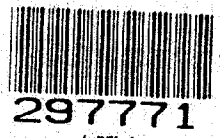


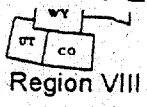
RCRA-1998-0003-0202



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
REGION VIII (8HWM-SM)  
999 18th STREET - SUITE 500  
DENVER, COLORADO 80202-2466



ADMINISTRATIVE  
RECORD



**ENDANGERMENT ASSESSMENT**

for

**Bingham Creek, Utah**

*Phase III Remediation*

prepared by

Superfund Management Branch, Hazardous Waste Management Division  
EPA Region VIII, Denver, CO

reviewed by

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## I. INTRODUCTION

This document comprises an *endangerment assessment* (EA), which is required by the US EPA Superfund Program to characterize potential health risks from contaminants at a site. Elevated levels of and exposures to hazardous waste contamination may require removal or other remedial actions to reduce risks and adequately safeguard current and future health of the exposed people. This EA describes Phase III of health risk assessments for the Bingham Creek residential area, and it follows two preliminary endangerment assessments (PEA) that were written in 1991 and 1993 to support earlier removal actions at the site. These prior actions were undertaken to reduce or eliminate imminent and substantial endangerments to health, which were described during the Phase I and II investigations. The Phase III EA is intended to adequately characterize remaining site hazards and risks to human health for the purpose of assisting risk managers in determining appropriate risk reduction actions, if any,

### A. Site Description

The physical site and exposure conditions being evaluated for human health risks are in the Bingham Creek residential areas that are located in and near West Jordan, Utah (with some properties in or nearer to South Jordan). The site has mostly residential properties with mixtures of single family houses having standard lot sizes for this region, some small acreages with pastures and/or gardens, and smaller areas with trailer parks or multi-family apartment housing. The residential properties of health concern are located in or near the historic flood plain of Bingham Creek extending eastward from the western city boundary at 4800 West to the Jordan River. The exact boundaries of historic mine waste contamination are not known, but past soil samplings by EPA in 1991 and by the University of Cincinnati in 1993 helped identify where the majority of contaminated properties are located which have elevated metals above background concentrations and at levels approaching or exceeding health concerns. A 1991 rendition of a USGS map shows the general site area along Bingham Creek (Fig. 1-1).

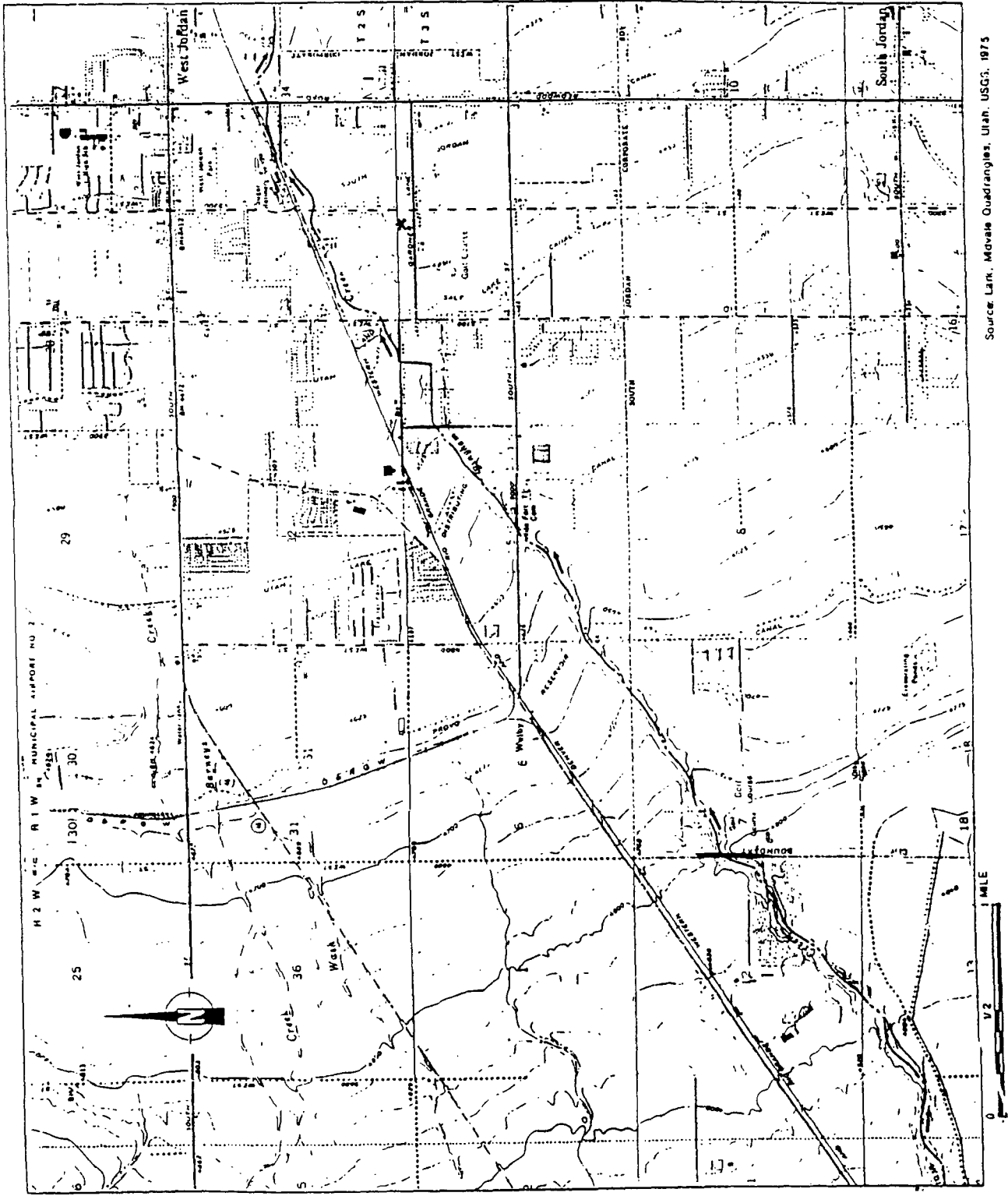
FIGURE 1-1: 1991 Map Showing General Area of Health Concern Around Bingham Creek.

TECHNICAL ASSISTANCE TEAM FOR EMERGENCY RESPONSE, REMOVAL AND PREVENTION  
EPA CONTRACT 68-07-1361

TITLE: BINGHAM CREEK  
West Jordan, Utah

T.O.P. 108-9103-003  
ecology & environment, inc.  
DENVER, COLORADO

Date: 04/91, 04/92, 04/93, RSM State.



Source: Lark, Movable Quadrangles, Utah, USGS, 1975

## B. Sources of Contamination

The main originating sources of contamination are the historic mining operations in the upper areas of Bingham Creek drainage in the Oquirrh Mountains to the west of Salt Lake City. Kennecott Utah Copper and ARCO own and are in the process of controlling or have controlled the releases of further contamination from these primary source areas. The ARCO Tailings and/or Anaconda Tailings near the boundary of Kennecott's eastern property line and along Bingham Creek is a discreet source area that is currently being addressed under Superfund's removal program with plans for eventual capping of the mine tailings piles.

Natural runoff and flooding of mine waste tailings have left surficial soil deposits of mine wastes in residential properties along the lower part of Bingham Creek. Much of the contamination was located in or near the Bingham Creek Channel, which had much of the contaminated mine wastes removed from the channel during Phase II actions in 1993-1994. The worst known contamination in residential properties was removed during Phase I in 1991 for soil lead levels  $\geq 2500$  ppm. These earlier removals also took care of most of the worst arsenic contamination, since the higher levels of arsenic have usually been found along with the higher lead concentrations. Other sources of contamination include wind-blown dusts from the contaminated surficial soil, disturbance of contaminated soils during construction or other development activities, irrigation canals that had the ability to transport contamination, and past practices of using contaminated channel contents and nearby soils for fill material in residential yards. Additional detail on sources and transport of mine waste contamination can be found in the Phase III Action Memorandum <sup>1</sup>, June 1995, with its appendices, and in the previous EPA Region VIII Action Memos with their PEAs for Phase I and Phase II actions.

■

## C. **Removal** Actions Performed by EPA

### 1. Phase I, 1991

Phase I of EPA Superfund investigations began in 1990 and removal actions **took** place in 1991 to remediate surface soils in about 50 residential properties between 3200 West and 2700 West that had soil lead levels  $\geq 2500$  ppm. Details of the contaminants being addressed, health **risks**, and removal actions can be found in the EPA Region VIII Superfund Action Memorandum <sup>2</sup> dated May 7, 1991, and in its Attachment C which contains the PEA <sup>3</sup>. The EPA's UBK (Uptake Biokinetic) model **was** used to estimate risks to young children of having blood lead levels exceeding 15.ug/dl (asserted then to be a more urgent level of concern than the 10 ug/dl health protective goal), using assumptions quite similar to those used in the Phase III IEUBK modeling effort: 100 mg/day soil ingestion apportioned as 45% outdoor soil and 55% indoor dust, 25% bioavailability based on East Helena data, and two dust concentrations based on soil-dust lead relationships observed at Midvale, UT, and at Leadville, CO (respectively,  $\text{dust Pb} = 0.735 * \text{soil Pb} + 191$  and  $\ln(\text{dust Pb}) = 0.435 * \ln(\text{soil Pb}) + 3.65$ ).

The conclusions were 1) that most risks were posed from exposures to lead in soils from yards with levels  $\geq 2500$  ppm, 2) that those exposures would result in risks of having the majority of young children exceeding 15 ug/dl blood lead (PbB), and 3) that the risks were considered to be in the form of an acute (single exposure season) hazard. Two other levels of soil lead ranges and **risks** were evaluated: 1000-2500 ppm and  $< 1000$  ppm. Lesser **risks** of elevated blood lead were estimated for these ranges that were still of chronic concern for the residences with 1000-2500 ppm and of minor concern for the  $< 1000$  ppm range of homes. Arsenic **was** not deemed to be an imminent health hazard in relationship to lead, but the large order-of-magnitude uncertainties surrounding the arsenic **risk** estimates were noted.

## 2. Phase II, 1993

Phase II of EPA Superfund investigations began in 1992 and removal actions took place in 1993 to remediate contaminated creek channel soils with soil lead levels  $\geq 2000$  ppm in or adjacent to Bingham Creek from 4800 West to Brookside Trailer Park just east of Redwood Road near the Jordan River. Details on the channel contamination, health **risks**, and removal action can be found in the EPA Region VIII Superfund Action Memorandum <sup>4</sup> dated Jan 28, 1993, and its Attachments C and D which contain a toxicological assessment <sup>5</sup> and the PEA <sup>6</sup>.

About two miles of Bingham Creek channel soils were randomly sampled during August 1992 in 85 zones (about 4000 sq. ft. each) between 4800 West and 3200 West, an area which **was** more densely populated with residences closest to the creek channel. Results showed that lead levels ranged from **290** to 23000 ppm and averaged 5660 ppm, while arsenic levels ranged from 19 to 890 ppm and averaged 200 ppm (distributions of these ranges of contaminant levels are graphed in the Phase II PEA). Children had unrestricted access to the creek and were observed and reported playing there frequently, and some areas in the channel had colored soil which could present an "attractive nuisance" to children who played in the creek. Risks were estimated for three potentially exposed residents: young children  $< 6$  years old, adults, and so-called *explorer* children aged 7 to 16 years old; and ranges of risks were determined based on ranges of average exposures and RMEs (reasonable maximum exposure). The UBK model **was** used to estimate **risks** to children for exceeding blood lead levels of 10 ug/dl, using default assumptions except for the dust to soil relationship which was adopted from Midvale, UT, as in the case for the Phase I PEA (dust Pb =  $0.735 \times \text{soil Pb} + 191$ ).

EPA concluded that exposures to channel contaminants in about half the zones posed excessive **risks** for arsenic (noncancer childhood hazard quotients  $> 1.0$  and adult cancer **risks**  $> 1 \times 10^{-4}$ ) and for lead to children (blood lead  $> 10$  ug/dl), with lead being the contaminant of major concern. Considerable uncertainties (bi-directional) were pointed out in the PEA. "4-

## D. **Current Efforts** to Address Remaining Contaminated Materials

### 1. Superfund Risk Assessment Approach

Region VIII toxicologists generally prefer the use of good site-specific data and defensible science over default assumptions and modeling to quantitatively assess health risk <sup>7</sup>. Knowledge of contaminant characteristics and presence (locations and amounts), realistic exposures of populations, and background contaminant levels from comparable reference areas are key elements in quantitating risks with the minimal amount of uncertainty possible. The four steps used in **risk** assessment as recommended by the National Academy of Sciences in 1983 are generally followed by EPA. CERCLA (1980) and SARA (1986) are the laws governing Superfund activities, while the revised 1988 NCP provides the regulations for Superfund. The 1989 Risk Assessment Guidance for Superfund (RAGS), Vol. 1, **Parts A, B, and C**, is the main guideline for the conduct of **baseline** risk assessments; whereas, the **RAGS**, Vol. 2, and the 1992 **EPA Framework** document are the main guidelines for assessing ecological **risks**. Other **EPA** guidance, technical information, and policy statements are also employed; such as, the 1991 Default Exposure Factors, the IRIS database on toxicity reference values and carcinogen slope factors, and the 1992 Calculation of the C-Term.

Data should be scientifically sound (representative exposure-based sampling, adequate detection limits for valid analytical methods, acceptable quality assurance / quality control and chain of custody), and results should be defensible with sufficient statistical power to help give reasonable assurance that risks are not being overlooked if they actually exist. Superfund baseline risk assessments should establish: 1) current baseline (**risks** that would exist without remediation) and future risks, 2) cause-and-effect relationships between contaminants and **risks** to **health**, 3) quantitative PRGs (preliminary remediation goals) for each media and exposure pathway, along with uncertainties of those risk-based concentrations, and 4) an assessment of ecotoxicological **risks** to wildlife and habitat from exposures to environmental contaminants.



## 2. Kennecott **Risk** Assessment **Task** Force

To improve communications and broad involvement of all parties concerned with the risk assessment for the Kennecott properties, Dr. Eva Hoffman (the Region VIII RPM, remedial project manager for the site) established a **Risk** Assessment **Task** Force (RATF) that first met in the spring of 1992. Members were restricted to technical representatives and project managers from **EPA** Region VIII staff, Utah State and County officials, West Jordan city representatives, Kennecott and **ARCO** staff, and consultants; while lawyers, media; upper management and politicians were discouraged from attending, so that an environment could be established that would foster more uninhibited and objective scientific discussions regarding the contaminant sampling and data, exposures, and **risks** at this site. Several meetings were held during each year as needed to broadly address risk assessment issues and findings.

It is noteworthy that the site is **as** yet only proposed for listing **as** a NPL (national priorities list) site under the EPA Superfund program, and HQ **EPA** allowed Region VIII to approach the site in a more creative and streamlined fashion compared to usual Superfund sites and processes. **A** streamlining approach for both human and ecological risks at the site was devised, and it resembles **EPA's** new soil screening level (**SSL**)<sup>8</sup> guidance approach. The RATF usually had 10 attendees and over 25 at some meetings that were held on major technical items. All parties have been involved in gathering and presenting data and other information that was used to assess health **risks** at the site, especially for Bingham Creek -- Phase III. Compared with other Superfund sites, this area has had an extensive amount of site-specific data generated and evaluated in a relatively short time that has served to better define and act upon health risks to exposed persons. The **RATF** was very useful in evaluating site information, and such a process should be employed at more NPL sites..

## II. ENDANGERMENT ASSESSMENT OF HEALTH RISKS

### A. Hazard Identification

#### 1. Nature and Extent of Soil Contamination

An accumulation of relatively comprehensive soil sampling to delineate mine waste contamination has occurred in the Bingham Creek area since the Utah Department of Health (UDOH) initially collected and analyzed 110 soil samples from near the base of Bingham Creek to the Jordan River during 1990. The sampling was part of the Superfund Site Investigation (SI) and showed lead concentrations in the channel to be as high as 30,000 ppm and in residential soils as high as 12,000 ppm with elevated arsenic levels that correlated with the lead levels<sup>2</sup>. EPA and the UDOH also collected 20 background samples near Bingham Creek for analysis of lead (Pb), arsenic (As) and cadmium (Cd), and found levels of about  $110 \pm 68$  ppm Pb,  $11 \pm 5$  ppm As, and  $0.7 \pm 0.3$  ppm Cd<sup>3</sup>.

During December 1990 through January 1991, the EPA Emergency Response Branch (ERT) along with the Bureau of Reclamation (BOR) and the Ecology and Environment, Inc. Technical Assistance Team (TAT) collected about 1000 more soil samples from residential properties along Bingham Creek from 4000 West to just east of Redwood Road in the Brookside Trailer Park area. Fifty-six properties comprising about 42 acres of residential land were found to be contaminated with surface soil lead  $\geq 2500$  ppm, and arsenic was found at levels of about 3% that of lead<sup>2</sup>. The Phase I PEA further analyzed these results statistically and by segregating them into neighborhoods and into higher ( $> 2500$  ppm), medium (1000 - 2500 ppm) and lower ( $< 1000$  ppm) levels of lead for assessing differential health risks<sup>3</sup>.

During August 1992, EPA's ERB with the BOR and E&E TAT randomly selected from systematically selected locations in the Bingham Creek channel from 4800 West to 3200 West

for collection of 85 composited soil samples that were analysed for Pb and As. Results showed that lead levels ranged from 290 to 23000 ppm and averaged about 5600 ppm, while arsenic levels ranged from 19 to 890 ppm and averaged about 200 ppm, with As having a fairly good correlation with lead levels (calculated to be about a **4% As to Pb ratio**)<sup>5</sup>.

From August to October of 1993, the University of Cincinnati (UC) **along** with the Salt Lake City-County Health Department collected multimedia residential soil and dust samples for an Environmental Health Lead Study (EHLS) and a childhood urine arsenic study that involved 907 families and about **1300** subjects; wherein **927** children were screened for Pb and 696 children were screened for urine **As** levels. The **EHLS** sampling extended ½ - 1 mile on either side of Bingham Creek from the western city limits eastward to the Jordan River.

While this was an extensive sampling effort that generated much useful information, it did have limitations for **EPA Superfund risk** assessment purposes, mostly since the EHLS area incorporated properties well beyond (with little or no contamination) those with elevated levels of metal contamination of health concern per **EPA** criteria ( $> 400$  ppm Pb), as demarcated by Phase I soil analyses which evaluated perimeter soils in an attempt to define the extent of mine waste contamination in surface soils near Bingham Creek. The **EHLS** design also excluded properties that did not have resident children, and these locations would **still** be of potential future health **risk** concern for **EPA** in evaluating environmental protectiveness of contaminated soils. Other possible limitations included the fact that two soil removals had taken place before most sampling, and that the subjects were reasonably aware of contaminant hazards and may have taken advisory steps to reduce contact with soil (such as vegetating bare areas, keeping dust down in homes, restricting children's access to the creek and washing their hands better, etc.). Under these conditions, though, the **EHLS** reported that **72** (6.9%) of about 900 homes evaluated had one or more samples with Pb  $> 400$  ppm and that **20** (1.9%) were found with at least one sample  $> 1000$  ppm Pb; also, arsenic levels were reported as "quite low" with **.3%** (**3** homes) yard soil **As**  $> 230$  ppm and **5%** (**46** homes) of yard soil **As**  $> 95$  ppm<sup>9</sup>. ||

It should also be noted that at this time, the complete data analyses and final reports of this EHLS work have not been obtained by EPA. A good effort was made to obtain/provide most of the relevant data and results from this work for incorporation into this EA and into EPA's 1995 Action Memorandum; however, EPA does not have all the data and details of the samples and results, so there remain considerable gaps in EPA's ability to accurately and fully assess the contamination found during this EHLS. Even so, EPA's toxicologist believes that sufficient information of good quality from this work is available to credibly use it to help define contaminant levels, exposures to most residents, and risks to much of the populace.

Further, because of the limitations (most subjects were not highly exposed, homes without children were excluded, recent soil removals in study area, and publicity impacts) noted above with this EHLS data set, EPA and the RATF focused their evaluations of contamination and exposure-based risks on smaller subsets from the larger EHLS data set. The residential areas surrounding Bingham Creek were prioritized as to the likelihood of having potentially elevated soil lead levels of health concern (based on results from the Phase I sampling and from preliminary soil sampling results in the EHLS). Dr. Gerry Henningsen submitted the criteria and prioritized locales for selecting a smaller subset of the entire EHLS data set (excluding properties having soil removed by EPA) to evaluate for more relevant exposures and potential health risks<sup>10</sup> (see EA appendix). It was agreed by the RATF principle members that such a subset should have about 200 residences in order to make more meaningful scientific evaluations of the results. Dr. Robert Bornschein of the UC selected 209 homes with 272 children aged  $\leq 6$  years by using the EPA criteria. This "exposed home" subset was later *trimmed* to 246 qualifying children for further PbB and other analyses<sup>11</sup>. Finally, a smaller subset of the 209 exposed subset was selected from homes that had any soil or dust lead  $\geq 400$  ppm, resulting in 20 homes with 25 children from the entire EHLS data set to focus upon for evaluation of health risks from Pb exposure. Of these 20 homes identified by the EHLS, only about 10 had average soil lead levels  $> 500$  ppm. Multimedia contaminant levels in the smaller and more relevant datasets can be found on pages 22, 28 and 39 of a

recent UC report <sup>12</sup> (see EA appendix)

## 2. Contaminants of Concern

The sampling, analyses, and reports described above have determined that lead and arsenic from mine wastes are the contaminants of concern and, as such, are the major *risk drivers* at this site. Levels exist in residential areas that substantially exceed nearby background levels that average about 110 ppm Pb and 11 ppm As in surface soils <sup>3</sup>. Contaminant levels are also present above estimated levels of reasonable safety (draft EPA soil screening levels and EPA Region III Risk-Based Concentrations) of 400 ppm soil Pb and 40 ppm soil arsenic ( $1 \times 10^{-4}$  extra cancer risk) <sup>8</sup>.

## 3. Metals Characterization

Because there is reason to believe that Pb and As from mine wastes in contaminated soils are in geophysical and chemical forms that are likely not as readily bioaccessible as the more soluble molecular-sized salts (e.g., lead acetate or sodium arsenate), several geochemical analyses of Bingham Creek contaminated soils have been conducted. Researchers from PTI <sup>13</sup> (Boulder, CO) performed a limited analysis of creek channel soil samples, as did Cannon Microprobe (Seattle, WA) <sup>3</sup>, but little definitive scientific credence was afforded these evaluations by EPA due to limitations in methodologies and scale.

More extensive characterizations of the metal contaminants in channel and residential soils were performed by Dr. John Drexler at the University of Colorado, Boulder, CO; where he measured stoichiometry, mass, frequency and the **matrix** of metal bearing particles. Excerpts from an EPA report of relevant Bingham Creek geochemical findings via use of an electron microprobe method are included in the EA appendix <sup>14</sup>. Two representative composite samples were sieved to <250 um, one collected from the creek channel and one

collected from yards with known soil lead > 400 ppm. The **small** sieved sub-samples were the subjects of the main analyses; additional individual sample analyses of metal phases were also performed by Dr. Drexler for **EPA** (preliminary graphical results <sup>14a</sup> attached in the appendix). The two speciated composite soils were also used for testing in bioavailability studies which are described later. The channel yard composite sample had **6330** ppm Pb and 149 ppm *As*, while the yard composite had 1590 ppm Pb and 51 ppm *As*.

Results of metal characterization analyses showed: 1) **small** particle sizes, with about half the particles < 5  $\mu\text{m}$  measured **as** the longest diameter, 2) lead was predominantly in **liberated** anglesite ( $\text{PbSO}_4$ ) in the yard sample and in **liberated** phosphate ( $\text{PbPO}_4$ ) in the composited channel sample, with cementing by Mn-oxide and Fe-sulfate particles. These results suggest that finding small particles (more surface area per unit mass) and more soluble lead salts (vs insoluble lead ores such **as** galena,  $\text{PbS}$ ) in liberated particles would tend to augment bioaccessibility, while the larger and cemented (unexposed) particles would tend to retard bioaccessibility. Final conclusions on relative bioaccessibility of these soil metals will not be possible until the final results of the metal speciation tests and accompanying bioavailability studies in young pigs are completed by **EPA** in 1996 for a suite of NPL sites.

## B. Dose-Response Evaluations of Toxicity

### 1. Susceptible Populations

Residential children under 7 years old are the most susceptible population to the toxic effects of lead, which is the main contaminant of health **risk** concern, and to arsenic **as** a secondary concern for noncancer effects. There are lesser causes for health concern to older children and adults who are less susceptible to these metal toxicities, but these groups are presently **not without** exposures and potential **risks** in the Bingham Creek area. The main reasons for higher susceptibility of the young children involve **behaviors** and **physiology** that

enhance their exposure and toxicity, as described below:

1) Toddler aged children experience substantial incidental ingestion of house-dust and residential soil through normal hand-to-mouth contact and outdoor plus indoor playing activities. Such activity is expected to transfer small particulates (250 microns or less in diameter) from hands, toys, or other objects into the mouth and subsequently into the digestive system. Low gastric pH can help solubilize lead from the soil and dust particles. 2) When dissolved Pb enters the small intestine of children and other young mammals, it can presumably be actively transported (as well as passively absorbed to a lesser extent) into the blood via the calcium active transport system<sup>15</sup>. This active transport availability coupled with the large surface area of the absorptive intestinal surface have the potential to kinetically drive insoluble Pb (at equilibrium with soluble lead) into solution for even greater uptake as Pb ions are actively moved out of the intestinal lumen<sup>16</sup>. 3) Young children generally receive a higher dose of metals than do older children and adults, since young children's soil/dust intake is as great or greater while they weigh less. This higher dose becomes important for lead neurotoxicity and arsenic noncancer effects which can arise from shorter, less than lifetime, exposures. 4) Young children also have more susceptible nervous systems which are still developing and are vulnerable to impairment from excessive lead exposure. If enough lead crosses the blood-brain barrier in young children, lead can interfere with normal mental development.

## 2. Lead


Several scientific studies have found that populations of children between ages 0 to 3 years old who have blood Pb levels  $\geq 10-15 \mu\text{g}/\text{dl}$  are at increased risk for adverse effects such as decreased cognitive function, compromised bone growth, hearing dysfunction, and behavioral alterations that may be irreversible. Children up to 7 years old are considered to be at higher risk to neurotoxicity of lead. Lead from mine waste that is able to be absorbed

into the blood stream should have the same ability to cause toxic effects as seen with other forms of absorbed lead; any difference in dose-response for similarly exposed individuals should largely be a function of differing bioavailability due to geochemical form and animal or human physiological factors (gastric pH, GI transit time, age, genetics, diet, etc.). Lead is also classified by EPA as a **B2** Probable Human Carcinogen<sup>17</sup>, but the uncertainty is quite large and the potential neurotoxic effects in children are generally of a much more realistic and serious concern. A Reference Dose has not been generated for lead by EPA; instead EPA relies upon the **IEUBK** model to predict a toxicologically safe level for lead exposures in children<sup>18</sup>. The proposed soil screening level for acceptably safe levels of lead in soil under default exposure conditions is 400 ppm<sup>8</sup>. A current and detailed review of the intricacies of lead toxicity can be found in the EPA Lead Workgroup document<sup>19</sup>.

### 3. Arsenic

Arsenic is a toxicological hazard both as a potential carcinogen (lung, internal organ, and skin cancers) and as a noncarcinogen (at high and/or chronic doses being able to damage skin, blood vessels, and the gastrointestinal tract)<sup>17</sup>. The current RfD presented by IRIS is 0.3 ug/kg/d (ppb) with a *medium* level of confidence regarding dermal and vascular effects. The unit risk for arsenic in drinking water is  $5 \times 10^{-5}$  excess cancer risk for each ug/l As. The majority of cancer risk information has come from studies reported by Chen on Taiwanese who drank water with elevated levels of inorganic arsenic. The proposed EPA soil screening level for acceptably safe levels of arsenic in soil under default exposure conditions ranges from 0.4 to 40 ppm, based on excess cancer risks of either  $1 \times 10^{-6}$  to  $1 \times 10^{-4}$  (1 in 10,000).

### C. Exposure Evaluation

Elevated blood Pb (PbB) levels in young children are thought to arise primarily from incidental ingestion of Pb in house-dust (even more-so when contaminated by leaded paint) 



and in residential soil by young children engaged in normal hand-to-mouth activities<sup>18</sup>. Such activity is expected to transfer small particles (250 microns or less in diameter) from hands, toys, or other objects into the mouth and subsequently into the digestive system. If the small particles contain lead in sufficient quantities, enough lead can be absorbed into the blood stream to result in unacceptably high blood Pb levels. Recent literature supports that soil Pb is bioavailable, but probably to varying degrees depending on several factors including: Pb chemical species, soil matrix, particle size, and physiological factors such as gastric pH and active transport present in young mammals. Currently, acceptable levels of lead exposure are based mainly upon predicted blood lead levels in young children, which should not exceed 10 ug/dl in more than 5% of exposed children (correspondingly, a childhood residential yard exposure unit shouldn't pose more than a 5% chance of a young exposed child's PbB exceeding 10 ug/dl) according to EPA<sup>20</sup>.

### 1. Exposure Pathways

Residents who live in the vicinity of Bingham Creek may be exposed to contaminants in the flood plain in various ways, including those listed below in expected order of significance:

- incidental ingestion of contaminated soil and dusts while children play or residents work in yards or homes located in the flood plain,
- ingesting metals taken up from contaminated soil by home-grown garden vegetables
- inhalation of dust particles eroded from contaminated media into air by wind or mechanical disturbances,
- incidental ingestion of contaminated surface water or sediment in low lying areas during storm water run-off events,
- dermal contact with contaminated soils or remaining contaminated sediments and tailings deposits while working or playing in the creek bed or flood plain, and
- ingestion of well water contaminated by metals leached from the waste materials into groundwater.

## 2. Blood Lead and Urine Arsenic Evaluations of Residents

### a. Environmental Health Lead Study Design

From August to October of 1993, the University of Cincinnati (UC) worked with the Salt Lake City-County Health Department to collect multimedia residential soil and dust samples for an Environmental Health Lead Study (EHLS) that tested blood lead levels in those area-families that had children. UC and SLCC simultaneously conducted a childhood urine arsenic study. The **EHLS** examined 907 families and about 1300 subjects, of which 927 children were screened for **PbB** and 696 children were screened for urine **As** levels; and extensive surveys were taken to record demographics, behavior, and other factors related to exposures to lead and arsenic from the mine wastes which contaminates residential yards. The EHLS sampling area extended for about  $\frac{1}{2}$  - 1 mile on either side of Bingham Creek, starting from the western city limits eastward to the Jordan River<sup>9</sup>.

EPA Region VIII had the opportunity to review and comment on this design, and several EPA technical concerns were addressed. This EHLS **was** perhaps one of the better conducted and more comprehensive blood lead studies to-date on mine waste exposures, but it still had serious problems that precluded its ability to confidently quantitate risk (the end product required by EPA Superfund). While the design was thorough in data collection and QA/QC, it was inherently limited (due to the small sample size of more highly exposed children) by not initially focusing upon residences with actual elevations of soil lead and arsenic that were high enough to expose residents to **toxic levels** of the contaminants. **Thus**, in EPA's scientific opinion, the majority of data were collected from relatively clean **background** residences where results were **near or below detection limits**, which greatly hinders the ability to extract out the potential problems experienced by a minority of the contaminated residences (essentially diluting and masking those fewer potential exposures and risks). In

other words, EPA views the EHLS study design most appropriately as largely a “background” PbB study, in reality. Under other theoretical site conditions where the majority of sampled residences would have been quite heavily contaminated (homogeneous), this design would have been nearly ideal; but at this site, the design did not account enough for the distinct differences (heterogeneity) in “exposures” in the study area and thus did not hone in on the true problem areas.

#### b. Residential Multimedia Environmental Sampling Results

The overall **EHLS** results showed that only a small percentage of the nearly 1300 subjects had elevated contaminants in nearly 900 residences tested (see above nature and extent discussion)<sup>9</sup>, which is good information to the larger Bingham Creek community for overall public health purposes. However, the relative levels of environmental contamination became substantially higher and more of a health risk concern as one sequentially focused in upon 1) the *more likely exposed areas* comprised of a subset of 209 homes (out of the 900 total homes) and 2) then focusing down further to the 20 homes (a subset of the 209 homes) with any lead level > 400 ppm or 3) the 10 homes from the prior 20-home subset ~~with~~ average soil Pb > 500 ppm (see **Figure 2-1** on next page). These latter 10-20 homes with higher environmental levels of Pb and As contamination were the extent of the hazardous waste problem identified out of the nearly 900 homes sampled by UC in the EHLS study; but one must note that there are *numerous other properties*, with known contaminant levels approximating or exceeding the levels found in these latter homes, which were not included in the EHLS study for various reasons only known to the UC investigators at **this** time.

As mentioned before, while EPA has obtained much of the pertinent EHLS data, EPA has not received all of the results or a final report. Furthermore, there has not been enough time since the **EHLS** results were presented at the May **23 RATF** meeting to thoroughly analyze all aspects of the contamination and effects found by UC investigators. Current

details of the larger and the several smaller subsets of the EHLS environmental results can be found in the attached May 23 RATF meeting report <sup>12</sup> that was presented by UC. Summaries of those results are shown in the table below for the purpose of demonstrating the probable misdirection and misinterpretation created when trying to extrapolate findings from a mostly non- or under-exposed population to a much smaller group of residents which truly have elevated levels of metal contamination that begin to pose a substantial health risk concern. The tabulated values have units of ppm, and some of the percentiles were calculated by EPA scientists when not provided by UC.

Measurement* (ppm Pb)	Large SEM Data set Pg 16A	Exposed 209 Homes Pg 22	20 Home Subset Pg 28	10 Home Subset Pg 39
# Children:	690	246	25	17
<b>Exterior soil</b>				
GM	71	86	452	870
GSD	2.27	2.53	2.04	-
Max	1414	1414	1414	1414
95%-tile	322	435	1844	-
Min	16	19	82	517
5 %-tile	13	17	111	-
<b>Interior dust</b>				
GM	117	128	265	334
GSD	1.78	1.75	2.00	-
Max	1451	1451	1451	645
95%-tile	416	448	1060	-
Min	13	18	93	99
5 %-tile	33	37	66	-

abbreviations: ppm = parts per million, or mg/kg; Pb = lead; SEM = structural equation model; GM = geomemc mean; GSD = geometric standard deviation; Max = maximum reported value; %-tile = 95th or 5th percentile of the distribution of data for that subset; Min = minimum reported value; - = not calculated.


As can be seen by the above table, the remaining Phase III contamination problem is relatively small when subsets of more highly contaminated homes are segregated from the larger data set. However, as cannot be seen from the **EHLS** datasets, there are approximately 75 known properties with average soil lead levels > 500 ppm as determined from past sampling that was located within the **EHLS** study area. Also, a few children under 7 years old who live at these properties were removed as subjects from the larger **EHLS** datasets for uncertain reasons. Further, the above table clearly shows that there are two distinct populations in the **EHLS** area in terms of environmental contamination of residences with mine wastes: 1) the majority with minimal contamination at or near background levels, and 2) a minority with elevated levels of metal contamination which are cause for potential health **risk** concern. It depends on the scale one wishes to examine the **EHLS** dataset as to what significance is placed on the results; i.e., rather misleadingly low percentages of PbB elevations are derived ~~from~~ reports on the whole dataset, vs more serious problems with PbB levels are evident when one focuses in on the subsets of homes with potential health **risk** problems due to elevated contaminant levels. It is this smaller set of homes that has always been of primary concern to **EPA**, and it is this group that may require some actual **risk** reduction via Superfund actions. Since the rest of the homes are not contaminated at high enough levels to pose health risks of concern, they are outside the scope and interest of Superfund even though they were subject to intense study by UC-SLCC. These "other" homes with low or near-background levels of Pb are viewed **as** good reference information to serve as a baseline for comparisons to homes Contaminated at higher levels of health risk concern.

### c. Blood Lead Results

A total of 6 PbB levels > 10 ug/dl out of 972 children (uncertain if ages are < 72 or < 84 months) were reported by UC<sup>9</sup>. The levels > 10 ug/dl were: 10.4, 13.1, 11.5, 11.9, 11.4, and 23.5 ug/dl **as** reported by UC at the May 5, 1995, RATF meeting. The first two

children exceeding 10 ug/dl were siblings with soil Pb = 705 ppm and dust Pb = 209 ppm and with possibly some lead in interior paint as an added source of environmental Pb. The middle two children of the 6 were not siblings and had no apparent environmental sources of lead to account for their PbB elevations. The last two of the 6 children were siblings with soil Pb = 1007 ppm and dust Pb = 280 ppm and also with probably some lead in interior paint and in exterior paint (perimeter soil Pb = 2291 ppm) as added sources of environmental lead. It was later learned that this property had removal performed in 1991, but the owners had reportedly disturbed the remedy by landscaping the lot.

Out of the 209 exposed home data subset, there were 14 children with PbB > 5 ug/dl and 4 children > 10 ug/dl (the 2 pairs of siblings described above, but the last two siblings were from a home that was remediated in 1991 and so they were dropped from further SEM (structural equation model) analyses along with a third older sibling who had a PbB = 6.2). In the 20-home subset with 25 children where "any" soil level was > 500 ppm (= 21 homes and 28 children if include the 3 siblings just discussed) and in the smaller set of 11 homes with 17 children (= 12 homes and 20 children if include the 3 siblings just discussed) having "average" soil Pb > 500 ppm, there were only the first two siblings described in the previous paragraph that exceeded 10 ug/dl PbB (= 4 children total if ignore the 1991 remediation). See pages 39-42 of the attached May 23 UC report to the RATF<sup>12</sup>.

Also included in the UC May 23 report was a summary of the QA/QC for the **EHLS**. Inter-laboratory comparisons of 93 samples with CDC were very good. A total of 75 blind field standards at a 5% rate showed good accuracy and read slightly higher compared to the CDC nominal values. Nearly 200 laboratory bench known controls in the range of the EHLS samples showed good consistency with about 0.5 ug/dl standard deviation for the 1.8 ug/dl and the 4.1 ug/dl standards that were run in duplicate. An additional 97 bench reference blind controls at 4 concentrations also showed good accuracy with slightly higher readings than the nominal values. No data was provided on trip or lab blanks to check for contamination, but 

the blind field standards appeared to be uncontaminated by way of their accuracy. In all cases, as expected, the % coefficient of variation ( $CV = SD \div \text{mean}$ ) increased as the standard values approached the lower levels of method detection limits for lead in blood.

A later submission of the method detection limit (MDL) and control charts over time were submitted via memo on June 8, 1995, from Dr. M. Kathryn Brown at the UC. The instrumental DL (IDL) had been verbally reported by Dr. Bornschein on May 5 as 0.6 ug/dl. Dr. Brown reported the average MDL as 1.4 ug/dl  $\pm$  0.4 ug/dl, with a range of 0.9 to 2.1 for 50 runs. The control charts for the CDC standards showed slightly high results for the 1.8 ug/dl standard and slightly lower results for the 4.1 ug/dl standard.

No method "quantitation" limit (**MQL**) was provided, and it may not have been established. UC reported that the **EPA** method from 40 CFR (7-1-92 Edition) was used to determine the MDL (3 SDs above the instrument blank signal). The MDL simply identifies that the analyte is "present", but it cannot be confidently quantitated at this signal level. This approach is fine, but a problem arises with any analytical method when results are obtained below the method **quantitation** limit, which is generally defined as 5 to 10 SDs above the blank signal. In such cases, there are several procedures offered by **EPA RAGS** to estimate what the values might actually be when the instrument and method give results between the MDL and **IDL**. For the UC **EHLS**, the lowest MQL would be estimated as about  $5 \times 0.4 = 2.0$  ug/dl. Variability has also been defined as about  $\pm 0.5$  ug/dl (1 SD) in this range.

The above QA/QC is good, but study findings are hampered by the many low sample results that are near or below the estimated MQL and reported MDL. In addition, the EHLS results were reported out to 2 decimal points, but it is doubtful that the method can generate accuracy to within 2 decimal points at these low analytical levels. **Based** on the measurements of accuracy with %CVs that ranged from about 12-33% in the EHLS sample PbB range, coupled with the average MDL SD of 0.4, these would argue against such purported accuracy; 23

it appears that at the GM of the PbB for the study that the results are only accurate to the nearest whole digit (no decimal points). As in past EHLS studies, it appears that results below the MQL and below the MDL were treated as quantitatively as were results above the MQL. This is improper as well, and those non-quantifiable values should be handled differently as is scientifically valid and in agreement with EPA RAGS and Data Usability Guidance for Superfund, 1992.

In 1990 a voluntary medical screen of PbB levels was conducted for about 100 children aged <7 years in the general Bingham Creek area. All results were < 10ug/dl, but the distributions were quite higher than in the 1993 EHLS study<sup>9</sup>. A major limitation with this study is the absence of residential soil Pb levels to correlate with the PbB levels so that exposure related cause and effect can be established.

#### d. Urine Arsenic Results

Little formal results on urine arsenic were provided to EPA by UC<sup>9</sup> or by Kennecott. The partial datasets that EPA had access to were sorted by Life Systems consultants for urinary arsenic levels above 10 ug/l (ppb). We found 124 urine samples with As at levels between 10 and 35 ug/l in our data set. Supposedly, 696 children had urine samples analyzed. 19 of the 124 children with the higher urine arsenic had As levels > 100 ppm in some media, with a few play areas containing around 500 ppm As; no correlations were run between urine As levels and media As levels. In the subset of 20 homes with average soil Pb > 400 ppm, there were 6 homes with average soil As > 100 ppm. The Phase I sampling estimated a 3% correlation of soil As to soil Pb levels, while in Phase II that relationship was about 4%.

### 3. Bioavailability Studies in Animal Models

EPA's default bioavailability estimate for lead is 30% absolute uptake (60% relative to



oral lead acetate) and for arsenic is 85% absolute uptake, assuming there are no site-specific data to justify alternate bioavailability factors. Two animal bioavailability studies were conducted at this site. One was performed by PTI Environmental Services, but review of the data at the May 5 RATF meeting showed that the design was too limited and results non-linear, which precluded EPA from being able to use the study (attached synopsis in appendix); however, PTI staff indicated that they had additional results that enabled better interpretation of the data, and that the conclusions were similar to EPA's estimates from the pig study.

EPA used the young juvenile swine model to evaluate relative (to soluble lead acetate) oral uptake of two representative composited soils, one resembling channel soils and the other typical of residential yard soil. Both were characterized as to particulate size, stoichiometry, and matrix, as described previously. The protocol from the EPA Project Manual for the pig study is attached, along with the final detailed experimental design for the bioavailability studies of the Bingham Creek soils. Also included is a spreadsheet with the results and a graph and calculations that show the bioavailability results. The results showed that the channel soil with higher concentrations of mostly anglesite had a blood bioavailability of about 17%, while the residential soil with lower concentrations of mostly Pb phosphate had a higher blood bioavailability of about 19%. Tissue bioavailability was also calculated (data and results not shown, but presented at the May 5 RATF meeting) for the two soils, which ranged from about 10% to 13% for liver, kidney and bone samples. Quality control showed that the data collections, processing, analyses, and management were reasonably good (scientifically sound). Some repeat analyses had to be performed due to slight contamination of newly prepared matrix modifier solutions used to dilute and prepare blood for analyses.

Because of the uncertainty as to which tissue is best to calculate bioavailability, the RATF agreed to use the blood level of 19% as an estimate akin to a RME value and to use the rough average for both tissue and blood bioavailability of 15% as an "average" bioavailability estimate. This would produce a range of PRGs in the UBK model when run at the two uptake

25

values of 15 % and 19%.

Some partial (days 4 and 7 for control pigs, and day 7 for high dosed pigs) arsenic data were available at the date of this EA report. The study collected urines on days 0, 7, and 14, but the entire urine As analyses have not been completed. Included in the bioavailability package are the preliminary results of the urine As generated bioavailability; the best initial estimate is that about 15% of the As was absorbed as of day 7 per the spot urine tests.

#### 4. Vegetative Uptake and Exposure Survey

Life Systems conducted a garden vegetable and soil sampling survey<sup>22</sup> for EPA during 1993 at 24 residences in Bingham Creek, with 17 gardens sampled in contaminated areas and 7 gardens sampled that had been remediated by EPA in 1991. The contaminated areas had the highest levels of soil lead, while the removal areas had the lowest; soil Pb ranged from 14-4100 ppm and soil As ranged from 5-67 ppm. A questionnaire was filled out by each gardener to study consumption and handling of garden produce. The highest lead found in a vegetable was 0.5 ppm and the highest arsenic was 0.07 ppm. The study concluded that on average there was no substantial contamination of garden vegetables to cause either cancer or noncancer health concerns. There is a possibility that much higher than normal vegetable consumption could contribute to an incremental risk from area exposures to soil Pb and As. At the highest areas of contamination, children who would eat a lot of root crops could be at increased health risk from this route of exposure.

#### D. Risk Characterization

##### 1. Integrated Exposure Uptake Biokinetic (IEUBK) Model

Life Systems performed the UBK model runs for EPA and provided two comprehensive

packages at the May 5 and the May 23 RATF meetings. The package from the May 5 meeting showed model inputs and outputs on a flow diagram, calculations of the site-specific GSDs for measured PbBs, dust to soil ratio calculations adjusted for mass loading, and preliminary UBK model predictions. The package from the May 23 meeting refined the initial UBK input estimates and outputs based upon clarifying information from the UC. A final report of all the UBK modeling work by Life Systems is attached <sup>23</sup>.

a. Default and Site-Specific Input Values

The 209 home database was *trimmed* per criteria established by the RATF, and **252** children were further evaluated by EPA ~~from~~ 171 residences; using similar criteria, UC arrived at a 209 home subset with 246 qualified children for their SEM (structure equation modeling) calculations <sup>12</sup>. Both the UC and Life Systems further evaluated the 20 home subset which was trimmed to 17 residences and **25** children by Life Systems.

The GSD derived From the larger dataset with about 768 children (from the two combined randomly split datasets of 384 each) was estimated at 1.56 while the **209** home GSD was calculated to be 1.43. These values were averages of the median and the weighted median values derived from a box model or 3-dimensional matrix method. The dust value was derived from a composite of ratios using different dust loading facotrs, and a best estimate equation was generated:  $\text{dust Pb} = 0.43 * \text{soil Pb} + 90$ . Water Pb was set at 1.4 ppb and the bioavailability ~~was~~ used at both the 15% and the 19% level. Air and dietary Pb were set at default values, and soil:dust intake ratios were used at the default ratio of 45%:55%. At the May **23** RATF meeting, it was decided to incorporate average dust Pb levels into the UBK model ~~outputs~~ since there was not greater confidence or justification to prefer either the site-specific equation or the constant average. A total of 24 model outputs as PRGs were run from these input values and at two age ranges: **0-72** and 0-84 months (the UC SEM ~~was~~ only run for children 0-72 months old). Individual **PbB** values were also predicted for each of the 209

homes and for the 20 homes to compare the observed vs predicted values as a cumulative frequency. These graphs showed that the predicted PbBs were lower than the observed PbBs at the higher levels of blood lead values.

#### b. Predicted Soil Levels that Prevent Excessive Risks

The UBK's predicted PRGs (soil levels of Pb estimated to keep >95% of exposed children PbB < 10 ug/dl) ranged from 815 to 1680 ppm from the 24 permutations of the UBK calculations. The *best estimate* was **1100** ppm (based on EPA's preferred estimates for bioavailability of 19%, a GSD of 1.43, and modeling 0-84 month old children) by averaging the two preferred estimates of about 900 ppm (when using the dust Pb equation) and of about 1300 ppm (when using the average dust Pbs). When a range was derived based upon the exact above scenarios but by changing only the bioavailability to 15%, a *second best estimate* from ranges of 1130ppm to about 1670 ppm equaled an average of **1400** ppm soil Pb.

## 2. Structural Equation Modeling (SEM) of Blood Lead

### a. Sources and Correlations of Soil Lead to Blood Lead

While the EHLS was an extensive sampling effort that generated much useful information, it did have limitations for EPA Superfund risk assessment purposes, mostly since the EHLS area incorporated relatively clean properties well beyond those with elevated levels of metals contamination of health concern per EPA criteria (>400 ppm Pb), as shown by Phase I soil analyses which evaluated perimeter soils in an attempt to demarcate the extent of mine waste contamination of surface soils near Bingham Creek. The EHLS design also excluded properties that did not have resident children, and these locations would still be of potential future health risk concern for EPA in evaluating environmental protectiveness of contaminated soils. Other possible limitations included the fact that two soil removals had

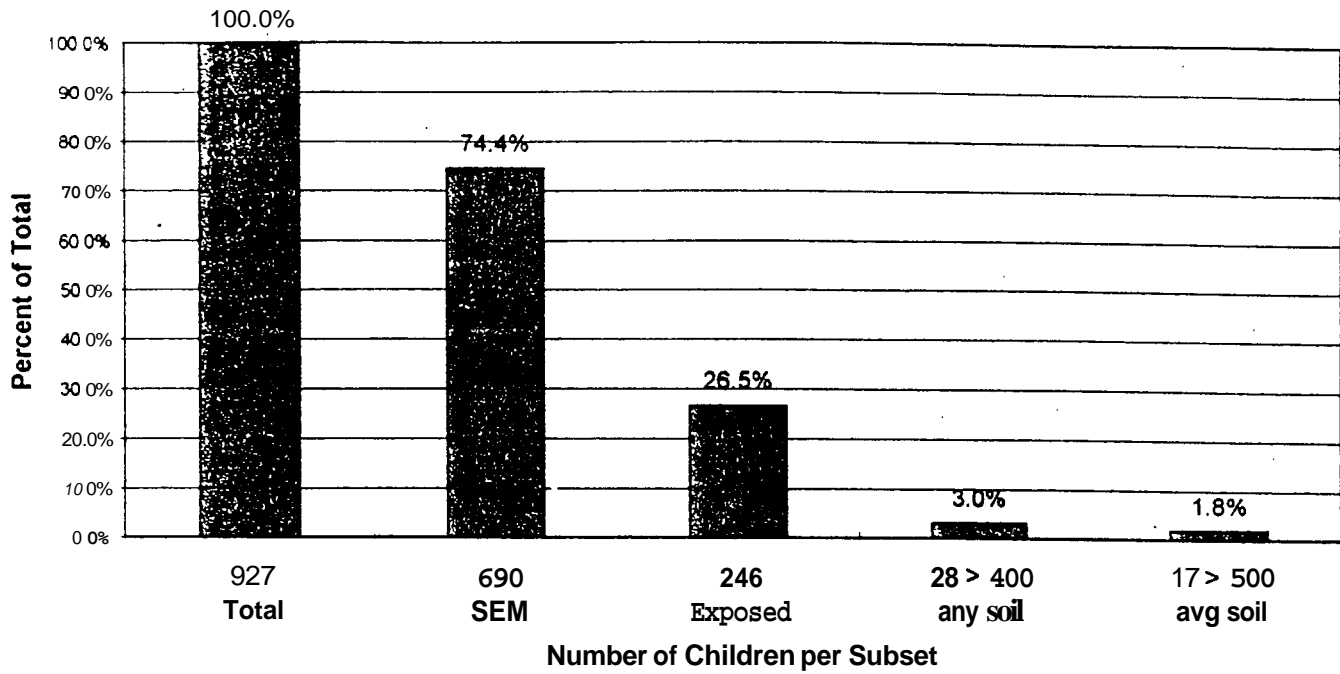
taken place before most sampling, and that the subjects were reasonably aware of contaminant hazards and may have taken advisory steps to reduce contact with soil (such as vegetating bare areas, keeping dust down in homes, etc.). Under these design and sampling conditions, the EHLS found 72 (6.9%) of about 900 homes evaluated with one or more samples of Pb > 400 ppm and that 20 (1.9%) homes were found with at least one sample > 1000 ppm Pb; also, arsenic levels were reported by UC as being "quite low", with 0.3% (about 3) of the yards containing soil As > 230 ppm and 5% (about 45) of yards having soil As levels > 95 ppm<sup>9</sup>.

#### b. Predicted Soil Levels that Prevent Excessive Risks

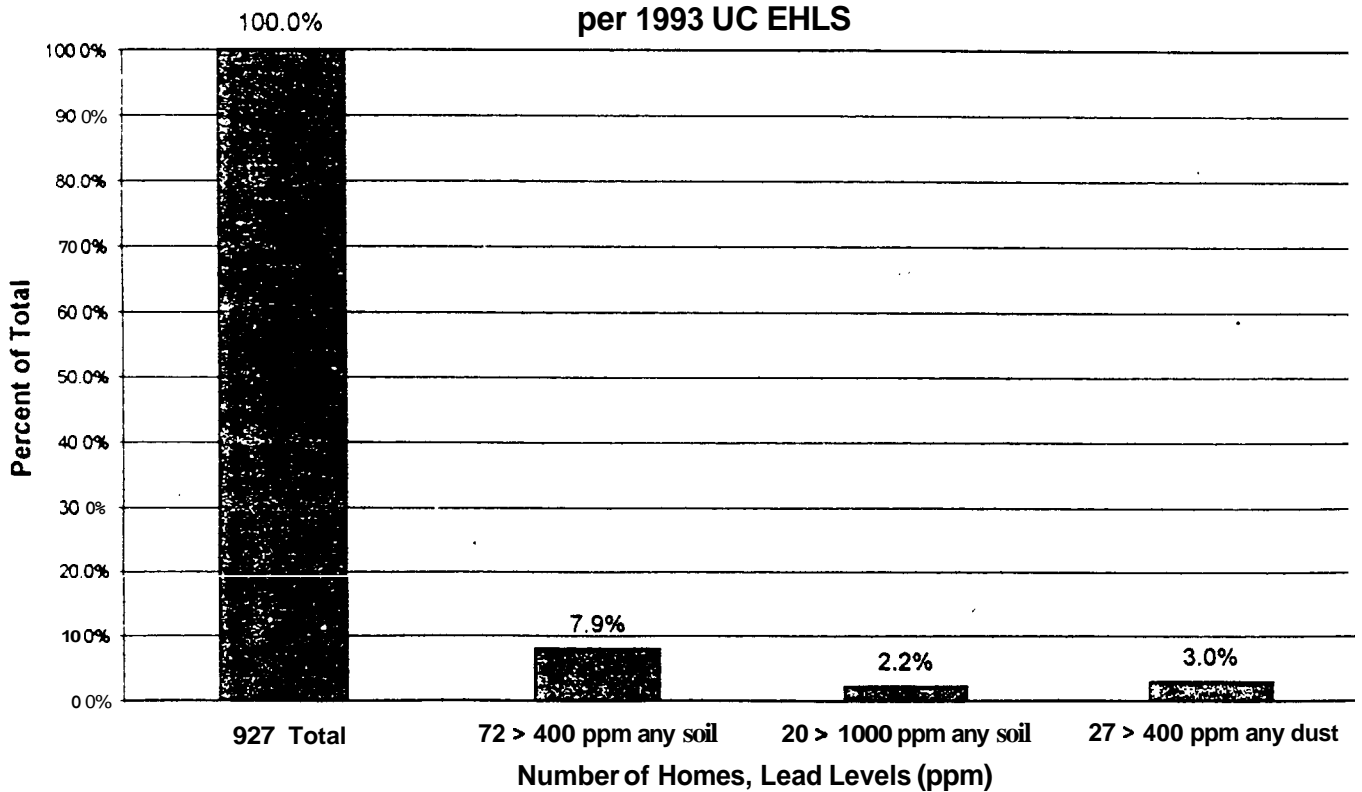
Estimates are between 2250 and 3000 ppm Pb based upon a forced regression model with subjective levels of protectiveness. Refer to the May 23 presentation<sup>12</sup> in the appendix for details of the SEM process. Because of the very low concentrations of Pb in media and in blood, the SEM did not have much success in predicting higher blood lead levels in future exposed children. It did an adequate job of characterizing the 1993 PbB levels, and the model appears to be more useful as a tool for identifying relative contributions of PbB from various "sources" as a form of a statistical sensitivity analysis. Because of the low PbB levels, there were problems with quantitation near the detection limits (DLs), which translates directly to problems with quantifying the results by giving diminished confidence to the SEM's accuracy and precision. In addition, the SEM's generated best fits of both the 209-home and 20-home subsets of "observed vs predicted" PbB data showed a slope of about 0.2, which could mean that the UC SE model consistently under-predicted blood lead values **5-fold**. If such a correction was made to the previously estimated PRGs of **2250** to 3000 ppm, then those PRGs would drop 20% back to about 450 to 750 ppm (see pages **27A** and 31 of the May 23 UC report<sup>12</sup>; included as **Figure 2-2** on the next page of this EA).

Figure 2-1.

Sample Sizes of Children in the 1993 EHLS



Relative Amounts of Residential Soil Contamination per 1993 UC EHLS



### 3. Strength of Scientific Evidence for Remedial **Risk** Reduction

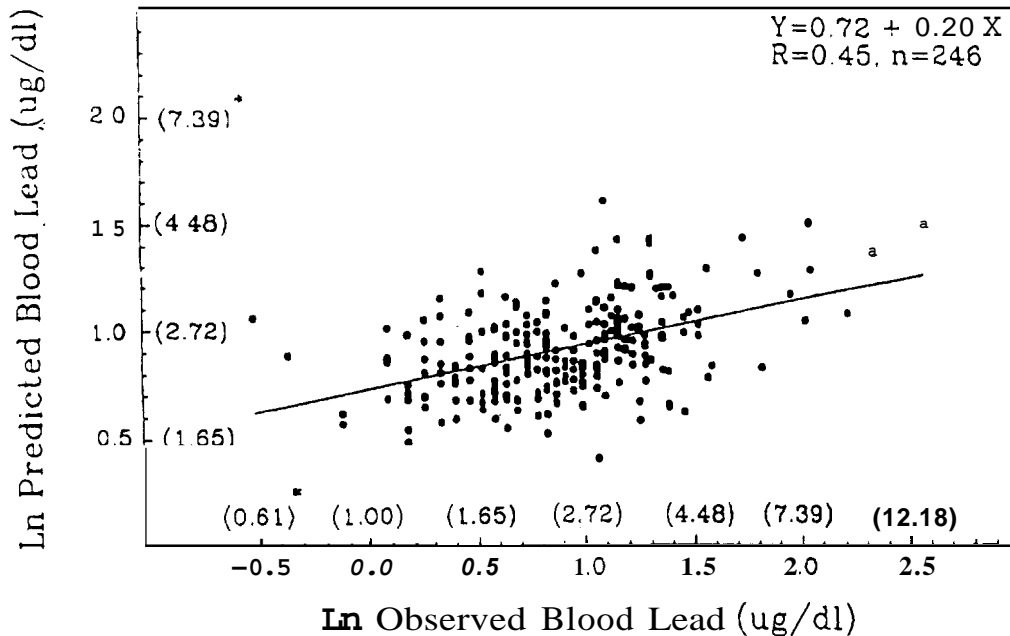
#### a. IEUBK, SEM, PbB, Arsenic, Vegetation, Publicity, etc.

The IEUBK model was given more strength and weight of evidence by EPA compared to the SEM, since the **UBK** model was composed of and built upon broader, repeated, and at least partially validated sets of data along with good verification of results for some NPL sites. The SEM was judged to simply not have the *resolutionpower* necessary to predict future PbB values **as well as** the UBK model can do that task.

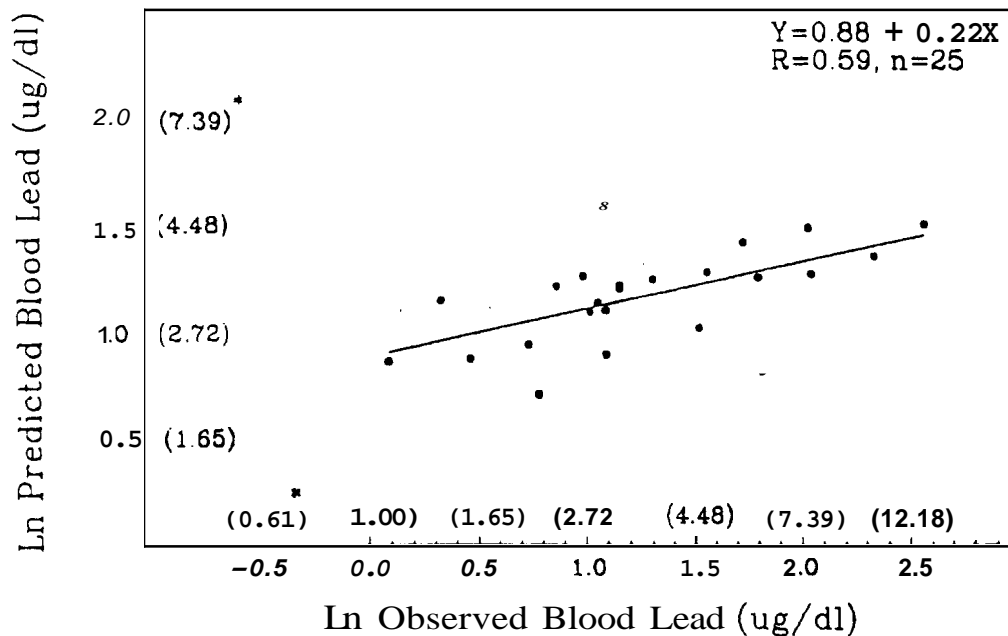
When comparing relative predictiveness of the two models (see Life Systems' report <sup>23</sup> pages 5-5 and 5-6 compared directly to the May **23** UC report's <sup>12</sup> pages 29 to 30 and **25-26**; these are included on the following pages as **Figures 2-3, 2-4 and 2-5**), it is readily apparent that the UBK model is superior in predicting the higher PbB values which are where the health concerns exist for **EPA** (even if the GM of the UBK model is not able to fit the poorly quantitative PbB results (due to MDL issues plus the general lack of exposure and the uncertain post-removal and publicity confounders). The cumulative frequencies shown by graphs for both models again support that, while both models underpredict higher-end PbBs which are the health **risk** concern region, the UBK model provides a closer fit to those upper end **risk** values. The problem for the **SEM** is that it **CAN'T** validly and scientifically **quantitate** the results with sufficient confidence and certainty as discussed above. As mentioned on page 19 of this **EA**, this EHLS design would have been nearly ideal for sites where *higher and more widely distributed* soil Pb and associated blood lead levels existed, but that is not the situation at Bingham Creek -- Phase III. While the UBK is not contended to necessarily be a perfect model (there is no such thing), it does have some strength and success in predicting PbBs that have shown good agreement with measured values at some sites, and it is currently the best available tool for predicting PbB from environmental sources of Pb.

Fig. 2-2

Bingham Creek Blood Lead  
Predicted vs. Observed



Bingham Creek Blood Lead  
Predicted vs. Observed



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Fig. 2-3

FIGURE 5-5 RANK-ORDERED CUMULATIVE DISTRIBUTIONS (209 Residences/252 Children Subset)

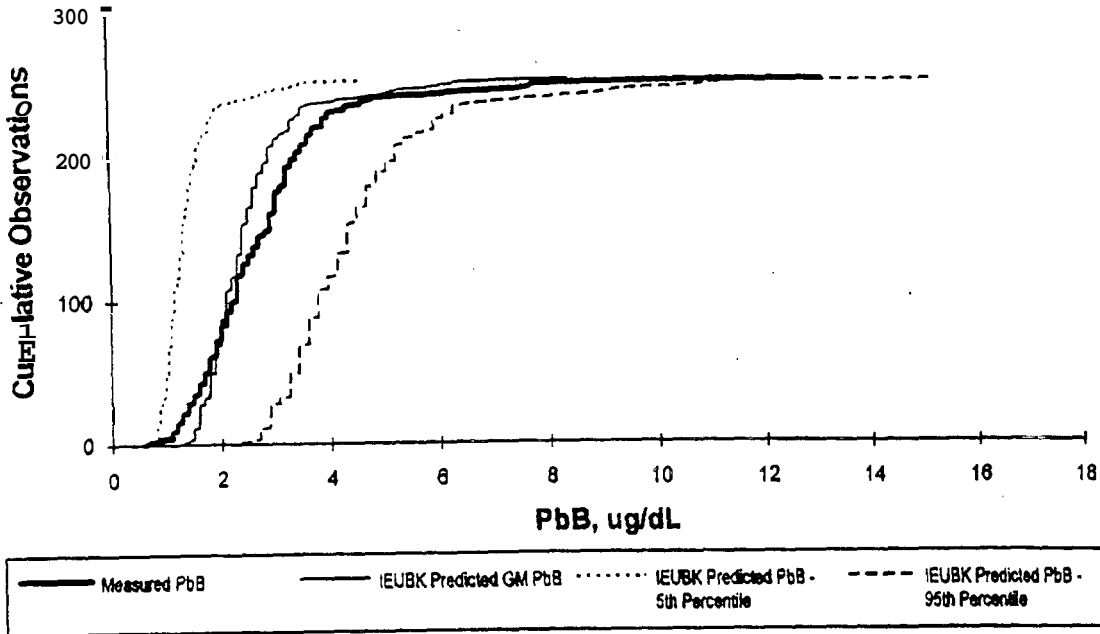


FIGURE 5-4 RANK-ORDERED CUMULATIVE DISTRIBUTIONS (20 Residences/25 Children Subset)

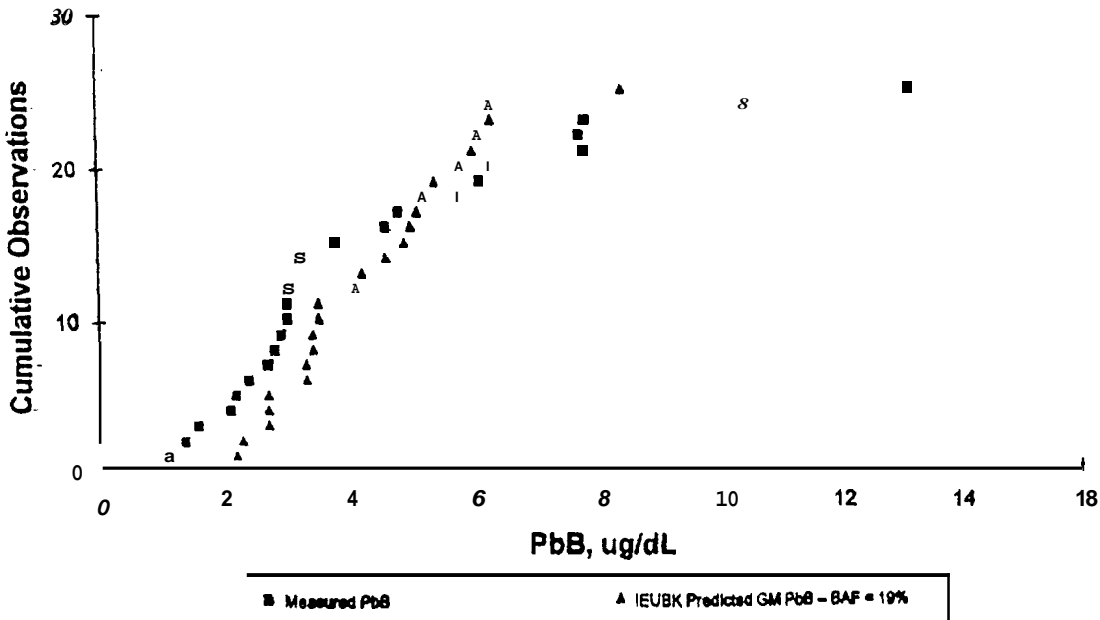
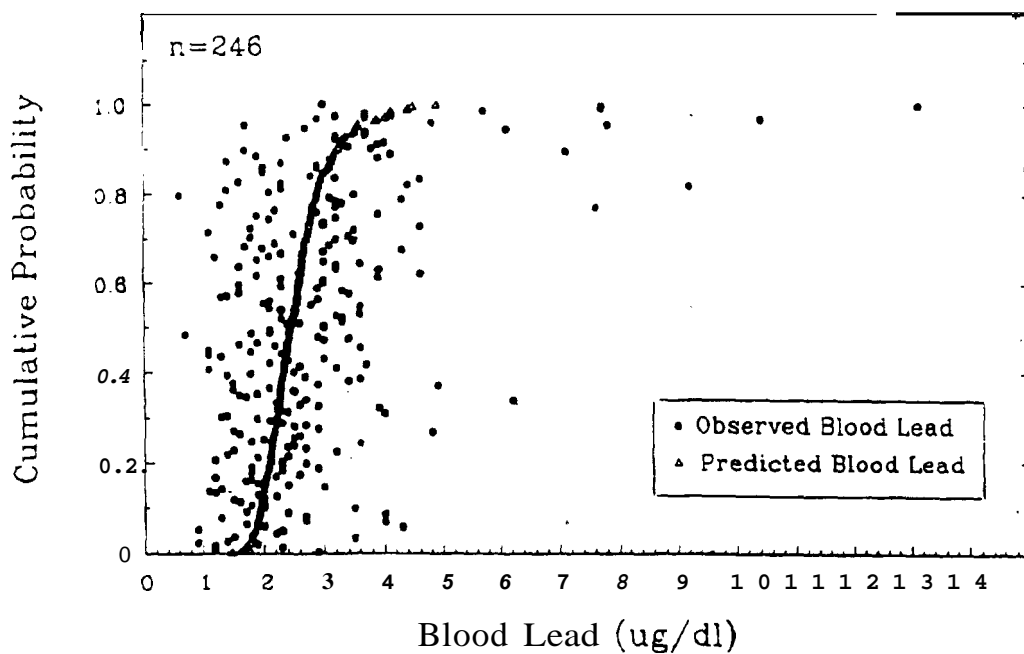
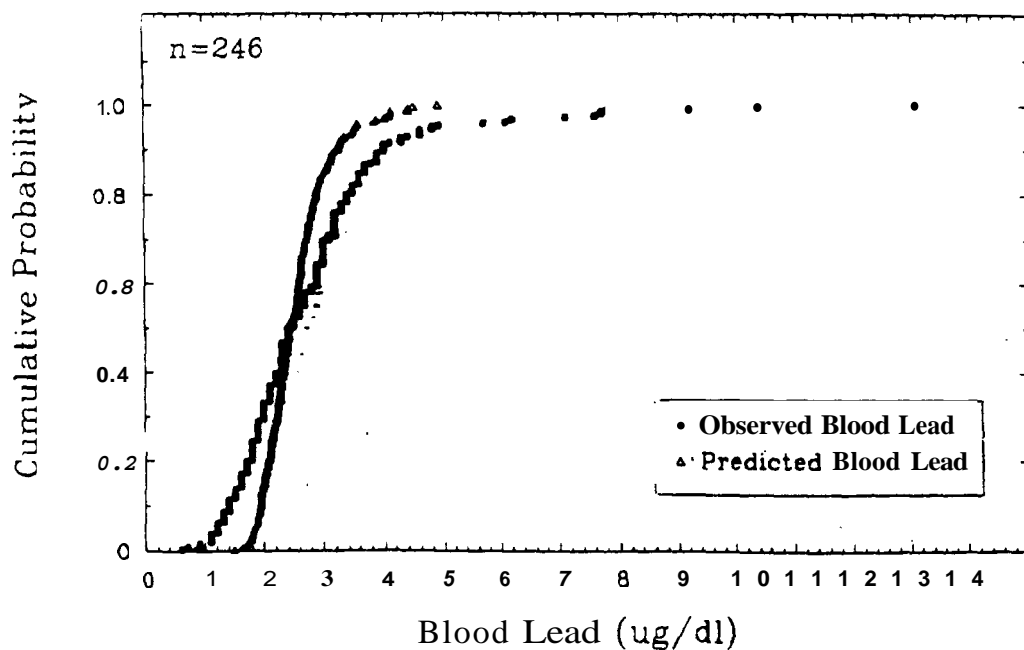


Fig. 2-4

Bingham Creek Study Blood Lead Cumulative Distribution



Bingham Creek Study Blood Lead Cumulative Distribution



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It is fortuitous that few children were found currently above EPA's health standard of 10 ug/dl, but the conclusiveness of the blood lead data is weakened by the design sampling limitations as described previously. The blood lead results can lend some weight of evidence towards qualitatively lowering risk concerns at the site, since (overall) very few children were found near or above the standard; however, in the smaller exposed groups there were enough elevations of PbB which could be argued to the contrary to be substantially at or above the protective level that qualitatively higher concern should be assumed, and thus the results for the more highly exposed subsets do not show conclusive safety for children based upon their PbB levels. Fortunately, they don't show greatly elevated levels either, which would certainly be cause for greater concerns. Because of the "dilution" of the exposed children with the overwhelming numbers of relatively unexposed children, the overall percentages of excess PbB have little meaning for EPA Superfund's quantitative risk assessment.

Rather good scientific arguments have been posed by Kennecott and ARCO scientists and their consultants for reducing concerns about risks to health posed by exposures to soil Pb and As in the Phase III scenario at Bingham Creek; however, the points raised have technical controversial, and an objective view of the scientific literature shows that nearly as convincing arguments can be made for heightening concerns about site health risks<sup>24, 25, 26</sup> or at least to remain somewhat more protective in view of the uncertainties of the true extent and variability of the soil Pb to PbB relationships. Another controversial topic is the possible impacts of publicity on potential deviations in PbB values that were measured after residents could have been more attentive to reducing exposures to soil and dust Pb. The smaller 20-home subset of survey results<sup>9</sup> was later submitted by UC to EPA to help with better examining the question of potential effects of "education" or public awareness on behavior and exposure related to the blood lead results. The smaller dataset of those 20 families living in the more contaminated properties appeared to have had more awareness and may have taken more action to reduce their residential Pb exposures, but the data is limited and conclusive results are elusive.

b. Uncertainty Analyses

The following summary briefly outlines various factors that **EPA** used to balance the **risk** of lead to children at the site and to arrive at recommended PRGs for risk managers. In terms of strength of scientific evidence, **EPA Region VIII** recommended that scientific preference be given to these site-specific results:

- 1) UBK model quantitative predictions (**used** over those predicted by the **SEM**)
  - UBK gives future risk, and is currently built upon a better basis
  - UBK predicts better than **SEM** at this site for higher blood leads
  
- 2) 19% bioavailability determined via the blood Pb (vs average of 15% in **all** tissues)
  - blood values, not other tissue levels, are **used** for health criteria
  - other animal studies proved inadequate for estimating soil Pb bioavailability
  
- 3) Use the smaller “exposed” subsets of blood lead data in the UBK model
  - « 25% of 907 ‘homes are possibly contaminated at potentially toxic levels
  - only 20 of the 209-home subset had  $\bar{x}$  soil Pb > 400 ppm, **EPA’s** default **PRG/SSL**
  - use a **GSD** of 1.43 from the exposed subset of about 250 children in the 209 homes
  - provide equal weight of average dust with the dust:soil ratio calculated<sup>23</sup>
  
- 4) Use the UBK predictions for the entire age range of principle concern (0-84 months) rather than the initially evaluated narrower ages of 0-72 months

In regards to site-specific **PRGs** vs defaults (which would give a **PRG** value of about 400 ppm Pb), this site has **EXTENSIVE** data to justify the use of higher than default soil levels for “equivalent health protection” of residents (where lesser site-specific data exists),

since site uncertainties are reduced and confidence in protectiveness of the higher PRG numbers is increased. The EHLS results were particularly useful in decreasing the **GSD** from a default of 1.6 to a site specific preferred value of **1.43** input (although the same quantitative uncertainties related to near DL values applies here **as** well). Dust exposure **was** also reduced from the default level of **70%** dust levels vs soil levels to a site-specific relationship that was based on the mean of average dust levels and the regression of soil to dust levels calculated **from** the EHLS multimedia data.

We also have much more than default knowledge of the arsenic at Bingham Creek, including: 1) metal speciation showing the presence of less soluble forms rather than more bioavailable smelter-derived arsenic oxides, 2) relatively low urine levels in children, 3) relatively low levels of arsenic in urine **from** pigs fed the highest dose of arsenic soil, and 4) an association of about **4%** arsenic vs lead in soil from Phase II analyses which would equate to average levels of arsenic remaining at about **44** ppm for a lead PRG level of 1100 ppm. Arsenic **risk** under assumptions used in the Phase I **PEA** was about  $7.5 \times 10^{-5}$  (or  $0.75 \times 10^{-4}$ ) at an average arsenic residential yard concentration of 100 ppm; however, the SSL for arsenic under more recent default exposure conditions is **37** ppm. The **WHO** standard is 50 ug/l, but it is not a solid health standard but is more of an exposure estimate of concern. All sampled urines from **EHLS** children were below the 50 ug/l level, with the highest at 35 ug/l. Thus, proposed levels of 100 ppm **As** in soils should be reasonably protective of health for this site.

Some biomedical or other factors to consider in properly balancing uncertainty and strength of evidence for data relevant to the site is briefly summarized in outline form below:

- **More protection** may be needed (arguing for a lower soil Pb) if or since:
  - vegetation Pb intake was not quantitatively added into the **risk** assessment
  - added arsenic **risk** was not quantitated or added, even though acknowledged as low

- the SEM "under-predicts" more so than does the UBK model
  - publicity may have had some impact to lessen exposure and blood lead levels
  - distributions of the the 1990 blood lead data showed that the range of measured PbB was higher compared to the range of distributed PbB values found in the 1993 EHLS, possibly due 1) to the dilution effect of including in 1993 such a large majority of minimally exposed children with the relatively smaller group of highly contaminated homes, 2) removal of residential surface soil before most 1993 sampling occurred, and 3) publicity and awareness by those families most exposed may have altered their children's behavior in regards to soil and dust exposure (reports for smaller subset of 20 homes appear different (may be more aware and concerned) vs the larger dataset
  - removal of soils may have had some impact to lower 1993 blood lead levels<sup>25</sup>
  - analytical uncertainty also exists and was minimally accounted for in sampled media
  - > 5% "exposed" children in smaller subsets had blood lead > 10ug/dl
  - future risk protection is not as strongly afforded by the SEM outputs vs the UBK model
  - land-use for pastures and vacant lots are zoned as residential and are under substantial pressure for development as residences where children could have more exposure
- **Less protection** may be needed (arguing for higher soil Pb) if or since:
    - low urine arsenic levels were found in children and pigs
    - some of the higher blood leads were possibly "contributed" to by Pb in paint for very few older homes, but these homes also had considerable levels of soil and dust Pb as well; so the resultant elevated PbBs are likely a result of exposure to combinations of these sources (such as seen at Butte, MT)
    - the 1990 and 1993 blood Pb levels were largely < 10ug/dl, but are not conclusive in terms of predicting future risks
    - less vulnerable social-economic status of residents would generally decrease exposure
    - bioavailability was more conservatively set at 19%, which was an upper protective range found from evaluating just the blood lead uptake in pigs, rather than using lower values found by evaluating other tissues; however, to help account for this uncertainty in lead uptake, the RATF agreed with EPA to use the range of bioavailabilities from 15% to 19%, where 15% was the mean between the 19% blood estimate and the 11% tissue averaged estimate. It's important therefore to note that bioavailability may be lower than the 19%, but is likely not any higher based on the results of the pig studies.

### E. Qualitative Risks to Ecological and Agricultural Receptors

Ecotoxicological and agricultural risks are estimated to be minimal based on professional judgement, since this highly urbanized area negates any significant exposure and risk to populations of terrestrial wildlife or habitat. Also, the remaining concentrations of the metals are not high enough to pose a credible concern for population scale health risks or impacts. Domestic animals kept in the more highly contaminated pastures on acreages are at some risk, especially ruminants (calves and lambs) if they would ingest too much of the more contaminated soil. Some dogs could experience potential blood problems from Pb, while As in soil at the measured concentrations is a lesser concern to animals. This is another area of considerable uncertainty, but based on the nature of the metal contaminant types and levels and the potential for toxicologic injury in exposed animals, there is little realistic cause for concern in most situations. Owners of livestock or pets on properties with some of the higher levels of contaminants may wish to more closely observe their animals for any unusual signs that a veterinary clinician could evaluate and quite easily rule in or out any contributions from environmental metal contamination.

### III. PRELIMINARY REMEDIATION GOALS

Please refer to the accompanying memo which describes the toxicological need for time critical soil removal action to help reduce imminent and substantial health threats to children potentially exposed to elevated ranges of soil Pb and As, especially for some remaining properties having levels of up to 16000 ppm Pb and 550 ppm As in some zones (25 known areas >2500 ppm, in respect to past EPA PEAs). As noted in the Phase I PEA (refer to page 6 of this present EA), Pb can pose more of a short-term health hazard for young children, which adds to the need to consider merits of time-critical actions to reduce their health risks.

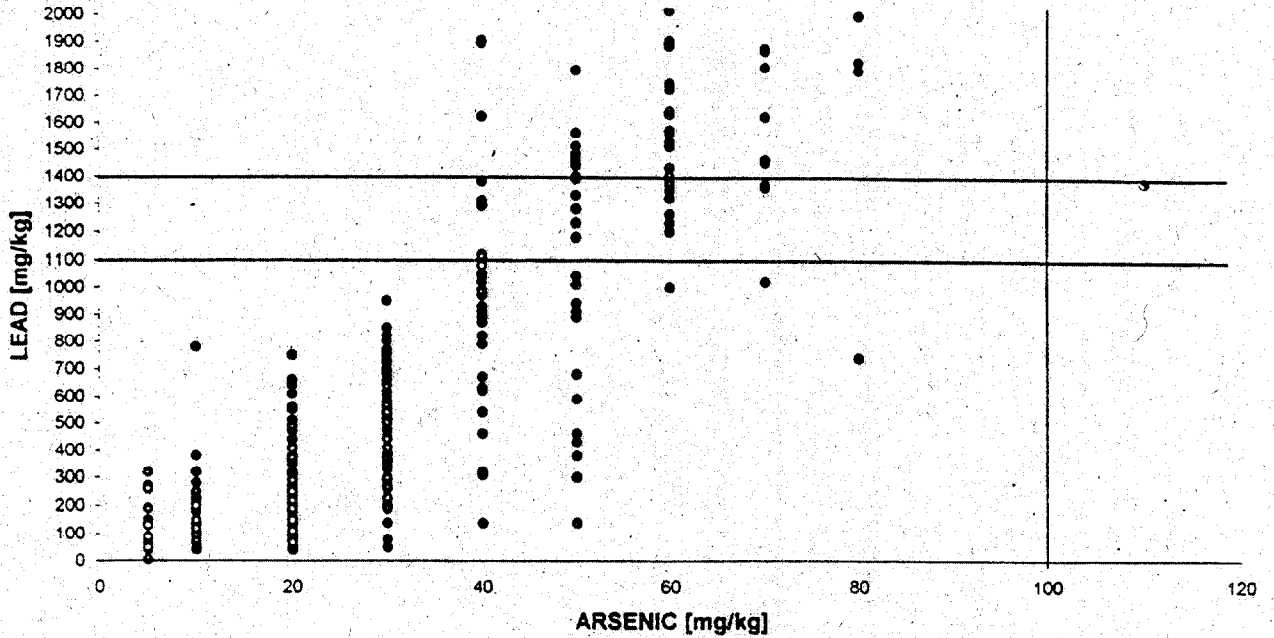
A summary of the "bottom line" (considering strength of site data and uncertainty) health risk-based concentrations that are proposed as PRGs by EPA technical staff are:

- **1100 ppm soil Pb** as a best estimate for a defensibly protective PRG (preliminary remediation goal), which could range up to **1400 ppm**, for current and future residential yard exposure units with soil levels that protect children from having >5% chance of exceeding >10ug/dl PbB, based upon best current information. Note that because of sampling and other measurement error, any confirmational sampling that rules out yards or zones as being under these PRGs should aim for such non-remediated properties to have average soil lead levels of (respectively)  $\leq 1000$  ppm and up to  $\leq 1400$  ppm to ensure the PRG has been achieved with adequate confidence in the soil sampling results.
- **1500 ppm soil Pb** (>1400 ppm upper range described above) is recommended for those areas comprising less than conceivably full residential exposure unit areas (such as in fractions of areas significantly less than usual lot sizes of 1/4 acre), since risk-based concentrations are derived from entire yard-wide average Pb exposures and not just smaller areas; however,
  - Relative "hot-spots" of soil metals found in smaller-than-standard exposure unit areas are recommended to be considered for remedial clean-up for As >100 ppm and Pb >1000 ppm in higher-than-usual childhood exposure areas (sand boxes, gardens, etc.)
  - Where such hot-spots may not be removed, then it is recommended that children *avoid those areas or otherwise reduce exposure* to higher levels of soil metal contaminants by washing children's hands, vegetating bare soils, reducing house dust levels, etc.
- **100 ppm soil As** is recommended as a semi-quantitative consideration for taking appropriate action to reduce potential risks from As over-exposures, which is substantially higher than EPA's SSLs but is supportable based on a balance of site-specific data and the uncertainties surrounding As exposure and health risks. In addition, **Figure 3-1** shows that per the Phase II Bingham Creek data, there is relatively good correlations between soil Pb and As levels, such that the soil Pb PRGs would be expected to coincidentally eliminate most soil As levels that exceed the suggested 100 ppm PRG for soil As. As elaborated more recently <sup>27</sup>, there are definitely some unacceptably high (300 - 400 ppm) levels in highly frequented areas by children that require action to reduce potential risks to tolerable levels. The EHLS report <sup>9</sup> stated that about 45 yards had soil As > 95 ppm; and if the past Pb:As ratios hold true, then the soil Pb PRG range of 1100 to 1400 should address most of the soil As levels greater than about 50 ppm on average (using the 3-4% ratio).



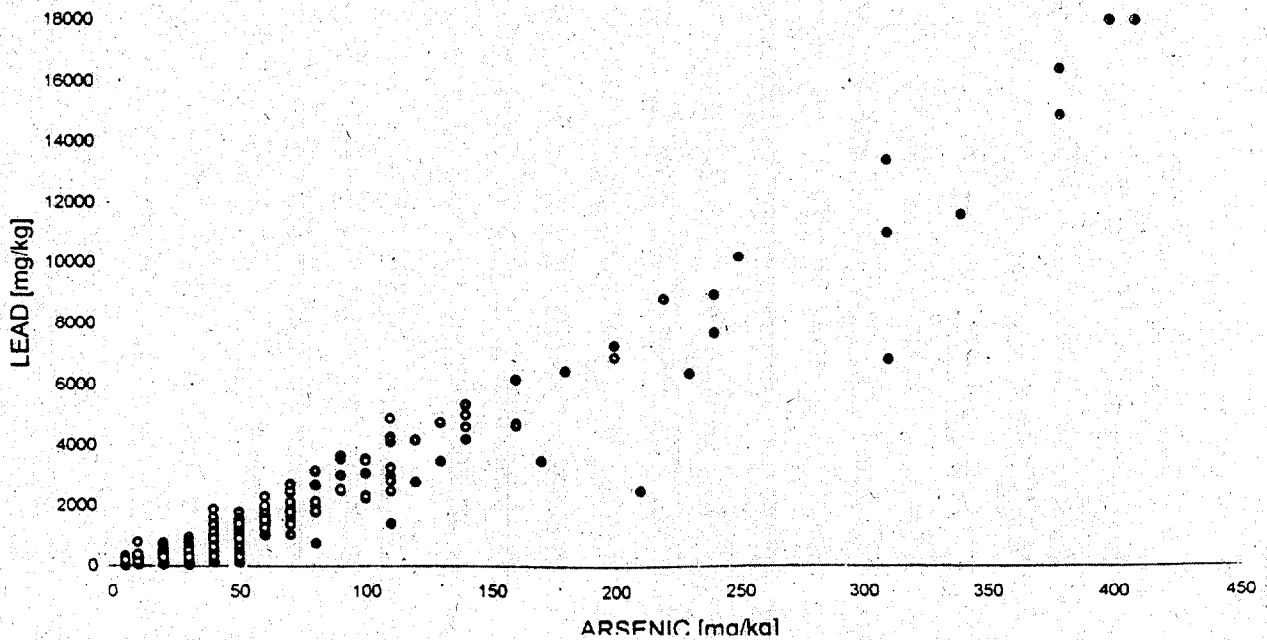
Fig 3-1

Arsenic to Lead Relationships in 1992 Bingham Creek Data compared to 1995 Proposed Action Levels of 1100-1400 ppm Pb and 100 ppm As



KENN2.XLC

Lead to Arsenic Relationships in Bingham Creek Channel Samples, Phase 2



#### IV. References and Attachments

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14. Phase II Bioavailability Studies: Sample Preparation and Analyses Report (draft), Roy F. Weston, Inc. (John Drexler, subcontractor), Region VIII EPA, Denver, CO, April, 1995.
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18. Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities, EPA OSWER Dir # 9355.4-12, Elliott Laws, Jul 14, 1994.
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