decreasing body mass index must be chiefly metabolic and/or due to anorexia. Kaposi's sarcoma, if disseminated, can involve the gastrointestinal tract, but this condition is also uncommon, making it unlikely that malabsorption mediates this relation.

The independent relation of a low CD4+ cell count with a decrease in body mass index raises some intriguing possibilities. The first is that advanced HIV infection itself may contribute directly to weight loss, even in the absence of clinically apparent opportunistic infections. However, it seems unlikely that patients with low CD4+ counts can remain completely free of opportunistic infections. A more likely explanation is that a low CD4+ count in our study is a marker for subclinical opportunistic infections (e.g., Microsporida, Cryptosporidium, cytomegalovirus) or neoplasia (e.g., lymphoma) that can lead to weight loss.

Oral thrush will almost certainly lead to anorexia (due to pain on chewing and swallowing). In addition, this condition is often associated with esophageal candidiasis, which is likely to exacerbate anorexia. In our study, oral thrush was also a stronger predictor of weight loss than diarrhea, although not of the same magnitude as fever or a CD4+ lymphocyte count of $<100/\mu l$. Herpes zoster was a relatively infrequent opportunistic infection in our cohort, but it was also inversely associated with change in body mass index.

In summary, these data suggest that fever, a low CD4+ lymphocyte count, oral thrush, and AIDS all contribute importantly to HIV-related wasting. Diarrhea, in comparison, was less important when the effects of these other factors were taken into account. Further studies are planned to collect data on levels of caloric and nutrient intake preceding weight loss to determine the relative importance of anorexia as a cause of HIV-related wasting.

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Assessment of Blood Lead Levels in Children Living in a Historic Mining and Smelting Community

Magdalena Cook,[1] Willard R. Chappell,[2] Richard E. Hoffman,[1] and Ellen J. Mangione[1]

Lead poisoning in childhood is an important public health problem, and thus, it is important to determine how children are exposed to lead. In 1987, the authors conducted an exposure assessment and blood lead screening for children aged 6–71 months living in Leadville, Colorado. High levels of lead had been found in the soil as a result of both past mining and smelting activities and natural mineralization. Blood was collected from each child for lead analysis, and behavioral characteristics were identified through an interview with a parent or guardian. Three sources of exposure to lead were associated with blood lead levels: lead in a core sample taken from the backyard of the family's home, lead brought home on the clothes of a miner, and lead from soldering in the home. Two pathways of exposure were associated with blood lead levels: the child swallowing things other than food, and taking food or a bottle outside to play. Multivariate regression using these variables found effect modification by age. For children aged 6–36 months, only sources of exposure were independent predictors of blood lead levels, while in children aged 37–71 months, a pathway of exposure in addition to sources of exposure independently predicted blood lead levels. *Am J Epidemiol* 1993;137:447–55.

child, preschool; environmental exposure; lead poisoning

The vulnerability of young children to lead toxicity has been increasingly recognized in recent years. In 1985, the Centers for Disease Control lowered its definition of an elevated blood lead level from 30 μg/dl to 25 μg/dl. In response to epidemiolog and clinical studies published between 19 and 1990 (1–9) demonstrating adver health effects at blood levels as low as 10 μ dl, the Centers for Disease Control recor mended in October 1991 (1) that childr with a blood lead level of ≥15 μg/dl recei nutritional and educational interventi and frequent monitoring of blood lead le els, and that when a large proportion children in a community have blood le levels in the range of 10–14 μg/dl, comm nity-wide, primary lead poisoning preve tion activities be initiated.

Leadville, Colorado, is the site of a histor mining district. Mining began with the di covery of placer gold in 1859, and gol silver, and zinc were mined, milled, an smelted in the community for over 1(years. An estimated 1 million tons of le concentrate were produced from 1860 1966. The first smelter was built in 187 and the last stopped operating in 196

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Abbreviation: ppm, parts per million.

[1] Division of Disease Control and Environmental Epidemiology, Colorado Department of Health, Denver, Colorado.

[2] Department of Physics, Environmental Sciences Program, University of Colorado at Denver, Denver, Colorado.

Reprint requests to Magdalena Cook, Manager, Injury Epidemiology Program, Division of Disease Control and Environmental Epidemiology, DCEED-IE-A3, Colorado Department of Health, 4300 Cherry Creek Drive South, Denver, CO 80222-1530.

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Waste materials from the mines, mills, and smelters were deposited in close proximity to or within present residential areas. At least 50 percent of the ore mined in the Leadville district contained lead carbonate (cerusite), lead sulfate (anglesite), and lead oxide (massicot); in addition, smelter wastes contained oxidized forms of lead.

This study was initiated because of concern that children in Leadville, a community with an estimated population of 2,600 in 1987, were at high risk of lead poisoning. A sampling of residential soils for lead was conducted in Leadville in 1986 as part of a remedial investigation for the Comprehensive Environmental Response Compensation and Liability Act (also known as "Superfund"). More than 60 percent of the surface soils had lead concentrations of >1,000 parts per million (ppm) and more than 80 percent had concentrations of >500 ppm (range, 110–12,000 ppm). The 1985 Centers for Disease Control statement on preventing childhood lead poisoning noted that "lead in soil and dust appears to be responsible for blood lead levels in children increasing above background levels when the concentration in the soil or dust exceeds 500–1,000 ppm" (10, p. 7).

In response to the environmental survey, the Colorado Department of Health, the University of Colorado at Denver, and the Agency for Toxic Substances and Disease Registry jointly designed and conducted an assessment of exposure including screening of blood lead levels for children aged 6–71 months in 1987. In this report, we describe the results of blood lead screening and assess both children's behavior and potential sources of lead as contributors to blood levels.

MATERIALS AND METHODS

Eligible participants were children aged 6–71 months who had resided in the community for at least 3 months preceding the study. We conducted a comprehensive, door-to-door census to identify all eligible children. The demographic and behavioral characteristics of each participating child and the socioeconomic status, education, and occupation of the child's parents or guardians were determined by interviewing a parent or guardian. After written consent was obtained from the parents or guardians, two venous blood samples of a minimum of 1.75 ml were collected from each participating child. Lead analysis by graphite furnace atomic absorption spectroscopy was performed by the Division of Environmental Health Laboratory Sciences of the Center for Environmental Health and Injury Control, Centers for Disease Control.

The following soil and dust samples were collected for lead level analysis by contract laboratories of the US Environmental Protection Agency: a composite sample of floor dust from the main entrance, the subject's bedroom, and the room where the subject spent most of his or her waking time (11); a composite sample of windowsill dust from all windowsills in the subject's bedroom and the subject's main room (11); two exterior surface scrapings from exposed dirt surfaces, one from the subject's primary outside play area and the other from near the main entrance; and core samples of exterior soil from the front and back yards.

Lead in paint in the interior and exterior of the house was measured using a portable x-ray fluorescence analyzer (XK-3 Lead-in-Paint Analyzer, Princeton Gamma Tech, Inc., Princeton, New Jersey). The sites for x-ray fluorescence analyzer readings were painted surfaces; with particular attention paid to any surfaces chipped or in poor condition. Occasional pieces of painted furniture, such as cribs or chairs, were also sampled. Typically, six to eight interior sites and four to six exterior sites were sampled. When readings deviated more than 0.5 mg/cm^2 from the calibration standard, the measurement was considered invalid and excluded. Elevated levels of lead in paint were defined as readings of >6.0 mg/cm^2.

A single sample of tap water was collected from each participating household. After the tap water was allowed to run for 10 seconds, an acid-washed, 500-ml polyethylene bottle containing 1 ml of triply distilled concentrated nitric acid was filled. The samples were analyzed by the Colorado Department of Health, Division of Laboratories.

Statistical analyses were performed using log-transformed levels of both blood lead and soil lead. Multivariate linear regression was used to examine and control for the independent contribution of various sources and pathways of lead exposure. The variables included in regression models were formulated using the results from univariate analyses and stepwise forward regression. Biologically plausible pathways and sources of exposure were included in the models. Stepwise forward regression was used to identify which environmental samples independently contributed to blood lead levels. Since there was no a priori evidence of a point source of airborne lead, we assumed that ingestion was the only pathway of exposure. In any given multivariate model, only one measure of a particular behavior was included. For example, models did not include both swallowing things other than food and taking food or a bottle outside to play. To assess for effect modification of age on blood lead level, separate models were constructed for children aged 6–36 months and children aged 37–71 months.

The percentage of change in the blood lead levels was calculated using results from the regression modeling. Values of 0 for a response of "no" and 1 for a response of "yes" were used for dichotomous variables, and values of 100 and 1,100 ppm were used for environmental sampling variables. The percentage of change reflects the change in the blood lead levels and not the log transformed levels.

RESULTS

Two hundred thirty-nine children 6 months of age from 174 households w identified by the census, and 150 (63 cent) children from 105 households par pated in the study.

Environmental sampling

Soil and dust samples collected aro the home from 105 households were c gorized by sample type and sampling l tion. The mean values ranged from 1,00 2,453 ppm. The highest mean lead valu 2,453 ppm was found in scrape sample the entryway, but the highest single envi mental sample was a composite dust san from a windowsill measuring 27,900 (table 1).

One of the 105 water samples (0.9 perc had lead concentrations above the leve detection of 5 μg/L. The concentratio lead in this sample was 13 μg/L.

Fifty-four of 105 households (51.4 cent) representing 49.3 percent of the pa ipants (74 of 150) had valid x-ray fluo cence analyzer measurements that were in the analysis. Lead levels were elevate 20 of 218 interior samples (9.2 percent) 21 of 146 exterior paint samples (14.4 cent).

Blood lead levels

The arithmetic mean blood lead level 10.1 μg/dl ± a standard deviation of (range, 0.5–30.1 μg/dl); the geometric m was 8.7 μg/dl ± 1.79. There were no sig

TABLE 1. Mean lead levels in environmental samples collected around the home: Leadville, Colorado, 1987

Type of sample and sampling location	No. tested	Lead levels in environmental samples (ppm*)				
		Arithmetic			Geometric	
		Mean	SD*	Range	Mean	SD*
Front yard core	104	1,674	1,460	49–7,400	1,110	2.7
Backyard core	103	1,884	3,133	27–27,800	1,034	3.1
Play area scraping	74	1,710	1,849	3–8,620	892	3.9
House entryway scraping	104	2,453	1,998	203–13,900	1,876	2.1
Windowsill dust	105	2,125	4,412	30–27,900	871	3.8
Floor dust	104	1,000	1,155	8–11,100	726	2.4

* SD, standard deviation; ppm, parts per million.

TABLE 2. Mean blood lead levels and percentage by blood lead group, by age group: Leadville, Colorado, 1987

Age group (months)	Blood lead levels (μg/dl)					Percentage of children with blood lead levels:		
	Arithmetic			Geometric		≥10 μg/dl	≥15 μg/dl	≥25 μg/dl
	Mean	SD*	Range	Mean	SD			
6–36	10.4	5.8	0.5–25.4	8.7	2.0	44.1	16.2	1.5
37–71	9.9	5.4	3.3–30.1	8.8	1.6	37.8	13.4	2.4
6–71	10.1	5.6	0.5–30.1	8.7	1.8	40.7	14.7	2.0

* SD, standard deviation.

icant differences in mean blood lead levels between males and females (10.3 vs. 10.0 μg/dl, $p > 0.05$) or between children aged 6–36 months as compared with children aged 37–71 months (10.4 vs. 9.9 μg/dl, $p > 0.05$) (table 2).

Overall, the percentages of children with blood lead levels greater than or equal to 10 μg/dl, 15 μg/dl, and 25 μg/dl were 40.7 percent, 14.7 percent, and 2.0 percent, respectively. The percentage of the 68 children aged 6–36 months with blood lead levels of ≥10 μg/dl, ≥15 μg/dl, and ≥25 μg/dl were 44.1 percent, 16.2 percent, and 1.5 percent, respectively; for the 82 children aged 37–71 months, these percentages were 37.8 percent, 13.4 percent, and 2.4 percent (table 2).

Exposure assessment

The log lead in the outdoor soil samples and in the scrape sample from the house entryway was associated with the log blood lead levels ($p < 0.001$) (table 3). There was no association between blood lead levels and the indoor dust samples. The largest percentage of change in blood lead values was found in the backyard core sample. A separate forward stepwise regression of the environmental sampling results found that after the backyard core sample was adjusted for, no other environmental sampling variables were associated with blood lead levels ($p < 0.001$, $R^2 = 0.19$). Thus, the result for the backyard core sample was used as the source of exposure to soil lead in the multivariate regression models.

No association was found between the x-ray fluorescence analyzer measurements of lead in paint and blood lead levels when the latter were compared with each of the 24 site measurements. Comparison of mean blood lead levels in children with valid x-ray fluorescence analyzer data (10.5 μg/dl ± 5.8) with those of children with invalid x-ray fluorescence analyzer data (9.5 μg/dl ± 5.0) showed no significant difference ($p > 0.10$).

Other variables measuring sources of exposure to lead in which the mean log blood lead values differed were defined as follows:

1. Whether a parent in the household who worked as a miner within the last 3 months (18.0 percent of 150) wore work clothes home (mean blood lead for those responding "yes" > mean blood lead for those responding "no") or showered before coming home ("no" > "yes"); this variable was labeled "mine."
2. Whether any household member soldered electronic equipment or pipes in the home ("yes" > "no"); this variable was labeled "solder."

The variable "mine" contributed to a larger percentage of change in blood lead levels than did "solder" (40.5 percent vs. 22.1 percent, respectively) (table 3).

Behavioral variables were viewed as indicating the pathway rather than the source of exposure. Two behavioral variables had statistically significant differences in mean log blood lead levels:

1. Whether a child took food or a bottle outside to play ("yes" > "no"); this variable was labeled "bottle."
2. Whether the child swallowed things other than food ("yes" > "no"); this variable was labeled "swallow."

TABLE 3. Percentage of change in blood lead level by predictor variable for children aged 6–71 months: Leadville, Colorado, 1987

Variable	Percentage of change in blood lead level per unit change in variable*	95% CI†	p value
Exposure			
Environmental samples			
Front yard core	61.5‡	55.6–67.7	<0.001
Backyard core	82.1‡	73.7–90.9	<0.001
Play area scraping	43.3‡	39.3–47.4	<0.001
House entryway scraping	69.5‡	59.3–80.3	<0.001
Windowsill dust	7.5‡	7.0–7.9	0.372
Floor dust	18.3‡	16.0–20.6	0.189
Other§			
Mine	40.5‖	28.0–54.2	0.013
Solder	22.1‖	17.0–27.5	0.068
Behavior¶			
Swallow	53.7‖	36.6–73.0	0.003
Bottle	40.5‖	31.4–50.2	0.001
Other#			
Dogs or cats	20.9‖	16.9–25.0	0.047
Ground type	19.7**	15.6–24.0	0.068

* Calculated from the results of linear regression analyses.
† CI, confidence interval.
‡ Percentage of change in blood lead between environmental values of 100 and 1,100 parts per million.
§ Mine, a miner in the household who did not shower or change clothes before coming home; solder, electronic equipment or pipes were soldered in the home.
‖ Percentage of change in blood lead between 0 = "no" and 1 = "yes."
¶ Swallow, the child swallowed things other than food; bottle, the child took food or a bottle outside to play.
# Dogs or cats, whether dogs or cats go in and out of the house; ground type, type of ground played on.
** Percentage of change in blood lead between 0 = concrete or grass and 1 = dirt.

Swallowing things other than food resulted in a larger percentage of increase in blood lead levels (53.7 percent) than taking food or a bottle outside to play (40.5 percent) (table 3).

Other variables associated with significant differences in mean log blood lead levels in the univariate analyses included the type of ground the child played on outside (dirt > concrete or grass) and whether dogs or cats went in and out of the house ("yes" > "no"). The type of ground played on and whether dogs or cats went in and out of the house were indirect measures of exposure to the lead in the soil, and were not included in the multivariate models.

Two multivariate linear models were constructed using each source of exposure to lead (log backyard core soil lead, parent being a miner and wearing work clothes home or not showering before comin home, and parent soldering) but differen behavioral pathways of exposure. Model in table 4 had the highest value of R^2 for a children aged 6–71 months (R^2 = 0.29 Parental occupation (labeled "mine"), swal lowing things other than food, and the so lead level all independently contributed t blood lead levels. Soldering in the home wa not a significant predictor.

When model 1 was stratified by age (6–3 months and 37–71 months), the variabl contributing to blood lead levels differe For children aged 6–36 months, only the lo soil lead level contributed independently whereas swallowing things other than foo and the log soil lead level contributed signi icantly to blood lead levels for children age 37–71 months.

Taking food or a bottle outside to pla

TABLE 4. Percentage of change in blood lead level* by two multivariate regression models for children aged 6–71 months: Leadville, Colorado, 1987

Variable	6–36 months			37–71 months			6–71 months		
	Percentage of change in blood lead level per unit change in variable	95% CI†	*p* value	Percentage of change in blood lead level per unit change in variable	95% CI	*p* value	Percentage of change in blood lead level per unit change in variable	95% CI	*p* value
Model 1									
Swallow‡	16.2§	10.2–22.5	0.418	120.3	67.7–191.2	<0.001	46.2	33.7–59.9	0.003
Mine‖	47.7§	24.8–74.7	0.080	28.4	19.9–37.5	0.079	35.0	25.0–45.7	0.019
Solder¶	10.5§	7.3–13.8	0.519	2.0	1.5–2.5	0.879	3.0	2.4–3.6	0.770
Log soil lead	100.4#	84.7–117.5	<0.001	43.3	39.3–47.4	<0.001	73.6	66.2–81.3	<0.001
Model 2									
Bottle**	29.7§	20.8–39.3	0.070	27.1	20.1–34.5	0.049	28.4	22.9–34.2	0.008
Mine	22.1§	14.7–30.0	0.211	24.6	16.8–32.9	0.154	20.9	16.1–26.0	0.090
Solder	25.9§	19.2–32.9	0.057	−7.6	−5.8 to −9.5	0.518	8.3	6.8–9.9	0.338
Log soil lead	54.0#	48.8–59.3	<0.001	50.3	44.4–56.4	<0.001	50.3	46.8–54.0	<0.001

* Calculated from results of multivariate regression analyses.
† CI, confidence interval.
‡ Swallow, the child swallowed things other than food.
§ Percentage of change in blood lead between 0 = "no" and 1 = "yes."
‖ Mine, a miner in the household who did not shower or change clothes before coming home.
¶ Solder, electronic equipment or pipes were soldered in the home.
# Percentage of change in blood lead between environmental values of 100 and 1,100 parts per million.
** Bottle, the child took food or a bottle outside to play.

was the behavioral pathway of exposure used in model 2 in table 4. For all children aged 6–71 months, taking food or a bottle outside to play and log soil lead were significant independent contributors to the log blood lead level ($R^2 = 0.27$) for this model. For the regression restricted to children 6–36 months of age, only the soil lead value was a significant predictor, but for the model restricted to children 37–71 months of age, the log soil lead level and taking food or a bottle outside to play were independently associated with the log blood lead level.

Model 1 was used to derive the slope of the line to calculate the increment of increase in the blood lead levels per 1,000 increment increase in soil lead levels. For the age groups 6–36 months, 37–71 months, and 6–71 months, the slope values were 4.7, 2.7, and 3.7 μg/dl, respectively.

DISCUSSION

This study was designed to determine whether a significant proportion of Leadville children had elevated levels of blood lead and, if so, what the important source(s) and pathway(s) of exposure were. The answer to the first question has changed over time. When the preliminary results were released in April 1990, the Centers for Disease Control definition of elevated blood lead had changed to ≥10 μg/dl, and 40.7 percent of the participants had elevated levels. In 1989, the Department of Health conducted surveys of children attending public health clinics in three Colorado communities in which there were no mining or smelting operations, and the proportion of children with blood lead levels ≥10 μg/dl was less than 9 percent in all three. When comparing these surveys with that of Leadville, however, it should be noted that they were conducted 2 years later and it would be expected that blood lead levels would be somewhat lower, due to the drop in lead in gasoline. There is no question, however, that based on the October 1991 Centers for Disease Control definition Leadville children are at increased risk of exposure to lead.

We used parental responses to several questions about the child's behavior to indicate how a child might be ingesting lead and to investigate seven sources of lead: soil and dust contamination, lead-based paint, drinking water, parental occupation, soldering, food served in clay pottery, and food stored in lead-soldered cans. Our findings were that two sources of lead (parental occupation and the level of lead in the soil) independently predicted blood lead levels, depending upon the child's age. For children aged 6–36 months and children aged 37–71 months, only the level of lead in the soil was an independent predictor. For the total group of children aged 6–71 months, the independent sources of exposure to lead were parental occupation and the level of lead in the soil. The association of the log of the soil lead from backyard core samples with the log of the blood lead level was found in both models tested. Soil lead contributed to a greater percentage of increase in the lead in the blood of children aged 6–36 months than that of children aged 37–71 months (100.4 percent vs. 43.3 percent).

There were two behaviors independently associated with increasing blood lead levels in children 6–71 months of age: 1) swallowing things other than food and 2) taking food or a bottle outside to play. Associations with these variables also varied by age. Presumably, these variables reflected pathways by which the soil lead was ingested.

X-ray fluorescence analyzer data were available for 49.3 percent of the participants. Thus, it is possible that the lack of association between lead-based paint and blood lead levels was due to a bias toward low levels of lead-based paint in homes with valid x-ray fluorescence analyzer measure-

association between blood lead levels and lead-based paint, one would expect children in the group with valid x-ray fluorescence analyzer measurements to have lower blood lead levels than children in the group with invalid x-ray fluorescence analyzer measurements. This was not the case, however, and there was no statistical difference between the mean log blood lead levels of those for whom x-ray fluorescence analyzer data were available and those for whom it was not.

Other studies have described an association between indoor dust lead levels and blood lead levels (12–14). Such an association was not found in this study, perhaps because environmental scientists had difficulty collecting indoor dust. This may have been due to study participants cleaning the house before the scheduled visit.

Our study did not find an association between blood lead levels and lead in drinking water. We collected water samples at whatever time the home visit was scheduled and did not collect first-draw morning samples, which would have reflected the highest lead concentrations in the drinking water of each home. It is possible that first-draw samples would have had higher lead concentrations. The child, however, would have had to consume this water before it was ingested by another family member or used for some other purpose.

The contribution to blood lead levels made by different pathways and sources of exposure varied by age, as has been shown in other studies (15, and W. R. Chappell et al., Department of Physics, Environmental Sciences Program, University of Colorado at Denver, Denver, Colorado, unpublished manuscript). In this study, only the soil lead contributed independently to elevating the blood lead levels of the younger children (6–36 months). For older children (37–71 months), however, both behavioral pathways of exposure (in this study, swallowing things other than food or taking a bottle or food outside to play) were important in addition to the soil lead level for the elevation of blood lead levels. These data would suggest developing different strategies for the prevention of lead exposure for different age groups of children. However, because of the consistent association of blood lead level and soil lead level, ingesting dirt at any age by any behavior is a source of some risk of exposure in this community.

This study is unique, in that it investigated the exposure of children to lead from several sources, including smelting, mine tailings, and natural mineralization, as opposed to just smelting or mining alone. Nevertheless, the slope values found in this study were similar to results from smelting communities (16) and are larger than those found in mining communities without smelters (15, and W. R. Chappell et al., Department of Physics, Environmental Sciences Program, University of Colorado at Denver, Denver, Colorado, unpublished manuscript).

The most effective individual prevention strategy seems to be one that leads to parental understanding of how children are most likely to be exposed to lead in this community—through ingestion of soil while playing outside or from objects, clothes, or pets carrying soil. This study provides evidence on a community level that preventing exposure to dirt would reduce exposure to lead. Furthermore, ongoing screening of infants and children with elevated lead levels is needed, as are education programs for children and their parents, consistent with the 1991 statement of the Centers for Disease Control.

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association between blood lead levels and lead-based paint, one would expect children in the group with valid x-ray fluorescence analyzer measurements to have lower blood lead levels than children in the group with invalid x-ray fluorescence analyzer measurements. This was not the case, however, and there was no statistical difference between the mean log blood lead levels of those for whom x-ray fluorescence analyzer data were available and those for whom it was not.

Other studies have described an association between indoor dust lead levels and blood lead levels (12–14). Such an association was not found in this study, perhaps because environmental scientists had difficulty collecting indoor dust. This may have been due to study participants cleaning the house before the scheduled visit.

Our study did not find an association between blood lead levels and lead in drinking water. We collected water samples at whatever time the home visit was scheduled and did not collect first-draw morning samples, which would have reflected the highest lead concentrations in the drinking water of each home. It is possible that first-draw samples would have had higher lead concentrations. The child, however, would have had to consume this water before it was ingested by another family member or used for some other purpose.

The contribution to blood lead levels made by different pathways and sources of exposure varied by age, as has been shown in other studies (15, and W. R. Chappell et al., Department of Physics, Environmental Sciences Program, University of Colorado at Denver, Denver, Colorado, unpublished manuscript). In this study, only the soil lead contributed independently to elevating the blood lead levels of the younger children (6–36 months). For older children (37–71 months), however, both behavioral pathways of exposure (in this study, swallowing things other than food or taking a bottle or food outside to play) were important in addition to the soil lead level for the elevation of blood lead levels. These data would suggest developing different strategies for the prevention of lead exposure for different age groups of children. However, because of the consistent association of blood lead level and soil lead level, ingesting dirt at any age by any behavior is a source of some risk of exposure in this community.

This study is unique, in that it investigated the exposure of children to lead from several sources, including smelting, mine tailings, and natural mineralization, as opposed to just smelting or mining alone. Nevertheless, the slope values found in this study were similar to results from smelting communities (16) and are larger than those found in mining communities without smelters (15, and W. R. Chappell et al., Department of Physics, Environmental Sciences Program, University of Colorado at Denver, Denver, Colorado, unpublished manuscript).

The most effective individual prevention strategy seems to be one that leads to parental understanding of how children are most likely to be exposed to lead in this community—through ingestion of soil while playing outside or from objects, clothes, or pets carrying soil. This study provides evidence on a community level that preventing exposure to dirt would reduce exposure to lead. Furthermore, ongoing screening of infants and children with elevated lead levels is needed, as are education programs for children and their parents, consistent with the 1991 statement of the Centers for Disease Control.

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