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BASELINE HUMAN HEALTH RISK ASSESSMENT VASQUEZ BOULEVARD AND I-70 SUPERFUND SITE DENVER, CO

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APPROVAL PAGE

This risk assessment has been prepared in accord with USEPA national and regional guidelines and is approved for release without condition.

8/3/01

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LIST OF ACRONYMS AND ABBREVIATIONS

CDC	Centers for Disease Control
CDPHE	Colorado Department of Public Health and Environment
cm	centimeter
COPC	chemical of potential concern
CTE	central tendency exposure
DI	daily intake
DL	detection limit
dL	deciliter (0.1 liter)
dw	dry weight
EC01	concentration in water that results in a 1% increase in excess lifetime cancer risk
EMPA	electron microprobe analysis
EPC	exposure point concentration
g	gram
GFAA	graphite furnace atomic absorption
GSD	geometric standard deviation
HIF	human intake factor
HQ	hazard quotient
ICP	inductively coupled plasma spectroscopy
m	meter
m^3	cubic meter
MS	mass spectrometry
IEUBK	Integrated Exposure, Uptake, and Biokinetic Model
IRIS	Integrated Risk Information System
ISE	Integrated Stochastic Exposure Model
kg	kilogram
LOAEL	lowest observed adverse effect level
mg	milligram
ng	nanogram
NOAEL	no observed adverse effect level
NPL	National Priorities List
PbB	blood lead level
PDF	probability density function
ppm	parts per million
RBA	relative bioavailability
RBC	risk-based concentration
RfD	reference dose
RME	reasonable maximum exposure
SF	slope factor
TAL	Target Analyte List
UCL	upper confidence limit
ug	microgram
USEPA	U.S. Environmental Protection Agency
VBI70	Vasquez Boulevard and I-70 Site
ww	wet weight
XRF	X-ray fluorescence

EXECUTIVE SUMMARY

1.0 BACKGROUND

1.1 Site Description

The Vasquez Boulevard and I-70 (VBI70) Superfund Site is an area of approximately four square miles located in the north-central section of Denver, Colorado. The site is composed of a number of neighborhoods that are largely residential, including Swansea, Elyria, Clayton, Cole, and portions of Globeville. Most residences at the site are single family dwellings, but there are also some multi-family homes and apartment buildings. The site also contains a number of schools, parks, and playgrounds, as well as a number of commercial and industrial properties. Figure ES-1 is a map which displays the site.

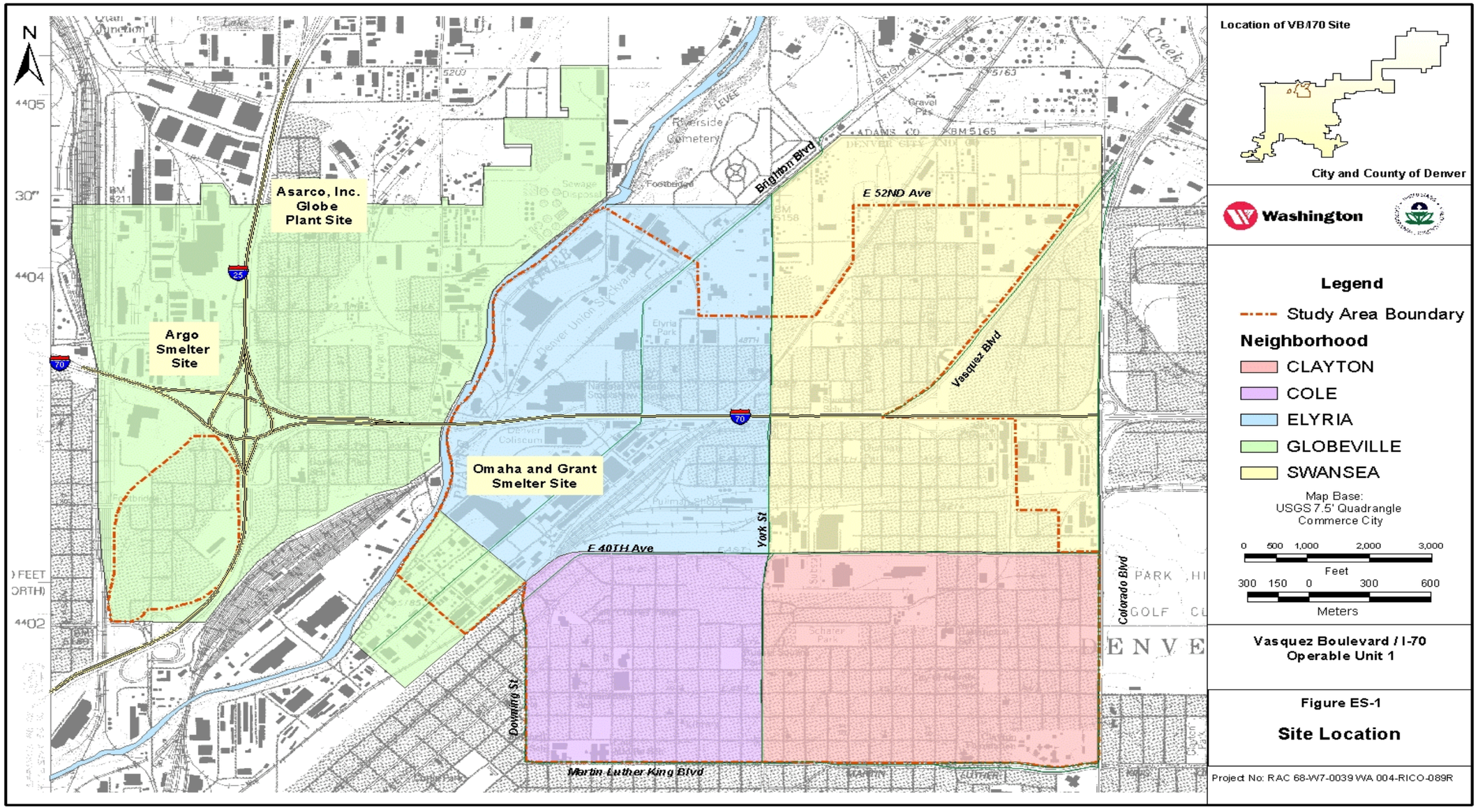
1.2 Basis For Potential Concern

The site came to the attention of the U.S. Environmental Protection Agency (USEPA) because studies directed by the Colorado Department of Public Health and Environment (CDPHE) at a nearby site (Globe Smelter) indicated that elevated concentrations of arsenic and/or lead occurred in the soil of some residential properties in the Swansea/Elyria area. The source of these elevated levels is not known, but *a priori,* it is considered plausible that the contamination is associated with releases either from the Globe facility and/or from one or both of two other smelters which previously existed in the area (the Argo Smelter and the Omaha and Grant Smelter). The locations of these three smelters in relation to the VBI70 site are also shown in Figure ES-1. Alternative potential sources include the historic application of arsenic- or lead-containing lawn care products, and/or (for lead) anthropogenic sources such as automobile exhaust, leaded paint, etc.

Based on the results of several rounds of soil sampling, USEPA concluded that the VBI70 site contained multiple residences where the concentration of arsenic and/or lead in yard soil could be above a level of potential human health concern. On this basis, USEPA proposed the VBI70 site for inclusion on the Superfund National Priorities List (NPL) in January, 1999, and the site was added to the NPL on July 22, 1999.

The process of evaluating the nature and extent of environmental contamination at the site and of estimating the potential risks to human and ecological receptors has been divided into several sub-projects, or "Operable Units". This risk assessment focuses on risks associated with soil contamination in residential areas of the site. This is referred to as Operable Unit 1 (OU1).

Figure ES-1 Site Map



2.0 SUMMARY OF SITE DATA AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN

2.1 Initial Studies

Phase I/Phase II

Once investigations at the nearby Globe site began to suggest that elevated levels of arsenic and/or lead might exist in soils at residential properties within the area of the VBI70 site, CDPHE requested assistance from USEPA Region VIII in characterizing the nature and extent of the contamination. In response, USEPA Region VIII undertook a study designed to identify properties that had levels of arsenic or lead that were sufficiently high that time-critical action (soil removal and replacement) might be warranted. Most of these samples were collected during the initial round of sampling (referred to as Phase I), with the remainder being obtained in a subsequent sampling effort (Phase II). In the majority of cases, two surface samples and one subsurface sample were collected per property, with additional surface samples at some locations (depending on the size of the property).

The action levels selected for time-critical soil removal were 450 ppm for arsenic and 2,000 ppm for lead. For arsenic, a majority of properties sampled (927 out of 1390) had maximum values that were below the limit of detection (average detection limit in Phase I/II = 51 ppm). However, arsenic was detected in one or more surface soil samples at a number of properties, with 40 of these properties having one or more samples above 450 ppm. For lead, most properties (1,153 out of 1,390) had concentration values in surface soil that were below 400 ppm, but 238 properties had one or more values above 400 ppm. Of these, six properties had one or more lead value above 2,000 ppm.

In order to help confirm the identity of properties which warranted time-critical soil removal actions, USEPA collected two or more composite samples (each consisting of five sub-samples) of surface soil from residential properties where one or more grab samples were above the removal level for arsenic. Based on the results of this composite sampling program, a total of 21 residences were identified where one or more composites confirmed that arsenic levels were above the action level. Using the authority provided under CERCLA 104, EPA performed soil removal and replacement at 18 of these properties in the fall of 1998. The owners of the other three properties refused permission for the removal. No properties were identified where lead levels in composite soil samples were high enough to warrant a time-critical soil removal action.

Risk-Based Sampling Program

One of the striking findings that emerged from the Phase I/Phase II sampling programs was that arsenic-affected properties did not appear to occur in a clear spatial pattern. That is, the occurrence of high arsenic levels in soil did not appear to be associated with proximity to one or more of the current or historic smelters, and properties with elevated levels of arsenic often occurred immediately adjacent to one or more residences that were not apparently affected.

In order to obtain additional information on the spatial pattern of contamination both within and between yards, USEPA selected eight properties to undergo detailed soil sampling. Five of the yards were locations where Phase I/Phase II sampling indicated the arsenic concentrations were above the removal level, while three of the properties had arsenic concentrations below the removal level. At each property, a high-density grid was established on 5-foot centers, and soil samples were collected wherever the grid node did not fall on a driveway, patio, etc. In addition, whenever access could be obtained, the sampling grid was extended 10-15 feet into adjacent properties in order to determine if there was a clear difference in contamination levels between adjacent properties.

The results for one property are shown in Figure ES-2. As seen at this location, there is a fairly clear boundary between the property of concern and the adjacent properties. Similar patterns are observed at other properties, although there are some locations where the contamination may extend somewhat into the adjacent property.

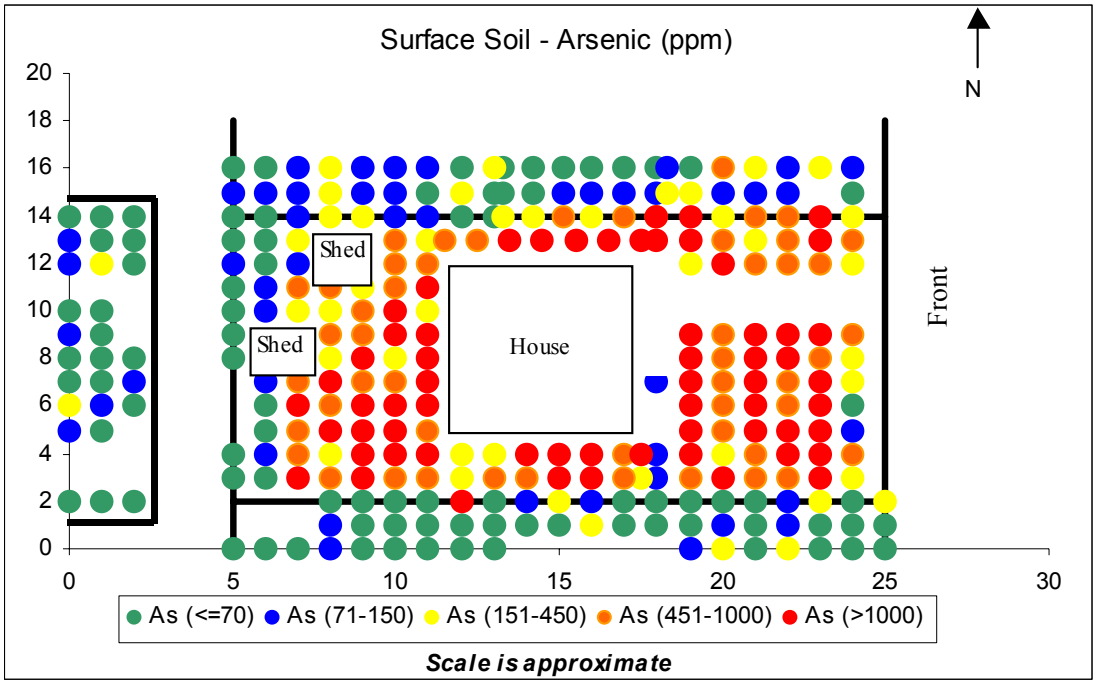
Other activities conducted under the Risk-Based Sampling Program included collection of a number of environmental samples (dust, water, paint, vegetables) at the eighteen properties selected for soil removal. Arsenic and lead levels in indoor dust were found to have no apparent relationship to levels in yard soil, suggesting that soil was not a predominant source of contaminant levels in indoor dust. Lead levels in tap water were all below the current USEPA action level for lead in drinking water (15 ug/L), suggesting that tap water is not likely to be a significant source of exposure. Lead was detected in paint at most locations, with 130 out of 144 samples having values above 1 mg/cm^2. These data suggest that interior and/or exterior leaded paint might be a source of lead exposure in area children, either directly (by paint chip ingestion), or indirectly (by ingestion of dust or soil containing paint chips). Only one of the 18 properties scheduled for soil removal had a vegetable garden. At this location, concentrations of arsenic and lead were below the level of detection in two vegetable samples. Because so few vegetable samples were obtained, no conclusions can be drawn from this data set.

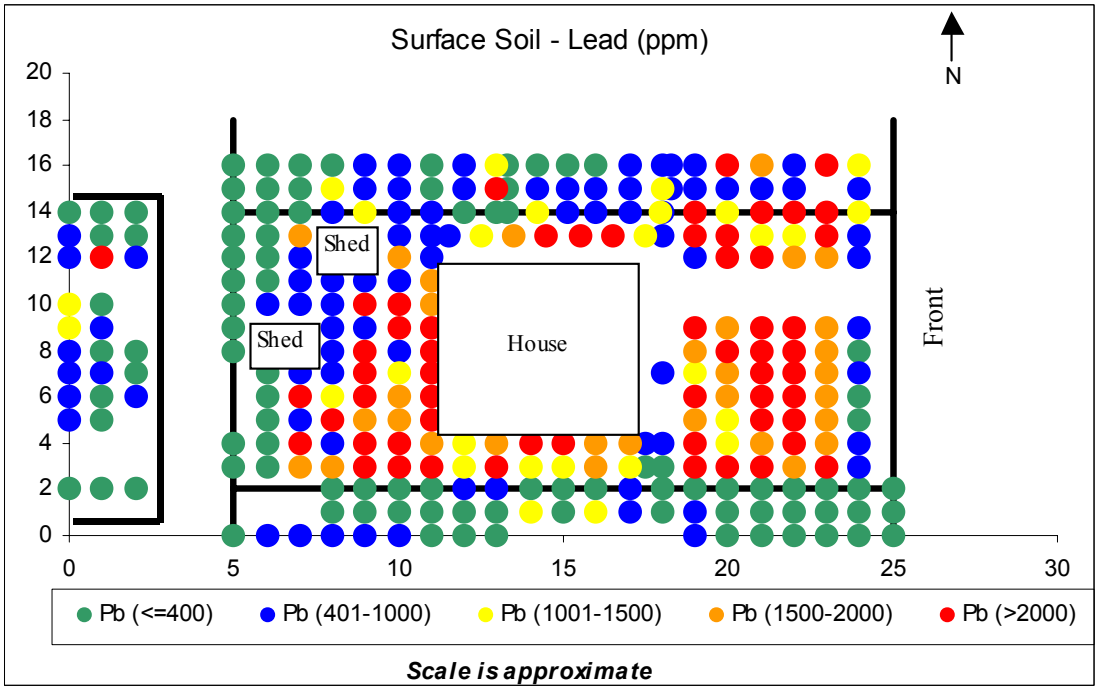
In addition to environmental sampling, a number of biological samples (hair, urine, blood) were also collected from residents in the properties selected for soil removal. A total of 15 individuals residing at six of the properties scheduled for soil removal volunteered to participate in the program. None of the samples collected exceeded the normal range for lead or arsenic. Although this data set is too small to draw firm conclusions, the results provide no indication that exposures at these locations were of immediate health concern.

Physical-Chemical Characterization

USEPA also undertook two studies to characterize the physical chemical attributes of the lead and arsenic contamination in residential site soils. These studies found that arsenic in site soils occurs mainly as arsenic trioxide, with a smaller but significant contribution from lead arsenic predominant form accounting for elevated lead levels in yard soils. Levels of lead phosphate and lead manganese oxide also tend to increase as total lead concentrations increase, but these phases may be secondary weathering products derived from the lead arsenic oxide.

Figure ES-2 Spatial Distribution of Contaminants–Property 1





In addition, the concentration of metals in bulk (unsieved) soil samples were compared to that in fine (sieved) samples. The slope of the best fit regression line through the paired data set was close to 1.0 for zinc, but was slightly higher for arsenic (slope = 1.21), lead (slope = 1.09) and cadmium (slope = 1.13). In all cases, these slopes were statistically different from 1.0 ($p < 0.001$). This indicates that the concentration of arsenic, lead and cadmium is about 10-20% higher in fines than in bulk samples of soil.

2.2 Selection of Chemicals of Potential Concern

Chemicals of potential concern (COPCs) are chemicals which a) are present at a site, b) occur at concentrations which are or might be of health concern to exposed humans, and c) are or might be due to releases from a Superfund site. USEPA assumes that any chemical detected at a site is a candidate for selection as a COPC, but identifies a number of methods that may be used for determining when a chemical is not of concern and may be eliminated from further consideration. Each risk assessment may choose to apply some or all of the methods identified by USEPA to select COPCs, as appropriate.

At this site, COPCs were selected based on available data from full-suite analyses of soil samples for the 23 metals included on USEPA's Target Analyte List (TAL). In accord with standard methods identified in USEPA risk assessment guidance, chemicals were eliminated if: a) the maximum value was below a level of health concern, b) the chemical is a beneficial mineral that is required for good health, and c) if the risk contributed is minor compared to other chemicals that will be retained. Based on these selection procedures, the COPCs selected for quantitative evaluation at the VBI70 site are arsenic and lead. All other chemicals measured in soil are either not of concern or are present at levels which contribute minimal risk compared to arsenic and lead.

2.3 Phase III Investigation

Because of the absence of any clear spatial pattern of soil contamination, USEPA concluded that the identity and location of properties with elevated levels of arsenic and/or lead could not be reliably predicted using traditional approaches, and that sampling of every yard was necessary. For this reason, USEPA undertook a large-scale sampling program designed to obtain data that would help evaluate health risks to residents in the area. This program is referred to as the Phase III investigation. The investigation consisted of four main parts:

- Sampling of residential yard soils
- Sampling of indoor dust at residences
- Sampling of residential vegetable gardens (vegetables and soil)
- Supplemental sampling of soil at local schools and parks

Phase III was implemented in two parts. The first part, referred to as Phase IIIa, focused mainly on properties (including residences, schools, and parks) which had not been investigated in Phases I or II, including a large portion of both the Cole and Clayton neighborhoods. The

second part, referred to as Phase IIIb, consisted of re-sampling at properties that had previously been sampled in Phase I or II, but for which the data were judged to be too limited to support clear risk-management decision making. This risk assessment is based on the combined data from Phase IIIa and IIIb.

Residential Soil Sampling

A total of 30 surface soil (0-2 in.) grab samples were collected from each property where access was granted. These 30 samples were combined into three composite samples, each containing 10 grab samples. The composites were prepared by combining every third grab sample, such that each composite represents an independent estimate of the yard-wide mean concentration. All composite samples were dried and mixed, and then analyzed for arsenic and lead by XRF.

The total number of properties sampled in Phase III was 2,986. Summary statistics, based on average values at each property and stratified by neighborhood, are summarized in Table ES-1. For arsenic, most properties (2,471 out of 2,986 = 83%) have average concentrations of 50 ppm or less, with 258 properties (9%) between 50-100 ppm, 183 (6%) between 100-200 ppm, and 74 (2%) above 200 ppm. For lead, 2,712 (91%) properties have mean lead concentrations lower than 400 ppm, with 266 (9%) between 400-800 ppm and 8 (0.3%) higher than 800 ppm. There is only a weak correlation between the occurrence of elevated lead and elevated arsenic in soil, suggesting that the main sources of lead and the main sources of arsenic in yard soil are not likely to be the same.

Residential Dust Sampling

In accord with the initial results obtained during the Risk-Based sampling program, only a weak correlation was detected between the level of either arsenic or lead in paired soil and dust samples ($R^2 = 0.14$ to 0.18, respectively). Nevertheless, the slopes of both regression lines are statistically different from zero ($p < 0.01$), with best estimate parameter values as follows:

Arsenic: $C_{dust} = 0.06 \cdot C_{soil} + 11$

Lead: $C_{dust} = 0.34 \cdot C_{soil} + 150$

These equations were used to estimate the concentration of arsenic and lead in dust at each property based on the measured values in soil.

USEPA collected 72 samples of different types of garden vegetables from 19 different properties around the site. Each vegetable sample was washed in de-ionized water to minimize the amount of adhering soil. Vegetables were not peeled before analysis. At each location where a vegetable sample was collected, a co-located sample of garden soil was also collected.

Table ES-1 Property Mean Summary Statistics for Phase III Soil Samples
Residential Garden Sampling

ARSENIC

Neighborhood	Total Properties	Distribution of Yard Average Concentration Values for Arsenic (ppm) (a)					
		5th	25th	50th	75th	95th	Maximum
Clayton	902	5.5	5.5	8.7	38.3	168.0	758
Cole	796	5.5	7.7	11.8	24.8	142.1	660
Elyria	59	5.5	8.5	12.3	22.3	97.2	431
Globeville	63	5.5	8.5	13.8	22.3	123.3	297
Swansea	1166	5.5	5.5	9.7	30.6	128.3	604
ALL	2986	5.5	5.5	10.5	30.3	144.9	758

LEAD

Neighborhood	Total Properties	Distribution of Yard Average Concentration Values for Lead (ppm) (a)					
		5th	25th	50th	75th	95th	Maximum
Clayton	902	76	106	140	193	337	1131
Cole	796	135	221	288	371	538	1130
Elyria	59	181	299	372	438	601	922
Globeville	63	171	257	332	482	633	835
Swansea	1166	76	119	164	250	410	776
ALL	2986	81	127	188	292	465	1131

(a) Yard average is the mean of composites collected from the yard

For arsenic, the mean concentration in vegetables (averaged across all samples) was 0.043 mg/kg wet weight (43 ng/g ww). One data point (an onion sample from property 6) appears to be substantially higher than expected based on the other samples. The basis for this apparently high value is not known, but might be attributable to incomplete removal of soil from the sample prior to analysis. If that sample is considered to be an outlier and is excluded, then the mean concentration of arsenic in vegetables is 30 ng/g wet weight. The slope of the best-fit regression line through the data (outlier excluded) is quite low (0.0014 mg/kg wet weight per mg/kg in soil), but the slope is statistically different from zero ($p < 0.001$).

For lead, the mean concentration across all samples was 0.15 mg/kg wet weight (150 ng/g ww). Again, one data point (a garlic sample from property 11) appears to be substantially higher than expected based on the other samples. If that sample is considered to be an outlier and is excluded, then the mean concentration of lead in vegetables is 62 ng/g wet weight. The slope of the best-fit regression line through the data (outlier excluded) is not statistically different from zero ($p > 0.5$).

There is only a weak relationship between the concentration of arsenic in yard soil and in garden soil (slope = 0.066, $R^2 = 0.265$), although the slope is statistically different from zero ($p < 0.01$). For lead, both the slope (0.60) and the correlation ($R^2 = 0.410$) are somewhat higher that for arsenic, but the correlation is still rather weak. These results indicate that garden soil is not equivalent to yard soil, with levels of arsenic and lead tending to be lower in the gardens than in the yard. This might be because the garden soil is prepared by amending yard soil with clean soil, peat moss, or other additives that dilute the yard soil contaminant level, or because the source(s) that have affected the yard did not equally affect the gardens.

Sampling at Schools and Parks

Samples of surface soil were collected at 10 schools and one park. Concentrations of arsenic are generally low, with average values ranging from 11-14 ppm, and maximum values less than 25 ppm. An exception to this pattern occurred at one school property where two values significantly higher than expected were detected (1,517 ppm and 70 ppm). These values occurred adjacent to each other, and were surrounded by values of 17-23 ppm, indicating the presence of a small "hot spot". Even though no children were exposed at this area, EPA Region VIII has worked with the property owner to address this area of contamination.

2.4 Data Selected For Use in This Risk Assessment

The data from the Phase III sampling program were selected for use in this risk assessment because 1) all Phase III data were collected in accordance with project plans that were developed with careful consideration of the Data Quality Objectives (DQOs) needed to support risk assessment calculations, and 2) all data collected during Phase III are accompanied by Quality Assurance (QA) samples that allow detailed evaluation of the reliability of the data. These

quality assurance data (USEPA 2000e) reveal that the data collected are of high quality, with adequate accuracy and precision to support a reliable evaluation of human health risk.

Data collected during Phase I/Phase II were not used because they were collected only with the intent of identifying locations that exceeded the removal action levels, and were not intended to support detailed risk calculations or remedial decision making. More specifically, data from Phase I/Phase II were not used because 1) many samples had elevated detection limits for arsenic (average = 51 ppm, range = 44 to 800 ppm), 2) the sampling density at each property was sometimes too low to ensure representativeness, and/or 3) exact sampling locations within a property were not always clear. However, despite these limitations, it is clear that the data from Phase I/Phase II and from Phase III are generally similar, each indicating the occurrence of scattered properties with elevated levels of lead and/or arsenic.

3.0 EXPOSURE ASSESSMENT

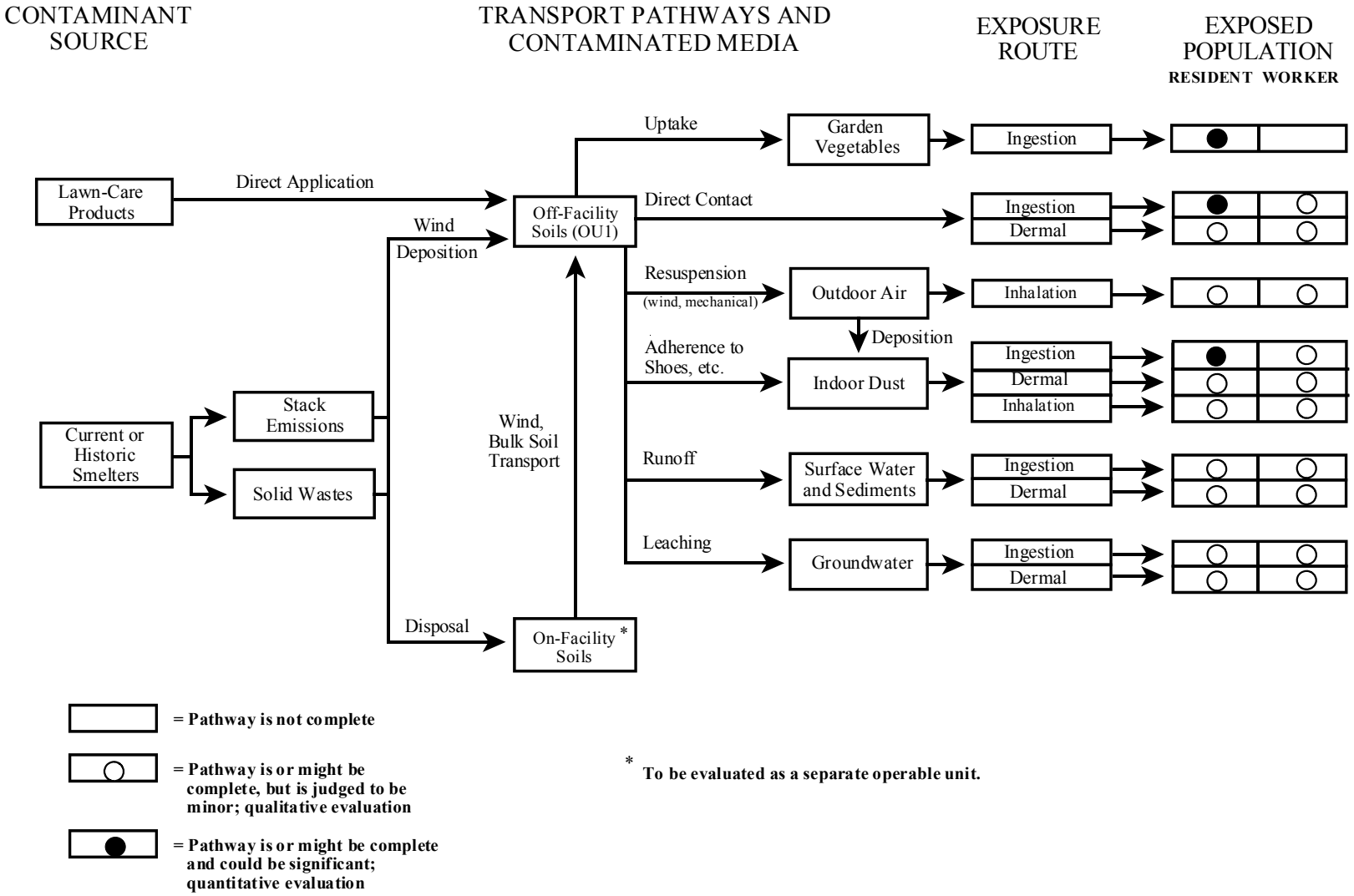
Figure ES-3 presents a conceptual model showing the main pathways by which contaminants present in surface soil may come into contact with area residents. This conceptual model was developed in consultation with local community groups as well as representatives from the City and County of Denver, the Colorado Department of Public Health and Environment, and the Agency for Toxic Substances and Disease Registry. Exposure scenarios that are considered most likely to be of concern are shown by boxes containing a solid circle, and greatest attention is focused on these pathways. Pathways which are judged to contribute only occasional and minor exposures are shown by boxes with an open circle. Incomplete pathways (i.e., those which are not thought to occur) are shown by open boxes. Based on this conceptual model, the following pathways are judged to be of sufficient potential concern to warrant quantitative exposure and risk analysis for this Operable Unit (OU1):

Exposure Pathways of Potential Concern for Quantitative Risk Analysis

Population	Medium and Exposure Route
Resident	Incidental ingestion of soil and dust in and about the home and yard Ingestion of home-grown vegetables

Other exposure pathways are judged to be sufficiently minor that further quantitative evaluation is not warranted.

Figure ES-3 Conceptual Site Model for Operable Unit 1
Exposure to Off-Facility Soils
Revision 2



4.0 QUANTIFICATION OF EXPOSURE AND RISK FROM ARSENIC

4.1 Quantification of Exposure

It is expected that different individuals who live in the VBI70 site will have a range of different exposure levels to arsenic. This is because they have different intake rates of soil, dust and vegetables, and live in areas of differing arsenic concentration. The risk assessment estimated the exposure for two different types of resident: a resident with average exposure, and one at the high end of the exposure distribution. These two cases are referred to as Central Tendency Exposure (CTE) and Reasonable Maximum Exposure (RME). Estimates of exposure for the CTE and RME cases were calculated for three different exposure scenarios: long-term (chronic/lifetime), short-term (subchronic), and acute (pica). Standard exposure equations identified in USEPA risk assessment guidance were used in all cases. When applicable, EPA defaults were used for exposure parameter input values. In accord with Agency guidelines, when reliable site-specific exposure data were available, these data were used in place of default exposure assumptions. All concentration values in soil, dust and garden vegetables were based on site-specific measurements.

4.2 Toxicity Assessment

The toxic effects of arsenic have been reasonably well established, based mainly on studies of humans exposed to elevated levels of arsenic from a variety of sources. The main effects are summarized below.

Acute Noncancer Effects

Very high doses of arsenic may cause acute lethality, but such exposures from environmental sources are very unlikely. Oral exposure to non-lethal but high acute doses of arsenic produces marked irritation of the gastrointestinal tract, leading to nausea and vomiting. Other signs may include neuritis and vascular effects.

Subchronic Noncancer Effects

Symptoms resulting from sub-chronic ingestion of lower doses of arsenic often begin with a vague weakness and nausea. As exposure continues, symptoms become more characteristic and may include signs such as diarrhea, vomiting, anemia, injury to blood vessels, damage to kidney and liver, and impaired nerve function that leads to "pins and needles" sensations in the hands and feet.

Chronic Noncancer Effects

Chronic exposure to arsenic is associated with all of the effects noted above. In addition, after exposure continues for a sufficient period of time, an unusual pattern of skin abnormalities, including dark and white spots and a pattern of small "corns" may occur, especially on the palms and soles.

Carcinogenic Effects

There is strong evidence from a number of human studies that oral exposure to arsenic increases the risk of skin cancer. The most common type of cancer is squamous cell carcinoma, which appears to develop from some skin corns. In addition, basal cell carcinoma may also occur, typically arising from cells not associated with the corns. Although these cancers may be easily removed, they can be painful and disfiguring and can be fatal if left untreated. More recent data indicate that chronic oral arsenic exposure also increases the risk of several types of internal cancer, including cancer of the bladder and lung.

Toxicity Factors for Arsenic

Based on the available toxicity data for arsenic, the USEPA has established a series of Reference Doses (RfDs) for evaluating risk of non-cancer effects, and a cancer slope factor for quantifying the risk of cancer. These values are summarized below.

USEPA Arsenic Toxicity Factors Utilized in the Risk Assessment

Toxicity Factor	Value	Source
Acute RfD	0.015 mg/kg-day	USEPA 2001f
Subchronic RfD	0.006 mg/kg-day	USEPA 1995b
Chronic RfD	0.0003 mg/kg-day	IRIS 2000
Oral Slope Factor	1.5 $(mg/kg\text{-}day)^{-1}$	IRIS 2000

Because the oral RfD values and the oral SF for arsenic are based on studies of humans exposed to arsenic either in drinking water or in other readily absorbable forms, solid forms of arsenic in site soils may be less well-absorbed and require adjustments in the toxicity factors to derive appropriate estimates of toxicity. In order to investigate the relative bioavailability (RBA) of arsenic in site soils, USEPA performed a study in which five separate samples were fed to swine for 12 days. The study found that arsenic in site soils was less well absorbed than a readily soluble form of arsenic (sodium arsenate), with RBA values for individual samples ranging from about 0.18 to 0.45, with a mean value of 0.31 for all site samples. In order to be conservative, exposure calculations were based on the upper confidence limit of the RBA for arsenic in site soils (0.42).

4.3 Risk Characterization for Arsenic

Risks from Soil and Dust

Cancer Risk

Cancer risks from exposure of residents to arsenic in yard soil and indoor house dust were calculated for each property using the basic equations recommended by USEPA. The risk

estimates are expressed as the probability that an individual exposed to arsenic at the site will develop a cancer by the age of 70 that would not otherwise have occurred. For example, a cancer risk of 2E-05 means that the probability is 2 out of 10^5 (2 out of 100,000) that the exposed individual might develop a tumor from site-related exposures. The results of these calculations are shown in Table ES-2.

For CTE exposure conditions, most properties have estimated excess cancer risks for exposures due to arsenic in soil plus dust that range from 1E-06 to 1E-05 (5th to 95th percentiles), with a maximum value of 9E-05. For RME exposure conditions, most properties have risks that range from 9E-06 to 1E-04 (5th to 95th percentiles), with 92 properties having risks of 2E-04 or higher. The highest RME risk value was 8E-04. The spatial pattern of properties with arsenic RME cancer risk levels of 2E-04 or higher is approximately uniform across the site, with a frequency of about 1%-4% in each neighborhood.

In interpreting these risk estimates, it is important to recognize that arsenic is a naturally occurring element in soil. Based on an analysis of the distribution of concentration values observed in Phase III soil samples, it is estimated that background levels are well-characterized as a lognormal distribution with a mean of 8 ppm and a standard deviation of 3.6 ppm. Based on this, background levels may range up to about 15 ppm or slightly higher. If so, lifetime cancer risks from naturally occurring levels of arsenic probably range from about 1E-06 for an average (CTE) person up to about 1E-05 for an upper-bound (RME) individual.

Chronic Noncancer Risks

In accord with standard EPA methods, the risk of non-cancer effects is expressed as the ratio of the dose resulting from exposure to site media compared to a dose that is believed to be without risk of effects, even in sensitive individuals. This ratio is called the Hazard Quotient (HQ). If the value of HQ is equal to or less than one (1E+00), it is believed there is no significant risk of noncancer effects. If the HQ exceeds one, then there is a chance that noncancer effects may occur, with the probability tending to increase as the value of HQ increases.

Estimated risks of non-cancer health effects from chronic exposure to arsenic in soil and dust are shown in Table ES-3. For individuals with CTE exposure, risks at most properties fall between 2E-02 and 2E-01 (5th to 95th percentile), while individuals with RME exposure have risks that lie mainly between 5E-02 and 6E-01. These results indicate that risk of noncancer effects from chronic exposure is below a level of concern for most individuals at most locations. However, a total of 20 properties have RME HQ values of 2E+00 or higher, with a maximum value of 4E+00. These locations where noncancer risks enter a range of concern (HQ > 1E+00) are also above the usual level of concern (1E-04) for cancer.

Subchronic Noncancer Risks

Estimated risks of non-cancer health effects from sub-chronic exposure of area children to arsenic in soil are shown in Table ES-4. As seen, the incidence of properties with subchronic HQ values above 1E+00 is relatively low (2 out of 2,986 = 0.07% for CTE individuals, 53 out of

Table ES-2 Estimated Cancer Risk from Arsenic in Soil and Dust

Neighborhood	Number of Properties Evaluated	Number and Percent of Properties Within the Specified Risk Range							
		CTE Cancer Risk				RME Cancer Risk			
		<=1E-05	2E-05 - 1E-04	2E-04 - 1E-03	> 2E-03	<=1E-05	2E-05 - 1E-04	2E-04 - 1E-03	> 2E-03
Clayton	902	858	44			479	385	38	
		95%	5%			53%	43%	4%	
Cole	796	772	24			344	429	23	
		97%	3%			43%	54%	3%	
Elyria	59	58	1			17	41	1	
		98%	2%			29%	69%	2%	
Globeville	63	61	2			25	36	2	
		97%	3%			40%	57%	3%	
Swansea	1166	1132	34			610	528	28	
		97%	3%			52%	45%	2%	
All Neighborhoods	2986	2881	105			1475	1419	92	
		96%	4%			49%	48%	3%	

CTE=Central Tendency Estimate

RME=Reasonable Maximum Exposure

Table ES-3 Estimated Chronic Noncancer Risk from Arsenic in Soil and Dust

Neighborhood	Number of Properties Evaluated	Number and Percent of Properties Within the Specified Risk Range							
		CTE Hazard Quotient				RME Hazard Quotient			
		<=1	2-5	6-10	> = 11	<=1	2-5	6-10	> = 11
Clayton	902	901	1	--	--	895	7	--	--
		100%	0.1%	--	--	99%	0.8%	--	--
Cole	796	796	0	--	--	786	10	--	--
		100%	0%	--	--	99%	1.3%	--	--
Elyria	59	59	0	--	--	59	0	--	--
		100%	0%	--	--	100%	0%	--	--
Globeville	63	63	0	--	--	63	0	--	--
		100%	0%	--	--	100%	0%	--	--
Swansea	1166	1166	0	--	--	1163	3	--	--
		100%	0%	--	--	100%	0.3%	--	--
All Neighborhoods	2986	2985	1	--	--	2966	20	--	--
		100%	0%	--	--	99%	0.7%	--	--

CTE=Central Tendency Estimate

RME=Reasonable Maximum Exposure

Table ES-4 Estimated Subchronic Noncancer Risks from Arsenic in Soil

Neighborhood	Number of Properties Evaluated	Number and Percent of Properties Within the Specified Risk Range							
		CTE Hazard Quotient				RME Hazard Quotient			
		<=1	2-5	6-10	> = 11	<=1	2-5	6-10	> = 11
Clayton	902	900	2			881	19	2	
		100%	0.2%			98%	2%	0.2%	
Cole	796	796	0			777	19	0	
		100%	0%			98%	2%	0.0%	
Elyria	59	59	0			58	1	0	
		100%	0%			98%	2%	0.0%	
Globeville	63	63	0			62	1	0	
		100%	0%			98%	2%	0.0%	
Swansea	1166	1166	0			1155	11	0	
		100%	0%			99%	1%	0.0%	
All	2986	2984	2			2933	51	2	
		100%	0.1%			98%	2%	0.1%	

2,986 = 1.8% for RME individuals). The maximum RME HQ value was 7E+00. All of the locations where subchronic noncancer risks enter a range of concern (HQ > 1E+00) are also above the usual level of concern (1E-04) for cancer.

Noncancer Risks from Acute Pica Behavior

Because of the substantial uncertainty which exists in most of the input parameters for the acute pica scenario, it is not possible to specify a single set of inputs that are "best". Rather, a range of HQ values were calculated for two different combinations of soil intake and RfD values:

Alternative Pica Exposure and Toxicity Values

Variable	**Case 1**		**Case 2**	
	CTE	**RME**	**CTE**	**RME**
Soil intake (mg/day)	5000	10000	2000	5000
Acute RfD (mg/kg-d)	0.005		0.015	

Case 1: RfD = 0.005 mg/kg; Pica soil intake = 10,000 mg/event
Case 2: RfD = 0.015 mg/kg; Pica soil intake = 5,000 mg/event

It should be understood that these cases represent an uncertainty range, and that the "true" acute risk from pica behavior could lie anywhere in the interval. Indeed, it is quite possible that the true value even lies outside the range, since the actual distribution of pica soil intakes is not known.

The results are summarized in Table ES–5. As seen, the screening calculations above suggest that a large number of properties (ranging from 662 to 1841, depending on which set of input assumptions is deemed to be most appropriate) are of potential concern for the RME acute pica scenario.

Because data are so sparse on the actual magnitude and frequency of soil pica behavior, and considering that discussions continue to occur nationally on the most appropriate acute RfD for arsenic, and it is difficult to judge which (if any) of these properties should be considered to be an authentic acute health risk to children. In this regard, it should be noted that even though many people are exposed to arsenic levels in soil that are predicted to be of acute concern, both within the VBI70 site and elsewhere across the country and around the world, to the best of USEPA's knowledge, there has never been a single case of acute arsenic toxicity reported in humans that was attributable to arsenic in soil. Thus, these results for the acute pica scenario are considered to be especially uncertain, since they predict a very substantial risk for which there is no corroborating medical or epidemiological evidence.

Table ES-5 Estimated Acute Noncancer Risk from Pica Behavior

Exposure Assumptions	Number and Percent of Properties Within the Specified Risk Range									
	CTE Hazard Quotient					RME Hazard Quotient				
	<=1	2-5	6-20	> 20	Total > 1	<=1	2-5	6-20	> 20	Total > 1
Case 1	1475	949	432	130	1511	1145	580	328	933	1841
	49%	32%	14%	4%	51%	38%	19%	11%	31%	62%
Case 2	2692	268	26	0	294	2324	487	162	13	662
	90%	9%	1%	0%	10%	78%	16%	5%	0%	22%

Case 1: RfD = 0.005 mg/kg; Pica intake rate = 10,000 mg

Case 2: RfD = 0.015 mg/kg; Pica intake rate = 5,000 mg

Risks from Home-Grown Vegetables

A total of 72 different samples of garden vegetables were collected from 19 different properties across the site. At each property, the 95% upper confidence limit (UCL) of the mean concentration of arsenic was calculated, and this value (or the maximum, whichever was lower) was used to estimate risks to residents. For individuals whose intake of home-grown garden vegetables is average (CTE) for the western United States, neither non-cancer nor cancer risks enter a range of concern at any property tested. For individuals whose intake is at the upper-bound (RME) of the distribution of garden vegetable consumption, cancer and non-cancer risks do enter a range of potential concern for two properties. However, these risks were driven either by a single value that appeared to be anomalous, or by the margin of safety introduced by use of the 95% UCL. Overall, it appeared that while risks from arsenic in garden vegetables could not be entirely excluded, the risks were likely to be low. This is supported by noting that the intake of arsenic from home-grown vegetables is predicted to be well within the normal dietary range observed in the United States.

Total Risks for Ingestion of Soil and Home-Grown Vegetables

As noted above, data on arsenic levels in soil are available for all 2,986 properties investigated in Phase III, but data on arsenic levels in gardens and vegetables were collected only at 19 of these properties. Therefore, in order to calculate total risk at all properties, it was necessary to estimate the concentration of arsenic in garden vegetables using site-specific data on the relationship between arsenic in yard soil and in garden soil, and between arsenic in garden soil and in vegetable tissues.

Because exposure and risk from soil ingestion and vegetable ingestion are both distributions, care must be taken in the summation process. In the case of the non-cancer or cancer risk to an individual who has average exposure to both soil and vegetables, the total risk is simply the sum of the two pathway-specific risks:

CTE(total) = CTE(soil) + CTE(vegetables)

In the case of an individual who has RME exposure to soil or to vegetables, the estimate of RME total risk is not the simple sum of the RME risk estimates, because the two pathways are independent of each other, and an individual with RME soil intake is not likely to also have RME vegetable intake (and vice versa). Thus, the estimate of RME total risk is calculated either as:

1: RME(total) = RME(soil) + CTE(vegetables)
2: RME(total) = CTE(soil) + RME(vegetables)

The results are shown in Table ES-6. As seen, based on the site-specific relationships between arsenic in yard soil and garden soil and between arsenic in garden soil and garden vegetables, individuals with CTE exposure to garden vegetables are predicted to have excess cancer risks that are less than or equal to 1E-05, while individuals that have RME intake of garden vegetables

Table ES-6 Estimated Total Cancer Risks from Soil and Vegetables

Statistic	Pathway	Number of Properties		
		<= 1E-05	2E-05 - 1E-04	2E-04 - 1E-03
CTE Risk	Soil alone	2881	105	
	Vegetables alone	2986		
	CTE Soil + CTE vegetables		2921	65
RME Risk	Soil alone	1475	1419	92
	Vegetables alone		2979	7
	RME Soil + CTE vegetables[a]	933	1954	99
	CTE Soil[a] + RME vegetables		2921	65

[a] Adjusted to account for RME exposure duration (30 years)

are expected to have risks mainly between 2E-05 and 1E-04, with only a few properties having risks that exceed 1E-04. When CTE risks are combined across pathways, there are 65 properties where total risk exceeds 1E-04. When RME risks are combined across pathways, the highest risks occur for case 1 (RME soil intake plus CTE vegetable intake). Based on this scenario, there are 99 properties where total RME risks exceed 1E-04.

4.4 Uncertainties in Arsenic Risk Assessment

It is important to recognize that the calculations of short-term and long term exposure and risk from arsenic ingestion in soil are based on a number of assumptions and estimates, and that these introduce uncertainty into the risk results. The most important of the sources of uncertainty in the calculations are summarized below.

Uncertainty in Yard-Wide Average Concentration

The concentration term that is appropriate for calculating chronic exposure and risk from ingestion exposure to arsenic is the true mean concentration in the medium of concern (soil, dust, vegetables), averaged over the area and time interval (averaging time) of concern. There are two important sources of uncertainty in this value. First, because the true mean cannot be calculated from a limited set of sample results, the USEPA utilizes the 95% upper confidence limit of the mean as a conservative (high end) estimate of the true mean. This approach helps ensure that the exposure and risk estimates that are derived are more likely to overestimate than underestimate the actual risk. Second, the basic exposure unit selected for evaluation in this risk assessment is the residential property. Using the UCL of the mean for a property is equal to assuming that an individual residing at that location does not ingest soil or dust from any other location, even over a time period of up to 30 years. While this might be true for a small sub-set of residents, it is believed that most residents are sufficiently mobile that exposures will occur over a wider area than just their own yard. This, in turn will result in lower exposures for people residing in homes with affected soils, and their true risks will be lower than calculated.

Uncertainty in Concentration Values at Sublocations

As noted earlier, the sampling and analysis design for Phase III was based on a set of three composite samples from each property. Consequently, there are no data that allow a direct estimation of the concentration value at any specific sub-location of the yard (these are needed to address risks from subchronic and acute exposures). To address this data limitation, the distribution of concentration values within a property was modeled by assuming a lognormal distribution, and the standard deviation within each property was estimated from a site-wide average coefficient of variation. Since the mean at each property was estimated using the 95% UCL or the maximum composite value, both the mean and the standard deviation are more likely to be high than low at each property. Thus, the values estimated for evaluation of subchronic and acute exposures are also more likely to be high than low.

Uncertainty in Intake Rates

Data on the amount of soil ingested by humans are very limited. Measurements are difficult to perform, and results vary significantly from study to study and from method to method. In addition, data are based mainly on short term studies, so estimates of long-term average intake rates are especially uncertain. Moreover, intake rates are likely to vary from site to site and property to property, depending on things such as climate, socioeconomic status, yard condition, etc., so the default intake rates used in these calculations may not reflect the true intake rates at the site. Because of the limitations in the data, the default values recommended by USEPA are intended to be on the high side (i.e., are more likely to overestimate than underestimate actual soil ingestion).

This is illustrated by comparing the default soil intake rates used by USEPA to data on soil intake rates measured in a group of 64 children in Anaconda, Montana (Stanek and Calabrese 2000). This study, which utilizes the latest and most refined analytical and statistical methods for estimating soil ingestion by children, estimated that the average (CTE) 7-day intake by children is about 31 mg/day (compared to the default of 100 mg/day), and that the 95th percentile intake for 7 days and 365 days are 133 and 106 mg/day, respectively (compared to the default assumption of 200 mg/day). If these values from the Anaconda site were judged to be a more reliable basis for estimation of risk from soil ingestion than the current default values, and if adult soil intake is assumed to be about ½ that of children, then there are only 23 properties (rather than 92 properties) in the VBI70 site where RME cancer risks from soil ingestion exceed a level of 1E-04.

Uncertainty in the Fraction of Total Intake that is Soil

One of the variables used to calculate risks from ingestion of soil plus dust is the fraction of the total intake that is soil (f_s). The EPA default value for this variable (45%) is based mainly on measurements in a set of 64 preschool children, but due to the difficulty in making these measurements, as well as potential differences between children and between sites, this value should be considered uncertain. It is not known whether the true value at the VBI70 site is more likely to be higher or lower than the default values. If the true site-specific value of f_s were lower (e.g., 20% rather than 45%), risks would be about 12% lower than calculated. Conversely, if the true site-specific value were higher (e.g., 70% rather than 45%), then the risks would be about 12% higher than calculated.

Uncertainty in Exposure Duration

Cancer risk calculations depend on the duration of exposure. Default exposure durations used in the risk assessment are not site-specific, and are estimated from data on the length of time that people own a particular residence. Thus, actual exposure durations of residents at the site may not be the same as the assumed exposure durations assumed, and might be either longer or shorter than assumed. For example, if the exposure duration were assumed to be 45 years (6 years as a child and 39 years as an adult) rather than the default value of 30 years, the estimated excess cancer risk level from soil ingestion would be about 19% higher than the values reported.

In addition, all of the exposure calculations presented here assume that exposure begins during childhood, when intake rates are higher than during adulthood. Thus, risks to individuals who move to the site after they are children will be lower than estimated. For example, risks to an individual exposed for 30 years as an adult are only 37% of the risks to an individual exposed for 6 years as a child and 24 years as an adult.

Uncertainty in RME Exposures

In the default point estimate approach for estimating exposure and risk to an RME individual, two exposure parameters (intake rate and exposure duration) are both assumed to be at their 95th percentile values. In reality, because these two exposure parameters are independent of each other, it is very unlikely that an individual with RME soil intake will also have RME exposure duration. Therefore, an individual with both RME soil intake and RME exposure duration represents not the 95th percentile of the risk distribution, but some significantly higher percentile. One way to estimate what the percentile of the default RME individual is, as well as the actual 95th percentile value, is through Monte Carlo modeling. Screening level calculations performed with this approach suggest that the RME risk estimate derived by the point estimate approach is about twice the Monte Carlo estimate of the 95th percentile value, and is located at approximately at or above the 99th percentile of the risk distribution. This supports the conclusion that RME point estimates of risk provide a substantial margin of safety.

Uncertainty in Toxicity Factors

One of the largest sources of uncertainty in most risk assessments stems from uncertainty in the toxicity factors used to predict responses from the calculated doses. In the case of arsenic, dose-response data are derived from studies in humans, which significantly reduces the degree of uncertainty compared to extrapolations based on animal data. However, a significant degree of uncertainty still remains in both the oral cancer slope factor and the chronic RfD. One of the most important sources of this uncertainty is lack of reliable data on actual arsenic ingestion rates by the human population used to quantify risk. There are also still large uncertainties in how to extrapolate the dose-response curve from relatively high exposure levels to lower exposure levels. For example, arsenic does not appear to cause cancer by a direct genotoxic mechanism (USEPA 2001d), suggesting that a sub-linear (and perhaps even a threshold) model might be reasonable. However, in the absence of information on the actual mode of action, an assumption of linearity is still deemed to be necessary and appropriate (USEPA 2001d). If the dose response curve is sub-linear, current risk estimates would be too high. Further, there is uncertainty in the importance of cultural, ethnic, dietary, and socioeconomic differences between different study populations. While little is known about the relative importance of these factors, it is likely that there are differences between people in their sensitivity to ingested arsenic, and it is for this reason that USEPA seeks to ensure an adequate margin of safety in the derivation of the RfD and the slope factor.

Uncertainty in Bioavailability

In order to cause an adverse response, arsenic that is ingested must be absorbed into the body. Measurements of the arsenic relative bioavailability have been performed for five soils from the VBI70 site. While measurements based on site soils significantly reduces uncertainty in this exposure parameter, uncertainty still remains. For example, variability was observed between different site soils, and a conservative estimate of the mean value was employed to represent the site-wide average absorption. This approach is expected to result in an over-estimate of true absorption. Another source of uncertainty is in the extrapolation of data from test animals to humans. The test animals (swine) were selected because they are believed to have a gastrointestinal system similar to that in humans, but it is also expected that absorption in humans may vary as a function of age, stomach contents, nutritional status, etc. Thus, the measurements in animals should be viewed as uncertain estimates of the true values in humans.

The RBA measured for soil was also assumed to apply to dust. This assumption is uncertain because the size distribution of arsenic-containing particles in dust may be different than for soil, and particle size might be one factor that influences RBA. If dust contains smaller particles than soil, and if this size difference tends to increase RBA, then the use of the soil RBA could underestimate the absorption of arsenic from dust. However, it should be remembered that the RBA value for soil was measured using only the fine fraction of soil (only particles smaller than 250 micrometers in diameter), so the difference in particle size distribution between dust and soil is not expected to be large. In addition, because arsenic concentrations in dust tend to be lower than in soil, the dose contributed by dust ingestion is relatively small compared to that for soil, so uncertainty in the absorption fraction for dust results in only a small uncertainty in the total absorbed dose.

Uncertainty in Pica Exposure and Risks

As noted above, screening-level calculations suggest that acute high-dose exposures to arsenic in soil (i.e., pica exposure) might be of concern at a number of properties within the site. However, data on the amount of soil ingested during pica behavior are very sparse. Based mainly on one study that observed an intake of 5-8 g/day by a single child, (Calabrese et al. 1989), USEPA has indicated that 5-10 grams might be a reasonable estimate. If this intake rate is correct, and if arsenic absorption from this mass of soil is similar to that estimated in site-specific studies (42%), then anywhere from 22% to 62% of all properties within the VBI70 site (and perhaps outside the site as well) could have arsenic levels above a level of acute concern. USEPA feels this conclusion is especially uncertain, since the Agency is not aware of any reported cases of acute arsenic toxicity attributable to ingestion of arsenic in soil. A more recent study of soil intake did not observe intake rates above 700 mg/day in a group of 64 children, suggesting that values of 5-10 grams might be unrealistically high. In addition, limited data on urinary arsenic levels in residents of the VBI70 area and the nearby Globe neighborhood do not reveal the occurrence of high soil intakes by children. These considerations suggest that arsenic risk from soil pica may not be as significant as the calculations suggest. On the other hand, if this type of exposure were to occur, it is possible the symptoms (transient upset stomach and general malaise) would not be recognized as being arsenic-related, and could easily go un-detected or

un-reported. In addition, if pica behavior is assumed to occur only infrequently during childhood, then the chances of observing the behavior in a study could be quite low. Because of the high uncertainty regarding the magnitude and frequency of soil pica behavior, more reliable risk estimates for this scenario will not be possible until better data are collected on pica intakes, along with direct measures of soil-related exposures to arsenic in soil.

Summary of Uncertainties in Arsenic Risk Characterization

Because of the uncertainties summarized above, none of the exposure and risk calculations for arsenic should be interpreted as accurate measures of the true risk, rather, all values should be interpreted as uncertain estimates. Because a majority of the approaches for dealing with uncertainty are more likely to overestimate than underestimate true risk, the final risk values above should be thought of as more likely to be higher than lower than the actual risks.

5.0 EXPOSURE AND RISK FROM LEAD

5.1 Overview

Risks from lead are evaluated using a somewhat different approach than for most other metals. First, emphasis is placed on evaluation of risks to young children because they are more likely to be exposed and are more susceptible to the effects of lead than adults. Second, risks are expressed as the probability that a child will have a blood lead value greater than 10 ug/dL. A blood lead of 10 ug/dL is a value identified by EPA as the level at which effects that warrant avoidance begin to occur, and EPA has set as a goal that there should be no more than a 5% chance that any child will have a blood lead value above 10 ug/dL.

5.2 IEUBK Model for Assessing Lead Risk

Risks from Soil and Dust

The USEPA has developed an Integrated Exposure Uptake Biokinetic (IEUBK) model for predicting the likely range of blood lead levels in a population of young children (age 0-6 years) exposed to a specified set of environmental lead levels. The IEUBK model was used to predict risks at each property that was sampled during Phase III, using the mean of the three composite values from each property as the best estimate of the average bulk lead concentration in soil at each property. This value was adjusted by a factor of 1.09 to estimate the concentration in fine soil. Other input parameters for the IEUBK model were the defaults recommended by EPA except for two site specific inputs: 1) the concentration of lead in dust as a function of the concentration in bulk soil, which were based on site-specific measurements, and 2) the relative bioavailability of lead, which was based on a test of site soils in an animal study. The site specific RBA was 84%, higher than the default assumption of 60%.

The IEUBK model was used to calculate the expected blood lead distribution for children (age 0-84 months) for each property. The results, characterized in terms of the probability of a random child exceeding a blood lead value of 10 ug/dL (this is referred to as "P10"), are shown in Table ES-7. As seen, a total of 1,655 out of 2,986 homes are predicted to have P10 values at or below the health-based goal of 5%, while 1,331 (45%) are predicted to exceed the health-based goal. Approximately 610 properties are predicted to have P10 values of 5-10%, slightly above the heath-based goal. However, about 518 properties would be expected to have P10 values between 10-20%, and 203 homes are predicted to have P10 values greater than 20% (substantially above the health-based goal). It should be noted that 1,057 of the 1,331 properties (79%) with P10 values above 5% have mean bulk lead concentrations lower than 400 ppm (the USEPA default level of concern). This is mainly because the site-specific RBA for lead (84%) is higher than the default value (60%), and also because of the use of the concentration value in the fine fraction rather than the bulk fraction in the risk calculations.

Although homes with elevated soil lead are found in all neighborhoods, the density of homes with P10 values greater than 5% tends to be higher in the central and western part of the site than in areas on the eastern side of the site.

In interpreting these risk estimates, it is important to recognize that lead is a naturally occurring element in soil, and that there are many current and historic anthropogenic sources of lead (e.g., automobile exhaust, leaded paint, generalized industrial emissions, etc.). Based on the extensive soil data set collected during Phase III, levels of lead in bulk soils at the VBI70 site range from below the detection limit (about 52 ppm) up to a maximum of more than 1,000 ppm. If it is assumed that the upper range of the lead from natural and area-wide anthropogenic sources is about 400 ppm, then the mean of all samples that are less than 400 ppm is about 195 ppm. Using this value (195 ppm in bulk soil) as a rough estimate of the mean concentration in urban background samples, and assuming the same site-specific input values described above, the IEUBK model predicts that blood lead levels attributable to urban background levels of lead probably average about 4.4 ug/dL for a typical (median) child, and might be as high as 9.5 ug/dL for a child with above-average (95th percentile) exposure to soil or dust.

Risks from Lead in Garden Vegetables

As noted previously, site-specific data show there is essentially no detectable uptake of lead from soil into garden vegetables at this site. On this basis, it is concluded that exposure to lead from ingestion of home grown garden vegetables is not of concern.

5.3 Uncertainties in Lead Risk Evaluation

It is important to stress that lead risk predictions based on the IEUBK model are uncertain. This uncertainty arises from a number of factors. First, there is inherent difficulty in providing the model with reliable estimates of human exposure to lead-contaminated media. For example, exposure to soil and dust is difficult to quantify because human intake of these media is likely to be highly variable, and it is very difficult to derive accurate measurements of actual intake rates. Likewise, site-specific data on exposure to lead through the diet are generally not available, and

Table ES-7 Estimated Risks to Children from Lead in Soil and Dust

Neighborhood	Total Number of Properties	Number and Percent of Properties Within Specified Risk Range				
		P10 <= 5%	P10 > 5% and <= 10%	P10 > 10% and <= 20%	P10 > 20%	Total P10>5%
Clayton	902	712	119	52	19	190
	100%	79%	13%	6%	2%	21%
Cole	796	169	248	273	106	627
	100%	21%	31%	34%	13%	79%
Elyria	59	6	9	28	16	53
	100%	10%	15%	47%	27%	90%
Globeville	63	7	18	21	17	56
	100%	11%	29%	33%	27%	89%
Swansea	1166	761	216	144	45	405
	100%	65%	19%	12%	4%	35%
All	2986	1655	610	518	203	1331
	100%	55%	20%	17%	7%	45%

P10=Prediced Risk of Exceeding Blood Lead of 10 ug/dL

because dietary lead levels have been decreasing over time, the default data used in the model may no longer be accurate. Second, it is often difficult to obtain reliable estimates of key pharmacokinetic parameters in humans (e.g., absorption fraction, distribution and clearance rates, etc.), since direct observations in humans are limited. Finally, the absorption, distribution and clearance of lead in the human body is an extremely complicated process, and any mathematical model intended to simulate the actual processes is likely to be an over-simplification. Consequently, IEUBK model calculations and predictions should not be thought of as being identical to actual risk.

One way to help characterize the uncertainty that may exist in the IEUBK model calculations is to investigate the effect of alternative (non-default) model inputs for some of the more uncertain parameters. Especially important is the GSD value, which has a very powerful effect on the number of properties of concern. Studies at other sites have shown that the GSD value may often be lower than the default of 1.6, and if that were to be the case at this site, risks to children from lead could be substantially overestimated. Another parameter that is uncertain is the soil intake rate, and if data from the most recent study of soil intake in children were used in place of the default soil intake values, risks from lead would be below a level of concern at most locations.

Another way that may sometimes help assess whether the IEUBK model is yielding reliable results at a particular site is to compare the IEUBK model predictions with actual observations of blood lead levels in the population of children currently living at the site. At the VBI70 site, only very limited blood lead data are available, with values from only 21 individuals available. In this group of individuals, the maximum blood lead concentration observed was 5 ug/dL. While this the data set is much too limited to support the conclusion that risks are absent, neither do the results signal any cause for alarm. Data from several blood lead surveillance programs conducted by the State suggest that lead in soil does contribute to blood lead in area children, but that soil lead is not the primary reason for blood lead concentrations greater than 10 ug/dL.

6.0 CONCLUSION

Arsenic

Some residential properties at the VBI70 site contain arsenic at concentrations substantially higher than the expected natural levels. Properties with elevated levels of arsenic occur at widely scattered locations across the site, with no clear spatial pattern. At an affected property, the contamination appears to be distributed across the yard area, with a fairly clear boundary between the affected property and the adjacent properties. The chemical form of the arsenic is predominantly arsenic trioxide.

In some cases, levels of arsenic in yard soil is sufficiently elevated to pose an RME excess lifetime cancer risk that is above a level of 1E-04. Based on current data, about 3% of all properties fall into this category. Chronic and subchronic non-cancer risks from arsenic are also

above a level of human health concern at some properties, mainly at the same locations where cancer risks are above 1E-04.

Screening level calculations suggest that acute high level (pica) intake of soil by children might be of acute non-cancer concern at a large number of properties at the site, but this finding is judged to be especially uncertain due to lack of reliable information on the magnitude and frequency of pica soil ingestion and on the most appropriate acute oral RfD value.

Lead

Lead also occurs at elevated levels in soil at some residential properties. Elevations occur in all neighborhoods of the site, but levels tend to be higher on the western part of the site than the eastern part. Using EPA's IEUBK model to evaluate the risk to children, it is estimated that about 45% of residences have levels that exceed EPA's health-based goal (no more than a 5% chance that a child will have a blood lead value above 10 ug/dL). Of these, many (about 79%) have mean lead concentrations lower than 400 ppm (the USEPA default level of concern). This is mainly because the site-specific RBA for lead (84%) is higher than the default value (60%).

SECTION 1
INTRODUCTION

1.1 SITE DESCRIPTION

The Vasquez Boulevard and I-70 (VBI70) Superfund Site is an area of approximately four square miles located in the north-central section of Denver, Colorado. The site is composed of a number of neighborhoods that are largely residential, including Swansea, Elyria, Clayton, Cole, and portions of Globeville. Most residences at the site are single family dwellings, but there are also some multi-family homes and apartment buildings. The site also contains a number of schools, parks, and playgrounds, as well as a number of commercial and industrial properties. Figure 1-1 is a map which displays the site.

The site is largely flat in topography, sloping gently towards the Platte River, which flows in a northeasterly direction through the site. Other than the Platte River, there are no other major surface water bodies within the site.

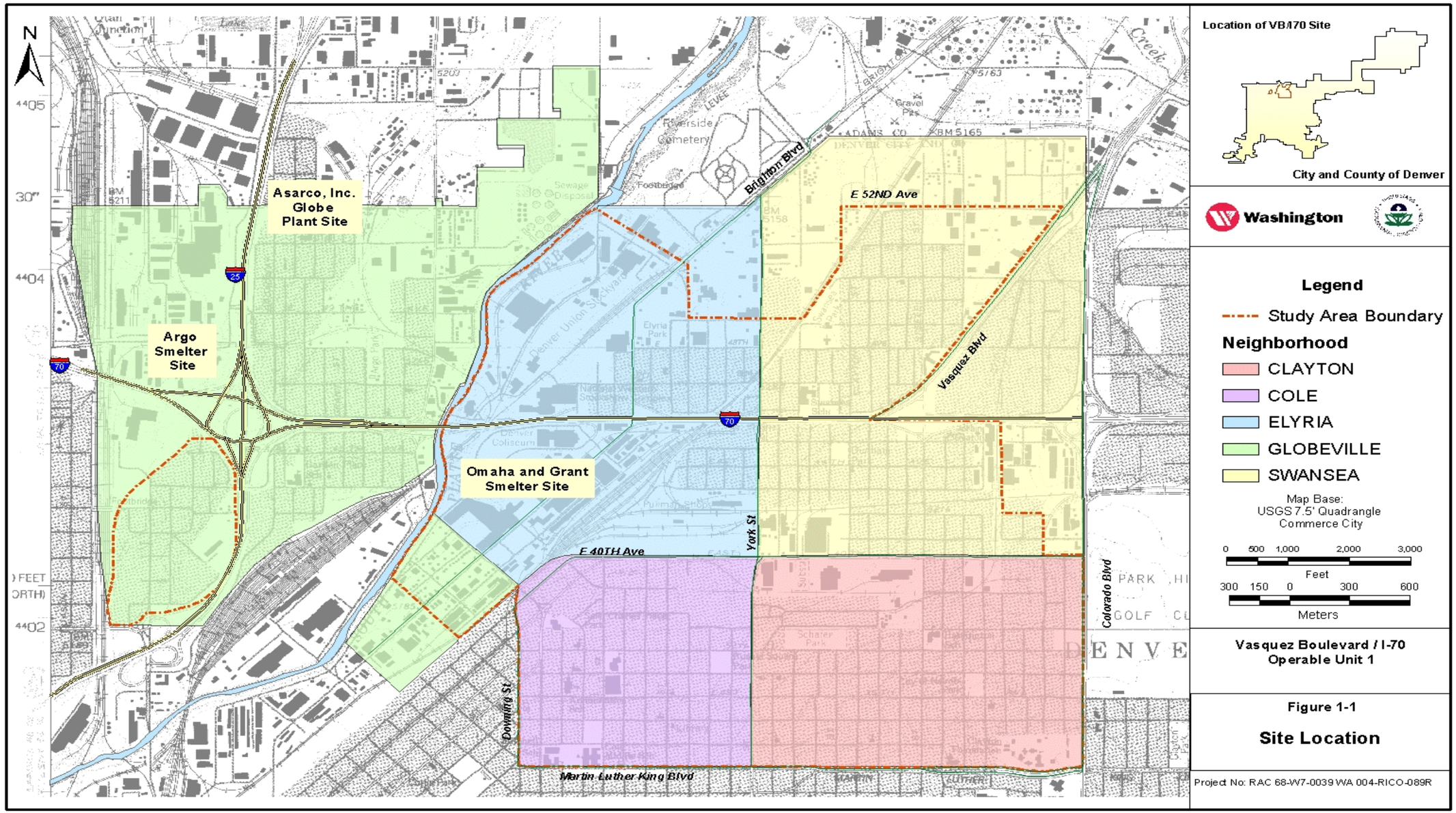
The climate of the site is generally typical of Colorado's semiarid eastern plains. Temperatures are moderate throughout the year, with monthly averages ranging from 30° F in January to 73° F in July. Annual rainfall measures 16 inches, 60% of which falls during the spring and summer. The rainiest month is May, with an average rainfall of 2.6 inches. Snowfall totals in the Denver Metro area average 60 inches, with March usually receiving the most snow (12.5 inches). The Rocky Mountain foothills, about 20 miles west of the site, help create a predominantly southern wind flow at the site, with an annual average velocity of about 8.5 mph. Peak winds can reach velocities of 30-50 mph, with the highest winds tending to be from the north-northwest (Colorado Climate Center 2000).

1.2 BASIS FOR POTENTIAL CONCERN

The site came to the attention of the U.S. Environmental Protection Agency (USEPA) because studies directed by the Colorado Department of Public Health and Environment (CDPHE) at a nearby site (Globe Smelter) indicated that elevated concentrations of arsenic and/or lead occurred in the soil of some residential properties in the Swansea/Elyria area. The source of these elevated levels is not known, but *a priori,* it was considered plausible that the contamination could be associated with releases either from the Globe facility and/or from one or both of two other smelters which previously existed in the area (the Argo Smelter and the Omaha and Grant Smelter). The locations of these three smelters in relation to the VBI70 site are also shown in Figure 1-1. Alternative potential sources include the historic application of arsenic- or lead-containing lawn care products, and/or (for lead) anthropogenic sources such as automobile exhaust, leaded paint, etc.

Based on the results of several rounds of soil sampling (see Section 2.0), USEPA concluded that the VBI70 site contained multiple residences where the concentration of arsenic and/or lead in

Figure 1-1 Site Map



yard soil could be above a level of potential human health concern. On this basis, USEPA proposed the VBI70 site for inclusion on the Superfund National Priorities List (NPL) in January, 1999, and the site was added to the NPL on July 22, 1999.

1.3 PURPOSE AND SCOPE OF THIS DOCUMENT

This document is a baseline human health risk assessment. The purpose of the assessment is to characterize the nature and magnitude of any risk to humans that may be attributable to contamination of site media, assuming that no steps are taken to remediate the environment or to reduce human contact with contaminated environmental media. More specifically, this assessment focuses on the direct and indirect risks to humans from contamination that is present in **soils** in current residential and commercial (non-smelter) areas of the site. This is referred to as the "Off Smelter Facility Operable Unit" (Operable Unit 1). The potential human health risks from exposure to other potentially contaminated environmental media (e.g., surface water, groundwater) and on-site soils (i.e., soils at former smelter areas) will be investigated and evaluated as separate Operable Units.

The results of this baseline risk assessment are intended to help inform risk managers and the public about the level of health risk which is attributable to contamination in site soils, to help determine the need for remedial action at the site, and to provide a basis for determining the levels of chemicals that can remain in site soils and still be adequately protective of public health (USEPA 1989).

The methods used to evaluate risks to humans and the environment employed in this assessment are consistent with current guidelines provided by the USEPA for use at Superfund sites (USEPA 1989, 1991a, 1991b, 1991c, 1992a, 1992b, 1993).

1.4 ORGANIZATION OF THIS DOCUMENT

In addition to this introduction, this report is organized into the following sections:

Section 2 This section provides a summary of the available data on the levels of chemical contaminants (metals) in site soils, and identifies which of these chemicals are of potential health concern to area residents or workers.

Section 3 This section discusses how residents and other people (workers, children at schools or playgrounds) may be exposed to site-related chemicals, now or in the future, and identifies exposure scenarios that are considered to be of potential concern.

Section 4 This section assesses the level of exposure and risk to humans from arsenic in site soils. This includes 1) a description of methods used to quantify exposure to arsenic, 2) data on the toxicity of arsenic to humans, 3) calculation of the level of noncancer and cancer risk that may occur as a result of exposure to arsenic in site

soils, and 4) a discussion of the uncertainties which limit confidence in the assessment.

Section 5 This section assesses the level of exposure and risk to area residents from lead in site soils. This includes 1) a description of the toxic effects of lead, 2) a summary of the method used by USEPA to evaluate risks from lead, 3) a summary of the estimated risks at this site attributable to lead in site soils, and 4) a discussion of the uncertainties which limit confidence in the assessment.

Section 6 This section provides full citations for USEPA guidance documents, site-specific studies, and scientific publications referenced in the risk assessment.

SECTION 2
SUMMARY OF SITE DATA AND
SELECTION OF CHEMICALS OF POTENTIAL CONCERN

Data on the level of arsenic, lead, and other metals which might have been released from area smelters into site soils have been collected in a phased series of investigations. A detailed summary and evaluation of these studies are presented in the Remedial Investigation/Feasibility Study (RI/FS) report for this site (USEPA 2001e). Each of these investigations is described briefly below, along with a summary of the key data collected during each phase.

2.1 PHASE I/PHASE II GRAB SAMPLE INVESTIGATION

Residential Soil Samples

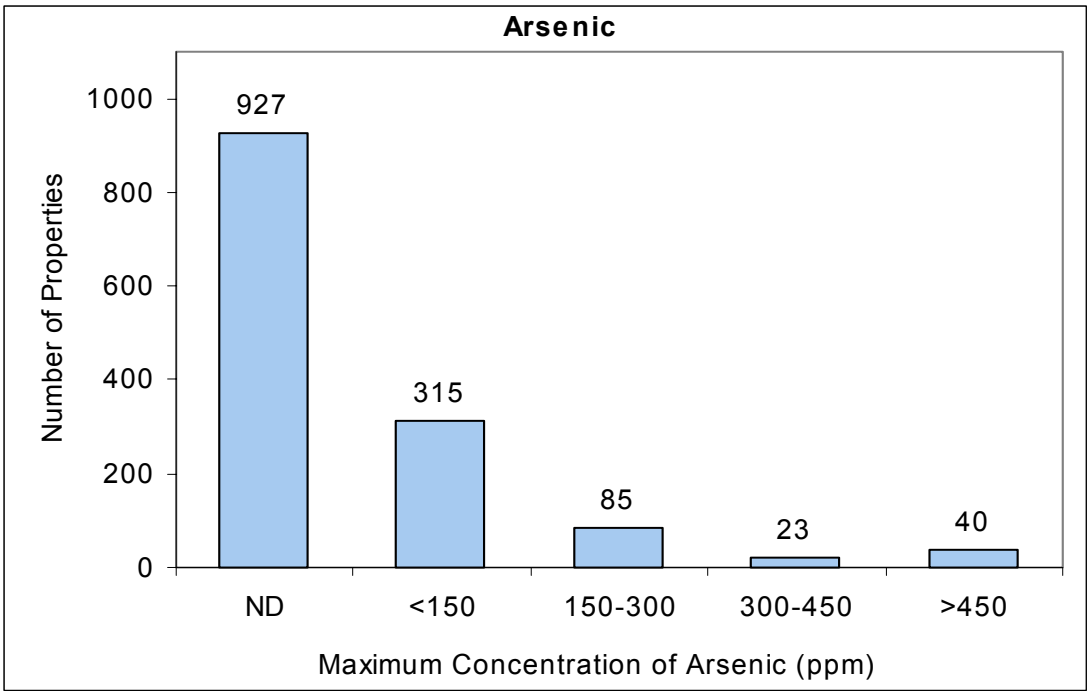
Once investigations at the nearby Globe site began to suggest that elevated levels of arsenic and/or lead might exist in soils at residential properties within the area of the VBI70 site, CDPHE requested assistance from USEPA Region VIII in characterizing the nature and extent of the contamination. In response, USEPA Region VIII undertook a study designed to identify properties that had levels of arsenic or lead that were sufficiently high that time-critical action (soil removal and replacement) might be warranted. The action levels selected for time-critical soil removal were 450 parts per million (ppm) for arsenic and 2,000 ppm for lead (USEPA 1998a).

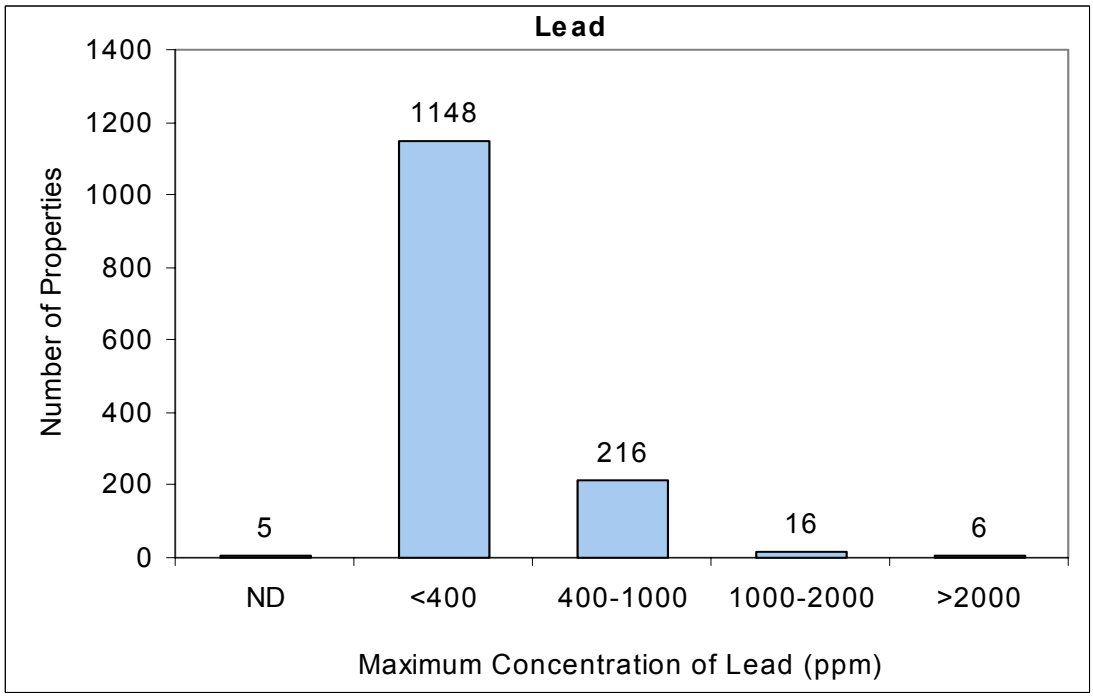
Details of the study are presented in UOS (1998a, 1998b). In brief, grab samples of surface soil and subsurface soil were collected from 1390 residential properties in the area of potential concern. Most of these samples were collected during the initial round of sampling (referred to as Phase I), with the remainder being obtained in a subsequent sampling effort (Phase II). In the majority of cases, two surface samples and one subsurface sample were collected per property, with additional surface samples at some locations (depending on the size of the property). All samples were analyzed for arsenic, lead, cadmium and zinc using X-ray fluorescence (XRF).

The results for arsenic in surface soil are summarized in Figure 2-1 (upper panel). As seen, a majority of properties sampled (927 out of 1390) had maximum arsenic values that were below the limit of detection (average detection limit = 51 ppm). However, arsenic was detected in one or more surface soil samples at a number of properties, with 40 of these properties having one or more samples above 450 ppm. Arsenic concentrations in subsurface samples were generally somewhat lower than the concentrations in surface soil, with an average ratio of subsurface to surface soil of about 0.8.

For lead (lower panel), most properties (1153 out of 1390) had maximum concentration values in surface soil that were below 400 ppm, but 238 properties had one or more values above 400

Figure 2-1 Phase I/Phase II Soil Grab Sample Data





Notes:
ND = Not Detected
Average detection limit for arsenic = 51 ppm (range = 44 to 800)
Average detection limit for lead = 32 ppm (range = 28 to 38)

ppm. Of these, 6 properties had one or more lead values above 2,000 ppm. Lead levels in subsurface soil tended to be lower than in surface soil, with an average ratio of subsurface to surface soil of about 0.7.

Any property with one or more arsenic values above 450 ppm and/or one or more lead values above 2,000 ppm were identified as candidates for soil removal, pending collection and analysis of composite soil samples to better characterize the true level of contamination (see below).

2.2 PHASE II CONFIRMATORY SAMPLING AND SOIL REMOVAL

In order to help confirm the identity of properties which warranted time-critical soil removal actions, USEPA collected two or more composite samples (each consisting of five sub-samples) of surface soil from residential properties where one or more grab samples were above the removal level for arsenic. This approach was employed because composites samples are judged to provide a more reliable and representative characterization of a yard than a single grab sample.

Based on the results of this composite sampling program, a total of 21 residences were identified where one or more composites confirmed that arsenic levels were above the action level. Of these, 18 underwent soil removal and replacement in the fall of 1998, while the owners of the other three properties refused permission for the removal. No properties were identified where lead levels in composite soil samples were high enough to warrant a time-critical soil removal action.

2.3 RISK-BASED SAMPLING PROGRAM

Following completion of the Phase I/Phase II sampling programs, USEPA undertook a number of additional studies in order to provide information that would help support long-term risk-based decision making at the site. One of these studies, referred to as the Risk-Based Sampling Program, collected more detailed data on metal contamination and exposure at the 18 properties that had been identified as requiring time-critical soil removal. Key elements of the program included: 1) detailed soil sampling to reveal the spatial pattern of contamination at some of the affected properties; 2) measurement of arsenic and lead levels in indoor dust, attic dust, and garden vegetables, as well as lead levels in paint and tap water; and 3) measurement of biomarkers of lead and/or arsenic exposure in residents at those locations. The details of the risk-based study design are presented in USEPA (1998b), and the results are detailed in USEPA (2001e). The main findings of this program are summarized below.

2.3.1 Spatial Patterns of Contamination

One of the striking findings that emerged from the Phase I/Phase II sampling programs was that properties that were affected by arsenic did not appear to occur in a clear spatial pattern. That is, the occurrence of high arsenic levels in soil did not appear to be associated with proximity to one or more of the smelters, and properties with elevated levels of arsenic often occurred immediately adjacent to one or more residences that were not apparently affected.

In order to obtain additional information on the spatial pattern of contamination both within and between yards, USEPA selected eight properties to undergo detailed soil sampling. Five of the yards were locations where Phase I/Phase II sampling indicated the arsenic concentrations were above the removal level, while three of the properties had arsenic concentrations below the removal level.

At each property, a high-density grid was established on 5-foot centers, and soil samples were collected wherever the grid node did not fall on a driveway, patio, etc. In addition, whenever access could be obtained, the sampling grid was extended 10-15 feet into adjacent properties in order to determine if there was a clear difference in contamination levels between adjacent properties. All samples were analyzed by XRF for arsenic, lead, cadmium, and zinc.

Diagrams which show the results for all four metals at all eight properties are presented in USEPA (2001e). Diagrams from this report that show the spatial patterns of arsenic and lead at two properties with high levels of arsenic contamination are shown in Figures 2-2 and 2-3. In both cases, arsenic levels vary from location to location, but are elevated across most of the yard. At property 1 (Figure 2-2), there is a fairly clear boundary between the property of concern and the adjacent properties. A similar pattern is observed at property 2 (Figure 2-3), although there are some locations where the contamination may extend somewhat into the adjacent property. The pattern of lead contaminations at these properties also showed a similar boundary effect. No clear boundary effect was observed for cadmium or zinc.

2.3.2 Contaminant Levels in Other Environmental Media

Samples of other environmental media were obtained at each removal property where access was granted. The results are summarized below.

Indoor Dust

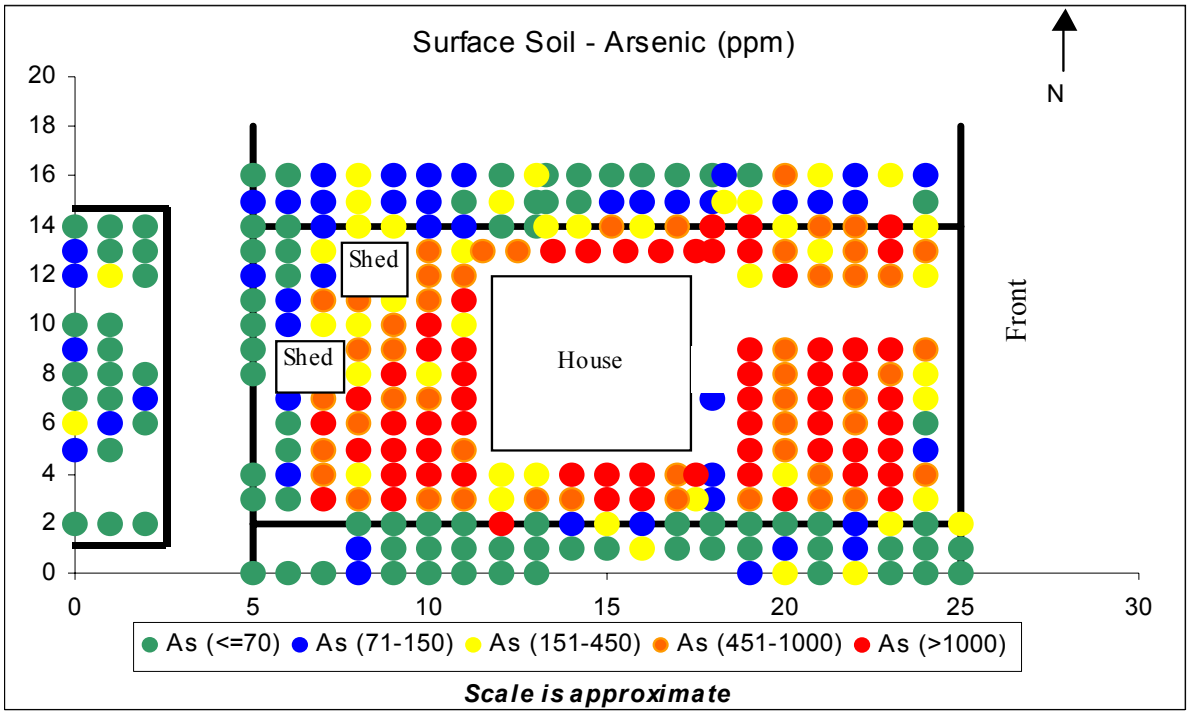
Dust from interior living spaces were collected at 15 properties, while attic dust was collected at 9 properties. Summary statistics are presented below.

Arsenic and Lead in Dust Samples from the Risk-Based Sampling Program

Medium	Arsenic			Lead		
	Detection Frequency	Mean (ppm)	Max (ppm)	Detection Frequency	Mean (ppm)	Max (ppm)
Interior dust (ppm)	14/15	107	172	15/15	243	1145
Attic dust (ppm)	7/9	230	499	9/9	1414	4106

Regression analysis between measured levels of arsenic and lead in indoor dust compared to the mean of the two five-point yard soil composites that were collected in Phase II revealed very little correlation between the concentration of either arsenic or lead in interior dust compared to that in outdoor soil, and the slopes of the best fit regression lines were not different from zero:

Figure 2-2 Spatial Distribution of Contaminants – Property 1



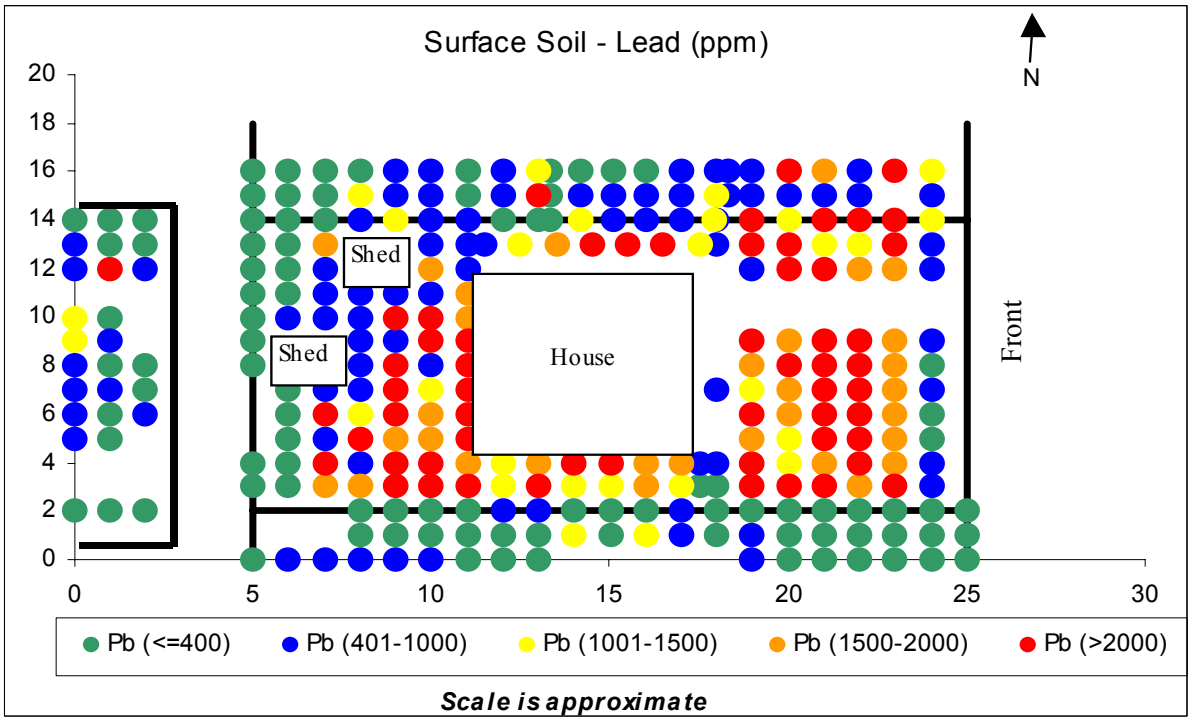
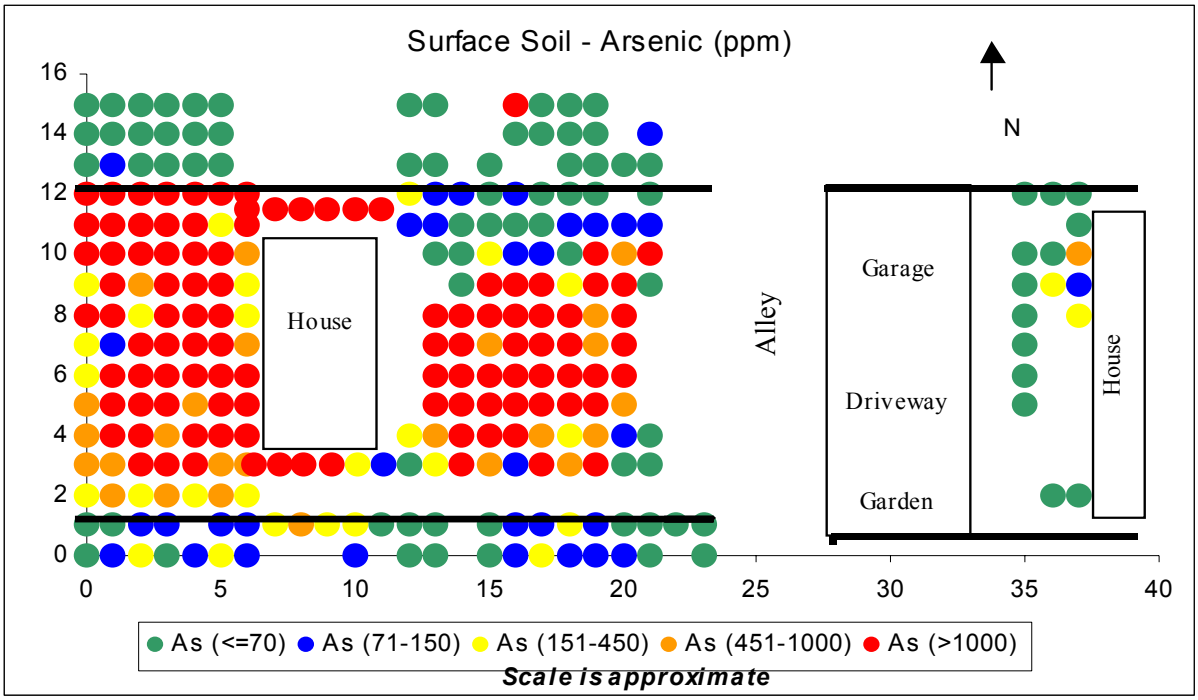
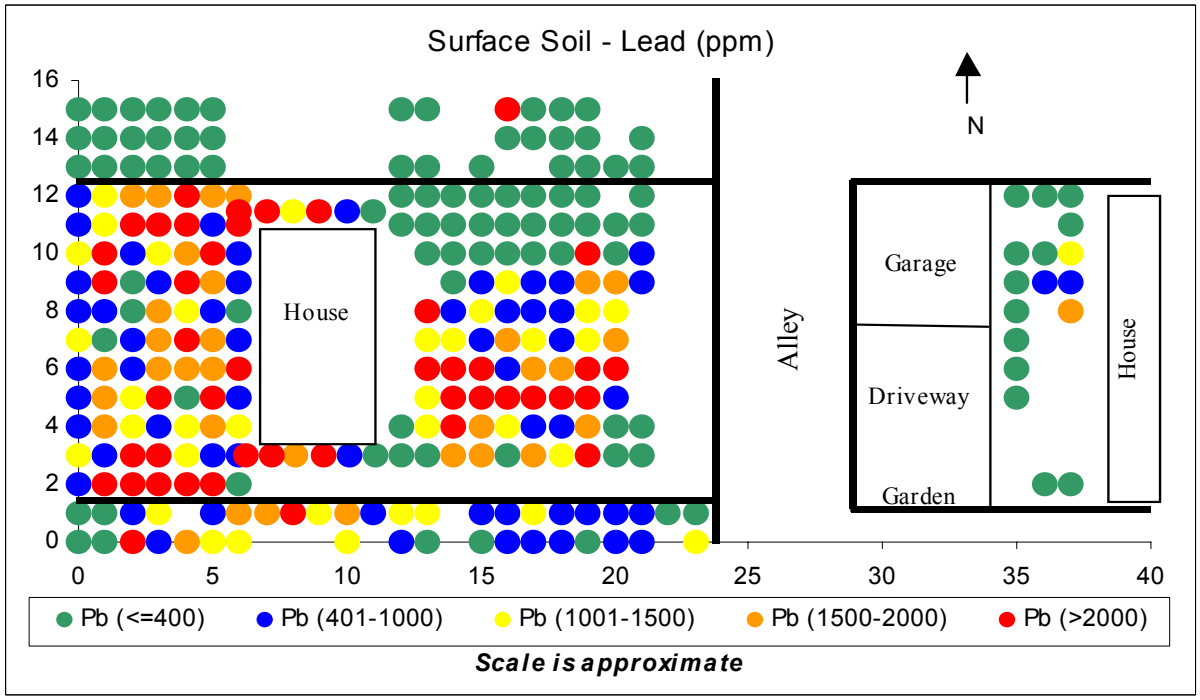


Figure 2-3 Spatial Distribution of Contaminants – Property 2





Correlation Between Yard Soil and Indoor Dust

Analyte	N	R^2	Slope	P Value
Arsenic	15	0.003	-0.004	>0.8
Lead	15	0.001	-0.0014	>0.9

Although this data set is too small to draw definite conclusions, the results suggest that outdoor soil is not a major determinant of arsenic or lead levels in indoor dust. There was also no significant correlation for arsenic or lead between the concentration in indoor dust and in attic dust. This suggests attic dust is not serving as an important source of indoor dust at this site.

Tap Water

Twelve properties allowed sampling and analysis of tap water for lead. Two types of water samples were collected: first flush and post-flush. Summary statistics are presented below.

Occurrence of Lead in Residential Water Samples

Medium	Detect. Freq.	Mean (ug/L)	Max (ug/L)
First-flush tap water	5/12	3.2	11.4
Post flush tap water	3/12	2.5	6.0

All of these values are below the current USEPA action level for lead in drinking water (15 ug/L), and are sufficiently low that tap water is not likely to be a significant source of lead exposure, at least in the 12 homes sampled.

Paint

Sixteen properties authorized analysis of lead levels in paint. Concentrations were measured by XRF at multiple locations on both interior and exterior surfaces. Summary statistics are presented below:

Occurrence of Lead in Residential Paint

Location	N	Mean (mg/cm2)	Range (mg/cm2)
Interior	89	4.2	0.3 - 19
Exterior	55	4.8	0.4 - 14

A total of 130 out of 144 samples had values above 1 mg/cm^2, the national default screening level for leaded paint (HUD 1995). These data suggest that interior and/or exterior leaded paint might be a source of lead exposure in area children, either directly (by paint chip ingestion), or indirectly (by ingestion of dust or soil containing paint-derived lead). In this regard, there is a weak but significant correlation between the concentration of lead in exterior leaded paint and the concentration of lead in yard soil ($R^2 = 0.283$, $p < 0.03$, $n = 16$), suggesting that some of the

lead in soil at the properties sampled may be attributable to exterior leaded paint. No significant correlation was detected between lead levels in interior paint and in indoor dust ($R^2 = 0.016$, $p > 0.5$, $n = 12$).

Garden Vegetables

Only one of the 18 properties scheduled for soil removal had a vegetable garden. At this location, one sample of potato and one sample of mint were collected. Concentrations of arsenic and lead were below the level of detection in both samples. Because so few samples were obtained, no conclusions can be drawn from this data set.

2.3.3 Phase II Biomonitoring

During Phase II, a total of 15 individuals residing at properties scheduled for soil removal (i.e., arsenic concentration above 450 ppm, or lead above 2,000 ppm) volunteered to have samples of hair, urine and/or blood analyzed for arsenic or lead The results are summarized in Table 2-1. For convenience, reference values indicating the typical and upper end of the normal range are also presented.

As seen, there were no cases where individuals living at the properties scheduled for soil removal had arsenic or lead levels that exceeded the "background" range typically seen in members of the general population, although one individual had a hair arsenic at the high end of the normal range. Although this data set is too small to draw firm conclusions, the results provide no indication that exposures at these locations were of immediate health concern.

2.4 PHYSICAL-CHEMICAL CHARACTERIZATION

In addition to the Risk-Based Sampling Program described above, USEPA also undertook two studies to characterize the physical and chemical attributes of the metal contamination in residential site soils, and to determine whether concentration estimates based on bulk (unsieved) soil samples were representative of concentrations in fine (sieved) samples. The design of these projects is presented in USEPA (1998c) and USEPA (1999e), and the results are detailed in USEPA (1998d) and USEPA (2001e). The main findings are summarized below.

2.4.1 Concentration in Sieved and Un-Sieved Soil Samples

As discussed in greater detail in Section 3, the main pathway by which humans are likely to be exposed to contaminants in soil is by incidental ingestion of soil particles adhering to the hand. Although data are limited, it is generally expected that small soil particles are more likely to adhere to the hands than coarse particles, and it is for this reason that USEPA Region VIII recommends that measurements of contaminant concentrations in soil generally be performed on samples that have been sieved to isolate the smaller particles (< 250 um). This sieved fraction is generally referred to as the “fine” fraction. Soil that has not been fine sieved but only coarse sieved (to remove particles larger than about 2 mm) is referred to as the “bulk” sample. Studies

Table 2-1 Biomonitoring Data for Residents at Phase II removal Properties

Demographic Data		Biomonitoring Data					
		Blood Lead		Hair Arsenic		Urinary Inorganic Arsenic	
Index Number	Age (years)	Value (ug/dL)	Qual.	Value (ug/g)	Qual.	Value (ug/L)	Qual.
1	3	2		0.43	U	20	U
2	7	2		1.32	U	20	U
3	9	2		0.39	U	20	U
4	13	2		0.39	U	10	U
5	16	1	U	0.3	U	20	U
6	17	2		0.45	U	10	U
7	22	1		0.41		10	U
8	43	3		0.28	U	20	U
9	43	2		0.29	U	10	U
10	47	2		1.16	U	20	U
11	51	3		0.41	U	20	U
12	56	4		0.26	U	20	U
13	58	3		0.32	U	20	U
14	65	2		0.91	U	20	U
15	70	2		0.38	U	20	U

Summary Statistics

Blood Lead

	Site Data (a)					Reference (b, c)	
Age (years)	N	Detect. Freq.	Geo. Mean (ug/dL)	Min (ug/dL)	Max (ug/dL)	Typical (ug/dL)	High End (ug/dL)
1-5	1	1/1	2.0	2.0	2.0	2.5 - 4.1	> 10
>=6	14	13/14	2.1	1.0	4.0	1.5 - 4.0	> 10
All	15	14/15	2.1	1.0	4.0	2.3 - 2.8	> 10

Hair Arsenic

	Site Data (a)					Reference (d)	
Age (years)	N	Detect. Freq.	Mean (ug/g)	Min (ug/g)	Max (ug/g)	Typical (ug/g)	High End (ug/g)
0-6	1	0/1	0.4	0.4	0.4		
>6	14	1/14	0.5	0.3	1.3	<0.2	1.0
All	15	1/15	0.5	0.3	1.3		

Urinary Inorganic Arsenic

	Site Data (a)					Reference (d)	
Age (years)	N	Detect. Freq.	Mean (ug/L)	Min (ug/L)	Max (ug/L)	Typical (ug/L)	High End (ug/L)
0-6	1	0/1	20	20	20		
>6	14	0/14	17	10	20	<10	20
All	15	0/15	17	10	20		

a Summary statistics calculated using unadjusted values for non-detects
b Brody et al 1994
c Pirkle et al 1998
d NRC 1999

ug = microgram
dL = deciliter (0.1 L)
g = gram
L = liter

at other sites have shown that concentrations of metals in the fine fraction can sometimes be somewhat higher (e.g, 10-30%) than in the bulk sample.

Because all of the samples collected during Phase I and Phase II were bulk samples, an investigation was performed to determine if the concentration values obtained for the bulk samples were likely to have values significantly different than if the samples had been sieved. During the Physical-Chemical Characterization study, a total of 120 samples were selected for a paired comparison of the concentration in bulk and fine samples, being sure to include samples with a wide range of arsenic and lead concentrations. All of these samples were analyzed for arsenic, lead, cadmium and zinc by XRF. For each analyte, only data pairs in which the analyte was above the detection limit in both the bulk and fine samples were used for correlation analysis.

The results are shown in Figure 2-4. As seen, the slope of the best fit regression line through the paired data set is close to 1.0 for lead and zinc, but is slightly higher for arsenic (slope = 1.21) and cadmium (slope = 1.13). This indicates that the concentration of at least some of the metals is about 10-20% higher in fines than in bulk samples of soil.

2.4.2 Speciation of Arsenic and Lead

Most metals, including arsenic and lead, can occur in a variety of different chemical and physical forms. These differences are of potential significance not only because they may help identify the source of contamination, but also because the toxicity of the metals may differ between different chemical forms. Therefore, USEPA undertook a study to obtain preliminary data on the chemical forms of arsenic and lead present in site soils. The details of the sample preparation and analysis methods are presented in USEPA (1998c). In brief, samples of site soil were chosen for analysis to span a range of arsenic and lead concentration values. Each sample was analyzed by electron microprobe analysis (EMPA), and the number and size of different chemical forms ("phases") of arsenic and lead-bearing particles were measured. From these data, the fraction of the total mass of arsenic and lead present in each phase was calculated. Samples evaluated in this way included a set of 22 residential soils evaluated under the Physical Chemical Characterization Study (USEPA 1998c), plus an additional 20 residential soils evaluated as part of the Soil Pilot-Scale Characterization Study (USEPA 1999e).

The results are shown in Figures 2-5 and 2-6. As seen, arsenic (Figure 2-5) occurs mainly as arsenic trioxide (As_2O_3), with a smaller but significant contribution from lead arsenic oxide (PbAsO) and a trace of arsenic antimony oxide (AsSbO). In most samples, the majority of all arsenic-bearing particles are 5-50 um in diameter. Lead (Figure 2-6) occurs in several phases, including lead arsenic oxide (PbAsO), lead phosphate (Pb Phosphate), and lead manganese oxide (PbMnO). The concentration of lead in lead arsenic oxide increases dramatically as total lead concentration increases, suggesting this is the predominant form accounting for elevated lead levels in yard soils. Levels of lead phosphate and lead manganese oxide also tend to increase as total lead concentrations increase, but these phases may be secondary weathering products derived from the lead arsenic oxide. In most samples, the majority of lead-bearing particles are 5-100 um in diameter.

Figure 2-4 Comparison of Concentration in Bulk and Fine Soil

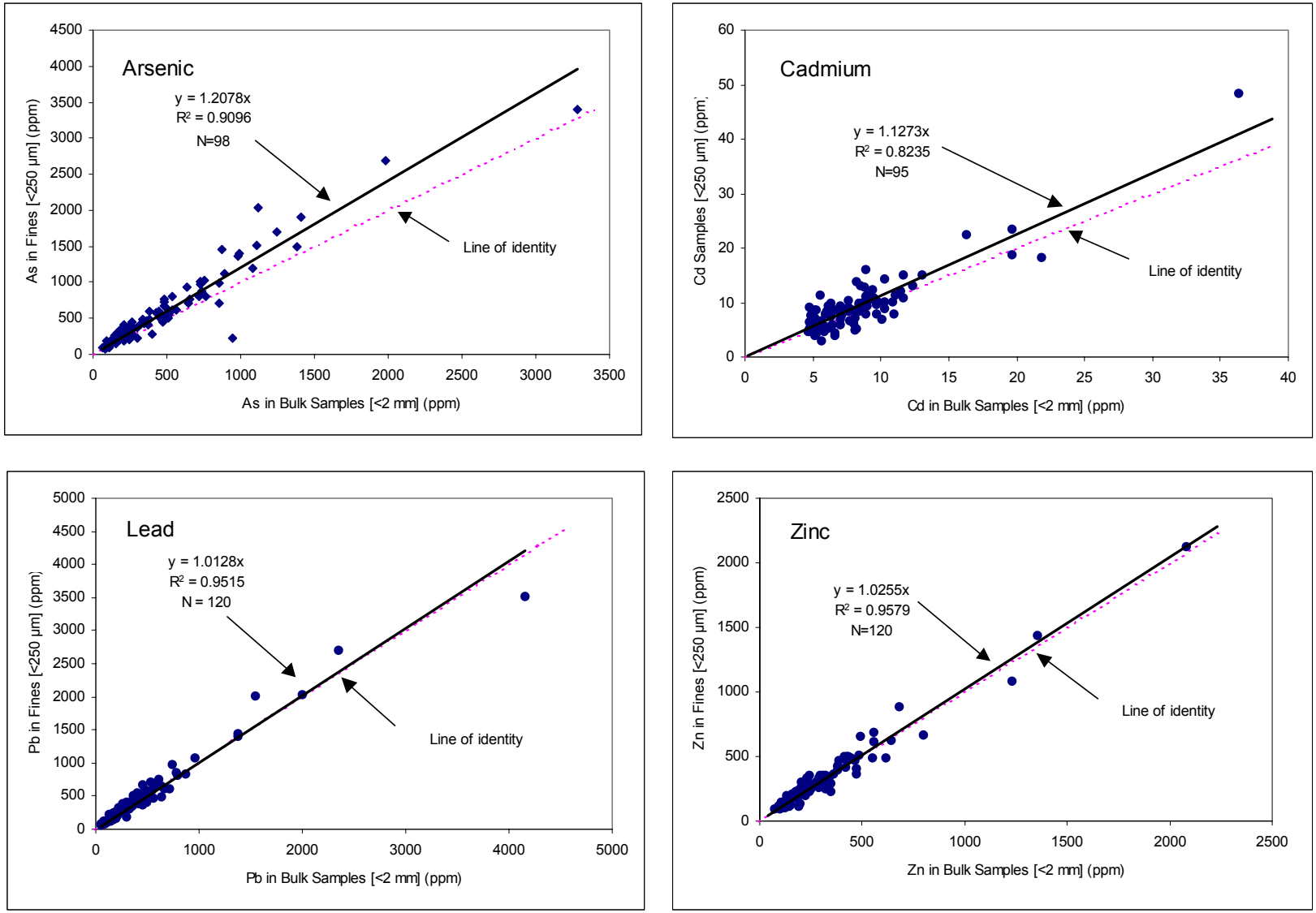


FIGURE 2-5 CHEMICAL FORMS OF ARSENIC IN SITE SOILS

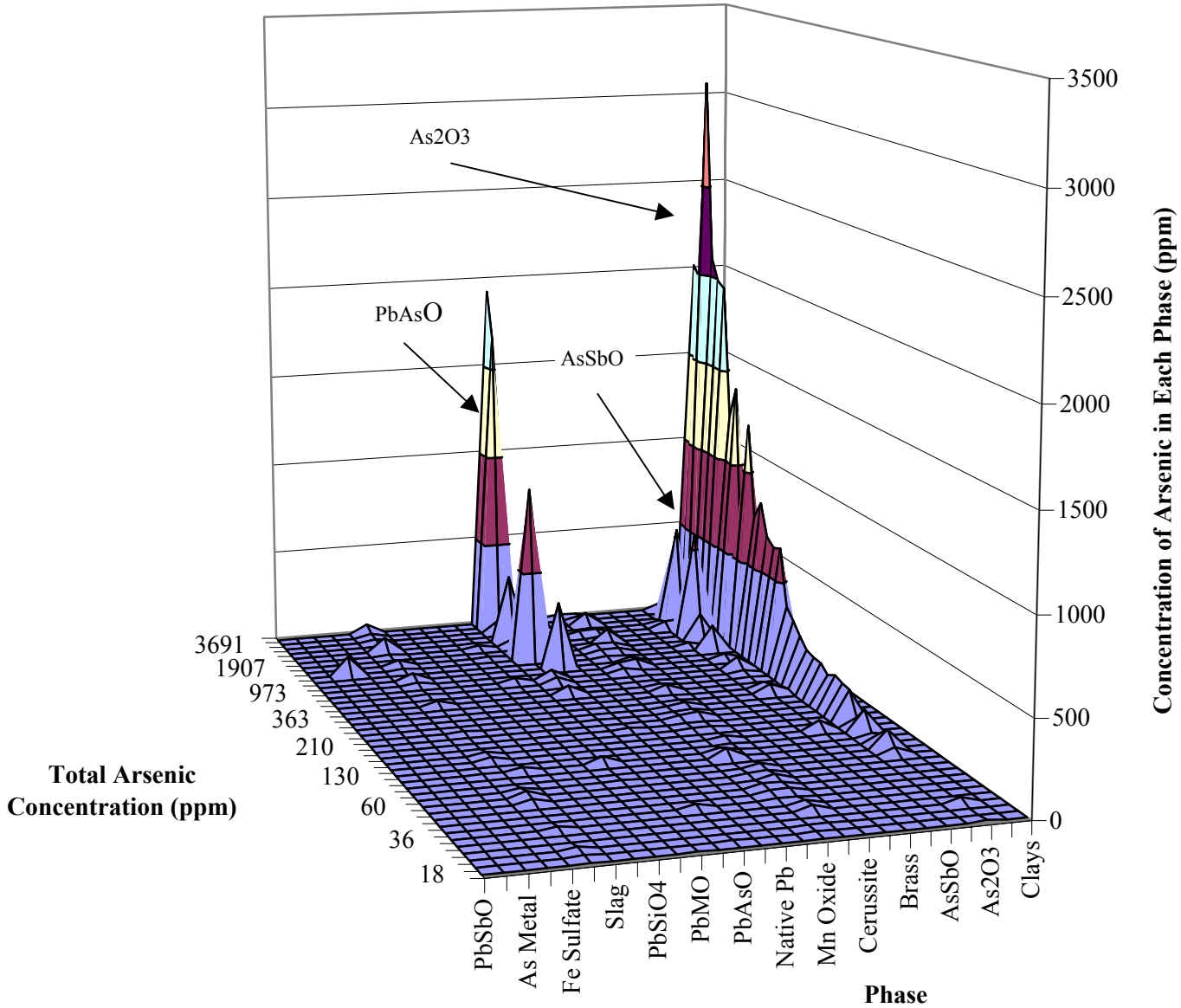
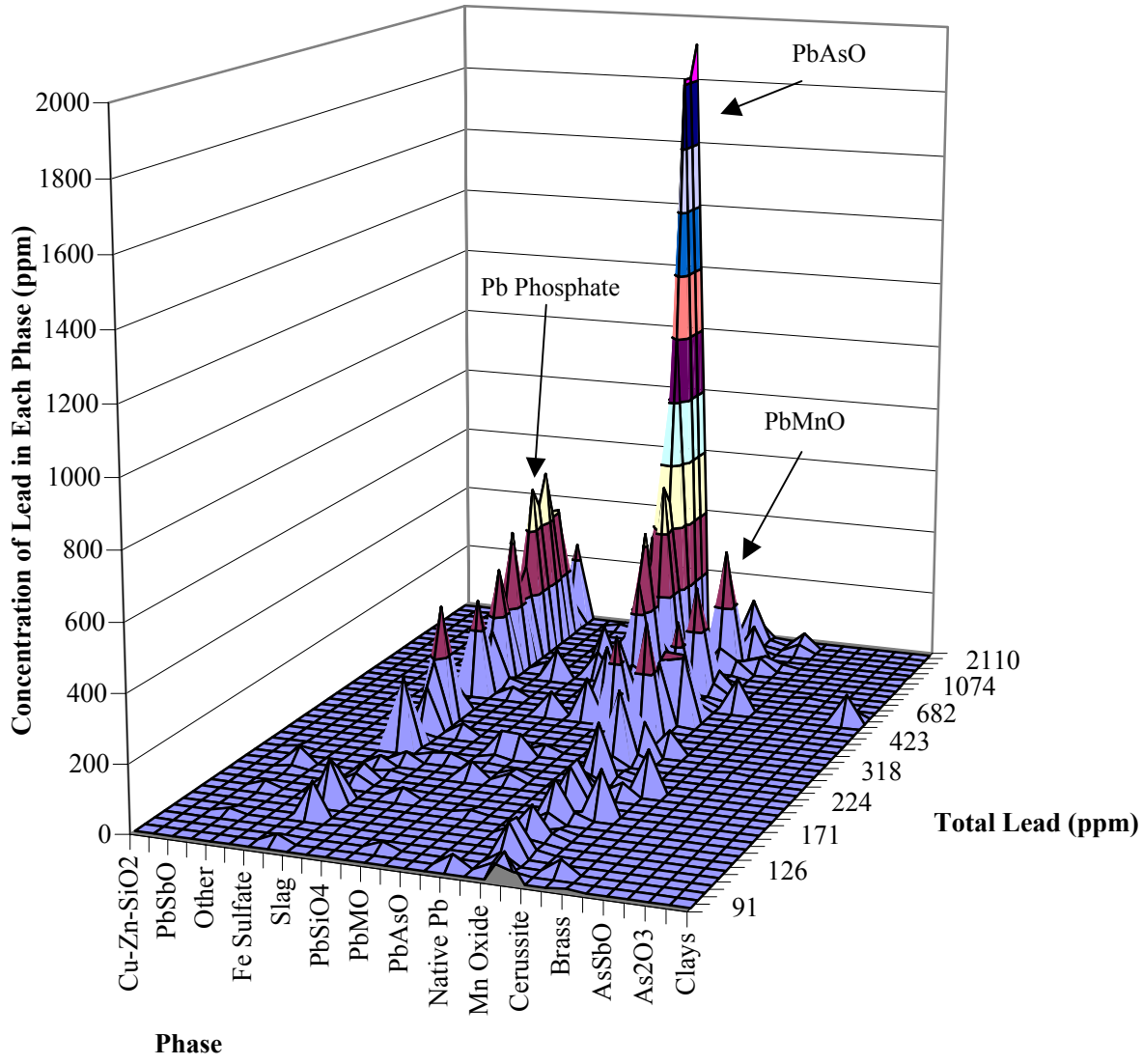


FIGURE 2-6 CHEMICAL FORMS OF LEAD IN SITE SOILS



2.5 SELECTION OF CHEMICALS OF POTENTIAL CONCERN

Chemicals of potential concern (COPCs) are chemicals which a) are present at a site, b) occur at concentrations which are or might be of health concern to exposed humans, and c) are or might be due to releases from a Superfund site. USEPA has derived a standard method for selecting COPCs at a site, as detailed in *Risk Assessment Guidance for Superfund: Human Health Evaluation Manual (Part A)* (USEPA 1989). In brief, USEPA assumes that any chemical detected at a site is a candidate for selection as a COPC, but identifies a number of methods that may be used for determining when a chemical is not of concern and may be eliminated from further consideration. Each risk assessment may choose to apply some or all of the methods identified by USEPA to select COPCs, as appropriate.

Data collected during Phase I and Phase II clearly indicated that arsenic and lead were both chemicals of potential concern at the VBI70 site. However, at that time no systematic evaluation had been performed to determine whether or not any other chemicals might also be of potential concern. For this reason, a careful review of the available data was undertaken to determine if other chemicals should be added to the list (USEPA 1999d). This review is summarized below.

2.5.1 Data Used to Select COPCs

As discussed above, most soil samples collected from the site were analyzed by XRF for only a few contaminants (mainly arsenic and lead). However, a sub-set of samples were analyzed by EPA Method 6010 (inductively coupled plasma atomic emission spectroscopy) (ICP) for the full suite of 23 metals included on USEPA's Target Analyte List (TAL), and these data are the basis of the COPC selection procedure. The data consist of two sub-sets:

- During Phase I, a total of 44 samples of soil were selected at random for ICP TAL analysis. The chief purpose of the analysis was to assess the accuracy of the XRF measurements for arsenic and lead. Because these samples were selected *a priori* and without regard to the level of contamination, there are only 9 of these samples that contain concentrations of arsenic above 100 ppm, with the maximum value being 1,200 ppm. Thus, these samples are helpful in the COPC selection procedure, but may not necessarily represent the chemicals of concern at the most contaminated properties.

- During the Risk-Based Sampling Program, USEPA performed an intensive study of arsenic and lead levels at 8 residential properties in the study area, including 5 properties with clearly elevated arsenic levels. Two samples from each of these five properties were selected for ICP TAL analysis, since these samples all contain high levels of arsenic (6,000 to 12,000 ppm) and are likely to reflect the contaminants most likely to be of concern.

These data are summarized in Table 2-2. In the case of copper, there is one sample whose analytical value (14,000 ppm) appears to be clearly inconsistent with all of the other 53 values (average = 37 ppm, max = 71 ppm). On this basis, the one extreme value for copper was

Table 2-2 Data Used to Select Chemicals of Potential Concern

Analyte	N	Detection Frequency	Summary Statistics		
			Min (ppm)	Max (ppm)	Mean (ppm)
ALUMINUM	54	100%	4900	15000	8761
ANTIMONY	54	22%	2.2	54	6.8
ARSENIC	54	93%	5	9940	543
BARIUM	54	100%	91	1000	251
BERYLLIUM	54	98%	0.3	1.1	0.7
CADMIUM	54	100%	0.9	19	5.9
CHROMIUM	54	100%	7.2	99	22
COBALT	54	98%	1.0	7.0	4.6
COPPER (a)	53	100%	12	71	37
LEAD	54	100%	36	3550	712
MANGANESE	54	100%	160	560	323
MERCURY	54	93%	0.1	11	1.0
NICKEL	54	100%	5.9	96	11
SELENIUM	54	19%	0.3	10	9
SILVER	54	69%	0.3	3	0.7
THALLIUM	54	89%	0.2	19	11
VANADIUM	54	100%	13	42	21
ZINC	54	100%	84	3680	499
CALCIUM	54	100%	1900	41000	6757
IRON	54	100%	7900	26000	13405
MAGNESIUM	54	100%	1400	4100	2400
POTASSIUM	54	100%	1400	4100	2350
SODIUM	54	5%	300	440	304

(a) Excludes one value (14,000 ppm) that is considered anomalous

excluded as an outlier, and screening was based on the remaining samples. All other data values were used. Non-detects were evaluated using the reported detection limit.

2.5.2 COPC Selection Process

Step 1: Eliminate Chemicals Whose Maximum Value Is Below a Level of Concern

This step involves comparing the maximum detected value in a medium to an appropriate Risk-Based Concentration (RBC). If the maximum value is less than the RBC, the chemical does not pose an unacceptable risk and can be eliminated.

The RBCs used in this evaluation were taken from USEPA's Region III Risk-Based Concentration (RBC) table for residential soil (USEPA 1999c). The value of each RBC depends on the specified Target Risk level. The Target Risk levels used in this evaluation are 1E-06 for carcinogenic chemicals and a hazard quotient (HQ) of 1.0 for noncarcinogenic chemicals.

Table 2-3 lists the Region III RBCs for each chemical and identifies those which can and cannot be eliminated at this step. Based on this screening step, the following chemicals were eliminated:

- Aluminum
- Barium
- Beryllium
- Cadmium
- Chromium
- Cobalt
- Copper
- Manganese
- Mercury
- Nickel
- Selenium
- Silver
- Vanadium
- Zinc

Step 2. Eliminate Beneficial Minerals

In accord with USEPA (1989), chemicals that are normal constituents of the body and the diet and are required for good health may be eliminated unless there is evidence that site-specific releases have elevated concentrations into a range where intakes would be potentially toxic. At this site, there is no reason to suspect this is the case, so the following chemicals were eliminated on this basis:

- Calcium
- Magnesium
- Potassium
- Sodium

Iron was also eliminated on this basis, since the average concentration of iron (13,400 ppm) is well below the screening level of 23,000 ppm. Additionally, only 1 of 54 samples exceeds the RBC for iron, and this only by a small amount (26,000 vs. 23,000 ppm).

Table 2-3 Comparison of Maximum Values in Soil to Soil Screening Levels

Analyte	Maximum Concentration (ppm)	Region III Soil Screening Level	Potential COCP
ALUMINUM	15000	78400	no
ANTIMONY	54	31	yes
ARSENIC	9940	0.43	yes
BARIUM	1000	5500	no
BERYLLIUM	1.1	160	no
CADMIUM	19	78	no
CALCIUM	41000	--	no
CHROMIUM	99	230	no
COBALT	7.0	4700	no
COPPER (b)	71	3100	no
IRON	26000	23000	yes
LEAD	3550	400	yes
MAGNESIUM	4100	--	no
MANGANESE	560	1600	no
MERCURY	11	23	no
NICKEL	96	1600	no
POTASSIUM	4100	--	no
SELENIUM	10	390	no
SILVER	3	390	no
SODIUM	440	--	no
THALLIUM	19	5.5	yes
VANADIUM	42	550	no
ZINC	3680	23000	no

COPC=Chemical of Potential Concern

(a) USEPA (1999c)

(b) Excludes one value (14,000 ppm) considered to be anomolous

Step 3. Eliminate Chemicals Whose Contribution is Minor Compared to Others

Following Steps 1 and 2, the list of chemicals remaining as potential COPCs was:

- Arsenic
- Antimony
- Lead
- Thallium

Antimony (a non-carcinogenic chemical) was eliminated because the magnitude of the non-cancer risk which it poses is very small compared to that posed by arsenic. For example, in the 10 samples most contaminated with arsenic, the average non-cancer risk contributed by antimony is less than 1% of that contributed by arsenic. That is, if antimony were retained and the non-cancer risk were quantified, the risk would be less than 1% larger than if antimony were not included. Because an increment of 1% is well within the uncertainty range of the risk assessment procedure, inclusion of antimony would not change any risk interpretations and therefore is judged to be unnecessary.

Step 4. Special Investigation for Thallium

Data on thallium available from the existing TAL analyses are internally inconsistent, as shown below:

Thallium Data from TAL Analyses

Parameter	Data Set 1	Data Set 2
Method	ICP-Trace	ICP-MS
Mean (ppm)	13.5	0.45
Max (ppm)	19	0.68
Detection Limit (ppm)	10	0.1

The basis for this internal inconsistency is not clear. One possibility is that differences in analytical methods are responsible. Data in Set 1 (collected during Phase I) utilized an analytical method (ICP-Trace, USEPA Method 6010) that had a relatively high detection limit, and most of the reported values were near that detection limit. In the second data set (collected during the risk-based sampling), thallium was analyzed by USEPA Method 6020 (ICP-MS), which has a much lower detection limit for thallium. In general, the results of the second analysis are thought to be more reliable, and are in accord with expected thallium levels in background soils (0.3-0.7 ppm) (ATSDR 1992). However, because it is not certain that the results from the second analysis are actually more reliable than from the first, a special study was performed in which thallium levels were measured in 10 site soils, including 6 samples from Set 1 (previously analyzed by ICP-Trace) and 4 samples from Set 2 (previously analyzed by ICP-MS). Each of the samples were analyzed for thallium by three analytical methods:

- Inductively Coupled Plasma Atomic Emission Spectroscopy [ICP-trace] (EPA SW-846 Method 6010B)
- Inductively Coupled Plasma-Mass Spectrometry [ICP-MS] (EPA SW-846 Method 6020)
- Graphite Furnace Atomic Absorption Spectroscopy [GFAA] (EPA SW-846 Method 7841)

The results of this analysis are provided in Table 2-4. A comparison of thallium levels in site soils as reported in past and present studies clearly indicate that results contained in the Phase I Investigation report (UOS 1998a) are biased high and are not reliable, with all of the 10 present site soil measurements having thallium values lower than 1 ppm. Based on the Region III (EPA 1999c) risk-based concentration for thallium in soil (5.5 ppm), it is concluded that thallium is not in a range of potential concern, and therefore it was eliminated as a COPC.

2.5.3 Summary: Chemicals Selected as COPCs at VBI70

Based on the methods and data detailed above, the COPCs selected for quantitative evaluation at the VBI70 site are arsenic and lead. All other chemicals are either not of concern or are present at levels which contribute minimal risk compared to arsenic.

2.6 PHASE III INVESTIGATION

Results from the Phase I/Phase II sampling programs, supplemented with the data and findings from the Risk-Based Sampling Program and the Physical Chemical Characterization Program, indicated that there are properties present in the VBI70 site where arsenic and/or lead could be in a range of health concern to exposed humans. However, because of the absence of any clear spatial pattern of soil contamination, the identity and location of such properties could not be reliably predicted using traditional approaches. For this reason, USEPA undertook a large-scale sampling program designed to obtain data that would help evaluate health risks to residents in the area. This program is referred to as the Phase III investigation. The investigation consisted of four main parts:

- Sampling of residential yard soils
- Sampling of indoor dust at residences
- Sampling of residential vegetable gardens (vegetables and soil)
- Supplemental sampling of soil at local schools and parks

The details of the Phase III sampling program are presented in USEPA (1999d).

Phase III was implemented in two parts. The first part, referred to as Phase IIIa, focused mainly on properties (including residences, schools, and parks) which had not been investigated in Phases I or II. The second part, referred to as Phase IIIb, consisted mainly of re-sampling at properties that had previously been sampled in Phase I or II, but for which the data were judged to be too limited to support clear risk-management decision making. The results of both IIIa and IIIb are summarized bellow.

Table 2-4 Comparison of Past and Present Data for Thallium in Soil

	Thallium Concentration (ppm)				
	Past Results		Present Study		
Sample ID	ICP-MS	ICP-Trace	ICP-Trace	ICP-MS	GFAA
C4690CYB-064	0.63		10 U	0.70	0.50 U
C4690CYB-046E	0.20		10 U	0.10	0.50 U
C4711THF-001	0.33		10 U	0.30	0.50 U
C4771VIN-001	0.33		10 U	0.30	0.50 U
D4145FIB10		12	10 U	0.20 U	0.50 U
D4715GYF10		17	10 U	0.30	0.50 U
D4050FIB10		11	10 U	0.20	0.50 U
D4701JOS10		10 U	10 U	0.10 U	0.50 U
D4780CBB10		16	10 U	0.50	0.80
D4785CLF10		15	10 U	0.20	0.50 U

U = not detected

2.6.1 Residential Soil Sampling

A total of 2,986 residential properties granted EPA access to collect soil samples during the Phase III program. At each of these properties, 30 surface soil (0-2 inch) grab samples were collected and combined into three composites samples, each containing 10 grab samples. The composites were prepared by combining every third grab sample, such that each composite represents an independent estimate of the yard-wide mean concentration. All composite samples were dried and thoroughly mixed, and then sieved through a coarse sieve (2 mm) to isolate the "bulk" fraction. A subset of samples were also sieved through a 250 um screen to isolate the "fine" fraction (see Section 2.4.1 above). All samples were analyzed for arsenic and lead by XRF.

Summary statistics for bulk soil samples, based on average values at each property and stratified by neighborhood, are summarized in Table 2-5. The distributions of arsenic and lead concentrations across the entire site are shown graphically in Figure 2-7. For arsenic, most properties (2,471 out of 2,986 = 83%) have average bulk soil concentrations of 50 ppm or less, with 258 properties (9%) between 50-100 ppm, 183 (6%) between 100-200 ppm, and 74 (2%) above 200 ppm. For lead, 2,712 (91%) properties have mean lead concentrations lower than 400 ppm, with 266 (9%) between 400-800 ppm and 8 (0.3%) higher than 800 ppm.

The relationship between the concentration of lead and arsenic in residential yard bulk soil samples is shown in Figure 2-8. As seen, there is a weak correlation between the concentration of lead and arsenic in soil, with a slope of about 0.6 ppm of lead per ppm of arsenic. However, this correlation accounts for only a small fraction of the variability in the lead concentration ($R^2 = 0.089$), and inspection of the figure indicates that samples with lead values above 400-600 ppm occur over a wide range of arsenic values, and are not associated predominantly with those where arsenic is above 100-200 ppm. This indicates that the main source of lead and the main source of arsenic in yard soil are not likely to be the same at most yards.

As noted earlier, data collected during one of the physical-chemical characterization studies (USEPA 1998d) indicated that both arsenic and lead might be slightly enriched in the fine fraction compared to bulk soil samples. In order to investigate this further, an additional set of 68 residential soil samples collected during the Phase III study were analyzed for lead and arsenic in both the bulk and fine soil fractions. The results of these 68 samples were combined with the results from the previous study (see Figure 2-4). The slope of the best fit linear regression line through the combined data set was 1.21 for arsenic, and 1.09 for lead. In both cases, these slopes were statistically different from 1.0 ($p < 0.001$). This confirms the earlier indication that at this site the concentration of metals are about 10-20% higher in fines than in bulk samples of soil.

Table 2-5 Property Mean Summary Statistics for Phase III Soil Samples
Residential Garden Sampling

ARSENIC

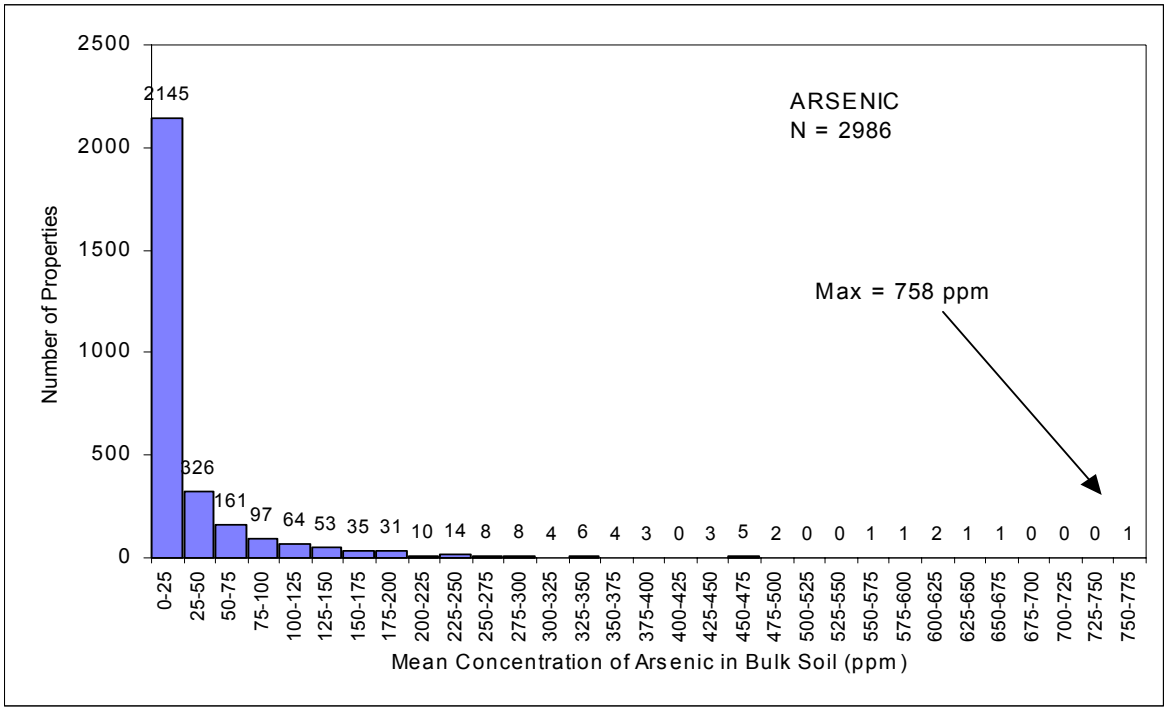
Neighborhood	Total Properties	Distribution of Yard Average Concentration Values for Arsenic (ppm) (a)					
		5th	25th	50th	75th	95th	Maximum
Clayton	902	5.5	5.5	8.7	38.3	168.0	758
Cole	796	5.5	7.7	11.8	24.8	142.1	660
Elyria	59	5.5	8.5	12.3	22.3	97.2	431
Globeville	63	5.5	8.5	13.8	22.3	123.3	297
Swansea	1166	5.5	5.5	9.7	30.6	128.3	604
ALL	2986	5.5	5.5	10.5	30.3	144.9	758

LEAD

Neighborhood	Total Properties	Distribution of Yard Average Concentration Values for Lead (ppm) (a)					
		5th	25th	50th	75th	95th	Maximum
Clayton	902	76	106	140	193	337	1131
Cole	796	135	221	288	371	538	1130
Elyria	59	181	299	372	438	601	922
Globeville	63	171	257	332	482	633	835
Swansea	1166	76	119	164	250	410	776
ALL	2986	81	127	188	292	465	1131

(a) Yard average is the mean of composites collected from the yard

Figure 2-7 Distribution of Property Mean Concentrations in Bulk Soils



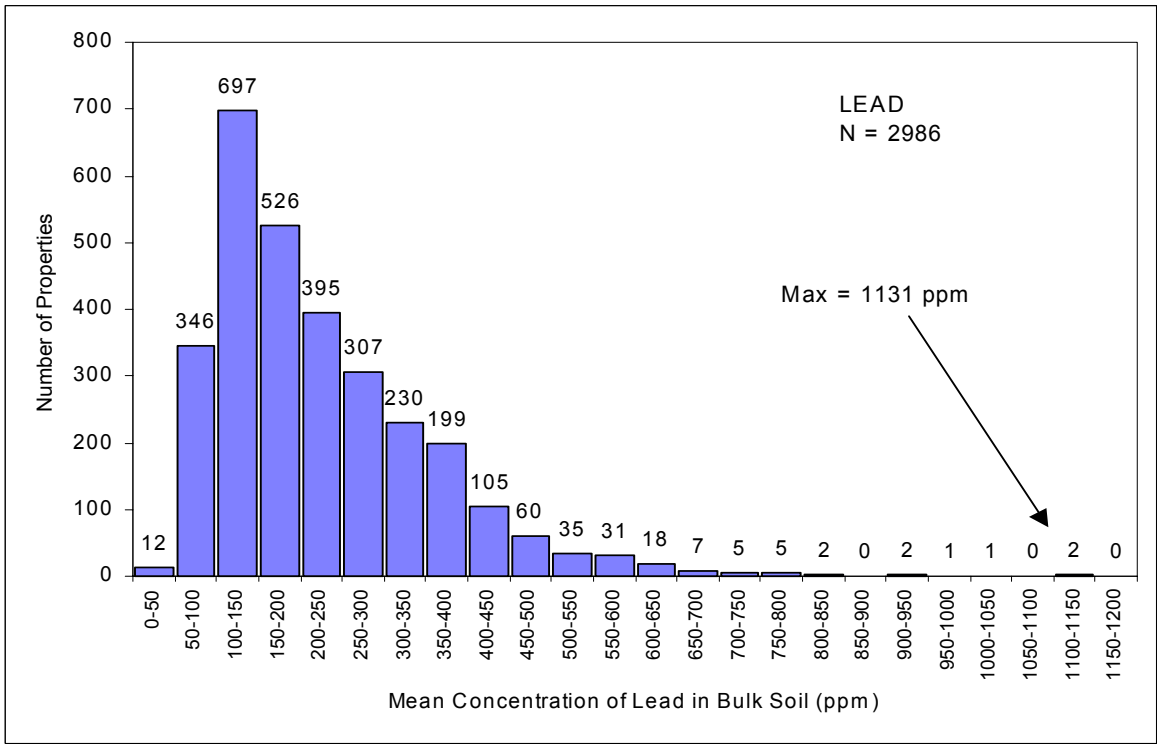
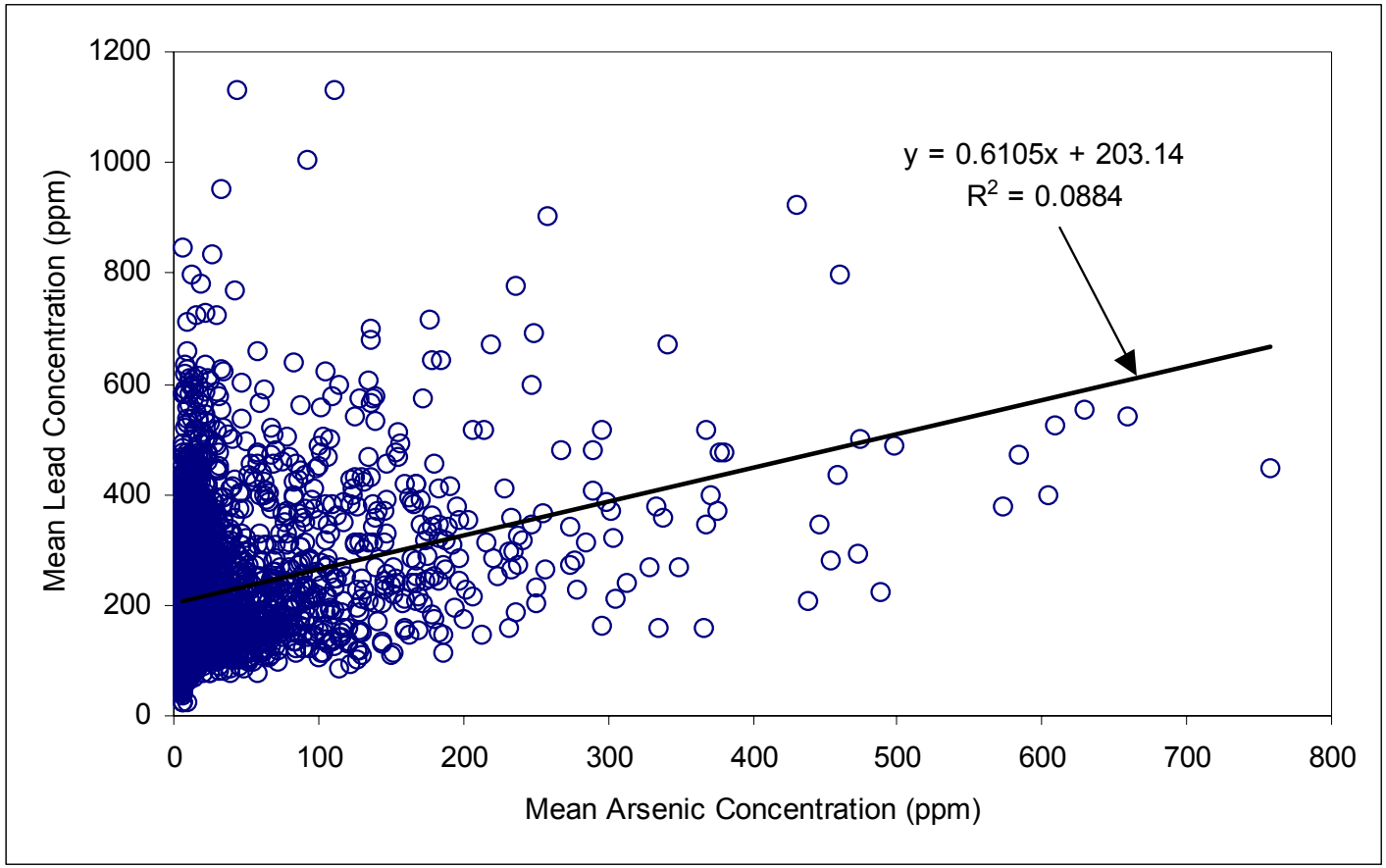


Figure 2-8 Correlation between Lead and Arsenic



2.6.2 Residential Dust Sampling

As discussed in greater detail in Section 3, one pathway by which residents may be exposed to contaminants in soil is by transport of outdoor soil into the house where it combines with other sources to form house dust. When data are absent, USEPA often assumes that the concentration of contaminants in house dust is the same as in yard soil. However, studies at other sites have shown that dust levels of metals are often lower in indoor dust than in outdoor soil. Therefore, USEPA Region VIII undertook a study to define the relationship between arsenic and lead levels in soil and dust at this site. The details of the sampling and analysis plan are presented in the Phase III Project Plan (USEPA 1999d). In brief, dust samples were collected from 74 properties. The locations of these properties were selected to span a range of arsenic and lead levels in soil, and to provide for spatial representativeness across the site. One composite sample was collected from each residence by vacuuming dust from 8-14 different living areas within the house, focusing on those areas judged to be most likely to be a source of dust exposure (e.g., bedroom, family room, kitchen, etc.). Samples were collected in October and November, 1999[1]. The results are shown in Figure 2-9. In the case of lead, two dust samples were excluded as outliers because they contained lead at concentration values (2,000 ppm and 9,900 ppm) that were much higher than that observed in yard soil (268 ppm and 320 ppm, respectively). The source of the high dust lead at these two locations is not known, but could be associated with releases from indoor leaded paint. Individuals living in these two homes were referred to the City and County of Denver's Department of Environmental Health to discuss the possible source of lead in the dust in their home, and the USEPA offered free blood lead testing to all family members.

As seen, there is only a weak correlation between the level of either arsenic or lead in paired soil and dust samples ($R^2 = 0.14$ to 0.18, respectively). Nevertheless, the slopes of both regression lines are statistically different from zero ($p < 0.01$), with best estimate parameter values as follows:

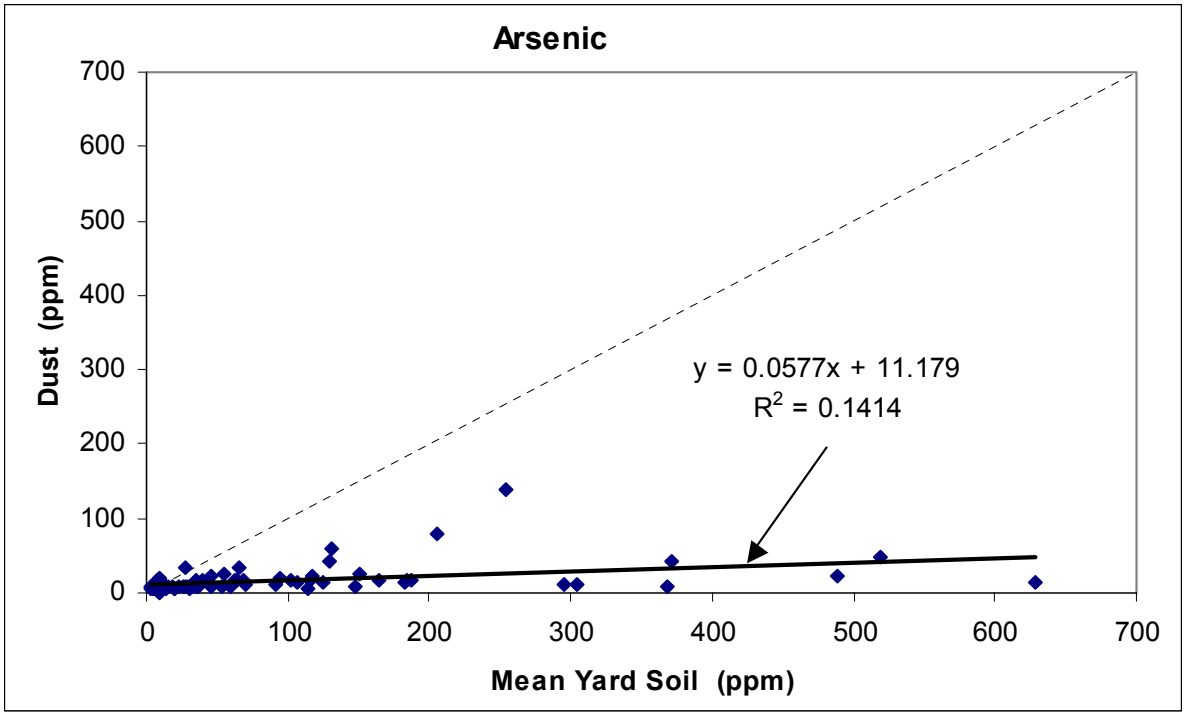
Arsenic: $C_{dust} = 0.06 \cdot C_{soil} + 11$

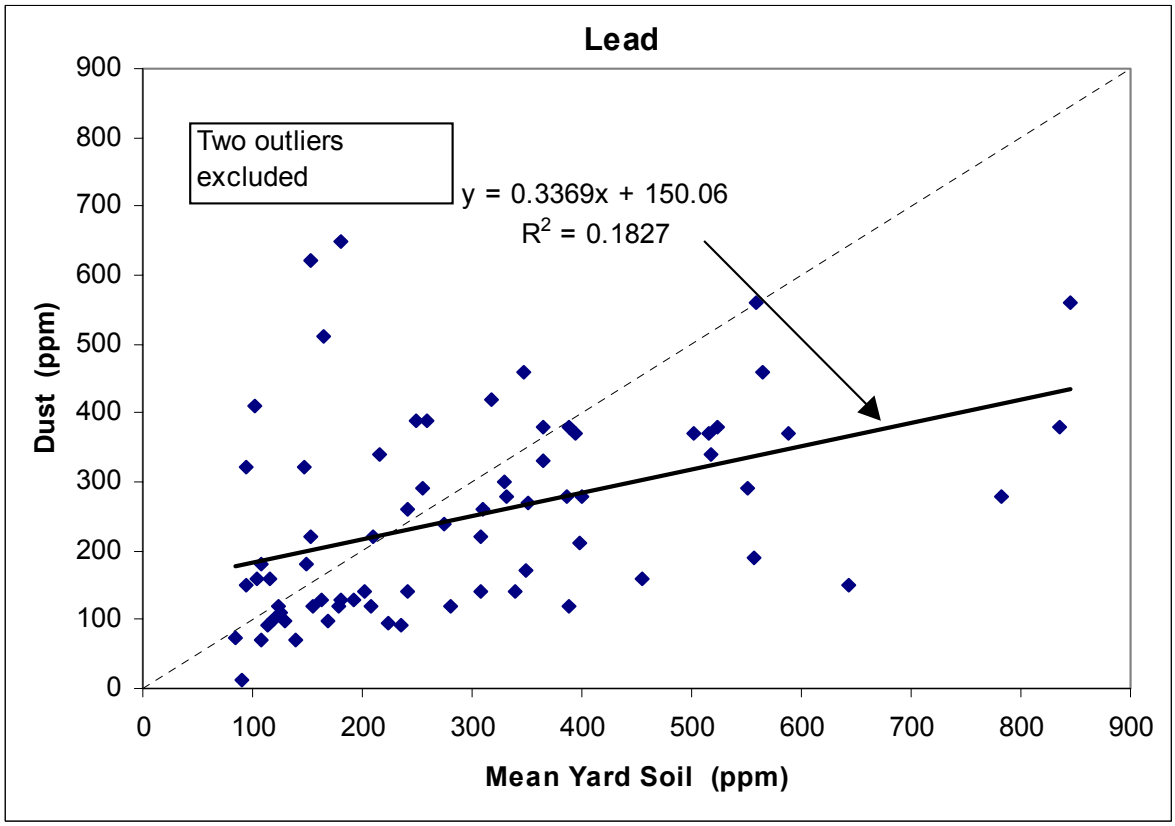
Lead: $C_{dust} = 0.34 \cdot C_{soil} + 150$

These slope values are somewhat higher than were observed in the Risk-Based sampling data (arsenic = -0.004 ppm per ppm, lead = -0.014 ppm per ppm) (see Section 2.3.2), perhaps because of the larger number of samples or perhaps because of differences in sampling and analysis methods for soil and dust. These slope values are within the range of values that have been observed at other sites investigated in Region VIII, as shown below:

[1] It is not known if dust concentrations at the site vary seasonally, but maximum impact from yard soil is suspected to occur in the late summer.

Figure 2-9 Relation between Concentration in Indoor Dust and Bulk Yard Soil





Soil-Dust Relationships at Other USEPA Region VIII Sites

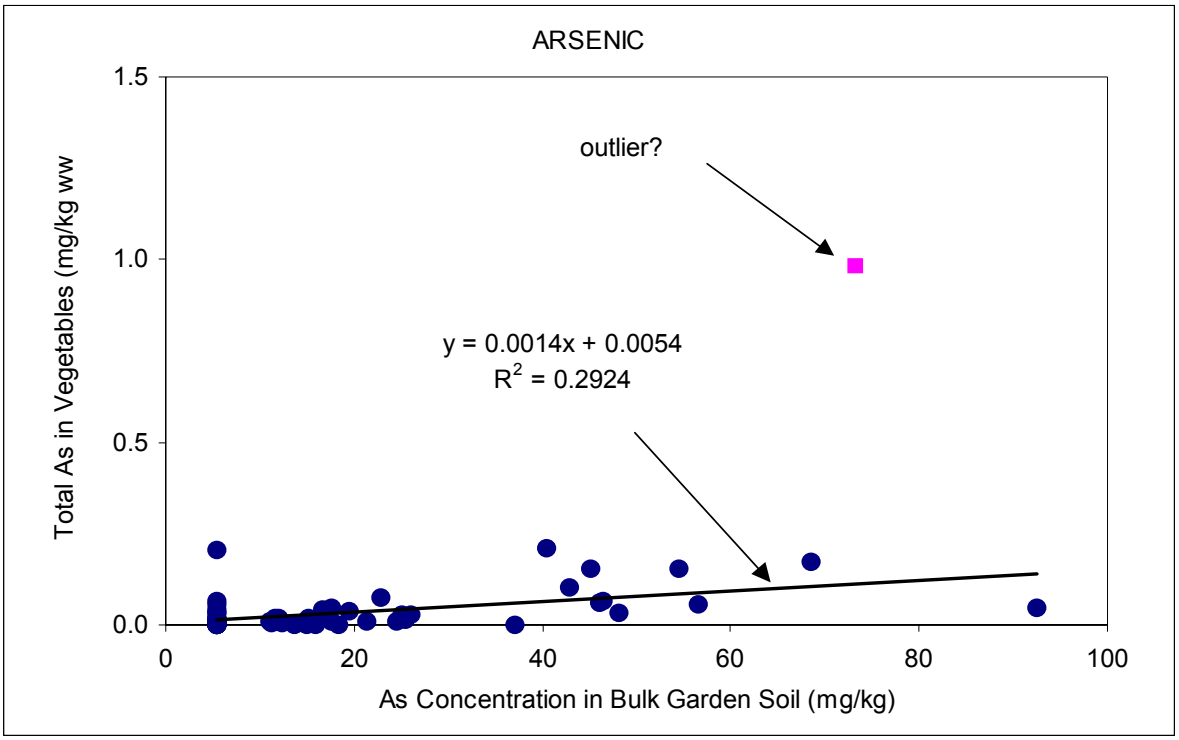
Site	Slope (ppm in dust per ppm in yard soil)	
	Arsenic	Lead
Anaconda	0.31	
Bingham Creek		0.43
Butte		0.24
Deer Lodge	0.001	-0.01
East Helena		0.88
Flagstaff/Davenport		0.06
Midvale OU1	0.03	0.04
Leadville	0.10	0.33
Murray Smelter	0.17	0.19
Sandy City		0.13
Sharon Steel		0.76

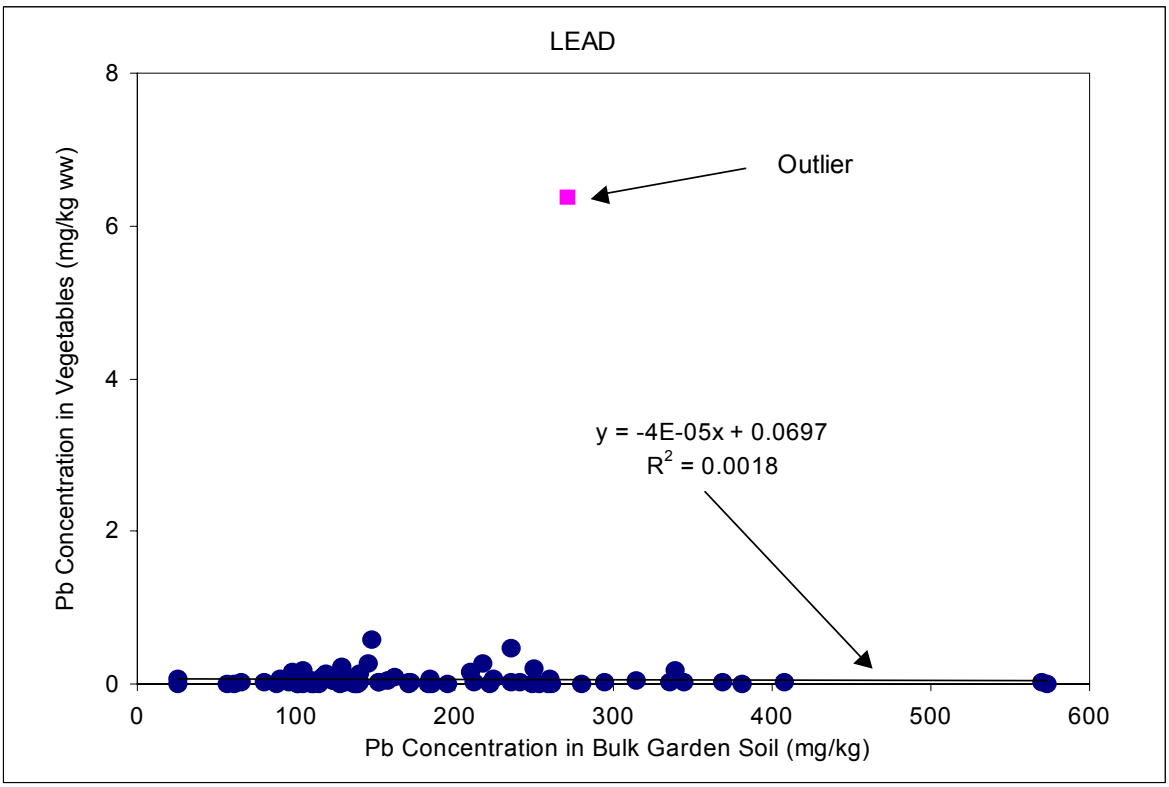
2.6.3 Residential Garden Sampling

Another pathway by which residents might be exposed to soil-related contaminants is ingestion of vegetables grown in home gardens that contain contaminated soil. In order to obtain site-specific data on this potential exposure route, USEPA Region VIII collected 72 samples of different types of garden vegetables from 19 different properties around the site. As detailed in the sampling plan (USEPA 1999d), each vegetable sample was washed in de-ionized water to minimize the amount of adhering soil. Vegetables were not peeled before analysis. At each location where a vegetable sample was collected, a co-located sample of garden soil was also collected. The detailed results for arsenic and lead levels in garden vegetables and soil are presented in Appendix A.

For arsenic, the mean concentration in vegetables (averaged across all samples) was 0.043 mg/kg wet weight (43 ng/g ww). A graph showing the relationship between the concentration of arsenic in garden soil and the corresponding concentration in garden vegetables is shown in Figure 2-10 (upper panel). As seen, one data point (an onion sample from property 6) appears to be somewhat higher than expected based on the other samples. The basis for this apparently high value is not known, but might be attributable to incomplete removal of soil from the sample prior to analysis, or to an uptake of arsenic into the outer skin of the onion. If that sample is considered to be un-representative of what would typically be ingested from home-grown garden vegetables (either because the vegetables would be more thoroughly washed and/or peeled before being eaten), then the mean concentration of arsenic in vegetables is 30 ng/g wet weight. The slope of the best-fit regression line through the data (outlier excluded) is quite low (0.0014 mg/kg wet weight per mg/kg in soil), but the slope is statistically different from zero ($p < 0.001$, $R^2 = 0.292$).

Figure 2-10 Relation between Total Arsenic in Garden Vegetables and Garden Soil





For lead, the mean concentration across all samples was 0.15 mg/kg wet weight (150 ng/g ww). A graph showing the relationship between the concentration of lead in garden soil and the corresponding concentration in garden vegetables is shown in Figure 2-10 (lower panel). As seen, one data point (a garlic sample from property 11) appears to be substantially higher than expected based on the other samples. As above, the basis for this apparently high value is not known, but might be attributable to incomplete removal of soil from the sample prior to analysis. If that sample is considered to be an outlier and is excluded, then the mean concentration of lead in vegetables is 62 ng/g wet weight. The slope of the best-fit regression line through the data (outlier excluded) is -4E-05 mg/kg wet weight per mg/kg in soil, which is not statistically different from zero ($p > 0.5$).The relationship between the concentration of arsenic and lead in garden soil and yard soil is shown in Figure 2-11. For lead (lower panel), the data are based on the mean garden soil values for the 19 gardens sampled collected during the garden vegetable sampling effort described above. For arsenic (upper panel), the data set includes the 19 properties described above, plus an additional 17 composite garden soil samples that were collected following the completion of the Phase III effort. These 17 samples were specifically selected to include properties with yard soil concentrations of arsenic greater than 100 ppm. As seen, there is only a weak correlation between arsenic levels in yard soil and garden soil (slope = 0.066, $R^2 = 0.265$), although the slope is statistically different from zero ($p < 0.01$). For lead, both the slope (0.60) and the correlation ($R^2 = 0.410$) are somewhat higher than for arsenic, but the correlation is still rather weak. These results indicate that garden soil is not equivalent to yard soil, with levels of arsenic and lead tending to be lower in the gardens than in the yards. This might be because the garden soil is prepared by amending yard soil with clean soil, peat moss, or other additives that dilute the yard soil contaminant level, or because the source(s) that have affected the yard did not equally affect the gardens.

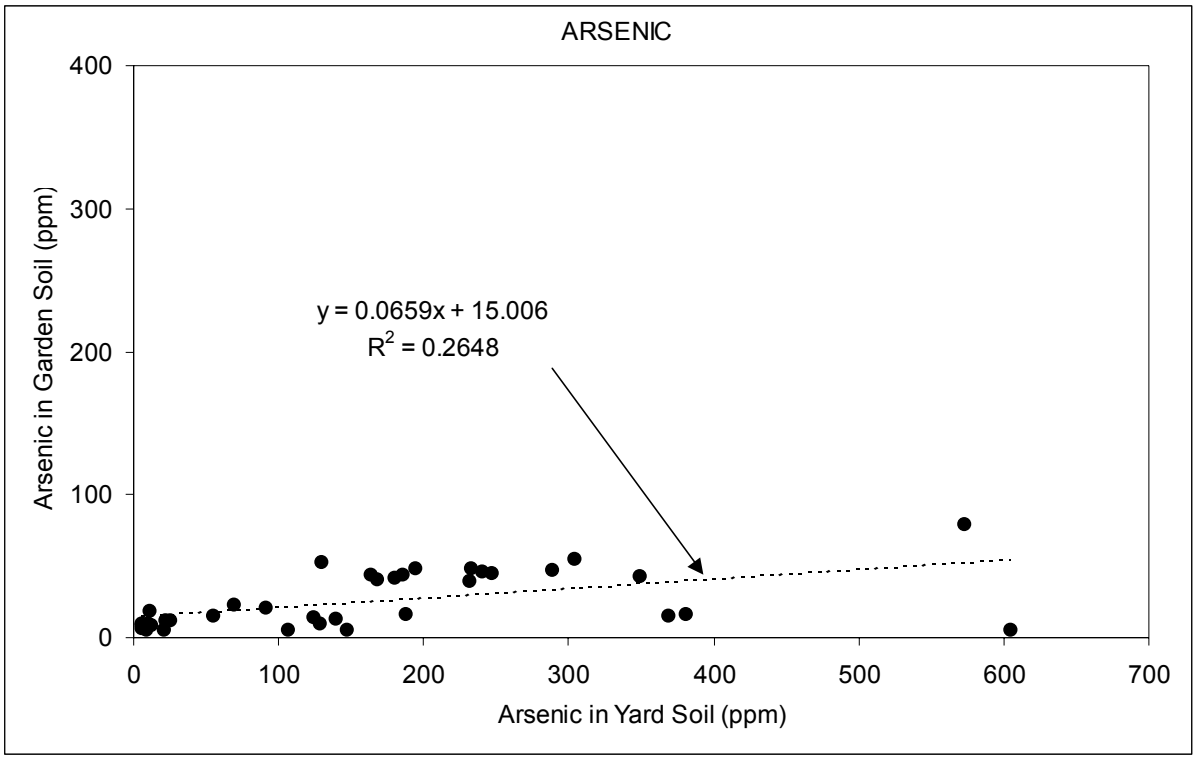
2.6.4 Sampling at Schools and Parks

As noted above, data on the levels of arsenic and lead in surface soil were collected at a number of schools and parks during the Phase I investigation. However, in most cases only a few samples were collected from each location, and not all schools and parks were sampled. Therefore, the Phase III Sampling and Analysis Plan included collection of 15-30 supplemental surface soil grab samples from each school and park within the site where access was granted. Samples were collected from a total of 7 parks or playgrounds and 15 schools. The results are shown in Table 2-6.

As seen, concentrations of lead are generally low, with average values ranging from 67-240 ppm. Mean concentrations of arsenic are also low in most locations (ranging from 11-14 ppm) and most maximum values are less than 25 ppm. An exception to this pattern occurred at one property owned by a school (location code S8). At this property, arsenic concentrations in two soil samples were significantly higher than the other samples (1517 ppm and 70 ppm)[2]. These values occur adjacent to each other near a sidewalk, and are surrounded by samples with arsenic

[2] These two samples were re-analyzed in triplicate to confirm the data. The mean values for the re-analyzed samples were 978 ppm and 114 ppm, respectively.

Figure 2-11 Relation between Contaminants in Garden Soil and Yard Soil



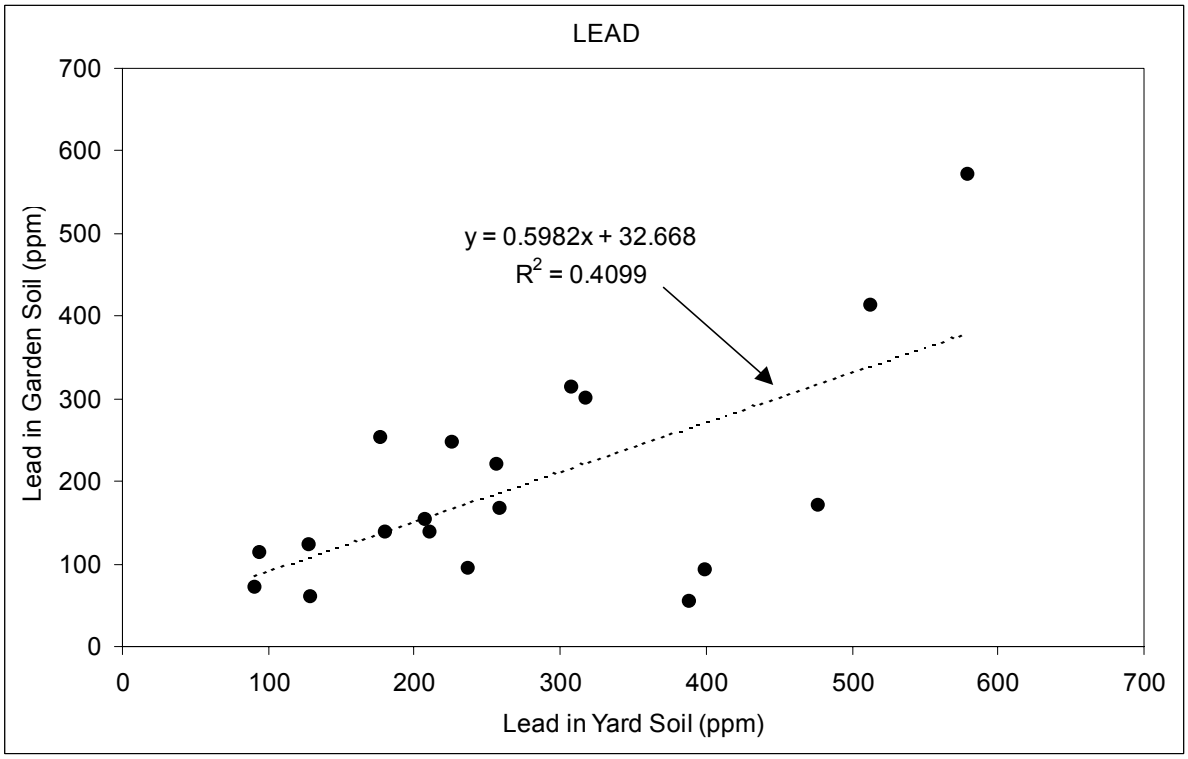


Table 2-6 Phase III soil Data for Schools and Parks

Category	Code	N	Arsenic (ppm)			Lead (ppm)		
			Mean (a)	Max	Min	Mean	Max	Min
School	S1	30	11	12	11	95	164	52
	S2	30	12	19	11	200	628	55
	S3	30	11	11	11	67	126	52
	S4	15	11	13	11	83	102	57
	S5	30	11	11	11	72	255	52
	S6	15	11	11	11	69	95	52
	S7	30	11	12	11	104	245	52
	S8	30	67	1517	11	310	1811	88
	S9	30	11	18	11	223	567	61
	S10	30	12	19	11	235	359	127
	S11	30	11	17	11	136	901	52
	S12	30	11	13	11	70	159	52
	S13	30	11	11	11	120	354	52
	S14	30	11	11	11	172	316	100
	S15	30	11	17	11	119	352	52
Park	P1	30	14	21	11	215	398	52
	P2	30	12	18	11	134	290	52
	P3	30	11	17	11	134	308	52
	P4	30	12	21	11	218	294	110
	P5	30	11	12	11	91	153	52
	P6	30	11	15	11	144	299	67
	P7	30	12	19	11	240	614	52

(a) Non-detects evaluated without adjustment

concentrations of 17-23 ppm. This suggests there might be a small arsenic "hot spot" at this location. The property was being developed for use as a school, but no children were present at the site at the time of sampling, so this was not a source of immediate concern. However, EPA Region VIII worked with the property owner to ensure that this location was re-landscaped and covered with a layer of clean topsoil during development so that future exposures would not be of concern.

2.6.5 Phase III Biomonitoring Program

In keeping with the approach established during Phase II, properties identified during Phase III which had an arsenic concentration above 400 ppm or a lead concentration above 2,000 ppm were scheduled for soil removal and replacement[3]. All residents at such properties were encouraged to participate in a voluntary biomonitoring program to evaluate if excess exposure was occurring to arsenic and/or lead. However, only seven individuals chose to participate. Summary statistics for those individuals are shown in Table 2-7. For convenience, reference values indicating the typical and upper end of the normal range are also presented.

As seen, similar to the observations obtained during Phase II, there were no cases where individuals living at the properties scheduled for soil removal had arsenic or lead levels that exceeded the "background" range typically seen in members of the general population. Although this data set is too small to draw firm conclusions, the results provide no indication that exposures at these locations were of immediate health concern.

2.7 DATA SELECTED FOR USE IN THIS RISK ASSESSMENT

The data from the Phase III sampling program were selected for use in this risk assessment because 1) all Phase III data were collected in accordance with project plans that were developed with careful consideration of the Data Quality Objectives (DQOs) needed to support risk assessment calculations, and 2) all data collected during Phase III are accompanied by Quality Assurance (QA) samples that allow detailed evaluation of the reliability of the data. A detailed review of these quality assurance data (USEPA 2000e) reveal that the data collected are of high quality, with adequate accuracy and precision to support a reliable evaluation of human health risk.

Data collected during Phase I/Phase II were not used because they were collected only with the intent of identifying locations that exceeded the removal action levels, and were not intended to support detailed risk calculations or remedial decision making. More specifically, data from Phase I/Phase II were not used because 1) many samples had elevated detection limits for arsenic (average = 51 ppm, range = 44 to 800 ppm), 2) the sampling density at each property was sometimes too low to ensure representativeness, and/or 3) exact sampling locations within a property were not always clear. However, despite these limitations, it is clear that the data from Phase I/Phase II and from Phase III are generally similar, each indicating the occurrence of scattered properties with elevated levels of lead and/or arsenic.

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[3] The concentration of arsenic that triggered an immediate cleanup during the Phase III program (400 ppm) was based on the lower limit of the range of concern identified by the USEPA Region VIII toxicologist.

Table 2-7 Biomonitoring Data for Residents at Phase III Removal Properties

Demographic Data		Biomonitoring Data					
		Blood Lead		Hair Arsenic		Urinary Inorganic Arsenic	
Index Number	Age (years)	Value (ug/dL)	Qual.	Value (ug/g)	Qual.	Value (ug/L)	Qual.
1	2	1	U	0.75		10	U
2	35	1	U	0.2	U	20	U
3	36	1	U	0.35	U	10	U
4	42	5		0.26	U	10	U
5	59	3		0.28	U	20	U
6	75	2		0.41	U	10	U
7	ND (adult)	NA		NA		10	U

U=Target analyte not detected

Summary Statistics

Blood Lead

	Site Data (a)					Reference (b, c)	
Age (years)	N	Detect. Freq.	Geo. Mean (ug/dL)	Min (ug/dL)	Max (ug/dL)	Typical (ug/dL)	High End (ug/dL)
1-5	1	0/1	1.0	1	1	2.5 - 4.1	> 10
>=6	5	3/5	2.0	1	5	1.5 - 4.0	> 10
All	6	3/6	1.8	1	5	2.3 - 2.8	> 10

Hair Arsenic

	Site Data (a)					Reference (d)	
Age (years)	N	Detect. Freq.	Mean (ug/g)	Min (ug/g)	Max (ug/g)	Typical (ug/g)	High End (ug/g)
0-6	1	1/1	0.75	0.75	0.75		
>6	5	0/5	0.3	0.20	0.41	<0.2	1.0
All	6	1/6		0.20	0.75		

Urinary Inorganic Arsenic

	Site Data (a)					Reference (d)	
Age (years)	N	Detect. Freq.	Mean (ug/L)	Min (ug/L)	Max (ug/L)	Typical (ug/L)	High End (ug/L)
0-6	1	0/2	10.0	10	10		
>6	6	0/6	13.3	10	20	<10	20
All	7	0/7	12.9	10	20		

a Summary statistics calculated using unadjusted values for non-detects
b Brody et al 1994
c Pirkle et al 1998
d NRC 1999

ug = microgram
dL = deciliter (0.1 L)
g = gram
L = liter

SECTION 3
EXPOSURE ASSESSMENT

Exposure is the process by which humans come into contact with chemicals in the environment. In general, humans can be exposed to chemicals in a variety of environmental media (e.g., soil, dust, water, air, food), and these exposures can occur through one or more of several pathways (ingestion, dermal contact, inhalation). Section 3.1 provides a discussion of possible pathways by which area residents and workers might come into contact with contaminants present in outdoor soil. Section 3.2 describes the basic methods used to estimate the amount of chemical exposure which humans may receive from direct and indirect contact with contaminants derived from outdoor soil.

3.1 CONCEPTUAL SITE MODEL

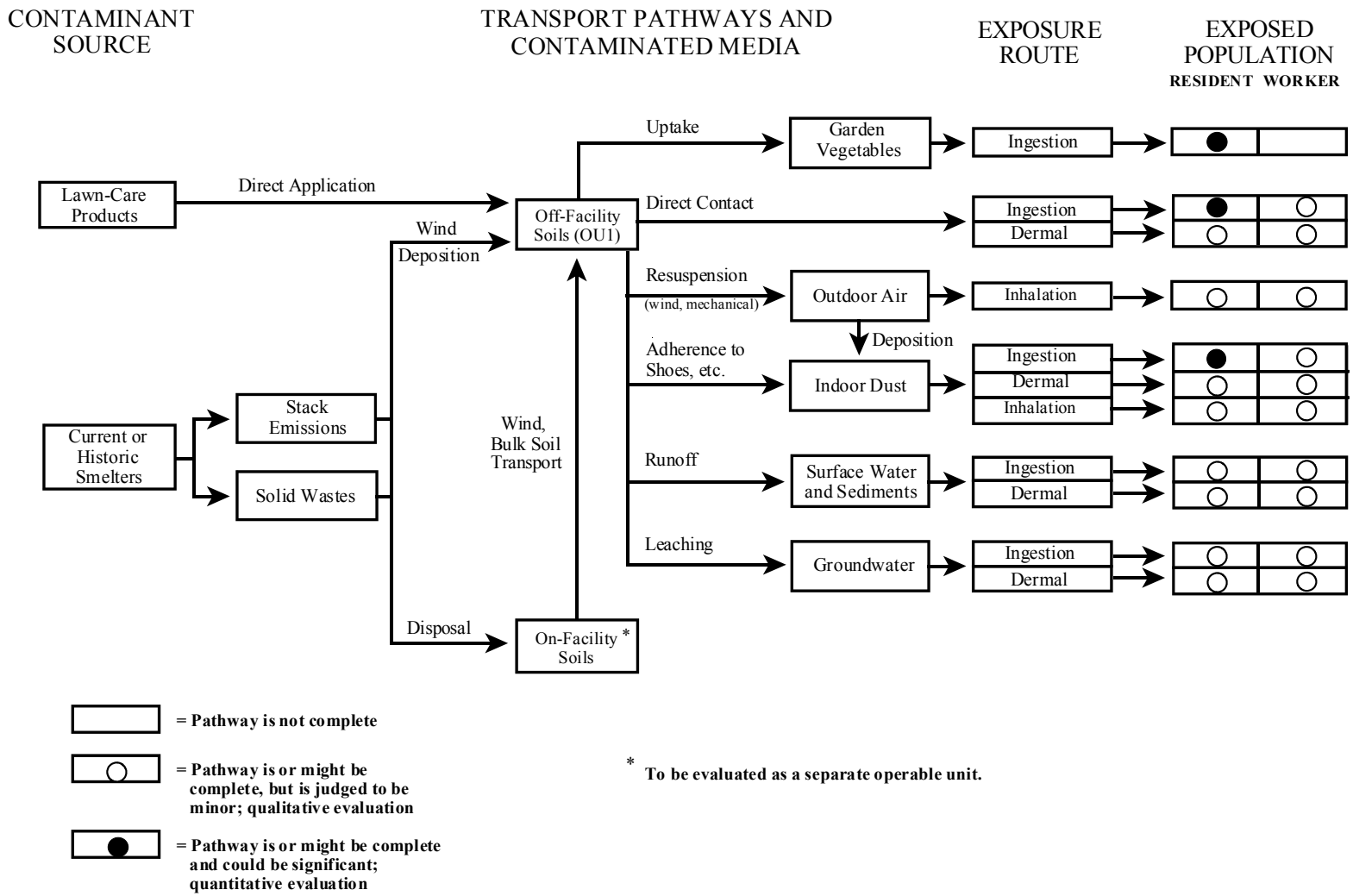
Figure 3-1 presents a conceptual site model for Operable Unit 1 (off-facility soils), showing the main pathways by which contaminants from current or former smelter activities and other sources might have reach off-facility soils, and the pathways by which people who live or work within the VBI70 site boundary might come into contact with those contaminants. This conceptual model was developed in consultation with local community groups as well as representatives from the City and County of Denver (CCOD), the Colorado Department of Public Health and Environment (CDPHE), and the Agency for Toxic Substances and Disease Registry (ATSDR). Exposure scenarios that are considered most likely to be of concern are shown in Figure 3-1 by boxes containing a solid circle, while pathways which are judged to contribute only minor exposures are shown by boxes with an open circle. Incomplete pathways (i.e., those which are not thought to occur) are shown by open boxes.

The following sections present a more detailed description of each of the exposure scenarios which are potentially relevant to the risk assessment for Operable Unit 1, and presents the basis for selecting the pathways that are of sufficient concern that quantitative evaluation is appropriate.

3.1.1 Potential Sources

The source of soil contamination at off-facility soils at the VBI70 site is not yet established. Two alternative hypotheses (which are not mutually exclusive) are that the contamination observed in off-facility soils (mainly residential soils) is due to 1) smelter-related releases (either airborne fallout from historic operations and/or bulk transport of contaminated waste material), or 2) application of some sort of pesticide or lawn care product (e.g., various herbicides or pesticides) that contained arsenic and/or lead. (Such products were commercially available and widely used in the period from the 1950s to the early 1970s). Studies are currently underway to obtain data that may help distinguish between these alternatives (USEPA 1999e). However, it is not necessary to know the source of the contamination in order to evaluate the potential human health risks from the contamination.

Figure 3-1 Conceptual Site Model for Operable Unit 1
Revision 2



3.1.2 Migration Pathways

Metals in soil tend to have relatively low mobility. Metals are not volatile, but may enter air attached to dust particles that are eroded from the yard soil into air by wind or mechanical forces. This is one pathway by which yard soil may enter a house and contribute to indoor dust. Another pathway by which yard soil may contribute to indoor dust is by bulk transport of soil adhering to shoes, clothing, pets, etc. Metals in soil can also leach downward toward groundwater, and can migrate as a function of surface water erosion. Finally, metals in soil can be taken up into home-grown garden vegetables.

3.1.3 Exposed Populations and Potential Exposure Scenarios

There are a number of different groups or populations of humans who may directly or indirectly come into contact with contaminants in area soils. This includes area residents and workers, as well as individuals who may be exposed at area schools or parks. The following text describes the scenarios which are considered plausible for each population, and identifies which are likely to be most important and which are sufficiently minor that they need not be evaluated quantitatively.

3.2 PATHWAY SCREENING

3.2.1 Residential Exposures

<u>Incidental Ingestion of Soil</u>

Few people intentionally ingest soil. However, it is believed that most people (especially children) do ingest small amounts of soil that adhere to the hands or other objects placed in the mouth. In addition, outdoor soil can enter the home and mix with indoor dust, which may also be ingested during meals or during hand-to-mouth activities. This exposure pathway is often one of the most important routes of human intake, so it was selected for quantitative evaluation.

<u>Dermal Contact with Soil</u>

Residents can get contaminated soil on their skin while working or playing in their yard. Even though information is limited on the rate and extent of dermal absorption of metals in soil across the skin, most scientists consider that this pathway is likely to be minor in comparison to the amount of exposure that occurs by soil and dust ingestion. This view is based on the following concepts: 1) most people do not have extensive and frequent direct contact with soil, 2) most metals tend to bind to soils, reducing the likelihood that they would dissociate from the soil and cross the skin, and 3) ionic species such as metals have a relatively low tendency to cross the skin even when contact does occur. These presumptions are supported by screening level calculations which indicate that dermal exposure of most metals is likely to be no larger (and probably much lower) than absorption due to soil ingestion (see Appendix B). Based on these considerations, along with a lack of data to allow reliable estimation of dermal uptake of metals

from soil, Region VIII generally recommends that dermal exposure to metals in soils not be evaluated quantitatively (USEPA 1995c). Therefore, this pathway was not evaluated quantitatively in this risk assessment.

Inhalation of Soil/Dust in Air

Particles of contaminated soil or dust become resuspended in air, and residents may breathe those particles both inside and outside their house. However, screening level calculations (presented in Appendix B) indicate that inhalation of soil particles released to air by wind erosion is likely to be a small source of risk (less than 0.2%) compared to the risk from incidental ingestion of soil. Likewise, monitoring data from a large construction project on the site indicate that mechanical erosion of soil into air is also likely to be of minimal concern. Based on this, it was concluded that inhalation exposure from airborne particulate matter is a sufficiently minor contributor to exposure and that it need not be included in the quantitative evaluation of residential exposure.

Ingestion of Home-Grown Vegetables

If a resident raises vegetables or fruits in a home garden that contains contaminated soil, some contamination may be taken up from the soil into the vegetable. If so, the resident would be exposed when those vegetables were consumed. Therefore, this pathway was selected for quantitative evaluation.

Contact with Surface Water and Sediment

There are no permanent surface water bodies within the VBI70 OU1 site boundary other than the Platte River. Although it is possible that site-related contaminants may be transported via surface water runoff and/or groundwater migration to the Platte, it is considered likely that human exposure levels to site-related contaminants would be relatively low at locations along the Platte. This is because human contact with surface water and sediments in the river is likely to be infrequent and relatively low in magnitude, at least compared with the level of exposure to residential yard soils and indoor dust. On this basis, exposure of residents to surface water and sediment is considered to be sufficiently minor that quantitative assessment is not warranted for Operable Unit 1.

Contact with Contaminated Groundwater

At present, there are no data to establish that metals in off-facility soils are a significant source of groundwater contamination. To the contrary, because the mass of contamination in the soil at any off-facility location is relatively small, it is not considered likely that off-facility soils are a significant source to groundwater. In addition, there are no known cases of area residents using a well for drinking water (the area is supplied with municipal water). On this basis, exposure to groundwater was not evaluated in this risk assessment.

3.2.2 Workplace Exposures

Workers at commercial or industrial locations within the site boundary may be exposed to soil while working in outdoor locations, so incidental ingestion, inhalation of particulates and/or dermal contact may occur. As is the case with residents, ingestion exposure is the most important of these exposure routes. Although only one soil sample has been collected from commercial properties at the VBI70 site[4], extensive sampling has been performed at commercial properties in the vicinity of the Globe plant (EnviroGroup 2000). This sampling has revealed that even the highest values detected during the sampling are below a level of potential health concern for workers, as shown below:

Summary of Soil Data from Commercial Properties in the Vicinity of the Globe Plant

Parameter	Arsenic	Lead
Number of commercial properties sampled	345	345
Average concentration (ppm)	20	145
Highest concentration (ppm) (average across property)	96	1064
Risk-based concentration for workers (ppm) (see Appendix C)	454	1104

Because there is no known reason why commercial properties in the vicinity of the Globe site should be less contaminated than commercial properties within Operable Unit 1 of the VBI70 site, these data are assumed to be representative of what would be obtained if sampling were to proceed at commercial properties within OU1. On this basis, it is concluded that sampling at commercial properties and detailed quantitative risk calculations for workers are not needed at OU1 of the VBI70 site. Therefore, the worker population is not evaluated further in this risk assessment.

3.2.3 Exposures at Schools and Parks

Area residents could also be exposed to contaminants in soil at community areas such as schools or parks. The pathway of primary concern for this scenario is direct ingestion of surface soil. As above, dermal contact and inhalation of airborne particles may occur, but these pathways are believed to be minor compared to the ingestion pathway.

As discussed above in Section 2.6.4 (see Table 2-6), concentrations of lead in surface soils from VBI70 schools and parks are generally low (67-240 ppm) and are below the EPA screening level (400 ppm) for health concern (USEPA 1994b). Mean concentrations of arsenic in soils from

[4] This sample was collected from a location that is currently commercial but is scheduled to be converted to a school.

schools and parks are also low in most locations (ranging from 11-14 ppm) and most maximum values are less than 25 ppm. An exception to this pattern occurred at one small location on one property owned by a school, but this apparent hot spot has been addressed by EPA and the property owner. On this basis, it is concluded that risks to children or other area residents are not of concern at area schools and parks, and further quantitative evaluation is not needed for this scenario.

3.3 SUMMARY OF PATHWAYS OF PRINCIPAL CONCERN

Based on the evaluations above, the following exposure scenarios are judged to be of sufficient potential concern to warrant quantitative exposure and risk analysis:

Exposure Scenarios of Potential Concern for Operable Unit 1

Population	Exposure Location	Medium and Exposure Route
Resident	Residences	Incidental ingestion of soil and dust in and about the home and yard Ingestion of home-grown vegetables

Other exposure pathways are judged to be sufficiently minor that further quantitative evaluation is not warranted.

SECTION 4
QUANTIFICATION OF EXPOSURE AND RISK FROM ARSENIC

4.1 OVERVIEW

The USEPA has established standard methods for estimating the level of exposure and risk to residents from a variety of chemical contaminants in soil. These methods are employed below to estimate the exposure and risk to residents at the VBI70 site from arsenic in soil. Whenever possible, site specific data are used in preference to non-site specific default assumptions.

Because the approach used to evaluate exposure and risk from lead is somewhat different than that used for arsenic, the assessment of lead risks is presented separately in Section 5.

4.2 QUANTIFICATION OF EXPOSURE

4.2.1 Basic Equation

The amount of a chemical which is ingested, inhaled, or taken up across the skin is referred to as "intake" or "dose", and is usually calculated using an equation of the following general form:

$$DI = C \cdot (IR/BW) \cdot (EF \cdot ED/AT)$$

where:

DI =	Daily intake of chemical (mg of chemical per kg of body weight per day)
C =	Concentration of the chemical in the contaminated environmental medium (soil, dust, etc.) to which the person is exposed. The units are mg of chemical per unit of environmental medium (e.g., mg/kg for soil, food, etc.).
IR =	Intake rate of the contaminated environmental medium. The units are usually kg/day for solid media (soil, dust, food).
BW =	Body weight of the exposed person (kg).
EF =	Exposure frequency (days/year). This describes how often a person is likely to be exposed to the contaminated medium over the course of a typical year.

ED = Exposure duration (years). This describes the exposure interval of concern (how long a person is likely to be exposed to the contaminated medium).

AT = Averaging time (days). This term specifies the length of time over which the average dose will be calculated. Usually, two different averaging times are considered:

- "Chronic" exposure includes averaging times on the scale of years (typically ranging from 7 years to 70 years). This exposure duration is used when assessing the non-cancer risks from chemicals of potential concern.

- "Lifetime" exposure employs an averaging time of 70 years. This exposure interval is selected when evaluating cancer risks.

In some cases (when the concentration of contaminants is sufficiently high that short-term exposures might be of concern), a separate evaluation of "subchronic" exposure (typically from several months to several years), or "acute" (single dose) exposure may also be performed.

Note that the last three factors (EF, ED, AT) combine to yield a factor between zero and one. Values near 1.0 indicate that exposure is nearly continuous over the specified averaging period, while values near zero indicate that exposure occurs only rarely.

For mathematical convenience, the general equation for calculating dose is often written as:

$$DI = C \cdot HIF$$

where:

HIF = Human Intake Factor. This term describes the average amount of an environmental medium contacted by the exposed person each day. The value of HIF is typically given by:

$$HIF = (IR/BW) \cdot (EF \cdot ED/AT)$$

The units of HIF are kg/kg-day for solid media such as soil, dust, and food.

4.2.2 Variability and Uncertainty in Exposure Calculations

For every exposure pathway of potential concern, it is expected that there will be differences between different individuals in the concentration of chemical to which they are exposed, as well as differences in intake rates, body weights, exposure frequencies and exposure durations. Thus, there is normally a wide range of average daily intakes between different members of an exposed population. Because of this, all daily intake calculations must specify what part of the range of doses is being estimated. Typically, attention is focused on two different parts of the exposure distribution:

> **Average or "Central Tendency" Exposure (CTE)** is either the arithmetic mean or the median exposure. It is calculated using the average values for all of the exposure parameters.
>
> **Reasonable Maximum Exposure (RME)** is the highest exposure that is reasonably expected to occur at a site. The intent of the RME is to estimate a conservative exposure case that is still within the range of possible exposures. This is done by using a combination of upper-bound estimates for some exposure parameters and average estimates for some exposure parameters.

This variability in exposure between different members of the population should not be confused with the difficulties that are often encountered in attempting to estimate either CTE or RME daily chemical intake levels. These difficulties arise because there are usually insufficient data to accurately define key exposure parameters such as typical and upper bound intake rates, exposure frequencies and exposure durations. Thus, the choice of values for average and upper-bound intakes are often rather uncertain.

4.2.3 Derivation of the Concentration Term

When people are exposed to a chemical in a medium such as soil, the level of exposure and risk is proportional to the average concentration in the area where exposure occurs. The location where exposure occurs (e.g., a specific residential yard or house) is usually referred to as the Exposure Unit (EU), and the average concentration within the EU is referred to as the Exposure Point Concentration (EPC). Typically, the EPC is estimated based on a set of measured values of the medium collected from the EU. However, the simple average of the measured values is only an estimate of the true mean, and the actual value could be either higher or lower. Because of this uncertainty, the USEPA typically recommends that, for chemicals such as arsenic, the EPC that is used to calculate exposure and risk be based on either the 95% upper confidence limit (UCL) of the mean concentration or the maximum concentration (whichever is lower) (USEPA 1989). Note that this approach is used for both the CTE and the RME exposure scenarios (USEPA 1992a). The equation used to calculate the UCL depends on what is known about the underlying distribution of values. In most cases, it is assumed the distribution is right-skewed, and the equation for a lognormal distribution is used (USEPA 1992a). However, when the data are described by a distribution that is more nearly symmetric, then the equation for a t-

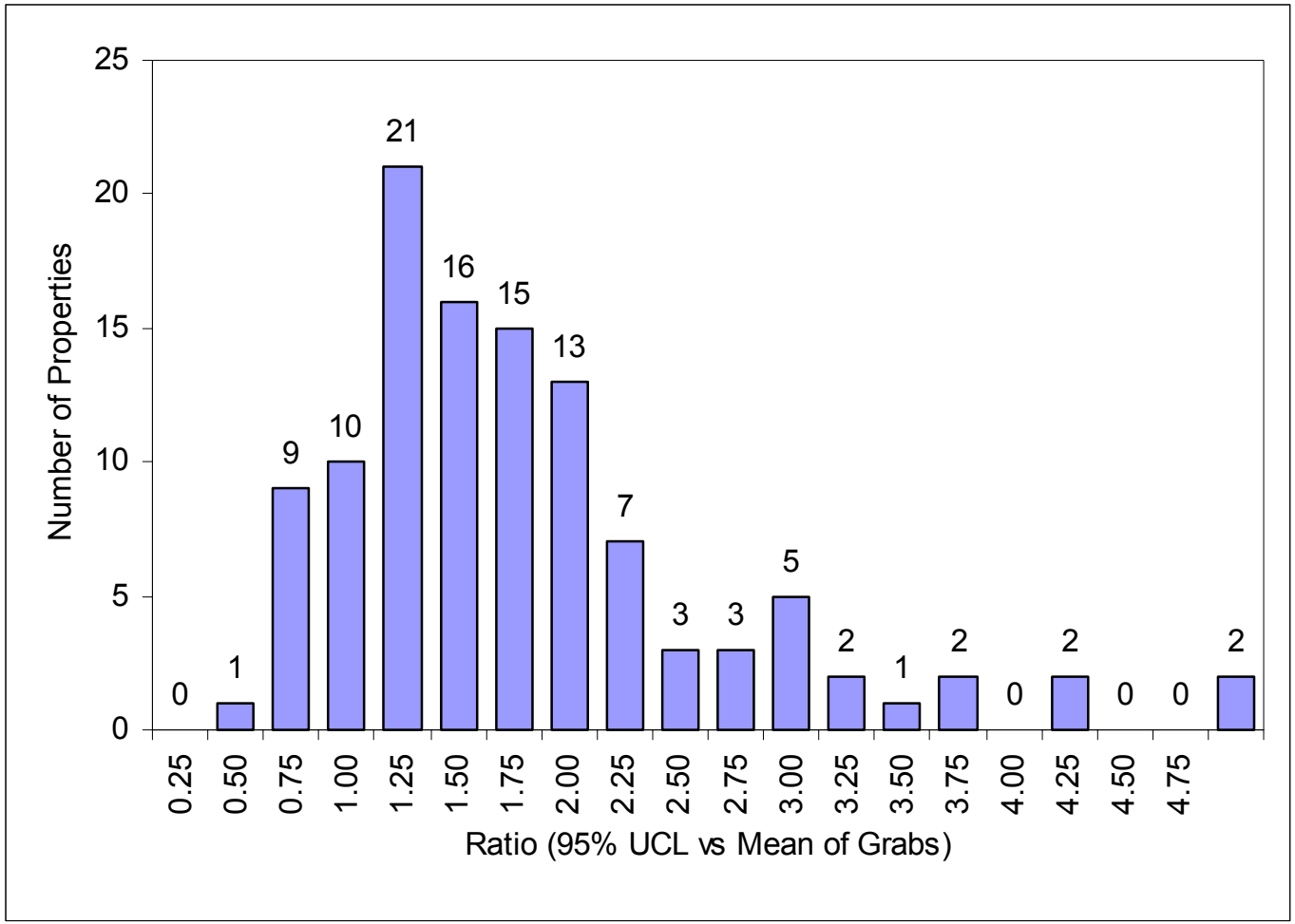
distribution is used (USEPA 1992a). Samples that are below the detection limit are evaluated using a value equal to one-half the detection limit.

As discussed in the Project Plan for the Phase III sampling (USEPA 1999d), preliminary data from the site indicated that although the distribution of grab samples from a property was likely to be approximately lognormally distributed, concentration values in 10-point composite samples drawn at random from a property were likely to be distributed approximately normally, indicating that the 95% UCL for a property could be calculated from the mean and standard deviation of the composite values using the t-equation. In most cases, data would not be available to test the validity of an assumption of this type. However, at this site, the assumption can be evaluated based on a supplemental set of data that were collected at 119 of the Phase III properties. At each of these properties (selected because it was suspected they might be of concern for short-term noncancer effects), a repeat set of 30 grab samples were collected, and the samples were analyzed individually rather than being composited. Because of the relatively large number of grab samples (30) collected at each property, the mean of the 30 samples at each property may be assumed to be relatively close to true mean at the property. Accepting the mean of the grab samples as a reliable estimate of the true mean, if the method used for calculating the 95% UCL for each property has worked correctly (i.e., if the assumption of normality of composite values is correct), approximately 95% of the UCL values should be larger than the mean of the 30 grab samples. The results of this check are shown in Figure 4-1, expressed as the frequency distribution of the ratio of the 95% UCL of the composites to the mean of the grab samples. The expected result is that approximately 95% of the ratios should have a ratio greater than 1.0. As seen, most samples (83%) had 95% UCL values greater than the mean of the grab samples (i.e., a ration greater than 1.0), but a total of 20 out of 119 (17%) had 95% UCL values less than the mean of the grab samples. When this same test was performed using the EPC rather than the 95% UCL, 75% of the samples had a ratio greater than 1.0. These results suggests that the approach used to calculate the UCL and the EPC from the composite samples may be slightly less conservative than intended. However, the actual number of UCL and EPC values that are not higher than the true mean may be somewhat less than estimated by the ratio test, since some of the sample means based on the grab samples may be significantly higher than the true means, even though the N value is 30. Further, in those cases where the 95% UCL or the EPC is not higher than the mean of the grab samples, the magnitude of the difference is relatively small (less than a factor of 2 in early all cases). On this basis, it is concluded that use of the t-equation to calculate 95% UCL and EPC values from the composite samples is reasonable and provides an adequate margin of safety.

4.2.4 Source of Exposure Parameters

The USEPA has collected a wide variety of data and has performed a number of studies to help establish reasonable values for many human exposure parameters. The chief sources of these standard default values are the following documents:

Figure 4-1 Comparison of the UCL based on Composites to the Mean of Grab Samples



1. Risk Assessment Guidance for Superfund (RAGS). Volume I. Human Health Evaluation Manual (Part A). USEPA 1989.

2. Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors". USEPA 1991a.
3. Superfund's Standard Default Exposure Factors for the Central Tendency and Reasonable Maximum Exposure. Draft. USEPA 1993.

4. Exposure Factors Handbook. Volumes I to III. USEPA 1997.

However, for some parameters, there is no guidance and there are few or no data to support the selection of CTE or RME values, so professional judgement and input from community members were utilized in some cases.

4.2.5 Quantification of Exposure of Residents to Soil

4.2.5.1 Long-Term (Chronic and Lifetime) Exposure

Basic Equation

Based on the assumption that the concentration of contaminants is approximately equal in outdoor yard soil and indoor house dust, the USEPA usually evaluates long-term average residential exposure to soil and dust in a single step. The basic equation is as follows:

$$DI_{sd} = EPC_{sd}\left(\frac{IR_{sd}}{BW} \cdot \frac{EF_{sd} \cdot ED}{AT}\right)$$

Both chronic and lifetime average intake rates are time-weighted to account for the possibility that an exposed individual may begin exposure as a child (USEPA 1989, 1991a, 1993), as follows:

$$TWA - DI_{sd} = EPC_{sd}\left(\frac{IR_c}{BW_c} \cdot \frac{EF_c \cdot ED_c}{AT} + \frac{IR_a}{BW_a} \cdot \frac{EF_a \cdot ED_a}{AT}\right)$$

where:

$TWA\text{-}DI_{sd}$ = Time-weighted Daily Intake from ingestion of soil and dust (mg/kg-d)
EPC_{sd} = Exposure Point Concentration of chemical in soil and dust (mg/kg)
IR = Intake rate of soil and dust (kg/day) when a child (IR_c) or an adult (IR_a)
BW = Body weight (kg) when a child (BW_c) or an adult (BW_a)
EF = Exposure frequency (days/yr) when a child (EF_c) or an adult (EF_a)
ED = Exposure duration (years) when a child (ED_c) or an adult (ED_a)
AT = Averaging time (days)

Default Exposure Parameters

Default values and assumptions recommended by USEPA (1989, 1991a, 1993, 1997) for evaluation of chronic and lifetime residential exposure to soil and dust are listed below:

USEPA Default Parameters for Long-Term Residential Exposure to Soil and Dust

Exposure Parameter	Unit	CTE	RME
IR as child	kg/day	1E-04	2E-04
IR as adult	kg/day	5E-05	1E-04
BW as child	kg	15	15
BW as adult	kg	70	70
EF as child or adult	days/yr	234	350
ED as child	years	2	6
ED as adult	years	7	24
AT (noncancer effects)	days	9·365	30·365
AT (cancer effects)	days	70·365	70·365

CTE=Central Tendency Exposure
RME=Reasonable Maximum Exposure

Based on the exposure parameters above, the time-weighted HIFs for chronic and lifetime exposure of residents to soil and dust are as follows:

Human Intake Factors (HIFs) for Long-Term Residential Exposure to Soil and Dust

Residential Exposure to Soil plus Dust	HIF_{sd} (kg/kg-d)	
	CTE	RME
TWA-chronic (non-cancer)	1.3E-06	3.7E-06
TWA-lifetime (cancer)	1.7E-07	1.6E-06

TWA = Time Weighted Average
HIF_{sd} = Human Intake Factor for soil and dust

Adjustment for Unequal Concentrations in Soil and Dust

As noted in Section 2, studies at a number of sites have revealed that the concentration of metals such as lead and arsenic is often not as high in indoor dust as in outdoor soil. In this situation, it is necessary and appropriate to evaluate exposure to soil and dust separately, as follows:

$$DI_{sd} = EPC_s \cdot HIF_s + EPC_d \cdot HIF_d$$

where:

EPC = Exposure Point Concentration in soil (EPC_s) or in dust (EPC_d)
HIF = Human Intake Factor for soil (HIF_s) or dust (HIF_d)

Derivation of the EPC_s Term

As noted above, the EPC for soil is the 95% UCL or the maximum detected value at an exposure area, whichever is lower. Because measurements of arsenic concentration in soil are based on bulk soil samples, and exposure is suspected to be associated mainly with the fine fraction, the value for EPC_s is adjusted to account for the enrichment of arsenic in the fine fraction compared to the bulk fraction as follows (see Section 2.6.1):

$$ECP_s = 1.21 \cdot EPC(bulk)$$

Derivation of the EPC_d Term

In general, the concentration of contaminants in dust can be expressed as a function of the concentration in outdoor bulk soil using the following equation:

$$EPC_d = D0 + ksd \cdot EPC(bulk\ soil)$$

where:

D0 = Concentration in dust (ppm) that is not attributable to yard soil
ksd = Fraction of indoor dust that is derived from outdoor soil

As discussed in Section 2.6.2, in order to derive a reliable site-specific estimate of the relation between yard soil and indoor dust, paired samples of yard soil and indoor dust were collected at 74 properties at the site. These data are presented in Figure 2-9. For arsenic, the best estimate of the relation between soil and dust is given by the equation:

$$EPC_d = 0.06 \cdot EPC_s + 11$$

That is, D0 = 11 ppm and ksd = 0.06.

Estimation of HIF_s and HIF_d

If f_s is defined as the fraction of total intake that is soil, then the HIF for soil and dust intake (combined) may be separated into its two component parts, as follows:

$$HIF_s = f_s \cdot HIF_{sd}$$
$$HIF_d = (1-f_s) \cdot HIF_{sd}$$

Data are sparse on the relative amounts of soil and dust ingestion by residents, but limited data support the view that total intake is composed of about 45% soil and 55% dust in children (Stanek and Calabrese 1992, USEPA 1994a). By extrapolation, this ratio is also assumed to apply to resident adults. Thus:

$$f_s = 0.45$$

Combined Final Equation

Combining all the relationships above yields the following final equation:

$$DI_{s,d} = 1.21 \cdot EPC(bulk) \cdot f_s \cdot HIF_{sd} + [D0 + ksd \cdot EPC(bulk)] \cdot (1 - f_s) \cdot HIF_{sd}$$

Substituting the exposure parameters above and simplifying yields the following:

Equations for Calculating Long-term Average Daily Intake of Arsenic from Soil (mg/kg-d)

Exposure Duration	CTE	RME
Chronic (noncancer)	7.5E-07·EPC(bulk) + 7.9E-06	2.1E-06·EPC(bulk) + 2.2E-05
Lifetime (cancer)	9.8E-08·EPC(bulk) + 1.0E-06	9.2E-07·EPC(bulk) + 9.7E-06

4.2.5.2 Sub-Chronic Exposure

In most cases, if chronic noncancer and cancer risks from arsenic are below a level of concern, risks from shorter term exposures will also be below a level of concern. However, there are some cases where this may not be so. For example, a child playing in the yard during the summer months might have soil intakes that are higher than the long-term average, and exposure might occur preferentially at a sub-area of the yard with arsenic levels that are higher than the yard-wide average. This is the scenario that is evaluated below.

Basic Equation

The basic equation used to evaluate noncancer risk from this type of scenario is the same as described previously, except that only soil exposure is considered (not dust). Thus, the basic equation is:

$$DI_s(\text{sub-chronic}) = EPC_s \cdot (IR/BW) \cdot (EF / AT)$$

Each of the inputs are discussed below.

EPC_s

It is assumed that during a relatively short exposure interval (e.g., a period of 1-3 months over the course of a summer), a child might play in a particular sub-location of the yard where soil

concentrations of arsenic are higher than the yard-wide average. Ideally, if data were available from multiple sampling points within each yard, it would be possible to calculate the average concentration for multiple sub-areas within the yard, and then evaluate exposure at a selected sub-location that is elevated compared to the average. The choice of which sub-area to assume for this exposure is a matter of judgement, but the ninth highest out of ten subareas (i.e., the 90th percentile of subarea means) seems reasonable. However, because the soil samples collected from each residential property during Phase III were all 10-point composites (each representing the yard wide average) rather than a set of discrete grab samples, these data are not suited for direct estimation of the distribution of mean concentration values in sub-areas of the yard. However, a conservative estimate of the 90th percentile sub-area mean may be derived as follows:

a) The distribution of grab sample values at a property can be estimated by extrapolation from the detailed grab sampling data collected during the Risk-Based sampling program and the Phase III grab sampling program. These data are shown in Figure 4-2. As seen, the standard deviation of the grab samples within a yard tends to increase in proportion to the mean value in the yard, and the ratio of the standard deviation compared to the mean (the coefficient of variation, or CV) may be estimated from the slope of the best-fit regression line through the data:

$$CV = s / m = 1.02 \quad \text{(where s = standard deviation and m = mean)}$$

Thus, at any yard where the mean concentration is known (as is the case for all properties sampled during Phase III), then the standard deviation for a set of grab samples from within that yard may be estimated using the equation above. Assuming the soil samples within the yard are distributed approximately lognormally, then the concentration corresponding to any specified percentile of all yard samples can be calculated.

b) Because the distribution of sub-area means will be narrower than the distribution of individual grab sample values, use of the 90th percentile of the underlying grab sample distribution will be a conservative estimate of the 90th percentile of the sub-area means. The 90th percentile is calculated as:

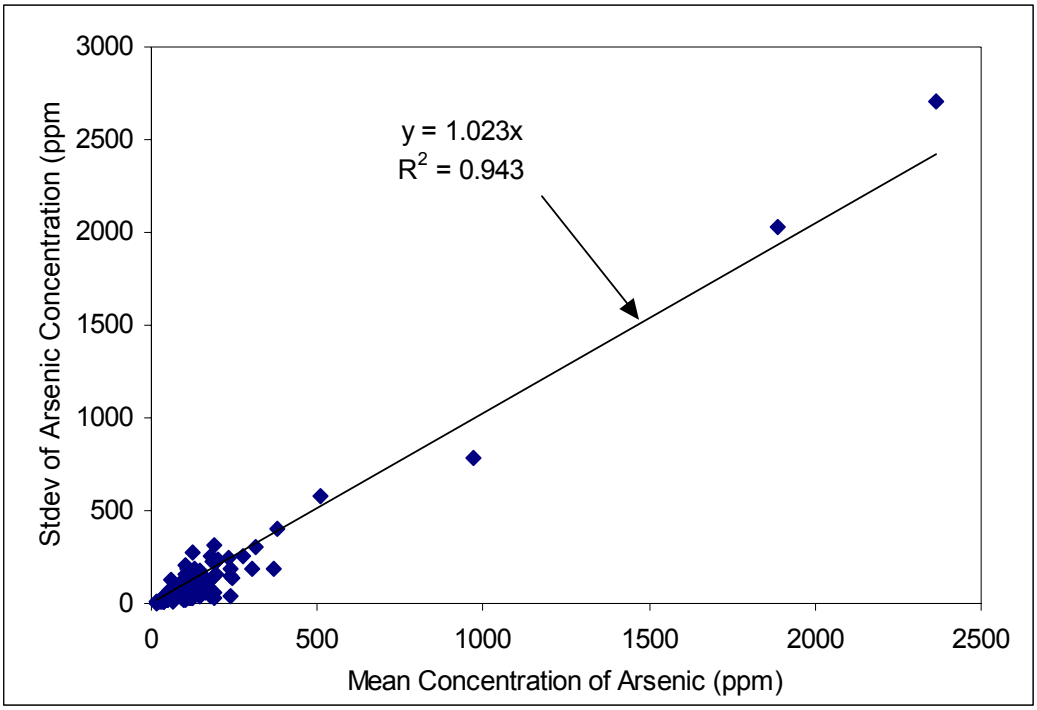
$$EPC_s(\text{sub-chronic}) = C(\text{90th percentile}) = GM \cdot GSD^{1.282}$$

where:

$$GM = \frac{m^2}{\sqrt{m^2 + s^2}}$$

$$GSD = \exp\left(\sqrt{\ln\left(\frac{m^2 + s^2}{m^2}\right)}\right)$$

Figure 4-2 Coefficient of Variation in Yard Soil Grab Samples



Solving and simplifying yields:

$$EPC_s(\text{subchronic}) = 2.07 \cdot \text{Mean}$$

As above, the EPC (the 95% UCL of the mean or the maximum value) is used as a conservative estimate of the mean, and this value is adjusted by a factor of 1.21 to account for potential enrichment of arsenic in the fine fraction compared to the bulk. Thus, the final equation for estimating the sub-chronic EPC in soil is:

$$EPC_s(\text{subchronic}) = 1.21 \cdot 2.07 \cdot EPC(\text{bulk}) = 2.50 \cdot EPC(\text{bulk})$$

Body Weight

The age at which soil ingestion by a child is most likely to occur is not known. Based on professional judgement, it is suspected that children ages 1-2 years are most at risk, so a body weight of 12.3 kg (the mean for boys and girls age 1-2) is assumed (USEPA 1997).

Soil Intake Rate

The average amount of soil ingested per day by a child during a short-time exposure is not known. As noted above, USEPA typically assumes a long-term (six year) average intake of 100 mg/day for a typical (CTE) child and a long-term average intake of 200 mg/day for an RME child. In the absence of data, it was assumed that the average intake over a period of several months (sub-chronic) might be about twice as high as the long-term average, so CTE and RME values of 200 mg/day and 400 mg/day were assumed, respectively. These values are consistent with the recommendations of USEPA (1997) which identifies 400 mg/day as an upper percentile for short term soil intake by children.

Exposure Frequency and Averaging Time

For the purposes of this evaluation, the sub-chronic exposure interval of chief concern is assumed to be the summer months when the child frequently plays outdoors and the soil is not frozen or snow-covered. In the absence of any site-specific data or USEPA guidance, the exposure frequency is assumed to be 15 days per month for the CTE child and 25 days per month for the RME child.

Summary of Sub-Chronic Exposure Assumptions

Sub-Chronic Exposure Assumptions

Variable	CTE	RME
EPC	2.50·EPC(bulk)	2.50·EPC(bulk)
Intake rate (mg/day)	200	400
Body weight (kg)	12.3	12.3
Exposure Frequency (days per month)	15	25
Averaging Time (days)	30	30
HIF (kg/kg-day)	8.1E-06	2.7E-05

Combined Final Equation

Substituting the exposure parameters above and simplifying yields the following:

CTE DI(subchronic) = 2.03E-05·EPC(bulk)
RME DI(subchronic) = 6.77E-05·EPC(bulk)

4.2.5.3 Acute Pica Exposure

Pica behavior is the intentional ingestion of non-food items, and this may include ingestion of soil. In this scenario, a child is envisioned as going to some location in the yard and ingesting a relatively large amount of soil over a short time period. The prevalence of soil pica behavior is not known, but is assumed to be low in the general population. However, it is plausible that many children exhibit some pica behavior if studied for long periods of time (USEPA 1997). For the purposes of this evaluation, the acute pica scenario focuses on the risks from a single event in which a child ingests a large mass of soil from a small location within a yard.

Basic equation

The basic equation used to evaluate risk from pica behavior is as follows:

$$DI_s(pica) = EPC_s \cdot IR/BW$$

Each of the inputs are discussed below.

EPC_s

Because exposure could occur at any location in the yard, the concentration value used as input could be the value from any sampling location where a child might play. In order to be conservative, it is assumed that this concentration could be a high value such as the 95th

percentile of the samples within the yard. As noted above, the composite samples collected during Phase III are not suited to estimating the 95th percentile directly, so the 95th percentile is estimated assuming a lognormal distribution and a CV of 1.02 (see Figure 4-2). The 95th percentile value is given by:

$$EPC_s(\text{acute}) = C(\text{95th percentile}) = GM \cdot GSD^{1.645}$$

Solving and simplifying yields:

$$EPCs(\text{acute}) = 2.81 \cdot \text{Mean}$$

As above, the EPC for the yard is used as a conservative estimate of the mean. However, in the case of pica behavior, it is assumed that bulk soil rather than fine soil is ingested. Thus, the EPC for acute exposure is calculated as follows:

$$EPC_s(\text{acute}) = 2.81 \cdot EPC(\text{bulk})$$

Body Weight

The age at which pica behavior is most likely to occur is not known. Based on professional judgement, it is suspected that children ages 1-2 years are more likely to engage in soil pica that either younger or older children, so a body weight of 12.3 kg (the mean for boys and girls age 1-2) is assumed (USEPA 1997).

Soil Intake Rate

Data on soil pica are very sparse. Based on the limited information that is available, USEPA (1997) has identified 10 grams as a reasonable value for use in an acute exposure assessment. However, this estimate is based on observations of only one child in one study, so this rate is considered to be especially uncertain. Because of this uncertainty, two alternative assumptions were evaluated in this risk assessment:

Assumed Pica Soil Intake (mg/event)

Case	CTE	RME
1	5,000	10,000
2	2,000	5,000

Summary of Acute Pica Exposure Assumptions

Acute Pica Exposure Assumptions

Variable	CTE	RME
EPC	2.81·EPC(bulk)	2.81·EPC(bulk)
Intake rate (mg/day) Case 1 Case 2	 5000 2000	 10000 5000
Body weight (kg)	12.3	12.3

Combined Final Equation

Substituting the exposure parameters above and simplifying yields the following:

Equations for Calculating Acute Pica Intake of Arsenic from Soil (mg/kg)

Scenario	CTE	RME
Case 1	1.14E-03·EPC(bulk)	2.28E-03·EPC(bulk)
Case 2	4.57E-04·EPC(bulk)	1.14E-03·EPC(bulk)

4.2.6 Quantification of Exposure of Residents to Home-Grown Vegetables

Basic Approach

Two basic options are available for evaluating exposure from home-grown garden vegetables. In the first approach, data are collected on the concentration of chemical in each type of vegetable grown at each garden, and these concentrations are multiplied by the intake rate appropriate for that specific type of vegetable. The second approach is to evaluate the intake from a garden as a whole, averaging concentration values across all vegetables from that garden, and multiplying by the estimated total intake rate for home-grown garden vegetables.

Each of these approaches has advantages and limitations. The strength of the first approach is that it can account for differences in concentration between vegetable types. However, this approach requires multiple measurements of concentration in each type of vegetable harvested from each garden, which is usually not possible when samples are collected at a single time point. In addition, the current or future resident might change the types of crops grown in a garden, invalidating the type-specific calculations for that garden. The advantage of the second approach is that it is less sensitive to the specific types of vegetables that happen to be present when samples are collected, but can be misleading if there are significant variations between vegetable types. After consideration of both options, the second approach was selected for use in this risk assessment, since there were not enough data for each vegetable type in each garden to support reliable type-specific dose calculations. Based on this approach, the equation for

evaluation of exposure from ingestion of home-grown vegetables or native vegetation is as follows:

$$DI_{gv} = EPC_{gv} \cdot IR_{gv} \cdot \left(\frac{EF_{gv} \cdot ED}{AT} \right)$$

where:

DI_{gv} = Average daily intake of chemical from home-grown garden vegetables (mg/kg-day)
EPC_{gv} = Concentration in garden vegetables (mg/kg wet weight), averaged across types
IR_{gv} = Average total intake rate of home-grown garden vegetables (kg wet weight per kg body weight per day)
EF_{gv} = Exposure frequency to home-grown garden vegetables (days/yr)
ED = Exposure duration (years)
AT = Averaging time (days)

Calculation of EPC_{gv}

At each of the 19 properties where garden vegetables samples were collected, the EPC for the garden vegetables was calculated as the 95% UCL of the mean concentration or the maximum detected value, whichever was smaller. The 95% UCL was calculated based on the assumption that the sample values were distributed lognormally, and non-detects were evaluated using an assumed concentration equal to ½ the detection limit.

At properties where no garden vegetables samples were collected, the concentration of arsenic in garden vegetables was estimated using site-specific data on the relationship between arsenic in yard soil and in garden soil, and between arsenic in garden soil and in vegetable tissues. These site-specific relationships have been presented previously (see Section 2.6.3), and are summarized below:

$$C(\text{garden}) = 0.066 \cdot C(\text{bulk yard soil}) + 15.01$$
$$C(\text{vegetable}) = 0.0014 \cdot C(\text{garden}) + 0.0054$$

Thus, given C(yard soil) at a property (i.e., the bulk soil EPC for the property), the concentration of arsenic in garden vegetables may be calculated using the equations above.

Adjustment for Organic Content

It is important to recognize that EPA measured the total arsenic content of each vegetable sample. However, some of the arsenic in vegetables occurs in an organic form that is believed to be substantially less hazardous than inorganic arsenic. The fraction of total arsenic that is inorganic varies from vegetable to vegetable, but a mean value is approximately 60% (Schoof et al. 1999). Thus, for arsenic ingestion in garden vegetables, the following adjustment is used:

$$EPC_{gv}(\text{inorganic}) = EPC_{gv}(\text{total}) \cdot 0.6$$

Intake Rates

A number of studies on the intake of homegrown garden vegetables are summarized in the Exposure Factors Handbook (USEPA 1997). Intake rates vary as a function of several parameters, including vegetable type, geographic region and age. For this evaluation, the intake rates were based on the seasonally-adjusted lifetime mean value of home-grown garden vegetable intakes by people living in the western region of the United States (USEPA 1997). These intake rates are summarized below:

Home Grown Vegetable Intake (kg wet weight/kg body wt/day)

Percentile	Value
50th (CTE)	4.92E-04
95th (RME)	5.04E-03

In this case, time-weighted averaging of intakes across childhood and adulthood is not needed since the lifetime average values above are essentially identical to the calculated time-weighted average values.

These intake rates are based on "household consumption", which reflects the amount of each type of food item purchased at the store. Thus, these rates do not account for loss of vegetable material during preparation. Therefore, USEPA (1997) recommends adjusting the intake rates above to account for the preparation loss, as follows:

$$IR(adj) = IR(unadjusted) \cdot \text{Loss Factor}$$

The mean preparation loss across multiple vegetable types is 14% (USEPA 1997), so the adjustment factor is 0.86.

Summary of Exposure Assumptions

These exposure parameters used to evaluate residential exposure from garden vegetable ingestion are summarized below:

Exposure Parameters for Residential Ingestion of Garden Vegetables

Parameter	CTE	RME
EPC (inorganic)	0.6·EPC(total)	0.6·EPC(total)
IR (kg wet weight/kg body wt/day)	4.92E-04	5.04E-03
Loss factor	0.86	0.86
EF (days/yr)	350	350
ED (years)	9	30
AT (noncancer effects) (days)	9·365	30·365
AT (cancer effects) (days)	70·365	70·365

Based on these exposure parameters, the HIF values for exposure of residents to home-grown vegetables are as follows:

Human Intake Factors for Exposure of Residents to Home-Grown Garden Vegetables

Residential Exposure to Home-Grown Garden Vegetables	HIF_{gv} (kg ww/kg-d)	
	CTE	RME
Chronic (non-cancer)	4.1E-04	4.2E-03
Lifetime (cancer)	5.2E-05	1.8E-03

Final Equations

Substituting the exposure parameters above and simplifying yields the following:

Equations for Calculating Exposure from Garden Vegetables

Effect	CTE (mg/kg-day)	RME (mg/kg-day)
Non-cancer	2.43E-04·EPCgv(total)	2.49E-03·EPCgv(total)
Cancer	3.13E-05·EPCgv(total)	1.07E-03·EPCgv(total)

Using the equations above to relate the EPC_{gv}(total) to bulk yard soil yields the following:

Equations for Calculating Exposure from Garden Vegetables Based on Bulk Yard Soil

Effect	CTE (mg/kg-day)	RME (mg/kg-day)
Non-cancer	2.25E-08*EPC(bulk soil) + 6.43E-06	2.30*EPC(bulk soil) + 6.59E-05
Cancer	2.89E-09*EPC(bulk soil) + 8.27E-07	9.88E-08*EPC(bulk soil) + 2.82E-05

4.3 TOXICITY ASSESSMENT

4.3.1 Overview

The objective of a toxicity assessment is to identify what adverse health effects a chemical causes, and how the appearance of these adverse effects depends on dose. In addition, the toxic effects of a chemical frequently depend on the route of exposure (oral, inhalation, dermal) and the duration of exposure (subchronic, chronic or lifetime). Thus, a full description of the toxic effects of a chemical includes a listing of what adverse health effects the chemical may cause, and how the occurrence of these effects depends upon dose, route, and duration of exposure.

The toxicity assessment process is usually divided into two parts: the first characterizes and quantifies the non-cancer effects of the chemical, while the second addresses the cancer effects of the chemical. This two-part approach is employed because there are typically major differences in the time-course of action and the shape of the dose-response curve for cancer and non-cancer effects.

Non-Cancer Effects

Essentially all chemicals can cause adverse health effects if given at a high enough dose. However, when the dose is sufficiently low, typically no adverse effect is observed. Thus, in characterizing the non-cancer effects of a chemical, the key parameter is the threshold dose at which an adverse effect first becomes evident. Doses below the threshold are considered to be safe, while doses above the threshold may cause an effect.

The threshold dose is typically estimated from toxicological data (derived from studies of humans and/or animals) by finding the highest dose that does not produce an observable adverse effect, and the lowest dose which does produce an effect, following some specified duration of exposure. These are referred to as the "No-observed-adverse-effect-level" (NOAEL) and the "Lowest-observed-adverse-effect-level" (LOAEL), respectively. The threshold is presumed to lie in the interval between the NOAEL and the LOAEL. However, in order to be conservative (protective), non-cancer risk evaluations are not based directly on the threshold exposure level, but on a value referred to as the Reference Dose (RfD). The RfD is a duration-specific estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects, even in sensitive individuals.

The RfD is derived from the NOAEL (or the LOAEL if a reliable NOAEL is not available) by dividing by an "uncertainty factor". If the data are from studies in humans, and if the observations are considered to be very reliable, the uncertainty factor may be as small as 1.0. However, the uncertainty factor is normally at least 10, and can be much higher if the data are limited. The effect of dividing the NOAEL or the LOAEL by an uncertainty factor is to ensure that the RfD is not higher than the threshold level for adverse effects. Thus, there is always a "margin of safety" built into an RfD, and doses equal to or less than the RfD are nearly certain to be without any risk of adverse effect. Doses higher than the RfD may carry some risk, but

because of the margin of safety, a dose above the RfD does not mean that an effect will necessarily occur.

Cancer Effects

For cancer effects, the toxicity assessment process has two components. The first is a qualitative evaluation of the weight of evidence that the chemical does or does not cause cancer in humans. Typically, this evaluation is performed by the USEPA, using the system summarized in the table below:

Cancer Weight of Evidence Categories

Category	Meaning	Description
A	Known human carcinogen	Sufficient evidence of cancer in humans.
B1	Probable human carcinogen	Suggestive evidence of cancer incidence in humans.
B2	Probable human carcinogen	Sufficient evidence of cancer in animals, but lack of data or insufficient data from humans.
C	Possible human carcinogen	Suggestive evidence of carcinogenicity in animals.
D	Cannot be evaluated	No evidence or inadequate evidence of cancer in animals or humans.

For chemicals which are classified in Group A, B1, B2, or C, the second part of the toxicity assessment is to describe the carcinogenic potency of the chemical. This is done by quantifying how the number of cancers observed in exposed animals or humans increases as the dose increases. Typically, it is assumed that the dose response curve for cancer has no threshold, arising from the origin and increasing linearly until high doses are reached. Thus, the most convenient descriptor of cancer potency is the slope of the dose-response curve at low dose (where the slope is still linear). This is referred to as the Slope Factor (SF), which has dimensions of risk of cancer per unit dose.

Estimating the cancer Slope Factor is often complicated by the fact that observable increases in cancer incidence usually occur only at relatively high doses, frequently in the part of the dose-response curve that is no longer linear. Thus, it is necessary to use mathematical models to extrapolate from the observed high dose data to the desired (but unmeasurable) slope at low dose. In order to account for the uncertainty in this extrapolation process, USEPA typically chooses to employ the upper 95th confidence limit of the slope as the Slope Factor. That is, there is a 95% probability that the true cancer potency is lower than the value chosen for the Slope Factor. This approach ensures that there is a margin of safety in cancer risk estimates.

4.3.2 Toxicity Summary for Arsenic

The toxic effects of arsenic have been reasonably well established, based mainly on studies of humans exposed to elevated levels of arsenic from a variety of sources. The findings from these studies are summarized briefly below.

Acute Noncancer Effects

The estimated LD50 (the dose that causes 50% lethality) from arsenic ingestion is about 1-4 mg/kg in humans (USEPA 2001d). Oral exposure to non-lethal but high acute doses of arsenic produces marked irritation of the gastrointestinal tract, leading to nausea and vomiting. Other signs may include neuritis and vascular effects (USEPA 2001d). Incidents of acute arsenic toxicity are generally associated with accidental exposures, but may sometimes occur from ingestion of herbal medicines.

USEPA has reviewed available data on the acute and short-term toxicity of arsenic (USEPA 2001f), and has concluded that a large cross-sectional study of arsenic-induced skin lesions in children (Mazumder et al.1998) identifies a NOAEL of 0.015 mg/kg/day. Because this value is based on observations in a large number of individuals, including those who are likely to be sensitive, an uncertainty factor of 1 is recommended, yielding an RfD of 0.015 mg/kg-day (USEPA 2001f). Likewise, ATSDR has reviewed the available data, and noted that a study by Mizuta et al. (1956) reported multiple signs of acute arsenic toxicity in people exposed to arsenic-contaminated soy sauce. The exposure level causing the effects was estimated to be about 3 mg/day, which corresponds to a dose of about 0.05 mg/kg-day. Based on this study, ATSDR (2000) derived an acute oral MRL of 0.005 mg/kg/day by using a safety factor of 10 to extrapolate from the LOAEL to a NOAEL. ATSDR recommends use of this MRL as a screening value.

Subchronic Noncancer Effects

Symptoms resulting from sub-chronic ingestion of lower doses of arsenic often begin with a vague weakness and nausea. As exposure continues, symptoms become more characteristic and may include signs such as diarrhea, vomiting, anemia, injury to blood vessels, damage to kidney and liver, and impaired nerve function that leads to "pins and needles" sensations in the hands and feet. The USEPA has developed a subchronic oral RfD for arsenic of 6E-03 mg/kg-d (USEPA 1995b). This value is based on an estimated LOAEL of 0.06 mg/kg-day in humans (both children and adults) exposed to arsenic for periods of time from six months up to about 15 years. An uncertainty factor of 10 is used to account for extrapolation from a LOAEL to a NOAEL.

Chronic Noncancer Effects

Chronic exposure to arsenic is associated with all of the effects noted above. In addition, after exposure continues for a sufficient period of time, an unusual pattern of skin abnormalities,

including dark and white spots and a pattern of small "corns" may occur, especially on the palms and soles (ATSDR 2000, USEPA 2001d).

The average daily intake of arsenic that produces these skin effects varies from person to person. In a large epidemiological study in Taiwan, Tseng et al. (1968) reported skin lesions in humans exposed to chronic oral doses of 0.014 mg/kg-day or higher. Intake was through the drinking water. These effects were not observed in a control population ingesting 0.0008 mg/kg-day.

The USEPA used the NOAEL of 0.0008 mg/kg/day for skin and vascular lesions (Tseng et al. 1968) to derive a chronic oral RfD of 3.0E-04 mg/kg/day (IRIS 2000). The NOAEL was divided by an uncertainty factor of 3 to account for both the lack of data to preclude reproductive toxicity as a critical effect and to account for some uncertainty in whether the NOAEL of the critical study accounts for all sensitive individuals (IRIS 2000). Confidence in the RfD is rated medium. A higher rating was not given due to uncertainties in dose estimates and other problems in the epidemiological data base (IRIS 2000).

Cancer Effects

There is strong evidence from a number of human studies that oral exposure to arsenic increases the risk of skin cancer (USEPA 1988, NRC 1999, ATSDR 2000, USEPA 2001d). The most common type of cancer is squamous cell carcinoma, which appears to develop from some skin corns. In addition, basal cell carcinoma may also occur, typically arising from cells not associated with the corns. Although these cancers may be easily removed, they can be painful and disfiguring and can be fatal if left untreated. More recent data indicate that chronic oral arsenic exposure may also increase the risk of internal cancers, including cancer of the bladder and lung (NRC 1999, USEPA 2001d).

Based on a study of skin cancer incidence in Taiwanese residents exposed mostly to arsenic in drinking water (Tseng et al. 1968), the USEPA has calculated a unit risk of 5E-5 $(\mu g/L)^{-1}$ corresponding to an oral slope factor of 1.5 $(mg/kg/day)^{-1}$ (IRIS 2000). Assuming a water intake of 2 L/day by a 70-kg person, a concentration of 10 ug/L corresponds to a lifetime excess cancer risk of about 4E-04. The NRC (1999) has reviewed a number of alternative approaches for quantification of cancer risk at low doses, and noted that the risk estimates depend heavily on the mathematical approach employed as well as the cancer data set utilized. Based on the incidence of bladder cancer in males in Taiwan, several different methods yield estimates of the EC01 (the concentration in water that results in a 1% increase in excess lifetime cancer risk) of about 400-450 ug/L. If the dose response curve is assumed to be linear and to have no threshold, this corresponds to an oral slope factor of about 0.8-0.9 $(mg/kg\text{-}day)^{-1}$, slightly lower than the value based on skin cancer. Assuming a water intake of 2 L/day by a 70-kg person, this slope factor would correspond to a risk of about 2E-04 at an exposure concentration of 10 ug/L.

More recently, Morales et al. (2000) used a number of alternative risk models to analyze the incidence of bladder and lung cancer in the Taiwanese population exposed to arsenic in drinking water. USEPA (2001d) reviewed these results and, after consultation with the authors, concluded that a model without a reference population was most appropriate, since the available

reference population (urban residents in Taiwan) are not considered to be a good control group for the rural workers exposed to the high arsenic levels. For people exposed at a concentration of 10 ug/L, the risk model preferred by USEPA yielded estimates of excess cancer risk of 0.6E-04 to 3.0E-04 for an average individual, and from 1.3E-04 to 6.1E-04 for an individual at the 90th percentile of the risk distribution. These risk estimates are similar to the risk estimates derived previously by USEPA and by NRC (1999). This indicates that the slope factor of 1.5 $(mg/kg\text{-}day)^{-1}$ based on the incidence of skin cancer in the Taiwanese population is likely to be generally appropriate for estimation of risks from other cancer types as well. This is probably because most individuals who develop arsenic-induced bladder or lung cancer also develop skin cancer, and so the total number of people with any type of arsenic induced cancer is similar to the number with skin cancer.

Potential Beneficial Effects

Several studies in animals suggest that low levels of arsenic in the diet may be beneficial for reproduction and normal postnatal development. The USEPA (1988) reviewed the evidence and concluded that the essentiality of low levels of arsenic in animals has not been established, but is plausible. The NRC (1999) also reviewed the evidence and noted that studies to date do establish that arsenic supplementation of low-arsenic semi-synthetic diets prevents the occurrence of abnormal reproductive or decreased growth in animals, but that there is no proof that arsenic is an essential element in humans or that it is required for any biochemical process.

If arsenic is beneficial or essential in animals, it is also likely to be so for humans. Based on the animal data, the estimated beneficial dose for humans would be approximately 10 to 50 µg/day (USEPA 1988). This level of arsenic intake is usually provided in a normal diet, and no cases of arsenic deficiency in humans have been reported (NRC 1999, ATSDR 2000).

Summary of Toxicity Values for Arsenic

Based on the information reviewed above, this risk assessment utilized the following toxicity factors for ingested arsenic:

Arsenic Toxicity Factors Utilized in the Risk Assessment

Toxicity Factor	Value	Source
Acute RfD	0.015 mk/kg-day	USEPA (2001f)
Subchronic RfD	0.060 mg/kg-day	USEPA (1995b)
Chronic RfD	0.0003 mg/kg-day	IRIS 2000
Oral Slope Factor	1.5 $(mg/kg\text{-}day)^{-1}$	IRIS 2000

RfD=Reference Dose

4.3.3 Adjustments For Relative Bioavailability

As discussed in USEPA (1989), most oral RfD and SF values developed by USEPA are based on the empirical relationship between the occurrence of toxic effects and the amount of chemical ingested, and the amount of chemical that is actually absorbed into the body is not explicitly considered. Thus, if it is expected that the absorption of a chemical from an on-site medium is significantly different than from the medium used in the study supporting the RfD or SF, then it is appropriate to adjust the RfD or SF to account for this difference in absorption. This adjustment increases the accuracy of the subsequent risk calculations while still being protective of public health.

The ratio of the absorption fraction for a chemical in site medium compared to the medium used in the key toxicity studies is referred to as the Relative Bioavailability (RBA). If reliable estimates of RBA are available for chemicals of potential concern in site media, these can be used to adjust the default RfD and SF values as follows:

$$RfD_{adj} = RfD_{default} / RBA$$
$$SF_{adj} = SF_{default} \cdot RBA$$

In the case of arsenic, all of the oral RfDs as well as the oral SF are based on studies of humans exposed to arsenic either in drinking water or in other readily absorbable forms. Thus, solid forms of arsenic in site soils may be less well-absorbed and require adjustments in the toxicity factors to derive appropriate estimates of toxicity.

In order to investigate the relative bioavailability of arsenic in site soils, USEPA performed a study in which five separate samples were fed to swine for 12 days. Swine were selected as the test species because it is believed the gastrointestinal system (and hence the behavior of ingested arsenic) in swine is similar to that in humans. The details of the study design and of the findings are presented in a separate report (USEPA 2001b). In brief, the study found that arsenic in site soils was less well absorbed than a readily soluble form of arsenic (sodium arsenate), with RBA values for individual samples of site soil ranging from about 0.18 to 0.45. Because it is believed that these differences in RBA reflect mainly experimental variation, a single site-wide RBA value was derived by calculating the 95% upper confidence limit of the mean RBA for all of the site soils tested. The resulting value was 0.42.

4.4 RISK CHARACTERIZATION FOR ARSENIC

4.4.1 Basic Approach

<u>Cancer Risk</u>

The risk of cancer from exposure to a chemical such as arsenic is described in terms of the probability that an exposed individual will develop cancer because of that exposure by age 70. For each chemical of concern, this value is calculated from the daily intake of the chemical from

the site, averaged over a lifetime (DI_L), and the slope factor (SF) for the chemical, as follows (USEPA 1989):

$$\text{Cancer Risk} = 1 - \exp(-DI_L \cdot SF)$$

In most cases (except when the product of $DI_L \cdot SF$ is larger than about 0.01), this equation may be accurately approximated by the following:

$$\text{Cancer Risk} = DI_L \cdot SF$$

Because of the uncertainty in both the exposure term and the slope factor term, USEPA guidance recommends that all cancer risk estimates be expressed to only one significant figure (USEPA 1989).

The level of total cancer risk that is of concern is a matter of personal, community and regulatory judgement. In general, it is the policy of the USEPA that remedial action is not warranted where excess cancer risks to the RME individual do not exceed a level of 1E-04 (USEPA 1991b).

Noncancer Risk

The potential for noncancer effects from exposure to a chemical is evaluated by comparing the estimated daily intake of the chemical over a specific time period (chronic, sub-chronic, acute) with the RfD for that chemical derived for the corresponding exposure period. This comparison results in a noncancer Hazard Quotient, as follows (USEPA 1989):

$$HQ = DI / RfD$$

where:

HQ = Hazard Quotient
DI = Daily Intake (mg/kg-day)
RfD = Reference Dose (mg/kg-day)

Because of the uncertainty in both the exposure term and the reference dose term, USEPA guidance recommends that all HQ values be expressed to only one significant figure (USEPA 1989). If the HQ for a chemical is equal to or less than one (1E+00), it is believed that there is no appreciable risk that noncancer health effects will occur, even in sensitive individuals. If an HQ exceeds 1E+00, there is some possibility that noncancer effects may occur, although an HQ above 1E+00 does not indicate an effect will definitely occur. This is because of the margin of safety inherent in the derivation of exposure estimates and RfD values. However, the larger the HQ value, the more likely it is that an adverse effect may occur.

4.4.2 Risks from Soil and Dust

4.4.2.1 Cancer Risk

Cancer risks from exposure of residents to arsenic in yard soil and indoor house dust were calculated for each property using the basic equations described above. The exposure point concentration (EPC) for soil at each property was the 95% UCL of the mean value of the three 10-point composite values or the maximum composite value (whichever was lower). The 95% UCL of the mean was calculated based on an assumption that the distribution of 10-point composite values at a property is likely to be approximately normally distributed (USEPA 1999d). Non-detects were evaluated by assuming a value equal to one-half the detection limit. The concentration in dust was calculated from the soil exposure point concentration as described in Section 4.2.5.1 (above). The resulting risk estimates are presented in Table 4-1.

For CTE exposure conditions, most properties have estimated excess cancer risks for exposures due to arsenic in soil plus dust that range from 1E-06 to 1E-05 (5th to 95th percentiles), with a maximum value of 9E-05. For RME exposure conditions, most properties have risks that range from 9E-06 to 1E-04 (5th to 95th percentiles), with 92 properties having risks of 2E-04 or higher. The highest RME risk value was 8E-04. As shown in Table 4-1, the spatial pattern of properties with arsenic RME cancer risk levels of 2E-04 or higher is approximately uniform across the site, with a frequency of about 1%-4% in each neighborhood.

In interpreting these risk estimates, it is important to recognize that arsenic is a naturally occurring element in soil. Figure 4-3 presents the distribution of mean arsenic concentrations in residential properties sampled during Phase III. As seen, the distribution is fairly well-characterized as the sum of two different lognormal distributions with the following statistics[5]:

Parameters of the Best Fit Lognormal Distributions

Statistic	Distribution 1	Distribution 2
GM (ppm)	7.3	28.4
GSD	1.5	3.5
AM (ppm)	8.0	62.5
Stdev (ppm)	3.6	123
95th (ppm)	15	224

[5] In order to estimate the parameters of the two lognormal distributions, the data were log-transformed and fit to an equation of the following form: Conc ~ k·N(a,b) + (1-k)·N(c,d), where a and b are the log-mean and log-standard deviation of distribution 1, and c and d are the log-mean and log-standard deviation of distribution 2. The parameter k is the mixing fraction of the two distributions. Parameter values (a, b, c, d, and k) were derived using least square regression on order statistics.

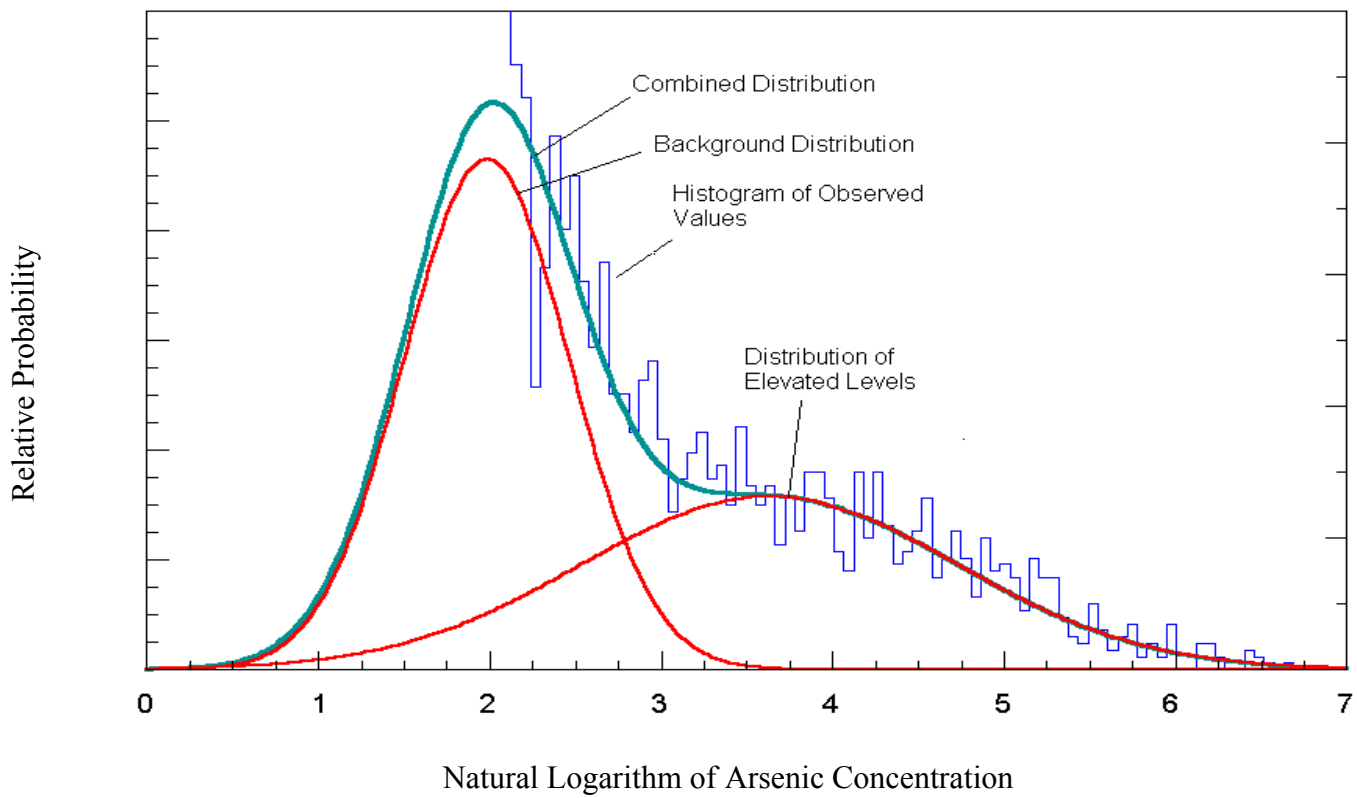
Table 4-1 Estimated Cancer Risk from Arsenic in Soil and Dust

Neighborhood	Number of Properties Evaluated	Number and Percent of Properties Within the Specified Risk Range							
		CTE Cancer Risk				RME Cancer Risk			
		<=1E-05	2E-05 - 1E-04	2E-04 - 1E-03	> 2E-03	<=1E-05	2E-05 - 1E-04	2E-04 - 1E-03	> 2E-03
Clayton	902	858	44			479	385	38	
		95%	5%			53%	43%	4%	
Cole	796	772	24			344	429	23	
		97%	3%			43%	54%	3%	
Elyria	59	58	1			17	41	1	
		98%	2%			29%	69%	2%	
Globeville	63	61	2			25	36	2	
		97%	3%			40%	57%	3%	
Swansea	1166	1132	34			610	528	28	
		97%	3%			52%	45%	2%	
All Neighborhoods	2986	2881	105			1475	1419	92	
		96%	4%			49%	48%	3%	

CTE=Central Tendency Estimate

RME=Reasonable Maximum Exposure

Figure 4-3 Distribution of Arsenic Values in Phase III Soils



Although this analysis cannot reveal the basis for the two-component nature of the distribution, the most straightforward interpretation is that the first distribution represents background, and the second distribution represents an extra amount of arsenic present in some yards due to some other (non-background) source. If so, the best estimate of the average background level of arsenic is about 8 ppm, although some background levels may range up to about 15 ppm or higher. Based on this, risks from naturally occurring levels of arsenic probably range from about 1E-06 for an average (CTE) person up to about 1E-05 for an upper-bound (RME) individual.

4.4.2.2 Chronic Noncancer Risks

Estimated risks of non-cancer health effects from chronic exposure to arsenic in soil and dust are shown in Table 4-2. For individuals with CTE exposure, risks at most properties fall between 2E-02 and 2E-01 (5th to 95th percentile), while individuals with RME exposure have risks that lie mainly between 5E-02 and 6E-01. These results indicate that risk of noncancer effects from chronic exposure is below a level of concern for most individuals at most locations. However, a total of 20 properties have RME HQ values of 2E+00 or higher, with a maximum value of 4E+00. These locations where noncancer risks enter a range of concern (HQ > 1E+00) are also above the usual level of concern (1E-04) for cancer.

4.4.2.3 Subchronic Noncancer Risks

Estimated risks of non-cancer health effects from sub-chronic exposure of area children to arsenic in soil are shown in Table 4-3. As seen, the incidence of properties with subchronic HQ values above 1E+00 is relatively low (2 out of 2,986 = 0.07% for CTE individuals, 53 out of 2986 = 1.8% for RME individuals). The maximum RME HQ value was 7E+00. All of the locations where subchronic noncancer risks enter a range of concern (HQ > 1E+00) are also above the usual level of concern (1E-04) for cancer.

4.4.2.4 Noncancer Risks from Acute Pica Behavior

Because of the substantial uncertainty which exists in most of the input parameters for the acute pica scenario, it is not possible to specify a single set of inputs that are "best". Rather, a range of HQ values were calculated for two different combinations of soil intake and RfD values:

Soil Intake and Arsenic Toxicity Factors for Calculating Non-Cancer Risks for two Acute Pica Scenarios

Variable	**Case 1**		**Case 2**	
	CTE	**RME**	**CTE**	**RME**
Soil intake (mg/day)	5000	10000	2000	5000
Acute RfD (mg/kg-d)	0.005		0.015	

Table 4-2 Estimated Chronic Noncancer Risk from Arsenic in Soil and Dust

Neighborhood	Number of Properties Evaluated	Number and Percent of Properties Within the Specified Risk Range							
		CTE Hazard Quotient				RME Hazard Quotient			
		<=1	2-5	6-10	> = 11	<=1	2-5	6-10	> = 11
Clayton	902	901	1	--	--	895	7	--	--
		100%	0.1%	--	--	99%	0.8%	--	--
Cole	796	796	0	--	--	786	10	--	--
		100%	0%	--	--	99%	1.3%	--	--
Elyria	59	59	0	--	--	59	0	--	--
		100%	0%	--	--	100%	0%	--	--
Globeville	63	63	0	--	--	63	0	--	--
		100%	0%	--	--	100%	0%	--	--
Swansea	1166	1166	0	--	--	1163	3	--	--
		100%	0%	--	--	100%	0.3%	--	--
All Neighborhoods	2986	2985	1	--	--	2966	20	--	--
		100%	0%	--	--	99%	0.7%	--	--

CTE=Central Tendency Estimate

RME=Reasonable Maximum Exposure

Table 4-3 Estimated Subchronic Noncancer Risks from Arsenic in Soil

Neighborhood	Number of Properties Evaluated	Number and Percent of Properties Within the Specified Risk Range							
		CTE Hazard Quotient				RME Hazard Quotient			
		<=1	2-5	6-10	> = 11	<=1	2-5	6-10	> = 11
Clayton	902	900	2			881	19	2	
		100%	0.2%			98%	2%	0.2%	
Cole	796	796	0			777	19	0	
		100%	0%			98%	2%	0.0%	
Elyria	59	59	0			58	1	0	
		100%	0%			98%	2%	0.0%	
Globeville	63	63	0			62	1	0	
		100%	0%			98%	2%	0.0%	
Swansea	1166	1166	0			1155	11	0	
		100%	0%			99%	1%	0.0%	
All	2986	2984	2			2933	51	2	
		100%	0.1%			98%	2%	0.1%	

It should be understood that these cases represent an uncertainty range, and that the "true" acute risk from pica behavior could lie anywhere in the interval. Indeed, it is quite possible that the true value even lies outside the range, since the actual distribution of pica soil intakes is not known.

The results are summarized in Table 4-4. As seen, the screening calculations above suggest that a large number of properties (ranging from 662 to 1841, depending on which set of input assumptions is deemed to be most appropriate) are of potential concern for the RME acute pica scenario. In the absence of reliable data on the magnitude and frequency of soil pica intake, and considering that national decisions continue on the most appropriate acute RfD for arsenic, it is difficult to judge which (if any) of these properties should be considered to be an authentic acute health risk to children. In this regard, it should be noted that even though many people are exposed to arsenic levels in soil that are predicted to be of acute concern, both within the VBI70 site and elsewhere across the country and around the world, to the best of USEPA's knowledge, there has never been a single case of acute arsenic toxicity reported in humans that was attributable to arsenic in soil. Thus, these results for the acute pica scenario are considered to be especially uncertain, since they predict a very substantial risk for which there is no corroborating evidence.

4.4.3 Risks from Home-Grown Vegetables

As discussed previously (see Section 2.6.3), a total of 72 different samples of home-grown garden vegetables were collected from 19 different properties across the site. At each property, the 95% UCL of the mean concentration of arsenic averaged across all vegetables samples from the garden was calculated using an assumption of lognormality. Non-detects were evaluated by assuming a value equal to one-half the detection limit. The EPC was then the 95% UCL or the maximum detected value (whichever was lower). As noted above, the concentration of inorganic arsenic was assumed to be 60% of total arsenic concentration.

Cancer and non-cancer risks from ingestion of home-grown vegetables at each of the 19 properties sampled were calculated by combining the EPC value with the estimated intake of garden vegetables described in Section 4.2.6, and calculating HI values and excess cancer risks as described in Section 4.4.1. The results are summarized in Table 4-5.

As seen, for individuals whose intake of home-grown garden vegetables is average (CTE) for the western United States, neither non-cancer nor cancer risks enter a range of concern at any property tested. For individuals whose intake is at the upper-bound (RME) of the distribution of garden vegetable consumption, cancer and non-cancer risks do enter a range of potential concern for two properties, as discussed below:

- At Property 6, a number of vegetables had arsenic concentration values that were higher than in samples from most other properties. The concentrations of arsenic in the garden

Table 4-4 Estimated Acute Noncancer Risk from Pica Behavior

Exposure Assumptions	Number and Percent of Properties Within the Specified Risk Range									
	CTE Hazard Quotient					RME Hazard Quotient				
	<=1	2-5	6-20	> 20	Total > 1	<=1	2-5	6-20	> 20	Total > 1
Case 1	1475	949	432	130	1511	1145	580	328	933	1841
	49%	32%	14%	4%	51%	38%	19%	11%	31%	62%
Case 2	2692	268	26	0	294	2324	487	162	13	662
	90%	9%	1%	0%	10%	78%	16%	5%	0%	22%

Case 1: RfD = 0.005 mg/kg; Pica intake rate = 10,000 mg

Case 2: RfD = 0.015 mg/kg; Pica intake rate = 5,000 mg

Table 4-5 Estimated Cancer and Noncancer Risk from Arsenic in Garden Vegetables

Property Number	Neighborhood	DF	EPC (based on inorganic arsenic) (a)	Chronic Noncancer Risk		Lifetime Cancer Risk	
				CTE	RME	CTE	RME
1	CLAYTON	1/10	3.2E-03	4E-03	4E-02	2E-07	8E-06
2	CLAYTON	0/1	2.5E-03	3E-03	3E-02	2E-07	7E-06
3	CLAYTON	0/1	2.6E-02	4E-02	4E-01	2E-06	7E-05
4	CLAYTON	3/6	3.3E-02	4E-02	5E-01	3E-06	9E-05
5	CLAYTON	1/2	1.2E-02	2E-02	2E-01	9E-07	3E-05
6	CLAYTON	12/12	3.3E-01	4E-01	5E+00	3E-05	9E-04
		11/11 (b)	1.3E-01	2E-01	2E+00	1E-05	3E-04
7	CLAYTON	0/2	9.6E-03	1E-02	1E-01	7E-07	3E-05
8	COLE	1/2	4.0E-02	5E-02	6E-01	3E-06	1E-04
9	COLE	1/2	1.1E-03	1E-03	1E-02	8E-08	3E-06
10	COLE	1/1	1.2E-03	2E-03	2E-02	9E-08	3E-06
11	COLE	4/6	1.2E-01	2E-01	2E+00	1E-05	3E-04
12	COLE	4/4	4.4E-02	6E-02	6E-01	3E-06	1E-04
13	COLE	3/9	2.0E-02	3E-02	3E-01	2E-06	5E-05
14	COLE	3/3	1.2E-02	2E-02	2E-01	9E-07	3E-05
15	COLE	0/4	1.9E-02	3E-02	3E-01	1E-06	5E-05
16	COLE	1/1	1.2E-02	2E-02	2E-01	9E-07	3E-05
17	SWANSEA/ELYRIA	0/2	2.0E-03	3E-03	3E-02	2E-07	5E-06
18	SWANSEA/ELYRIA	1/1	8.7E-04	1E-03	1E-02	7E-08	2E-06
19	SWANSEA/ELYRIA	1/3	2.9E-03	4E-03	4E-02	2E-07	8E-06

EPC=Eposure Point Concentration

CTE=Central Tendency Estimate

RME=Reasonable Maximum Exposure

Notes:

Shading indicates that vegetable concentration and resulting risk may exceed protective levels.

(a) Units are mg arsenic per kg wet weight of vegetable. Inorganic arsenic is assumed to be 60% of the total arsenic content.

(b) Outlier excluded

soil samples at this location were also somewhat higher (mean = 51 ppm) than for most other gardens (average = 11 ppm, range = 6 to 23 ppm), suggesting the elevated values in vegetables were likely attributable to the elevated soil levels[6]. One vegetable sample (an onion) from this property was especially high in arsenic (see Figure 2-10, upper panel), possibly because of soil adhering to the sample or because of uptake from soil into the outer layer of the onion. If this one sample is judged to be un-representative of what a resident is likely to ingest (either because of washing and/or peeling before ingestion) and is excluded from the risk calculations, the estimates of noncancer and cancer risks are both reduced, but are still slightly above the usual USEPA level of concern. These results indicate that ingestion of garden vegetable samples from this location could be of potential concern for an RME (but not a typical) consumer, but it is possible these risks are not real, being attributable either to an anomalous analytical result and/or to the extra safety margin introduced by use of the EPC rather than the simple mean.

- At Property 11, the RME cancer risk estimate of 3E-04 is attributable to a single vegetable sample (garlic) that was significantly higher than the remainder of the samples from this location. This caused the 95% UCL of the mean to exceed the maximum value (the garlic sample), so the risk calculation was based on the garlic sample. Because this value seemed to be questionable compared to other samples from the garden, USEPA returned to the property and collected a second sample of garlic. This sample yielded a lower concentration for arsenic (0.2 ppm vs 1.24 ppm dry weight), suggesting the first result may have been anomalous. This is supported by the observation that soil arsenic concentrations at this location are quite low (mean = 12 ppm), and elevated concentrations in vegetables are not expected at such low soil levels. Even if the concentration in the one garlic sample were considered to be reliable, because the average mass of garlic ingested per day is relatively small compared to other vegetable types, risks from garden vegetables at this location are not likely to be of concern.

An alternative approach for evaluating the potential health risks from arsenic in home grown garden vegetables from the site is to compare the average daily intakes of arsenic in site vegetables to intakes that occur in the average United States diet. These data are summarized below:

[6] The property owner was not aware of any additions or treatments of the garden that would account for the moderately elevated arsenic levels in garden soil.

Comparison of Average Daily Intakes of Arsenic in the Average United States Diet and from Vegetables at VBI70 Properties

Parameter	Value (ug/kg-day)	
	Total Arsenic	Inorganic Arsenic
Typical dietary intake of arsenic		
Gunderson 1995	0.36 - 0.81	
Yost et al. 1998	0.75	0.20
Estimated Average Intake at VBI70 Properties		
Property 6 (including outlier)	0.07	0.04
Property 6 (excluding outlier)	0.04	0.02
Property 11	0.02	0.01
All other properties	0.01	0.004

As seen, even at Property 6, predicted mean intake of arsenic from site vegetables is a relatively small fraction of the normal average intake of arsenic from the diet, both for total and inorganic forms. This supports the conclusion that ingestion of home-grown vegetables from the site is not likely to cause doses that are outside the normal dietary range.

Overall, the data and calculations above indicate that ingestion of arsenic in home-grown vegetables is not likely to be a source of significant exposure or risk to most area residents. A limitation to this conclusion is that garden vegetable samples were not obtained from gardens with soil arsenic levels higher than about 90 ppm. As noted above, it appears that arsenic concentrations in garden soils are only weakly correlated with and are substantially lower than arsenic levels in yard soils (see Figure 2-11), even at yard soil concentrations up to 600 ppm. On this basis, it is considered that arsenic levels substantially above 90 ppm are not likely to occur in garden soil, even when yard soils are much higher. However, if vegetables were to be grown in garden (or yard) soil with high arsenic concentrations, then uptake into vegetables might be higher than in the samples evaluated.

4.4.4 Combined Risks from Soil and Home-Grown Vegetables

Residents may be exposed to contaminants in soil both by incidental ingestion of soil and by ingestion of home-grown garden vegetables. Thus, the total risk attributable to contaminants in soil is the sum of these two pathways:

$$\text{Risk(total)} = \text{Risk(soil)} + \text{Risk (vegetables)}$$

Data on arsenic levels in soil are available for all 2,986 properties investigated in Phase III, but data on arsenic levels in vegetables were collected only at 19 of these properties. Therefore, in order to calculate total risk at all properties, the concentration of arsenic in garden vegetables was estimated at each property as described in Section 4.2.6.

Because exposure and risk from soil ingestion and vegetable ingestion are both distributions, care must be taken in the summation process. In the case of the risk to an individual who has average exposure to both soil and vegetables, the total risk is simply the sum of the two pathway specific risks:

CTE(total) = CTE(soil) + CTE(vegetables)

In the case of an individual who has RME exposure to soil or to vegetables, the estimate of RME total risk is not the simple sum of the RME risk estimates, because the two pathways are independent of each other, and an individual with RME soil intake is not likely to also have RME vegetable intake (and vice versa). Thus, the estimate of RME total risk is calculated either as:

1: RME(total) = RME(soil) + CTE(vegetables)
2: RME(total) = CTE(soil) + RME(vegetables)

However, because the RME individual is assumed to have 30 years of exposure to soil, it is also necessary to assume the individual has 30 years of exposure to garden vegetables (rather than 9 years, which is the usual CTE exposure duration). To account for this, the equations above are modified as follows:

1': RME(total) = RME(soil) + (30/9)*CTE(vegetables)
2': RME(total) = (30/9)*CTE(soil) + RME(vegetables)

The results are shown in Table 4-6. As seen, based on the site-specific relationships between arsenic in yard soil and garden soil and between arsenic in garden soil and garden vegetables, individuals with CTE exposure to garden vegetables are predicted to have excess cancer risks that are less than or equal to 1E-05, while individuals that have RME intake of garden vegetables are expected to have risks mainly between 2E-05 and 1E-04, with only a few properties having risks that exceed 1E-04. When CTE risks are combined across pathways, there are 65 properties where total risk exceeds 1E-04. When RME risks are combined across pathways, the highest risks occur for case 1 (RME soil intake plus CTE vegetable intake). Based on this scenario, there are 99 properties where total RME risks exceed 1E-04.

4.5 UNCERTAINTIES IN ARSENIC RISK ASSESSMENT

It is important to recognize that the calculations of short-term and long term exposure and risk from arsenic ingestion in soil are based on a number of assumptions and estimates, and that these introduce uncertainty into the risk results. The most important of the sources of uncertainty in the calculations are summarized below.

Uncertainty in Average Concentration Terms

The concentration term that is appropriate for calculating chronic exposure and risk from ingestion exposure to arsenic is the true mean concentration in the medium of concern (soil, dust,

Table 4-6 Estimated Total Cancer Risks from Soil and Vegetables

Statistic	Pathway	Number of Properties		
		<= 1E-05	2E-05 - 1E-04	2E-04 - 1E-03
CTE Risk	Soil alone	2881	105	
	Vegetables alone	2986		
	CTE Soil + CTE vegetables		2921	65
RME Risk	Soil alone	1475	1419	92
	Vegetables alone		2979	7
	RME Soil + CTE vegetables[a]	933	1954	99
	CTE Soil[a] + RME vegetables		2921	65

[a] Adjusted to account for RME exposure duration (30 years)

vegetables), averaged over the area and time interval (averaging time) of concern. There are two important sources of uncertainty in this value. First, because the true mean cannot be calculated from a limited set of sample results, the USEPA utilizes the 95% upper confidence limit of the mean as a conservative estimate of the true mean. This approach helps ensure that the exposure and risk estimates that are derived are more likely to overestimate than underestimate the actual risk. Second, the basic exposure unit selected for evaluation in this risk assessment is the residential property. Using the UCL of the mean for a property is equal to assuming that an individual residing at that location does not ingest soil or dust from any other location, even over a time period of up to 30 years. While this might be true for a small sub-set of residents, it is believed that most residents are sufficiently mobile that exposures will occur over a wider area than just their own yard. This, in turn will result in lower exposures for people residing in homes with affected soils, and their true risks will be lower than calculated.

Uncertainty in Concentration Values for Sublocations

As noted earlier, the sampling and analysis design for Phase III was based on a set of three composite samples from each property. Consequently, there are no data that allow a direct estimation of the concentration value at any specific sub-location of the yard (these are needed to address risks from subchronic and acute exposures). To address this data limitation, the distribution of concentration values within a property was modeled by assuming a lognormal distribution, and the standard deviation within each property was estimated from the mean value by multiplying by a site-specific average coefficient of variation of 1.02. This approach should be considered to yield only approximate values, but since the mean at each property was estimated using the 95% UCL or the maximum composite value, both the mean and the standard deviation are more likely to be high than low at each property. Thus, the values estimated for evaluation of subchronic and acute exposures are also more likely to be high than low.

This expectation is supported by a comparison of the estimated and actual sub-location concentrations at the eight intensively samples properties from the Risk-Based sampling program. This comparison was performed as follows. First, at each of these eight properties the yard was divided into 16-20 sub-areas, and the mean concentration in each sub-area was calculated based on the values of the grab samples that fell within the sub-area (typically 5-20). Second, the mean values for these 16-20 sub-areas were rank ordered and used to estimate the mean at the 90th and 95th percentile sub-area. Third, the 90th and 95th percentile values of the underlying distribution of grab samples was calculated using the estimation method described above. In order to complete step 3, it was necessary to estimate what the EPC at these properties would have been if only three composites had been collected instead of a large number of grab samples. Based on the Phase III data, the typical ratio of the EPC to the mean is about 1.4. Thus, an EPC value equal to 1.4-times the mean was assumed for each of the eight properties. Based on this, the calculated values of the 90th and 95th percentile values of the underlying distribution of grab samples were, on average, about 2-times higher than the 90th and 95th percentile values for subarea means. These results support the conclusion that the method used to estimate EPC values for the subchronic and acute risk calculations is conservative (more likely to be high than low).

Uncertainty in Intake Rates

Data on the amount of soil ingested by humans are very limited. Measurements are difficult to perform, and results vary significantly from study to study and from method to method. In addition, data are based mainly on short term studies, so estimates of long-term average intake rates are especially uncertain. Moreover, intake rates are likely to vary from site to site and property to property, depending on things such as climate, socioeconomic status, yard condition, etc., so the default intake rates used in these calculations may not reflect the true intake rates at the site. Because of the limitations in the data, the default values recommended by USEPA are intended to be on the high side (i.e., are more likely to overestimate than underestimate actual soil ingestion).

This is illustrated by comparing the default soil intake rates used by USEPA to data on soil intake rates measured in a group of 64 children in Anaconda, Montana (Stanek and Calabrese 2000). This study, which utilizes the latest and most refined analytical and statistical methods for estimating soil ingestion by children, estimated that the average (CTE) 7-day intake by children is about 31 mg/day (compared to the default of 100 mg/day), and that the 95th percentile intake for 7 days and 365 days are 133 and 106 mg/day, respectively (compared to the default assumption of 200 mg/day). If these values from the Anaconda site were judged to be a more reliable basis for estimation of risk from soil ingestion than the current default values, and if adult soil intake is assumed to be about ½ that of children, then there are only 23 properties (rather than 92 properties) in the VBI70 site where RME cancer risks from soil ingestion exceed a level of 1E-04.

Uncertainty in the Fraction of Total Intake that is Soil

One of the variables used to calculate risks from ingestion of soil plus dust is the fraction of the total intake that is soil (f_s). When concentrations of a contaminant in dust are similar to the concentration in yard soil, the exact value of f_s has very little impact on the calculated risks. However, at this site, concentrations of arsenic in dust are substantially lower than soil, so the value for f_s is important (the larger the value, the higher the risk). The EPA default value for this variable (45%) is based mainly on measurements in a set of 64 preschool children (Stanek and Calabrese 1992). However, due to the difficulty in making these measurements, as well as potential differences between children and between sites, this value should be considered to be uncertain. It is not known whether the true value at the VBI70 site is more likely to be higher or lower than the default values. If the true site-specific value of f_s were lower (e.g., 20% rather than 45%), risks would be about 12% lower than calculated. Conversely, if the true site-specific value were higher (e.g., 70% rather than 45%), then the risks would be about 12% higher than calculated.

Uncertainty in Exposure Duration

Cancer risk calculations depend on the duration of exposure. Default exposure durations used in the risk assessment are not site-specific, and are estimated from data on the length of time that

people own a particular residence. Thus, actual exposure durations of residents at the site may not be the same as the assumed exposure durations assumed, and might be either longer or shorter than assumed. For example, preliminary data collected at the Globe site suggest that 10-15% of the residents have lived in their homes for more than 30 years (Mitchell 2001a). Likewise, analysis of available demographic data by an contractor for ATSDR indicates that about 13-20% of the residents in the VBI70 area may have resided in their home for more than 30 years (Claritas 2000). These data suggest that an assumed 95th percentile exposure duration of 30 years may be somewhat too low. However, the data from the Globe site (Mitchell 2001a) suggest that of the residents who have lived at the site for more than 30 years, only a fraction resided at the home as a child (when exposure rates are assumed to be highest).

If the RME exposure duration were assumed to be 45 years (6 years as a child and 39 years as an adult) rather than the default value of 30 years, the estimated excess cancer risk level from soil ingestion would be about 19% higher than the values reported. In addition, all of the exposure calculations presented here assume that exposure begins during childhood, when intake rates are higher than during adulthood. Thus, risks to individuals who move to the site after they are children will be lower than estimated. For example, risks to an individual exposed for 30 years as an adult are only 37% of the risks to an individual exposed for 6 years as a child and 24 years as an adult.

Uncertainty in RME Exposures

In the default point estimate approach for estimating exposure and risk to an RME individual, two exposure parameters (intake rate and exposure duration) are both assumed to be at their 95th percentile values. In reality, because these two exposure parameters are independent of each other, it is very unlikely that an individual with RME soil intake will also have RME exposure duration. Therefore, an individual with both RME soil intake and RME exposure duration represents not the 95th percentile of the risk distribution, but some significantly higher percentile. One way to estimate what the percentile of the default RME individual is, as well as the actual 95th percentile value, is through Monte Carlo modeling. These calculations (described in detail in Appendix D) characterize the variability in risk to different individuals in a hypothetical population of people exposed at a specified exposure location. For an arbitrary exposure point concentration of 200 ppm arsenic in fine soil (165 ppm in bulk soil), the results of the point estimate calculation and the Monte Carlo calculations are as shown below:

Cancer Risk Estimates for 200 ppm Arsenic in Fine Soil

Method	Statistic	Soil Alone	Vegetables Alone	Total Risk
Point Estimate	RME cancer risk	1E-04	7E-05	1E-04
Monte Carlo (a) (see Appendix D)	90th percentile	1E-05 to 4E-05	9E-06	2E-05 to 5E-05
	95th percentile	2E-05 to 6E-05	1E-05	3E-05 to 7E-05
	99th percentile	5E-05 to 1E-04	3E-05	6E-05 to 1E-04
	99.9th percentile	1E-04 to 2E-04	8E-05	1E-04 to 2E-04

(a) Range is based on two alternative PDFs for soil intake rate (see Appendix D)

As seen, the RME risk estimate derived by the point estimate approach is about twice the Monte Carlo estimate of the 95th percentile value, and is located at or above the 99th percentile of the risk distribution. This supports the conclusion that the RME point estimate of risk is conservative and will provide protection to nearly all individuals in the exposed population.

Uncertainty in Toxicity Factors

One of the largest sources of uncertainty in most risk assessments stems from uncertainty in the toxicity factors used to predict responses from the calculated doses. In the case of arsenic, dose-response data are derived from studies in humans, which significantly reduces the degree of uncertainty compared to extrapolations based on animal data. However, a significant degree of uncertainty still remains in both the slope factor and the chronic RfD. One of the most important sources of this uncertainty is lack of reliable data on actual arsenic ingestion rates by the Taiwanese population used to quantify risk. For example, dose-response curves in the key studies are based on village-based estimates of the concentration of arsenic in well water, rather than individual specific intake rates (USEPA 2001d). This type of approach, referred to as an ecological study, is well-known to have a number of limitations, and might either overestimate or underestimate the true dose-response relationship. In addition, exposures to arsenic through the diet are believed to be significant, but the magnitude of this contribution can only be estimated. There are also still large uncertainties in how to extrapolate the dose-response curve from relatively high exposure levels to lower exposure levels. For example, arsenic does not appear to cause cancer by a direct genotoxic mechanism (USEPA 2001d), suggesting that a sub-linear (and perhaps even a threshold) model might be reasonable. However, in the absence of information on the actual mode of action, an assumption of linearity is still deemed to be necessary and appropriate (USEPA 2001d). If the dose response curve is sub-linear, current risk estimates would be too high. Further, there is uncertainty in the importance of cultural and ethnic differences between different study populations. These differences could include factors such as inherent differences in the level and capacity of liver enzymes to methylate (and hence detoxify) ingested inorganic arsenic. Likewise, because methylation requires an adequate supply of the methyl group (usually derived from dietary methionine), it is plausible that people with poor diets (especially diets that are low in methionine) might have decreased ability to methylate arsenic. Differences in diet might also influence the relative amount of arsenic that is absorbed

from the gastrointestinal tract into the blood. While little is known about the relative importance of these factors, it is likely that there are differences between people in their sensitivity to ingested arsenic, and it is for this reason that USEPA seeks to ensure an adequate margin of safety in the derivation of the RfD and the slope factor.

Uncertainty in Bioavailability

In order to cause an adverse response, arsenic that is ingested must be absorbed into the body. As detailed in USEPA (2001b), measurements of the arsenic relative bioavailability have been performed for five soils from the VBI70 site. While measurements based on site soils significantly reduces uncertainty in this exposure parameter, uncertainty still remains. For example, variability was observed between different site soils, and a conservative estimate of the mean value was employed to represent the site-wide average absorption. This approach is expected to result in an over-estimate of true absorption. Another source of uncertainty is in the extrapolation of data from test animals to humans. The test animals (swine) were selected because they are believed to have a gastrointestinal system similar to that in humans, but it is also possible that absorption in humans might vary as a function of age, stomach contents, nutritional status, etc. Thus, the RBA value measured in the site-specific study should be viewed as an approximation of the true RBA value in humans.

The RBA measured for soil was also assumed to apply to dust. This assumption is uncertain because the size distribution of arsenic-containing particles in dust may be different than for soil, and particle size might be one factor that influences RBA. If dust contains smaller particles than soil, and if this size difference tends to increase RBA, then the use of the soil RBA could underestimate the absorption of arsenic from dust. However, it should be remembered that the RBA value for soil was measured using only the fine fraction of soil (only particles smaller than 250 micrometers in diameter), so the difference in particle size distribution between dust and soil is not expected to be large. In addition, because arsenic concentrations in dust tend to be lower than in soil, the dose contributed by dust ingestion is relatively small compared to that for soil, so uncertainty in the absorption fraction for dust results in only a small uncertainty in the total absorbed dose.

Uncertainty Due to Potential Chemical Interactions

All of the risk calculations presented in Section 4 predict the health effects of arsenic acting alone. However, most people are exposed to many different chemicals in air, water, soil and the diet, and the possibility exists that some of these chemicals might either increase or decrease the toxicity of ingested arsenic. Very few data are available on toxicokinetic or toxicologic interactions of arsenic with other chemicals, although some epidemiological studies suggest that lead and arsenic might both be associated with behavioral deficits (Moon et al. 1985). This lack of detailed knowledge on chemical interactions is a general source of uncertainty, but it is not considered likely that risk is significantly underestimated as a result of any such (hypothetical) interactions.

Uncertainty in Risks Combined Across Exposure Pathways

When exposure of an individual occurs by more than one exposure pathway (e.g., ingestion of soil and ingestion of home-grown produce), the total exposure and risk is given by the sum across the pathways. However, calculation of this sum is difficult in the standard point estimate approach, especially for the RME individual, and the value must be estimated by assuming CTE intake of one pathway plus RME intake of the other pathway. Appendix D presents an evaluation of total risks calculated using Monte Carlo modeling which demonstrates that the point estimate of RME risk that is calculated by summing the RME soil risk with the CTE vegetable risk will result in a risk estimate that exceeds the 95th percentile of the combined Monte Carlo distribution. This demonstrates that the RME point estimate is likely to be conservative and will protect more than 99% of the exposed population.

Uncertainty in Pica Exposure and Risks

As noted earlier, screening-level calculations suggest that acute high-dose exposures to arsenic in soil (i.e., pica exposure) might be of concern at a number of properties within the site (see Section 4.4.2). However, data on the amount of soil ingested during pica behavior are very sparse. Based mainly on one study that observed an intake of 5-8 g/day by a single child (Calabrese et al. 1989), USEPA has indicated that 5-10 grams might be a reasonable estimate. If this intake rate is correct, and if arsenic absorption from this mass of soil is similar to that estimated in site-specific studies (42%), then anywhere from 22% to 62% of all properties within the VBI70 site (and perhaps outside the site as well) could have arsenic levels above a level of acute concern. USEPA feels this conclusion is especially uncertain, since the Agency is not aware of any reported cases of acute arsenic toxicity attributable to ingestion of arsenic in soil. The most recent study of soil intake by children (Stanek and Calabrese 2000) did not observe intake rates above 700 mg/day in a group of 64 children, suggesting that values of 5-10 grams might be unrealistically high. In addition, limited data on urinary arsenic levels in residents of the VBI70 area and the nearby Globe neighborhood do not reveal the occurrence of high soil intakes by children (Mitchell 2001b). For example, two children from the VBI70 area who were exposed to high soil arsenic levels (above 400 ppm) both had urinary arsenic levels below the limit of detection (see Table 2-1 and Table 2-7). In the Globe area, 7 out of 62 children exposed to soil arsenic concentrations of 5-200 ppm had urinary arsenic levels that were above the detection limit, but the maximum concentration value was only about 15 ug/L. This concentration may be contrasted to a value of 100-1000 ug/L which is what would be expected to occur in a child who ingested 5,000 to 10,000 mg of soil at a location that contained 50 mg/kg arsenic in soil.

These considerations suggest that arsenic risk from soil pica may not be as significant as the calculations suggest. On the other hand, if this type of exposure were to occur, it is possible the symptoms (transient upset stomach and general malaise) would not be recognized as being arsenic-related, and could easily go un-detected or un-reported. In addition, if pica behavior is assumed to occur only infrequently during childhood (e.g., 1 day out of 500-1000), then the chances of observing the behavior in a study of only a few hundred children could be quite low. That is, it is possible that exposure to arsenic via pica ingestion of soil might be occurring in the

children evaluated in these studies, but that the exposure was not detected because it is an infrequent event. Because of the high uncertainty regarding the magnitude and frequency of soil pica behavior, more reliable risk estimates for this scenario will not be possible until better data are collected on pica intakes, along with direct measures of soil-related exposures to arsenic in soil.

Summary

Because of the uncertainties summarized above, none of the exposure and risk calculations for arsenic presented above should be interpreted as accurate measures of the true risk. Rather, all values should be interpreted as uncertain estimates. Because most of the approaches for dealing with uncertainty are intended to be conservative (i.e., are more likely to overestimate than underestimate), the risk values above should generally be thought of as high-end estimates of the true risk, and actual risks are more likely to be lower than the calculated values.

SECTION 5
EXPOSURE AND RISK FROM LEAD

5.1 OVERVIEW

As noted earlier, risks from lead are evaluated using a somewhat different approach than for most other metals. First, because lead is wide-spread in the environment, exposure can occur by many different pathways. Thus, lead risks are usually based on consideration of total exposure (all pathways) rather than just to site-related exposures. Second, because studies of lead exposures and resultant health effects in humans have traditionally been described in terms of blood lead level[7], lead exposures and risks are typically assessed using an uptake-biokinetic model rather than calculating an estimated dose and comparing that dose to an appropriate reference dose (RfD). Therefore, calculating the level of exposure and risk from lead in soil also requires assumptions about the level of lead in other media, and also requires use of pharmacokinetic parameters and assumptions that are not needed in traditional methods.

For residential land use, the sub-population of chief concern is young children. This is because young children 1) tend to have higher exposures to lead in soil, dust and paint, 2) tend to have a higher absorption fraction for ingested lead, and 3) are more sensitive to the toxic effects of lead than are older children or adults.

It is currently difficult to identify what degree of lead exposure, if any, can be considered safe in young children. Some studies report subtle signs of lead-induced neurobehavioral effects in children beginning at blood lead levels around 10 ug/dL or even lower, with population effects becoming clearer and more definite in the range of 30-40 ug/dL (CDC 1991, ATSDR 1999). On the other hand, some researchers and clinicians believe the effects that occur in children at low blood lead levels are so minor that they need not be cause for concern. After a thorough review of all the data, the USEPA has identified 10 ug/dL as the blood lead level at which effects that warrant avoidance begin to occur, and has set as a goal that there should be no more than a 5% chance that any child will have a blood lead value above 10 ug/dL (USEPA 1994a, 1994b). This approach focuses on the risks to a child at the upper bound (about the 95th percentile) of the exposure distribution, very much the same way that the approach used for other chemicals focuses on risks to the RME individual. The Centers for Disease Control (CDC) has also established a guideline of 10 ug/dL in preschool children which is believed to prevent or minimize lead-associated cognitive deficits (CDC 1991).

5.2 IEUBK MODEL FOR ASSESSING LEAD RISK

The USEPA has developed an Integrated Exposure Uptake Biokinetic (IEUBK) model for predicting the likely range of blood lead levels in a population of young children (age 0-6 years)

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[7] The concentration of lead in the blood is usually abbreviated "PbB", and is expressed in units of micrograms of lead per deciliter of blood (ug/dL). One dL is equal to 100 mL.

exposed to a specified set of environmental lead levels (USEPA 1994b). This model requires as input data on the levels of lead in soil, dust, water, air, and diet at a particular location, and on the amount of these media ingested or inhaled by a child living at that location. All of these inputs to the IEUBK model are central tendency point estimates. These point estimates are used to calculate an estimate of the central tendency (the geometric mean) of the distribution of blood lead values that might occur in a population of children exposed to the specified conditions. Assuming the distribution is lognormal, and given (as input) an estimate of the variability between different children (this is specified by the geometric standard deviation or GSD), the model calculates the expected distribution of blood lead values, and estimates the probability that any random child might have a blood lead value over 10 ug/dL.

If all of the IEUBK model exposure levels and intake rates are set at their default values, and if the concentration of lead in dust is assumed to be 70% of that in soil (the default assumption), then the IEUBK model predicts that a soil lead level of about 350 ppm corresponds to the target risk level (no more than a 5% chance of exceeding a blood lead level of 10 ug/dL) for children age 0-84 months. If default estimates of dietary intake are adjusted downwards by a factor of 0.7 to partially account for the lower lead levels in the current food supply (Bolger et al. 1996, Gunderson et al. 1995, Griffin et al. 1999) than are assumed in the default IEUBK model, then the soil lead level that corresponds to the target risk level is about 400 ppm. Based in part on these results, USEPA has established a national policy that soil lead levels below 400 ppm may be assumed to be below a level of health concern (USEPA 1994a). Soil lead levels above 400 ppm may or may not be of concern, depending on site-specific factors. At 400 ppm in soil, the IEUBK model predicts that exposure from soil (including ingestion of both soil and dust) accounts for 73-78% of the total absorbed dose of lead, with even larger relative contributions at higher soil lead levels. Of the non-soil exposure, food is about three times larger than water, and intake from air is negligible.

Whenever reliable site-specific data are available on any of the IEUBK model input parameters, these are used in preference to the assumptions employed in the default case. At this site, three types of site-specific data are available, as follows:

Adjustment for Lead Enrichment in the Fine Fraction

As discussed in Section 2.4.1, it is suspected that exposure to soil occurs mainly via ingestion of the fine fraction. Since Phase III data on the concentration of lead in soil are based on the concentration in bulk soil, the mean concentration in bulk soil at each property was adjusted to account for the enrichment of lead in the fine fraction, as follows (see Section 2.6.1):

$$C_s(\text{fine}) = 1.09 \cdot C_s(\text{bulk})$$

Soil-Dust Relationship

The site-specific relationship between lead in bulk yard soil and lead in indoor dust was presented earlier in Figure 2-9. As shown in this figure, the average relationship is described by an equation of the form:

$$C_d = 0.34 \cdot C_s(\text{bulk}) + 150$$

Lead Bioavailability

In order to investigate the relative bioavailability of lead in site soils, USEPA Region VIII performed a study in which two separate samples of site soil were fed to swine for 15 days. Swine were selected as the test species because it is believed the gastrointestinal system (and hence the behavior of ingested lead) in swine is similar to that in humans. The details of the study design and of the findings are presented in a separate report (USEPA 2001c). In brief, the study found that lead in site soils was absorbed by swine about 81-87% (mean = 84%) as well as a readily soluble form of lead (lead acetate). This *in vivo* estimate is supported by the bioaccessability measured *in vitro*:

In Vivo Bioavailability and In Vitro Bioaccessability Measurements of VBI70 Site Soils

Test Material	Sample Location	In Vivo Relative Bioavailability (%)	In Vitro Bioaccessability (%)
Sample 1	Eastern part of site	87%	86%
Sample 2	Western part of site	81%	85%*

* Mean of duplicate analyses

This RBA value is somewhat higher than the typical USEPA default value of 60%, suggesting that the lead in site soils is in a form that can be readily absorbed. Based on this site-specific finding, an RBA of 0.84 was used in the evaluation of lead risks. Based on a default absorption fraction of 50% for lead in water and food, this RBA corresponds to an absolute bioavailability (ABA) of 42% (0.42).

These adjustments to the model, along with the other model inputs, are summarized in Table 5-1. This site-specific adjusted model was used to evaluate risks to children from lead in soil and dust, as described below.

5.3 RISK CHARACTERIZATION FOR LEAD

5.3.1 Risks from Lead in Soil and Dust

The expected blood lead distribution for children (age 0-84 months) was calculated for each property using IEUBKwin v1.0 (build 241). The soil value at each property was the estimated concentration in fine soil (1.09 times the mean bulk concentration), and the dust lead concentration was predicted using the equation above. The results, characterized in terms of the probability of a random child exceeding a blood lead value of 10 ug/dL (this is referred to as "P10"), are shown in Table 5-2.

As seen, a total of 1,655 out of 2,986 homes are predicted to have P10 values at or below the health-based goal of 5%, while 1,331 (45%) are predicted to exceed the health-based goal. Approximately 610 properties are predicted to have P10 values of 5-10%, slightly above the

Table 5-1 IEUBK Model Inputs
SOIL/DUST INPUTS

Csoil = 1.09 · Bulk property-specific average (ppm) (a)
Cdust = 0.34 · Csoil + 150 (ppm) (a)

CONSTANTS

PARAMETER	VALUE
Air concentration (ug/m3)	0.10
Indoor air concentration	30% of outdoors
Drinking water concentration (ug/L)	4.0
Absorption Fractions: Air Diet Water Soil/Dust (a)	 32% 50% 50% 42%
Fraction soil	45%
GSD	1.6

AGE DEPENDENT

Age	AIR		DIET	WATER	SOIL
	Time Outdoors (hrs)	Vent. Rate (m3/day)	Dietary intake (ug/day)	Intake (L/day)	Intake (mg/day)
0-1	1.0	2.0	3.87	0.20	85
1-2	2.0	3.0	4.05	0.50	135
2-3	3.0	5.0	4.54	0.52	135
3-4	4.0	5.0	4.37	0.53	135
4-5	4.0	5.0	4.21	0.55	100
5-6	4.0	7.0	4.44	0.58	90
6-7	4.0	7.0	4.90	0.59	85

(a) Values based on site-specific data

Table 5-2 Estimated Risks to Children from Lead in Soil and Dust

Neighborhood	Total Number of Properties	Number and Percent of Properties Within Specified Risk Range				
		P10 <= 5%	P10 > 5% and <= 10%	P10 > 10% and <= 20%	P10 > 20%	Total P10>5%
Clayton	902	712	119	52	19	190
	100%	79%	13%	6%	2%	21%
Cole	796	169	248	273	106	627
	100%	21%	31%	34%	13%	79%
Elyria	59	6	9	28	16	53
	100%	10%	15%	47%	27%	90%
Globeville	63	7	18	21	17	56
	100%	11%	29%	33%	27%	89%
Swansea	1166	761	216	144	45	405
	100%	65%	19%	12%	4%	35%
All	2986	1655	610	518	203	1331
	100%	55%	20%	17%	7%	45%

P10=Prediced Risk of Exceeding Blood Lead of 10 ug/dL

heath-based goal. However, about 518 properties would be expected to have P10 values between 10-20%, and 203 homes are predicted to have P10 values greater than 20% (substantially above the health-based goal). It should be noted that 1,057 of the 1,331 properties (79%) with P10 values above 5% have mean bulk lead concentrations lower than 400 ppm (the USEPA default level of concern). This is mainly because the site-specific RBA for lead (84%) is higher than the default value (60%), and also because of the use of the concentration value in the fine fraction rather than the bulk fraction in the risk calculations.

Although homes with elevated soil lead are found in all neighborhoods, the density of homes with P10 values greater than 5% tends to be higher in the central and western part of the site than in areas on the eastern side of the site. This is illustrated the following table:

Count of Properties

Location	Total Number	Number with P10 > 5%	%
Western (a)	918	736	80%
Eastern (b)	2068	595	29%

(a) Western = Cole, Elyria, Globeville
(b) Eastern = Clayton and Swansea

In interpreting these risk estimates, it is important to recognize that lead is a naturally occurring element in soil, and that there are many current and historic anthropogenic sources of lead (e.g., automobile exhaust, leaded paint, generalized industrial emissions, etc.). As noted earlier (see Figure 2-7), levels of lead in bulk soils at the VBI70 site range from below the detection limit (about 52 ppm) up to a maximum of more than 1,000 ppm. In contrast to the situation that was found for arsenic (see Figure 4-3), analysis of this distribution does not reveal the presence of two distinct components, so the boundary between the values that are "background" (including both natural and area-wide anthropogenic sources) and those that are elevated due to site-specific sources is difficult to judge. If it is assumed that the upper range of the typical urban background levels is about 400 ppm, then the mean of all samples that are less than 400 ppm is about 195 ppm. Using this value (195 ppm in bulk soil) as a rough estimate of the mean concentration in urban background samples, and assuming the same site-specific input values as shown in Table 5-1, the IEUBK model predicts that blood lead levels attributable to urban background levels of lead probably average about 4.4 ug/dL for a typical (median) child, and might be as high as 9.5 ug/dL for a child with above-average (95th percentile) exposure to soil or dust.

5.3.2 Risks from Lead in Garden Vegetables

As shown previously (see Figure 2-10), there is essentially no uptake of lead from soil into garden vegetables at this site. On this basis, it is concluded that exposure to lead from ingestion of home grown garden vegetables is not of concern.

5.4 UNCERTAINTIES IN LEAD RISK EVALUATION

It is important to stress that lead risk predictions based on the IEUBK model are uncertain. This uncertainty arises from a number of factors. First, there is inherent difficulty in providing the model with reliable estimates of human exposure to lead-contaminated media. For example, exposure to soil and dust is difficult to quantify because human intake of these media is likely to be highly variable, and it is very difficult to derive accurate measurements of actual intake rates. Likewise, site-specific data on exposure to lead through the diet is generally not available, and because dietary lead levels have been decreasing over time, the default data used in the model may no longer be accurate. Second, it is often difficult to obtain reliable estimates of key pharmacokinetic parameters in humans (e.g., absorption fraction, distribution and clearance rates, etc.), since direct observations in humans are limited. Finally, the absorption, distribution and clearance of lead in the human body is an extremely complicated process, and any mathematical model intended to simulate the actual processes is likely to be an over-simplification. Consequently, IEUBK model calculations and predictions should not be thought of as being identical to actual risk.

Alternative IEUBK Model Runs

In order to investigate some of these sources of uncertainty in the IEUBK model predictions, a series of three alternative IEUBK model runs were performed using several alternative model input values, including the following:

a) Dietary lead intake values based on the latest market-basket study by the FDA (Bolger et al. 1996, Gunderson et al. 1995, Griffin et al. 1999). These values are listed below:

FDA Dietary Lead Intake Values

Age	Dietary Intake (ug/d)
6-11 months	1.82
1 year	1.90
2 years	1.87
3 years	1.80
4 years	1.73
5 years	1.83
6 years	2.02

b) A series of alternative GSD values ranging from 1.2 to 1.5. The GSD is the most sensitive input parameter in the IEUBK model, and a small change in the GSD can result in a large change in the calculated P10 value. As discussed below, there is some reason to think that the default GSD value of 1.6 used by the IEUBK model might be somewhat

too high, so these runs were performed to investigate how the results would change if the GSD were indeed smaller than the default.

c) A mean soil intake value based on the soil intake study by Stanek and Calabrese (2000). In this study the estimate of the long-term average soil intake rate was 31 mg/day. Age-specific intake values were estimated by multiplying the mean value (31 mg/day) by the ratios of the IEUBK age-specific intake rates (see Table 5-1) compared to the IEUBK age-averaged intake rate (109 mg/day).

The results of these alternative IEUBK model runs are shown below:

Uncertainty Analysis Results for Alternative IEUBK Model Inputs

Model Run (a)	P 10 Value (%)				Total with P10>5%
	< 5%	5-10%	10-20%	> 20%	
Default (see Table 5-2)	1655	610	518	203	1331
Revised dietary intakes (see above)	1937	507	402	140	1049
GSD = 1.5	2058	450	345	133	928
GSD = 1.4	2413	315	171	87	573
Revised dietary intakes (see above) and GSD=1.4	2572	229	118	67	414
GSD = 1.3	2728	134	67	57	258
Revised dietary intakes (see above) and GSD=1.3	2801	91	59	35	185
GSD = 1.2 (b)	2911	37	19	19	75
Revised dietary intakes (see above) and GSD=1.2 (b)	2931	30	12	13	55
Soil intake based on Stanek and Calabrese (2000)	2986	0	0	0	0

(a) All runs include site-specific adjustments for lead enrichment in the fine fraction (1.09), RBA (0.84), and for soil-dust relationships.
(b) Calculations performed using the DOS version (0.99d) of the IEUBK model

These calculations help illustrate the range of potential uncertainty in risk estimates for lead that may be associated with uncertainty in the IEUBK model inputs, especially the dietary intake of lead, the soil/dust intake rate, and the GSD.

ISE Model Predictions

Another approach for assessing hazard from lead in soil is currently under development by USEPA Region VIII. This approach, referred to as the Integrated Stochastic Exposure (ISE) Model for Lead, uses the same basic equations and algorithms for calculating exposure and

blood lead values as the IEUBK model, except that it uses probability distribution functions (PDFs) rather than point estimates as inputs for a number of exposure parameters. These distributions are combined using Monte Carlo simulation techniques to yield a predicted distribution of absorbed lead doses (ug/day) for different members of the exposed population. These doses are then used as input to the biokinetic portion of the IEUBK model in order to generate the predicted distribution of blood lead values in the population. Thus, the variability between children is evaluated in the ISE model based on the variability in environmental and exposure parameters, rather than by application of an assumed or estimated GSD value as in the IEUBK model. Because this model has not yet undergone peer review or validation, it is considered to be only an investigative tool.

The input distributions used in the ISE model runs are summarized in Table 5-3. The distribution for soil ingestion is based on reliable data and a well-characterized empirical distribution function (EDF) reported by Stanek and Calabrese (1995). The mean soil intake value assumed by the IEUBK model (about 109 mg/day) is located between the 75th and 80th percentile of the EDF reported by Stanek and Calabrese (1995). Variability in the RBA term is based on the observed inter-individual variability in response in the animal study used to develop the RBA. In this study, the mean coefficient of variation (standard deviation divided by the mean response) across dose groups was about 0.2. based on the logic that variability is likely to be higher in a group of children than in a group of test animals, a coefficient of variation of 0.3 was assumed. Thus, given a mean RBA of 84% and a mean absolute absorption fraction of 42%, the standard deviation was assumed to be 12.6%. The basis of the other distributions is provided in Goodrum et al. (1996). It is important to note that these other distributions are screening-level only. In most cases a distribution is assumed to be lognormal, even though the true shape is not known. Likewise, the mean value of the distribution is selected to match the mean value used by the IEUBK model, but the estimate of the standard deviation is often an estimate based mainly on professional judgement.

The results of a risk evaluation based on the ISE model compared to the predictions of the IEUBK model are presented below:

Comparison of ISE and IEUBK Model Predictions

Model	P10 Value (%)				Total with P10>5
	< 5%	5-10%	10-20%	> 20%	
IEUBK Model	1655	610	518	203	1331
ISE Model	2986	0	0	0	0

Table 5-3 ISE Model Inputs

AT/EF:
=====

Exposure Frequency	Point	365.00	days/yr
Averaging Time	Point	365.00	days/yr

SOIL:
====

C_soil (soil Pb conc)	Point	600	ug Pb/g
IRsd (soil+dust IR)	PDF-Cumulative	----------	mg/day
Number: 8 Min: 0 Max: 7000			
Values: {0,10,45,88,186,208,225,7000}			
Percen: {0,0.25,0.5,0.75,0.9,0.95,0.99,1}			
Age: 0-1 IR scale factor	Point	0.6296	
Age: 1-2 IR scale factor	Point	1	
Age: 2-3 IR scale factor	Point	1	
Age: 3-4 IR scale factor	Point	1	
Age: 4-5 IR scale factor	Point	0.7407	
Age: 5-6 IR scale factor	Point	0.6666	
Age: 6-7 IR scale factor	Point	0.6296	
Fs (frac ingest as soil)	PDF-Triangular	(0.1,0.45,0.8)	

DUST:
====

C_dust (dust Pb conc)	PDF-Log Normal	(315, 307)	ug Pb/g soil
Regression Variable A	PDF-Log Normal	(150, 50)	C_dust=A+B*C_soil
Regression Variable B	PDF-Log Normal	(0.34, 0.2)	C_dust=A+B*C_soil

WATER:
=====

C_water (water Pb Conc)	PDF-Log Normal	(4, 3)	ug Pb/L
Age: 0-1 IR Water	PDF-Log Normal	(0.2, 0.2)	L/day
Age: 1-2 IR Water	PDF-Log Normal	(0.5, 0.4)	L/day
Age: 2-3 IR Water	PDF-Log Normal	(0.52, 0.4)	L/day
Age: 3-4 IR Water	PDF-Log Normal	(0.53, 0.4)	L/day
Age: 4-5 IR Water	PDF-Log Normal	(0.55, 0.4)	L/day
Age: 5-6 IR Water	PDF-Log Normal	(0.58, 0.4)	L/day
Age: 6-7 IR Water	PDF-Log Normal	(0.59, 0.4)	L/day

Table 5-3 (Continued)

DIET:
====

Age: 0-1 Diet Intake	PDF-Log Normal	(3.87, 2)	ug Pb/day
Age: 1-2 Diet Intake	PDF-Log Normal	(4.05, 2)	ug Pb/day
Age: 2-3 Diet Intake	PDF-Log Normal	(4.54, 2)	ug Pb/day
Age: 3-4 Diet Intake	PDF-Log Normal	(4.37, 2)	ug Pb/day
Age: 4-5 Diet Intake	PDF-Log Normal	(4.21, 2)	ug Pb/day
Age: 5-6 Diet Intake	PDF-Log Normal	(4.44, 2)	ug Pb/day
Age: 6-7 Diet Intake	PDF-Log Normal	(4.9, 2)	ug Pb/day

OTHER:
=====

Age: 0-1 Other Intake	Point	0	ug Pb/day
Age: 1-2 Other Intake	Point	0	ug Pb/day
Age: 2-3 Other Intake	Point	0	ug Pb/day
Age: 3-4 Other Intake	Point	0	ug Pb/day
Age: 4-5 Other Intake	Point	0	ug Pb/day
Age: 5-6 Other Intake	Point	0	ug Pb/day
Age: 6-7 Other Intake	Point	0	ug Pb/day

ABSORPTION:
==========

Soil: % accessible	PDF-Log Normal	(42,12.6,100,10)	percent
Dust: % accessible	PDF-Log Normal	(42,12.6,100,10)	percent
Water: % accessible	PDF-Log Normal	(50, 20)	percent
Diet: % accessible	PDF-Log Normal	(50, 20)	percent
Other: % accessible	Point	30	percent
Passive Fraction	Point	0.2	
Half Saturation Level	Point	100	ug/day

AIR:
===

Air Pb Conc Outdoors	PDF-Log Normal	(0.1, 0.05)	ug Pb/m3 air
Age: 0-1 Ventilation Rate	PDF-Log Normal	(2, 1.2)	m3 air/day
Age: 1-2 Ventilation Rate	PDF-Log Normal	(3, 1.4)	m3 air/day
Age: 2-3 Ventilation Rate	PDF-Log Normal	(5, 2.4)	m3 air/day
Age: 3-4 Ventilation Rate	PDF-Log Normal	(5, 2.4)	m3 air/day
Age: 4-5 Ventilation Rate	PDF-Log Normal	(5, 2.4)	m3 air/day
Age: 5-6 Ventilation Rate	PDF-Log Normal	(7, 3.4)	m3 air/day

Table 5-3 (Continued)

Age: 6-7 Ventilation Rate	PDF-Log Normal	(7, 3.4)	m3 air/day
Indoor Conc (% of Outdoor)	Point	30	percent
Age: 0-1 Time Outdoors	Point	1	hr/day
Age: 1-2 Time Outdoors	Point	2	hr/day
Age: 2-3 Time Outdoors	Point	3	hr/day
Age: 3-4 Time Outdoors	Point	4	hr/day
Age: 4-5 Time Outdoors	Point	4	hr/day
Age: 5-6 Time Outdoors	Point	4	hr/day
Age: 6-7 Time Outdoors	Point	4	hr/day
Lung Absorption Age 0-1	Point	32	percent
Lung Absorption Age 1-2	Point	32	percent
Lung Absorption Age 2-3	Point	32	percent
Lung Absorption Age 3-4	Point	32	percent
Lung Absorption Age 4-5	Point	32	percent
Lung Absorption Age 5-6	Point	32	percent
Lung Absorption Age 6-7	Point	32	percent

As seen, the ISE model predicts that there are no homes above the level of health concern. This is in marked contrast to the IEUBK model, which predicts that there are 1,331 homes of concern. The main reason for this difference is that the estimate of long-term average inter-individual variability generated by Monte Carlo simulation (GSD = 1.2) is substantially lower than the assumed variability in the IEUBK model (GSD = 1.6). If the variability between individuals is examined over shorter time scales (e.g., 24-36 months rather than 1-84 months), the GSD predicted by the ISE model approaches that assumed by the IEUBK model:

ISE Model GSD Values Calculated Over Various Averaging Times

Model	GSD as a Function of Averaging Time		
	Months 1-84	Months 24-36	Month 24
IEUBK	1.6	1.6	1.6
ISE	1.2	1.4	1.5

These results highlight the sensitivity of both models to the degree of inter-individual variability (as reflected in the assumed or calculated GSD), and suggests that the GSD value used by the IEUBK model may be more nearly appropriate for short-term (one-month) exposure intervals than for estimating variability in long-term average blood lead values.

Another factor which may contribute to the apparent difference between the models is that the blood lead point estimate calculated by the IEUBK model is not likely to be equivalent to the true geometric mean of the distribution of values among members of the exposed population. This is because the input point estimates used by the model are usually more likely to be arithmetic means than geometric means. If all of the inputs were arithmetic means, then the expected value of the IEUBK point estimate would be closer to the arithmetic mean blood lead than the geometric mean. Because inter-individual variability in blood leads is represented by a lognormal distribution in the IEUBK model, the arithmetic mean will always be greater than the geometric mean, so treating the IEUBK point estimate as the geometric mean may tend to shift the distribution to the right, thereby tending to increase the percent of the distribution above the health-based level of concern (10 ug/dL).

Comparison of IEUBK Results to Observed Blood Lead Values

Another way to help determine whether the IEUBK model is yielding reliable results at a particular site is to compare the IEUBK model predictions with actual observations of blood lead levels in the population of children currently living at the site. This approach has been used at a number of other sites in the Rocky Mountain west (e.g., Aspen, Leadville, Midvale), and it is often found that the observed incidence of elevated blood lead values is not as high as predicted by the model. There are a number of reasons why this might be so, including potential limitations in the blood lead study itself. However, the consistency of this pattern across sites suggests that, on average, the default IEUBK model may tend to be somewhat over-conservative. If so, this would presumably stem from imprecision in one or more of the model inputs (e.g., soil

or dust intake rates, biokinetic factors, GSD, etc.), but the actual basis of the apparent discrepancies between predicted and observed blood lead values remains uncertain and controversial.

At the VBI70 site, biomonitoring programs offered by the USEPA have resulted in collection of only very limited blood lead data. These data were derived by recruiting individuals living at homes selected for soil removal as part of the Phase II and Phase III programs to allow sampling of hair and urine to assess arsenic exposure, and sampling of blood to assess lead exposure. A total of 21 individuals participated. The results for blood lead are summarized below:

Blood Lead and Residential Soil Lead Levels at VBI70 Removal Properties

Age category	Number of Participants	Lead in Bulk Soil (ppm)		PbB (ug/dL)	
		Mean	Maximum	Geometric mean	Maximum
Child (0-6 years)	2	263	499	1.0	2.0
Adult (>6 Years)	19	459	1700	1.8	5.0
All	21	439	1700	1.7	5.0

This data set is much too limited to support any strong conclusion, especially because the number of children participating was so low, and because many of the properties had lead levels in soil that were only moderately elevated. However, the data do not provide an indication that lead exposures are above a level of concern.

Another source of potentially relevant blood data is from three different blood lead testing programs sponsored by the State. The Colorado Department of Public Health and Environment consolidated the data from these studies and provided the results to USEPA for evaluation in this risk assessment. A brief description of these three studies is provided below:

1. Denver Childhood Lead Survey. In this study, children age 0-3 years were tested from targeted neighborhoods where the risk of finding elevated blood lead levels was thought to be highest. Testing was conducted from June through September 1995.

2. Globe Medical Monitoring Program. In this study, children age 6 years and under have been tested through the Globe Medical Monitoring Program. The majority of the children were tested in the spring of 1994 at the Globe field office. These children were recruited via door-to-door outreach. Four additional children were tested at local clinics held in the south Globeville neighborhood in April of 2000.

3. State Lead Surveillance Program. This study includes blood lead results for children tested between 1995 and 2000. Most of the data collected between 1995 and 1999 were reported to the State as part of mandatory reporting of elevated blood lead levels (> 10 ug/dL) by state laboratories, and most values below 10 ug/dL were not reported.

Beginning in 2000, the State requires the laboratories to report all data, not just values above 10 ug/dL. Because of this important difference, the results for this study have been divided into Part 3A (1995-1999) and Part 3B (2000). Test results from 2000 were primarily collected at targeted clinics held specifically to recruit children living in the VBI70 area.

Because blood lead data are confidential medical information, the only information provided to USEPA on the study subjects besides their the blood lead level (ug/dL) was their age (years), the soil lead level (mg/kg) at the child's residence, and whether the residence is located within or outside the VBI70 study area.

The results are summarized in Table 5-4. Data from Phase 2 of the Third National Health and Nutrition Examination Survey (NHANES III) are also shown to provide a frame of reference (Pirkle et al. 1998). Inspection of this table reveals the following main points:

a) Within a study, there is no consistent pattern of difference in blood lead values for children living within VBI70 and those residing outside of the study area boundary. This suggests that residents living within the site do not have a substantially higher risk of lead exposure than people living in locations adjacent to the site.

b) For children age 0-5 residing within the VBI70 area, geometric mean blood lead levels observed in Study 1 (5.7 ug/dL) and Study 3A (15.6 ug/dL) are clearly higher than the national average for children age 1-5 (2.7 ug/dL). However, an elevation over average may be expected in these cases because the children in these studies do not represent a random set of children but a set selected for study because they were believed to have high risk of exposure (study 1) or were included in the study specifically because they have elevated blood lead levels (study 3A). The results for study 2 and study 3B (these studies are more nearly random than the other studies) suggest that blood lead levels for children age 0-5 residing within VBI70 (GM = 3.2 to 4.6) are somewhat higher than the national average for children age 1-5 (2.7 ug/dL), but are not clearly distinct from values seen elsewhere in the nation for children age 1-5 residing in old housing (GM = 3.8 ug/dL) or in families with low income (GM = 3.8 ug/dL) (Pirkle et al. 1998).

c) Geometric standard deviations within the different studies for children age 0-5 within the VBI70 area are range from 1.5 to 2.4. These values tend to be somewhat higher than the default GSD value of 1.6 assumed in the IEUBK model, but this is not considered to be evidence that the IEUBK default GSD value is too low. Rather, GSD values measured in most blood lead studies are expected to be higher than the true GSD for two main reasons: 1) the observed GSD includes variability in blood lead attributable to variability in environmental levels as well as variability in childhood contact with those media, while the desired value includes only variability in contact parameters; and 2) the variability in blood lead values between children is based on a single measurement in each child, rather than the long term average value in each child. As noted above,

Table 5-4 Comparison of State Blood Lead Data to National Statistics

STATE BLOOD LEAD DATA

	Age (yrs)	Within VBI70			Outside VBI70		
		N	Geomean (ug/dL)	GSD	N	Geomean (ug/dL)	GSD
Denver Survey (1)	0-4	83	5.7	1.8	83.0	6.0	2.0
Globe Program (2)	0-5	32	3.2	1.7	69	3.3	1.8
	6-11	6	3.4	1.5	17	2.8	1.3
State Surveillance Program (ALL) (3)	0-5	156	6.6	2.4	99	7.1	2.4
	6-11	17	3.9	2.0	8	6.7	2.9
State Surveillance Program (Prior to 2000) [a] (3A)	0-5	47	15.6	1.5	46	10.3	2.0
	6-11	--	--	--	4	12.7	1.5
State Surveillance Program (2000 Results) (3B)	0-5	99	4.6	2.2	53	5.1	2.4
	6-11	17	3.9	2.0	4	3.5	3.2

a This data set excludes 10 samples collected prior to 2000 with PbB < 10 ug/dL.

NATIONAL GEOMETRIC MEAN BLOOD LEAD LEVELS

Demographic Variable		Age (years)	Geomean (ug/dL)
ALL		1-5	2.7
		6-11	1.9
HOUSING	Pre 1946	1-5	3.8
	1946-1973		2.8
	Post 1973		2.0
INCOME	Low	1-5	3.8
	Middle		2.3
	High		1.9

NHANES III, Phase 2: 1991-1994. (Pirkle et al., 1998)

between-children variability in instantaneous measurements of blood lead values will always be larger than variability in long-term average values, and this results in an overestimate of the GSD. Thus, without more detailed data (e.g., repeated blood lead measurements in each child, data on the level of lead exposure in several environmental media for each child), it is considered that these data do not provide a way to estimate a reliable site-specific GSD value.

Figure 5-1 (upper panel) plots the blood lead levels across all three studies as a function of the mean soil lead level (based on data collected during the Phase III Program) at the child's residence. As seen, there is only a low degree of correlation ($R^2 = 0.019$), with high blood lead values occurring at low soil lead concentrations, and low blood lead values occurring at high soil lead concentrations. This observation establishes that soil lead is not the only source of lead exposure in children, and that soil lead is likely to explain only a small amount of the variability in blood lead levels between different children. Although the slope of the line that relates blood lead to soil lead (0.0075 ug/dL per ppm) is not statistically significant ($p = 0.07$), the slope is similar to that predicted by the IEUBK model, supporting the conclusion that soil lead probably does contribute to childhood lead exposures at the site.

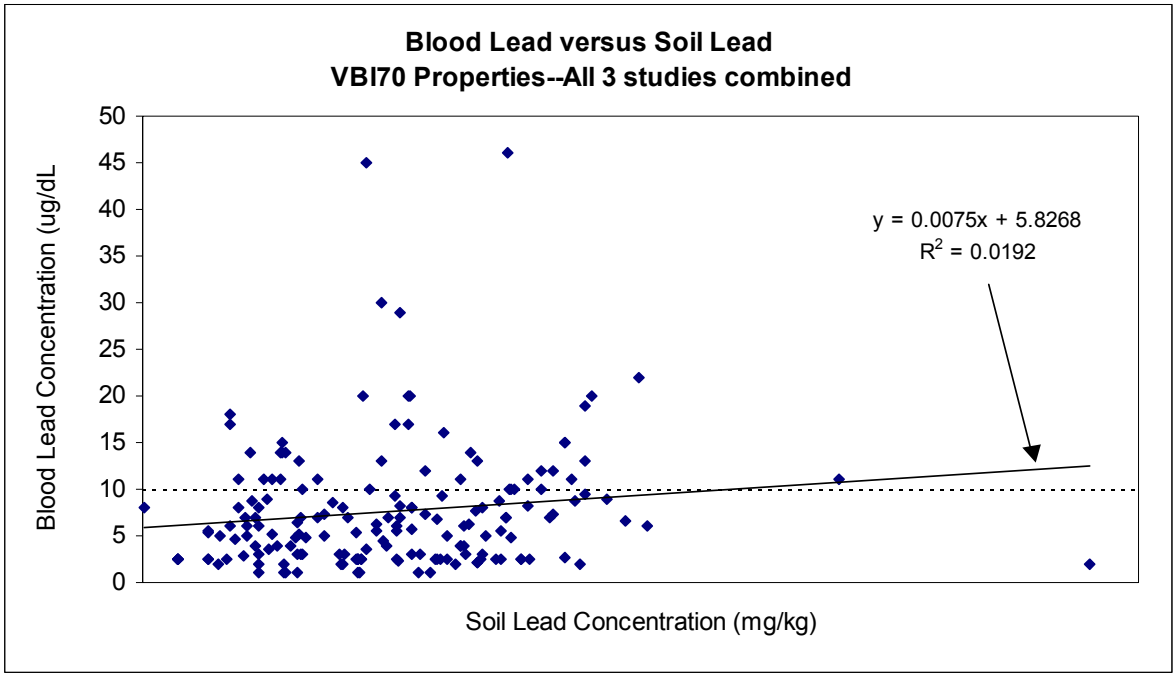
Figure 5-1 (lower panel) compares the blood lead values predicted by the IEUBK model with those actually observed in study participants. As seen, there is only a weak correlation ($R^2 = 0.059$), with the IEUBK model tending to over-predict the lower blood lead values and under-predict the higher blood lead values. This suggests that the IEUBK model may be over-estimating the contribution of the common sources of lead exposure (soil, dust, water, diet), and is not accounting for one or more large sources of lead exposure (most likely leaded paint ingestion).

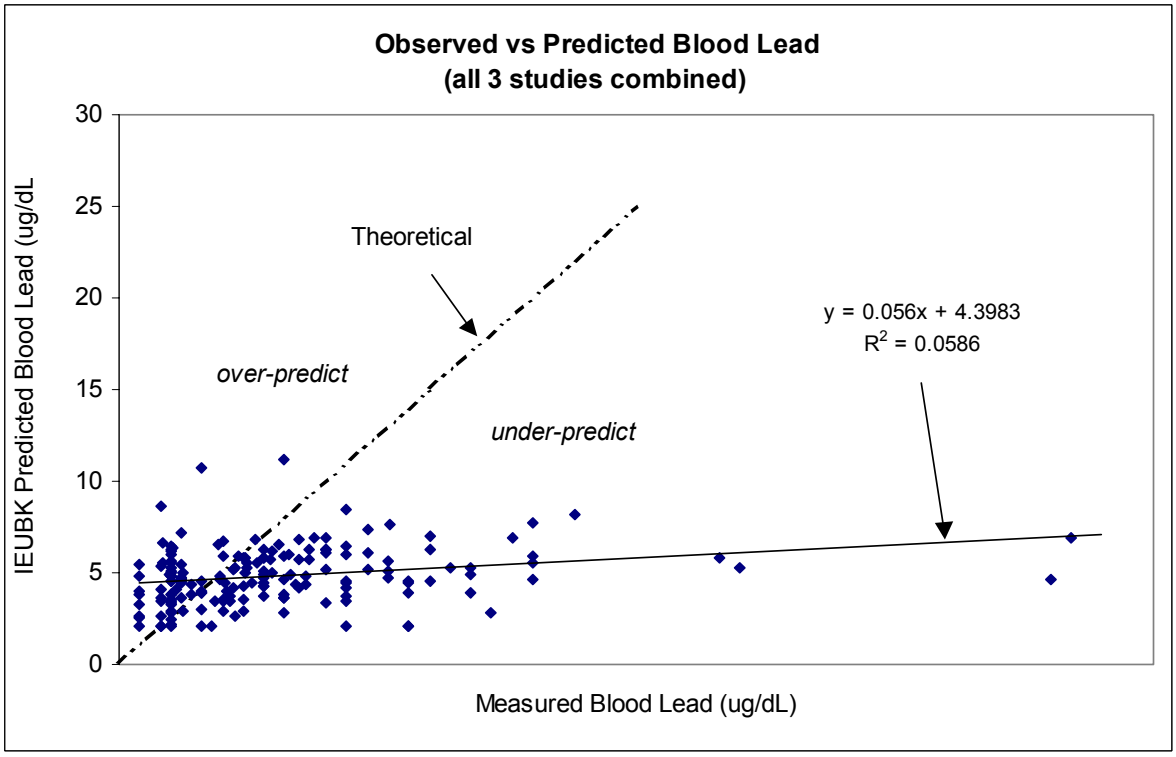
In conclusion, even though these blood lead studies were not designed or intended to support risk assessment purposes, they do support the following broad conclusions: a) elevated blood lead levels do occur in children residing within the site, b) soil is not likely to be the main source of elevated blood lead levels, and c) the elevations are not clearly different from areas outside the site.

Summary of Uncertainties

As discussed above, there are a number of sources of uncertainty in any evaluation of lead risks to children. When mathematical modeling is used to evaluate risks, the most important sources of uncertainty are in average soil ingestion rates, and in the degree of variation between the exposure rates of different children. As shown, the range of results across different sets of input values and different models can be quite large. When direct observation of blood lead values is used as the basis for evaluating risk, the main source of uncertainty is whether the study population is sufficiently large and sufficiently representative to allow correct interpretation. At this site, the available blood lead data set is clearly too small to provide a basis for any firm conclusions, but the data do not reveal any large hazard.

Figure 5-1 State Blood Lead Analysis Results





SECTION 6
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APPENDIX A

GARDEN VEGETABLE AND SOIL DATA

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Property ID	strPropAddress	Garden Vegetables										Garden Soils (mg/kg)					
		Sample ID	Vegetable Type	Dry Wt. Conc (mg/kg dw) Total As		Dry Wt. Conc (mg/kg dw) Pb		% Solid	Adj. Wet Wt. Conc (mg/kg ww) Total As	Adj. Wet Wt. Conc (mg/kg ww) Pb		Sample Number	Raw Concentration As		Raw Concentration Pb		Adjusted Conc As	Adjusted Conc Pb
1	3340 MONROE ST	3-04156-B	Rhubarb	0.05	U	0.61		8.70	2.2E-03	5.31E-02		3-04156-B	11.0	U	122.9		5.5	122.9
1	3340 MONROE ST	3-04159-B	Chard	0.10	J	0.57		6.52	6.5E-03	3.72E-02		3-04159-B	11.0	U	110.2		5.5	110.2
1	3340 MONROE ST	3-04151-B	Peppers	0.05	U	0.11	J	10.60	2.7E-03	1.17E-02		3-04151-B	11.0	U	152.2		5.5	152.2
1	3340 MONROE ST	3-04166-B	Squash	0.06	J	0.20		3.64	2.18E-03	7.28E-03		3-04166-B	15.0		248.8		15.0	248.8
1	3340 MONROE ST	3-04169-B	Squash	0.05	U	0.05	U	13.70	3.43E-03	3.43E-03		3-04169-B	11.0	U	100.8		5.5	100.8
1	3340 MONROE ST	3-04157-B	Eggplant	0.08	J	0.05	U	10.10	8.08E-03	2.53E-03		3-04157-B	11.0	U	127.3		5.5	127.3
1	3340 MONROE ST	3-04158-B	Cabbage	0.05	U	0.05	U	10.10	2.53E-03	2.53E-03		3-04158-B	11.0	U	111.2		5.5	111.2
1	3340 MONROE ST	3-04154-B	Cauliflower	0.05	U	0.05	U	9.98	2.50E-03	2.50E-03		3-04154-B	11.0	U	100.8		5.5	100.8
1	3340 MONROE ST	3-04155-B	Tomatoes	0.05	U	0.05	U	7.71	1.93E-03	1.93E-03		3-04155-B	11.0	U	104.8		5.5	104.8
1	3340 MONROE ST	3-04162-B	Squash	0.05	U	0.07	J	4.26	1.07E-03	2.98E-03		3-04162-B	11.0	U	222.6		5.5	222.6
2	3412 CLAYTON ST	3-04602-B	Tomatillo	0.05	U	0.05	U	16.50	4.13E-03	4.13E-03		3-04602-B	11.0	U	114.0		5.5	114.0
3	3510 SAINT PAUL ST	3-04600-B	Collard Greens	0.34		0.24		12.90	4.39E-02	3.10E-02		3-04600-B	11.0	U	95.9		5.5	95.9
4	3534 COLUMBINE ST	3-04620-B	Lettuce	0.10	J	2.20		10.50	1.05E-02	2.31E-01		3-04620-B	11.5		128.4		11.5	128.4
4	3534 COLUMBINE ST	3-04618-B	Carrots	0.06	J	0.96		13.20	7.92E-03	1.27E-01		3-04618-B	11.0	U	119.2		5.5	119.2
4	3534 COLUMBINE ST	3-04615-B	Beets	0.05	U	0.94		12.70	3.18E-03	1.19E-01		3-04615-B	11.3		129.7		11.3	129.7
4	3534 COLUMBINE ST	3-04617-B	Turnip Greens	0.08	J	0.68		13.60	1.09E-02	9.25E-02		3-04617-B	11.0		130.2		11.0	130.2
4	3534 COLUMBINE ST	3-04619-B	Rutabaga	0.17		0.80		11.40	1.94E-02	9.12E-02		3-04619-B	11.0	U	116.4		5.5	116.4
4	3534 COLUMBINE ST	3-04614-B	Collard Greens	0.32		0.20		17.10	5.47E-02	3.42E-02		3-04614-B	11.0	U	115.5		5.5	115.5
5	3546 HARRISON ST	3-04625-B	Collard Greens	0.16		0.50		12.10	1.94E-02	6.05E-02		3-04625-B	11.0	U	52.0	U	5.5	26.0
5	3546 HARRISON ST	3-04623-B	Peppers	0.05	U	0.05	U	15.00	3.75E-03	3.75E-03		3-04623-B	12.3		87.3		12.3	87.3
6	3604 BRUCE RANDOLPH AVE	3-04749-B	Onions	6.30		1.78		15.60	9.83E-01	2.78E-01		3-04749-B	73.3		145.5		73.3	145.5
6	3604 BRUCE RANDOLPH AVE	3-04768-B	Carrots	0.50		1.34		13.10	6.55E-02	1.76E-01		3-04768-B	46.5	J	103.9	J	46.5	103.9
6	3604 BRUCE RANDOLPH AVE	3-04758-B	Beets	1.09		1.13		13.90	1.52E-01	1.57E-01		3-04758-B	54.5		98.1		54.5	98.1
6	3604 BRUCE RANDOLPH AVE	3-04755-B	Turnips	3.45		1.21		6.11	2.11E-01	7.39E-02		3-04755-B	40.4		89.7		40.4	89.7
6	3604 BRUCE RANDOLPH AVE	3-04753-B	Cauliflower	0.46		0.50		10.00	4.60E-02	5.00E-02		3-04753-B	92.5		123.6		92.5	123.6
6	3604 BRUCE RANDOLPH AVE	3-04762-B	Collard Greens	0.37		0.12	J	15.50	5.74E-02	1.86E-02		3-04762-B	56.6		140.4		56.6	140.4
6	3604 BRUCE RANDOLPH AVE	3-04756-B	Collard Greens	0.63		0.11	J	16.20	1.02E-01	1.78E-02		3-04756-B	43.0		131.5		43.0	131.5
6	3604 BRUCE RANDOLPH AVE	3-04757-B	Cucumbers	2.92		0.27		5.92	1.73E-01	1.60E-02		3-04757-B	68.6		172.1		68.6	172.1
6	3604 BRUCE RANDOLPH AVE	3-04745-B	Zucchini	1.63		0.11	J	9.54	1.56E-01	1.05E-02		3-04745-B	45.1		280.3		45.1	280.3
6	3604 BRUCE RANDOLPH AVE	3-04743-B	Squash	0.63		0.08	J	9.70	6.11E-02	7.76E-03		3-04743-B	46.2		137.0		46.2	137.0
6	3604 BRUCE RANDOLPH AVE	3-04748-B	Tomatoes	0.08	J	0.05	U	13.30	1.06E-02	3.33E-03		3-04748-B	24.5		138.5		24.5	138.5
6	3604 BRUCE RANDOLPH AVE	3-04769-B	Cabbage	0.31		0.05	U	9.95	3.08E-02	2.49E-03		3-04769-B	48.2		110.1		48.2	110.1
7	3650 COOK ST	3-05234-B	Cabbage	0.08	J	0.08	J	19.90	1.59E-02	1.59E-02		3-05234-B	11.0	U	65.8		5.5	65.8
7	3650 COOK ST	3-05225-B	Tomatillo	0.05	U	0.06	J	16.30	4.08E-03	9.78E-03		3-05225-B	11.0	U	56.6		5.5	56.6
8	3223 RACE ST	3-05239-B	Beets	0.34		2.32		19.70	6.70E-02	4.57E-01		3-05239-B	11.0	U	236.0		5.5	236.0
8	3223 RACE ST	3-05240-B	Turnips	0.52		0.98		7.29	3.79E-02	7.14E-02		3-05240-B	19.5		260.5		19.5	260.5
9	3244 VINE ST	3-05237-B	Tomatoes	0.05	U	0.05	U	7.07	1.77E-03	1.77E-03		3-05237-B	11.0	U	137.2		5.5	137.2
9	3244 VINE ST	3-05238-B	Tomatoes	0.05	U	0.05	U	6.54	1.64E-03	1.64E-03		3-05238-B	37.1		170.6		37.1	170.6
10	3250 HIGH ST	3-04585-B	Tomatoes	0.05	U	0.62		7.67	1.92E-03	4.76E-02		3-04585-B	18.4		314.1		18.4	314.1
11	3310 VINE ST	3-04792-B	Garlic	1.24		38.60		16.50	2.05E-01	6.37E+00		3-04792-B	11.0	U	270.6		5.5	270.6
11	3310 VINE ST	3-05226-B	Chard	0.14	J	4.31		13.20	1.85E-02	5.69E-01		3-05226-B	12.0		147.5		12.0	147.5
11	3310 VINE ST	3-05222-B	Onions	0.10	J	1.67		12.40	1.24E-02	2.07E-01		3-05222-B	11.0	U	250.1		5.5	250.1
11	3310 VINE ST	3-05230-B	Collard Greens	0.07	J	0.53		13.80	9.66E-03	7.31E-02		3-05230-B	17.7		184.9		17.7	184.9
11	3310 VINE ST	3-04791-B	Collard Greens	0.12	J	0.16		13.60	1.63E-02	2.18E-02		3-04791-B	16.4		212.7		16.4	212.7
11	3310 VINE ST	3-04799-B	Cucumbers	0.67		0.18		6.02	4.03E-02	1.08E-02		3-04799-B	16.7		259.1		16.7	259.1

Property ID	strPropAddress	Garden Vegetables									Garden Soils (mg/kg)						
		Sample ID	Vegetable Type	Dry Wt. Conc (mg/kg dw) Total As		Dry Wt. Conc (mg/kg dw) Pb		% Solid	Adj. Wet Wt. Conc (mg/kg ww) Total As	Adj. Wet Wt. Conc (mg/kg ww) Pb	Sample Number	Raw Concentration As		Raw Concentration Pb		Adjusted Conc As	Adjusted Conc Pb
12	3315 RACE ST	3-04773-B	Carrots	0.27		1.15		11.00	2.97E-02	1.27E-01	3-04773-B	26.0		140.1		26.0	140.1
12	3315 RACE ST	3-04765-B	Collard Greens	0.38		0.58		12.00	4.56E-02	6.96E-02	3-04765-B	17.5		224.9		17.5	224.9
12	3315 RACE ST	3-04776-B	Collard Greens	0.56		0.30		13.00	7.28E-02	3.90E-02	3-04776-B	22.8		157.4		22.8	157.4
12	3315 RACE ST	3-04775-B	Collard Greens	0.27		0.29		11.00	2.97E-02	3.19E-02	3-04775-B	25.1		152.4		25.1	152.4
13	3322 VINE ST	3-04789-B	Onions	0.17		1.87		14.80	2.52E-02	2.77E-01	3-04789-B	16.9		217.7		16.9	217.7
13	3322 VINE ST	3-04798-B	Celery	0.19		2.05		8.25	1.57E-02	1.69E-01	3-04798-B	11.0	U	338.8		5.5	338.8
13	3322 VINE ST	3-04794-B	Turnips	0.33		1.57		10.20	3.37E-02	1.60E-01	3-04794-B	11.0	U	210.3		5.5	210.3
13	3322 VINE ST	3-04779-B	Collard Greens	0.11	J	0.16		13.70	1.51E-02	2.19E-02	3-04779-B	25.4		344.0		25.4	344.0
13	3322 VINE ST	3-04786-B	Squash	0.05	J	0.29		6.17	3.09E-03	1.79E-02	3-04786-B	11.0	U	240.8		5.5	240.8
13	3322 VINE ST	3-04782-B	Peas	0.05	U	0.06	J	22.50	5.63E-03	1.35E-02	3-04782-B	11.0	U	294.0		5.5	294.0
13	3322 VINE ST	3-04771-B	Cabbage	0.13	J	0.05	U	11.60	1.51E-02	2.90E-03	3-04771-B	11.5		186.0		11.5	186.0
13	3322 VINE ST	3-04784-B	Tomatoes	0.05	U	0.05	U	9.96	2.49E-03	2.49E-03	3-04784-B	11.0	U	253.0		5.5	253.0
13	3322 VINE ST	3-04781-B	Cabbage	0.05	U	0.05	U	9.24	2.31E-03	2.31E-03	3-04781-B	11.0	U	195.5		5.5	195.5
14	3351 GAYLORD ST	3-04148-B	Onions	0.14	J	0.68		13.80	1.93E-02	9.38E-02	3-04148-B	11.6		162.2		11.6	162.2
14	3351 GAYLORD ST	3-04144-B	Peppers	0.05	U	0.20		9.68	2.42E-03	1.94E-02	3-04144-B	15.3		171.6		15.3	171.6
14	3351 GAYLORD ST	3-04150-B	Broccoli	0.08	J	0.06	J	12.20	9.76E-03	7.32E-03	3-04150-B	21.3		183.5		21.3	183.5
15	3511 LAFAYETTE ST	3-05249-B	Cucumbers	0.68		0.66		4.55	3.09E-02	3.00E-02	3-05249-B	11.0	U	369.3		5.5	369.3
15	3511 LAFAYETTE ST	3-05247-B	Tomatoes	0.05	U	0.33		5.84	1.46E-03	1.93E-02	3-05247-B	11.0	U	570.0		5.5	570.0
15	3511 LAFAYETTE ST	3-05248-B	Tomatillo	0.10	J	0.16		7.37	7.37E-03	1.18E-02	3-05248-B	11.0	U	335.1		5.5	335.1
15	3511 LAFAYETTE ST	3-05244-B	Tomatoes	0.05	U	0.10	J	6.63	1.66E-03	6.63E-03	3-05244-B	11.0	U	381.1		5.5	381.1
16	3630 RACE ST	3-04608-B	Peppers	0.15		0.15	J	13.10	1.97E-02	1.97E-02	3-04608-B	15.2		79.5		15.2	79.5
17	4300 STEELE ST	3-04588-B	Tomatoes	0.05	U	0.18		5.90	1.48E-03	1.06E-02	3-04588-B	11.0	U	52.0	U	5.5	26.0
17	4300 STEELE ST	3-04589-B	Peppers	0.05	U	0.05	U	13.60	3.40E-03	3.40E-03	3-04589-B	11.0	U	61.2		5.5	61.2
18	4314 JOSEPHINE ST	3-04744-B	Tomatoes	0.05	U	0.11	J	5.77	1.44E-03	6.35E-03	3-04744-B	13.7		572.9		13.7	572.9
19	4755 GAYLORD ST	3-04597-B	Beans	0.05	U	0.13	J	19.00	4.75E-03	2.47E-02	3-04597-B	11.0	U	408.3		5.5	408.3
19	4755 GAYLORD ST	3-04595-B	Tomatillo	0.05	U	0.20		6.47	1.62E-03	1.29E-02	3-04595-B	16.0		236.1		16.0	236.1
19	4755 GAYLORD ST	3-04592-B	Tomatoes	0.05	U	0.05	U	6.05	1.51E-03	1.51E-03	3-04592-B	11.0	U	260.8		5.5	260.8

U=Analyte not detected
J=Estimated

APPENDIX B

SCREENING LEVEL EVALUATION OF RELATIVE RISK FROM ARSENIC VIA INHALATION OF DUST OR DERMAL CONTACT WITH SOIL COMPARED TO SOIL INGESTION

APPENDIX B

SCREENING LEVEL EVALUATION OF RELATIVE RISK FROM ARSENIC VIA INHALATION OF DUST OR DERMAL CONTACT WITH SOIL COMPARED TO SOIL INGESTION

1.0 INHALATION OF PARTICULATES IN AIR

The basic equations recommended by USEPA (1989) for evaluation of risk from inhalation exposure of soil particles in air and for incidental ingestion of soil are as follows:

<u>Inhalation Exposure</u>

$$Risk_{air} = C_a \cdot BR_a \cdot EF \cdot ED/(BW \cdot AT) \cdot SF_{inh}$$

<u>Ingestion Exposure</u>

$$Risk_{soil} = C_{soil} \cdot IR_{soil} \cdot EF \cdot ED/(BW \cdot AT) \cdot SF_{oral}$$

where:

C = Concentration of contaminant in air (C_a, mg/m^3) or soil (C_{soil}, mg/kg)
BR = Breathing rate (m^3/day)
IR_{soil} = Ingestion rate for soil (kg/day)
EF = Exposure frequency (days/yr)
ED = Exposure duration (years)
BW = Body weight (kg)
AT = Averaging time (days)
SF = Cancer slope factor for inhalation or oral exposure

Assuming that the values of BW, EF, ED, and AT are all the same for inhalation and oral exposure, the ratio of the risk from inhalation of particulates in air to that from ingestion of soil is then:

$$\text{Relative risk (inhalation/oral)} = (C_{air}/C_{soil})(BR/IR)(SF_{inhal}/SF_{oral})$$

Soil particles may be released from soil and enter air due either to wind-based erosion or mechanical disturbance. A screening level evaluation of each type of scenario is presented below.

Exposure from Wind-Based Soil Erosion

The amount of soil released to air by wind is a complex function of wind speed, soil characteristics, and the surface features of the site. The USEPA has developed a conservative screening level approach for evaluating wind-based releases, as described in USEPA (1996). Screening level defaults inputs for this equation are as follows:

- The ratio C_{air}/C_{soil} (ug/m^3 per ug/kg) is given by the inverse of the Particulate Emission Factor (PEF), calculated in accord with the equation and region-specific intake values identified in USEPA (1996). The resulting value is 9.1E-10 kg/m^3 (0.91 ug/m^3).

- The ratio of BR/IR for a resident is 20 m^3/day / 1E-04 kg/day = 2E+05 m^3/kg (USEPA 1989, 1991b)

- For arsenic, the ratio of the inhalation slope factor to the oral slope factor is 15/1.5 = 10 (IRIS 2000).

Based on these values, the ratio of the risk from inhalation exposure to arsenic in airborne soil particles compared to that from ingestion exposure is:

Relative risk = 9.1E-10 · 2E+05 · 10 = 0.0018 (0.18%)

As seen, the risk from inhaled arsenic is very small (< 0.2%) compared to that from ingested soil, so this pathway is considered to be sufficiently minor that quantitative evaluation is not required at this site.

Exposure from Mechanical Disturbances

The amount of soil which enters air as a result of mechanical disturbances (e.g., automobile traffic on a dirt road, agricultural tilling of a field, etc) is a complex function of the type and frequency of the disturbance. At the VBI70 site, data are available from a large highway construction project being carried out by the Colorado Department of Transportation for the Brighton Road Interchange on I-70 (CDOT 2000a, 2000b, 2000c). These data include 79-82 samples collected at each of three different monitoring stations over the interval from January through September, 2000. Each sample was analyzed for PM10 (particulate matter less than 10 um in diameter) and/or TSP (total suspended particulates). In addition, the levels of arsenic and lead in PM10 and TSP were measured.

The average level of PM10 measured at one station was 65 ug/m^3. This level is nearly two orders of magnitude higher than the default level of 0.9 ug/m^3 used to evaluate wind-erosion (see above). If this airborne matter were all attributable to mechanical erosion of soil into air, the relative cancer risk from inhalation compared to ingestion might be as large as about 13% (still a relatively small fraction). However, it is important to note that not all PM10 particles

in air are derived from soil. In support of this, average arsenic levels in PM10s and/or in TSP ranged from 2.4 to 3.2 ng/m^3, a level that is lower than the average of 20-30 ng/m^3 for urban areas across the United States (ATSDR 2000). Likewise, the average level of lead was 28-37 ng/m^3, lower than the default value of 100 ng/m^3 used by USEPA in the IEUBK model. These data indicate that even under conditions of mechanical disturbance, airborne levels of arsenic and lead from soil are still quite low and are not a source of significant health concern.

2.0 DERMAL EXPOSURE VIA SOIL

The basic equations recommended for estimation of risk from dermal contact with soil and ingestion of soil are as follows (USEPA 1989, 1992):

<u>Dermal Exposure</u>

$$Risk_{dermal} = C_s \cdot SA \cdot AF \cdot ABS \cdot EF \cdot ED/(BW \cdot AT) \cdot (SF_{oral}/AFo)$$

<u>Oral Exposure</u>

$$Risk_{soil} = C_s \cdot IR_{soil} \cdot EF \cdot ED/(BW \cdot AT) \cdot SF_{oral}$$

where:

C_s	=	concentration of chemical in soil (mg/kg)
SA	=	surface area in contact with soil (cm^2)
AF	=	soil adherence factor (kg/cm^2)
ABS	=	dermal absorption fraction (unitless)
AFo	=	oral absorption fraction
IR_{soil}	=	ingestion rate for soil (kg/day)
BW	=	body weight (kg)
EF	=	exposure frequency (days/yr)
ED	=	exposure duration (years)
AT	=	averaging time (days)
SF_{oral}	=	cancer slope factor for oral exposure

Thus, assuming the values of BW, ED, and AT are the same for dermal and oral exposure, the ratio of the risk for dermal contact compared to that for soil ingestion is given by:

$$\text{Relative risk (dermal/oral)} = (SA \cdot AF \cdot EF_{dermal} \cdot ABS)/(IR \cdot EF_{oral} \cdot AFo)$$

Screening level inputs for this equation are as follows:

- SA = 10% of whole body = 2,000 cm^2 (USEPA 1991b).
- AF = 1E-06 kg/cm^2 (USEPA 1992)
- EF_{dermal} = 50 days/yr (assumed)
- ABS is not known for arsenic, but is likely to be no higher than 0.01 (USEPA 1992)
- IR = 1E-04 kg/day (USEPA 1989, 1991b)

- EF_{oral} = 350 days/yr (USEPA 1989, 1991b)
- AFo = 1.0 for arsenic (assumed)

Based on these inputs, the estimated ratio of dermal risk to ingestion risk for arsenic in soil is:

Relative Risk = (2E+03·1E-06·50·0.01)/(1E-04·350·1.0) = 0.029 (2.9%)

Thus, the relative risk from dermal contact with arsenic in soil compared to ingestion exposure is likely to be no more than about 3%, and could be less if the frequency or extent of dermal contact is lower than assumed, or if the dermal absorption fraction for arsenic is lower than 0.01. On this basis, it is concluded that dermal absorption is a minor contributor of risk compared to oral exposure, and that this pathway may be excluded from quantitative evaluation.

3.0 REFERENCES

USEPA. 1989. Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual Part A. Interim Final. Office of Solid Waste and Emergency Response (OSWER), Washington, DC. OSWER Directive 9285.701A.

USEPA. 1991a. Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual (Part B, Development of Risk-Based Preliminary remediation Goals). Interim. Office of Research and Development, Washington, DC. EPA/540/R-92-003.

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APPENDIX C

RISK-BASED CONCENTRATION VALUES FOR WORKERS

APPENDIX C

RISK-BASED CONCENTRATION VALUES FOR WORKERS

1.0 OVERVIEW

A Risk-Based Concentration (RBC) is a concentration of a chemical in a medium that is not of health concern to a specified population under a specified set of exposure assumptions. RBC values are derived by reversing the risk assessment process, solving for the concentration of a chemical that corresponds to a specified target risk value. This Appendix calculates the RBC values for exposure of workers to arsenic and lead in soil. These values may then be used to assess whether there is a need for quantitative evaluation of risk to this population.

2.0 RBC FOR ARSENIC

The basic equation used to calculate the RBC for exposure of workers to arsenic in soil is:

$$\text{RBC} = \frac{\text{Target Risk}}{\left(\frac{\text{IR}}{\text{BW}}\right)\left(\frac{\text{EF} \cdot \text{ED}}{\text{AT}}\right)(\text{oSF} \cdot \text{RBA})}$$

Input values applicable to worker exposure to soil are listed below, along with the resulting RBC value.

Parameter	Default Value	Source
Target Risk	1E-04	USEPA 1991b
IR (kg/day)	1E-04	USEPA 1991a
BW (kg)	70	USEPA 1991a
EF (days/yr)	250	USEPA 1991a
ED (years)	25	USEPA 1991a
AT (years)	70	USEPA 1991a
RBA	0.42	USEPA 2001b
oSF $(\text{mg/kg-d})^{-1}$	1.5	IRIS 2000
RBC (mg/kg)	454	Calculated

3.0 RBC FOR LEAD

The EPA has not established a default soil action level for lead for protection of workers. However, the EPA has developed an interim method for calculating the risk to workers from lead in soil (USEPA 1996). The basic equation is:

$$GM\ PbB = PbB0 + PbS \cdot BKSF \cdot IR_s \cdot AF_s \cdot EF_s / AT$$

where:

GM PbB	=	Geometric mean blood lead (ug/dL) in a population of workers
PbB0	=	Baseline geometric mean blood lead value (ug/dL) in the workers in the absence of occupational exposure
BKSF	=	Biokinetic slope factor (ug/dL increase in blood lead per ug/day of lead absorbed)
PbS	=	Concentration of lead in soil (ug/g)
IR_s	=	Intake rate of soil (g/day)
AF_s	=	Absorption fraction for lead from soil. This value is given by: $AF_s = AF_{food} \cdot RBA_{soil}$
EF_s	=	Exposure frequency to soil (days/yr)
AT	=	Averaging time (days)

Given the GM PbB, and assuming the distribution of PbB values is lognormal with a geometric standard deviation of GSD, the 95th percentile of the distribution is given by:

$$95th = GM \cdot GSD^{1.645}$$

The subpopulation of primary concern for protection of workers from excessive lead exposure is pregnant females. The goal is to ensure that there is no more than a 5% chance that the blood lead level of the fetus will exceed 10 ug/dL. The ratio between the blood lead concentration in the mother and the fetus is given by:

$$R(fetal/maternal) = PbB(fetus) / PbB(mother)$$

Default input values recommended by USEPA for each of these parameters are summarized in Table C-1. Using these inputs, the concentration of lead in soil which yields a 95th percentile value of 10 ug/dL in the blood of the fetus may be calculated. This value is 1,545 ppm.

TABLE C-1 DEFAULT INPUT PARAMETERS
ADULT WORKERS LEAD EXPOSURE MODEL

INPUTS		
PbB0	2.0	ug/dL
BKSF	0.4	ug/dL per ug/day
IRsoil	0.05	g/day
EFsoil	219	days/yr
AT	365	days/yr
AFfood	0.2	
RBAsoil (a)	0.84	
R(fetal/maternal)	0.9	
GSD	1.8	
CALCULATED VALUES		
Target 95th (maternal)	11.1	ug/dL
Target GM (maternal)	4.23	ug/dL
AFsoil	0.17	
RESULT		
RBC	1104	ug/g

(a) Site-specific value estimated from studies in animals (USEPA 2001c)

APPENDIX D

MONTE CARLO MODELING OF EXPOSURE AND RISK FROM ARSENIC IN SOIL AT THE VBI70 SITE

APPENDIX D

SCREENING LEVEL MONTE CARLO MODELING OF EXPOSURE AND RISK FROM ARSENIC IN SOIL AT THE VBI70 SITE

1.0 INTRODUCTION

Monte Carlo modeling is a computer-based mathematical technique that may be used for calculating exposure and risk where input terms are characterized as Probability Density Functions (PDFs) rather than point estimates. This approach has the advantage that the full distribution of exposure and risk may be predicted (as opposed to two point estimates, the CTE and RME values), and that the percentiles of those estimates may be quantified. In addition, the Monte Carlo approach helps guard against "compounding conservatism", whereby a series of conservative assumptions are combined into a single but unlikely scenario.

2.0 BASIC EQUATIONS

The basic equations used to calculate risk using the Monte Carlo approach are identical to those used in the point estimate approach. These equations are presented in Section 4.2 of the main risk assessment.

3.0 SELECTION OF INPUT VARIABLES

In concept, every term used in the point estimate equation is a variable, and could be modeled as a probability density function (PDF). However, for simplicity, it is generally not necessary to evaluate every term as a PDF. Rather, only those terms that are the most variable and which are the primary sources of variability in the output (exposure and risk) need be modeled as PDFs.

For this screening level evaluation, the following inputs are judged to be the chief sources of variability in exposure and risk among individuals:

Exposure frequency (EF)
Exposure duration (ED)
Intake rate for soil and dust (IRsd)
Fraction of intake that is soil (Fs)
Vegetable intake rate (IRveg)

The distribution functions selected to model each of these variables are described below.

Exposure Frequency (EF)

Exposure frequency is the average number of days per year spent at home. No data were located on the distribution this variable, so a triangular distribution was selected, as follows:

$$EF \sim TRI(200, 234, 365)$$

The central tendency value of 234 days/yr is based on the default CTE value recommended by EPA, while the upper bound would represent the case where a person was at home continuously. This distribution yields a average value of 266 days per year (somewhat higher than the EPA default of 234 days/year for the CTE resident), and a 95th percentile value of 332 days per year (slightly lower than the EPA default of 350 days/year for the RME receptor).

Exposure Duration (ED)

Data on the length of time that people live in a specific residence are available in the Exposure Factors Handbook (USEPA 1997) (see Table 15-167). The empiric cumulative distribution based on data from 500,000 individuals is shown in Table D-1.

Soil and Dust Intake Rate (IRsd)

Two alternative distributions were used to evaluate soil and dust intake by children. The first is a lognormal distribution selected to match the USEPA default values of 100 and 200 mg/day for the CTE and RME child. The parameters of this distribution (mean and standard deviation) are as follows:

$$IR(soil,dust)_{child} \sim LN(100,53)$$

The second distribution is an empiric cumulative distribution based on the recent study by Stanek and Calabrese (1999). The study included observations on 64 children for a period of 2-7 days (a total of 331 child-days). The parameters of this distribution are shown in Table D-1.

Fraction Soil (Fs)

Data on the fraction of total intake of soil plus dust that is soil are very limited. Stanek and Calabrese (1992) analyzed data from 64 pre-school children over a 2-week period. The data ranged from a minimum of zero percent up to a maximum of 100%, and the cumulative distribution was very nearly equal to a straight line. On this basis, Fs was modeled as a uniform distribution with parameters (0,1).

Vegetable Intake Rate

Data on seasonally adjusted consumer-only intake of home grown vegetables, stratified by region, are provided in the Exposure Factors Handbook (Table 13-33). The empiric cumulative distribution function is shown in Table D-1.

Other Inputs

All other exposure and risk model terms were the same as used in the point estimate calculations.

4.0 RESULTS

Table D-2 shows the results of a Monte Carlo simulation at an exposure point where the concentration of arsenic in soil (fine fraction) is assumed to be 200 ppm. Similar results are obtained at other soil concentrations.

Figure D-1 plots the distribution of cancer risks from ingestion of soil and dust at this location (concentration in fines = 200 ppm). The two curves shown in the figure represent the results for the two different PDFs assumed for soil intake (see above). Inspection of this figure reveals the following main points:

1. The distribution of risks based on the soil intakes reported by Stanek and Calabrese (2000) are substantially lower than the values based on the EPA default intake parameters

2. Compared to the distribution that assumes default EPA intake rates, the CTE point estimate is lower than the mean of the distribution, and corresponds to the 56th percentile. The RME point estimate is substantially higher than the 95th percentile of the distribution, and corresponds to a value above the 99th percentile.

3. Compared to the distribution that assumes the soil intake data of Stanek and Calabrese (2000), the CTE point estimate corresponds to the 86th percentile, while the RME point estimate corresponds to a value well above the 99.9th percentile.

These results indicate that RME point estimates of risk are likely to be conservative (i.e., will provide protection to more than 95% of the exposed population), especially if soil intake is actually closer to the data of Stanek and Calabrese (2000) than to the EPA defaults.

Figure D-2 compares point estimates and Monte Carlo estimates of total risk from arsenic (the sum of exposure via vegetable intake and soil/dust intake) across a range of soil concentrations. In all cases, the Monte Carlo calculations assume a soil intake that is lognormal and the parameters are matched to the EPA defaults. The upper panel compares the CTE point estimate of risk (CTE soil + CTE vegetable) with the mean of the Monte Carlo simulation. As noted above, at any specified soil level, the point estimate of CTE risk is below the mean value of the

MCA. The lower panel compares the 95th percentile of the MCA with three alternative estimates of the total RME risk:

Method 1 = RME soil + CTE vegetables
Method 2 = CTE soil + RME vegetables
Method 3 = RME soil + RME vegetables

As seen, the 95th percentile of total risk calculated by MCA is lower than the point calculations of RME total based on Method 1 (used in this risk assessment) at all soil levels. As expected, Method 3 (RME soil + RME vegetable) yields a result much higher than Method 1 or the MCA value. These results provide assurance that the estimates of total risk calculated across pathways calculated using Method 1 are likely to be conservative (higher than actual).

TABLE D-1 EMPIRIC DISTRIBUTION FUNCTIONS USED IN MONTE CARLO MODELING

Exposure Duration		Soil Intake		Veg Intake	
Years		mg/day		kg ww/kg bw/day	
EFH Table 15-167		(Stanek and Calabrese 1999)		EFH Table 13-33	
1	0.00	0	0.00	1.80E-03	0.00
1.9	0.05	2	0.10	1.91E-02	0.05
2	0.10	9	0.20	3.83E-02	0.10
3	0.25	16	0.30	1.14E-01	0.25
9	0.50	21	0.40	4.92E-01	0.50
16	0.75	24.5	0.50	1.46E+00	0.75
26	0.90	29	0.60	2.99E+00	0.90
33	0.95	35	0.70	5.04E+00	0.95
41	0.98	53	0.80	8.91E+00	0.99
47	0.99	75	0.90	1.12E+01	1.00
55	0.998	91	0.95		
59	0.999	137	0.99		
87	1.000	173	1.00		

TABLE D-2 MONTE CARLO RESULTS
Soil Conc = 200 ppm in fines

Soil IR	Percentile	Risk(s+d)	Risk(veg)	Risk(total)
LN(100,53)	0.050	1E-06	2E-08	1E-06
	0.250	4E-06	2E-07	5E-06
	0.500	9E-06	9E-07	1E-05
	0.750	2E-05	3E-06	3E-05
	0.900	4E-05	9E-06	5E-05
	0.950	6E-05	1E-05	7E-05
	0.990	1E-04	3E-05	1E-04
	0.999	2E-04	8E-05	2E-04
Empiric (see Table D-1)	0.050	7E-08	2E-08	3E-07
	0.250	6E-07	2E-07	1E-06
	0.500	2E-06	9E-07	4E-06
	0.750	6E-06	3E-06	1E-05
	0.900	1E-05	8E-06	2E-05
	0.950	2E-05	1E-05	3E-05
	0.990	5E-05	4E-05	6E-05
	0.999	1E-04	7E-05	1E-04

FIGURE D-1

MONTE CARLO RESULTS FOR EXPOSURE TO ARSENIC IN SOIL/DUST

Concentration in Fine Fraction = 200 ppm

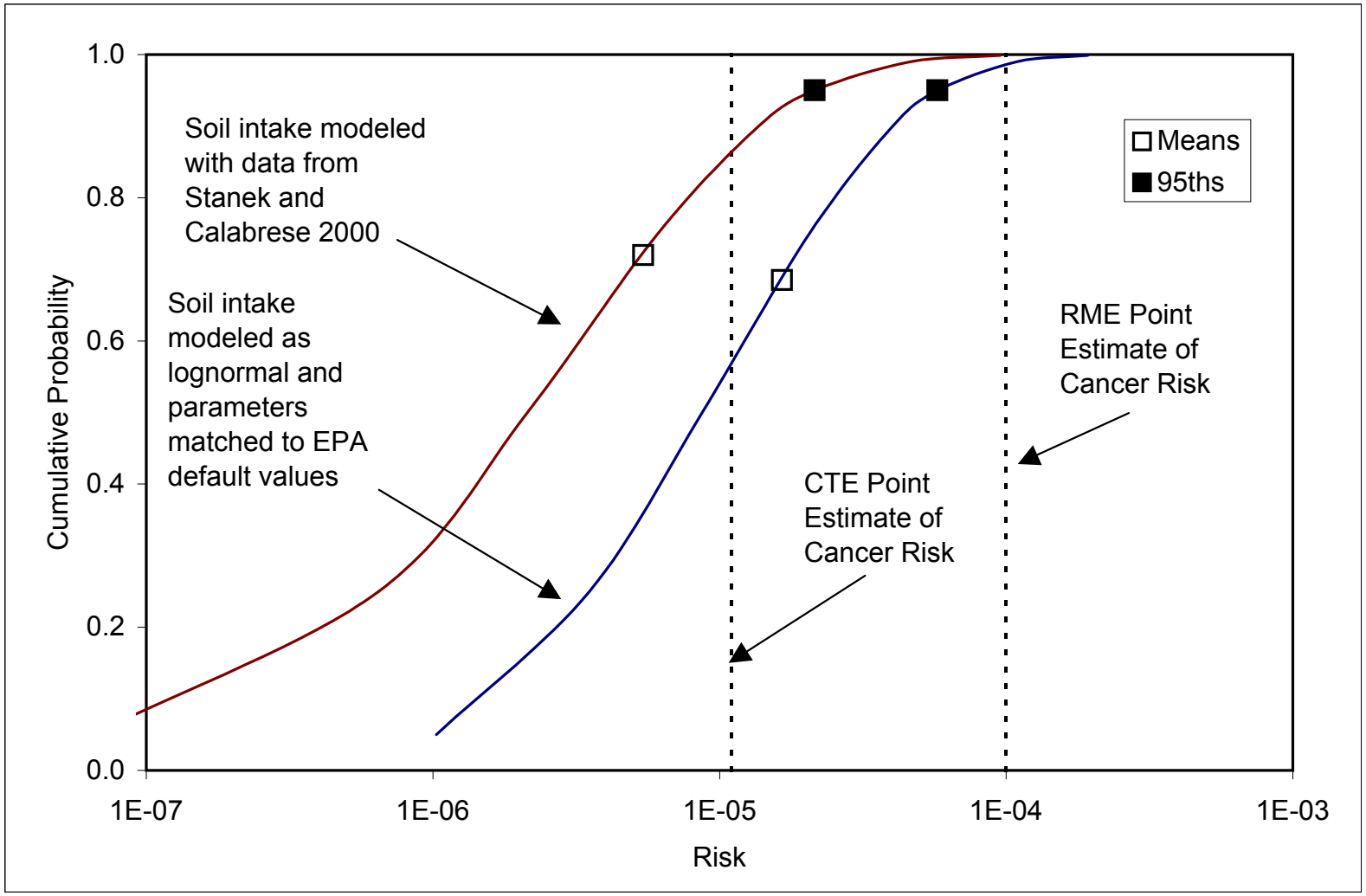
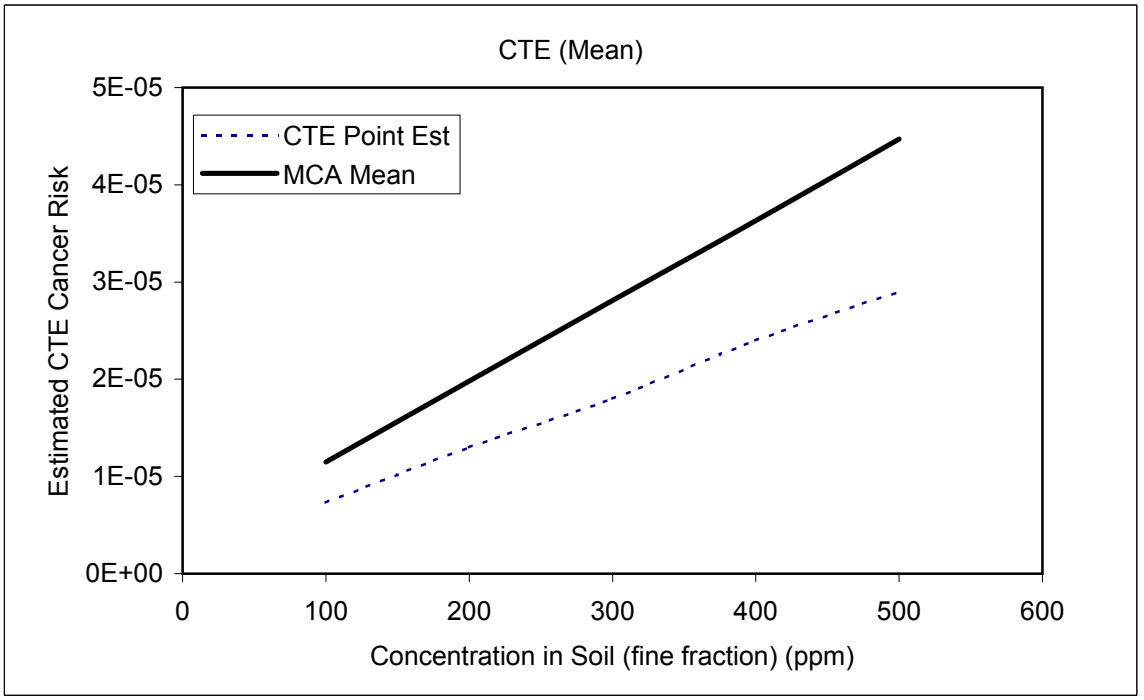
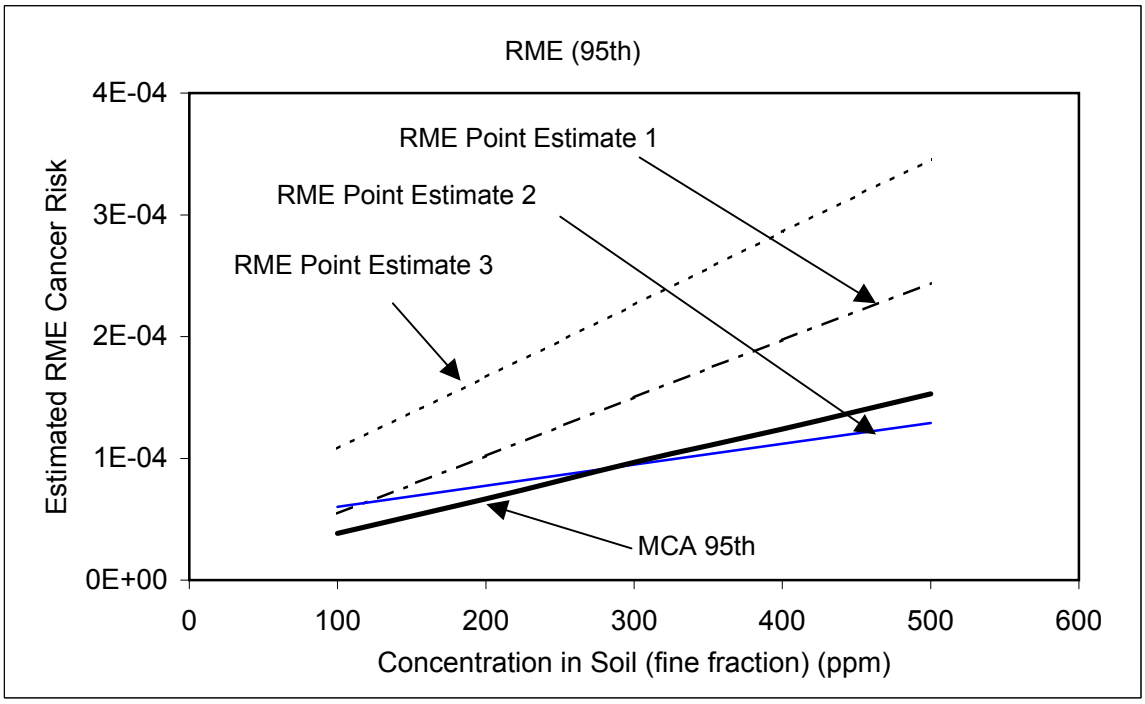


FIGURE D-2
COMPARISON OF POINT ESTIMATE AND MONTE CARLO ESTIMATES OF TOTAL RISK ACROSS A RANGE OF ARSENIC CONCENTRATIONS IN SOIL





Monte Carlo evaluation assumes soil intake is distributed lognormally with a mean of 100 mg/day and a standard deviation of 53 mg/day (95th percentile = 200 mg/day)