Environmental Arsenic Exposure of Children around a Former Copper Smelter Site

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Arsenic residues in the communities surrounding former smelters remain a public health concern, especially for infants and children. To evaluate environmental exposure among these children, a population-based cross-sectional study was conducted in the vicinity of a former copper smelter in Anaconda, Montana. A total of 414 children less than 72 months old were recruited. First morning voided urine samples and environmental samples were collected for arsenic measurements. The geometric mean of speciated urinary arsenic was 8.6 $\mu\text{g/liter}$ (GSD = 1.7, $N = 289$). Average arsenic levels of different types of soil ranged from 121 to 236 $\mu\text{g/g}$, and were significantly related to proximity and wind direction to the smelter site. The same significant relationship was observed for interior dust arsenic. Speciated urinary arsenic was found to be significantly related to soil arsenic in bare areas in residential yards ($P < 0.0005$). In general, elevated excretion of arsenic was demonstrable and warranted parents' attention to reduce exposure of their children to environmental arsenic. © 1997

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INTRODUCTION

Arsenic is a terrestrial element and is naturally distributed in air, water, soil, and food at a low or negligible level. A major concern for health effects is raised by environmental contamination resulting from human activities, such as copper smelting. Epidemiological studies have shown that arsenic is associated with several cancers, such as lung, skin, liver, bladder, and kidney (Enterline *et al.*, 1987; Smith *et al.*, 1992; Roat *et al.*, 1982; Zaldivar *et al.*, 1981; Chen *et al.*, 1988; Wu *et al.*, 1989). Also, long-

term exposure may lead to vascular disease and a characteristic pattern of skin abnormalities including patches of hyper- and hypopigmentation along with multiple hyperkeratotic "warts."

It has been recognized that consumption of contaminated soil by children may present a significant public health problem. Several studies have reported mean soil ingestion values ranging from 9 to 1834 mg/day, depending on the soil tracer elements used (Binder *et al.*, 1986; Clausen *et al.*, 1987; Calabrese *et al.*, 1989). Accordingly, assuming average arsenic levels in soil of 60 to 150 $\mu\text{g/g}$ in the high arsenic exposure areas (Binder *et al.*, 1987), the resulting average daily arsenic intake from soil could range from 1 to 275 μg per child.

Prior to the present study, several studies have been conducted in Anaconda, Montana, in which nearly 100 years of smelter operations resulted in waste materials including tailings and flue dust that now cover approximately 4000 acres (Atlantic Richfield Co., 1993). Following cessation of copper smelting in 1980, there has been no comprehensive survey of environmental and biological arsenic levels in this population. In order to evaluate current arsenic exposure in this area, a survey is required to assess the urinary arsenic levels, the environmental arsenic levels, the consumption of arsenic-containing food, and the children's behavior and/or daily activities which contribute to arsenic intake.

The present study took place in Anaconda, Montana, from the summer of 1992 through the summer of 1993. The study samples were gathered specifically so that they could be utilized to obtain more precise information on environmental arsenic levels and exposure pathways. The study sought to ascertain whether or not the children of Anaconda, living in the vicinity of the former smelter and waste dump sites, are currently exhibiting elevated urinary arsenic concentrations.

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MATERIALS AND METHODS

Prior to initiating the current study, a door-to-door census was undertaken in April and May, 1992, to determine how many families and children less than 72 months old were in each of the proposed study subareas. Six two-member teams were used in the collection of census data, and also in the subsequent study subject recruitment and in household visits for the collection of environmental and biological samples. All teams were uniformly trained in the questionnaire administration and the collection of environmental and biological samples. All families with age-appropriate and eligible children (less than 72 months and potty trained) were invited to participate. The children must have resided at their current address for at least 3 months. Study participation was strictly voluntary. Following a verbal and written description of the study, participants were asked to sign an Informed Consent Statement. Individual and family information was collected by administration of questionnaires during the study period. We enquired about children's demographic and behavioral information, participating families' socioeconomic status, residence location, house history and condition, and general environmental conditions regarding potential arsenic exposure. When the monitoring team collected soil, dust, water, and urine samples, corresponding forms were filled out to record the detailed collection information regarding each child-specific sample, e.g., date, time, place, area, information about the child's indoor and outdoor activities, and food consumption.

Exterior soil samples of the top 2 cm soil were collected with a coring device from each residential parcel occupied by the participating family. The five types of soil samples included perimeter soil sample, bare-area composite soil sample, garden-area composite soil sample, sand box or dirt play area sample, if present, and gravel or hardpack drive/parking areas sample. After air-drying and sieving with a mesh size of 250 μm , approximately 2 g of loose soil sample was measured for arsenic by the Kevex Delta Analyst Energy Dispersive X-ray Spectrometer (Hwang, 1994). Interior surface dust was collected according to a previously developed method (Que-Hee *et al.*, 1985) and consisted of a composite of at least three subsamples taken from the following areas in the residence, i.e., an area adjacent to the main entrance, a floor area in the room utilized most by the subject child, and a floor area in the subject child's bedroom. The dried and weighted dust sample was then processed for acid digestion and submitted for arsenic determination by a graphite

furnace atomic absorption spectrometer (GFAAS) (Hwang, 1994). The water samples were collected from the Anaconda municipal water supply system and from those households using private well water. Arsenic in water was also analyzed on a GFAAS (Hwang, 1994).

The first morning voided (FMV) urine samples were collected from each child participant for 2 consecutive days. After collection, samples were acidified with HNO_3 and then frozen for storage. A stratified random sample of 25 children drawn from the main study cohort was recruited to collect 24-hr urine samples. In addition to two samples of the consecutive first morning urine voids as described above, the third sample was designated the midday urine sample and was a composite of subsamples voided between the two consecutive first morning voids. Urine samples were analyzed for specific gravity by an optical refraction method at the field office (Walters *et al.*, 1990). Creatinine was measured with a colorimetric method using the Sigma Diagnostics Creatinine Kit (Henry, 1965). Two methods were used for urinary arsenic measurement. First, a modified digestion method was used to analyze total urinary arsenic, which includes inorganic arsenic, methylated arsenic, and fish arsenic (e.g., arsenobetaine, arsenocholine). The samples were acid digested with a mixture of nitric, perchloric, and sulfuric acids and analyzed on a hydride generation system attached to a flame atomic absorption spectrometer (HG-FAAS) with a detection limit of 1 $\mu\text{g/liter}$ (Yamamoto, 1985; NIOSH, 1977). The second method was used for the determination of combined As^{3+} , As^{5+} , monomethylarsonic acid (MMA), and dimethylarsinic acid (DMA), which are the major arsenic species and/or metabolites found in urine after exposure to inorganic arsenic, and referred as speciated urinary arsenic. The analytical method for the speciated urinary arsenic was based on the direct analysis of the urine sample on the HG-FAAS with a detection limit of 0.5 $\mu\text{g/liter}$ (Norin and Vahter, 1981).

For the purpose of quality control, field blanks and shipment blanks were obtained for each batch of environmental and biological samples. In addition, different levels of NBS Standards were used as references for arsenic determination. Detailed description of the QA/QC process can be found elsewhere (Hwang, 1994).

Data collected from questionnaires and results from laboratory analyses were entered into the Fox-BASE+/Mac data base system (Fox Software, 1991). The data analyses were performed by the Statistical Analysis System for Personal Computers (SAS

1989). Prior to major statistical analysis being performed on any of the data, univariate analyses of the frequency distributions were reviewed. Due to the log-normal distribution, urinary arsenic and environmental arsenic data were transformed to their log equivalent, and estimates of the sample geometric mean and geometric standard deviation were obtained. Simple bivariate correlations among the exposure variables, covariates, confounders, and dependent variables were then calculated.

RESULTS

The door-to-door census identified a total of 480 families with 642 children less than 72 months of age in the study area. With some families refusing to participate or moving out prior to the environmental sampling, only 412 (85.8%) families participated in the present study. However, only age-appropriate potty-trained children were recruited for urine collection. Thus, 414 children in 334 families were recruited as the study subjects in the urine arsenic evaluation.

As previously noted, urine samples were intended to be collected on 2 consecutive days in order to provide a more stable estimate of arsenic excretion. However, not all children provided two consecutive first morning urine samples. Also, if a sample contained an abnormally low level of creatinine, indicative of a very dilute urine specimen, the arsenic result was excluded from analysis since the arsenic concentration might be spuriously low. In addition, the sample sizes of speciated urinary arsenic and total urinary arsenic in the data analysis might be different due to the troubles encountered in each analytic process. As a result, urinary arsenic levels were available for 404 children. Of these 404 children, total and speciated urinary arsenic levels available on the 2 consecutive days were obtained from 312 and 289 children, respectively. Since the intent was to perform statistical analyses based on two consecutive first morning urine samples, those children who provided only one urine sample were excluded from the analyses. Nevertheless, there was no significant difference between the children providing two urine samples and the excluded ones in most demographic factors, such as height, weight, gender, and race. Only the average age of the former (52.6 months) was slightly older than that of the latter (47.8 months).

Table 1 shows descriptive data for recruited children with two-consecutive first morning voided urine samples. Of them, 97.4% were white, while native American, black, Asian, and Hispanic chil-

TABLE 1
Descriptive Statistics on Study Subjects^a

Variable	Mean	SD	5th percentile	95th percentile
Age (months)	52.6	14.5	28.6	74.0
Height (cm)	104.2	10.6	88.3	121.3
Weight (kg)	16.5	3.6	11.4	22.7
Gender (% male)	48.3			
Race (% white)	97.4			
Body surface area (m ²)	0.704	0.123	0.515	0.895
First morning void (ml)	54.4	23.6	17.0	94.0
Creatinine (g/liter)	1.092	0.396	0.480	1.800
Specific gravity	1.026	0.005	1.017	1.033

^a N = 271 children. Only children with complete data for all these nine variables are presented.

dren constituted the remaining 2.6% of participants. Meanwhile, the body surface area was determined from a nomogram based on children's height and weight (Slovic *et al.*, 1989) and an average of 0.704 m² was obtained. On the other hand, the mean urinary creatinine level and mean specific gravity were close to those of the general populations at this age.

In order to present the characteristics of the residential area in finer detail, the study area has been categorized into subareas A through K as shown in Fig. 1. The highest soil arsenic levels were found in central (subareas C, E) and east (subareas D, F) Anaconda, while the lowest arsenic levels were found upwind of and far from Anaconda (subareas A, H). Teresa Ann Terrace (subarea I) and Cedar Park Homes (subarea J), within subarea D and next to the old smelter site, did not have high levels of soil arsenic at the surface although these two areas were located on the EPA's priority list for soil abatement. This was probably due to the fact that they were new communities (established after 1970) and that the previously contaminated soil had been replaced or mixed with clean soil during new house construction. Perimeter soils and bare area soils in yards tended to have higher levels of arsenic contamination than soil samples collected in hardpack areas, garden areas, and play and/or sand box areas. This suggests that the soil in garden and play areas might have been brought onto the property. With respect to proximity and wind direction from the residence to the smelter site (categorized as Close, Intermediate, and Remote), a significant trend of soil arsenic levels is shown in Table 2 and Fig. 2. Also shown in Table 2 and Fig. 2 is the interior dust arsenic level. The geometric mean of arsenic levels in all interior dust samples was 73 $\mu\text{g/g}$ (GSD =

FIG. 1. Map of study subareas in Anaconda.

1.84). The highest dust arsenic levels were found in east Anaconda (subareas D, F). However, dust arsenic concentrations did not exhibit as much interarea variation as the soil arsenic levels.

In Table 3, when broken down by subareas, the average total urinary arsenic levels varied randomly. Meanwhile, total urinary arsenic levels in the three levels of proximity and wind direction categories did not differ significantly from each other and only a slight trend was observed. Also shown, in Table 3, the highest speciated arsenic levels were present in the northeast of Anaconda (subareas D, I, J) and far downwind of Anaconda (subarea K). However, any interpretation of differences between these subareas must be tempered by the small sample size in some study subareas. Nevertheless, the speciated urinary arsenic levels did show an association along with the combined categories of proximity and wind direction since the geometric mean of speciated urinary arsenic level in the CLOSE group was much higher than that of the other two groups.

The summary data of the higher value of the two consecutive first morning urinary arsenic levels of each child are also reported as Highest in Table 3. The differences between Average and Highest uri-

nary arsenic level means varied randomly across study areas. This result indicates that it did not make any difference in this study if the Average or Highest value was used to express urinary arsenic levels.

Table 4 presents a correlation coefficient matrix of the pertinent arsenic exposure and urinary arsenic levels. Soil in bare areas in yards had the highest correlation with speciated urinary arsenic levels ($r = 0.25$) (Fig. 3) while the other four types of soil samples had weaker but still significant correlations ($r = 0.12$ to 0.20). The correlation of total urine arsenic with soil arsenic levels was much weaker (no significant correlations found). In contrast to initial expectation, arsenic in interior dust did not correlate well with urinary arsenic.

DISCUSSION

The present study was a population-based exposure survey directly measuring the environmental exposure and its impact on children's arsenic body burden. The purpose of the present study was not simply to demonstrate that the average urinary arsenic in a study area was significantly different from

TABLE 2
Geometric Means of Arsenic Levels in Soil and Interior Dust by Study Subareas^a

Study area	Soil As ($\mu\text{g/g}$) ^b					Interior dust As ($\mu\text{g/g}$)
	P	B	H	G	S	
Close						
C	309 (20)	346 (18)	98 (8)	294 (8)	201 (15)	43 (36)
D	353 (23)	306 (19)	223 (12)	251 (9)	294 (16)	108 (23)
E	341 (45)	317 (39)	193 (22)	235 (20)	276 (34)	81 (68)
F	377 (133)	356 (121)	215 (67)	298 (44)	286 (116)	105 (134)
I	183 (12)	143 (10)	93 (1)	83 (2)	63 (4)	72 (12)
J	209 (21)	239 (17)	131 (2)	—(0)	108 (18)	52 (20)
Close	333 (254)	320 (224)	197 (112)	267 (83)	247 (203)	83 (293)
Intermediate						
B	197 (69)	174 (54)	107 (25)	136 (48)	134 (65)	58 (70)
G	174 (28)	138 (27)	71 (27)	116 (14)	142 (20)	71 (28)
K	207 (8)	237 (7)	113 (8)	236 (3)	243 (2)	94 (10)
Intermediate	191 (105)	166 (88)	89 (60)	135 (65)	137 (87)	64 (108)
Remote						
A	102 (59)	102 (46)	66 (49)	84 (30)	81 (54)	49 (59)
H	95 (17)	115 (15)	83 (16)	76 (6)	119 (10)	63 (17)
Remote	100 (76)	105 (61)	70 (65)	83 (36)	86 (64)	52 (76)
All areas	236 (435)	229 (373)	121 (237)	167 (184)	177 (354)	73 (477)

^a Numbers in parentheses indicate the sample sizes.

^b P, perimeter soil samples; B, bare area soil samples; H, hardpack area soil samples; G, garden area soil samples; S, sand box soil samples.

that of a "control" area. Also, due to the multivariate nature of factors affecting childhood arsenic exposure (such as socioeconomic status, parenting style, behavior, dietary differences) and the uncertainty regarding soil arsenic concentrations in any potential control area, this study does not employ a con-

trol group selected from outside the proposed study area. Rather, child-specific arsenic sources in each home were measured to determine the relative impact of these sources on the arsenic exposure of residents. Estimates of the impact of various independent variables (i.e., soil arsenic, dust arsenic) could be developed using statistical techniques. Thus, with a sufficiently wide range of exposures, which encompasses background exposure levels, it was possible to obtain estimates of the impact of environmental arsenic sources through data derived from within the study areas.

Also, the current study contained several features which maximized the probability that the highest urinary arsenic levels would be observed, the highest arsenic sources would be detected, and the relationship between environmental sources and urinary arsenic would be correctly characterized. For example, the recruitment strategy focused on young children (less than 72 months of age), the group known to be at greatest risk for elevated arsenic exposure. Urinary arsenic sampling was carried out during the summer, a time when seasonal factors combined to yield the highest urinary arsenic levels. The focus of measurement was on those sources and forms of arsenic which were immediately accessible to a young child, for example arsenic in dust, in soil, and in drinking water. In the course of analyzing samples for arsenic content, attention was focused

FIG. 2. Arsenic levels in soil of bare areas in yards, in interior floor dust and speciated urinary arsenic by proximity index of residence to smelter site (GM $\pm$ GSE).

TABLE 3
Total and Speciated Urinary Arsenic Levels by Study Subareas ($\mu\text{g/liter}$)^a

Study area	Total As			Speciated As		
	N	Average	Highest	N	Average	Highest
Close						
C	22	22.0 (1.6)	25.2 (1.7)	18	10.3 (1.6)	11.9 (???)
D	12	23.0 (1.8)	27.5 (1.8)	11	13.3 (1.8)	16.4 (???)
E	46	19.7 (2.1)	23.9 (2.3)	46	8.5 (1.8)	1.2 (???)
F	91	19.0 (1.7)	23.3 (1.8)	83	8.9 (1.6)	10.8 (???)
I	6	25.0 (2.1)	27.9 (2.0)	6	12.2 (1.6)	14.5 (???)
J	12	26.9 (1.9)	33.9 (1.9)	13	12.2 (2.0)	14.3 (2.???)
Close	189	20.3 (1.8)	24.6 (1.9)	177	9.5 (1.7)	11.4 (1.8)
Intermediate						
B	55	15.3 (2.0)	19.0 (2.2)	49	7.5 (1.5)	9.3 (1.5)
G	22	23.1 (2.4)	29.5 (2.7)	20	6.9 (1.8)	8.8 (1.8)
K	2	30.0 (1.8)	50.5 (1.7)	1	12.0 (—)	19.0 (—)
Intermediate	79	17.4 (2.2)	22.0 (2.4)	62	7.5 (1.5)	9.2 (???)
Remote						
A	40	17.0 (1.7)	20.9 (1.7)	38	6.9 (1.8)	8.5 (1.9)
H	4	22.6 (1.5)	28.2 (1.5)	4	9.6 (1.5)	12.1 (1.6)
Remote	44	17.4 (1.7)	21.4 (1.7)	42	7.1 (1.8)	9.0 (1.9)
All	312	19.1 (1.9)	23.5 (2.0)	289	8.6 (1.7)	10.4 (1.8)

^a Only children with two consecutive first morning urine samples were included. Geometric mean is presented with GSD in parentheses for urinary arsenic level.

on that fraction of the environmental source which was thought to produce the greatest risk to the child, i.e., arsenic in small particles ($<250\ \mu\text{m}$) that most readily adhere to hands and toys, and could be inadvertently ingested. Finally, the biological indicator of children's arsenic body burden was based on the two consecutive first morning urine samples in order to yield a more stable estimate. Because of these design features, it was unlikely that a higher community mean or wider range of urinary arsenic could be found. Furthermore, since the half-life of most absorbed arsenic species in the human body is about 1–3 days (Crecelius, 1977; Tam *et al.*, 1979; Buchet *et al.*, 1981), this cross-sectional study design

was appropriate and allowed evaluation of constant and low-level arsenic exposure of young children.

The mean total urinary arsenic levels of the present study was 11–22 $\mu\text{g/liter}$ lower than those reported in two previous studies in Anaconda, Montana (Table 5) (Baker *et al.*, 1977; Hartwell *et al.*, 1983). Both studies were focused on children less than 6 years old and were conducted in the summer when the smelter was in operation. Thus, the decrease in urinary arsenic levels among children relative to the two previous studies might be attributed to the shut-down of this smelter. On the other hand, mean total urinary arsenic level in the present study (19.1 $\mu\text{g/liter}$) was slightly, but not significantly,

TABLE 4
Correlation Coefficient Matrix of Urinary Arsenic, Soil Arsenic, and Interior Dust Arsenic^a

	Total As		Speciated As		Soil P	Soil B	Soil H	Soil G	Soil S
Speciated As ($\mu\text{g/liter}$)	0.59***	(282)							
Soil P ($\mu\text{g/g}$)	0.04	(282)	0.17*	(260)					
Soil B ($\mu\text{g/g}$)	0.00	(243)	0.25***	(226)	0.77***	(404)			
Soil H ($\mu\text{g/g}$)	-0.09	(146)	0.20*	(134)	0.60***	(256)	0.67***	(224)	
Soil G ($\mu\text{g/g}$)	0.10	(132)	0.20*	(121)	0.64***	(202)	0.55***	(165)	0.52*** (112)
Soil S ($\mu\text{g/g}$)	0.01	(280)	0.12*	(257)	0.65***	(389)	0.64***	(335)	0.50*** (206)
Int. Dust ($\mu\text{g/g}$)	0.01	(310)	0.03	(286)	0.45***	(470)	0.47***	(404)	0.47*** (256)
									0.42*** (203)
									0.52*** (391)

^a Numbers in parentheses indicate the sample sizes. All variables were log-transformed before correlation analysis.

* $P < 0.05$.

** $P < 0.005$.

*** $P < 0.0005$.

FIG. 3. Scatter plot of speciated urinary arsenic against soil arsenic level of bare areas in yards.

higher than that ($17.7 \mu\text{g/liter}$) of the survey conducted in Anaconda in 1985 when the smelter had already been shut down (Table 5) (Binder *et al.*, 1986). Furthermore, a nationwide survey on arsenic exposure in the vicinity of smelter sites showed that children without excess arsenic exposure have average total urinary arsenic levels ranging from 5 to $10 \mu\text{g/liter}$. Compared to these values, the mean total urinary arsenic of the present study was markedly higher than this "background" level. Assuming the average arsenic intake from dietary sources was comparable among those children in these studies, this difference might be attributed to the arsenic residues in residential soils, coming from the former smelter site. Nevertheless, these levels were still well below the World Health Organization's recommended maximum excretion level for total arsenic of $100 \mu\text{g/liter}$ as an action level for intervention (Fig. 3). Also, 90% of the samples were below $50 \mu\text{g/liter}$, which is an action level previously employed by the Centers for Disease Control and Prevention. In addition to total urinary arsenic, speciated urinary arsenic levels in the present study were comparable to that of the control group measured in a similar study conducted in Tacoma, Washington, in 1985, and markedly less than that of the study group living closest to the smelter site, whose median of uncorrected speciated urinary arsenic for children aged 0–6 years was $19.4 \mu\text{g/liter}$ (Table 5) (Polissar *et al.*, 1990). The smelter in Tacoma was shut down during the first quarter of that environmental arsenic survey while the smelter in Anaconda ceased operation

about 12 years prior to the present study. The difference in speciated urinary arsenic levels between these two studies might be attributed, in part, to the time elapsed between the smelter shut-down and the study execution, and to variable times for elimination or chemical alteration of arsenic sources.

Low urinary arsenic levels found in the study area were probably a reflection of low bioavailability of some forms of arsenic in contaminated soil. Recent microanalysis of soil particles in the residential study area, using an electron beam microprobe, suggested limitations of arsenic bioavailability due to encapsulation within insoluble matrices, to grinding of the arsenic grain by precipitation reaction, or to low solubility of the arsenic-bearing phases (Davis *et al.*, 1993). An animal feeding study provided further evidence of low bioavailability of arsenic found in Anaconda soils (Freeman *et al.*, 1993). This study reported that, for the animals fed with test soils, approximately 80% of the administered arsenic dose was eliminated in feces, indicating that little was absorbed. This bioavailability study of soil arsenic based on site-specific data implied a low percentage of arsenic intake through soil ingestion by children in the present study. Furthermore, bioavailability of arsenic in the actual soils may be less than that determined in the above-mentioned study. It was noted that actual particle sizes of the test soils in this bioavailability study were within the range of the size of soil particles reported to adhere to children's hands ($<100 \mu\text{m}$) (Chaney *et al.*, 1989). These particles were of a size that optimized dissolution rate and the extent of absorption. In contrast, arsenic which is found in large particles ($>250 \mu\text{m}$) is less subject to inadvertent ingestion. If ingested, large particles release less of their arsenic content per unit time than smaller particles. Therefore, bioavailability of arsenic in actual bulk soil ingestion from the study area might be even less than that determined in the bioavailability study due to the small size fraction of material used in that study, which represented only the finer percentage of the bulk soil. Thus, for a child with pica, the environmental arsenic levels measured in the present study represent a worst-case scenario in terms of the extent of arsenic exposure.

Arsenic exposures other than environmental soil and dust are also of concern in the present study. Some undefined fraction of arsenic might come from dietary arsenic, i.e., water and food, while arsenic exposure through inhalation and skin contact may be alternative pathways for the environmental sources.

Respiratory intake has been thought to be a pri-

TABLE 5
Childhood Arsenic Exposure Studies at Copper Smelters

Location	Soil arsenic (ppm)	N	Urinary arsenic (GM) ($\mu\text{g/liter}$)	Reference
Anaconda, MT			Total As	Baker <i>et al.</i> (1977)
3.6 mi ^a	—	40	30.8	
Comparison towns	—	193	5.8	
Anaconda, MT			Total As	Hartwell <i>et al.</i> (1983)
3 mi	171	9	40.0	
6 mi	65	38	10.0	
Anaconda, MT			Total As	Binder <i>et al.</i> (1987)
Mill Creek	398	8	54.0	
Anaconda	144	61	17.7	
Opportunity	136	21	15.3	
Livingston	44	33	16.6	
Ruston, WA			Speciated As	Polissar <i>et al.</i> (1990)
Ruston, 0.3 mi	353	649	19.4	
Vashon, 6.3 mi	30	351	11.7	
Bellingham, 105 mi	—	61	10.1	
Anaconda, MT			Total As	Present study
Close	319	140	19.9	
Intermediate	201	104	18.7	
Remote	114	46	17.8	
			Speciated As	
Close	319	134	9.1	
Intermediate	201	94	8.6	
Remote	114	43	7.2	

^a Miles, distance from smelter site.

mary pathway of arsenic exposure for workers occupationally exposed to arsenic. However, this situation does not hold true for the general population since ambient air concentrations of arsenic are usually well below that attained in arsenic-related work sites. In the Anaconda study, the air arsenic level was quite stable and well below the national average level for remote areas. Based upon an average air arsenic concentration of $0.0021 \mu\text{g}/\text{m}^3$ during the year prior to the present study (McVehil-Monett Associates, 1992), the respiratory arsenic excreted in urine can be estimated conservatively as $0.017 \mu\text{g}/\text{day}$ by assuming a tidal volume of 0.5 liters, breathing frequency of 18/min, 80% bioavailability of inhaled arsenic, and 80% excretion rate of absorbed arsenic in urine. This amount of estimated respiratory arsenic accounted for only 0.7% of the amount of the study children's daily speciated urinary arsenic output, which ranged 2.3 to $2.7 \mu\text{g}/\text{day}$, based on the subgroup with 24-hr urine data. This value is so small that its contribution to urine arsenic may be neglected.

In contrast, drinking water is one of the most important sources of arsenic exposure. Since most of the arsenic in water is dissolved and present in inorganic forms, such as As^{3+} and As^{5+} , water arsenic is expected to be absorbed readily through the hu-

man gastrointestinal tract. In this study, the measured water arsenic level was $1.36 \mu\text{g}/\text{liter}$ in the municipal water supply system, which covered 81.3% of the studied households. Most families using the public water supply were located in the subareas close to smelter site, which had higher soil arsenic levels than other subareas. For those with private wells, most of the water arsenic levels were below $5 \mu\text{g}/\text{liter}$, with an average of $2.5 \mu\text{g}/\text{liter}$. Assuming daily water consumption of 0.65 liter, 80% bioavailability of water arsenic, and 80% excretion rate of absorbed arsenic in urine, that portion of urinary arsenic coming from drinking water was estimated to range from 0.57 to $1.04 \mu\text{g}/\text{day}$. This accounts for a significant portion (about 21 to 45%) of the amount of the study children's daily speciated urinary arsenic output. However, the contribution of water arsenic to urinary arsenic would dilute the correlation between soil arsenic and speciated urinary arsenic because arsenic levels in drinking water were generally lower in the subareas with higher soil arsenic levels. Thus the significant association of soil arsenic with speciated urinary arsenic in the study area cannot be confounded by water arsenic levels. The arsenic that tap water contributed to total arsenic intake was therefore considered to be reflected in background urinary arsenic levels, and not

the primary factor in explaining differences in speciated urinary arsenic levels among the study children.

On the other hand, although skin contact could be an alternative pathway for arsenic intake, dermal absorption of the As^{5+} -form of arsenic in soil is only about 3–5% *in vivo* (Wester *et al.*, 1993). Due to low solubility of arsenic compounds in soils, the skin arsenic absorption rate for children in the study area was believed to be well below 3–5%. Furthermore, dermal exposure leads to arsenic binding to skin, and thus arsenic may not get into the blood stream to be excreted by the urine (ATSDR, 1992). Therefore, arsenic intake through skin contact is insignificant and may be neglected in the assessment of childhood arsenic exposure.

For the general population, food is thought to be the greatest source of arsenic exposure, especially seafoods. These “fish arsenics” are mostly organic arsenic and essentially nontoxic (e.g., arsenobetaine and arsenocholine in fish and shellfish). In the present study, the effect of eating seafoods on urinary arsenic has been examined for both total arsenic and speciated arsenic. Results showed that eating seafoods in the 2 days prior to urine sampling did not affect the speciated but rather the total urinary arsenic levels. This is consistent with our understanding of fish arsenic’s contribution to urinary arsenic, in that fish arsenic added to the amount of total urinary arsenic excreted rather than speciated urinary arsenic. Another issue of concern is crops grown in gardens with contaminated soil, which can themselves become contaminated either via arsenic contaminants adhering to the outer surface of crops or via uptake of arsenic into the plant. However, when crops are properly cleaned prior to eating, it is believed that very little arsenic is actually ingested as a result of arsenic taken up by the plant. Available data suggest that inorganic arsenic absorbed by the plant may be converted to organic arsenic compounds similar to water-soluble organo-arsenicals found in marine organisms (Nissen and Benson, 1982; Pyles and Woolson, 1982). Thus, methylation of arsenic in vegetables should substantially reduce risk estimates associated with the ingested dose. In the present study, the failure of eating locally grown crops to account for any of the variance in speciated urinary arsenic levels provides evidence for reduced risk due to this source.

In general, average environmental arsenic exposure in the present study group was low, without evident and imminent threat of adverse health effect. However, elevated excretion of arsenic was still

demonstrable and warranted parents’ attention to reduce exposure of their children to environmental arsenic. In addition, the results of the present study do show a dose–response relationship of environmental arsenic with internal exposure dose, i.e., speciated urinary arsenic. Since children, with the characteristics of energetic activity, have direct access to the environmental contaminants in surroundings, parents or guardians need to pay more attention to their children’s activity, especially hand-to-mouth behavior, even though the environmental contaminants might be elevated only slightly. In other words, people who live near a contaminated site should be advised to act conservatively and to make efforts to reduce exposure to environmental contaminants in order to minimize the potential health effects on themselves.

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