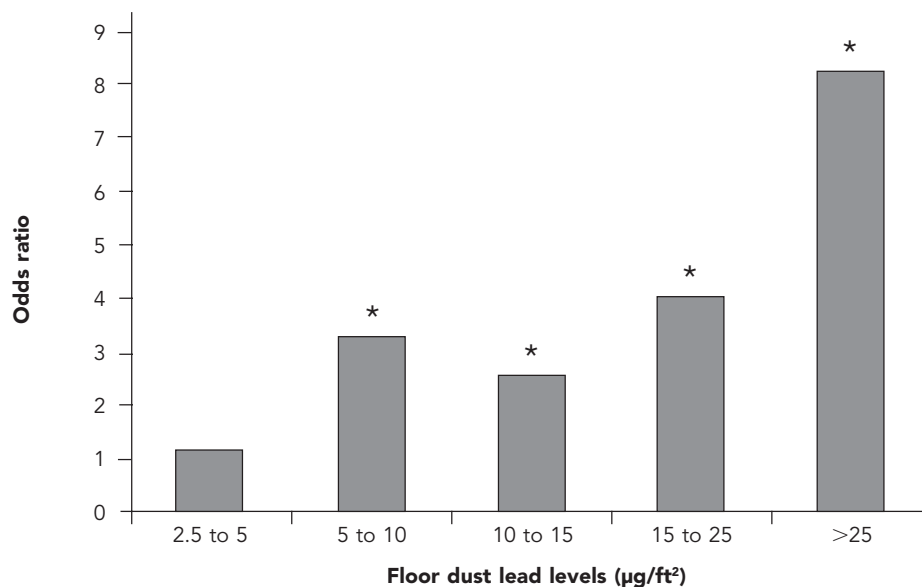
This study demonstrates that various characteristics—including lead-contaminated floor dust, condition of housing, and rental status—can be used to identify housing that contains residential lead hazards. These data, in combination with research identifying census level characteristics that can be

Table 1. Characteristics of children, families, and housing in the two Rochester Lead Studies

Characteristic	Number (percent)
Total	481 (100)
Race or ethnicity	
African American	252 (52.4)
White	140 (29.1)
Other	89 (18.5)
Housing condition	
Poor	136 (29)
Good	331 (71)
Household income	
<\$15,500	298 (64)
Rental housing	366 (76)
Marital status	
Single	238 (50)
Married	161 (34)
Single, living together	43 (9)
Divorced, separated, or widowed	34 (7)
Behaviors	
Soil ingestion	82 (17%)
Paint chip ingestion	12 (2.5%)
Mouthing window sill	94 (19.5%)
Environmental exposures	Mean (95% CI)
Floor dust lead (µg/ft ²) ^a	16.8 (13.5, 20.1)
Window sill lead (µg/ft ²) ^a	1,352 (434, 2,270)
Window trough lead (µg/ft ²) ^a	33,871 (26,510, 41,233)
Soil lead (µg/g) ^a	1,880 (1,627, 2,133)

^aReported values are arithmetic means; 95% confidence intervals (CI) are in parentheses.

Figure. Odds ratio of blood lead concentration ≥ 10 $\mu\text{g}/\text{dl}$ by various floor dust lead levels ($\mu\text{g}/\text{ft}^2$) compared with children exposed to floor dust lead levels below 2.5 $\mu\text{g}/\text{ft}^2$ (reference group). Asterisk indicates significant difference ($p < .05$).



used to identify neighborhoods with a high proportion of housing containing lead hazards (17–22), can be used to target screening and lead hazard control efforts before a child develops lead toxicity. Screening for lead hazards, including a visual inspection and dust testing, should be conducted in older housing prior to the purchase or rental of a housing unit. Dust lead testing should also be considered after renovation of older housing units. Finally, while these data were published previously, they bolster earlier research showing that the existing U.S. EPA residential lead standards and HUD post-abatement clearance levels are not set low enough to protect children.

These data indicate that the U.S. EPA's residential lead standards will not protect the vast majority of children from lead toxicity.^{10,12,23–25} We found that floor dust lead levels considerably lower than the floor standard of 40 $\mu\text{g}/\text{ft}^2$ were

associated with a considerable excess risk of children having blood lead levels ≥ 10 $\mu\text{g}/\text{dl}$. Children were at 3.5-fold greater risk for having a blood lead concentration ≥ 10 $\mu\text{g}/\text{dl}$ if they were exposed to floor dust lead levels of 5 $\mu\text{g}/\text{ft}^2$ to 10 $\mu\text{g}/\text{ft}^2$ compared with levels < 2.5 $\mu\text{g}/\text{ft}^2$.²⁵ Moreover, if the U.S. EPA floor standard of 40 $\mu\text{g}/\text{ft}^2$ was used, only 15% of children would be protected from residential lead hazards.²⁶ Finally, these standards are based on the probability of children having blood lead levels of 10 $\mu\text{g}/\text{dl}$ or higher; there is increasing evidence that blood lead levels below 10 $\mu\text{g}/\text{dl}$ are associated with cognitive deficits.^{7–9}

We found that housing characteristics and dust lead tests could be used as a screening tool to identify houses that contain lead hazards. In particular, rental status, floor dust lead levels, and housing condition were predictors of lead hazards. A floor dust lead standard of 5 $\mu\text{g}/\text{ft}^2$ identified

Table 2. Logistic regression analysis of characteristics associated with blood lead concentration > 10 $\mu\text{g}/\text{dl}$ among 481 children

Characteristics	Adjusted OR	95% CI	Estimate	Standard error	p value
Housing characteristics					
Rental housing	3.2	(1.3, 7.5)	1.16	0.44	.009
Floor dust lead > 15 $\mu\text{g}/\text{ft}^2$	2.2	(1.3, 3.8)	0.78	0.27	.004
House in poor condition	2.1	(1.2, 3.6)	0.73	0.28	.008
Individual characteristics					
Paint chip ingestion	5.8	(1.3, 26.5)	1.76	0.77	.02
African American race	3.3	(1.9, 6.1)	1.19	0.31	.0001
Soil ingestion	2.2	(1.1, 4.2)	0.78	0.33	.01
Age of children	1.1	(1.07, 1.16)	0.108	0.02	$< .0001$

OR = odds ratio

CI = confidence interval

Table 3. Screening characteristics for factors associated with children having a blood lead level ≥ 10 $\mu\text{g}/\text{dl}$

Characteristics	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Area under the curve (standard error)
Total					0.81
Rent home	0.92	0.28	0.25	0.93	0.60
African American	0.75	0.53	0.29	0.89	0.64
Poor housing condition	0.47	0.76	0.34	0.85	0.61
Soil ingestion	0.29	0.86	0.34	0.82	0.57
Paint chips	0.08	0.99	0.67	0.81	0.54
Housing index					
Rental housing AND floor >5 $\mu\text{g}/\text{ft}^2$	0.78	0.57	0.32	0.91	0.67
Rental housing AND floor >5 $\mu\text{g}/\text{ft}^2$ AND poor housing condition	0.42	0.87	0.46	0.85	0.65
Rental housing AND floor >15 $\mu\text{g}/\text{ft}^2$	0.49	0.79	0.38	0.86	0.64
Rental housing AND floor >15 $\mu\text{g}/\text{ft}^2$ AND poor housing condition	0.32	0.92	0.50	0.84	0.62

about 90% of housing units that pose a risk of a child developing a blood lead level ≥ 10 $\mu\text{g}/\text{dl}$, but there is an obvious trade-off between sensitivity and specificity. Policy makers, housing agencies, and communities will need to decide the acceptable number of housing units the system fails detect. It is not necessary or even particularly useful to use children's characteristics or behaviors to identify residential lead hazards. Instead, public health agencies, home-buyers, and housing agencies can use rental status, housing condition, and dust lead levels to prevent undue lead exposure from residential lead hazards. Other research shows that lead-contaminated water is a predictor of children's lead intake.²⁵ Thus, even though lead-contaminated water wasn't a predictor of children having blood lead levels >10 $\mu\text{g}/\text{dl}$ in this study, parents and public health officials should consider testing water for lead in certain geographical areas. Finally,

although we did not specifically identify lead-contaminated soil as a predictor in this analysis, we did show indirectly that ingestion of lead-contaminated soil was an important source of lead intake.

Consistent with earlier research, African American children were at higher risk for having blood lead levels ≥ 10 $\mu\text{g}/\text{dl}$. Racial disparity in blood lead levels among children is due, in large part, to poor housing conditions and higher lead exposure among minority children.^{25,27} But these children remained at increased risk even after controlling for environmental exposures and dietary intake of iron or calcium.²⁵ The reason for this striking disparity remains unclear. Nevertheless, reducing environmental lead exposure should reduce racial differences in blood lead concentration whether these differences are due to enhanced lead absorption or retention.

Table 4. Screening characteristics associated blood lead level ≥ 10 $\mu\text{g}/\text{dl}$ for various levels of lead-contaminated floor dust

Floor dust	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Area under the curve (standard error)
>2.5 $\mu\text{g}/\text{ft}^2$	0.95	0.16	0.23	0.92	0.56 (.03)
>5.0 $\mu\text{g}/\text{ft}^2$	0.87	0.38	0.26	0.92	0.62 (.03)
>10 $\mu\text{g}/\text{ft}^2$	0.68	0.55	0.28	0.87	0.61 (.03)
>15 $\mu\text{g}/\text{ft}^2$	0.54	0.72	0.33	0.86	0.63 (.03)
>20 $\mu\text{g}/\text{ft}^2$	0.41	0.83	0.39	0.85	0.62 (.03)
>25 $\mu\text{g}/\text{ft}^2$	0.33	0.88	0.42	0.84	0.61 (.03)
>30 $\mu\text{g}/\text{ft}^2$	0.24	0.91	0.40	0.82	0.57 (.03)
>35 $\mu\text{g}/\text{ft}^2$	0.19	0.93	0.42	0.82	0.56 (.03)
>40 $\mu\text{g}/\text{ft}^2$	0.16	0.94	0.42	0.81	0.55 (.03)
>45 $\mu\text{g}/\text{ft}^2$	0.10	0.96	0.40	0.81	0.53 (.03)
>50 $\mu\text{g}/\text{ft}^2$	0.08	0.96	0.36	0.80	0.52 (.03)

CONCLUSIONS

The findings of this study may not be generalizable to other cities or states. The prevalence of children having blood lead levels ≥ 10 $\mu\text{g}/\text{dl}$, for example, is higher in Rochester than many other U.S. cities. Still, the findings of the relationship of lead-contaminated dust and soil were comparable with a pooled analysis that involved over 1,200 children who lived in urban or lead-contaminated smelter, mining, or milling communities.²³ Moreover, the risk factors identified in this analysis, including African American race and soil ingestion, are consistent with other published research.^{27–29} Thus, while the screening characteristics would vary by the prevalence of children having a blood lead concentration in excess of 10 $\mu\text{g}/\text{dl}$, the sensitivity and specificity should be similar. Finally, we did not have a measure of age of housing; age of housing is an excellent indicator of lead toxicity that can be used to further target screening of housing for lead hazards.^{28,30}

In summary, this study demonstrates that housing characteristics—including poor condition, levels of lead-contaminated floor dust, and rental status—can be used to screen housing to identify lead hazards before children are unduly exposed. To prevent children from developing lead toxicity, older housing, especially rental housing, should be screened prior to occupancy, after renovation and abatement

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REFERENCES

- Pirkle JL, Kaufmann RB, Brody DJ, Hickman T, Gunter EW, Paschal DC. Exposure of the U.S. population to lead, 1991–1994. *Environ Health Perspect* 1998;106:745–50.
- Burns JM, Baghurst PA, Sawyer MG, McMichael AJ, Tong SL. Lifetime low-level exposure to environmental lead and children's emotional and behavioral development at ages 11–13 years. The Port Pirie Cohort Study. *Am J Epidemiol* 1999;149:740–9.
- Dietrich KN, Ris D, Succop PA, Berger OG, Bornschein RL. Early exposure to lead and juvenile delinquency. *Neurotoxicol Teratol* 2001;23:511–8.
- Bellinger DC, Stiles KM, Needleman HL. Low-level lead exposure, intelligence and academic achievement: a long-term follow-up study. *Pediatrics* 1992;90:855–61.
- Bellinger D, Needleman HL. Intellectual impairment and blood lead levels. *New Engl J Med* 2003;349:500–2.
- Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN. The long-term effects of exposure to low doses of lead in childhood. An 11-year follow-up report. *N Engl J Med* 1990;322(2):83–8.
- Schwartz J. Low-level lead exposure and children's IQ: a meta-analysis and search for a threshold. *Environ Res* 1994;65:42–55.
- Canfield RL, Henderson CR Jr, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentrations below 10 micrograms per deciliter. *N Engl J Med* 2003;348:1517–26.
- Lanphear BP, Dietrich KN, Auinger P, Cox C. Cognitive deficits associated with blood lead concentrations <10 $\mu\text{g}/\text{dl}$ in U.S. children and adolescents. *Pub Health Rep* 2000;115:521–9.
- Lanphear BP. The paradox of lead poisoning prevention. *Science* 1998;281:1617–8.
- Gibson JL. A plea for painted railings and painted rooms as the source of lead poisoning amongst Queensland children. *Australasian Medical Gazette* 1904;23:149–53.
- Lanphear BP, Weitzman M, Winter NL, Eberly S, Yakir B, Tanner M, et al. Lead-contaminated house dust and urban children's blood lead levels. *Am J Public Health* 1996;86:1416–21.
- Lanphear BP, Howard CR, Eberly S, Auinger P, Kolassa J, Weitzman M, et al. Primary prevention of childhood lead exposure: a randomized trial of dust control. *Pediatrics* 1999;103(4 Pt 1):772–7.
- Lanphear BP, Emond M, Jacobs DE, Weitzman M, Tanner M, Winter ML, et al. A side-by-side comparison of dust collection methods for sampling lead-contaminated house-dust. *Environ Res* 1995;68:114–23.
- Lanphear BP, Burgoon DA, Rust SW, Eberly S, Galke W. Environmental exposures to lead and urban children's blood lead levels. *Environ Res* 1998;76:120–30.
- SAS Institute, Inc., SAS/STAT User's Guide, Version 6, Fourth Edition, 2nd volume, Cary (NC): SAS Institute, Inc.; 1989.
- Norman EH, Bordley WC, Hertz-Picciotto I, Newton DA. Rural-urban blood lead differences in North Carolina children. *Pediatrics* 1994;94:59–64.
- Sargent JD, Brown MJ, Freeman JL, Bailey A, Goodman D, Freeman DH Jr. Childhood lead poisoning in Massachusetts communities: its association with sociodemographic and housing characteristics. *Am J Public Health* 1995;85:528–34.
- Lanphear BP, Byrd RS, Auinger P, Schaffer SJ. Community characteristics associated with elevated blood lead levels in children. *Pediatrics* 1998;101:264–71.
- Litaker D, Kippes CM, Gallagher TE, O'Connor ME. Targeting lead screening. The Ohio Lead Risk Score. *Pediatrics* 2000;106:e69 [cited 2004 Dec]. Available from: URL: <http://www.pediatrics.org/cgi/content/full/106/5/e69>
- Binns HJ, LeBailly SA, Fingar AR, Saunders S. Evaluation of risk assessment questions used to target blood lead screening in Illinois. *Pediatrics* 1999;103:100–6.
- Miranda ML, Dolinoy DC, Overstreet MA. Mapping for prevention: GIS models for directing childhood lead poisoning prevention programs. *Environ Health Perspect* 2002;110:947–53.
- Lanphear BP, Matte TD, Rogers J, Clickner RP, Dietz B, Bornschein RL, et al. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels. A pooled analysis of 12 epidemiologic studies. *Environ Res* 1998;79:51–68.
- Malcoe LH, Lynch RA, Keger MC, Skaggs VJ. Lead sources, behaviors, and socioeconomic factors in relationship to blood lead of native American and white children: a community-based assessment of a former mining area. *Environ Health Perspect* 2002;110 Suppl 2:221–31.
- Lanphear BP, Hornung R, Ho M, Howard CR, Eberly S, Knauf K. Environmental lead exposure during early childhood. *Pediatrics* 2002;140:40–7.
- U.S. Environmental Protection Agency. 40 CFR part 745. Lead; identification of dangerous levels of lead: Final Rule. *Federal Register* 2001;66:1206–40.
- Lanphear BP, Weitzman M, Eberly S. Racial differences in environmental exposures to lead. *Am J Public Health* 1996;86:1460–3.
- Clark CS, Bornschein RL, Succop P, Que Hee SS, Hammond PB, Peace B. Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. *Environ Res* 1985;38:46–53.
- Rabinowitz M, Leviton A, Needleman H, Bellinger D, Wateraux C. Environmental correlates of infant blood lead levels in Boston. *Environ Res* 1985;38:96–107.
- Jacobs DE, Clickner RP, Zhou JY, Viet SM, Marker DA, Rogers JW, et al. The prevalence of lead-based paint hazards in U.S. housing. *Environ Health Perspect* 2002;110:A599–606.