

# The Effect of Soil Abatement on Blood Lead Levels in Children Living Near a Former Smelting and Milling Operation

---

BRUCE P. LANPHEAR, MD, MPH<sup>a</sup>  
PAUL SUCCOP, PhD<sup>b</sup>  
SANDRA RODA, MS<sup>b</sup>  
GERRY HENNINGSEN, DVM, PhD<sup>c</sup>

## SYNOPSIS

**Objective.** To evaluate the effect of soil abatement on children's blood lead concentrations and on environmental levels of lead and arsenic.

**Methods.** Two cross-sectional surveys were conducted. The first (1989) was of a random sample of 6- to 72-month-old children ( $n=112$ ). The second (1998) included all 6- to 72-month-old children whose parents agreed to participate in the survey ( $n=215$ ). From 1993 to 1996, soil abatement was conducted around homes with average soil lead concentration  $>500$  parts per million (ppm). Venipuncture blood samples were taken, interviews were conducted, and samples of house dust, soil, water, and paint were tested for lead and arsenic, using identical protocols in both surveys. The expected decline in blood lead concentrations were calculated for children who lived in houses that were abated, compared with children who lived in houses that were not abated.

**Results.** Lead and arsenic in soil and interior dust in homes that underwent soil abatement declined significantly compared to unabated homes ( $p<.05$ ). After adjustment for potential confounders, the blood lead concentration in children ages 6 to 72 months who lived in soil-abated housing declined 42.8% faster than children who lived in unabated housing ( $p=0.14$ ). In children ages 6 to 36 months, the decline was 45.4% faster ( $p=0.03$ ). The estimated reduction in blood lead for children ages 6 to 36 months was 3.5  $\mu\text{g/dL}$  for every 1,000 ppm reduction in soil lead concentration (95% confidence interval [CI]=2.4  $\mu\text{g/dL}$ , 4.6  $\mu\text{g/dL}$ ).

**Conclusion.** Soil abatement was associated with a significant decline in children's blood lead and indoor environmental levels of lead and arsenic.

---

<sup>a</sup>Cincinnati Children's Environmental Health Center, Children's Hospital Medical Center, Cincinnati OH

<sup>b</sup>Department of Environmental Health, University of Cincinnati, Cincinnati, OH

<sup>c</sup>Region VIII, U.S. Environmental Protection Agency, Denver, CO

Address correspondence to: Bruce P. Lanphear, MD, MPH, Children's Hospital Medical Center, 3333 Burnet Ave., Cincinnati, OH 45229; tel. 513-636-3778; fax 513-636-4402; e-mail <bruce.lanphear@chmcc.org>.

© 2003 Association of Schools of Public Health

Children who live near lead mining, milling, or smelting industries are at increased risk for lead poisoning and lead toxicity from both industrial emissions and lead-contaminated soil.<sup>1-11</sup> Children's blood lead concentrations fell dramatically after the phase-out of leaded gasoline, lead-soldered canned foods, and emission controls.<sup>1,8,12</sup> But lead-contaminated soil remains an ongoing source of lead intake for many children who live in these contaminated communities.<sup>13</sup>

In 1980, the United States Congress passed the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) establishing federal authority for emergency response and cleanup of hazardous substances, including lead contaminated soil. The Agency for Toxic Substances and Disease Registry (ATSDR) estimated that 1,026 (71%) of 1,445 current or former Superfund sites contain lead hazards.<sup>13</sup> Over \$20 billion has been spent to remediate lead hazards,<sup>13</sup> but the effect of soil abatement on blood lead concentration for children who live in mining, milling, or smelting communities has not been rigorously evaluated.

The purpose of this study was to determine the effect of soil abatement for residential soil with mean soil lead concentrations greater than 500 parts per million (ppm) microgram/gram ( $\mu\text{g/g}$ ) on blood lead concentration in children during their peak age of susceptibility to lead ingestion and absorption, and environmental levels of lead and arsenic.

## METHODS

The city of Midvale (pop. 12,000) is a former site of a smelting and milling operation located 12 miles south of Salt Lake City, Utah. The smelter and milling operation began in 1910. The smelter, located adjacent to the mill site, closed in 1958 and the milling operation closed in 1971. Sulfide concentrates of lead, copper, and zinc were extracted or "milled" from ore by froth flotation. The waste product or "tailings" containing high concentrations of lead and arsenic were dumped adjacent to the mill site in piles up to 50 feet deep, covering over 250 acres. Soil containing lead and arsenic from the tailings was dispersed by wind, carried home in worker's clothing, and used for gardening, driveways, or play areas in and around residential dwellings.

Two cross-sectional studies were employed to estimate the effect of soil abatement on children's blood lead concentration and lead contamination of soil and house dust. Environmental arsenic contamination was also measured in both studies. Children who were 6 to 72 months old and lived in the city of Midvale, Utah, for at least two months were deemed eligible for the

studies. Children were excluded from the study if they had taken a prescribed iron supplement in the past two months or if there had been a major renovation of their residence during the past 12 months. The study was approved by the Investigational Review Board of Cincinnati Children's Hospital Medical Center.

The first cross-sectional study, conducted in October 1989, was a random survey.<sup>11</sup> After completing a door-to-door census, a proportional random sampling strategy was used to ensure that the sample was representative of the community with respect to the location of the residence and type of housing. In 1993, a clay cap was constructed over the tailings at the former mining and milling site to contain the waste and reduce the potential for wind transport of heavy metals into the adjacent community of Midvale. Soil abatement of yards with average soil lead concentrations greater than 500 ppm began in 1993 and continued until 1996. Soil abatement consisted of excavation to a depth of 18 inches and backfill with clean soil. The second cross-sectional survey, conducted in October 1998, was a full population survey of all eligible children. After completing a door-to-door census, all eligible children and their families were invited to participate.

Once a family was determined to be eligible and agreed to participate, an appointment was scheduled to conduct an interview and obtain a venipuncture blood sample from eligible children. A trained interviewer conducted a face-to-face survey with the primary caretaker to assess risk factors for lead exposure, including mouthing behaviors, soil ingestion, time spent outdoors, and where the child played. After the interview, an environmental health team visited each residence, visually inspected the home for possible sources of lead exposure, and collected environmental samples. If more than one eligible child lived in a residence, one child was chosen at random for the analysis.

Specific protocols and techniques were followed for the collection of blood and environmental samples to minimize contamination of the samples and ensure comparability between the studies. The puncture site was thoroughly cleansed to prepare the patient for sampling. Blood collection equipment and supplies were lot tested for the presence of lead contamination, then stored to keep them free from dust and contaminants. Samples were kept refrigerated and shipped overnight in coolers to the laboratory for analysis.

### Environmental samples

All environmental samples taken in 1998 were collected using protocols identical to those taken in 1989.



Photo by Earl Dotter

Dust sampling was conducted to characterize children's potential exposure to lead from house dust. We used the Dust Vacuum Method, or DVM, to collect dust samples and determine lead concentration and lead loading.<sup>14</sup> The DVM is an in-line filter cassette device which uses a portable personal air sampler with an air flow rate of 2.5 liters/minute.<sup>14</sup> The DVM was used in both 1989 and 1998. A dust wipe sample, the standard method of sampling house dust, was collected only in 1998. The dust wipe sample was taken for comparison with the U.S. Department of Housing and Urban Development's National Lead Survey and the U.S. Environmental Protection Agency's Residential Lead Standard.<sup>15-16</sup>

In each housing unit, a composite dust sample was collected from three floors in each residence: a floor directly inside the entryway of the residence, the child's bedroom floor, and the floor in the main activity room (e.g., living room or kitchen). Dust lead loading ( $\mu\text{g}/\text{ft}^2$ ), dust arsenic loading ( $\mu\text{g}/\text{ft}^2$ ), dust lead concentration ( $\mu\text{g}/\text{g}$ ), and dust arsenic concentration ( $\mu\text{g}/\text{g}$ ) were measured with the DVM Sampler.

Lead content of interior and exterior painted surfaces was measured for each housing unit, using a portable x-ray fluorescence analyzer. For each residence, at least one wall and one trim measurement were made for the entry room, the child's bedroom, and the main activity room. Three exterior paint lead measures were taken for each residence. At each location, three readings were made and averaged. The condition of each surface was rated as either intact [1], worn [2], or deteriorated [3].

Two composite samples of surface soil were taken—one from foundation and one from yard locations. Three shallow core sub-samples of soil were collected on each side of the house around the perimeter of the foundation where bare soil was present (a maximum of 12 surface samples). The foundation sub-samples were then combined for a composite foundation sample. If bare soil was present, composite samples were also taken from a garden, child's play area, or other bare soil areas in the yard. All surface soil samples were taken at a depth of 2.5 inches, but only the top two centimeters of the core sample was retained for



analysis. A first-draw water sample was taken for each child enrolled.

### Laboratory analyses

Blood samples were analyzed for lead concentrations by Anodic Stripping Voltammetry (ASV).<sup>17</sup> All samples were split and analyzed in duplicate. Calibration of the ASV instrument was done by referencing against two individual standard curves. Approximately 30 unknowns were run between these two curves, which were combined and averaged. This composite curve forms the linear regression equation used to calculate the blood lead concentration of the samples analyzed between them. A minimum of five standards were used for each curve and consisted of bovine or whole human blood samples whose lead value had been previously determined by isotope-dilution mass spectroscopy (IDMS), the definitive method for lead determination. The method detection limit for blood lead by ASV in this laboratory was 1.0 µg/dL.

Interior dust vacuum samples were collected by using a personal air monitoring pump connected by Nalgene tubing to a three-piece air monitoring cassette with a 0.8 mm mixed cellulose ester filter.<sup>14</sup> The dust was carefully rinsed from each cassette into a pre-weighed beaker. The sample contents of each beaker were oven-dried to constant weight and the total weight of the dust was calculated. The entire sample was then digested and analyzed using a modification of NIOSH 7082. Samples were analyzed for lead using a 5000 or 5100 Perkin-Elmer Flame Atomic Absorption Spectrometer (FAAS). Samples were analyzed for arsenic using a 5100 Perkin-Elmer Graphite Furnace Atomic Absorption Spectrometer with Zeeman background correction (ZGFAAS). The method detection limit (MDL) was 1.0 µg for lead sample analysis and 0.1 µg for arsenic.

Dust wipe samples were collected following the HUD dust wipe collection procedure.<sup>15–16</sup> The wipe samples were carefully removed from their containers and digested using a HNO<sub>3</sub> and HCl hot plate digestion procedure.<sup>22</sup> The entire sample was used in this procedure. Samples were analyzed for lead using a 5000 or 5100 Perkin-Elmer Flame Atomic Absorption Spectrometer (FAAS). Samples were analyzed for arsenic using a 5100 Perkin-Elmer Graphite Furnace Atomic Absorption Spectrometer with Zeeman background correction (ZGFAAS). The method detection limit (MDL) for lead sample analysis was 5.0 µg for 1–2 wipe samples and 10 µg for samples containing 3–4 wipes. Samples that were below the FAAS detection limit were re-analyzed by GFAAS, which resulted in

greater sensitivity and a lower MDL (0.16 µg). The MDL for arsenic was 0.1 µg.

Soil and entry dust samples were oven-dried, weighed, and sieved to a fraction that contained particles of less than 250 µm. Aliquots were taken from this soil fraction, digested, and analyzed according to a modification of NIOSH method 7082. Samples were analyzed for lead as described above. Samples were analyzed for arsenic using a 5100 Perkin-Elmer Graphite Furnace Atomic Absorption Spectrometer with Zeeman background correction (ZGFAAS). The method detection limit (MDL) was 1.0 µg for lead sample analysis and 0.1 µg for arsenic.

Water samples were collected in 250 ml polyethylene containers with a polyethylene cap. These containers were supplied by the analytical lab and were certified to follow all U.S. EPA analyte specifications for metals analysis. Within seven days of collection, water samples were acidified with concentrated HNO<sub>3</sub> to a pH <2. Water samples were analyzed for lead using a 5100 Perkin-Elmer Graphite Furnace Atomic Absorption Spectrometer with Zeeman background correction (ZGFAAS) following EPA method 200.9. The calculated MDL for the water sample analysis was 0.5 ppb.

The University of Cincinnati laboratory that analyzed the samples was accredited for lead analysis by the American Industrial Hygiene Association and the National Lead Laboratory Accreditation Program.

### Statistical analyses

Descriptive statistics were calculated for all measured variables to examine their distributions for normality to determine whether particular variables should be log transformed. For all statistical analyses, blood lead levels and all environmental lead measurements were log transformed (base 10) to normalize the data. Scatter plots of log<sub>10</sub> blood lead (BPb) versus independent variables were examined to visually detect linear trends. In a bivariate analysis, Pearson correlation coefficients between log(BPb) and independent continuous variables were calculated, and *t*-tests were used to statistically test the association between log (BPb) and dichotomous variables.

Our primary hypothesis was to test whether soil abatement was associated with a greater than expected decline in blood lead concentrations among children 6 to 72 months of age, after adjustment for covariates significantly associated with blood lead concentration (*p* < 0.20). These variables included child's age, mouth-ing behaviors, year of study, and socioeconomic status. Socioeconomic status was calculated using the Hollings-

head 4 factor score. We also tested whether soil abatement was associated with a greater than expected decline in blood lead concentrations among children ages 6 to 36 months who were born after soil abatement was completed. This younger group provides a better estimate of the benefit of soil abatement for children who were most vulnerable to exposure (i.e., younger than 36 months of age) and who had not yet been exposed.

## RESULTS

### Population characteristics

For the survey conducted in 1989, the participation rate was 112 (90%) of the 128 randomly identified children living in the study area. For the survey conducted in 1998, the participation rate was 215 (70%) of 257 eligible children living in the same area. Of the 215 children who participated in the 1998 study, 17 (7.9%) were siblings and were excluded from the following analyses. Thus, there were 198 index children in the 1998 study. Population characteristics of children are shown by year of study and by abatement status (Table 1).

In 1989, the geometric mean blood lead concentrations of the 73 children who lived in homes with an average soil lead concentration exceeding 500 ppm and that were subsequently abated, was 5.6  $\mu\text{g}/\text{dL}$ ; 11% had a blood lead  $\geq 10 \mu\text{g}/\text{dL}$ . In contrast, the geometric mean blood lead concentration for the 39 children who lived in homes with an average soil lead concentration below 500 ppm that were not abated was 3.9  $\mu\text{g}/\text{dL}$ ; 2.6% had a blood lead  $\geq 10 \mu\text{g}/\text{dL}$  (Table 1). In 1998, the geometric mean blood lead concentration for the 167 children who lived in homes that had undergone soil abatement was 3.0  $\mu\text{g}/\text{dL}$ ; 1% had a blood lead  $\geq 10 \mu\text{g}/\text{dL}$ . The geometric mean blood lead concentration for the 31 children who lived in homes with yards that had not been abated was 2.6  $\mu\text{g}/\text{dL}$  ( $p=0.06$ ).

In 1989, geometric mean levels of lead-contaminated soil and interior dust and arsenic in soil and interior dust were significantly higher in and around housing that later underwent soil abatement ( $p<0.05$ ). Socioeconomic status differed by abatement status in both 1989 and 1998, but mouthing behaviors and age did not differ by abatement status (Table 1).

For children who were 6 to 72 months of age, the rate of decline in blood lead concentrations among children who lived in yards that underwent soil abatement was not significantly different than the decline among children who lived in unabated yards. After adjustment for child's age, mouthing behavior, socio-

economic status, and year of study, there was an estimated 2.3  $\mu\text{g}/\text{dL}$  decline in blood lead concentration associated with soil abatement for children who were 6 to 72 months of age ( $p=0.14$ ) (Table 2).

We next evaluated the reduction in blood lead concentration associated with soil abatement for different age groups. After adjustment, there was a 2.0  $\mu\text{g}/\text{dL}$  (non-significant) difference in the decline in blood lead concentration associated with soil abatement for children ages 36 to 72 months ( $p=0.73$ ). In contrast, the rate of decline in blood lead concentrations due to soil abatement was significant for children ages 6 to 36 months; for children in this age group, there was a 2.5  $\mu\text{g}/\text{dL}$  difference in the decline in blood lead concentration among children whose yards were abated compared with children whose yards were not abated ( $p=0.03$ ) after adjustment for age of children, mouthing behaviors, socioeconomic status, and year of study. This was equivalent to a 3.5  $\mu\text{g}/\text{dL}$  (95% confidence interval [CI] = 2.4 – 4.6  $\mu\text{g}/\text{dL}$ ) decline in blood lead concentration for every 1000 ppm reduction in soil lead concentration (Table 2).

There was a significant decline in environmental levels of lead and arsenic by abatement status. In the yards that had been abated, there was a dramatic decline in soil lead and soil arsenic concentration ( $p<0.001$ ). Indeed, in contrast to the 1989 study, there was no longer a significant difference in environmental levels of lead and arsenic, and foundation soil lead concentrations were significantly higher for the unabated homes. The absolute decline in soil lead concentration in the abated yards was 439  $\mu\text{g}/\text{g}$  greater than in the yards that were not abated (Table 3). The decline in soil lead concentration by abatement status remained statistically significant after adjusting for year (1989 vs. 1998) to account for secular trends associated with reductions in the use of leaded gasoline or other sources of lead contamination ( $p<0.001$ ) (Table 3). The absolute decline in lead concentration and loading in house dust associated with soil abatement was 252  $\mu\text{g}/\text{g}$  ( $p=0.0005$ ) and 7.3  $\mu\text{g}/\text{ft}^2$  ( $p=0.03$ ), respectively (Table 3). Soil abatement was associated with an 11  $\mu\text{g}/\text{g}$  decline in interior dust arsenic concentration ( $p=0.03$ ), but the 0.3  $\mu\text{g}/\text{ft}^2$  decline in arsenic dust loading was not statistically significant ( $p=0.12$ ).

To ensure that differences in mobility did not alter our findings, we examined the subjects' duration in their current residence for two to six months by study year and abatement status. In 1989, 24 (33%) of 73 children in the group whose yards underwent soil abatement lived in their current residence for less than six months compared with 11 (28%) of 39 children whose

**Table 1. Comparisons of children's characteristics in the Midvale Abatement Study, by year of study and soil abatement status**

Characteristic	Soil abatement status 1989		p value	Soil abatement status 1998		p value
	Intervention group (n = 73)	Control group (n = 39)		Intervention group (n = 167)	Control group (n = 31)	
	Geometric mean (95% CI)	Geometric mean (95% CI)		Geometric mean (95% CI)	Geometric mean (95% CI)	
Blood lead (µg/dL)	5.6 (4.9, 6.3)	3.9 (3.2, 4.7)	0.0001	3.0 (2.8, 3.2)	2.6 (2.2, 3.1)	0.06
% Blood lead > 10 (µg/dL)	8 (11%)	1 (3%)	0.12	2 (1%)	0	0.67
Age (months) (mean)	33.4 (29.1, 37.8)	34.5 (28.5, 40.5)	0.77	35.2 (32.4, 38.0)	36.1 (28.6, 43.6)	0.80
Floor dust lead (µg/ft <sup>2</sup> )	14.6 (11.8, 18.0)	7.0 (5.4, 9.0)	0.0001	1.5 (1.3-1.8)	1.2 (0.9, 1.6)	0.34
Floor dust lead (µg/g)	551 (476, 639)	284 (249, 325)	0.0001	142 (129, 156)	127 (113, 143)	0.34
Floor dust arsenic (µg/ft <sup>2</sup> )	0.7 (0.6, 0.9)	0.4 (0.3, 0.5)	0.0001	0.1 (0.1, 0.14)	0.1 (0.06, 0.13)	0.24
Floor dust arsenic (µg/g)	28 (24, 33)	15 (13, 17)	0.0001	11 (10, 13)	9 (7, 13)	0.34
Interior paint lead	2.5 (2.2, 2.8)	1.9 (1.7, 2.0)	0.004	0.3 (0.3, 0.4)	0.3 (0.2, 0.3)	0.30
Exterior paint lead	2.2 (1.9, 2.6)	1.6 (1.5, 1.8)	0.006	0.4 (0.3, 0.5)	0.3 (0.2, 0.5)	0.49
Foundation soil lead (µg/g)	542 (466, 631)	144 (118, 175)	0.0001	54 (45, 65)	95 (64, 141)	0.02
Foundation soil arsenic (µg/g)	50 (44, 56)	19 (17, 22)	0.0001	13 (12, 14)	14 (11, 19)	0.32
Water lead (µg/L)	1.4 (1.2, 1.6)	1.5 (1.2, 2.0)	0.35	1.2 (1.1, 1.3)	1.2 (1.0, 1.4)	0.88
Bare soil present at residence	59 (81%)	29 (74%)	0.43	100 (60%)	16 (52%)	0.39
Hollingshead score (mean)	22.9 (20.7, 25.2)	31.8 (27.8, 35.9)	0.0001	24.6 (23.0, 26.2)	29.2 (25.2, 33.1)	0.03
Mouthing index (mean)	7.3 (6.0, 8.6)	6.5 (5.0-8.0)	0.40	4.2 (3.6, 4.9)	4.5 (2.8, 6.2)	0.73

CI=confidence interval

yards did not have soil abatement. In 1998, 26 (16%) of 160 children in the group whose yards were abated lived in their current residence for less than six months compared with six (20%) of 30 children whose yards did not have soil abatement.

## DISCUSSION

We conclude that soil abatement was associated with a significantly greater reduction in blood lead concentrations than expected among children ages 6 to 36 months who had not been exposed to lead-contaminated yards in early childhood. In contrast, soil abate-

**Table 2. Estimated change in blood lead concentrations ( $\mu\text{g}/\text{dL}$ ) associated with soil abatement and estimated increase in blood lead concentration ( $\mu\text{g}/\text{dL}$ ) per 1000 ppm increase in soil lead concentration**

Age group (months)	Unadjusted absolute decline in blood lead concentration (95% CI)	Adjusted absolute decline in blood lead concentration (95% CI)	Percent decline in blood lead concentration (95% CI)	p value <sup>a</sup>	Change in blood lead in concentration per 1000 ppm in soil lead (95% CI)
6 to 72	2.6 (2.0, 3.3)	2.3 (1.8, 2.9)	42.8 (33.2, 55.0)	0.14	2.1 (1.6, 2.6)
36 to 72	2.2 (1.5, 3.3)	2.0 (1.3, 3.0)	39.7 (26.4, 59.9)	0.73	1.7 (1.2, 2.3)
6 to 36	2.8 (2.0, 4.0)	2.5 (1.8, 3.5)	45.4 (32.8, 62.9)	0.03	3.5 (2.4, 4.6)

<sup>a</sup>p value for interaction of year by remediation status, adjusted for age, mouthing behavior score, and socioeconomic status.

ment was not associated with a significantly greater reduction in blood lead concentrations than expected for children ages 36 to 72 months. We further conclude that abatement of residential soil resulted in a significant decline in levels of lead and arsenic in soil, and levels of lead in interior house dust.

Soil abatement has been conducted extensively in Superfund sites to reduce human exposure to lead, arsenic, and other contaminants. This is the first controlled study to measure the reduction in blood lead concentration associated with soil abatement for children who live in former mining, milling, or smelting communities. Although the cost of soil abatement is high, there is emerging evidence that the cognitive deficits occur at blood lead levels below 10  $\mu\text{g}/\text{dL}$ .<sup>18-20</sup> Indeed, these studies suggest that there is no threshold for lead-associated cognitive deficits in children.<sup>18-20</sup>

The observed decline in blood lead concentration among children ages 6 to 36 months was consistent with earlier research. In a pooled analysis of 12 lead-exposed cohorts, the mean increase in blood lead levels was 3.8  $\mu\text{g}/\text{dL}$  for every 1000 ppm increase in soil lead levels.<sup>21</sup> In contrast, the decline in blood lead concentration was higher than those reported from a randomized, controlled trial of soil abatement among children in Boston.<sup>22,23</sup> In a two-year follow-up of soil

abatement, the investigators observed a 1.12 to 1.35  $\mu\text{g}/\text{dL}$  decline in blood lead levels for every 1000 ppm reduction in soil lead concentration.<sup>26</sup> This lower estimate was probably due to the older average age of children in the Boston study compared with this present study and the pooled analysis.<sup>21</sup> The body burden of lead is higher in older children and they have outgrown many high-risk mouthing behaviors. Thus, older children are less likely to benefit from a reduction in environmental lead exposure. Indeed, the findings of the Boston study were similar to the results in children ages 36 to 72 months (Table 3). There were no significant effects of soil abatement in the two other cities that composed the EPA-funded three-city soil abatement study, but the exterior source of lead in the latter two studies was more properly defined as dust, not soil.<sup>24,25</sup>

This study had several limitations. First, it was an observational study involving two distinct cohorts of children. Soil abatement was determined by soil lead concentrations  $>500$  ppm, not by random assignment. Despite the absence of any obvious difference in characteristics of children living in soil-abated yards compared with non-abated yards (with the exception of socioeconomic status), unrecognized differences in population characteristics may exist that bias the find-

**Table 3. Geometric mean decline in environmental exposures to lead and arsenic in house dust and soil**

	Intervention group	Control group	Absolute difference in decline	Adjusted p value
Floor dust lead ( $\mu\text{g}/\text{g}$ )	409	157	252	0.0005
Floor dust lead ( $\mu\text{g}/\text{ft}^2$ )	13.1	5.8	7.3	0.03
Foundation soil lead ( $\mu\text{g}/\text{g}$ )	488	49	439	0.0001
Floor dust arsenic ( $\mu\text{g}/\text{ft}^2$ )	0.6	0.3	0.3	0.12
Floor dust arsenic ( $\mu\text{g}/\text{g}$ )	17	6.0	11	0.03
Foundation soil arsenic ( $\mu\text{g}/\text{g}$ )	37	5.0	32	0.0001

ings of these analyses. Furthermore, the study does not demonstrate that soil abatement led to a decline in blood lead concentrations of children followed longitudinally. Instead, we evaluated the effect of soil abatement in a new cohort of children who resided in housing with either abated or non-abated properties. Still, these data arguably provide the most precise estimate of the contribution of lead intake from residential soil because the children in Midvale were exposed to minimal levels of leaded paint and water. Moreover, we measured the effect of soil abatement in a new cohort of children before they were unduly exposed. Thus, our estimates do not have to be adjusted for other residential sources of lead intake or existing body stores of lead.

## CONCLUSIONS

The results of this study suggest that soil abatement in yards with lead concentration exceeding 500 ppm resulted in a 2.5 µg/dl reduction in blood lead concentration among children ages 6 to 36 months. In contrast, there was no significant difference in the decline in mean blood lead concentration for older children who lived on abated properties compared with those who lived on non-abated properties. The analyses for children ages 6 to 36 months are arguably more relevant because these children represent an unexposed cohort, whereas many of the children older than 36 months were exposed to lead-contaminated soil prior to abatement. These data help refine the estimated contribution of lead-contaminated soil to children's lead intake and provide information for communities and federal agencies to make informed decisions about the benefits of soil abatement.

The authors thank Robert Bornschein, William Menrath, William Brattin, Joanne Grode, Diana Hammer, Tracy Hammon, Mary Goldade, Ken White and the field staff of IHI, and the Midvale Study team for their contributions to this study.

This study was funded by the United States Environmental Protection Agency.

## REFERENCES

1. Hertzman C, Ward H, Ames N, Kelly S, Yates C. Childhood lead exposure in Trail revisited. *Can J Public Health* 1991;82:385-91.
2. Baker EL Jr, Hayes CG, Landrigan PJ, Handke JL, Leger RT, Housworth WJ, Harrington JM. A nationwide survey of heavy metal absorption in children living near primary copper, lead, and zinc smelters. *Am J Epidemiol* 1977;106:261-73.
3. Landrigan PJ, Gehlbach SH, Rosenblum BF, Shoultz JM, Candelaria RM, Barthel WF, et al. Epidemic lead absorption near an ore smelter. The role of particulate lead. *N Engl J Med* 1975;292:123-9.
4. Kimbrough R, LeVois M, Webb D. Survey of lead exposure around a closed lead smelter. *Pediatrics* 1995;95:550-4.
5. Gulson BL, Mizon KJ, Korsch MJ, Howarth D. Non-ore body sources are significant contributors to blood lead of some children with low to moderate lead exposure in a major lead mining community. *Sci Total Environ* 1996;181:223-30.
6. Gulson BL, Howarth D, Mizon KJ, Law AJ, Korsch MJ, Davis JJ. Source of lead in humans from Broken Hill mining community. *Environ Geochem Health*. 1994; 16:19-25.
7. Cook M, Chappell WR, Hoffman RE, Mangione EJ. Assessment of blood lead levels in children living in a historic mining and smelting community. *Am J Epidemiol* 1993;137:447-55.
8. Hilts SR. A co-operative approach to risk management in an active lead/zinc smelter community. *Environ Geochem and Health* 1996;18:17-24.
9. Angle CR, Manton WI, Stanek KL. Stable isotope identification of lead sources in preschool children—the Omaha Study. *J Toxicol Clin Toxicol* 1995;33:657-62.
10. Angle CR, McIntire MS. Environmental lead and children: the Omaha Study. *J Toxicol Environ Health* 1979; 5:855-70.
11. Bornschein R, Clark S, Pan W, Succop P. Midvale Community Lead Study. *Chemical Speciation Bioavailability* 1991;3:149-162.
12. Pirkle JL, Brody DJ, Gunter EW, Kramer RA, Paschal DC, Flegal KM, Matte TD. The decline in blood lead levels in the United States. The National Health and Nutrition Examination Surveys (NHANES). *JAMA* 1994;272: 284-91.
13. Agency for Toxic Substances and Disease Registry. The nature and extent of lead poisoning in children in the United States: a report to congress, 1988. Atlanta: Department of Health and Human Services (US), Public Health Service.
14. Que Hee SS, Peace B, Clark CS, Boyle JR, Bornschein RL, Hammond PB. Evolution of efficient methods to sample lead sources, such as house dust and hand dust, in the homes of children. *Environ Res* 1985;38:77-95.
15. Lanphear BP, Emond M, Jacobs DE, Weitzman M, Tanner M, Winter NL, et al. A side-by-side comparison of dust collection methods for sampling lead-contaminated house-dust. *Environ Res* 1995;68:114-23.
16. Department of Housing and Urban Development (US). Guidelines for the evaluation and control of lead-based paint hazards in housing. Washington: Government Printing Office (US); 1995.
17. Roda S, Greenland R, Bornschein R, Hammond P. Anodic stripping voltammetry procedure modified for improved accuracy of blood lead analysis. *Clin Chem* 1988;34:563-7.



18. Schwartz J. Low-level lead exposure and children's IQ: a meta-analysis and search for a threshold. *Environ Res* 1994;65:42-55.
19. Lanphear BP, Dietrich K, Auinger P, Cox C. Cognitive deficits associated with blood lead concentrations <10 µg/dl in US children and adolescents. *Public Health Rep* 2000;115:521-9.
20. Lanphear BP, Canfield R, Henderson C, Cory-Slechta D, Cox C. Environmental exposure to lead and children's intelligence at blood lead concentrations below 10 micrograms per deciliter. *Pediatr Res* 2001;49:16A.
21. 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.
22. Weitzman M, Aschengrau A, Bellinger D, Jones R, Hamlin JS, Beiser A. Lead-contaminated soil abatement and urban children's blood lead levels. *JAMA* 1993;269:1647-54.
23. Aschengrau A, Beiser A, Bellinger D, Copenhafer D, Weitzman M. The impact of soil lead abatement on urban children's blood lead levels: phase II results from the Boston Lead-in-Soil Demonstration Project. *Environ Res* 1994;67:125-48.
24. Farrell KP, Brophy MC, Chisolm JJ, Rohde CA, Strauss WJ. Soil lead abatement and children's blood lead levels in an urban setting. *Am J Public Health* 1998;88:1837-9.
25. Environmental Protection Agency (US). Urban soil lead abatement demonstration project, Volume I. 1996, EPA/600/P-93/001aF, Washington, D.C.