Engineering a Sustainable Future

EPA Region 1 RAC2 Contract No. EP-S1-06-03

May 19, 2011
Nobis Project No. 80024

Via Electronic Submittal

U.S. Environmental Protection Agency, Region 1
Attention: Mr. Ed Hathaway, Task Order Project Officer
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Boston, Massachusetts 02109-3919

Subject: Transmittal of the Final Human Health Risk Assessment
Ely Copper Mine Superfund Site, Vershire, Vermont
Remedial Investigation / Feasibility Study
Task Order Number 0024-RI-CO-017L

Dear Mr. Hathaway:

Attached with this correspondence is the Final Human Health Risk Assessment report for the Remedial Investigation/Feasibility Study at the Ely Copper Mine Superfund Site located in Vershire, Vermont.

Should you have any questions or comments, please contact me at (603) 724-6224, or by email at aboeckeler@nobisengineering.com.

Sincerely,

NOBIS ENGINEERING, INC.

Andrew J. Boeckeler, P.G.
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Human Health Risk Assessment

Ely Copper Mill Superfund Site
Vershire, Vermont

Remedial Investigation / Feasibility Study
EPA Task Order No. 0024-RI-CO-017L

REMEDIAL ACTION CONTRACT
No. EP-S1-06-03

FOR

US Environmental Protection Agency
Region 1

BY

Nobis Engineering, Inc.

Nobis Project No. 80024

May 2011

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Vershire, Vermont
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Andrew J. Boeckeler, P.G.
Senior Project Manager

Tim Woods
Risk Assessor (Avatar)

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ELY COPPER MINE SUPERFUND SITE
VERSHIRE, VERMONT

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ACRONYMS AND ABBREVIATIONS

ADD	Average Daily Dose
AT	Averaging Time
bgs	below ground surface
CLT	Central Limit Theorem
cm^2	square centimeters
cm^3	cubic centimeters
cm/hour	centimeters per hour
COPC	Contaminant of Potential Concern
CSF	Cancer Slope Factors
CSM	Conceptual Site Model
CTE	Central Tendency Exposure
DABS	Dermal Absorption Factors
DAD	Dermally Absorbed Dose
DA_{event}	Absorbed Dose per Event
ED	Exposure Duration
EPA	U.S. Environmental Protection Agency
EPC	Exposure Point Concentration
HEAST	Health Effects Assessment Summary Tables
HHRA	Human Health Risk Assessment
HI	Hazard Index
HQ	Hazard Quotient
Hr	hour
IEUBK	Integrated Exposure Uptake and Biokinetic
ILCR	Incremental Lifetime Cancer Risk
IRIS	Integrated Risk Information System
kg	kilogram
kg/mg	kilogram per milligram
KM	Kaplan-Meier
Kp	Permeability Coefficient
L	liter
LADD	Lifetime Average Daily Dose
LOAEL	Lowest-Observed-Adverse-Effect-Level

ACRONYMS AND ABBREVIATIONS (cont.)

MCL	Maximum Contaminant Level
m^3/kg	cubic meters per kilogram
mg	milligrams
mg/cm^2	milligrams per square centimeter
mg/kg	milligrams per kilogram
mg/kg-day	milligrams per kilogram per day
mg/L	milligrams per liter
mg/m^3	milligrams per cubic meter
MVUE	Minimum Variance Un-biased Estimators
NCEA	National Center for Environmental Assessment
Nobis	Nobis Engineering, Inc.
NOAEL	No-Observed-Adverse-Effect-Level
OABS	Oral Absorption Factors
OSWER	Office of Solid Waste and Emergency Response
PCB	polychlorinated biphenyl
PPRTV	Provisional Peer Reviewed Toxicity Value
RAGS	Risk Assessment Guidance for Superfund
RfC	Reference Concentration
RfD	Reference Dose
RI/FS	Remedial Investigation/Feasibility Study
RME	Reasonable Maximum Exposure
RSL	Regional Screening Levels
RSR	Remediation Standard Regulations
Site	Ely Cooper Mine Superfund Site
SQL	Sample Quantitation Limit
UCL	95 percent upper confidence limit on the mean
URF	Unit Risk Factor
USACE	United States Army Corps of Engineers
USGS	United States Geological Service
µg/dL	micrograms per deciliter
µg/kg	micrograms per kilogram
µg/L	micrograms per liter

ACRONYMS AND ABBREVIATIONS (cont.)

$\mu g/m^3$	micrograms per cubic meter
XRF	x-ray fluorescence

1.0 INTRODUCTION

This Human Health Risk Assessment (HHRA) of the Ely Copper Mine Site (the Site) was prepared by Nobis Engineering, Inc. (Nobis) for the United States Environmental Protection Agency (EPA) under contract No. EP-S1-06-03, Task Order No. 0024-RI-CO-017L. This report presents a description of the risk assessment methods employed for the Site, as well as a summary of the results. The objective of the HHRA was to estimate potential current and future human health risks from the presence of contamination in the soil, groundwater, sediment, and surface water. The HHRA evaluates non-cancer health hazards, cancer risks, and lead exposures through quantitative assessments.

1.1 Document Organization

The HHRA is organized as follows:

- Section 2.0 Site Description and Setting provides a general description of the Site, including Site background and history.
- Section 3.0 Site Data Summary includes a summary of the existing Site data and the data used in the contaminant of potential concern (COPC) screening analysis.
- Section 4.0 Data Evaluation Approach presents the methods that were followed to evaluate and summarize the analytical data.
- Section 5.0 Identification of Contaminants of Potential Concern presents the process followed to identify COPCs in the evaluated media and presents the resultant COPCs.
- Section 6.0 Toxicity Assessment presents the toxicity values that were identified for the COPCs.
- Section 7.0 Exposure Assessment presents the exposure pathways, the routes of exposure, the exposure parameters, and the exposure point concentrations.
- Section 8.0 Risk Characterization presents the methodologies followed to estimate cancer risks and non-cancer health effect and presents the risk results.
- Section 9.0 Uncertainty Analysis presents the major uncertainties associated with the risk assessment process and the potential impact on the calculated risks.
- Section 10.0 Summary presents an overall summary of the results of the HHRA.
- Section 11.0 References.

2.0 SITE DESCRIPTION AND SETTING

This Section provides a brief description of the Site, its general surroundings, and pertinent historical facts regarding the mining history as a context for subsequent sections discussing Site details.

2.1 Site Description and Setting

The Site is located approximately 5 ½ miles southeast of the village of Vershire Center, and approximately 1 ½ miles northwest of the village of West Fairlee in Vershire, Orange County, Vermont (see Figure 2-1). The Site encompasses approximately 350 acres along the south slope of Dwight Hill, to the north of Schoolhouse Brook and South Vershire Road. The mine area includes features such as intact and collapsed adits, shafts, reservoirs, over 3,000 linear feet of underground workings (largely flooded) and remnant foundations of former mine operation buildings. Waste areas are sparsely vegetated and include former ore roast beds, waste rock and tailings piles, a former smelter area, and a slag pile. No buildings exist on the Site. The locations of the former Main Shaft Hoist, the Westinghouse Hoist House, smelter buildings, a World War I-era ore flotation separation plant, and other structures associated with historic mining operations have been documented at the Site (PAL 2005).

The Site landscape is a combination of barren open areas and patches of birch and evergreen trees. The south slope of Dwight Hill, which contains most of the waste rock associated with the mines, lies within the watershed of a small stream, Ely Brook, which flows 0.75 miles south to join Schoolhouse Brook on the south side of South Vershire Road. Schoolhouse Brook borders the southern margin of the Site and flows eastward approximately 1.75 miles to its confluence with the east branch of the Ompompanoosuc River. Schoolhouse Brook and the E. Branch of the Ompompanoosuc River are used for recreational purposes and contain edible fish.

Site topography is dominated by the peak and steep south slope of Dwight Hill extending from an elevation of approximately 1,600 feet above mean sea level down to Schoolhouse Brook at an elevation of approximately 940, some 660 feet of relief. The main shaft and several adits leading to the underground workings are located along the steep, upper portion of this slope at the head of the valley. Most of the mine wastes lie within the more gently sloping, lower portions of this valley. The crest of Dwight Hill occurs along a northwest trending ridge which forms the northern boundary of the Ely Brook watershed. Underground workings extend

approximately 3,000 feet to the northeast of the mine openings beneath and beyond the top of the ridge. North-south trending ridges to the west and east of the mine areas define two smaller upland valleys that merge into an open U-shaped valley facing south-southwest which define the Ely Brook watershed. The headwaters of Ely Brook are located in the western tributary valley, northwest of the mine areas. A tributary to Ely Brook drains the eastern tributary valley which contains a former reservoir and a series of beaver ponds located east of the Site. The northeast slope of Dwight Hill is moderately steep with an elevation drop of approximately 800 feet down to Route 113 to the east.

The Site is located in a Town of Vershire, Orange County, Vermont with a population of approximately 630 people (Town of Vershire website, 2000 Census). The Site is located in a rural, sparsely populated area of the town accessed by Beanville and South Vershire Roads, approximately 1.5 miles northwest of the Village of West Fairlee. It is estimated that less than 100 people live within a 1-mile radius of the Site. The nearest residents are located approximately ¼ mile east of the Site along Beanville Road. The Site and vicinity are forested with the exception of open areas occupied by mine waste rock piles. The Site is privately owned. The land is undeveloped and generally undisturbed since cessation of mining activities. The current land use of the Site is reportedly limited to management for commercial timber harvest. There are no residents or buildings on the Site. The Site is reportedly frequented for limited recreational use by off-road vehicles, hunters, hikers, and spelunkers.

3.0 SITE DATA SUMMARY

A number of field investigations have been performed at the Site. The available data from each investigation was reviewed and a determination was made as to its usability for HHRA purposes. The approach followed to determine the datasets for the HHRA was to ensure suitable spatial coverage, to utilize relatively recent data, and to provide adequate sample size.

3.1 URS Data

From 2004 through 2007, URS, in conjunction with the U.S. Army Corps of Engineers (USACE) and EPA, completed preliminary field sampling investigations. These investigations included: test pits and borings in waste areas; monitor well installation; and collection of surface water, sediment, surface and subsurface soil, and groundwater samples from the Site. In addition, one off-site residential groundwater sample was collected. All of the URS collected data were

analyzed for Target Analyte List (TAL) metals. These data have been validated. The data collected during 2006 and after were used in the HHRA.

3.2 USGS Data

The U.S. Geological Survey (USGS) completed a number field studies dating back to 2000. In 2002, a study of spring runoff from the Site to characterize the geochemical diversity of water sources in the Ely Brook watershed that included sampling seeps from mine waste areas, Ely Brook and tributaries, Schoolhouse Brook, and Ompompanoosuc River was performed. In 2004 and 2007, USGS completed a series of studies of mine wastes at the Site that included sampling and analysis of the various solid mine waste materials and sediment to characterize the materials, assess their acid-generating potential, and evaluate their potential for leaching metals. A separate study of the geochemistry of the slag material deposited along S. Vershire Road in comparison to slags at other mine sites in North America was completed by USGS in 2004. This study included an evaluation of secondary minerals formed on the slag and the potential leachability of metals from the slag. These data collected during 2006 and after were used in the HHRA.

In 2006 and 2007, USGS, in conjunction with EPA, collected data to support ongoing work on the aquatic Baseline Environmental Risk Assessment (BERA) for the Site. The study included a detailed characterization of surface water and sediment quality, an evaluation of fish and macroinvertebrate communities, and an evaluation of the toxicity of surface water, sediment and pore water. Whole body fish samples (blacknose dace and trout) were collected from Schoolhouse Brook and the East Branch of the Ompompanoosuc River. The data collected by USGS were analyzed for TAL metals along with other elements. The USGS data collected during 2006 and 2007 were used in the HHRA.

3.3 Nobis Data

Nobis conducted extensive field sampling investigations in 2009 in support of the current remedial investigation/feasibility study (RI/FS). The Nobis-collected data were used in the HHRA with the exceptions noted in the following paragraphs. Soil sampling was conducted during several phases of Site investigation activities including surface soil transects for x-ray fluorescence (XRF) field screening and laboratory analysis to assess the limits of waste areas. Additional surface soil samples were collected from selected locations within the transitional

zones bordering unvegetated waste areas to assess the potential impact beyond the waste area footprints. Surface and subsurface soil samples were also collected from soil boring and monitoring well locations to assess the characteristics of waste materials and underlying natural soils. While useful for characterizing the Site, the XRF data were not used for the HHRA. However, the XRF data were summarized and a statistical evaluation was performed of the paired XRF data and the laboratory data to determine the comparability and consistency of the data. This evaluation is presented in Appendix A. The results of the comparison are discussed in Section 9.0.

Surface soil samples (including duplicates) were collected from 39 locations throughout the Site. Sample intervals ranged from 0 to 0.2 feet (ft) below ground surface (bgs) to 0 to 2 ft bgs. In several locations, multiple samples were collected at consecutive depths. With the exception of two samples, all surface soil samples were submitted for Contract Laboratory Program (CLP) laboratory analysis of TAL metals. Select samples were also submitted for Delivery of Analytical Services (DAS) laboratory analysis of acid-base accounting (ABA), paste pH, paste conductivity, total organic carbon (TOC), synthetic precipitate leachate procedure (SPLP) metals, and cation exchange capacity (CEC).

Soil borings were advanced at 8 locations. Seven of the soil borings were completed as overburden well installations and one soil boring was completed without a well installation. During boring advancement, multiple soil samples were collected at discrete intervals in each location. A total of 25 samples were collected (approximately 3 from each boring) and submitted for CLP and DAS laboratory analysis of TAL metals, ABA, paste pH, and paste conductivity. In addition, 6 samples were submitted for SPLP metals analysis.

Over 250 surface soil samples (including duplicates) were collected along 25 transects located within the waste piles areas and extending into the surrounding wooded areas. The sample intervals ranged from 0 to 0.5 ft bgs to 2.5 to 4 ft bgs. The samples were submitted to the EPA's Office of Environmental Measurement and Evaluation (OEME) on-site mobile laboratory and screened for select metals using XRF. Based on the results of the XRF screening, 32 of the samples were submitted for CLP laboratory analysis of TAL metals. Select samples were also submitted for DAS analysis of ABA, paste pH, and paste conductivity.

Five vernal pool samples (including duplicates) were collected from 4 locations. Surface water samples were collected at each location and submitted for laboratory analysis of TAL metals (total and dissolved), pH, conductivity, alkalinity, sulfate, chloride, and nitrate. The vernal pool samples were not used in the HHRA.

Two surface water sampling events occurred in 2009. In July and November, 35 surface water samples (including duplicates) were collected from 33 locations. The samples were submitted for laboratory analysis of TAL metals (total and dissolved), chloride, sulfate, carbonate, bicarbonate, hydroxide, sulfide, nitrate/nitrite, nitrogen, total acidity, alkalinity, total cyanide, total suspended solids (TSS), total dissolved solids (TDS), and hardness. Additionally, directly following sampling, surface water at each corresponding location was field screened for temperature, pH, conductivity, dissolved oxygen (DO), oxidation reduction potential (ORP), and turbidity with the appropriate meters.

In August 2009, 17 sediment samples (including duplicates) were collected from 16 locations. The samples were submitted for laboratory analysis of TAL metals, ABA, paste pH, paste conductivity, TOC, and grain size. In addition, one sediment sample collected was submitted for laboratory analysis of SPLP metals.

In August 2009, 42 overburden and shallow bedrock groundwater samples (including duplicates) were collected using EPA low-flow sampling protocol from 40 monitoring wells (overburden and shallow bedrock). The samples were submitted for laboratory analysis of TAL metals (total and dissolved), total cyanide, sulfate, and hardness. In addition, 21 samples (50 percent) were submitted for chloride, carbonate, bicarbonate, hydroxide, sulfide, nitrate/nitrite, nitrogen, total acidity, alkalinity, total cyanide, TSS, and TDS.

In August 2009, 8 porewater samples (including duplicates) were collected at 7 locations along a portion of the upper reach of Ely Brook. The samples were submitted for laboratory analysis of TAL Metals (total and dissolved), chloride, sulfate, sulfide, nitrate/nitrite, nitrogen, alkalinity, and total cyanide. The porewater samples were not used in the HHRA.

In September 2009, 12 groundwater samples (including duplicates) were collected from 7 intervals in 3 deep bedrock wells during packer testing. The samples were submitted for CLP laboratory analysis of TAL Metals (total and dissolved).

In November 2009, 46 overburden and bedrock (shallow and deep bedrock) groundwater samples (including duplicates) were collected via EPA low-flow sampling protocol from 43 monitoring wells. The samples were submitted for laboratory analysis of TAL metals (total and dissolved), total cyanide, and sulfate. In addition, 11 samples were submitted for analysis of volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs), pesticides, and poly-chlorinated biphenyls (PCBs). Additionally, as part of the low-flow sampling protocol, geochemical parameters were recorded for temperature, pH, conductivity, DO, ORP, and turbidity with the applicable meters.

In December 2009, 6 drinking water samples were collected from 6 off-site residential locations. Samples were collected within a 0.5 mile distance from the Site to assess local groundwater quality relative to the Site and evaluate its potential impact on local drinking water. Drinking water samples were submitted for laboratory analysis of TAL metals (total and dissolved), chloride, sulfate, carbonate, bicarbonate, hydroxide, sulfide, nitrate/nitrite, nitrogen, total acidity, alkalinity, total cyanide, and hardness.

In September 2009, biota samples consisting of small mammals and soil invertebrates were collected in support of the ecological risk assessment. The biota samples were not used in the HHRA.

3.3.1 November 2010 Samples

In November 2010, 19 soil and sediment samples (including a field duplicate) were collected from the Site to identify the type of chromium present in the Site media. The samples were analyzed for total chromium and hexavalent chromium (Cr VI). The sample results (see Table 3-1) indicate the presence of total chromium in all samples and no detectable levels of Cr VI. This information provides sufficient evidence that Cr VI, the more toxic valence state of chromium, is not present in the Site media and enables the HHRA to evaluate the exposure and risks presented in the following sections of this report based on the less toxic trivalent chromium (Cr III) (see the risk calculation tables discussed in Section 8) .

3.4 Data Used in the Human Health Risk Assessment

Following the approach to identifying data for the HHRA described in Section 3.0, the available analytical data collected between 2006 and 2009 by URS, USGS, and Nobis were used (with the noted exceptions) in the HHRA. For the surface water and groundwater, the total metals results were used. The data selection criteria resulted in datasets that are both spatially representative and sufficiently robust for HHRA objectives.

The sediment and surface water datasets from the downstream areas (Schoolhouse Brook and Ompompanoosuc River) are not as robust as other areas. Historic sediment and surface water data collected prior to 2006 were considered for these areas but deemed too old for the HHRA as it does not reflect current conditions.

Tables 3-2 through 3-6 present summaries of the sample locations. Figures 3-1 through 3-6 show the sample locations.

4.0 DATA EVALUATION APPROACH

This section presents the approach that was followed to prepare the analytical data for use in the HHRA. It also discusses the exposure areas for the HHRA and presents the data summaries for each medium.

4.1 Data Reduction

Data reduction involves the evaluation of data qualifiers and their potential use in the HHRA process and describes the treatment of field duplicate samples. The following guidelines were used in developing the data sets to evaluate Site risk:

- If an analyte was not detected in any sample (U qualified) from a given medium, it was not considered further for that medium.
- All “U” qualified data represent samples for which the analyte was not present or was below the sample quantitation limit (SQL) and reported as a non-detect (ND).
- Estimated values (J qualified) were used at the reported value.

When field duplicate samples were collected, the following approach was used to calculate the concentrations to be evaluated in the HHRA:

- If the analyte was detected in both the original (primary) sample and the field duplicate, the average concentration was used in the HHRA unless there was a greater than 50 percent relative percent difference in solid media or a 30 percent relative percent difference in aqueous media in which case the higher of the two concentrations was used.

- If the analyte was detected in either the primary or duplicate sample and was ND in the other sample, the detected concentration was used.

- If the analyte was ND in the primary and duplicate sample, the average SQL concentration was used.

4.2 Exposure Areas

The entire Site was evaluated for soil exposure. There were no unique areas of exposure potential or contamination patterns that would warrant dividing the Site into multiple soil exposure areas (EAs). The surface water and sediment EAs were identified based on the waterbody and considered the length of the EA and waterbody characteristics such as morphology and flow regimes. A total of three surface water and sediment EAs were identified:

- Ely Brook and tributaries including the Beaver Ponds complex.
- Schoolhouse Brook – downstream to the East Branch of the Ompompanoosuc River.
- E. Branch of the Ompompanoosuc River.

A number of monitoring wells are present on the Site. The Site groundwater was evaluated by water-bearing unit: overburden, shallow bedrock, and deep bedrock. The off-site residential groundwater was evaluated separately.

Previous studies by EPA Region 1 and USGS have determined that Schoolhouse Brook and the Ompompanoosuc River support edible fish communities. In areas where only a limited number of trout were collected, blacknose dace were used as a proxy for trout.

4.3 Data Summary

Tables 4-1 through 4-7 present the data summaries for the Site media. The summary tables present the following information:

- List of detected analytes.
- The frequency of detection (FOD). The FOD for soil, sediment, surface water, and fish represents the number of detected samples out of the total number of samples. For groundwater, the FOD represents the number of times that an analyte was detected at least once in a well out of the total number of wells sampled.
- The range of detected concentrations.
- The location of the maximum detected concentration.
- Background value.
- Screening toxicity value.
- Whether or not the analyte was identified as a COPC.

The following data summary tables were developed:

- Surface soil (0 to 1 ft bgs) – Table 4-1
- Total soil (0 to 10 ft bgs) – Table 4-2
- Sediment – Table 4-3
- Surface water – Table 4-4
- Site groundwater – Table 4-5
- Off-site residential groundwater – Table 4-6
- Fish – Table 4-7

5.0 IDENTIFICATION OF CONTAMINANTS OF POTENTIAL CONCERN

The maximum detected concentrations in the affected media were compared to the EPA Regional Screening Levels (RSLs) (EPA, 2010a) for each EA. The residential soil RSLs were used for the soil evaluation. The tap water RSLs were used for the groundwater water evaluation. RSLs are not available for sediment and surface water exposure. In the absence of RSLs for these media, the residential soil and tap water RSLs were conservatively used for sediment and surface water exposure, respectively. The risk-based fish tissue RSLs were used for the fish evaluation. For screening all media, the cancer based RSLs were set at a target cancer risk of 1E-06. The non-cancer based RSLs were set at a target hazard quotient of 0.1. This was done to account for potential additivity or cumulative effects of multiple contaminants on similar organs.

If the medium-specific maximum detected concentration was less than the RSL, the analyte was eliminated from further consideration in the HHRA. If the maximum concentration exceeded the RSL, the analyte was identified as a COPC.

Analytes considered to be essential nutrients (calcium, magnesium, potassium, and sodium) were not included in the COPC screening. The RSLs for hexavalent chromium were conservatively used to screen chromium. There were a number of analytes for which RSLs were not available and suitable surrogate RSLs could not be identified. In these cases, the analytes were noted on the screening tables but not carried forward in the HHRA process due to a lack of toxicity information.

Site-specific background data were available. While not used in the COPC selection process, the background data were considered in the Uncertainty Analysis (Section 9.0) to determine how Site levels compared to the surrounding area levels.

Tables 4-1 through 4-7 present the results of the COPC screening process. The following sections present the results by medium.

5.1 Soil

The following metals were identified as COPCs in the soil:

Aluminum	Lead
Antimony	Manganese
Arsenic	Mercury
Cadmium	Molybdenum
Chromium	Nickel (total soil only)
Cobalt	Selenium
Copper	Vanadium
Iron	Zinc (total soil only)

5.2 Sediment

The following metals were identified as COPCs in Ely Brook sediment:

Aluminum	Iron
Arsenic	Lithium
Chromium	Manganese
Cobalt	Selenium
Copper	Vanadium

The following metals were identified as COPCs in Schoolhouse Brook sediment:

Aluminum	Iron
Arsenic	Lithium
Chromium	Manganese
Cobalt	Vanadium
Copper	

The following metals were identified as COPCs in Ompompanoosuc River sediment:

Aluminum	Iron
Arsenic	Lithium
Chromium	Manganese
Cobalt	Vanadium

5.3 Surface Water

The following metals were identified as COPCs in Ely Brook surface water:

Aluminum	Lithium
Arsenic	Manganese
Cadmium	Mercury
Chromium	Nickel
Cobalt	Vanadium
Copper	Zinc
Iron	

The following metals were identified as COPCs in Schoolhouse Brook surface water:

Chromium	Manganese
Cobalt	Mercury
Copper	Vanadium
Lead	

Mercury was the only COPC identified in the East Branch of the Ompompanoosuc River surface water.

5.4 Groundwater

The groundwater COPCs are summarized below.

Overburden

Aluminum	Iron
Arsenic	Lead
Barium	Manganese
Cadmium	Molybdenum
Chromium	Nickel
Cobalt	Vanadium
Copper	Zinc

Shallow Bedrock

Aluminum	Iron
Antimony	Lead
Arsenic	Manganese
Beryllium	Molybdenum
Cadmium	Nickel
Chromium	Vanadium
Cobalt	Zinc
Copper	

Deep Bedrock

Bis(2-ethylhexyl)phthalate	Cobalt
Aluminum	Copper
Arsenic	Iron
Cadmium	Manganese
Chromium	Nickel
	Vanadium

As shown in Table 4-5, Aroclor 1248 was detected in the deep bedrock groundwater. Although the maximum detected concentration of Aroclor 1248 exceeded its RSL, it was eliminated from consideration as a COPC in the HHRA. The detected sample result was considered suspect. PCBs were re-sampled and were not detected in subsequent samples. Further, the time period of mining operations pre-dates PCBs. For these reasons, PCBs were eliminated as COPCs.

Off-Site Residential

Copper
Vanadium

5.5 Fish

A small subset of the detected metals in the whole body fish tissue data was greater than the RSLs. These included the following:

- Antimony – Schoolhouse Brook blacknose dace.
- Chromium – brook trout and blacknose dace from Schoolhouse Brook and the East Branch of the Ompompanoosuc River.
- Cobalt – brook trout and blacknose dace from Schoolhouse Brook and the East Branch of the Ompompanoosuc River.
- Zinc – blacknose dace from the East Branch of the Ompompanoosuc River.

There are a few items that need to be discussed to put these exceedances into perspective. First, the fish tissue RSLs were based on a conservative default fish ingestion rate of 54 g/day. Given the characteristics of Schoolhouse Brook and the Ompompanoosuc River, this is an over-estimate of the level of fish consumption likely to occur in the evaluated area. The 95th percentile fish ingestion rates for rivers/streams in the northeastern United States are in the 10 to 30 g/day range (Ebert et al. 1993 and 1996; MDPH, 1997); depending upon how the catch is shared. Given the size and productivity of these fisheries, even this range is likely to overestimate realistic exposure. Using a more realistic ingestion rate would result in the RSLs approximately 2 to 5 times greater than the values used in this screening analysis. Assuming the adjusted RSLs are only twice as high, antimony, cobalt, and zinc would be eliminated as COPCs for the following reasons:

- Antimony – 2 of the 4 detected concentrations in fish would exceed the adjusted RSL of 0.108 mg/kg with a maximum ratio of detected concentration to RSL of 3.7. The other two detected concentrations would be below the adjusted RSL.

- Cobalt – only 4 of the 17detected concentrations in fish would exceed the adjusted RSL of 0.082 mg/kg with a maximum ratio of 1.3.

- Zinc - none of the 17 detected concentrations in fish would exceed the adjusted RSL of 82 mg/kg. (Using the RSL as currently is, only 2 of the 17 detected concentrations exceeded the RSL with a maximum ratio of only 1.01.)

Second, it was conservatively assumed for screening purposes that chromium was entirely in the hexavalent state (Cr VI), which has been shown to be non-detect in samples collected for chromium speciation (see Table 3-1). If the RSL for the more prevalent trivalent form was used for chromium, there would be no exceedances.

Lastly, all of the fish used in this screening evaluation were analyzed as whole body tissue. Metals tend to be excreted in the bile, by the gills, and/or be sequestered in bones of fish (Meyer 2005). Therefore, whole body concentrations are likely higher than what would be found in the fillet, which is more commonly consumed than whole fish, particularly for sport fish such as trout.

For these reasons, the levels observed in the fish tissue data are not a concern for human health and fish were not evaluated further in the HHRA.

6.0 TOXICITY ASSESSMENT

The toxicity assessment examines information concerning the potential human health effects of exposure to COPCs. The goal of the toxicity assessment is to provide, for each COPC, a quantitative estimate of the relationship between the magnitude and type of exposure and the severity or probability of human health effects. The toxicity values presented in this section are integrated with the information presented in the exposure assessment (Section 7.0) to characterize the potential for the occurrence of adverse health effects.

Cancer slope factors (CSFs) and unit risk factors (URFs) are the dose-response values used to evaluate potential carcinogens. Non-carcinogenic effects, such as organ damage or reproductive effects, are evaluated by reference doses (RfDs) for oral exposure and reference concentrations (RfCs) for inhalation exposure. The EPA's Integrated Risk Information System (IRIS) database (U.S. EPA 2010b) was consulted as the primary source for RfD and RfC values, as well as for CSFs and URFs. The IRIS database supersedes all other sources of

toxicity information for risk assessment. Additional sources for the dose-response values used in the risk assessment were the EPA National Center for Environmental Assessment (NCEA) and the Health Effects Assessment Summary Tables (HEAST) (U.S. EPA 1997b). The following hierarchy was used for selection for toxicity values:

- IRIS.
- Provisional Peer Reviewed Toxicity Values (PPRTVs) (NCEA values in database).
- HEAST.
- Other values including other NCEA values that are not in the PPRTV database along with values used by state agencies as presented on the RSL table (EPA, 2010a).

Tables 6-1 and 6-2 present oral non-carcinogenic and carcinogenic toxicity values, respectively. Tables 6-3 and 6-4 present inhalation non-carcinogenic and carcinogenic toxicity values, respectively. Appendix B includes the supporting documentation for the COPCs with PPRTVs.

6.1 Non-Carcinogenic Effects

For non-carcinogenic effects, it is assumed that there exists a dose below which no adverse health effects would occur. Below this "threshold" dose, exposure to a COPC can be tolerated without adverse effects. Therefore, for non-carcinogenic effects, a range of exposures exists that can be tolerated. Toxic effects are manifested only when physiologic protective mechanisms are overcome by exposures to a COPC above its threshold level.

The potential for non-carcinogenic health effects resulting from oral or dermal exposure to COPCs is assessed by comparing an exposure estimate (intake or dose) to an RfD. The RfD is expressed in units of mg/kg-day and represents a daily intake of COPC per kilogram of body weight that is not sufficient to cause the threshold effect of concern. An RfD is specific to the COPC, the route of exposure, and the duration over which the exposure occurs.

The potential for non-carcinogenic health effects resulting from inhalation exposure to COPCs is assessed by comparing an exposure concentration in air to an RfC. The RfC is expressed in units of milligrams per cubic meter (mg/m^3).

Dermal RfDs are derived from the corresponding oral values. To derive the dermal RfD, the oral RfD (based on an administered dose) is multiplied by the gastrointestinal tract absorption efficiency factor to determine an RfD based on an absorbed dose rather than an administered dose. The resulting dermal RfD is used to evaluate the dermal (absorbed) dose calculated by the dermal exposure algorithms.

Oral RfDs are presented in Table 6-1. Dermal RfDs and the absorption efficiencies used in their determination are also included in Table 6-1. The absorption efficiencies were obtained from EPA's Risk Assessment Guidance for Superfund (RAGS) Part E Guidance (EPA, 2004). Inhalation RfCs are presented in Table 6-3. The tables also include the primary target organs affected by each listed COPC, where information is available. This information may be used in the risk characterization to segregate risks by target organ effects when the total hazard index is greater than 1.0.

6.2 Carcinogenic Effects

The toxicity information considered in the assessment of potential carcinogenic risks includes slope factors, unit risk factors, and a weight-of-evidence narrative consistent with EPA's 2005 Guidelines for Carcinogenic Risk Assessment (EPA, 2005). These guidelines use standard narrative descriptors (Carcinogenic to Humans, Likely to Be Carcinogenic to Humans, Suggestive Evidence of Carcinogenic Potential, Inadequate Information to Assess Carcinogenic Potential, and Not Likely to Be Carcinogenic to Humans) to describe the likelihood that a COPC is a human carcinogen and are based on an evaluation of the available data from human and animal studies.

The CSF is the toxicity value used to quantitatively express the carcinogenic hazard of cancer-causing COPCs via oral and dermal routes. It is defined in the IRIS glossary as: "An upper-bound, approximately a 95 percent confidence limit, on the increased cancer risk from a lifetime exposure to an agent. This estimate, usually expressed in units of proportion (of a population) affected per mg/kg-day, is generally reserved for use in the low-dose region of the dose-response relationship, that is, for exposures corresponding to risks less than 1 in 100" (EPA 2010b).

Dermal CSFs are derived from the corresponding oral values. To derive the dermal CSF, the oral CSF is divided by the gastrointestinal absorption efficiency factor to determine a CSF based on an absorbed dose rather than an administered dose.

Inhalation risks are determined through use of inhalation URFs (EPA 2009). Inhalation URF, measured in units of $(\mu g/m^3)^{-1}$, is the incremental lifetime cancer risk per unit chemical concentration in air for a 70-kilogram adult with lifetime exposure at an inhalation rate of 20 m^3/day.

Oral CSFs are presented in Table 6-2. Dermal CSFs and the absorption efficiencies used in their determination are also included in Table 6-2. The absorption efficiencies were obtained from EPA's RAGS Part E Guidance (EPA, 2004). Inhalation URFs for the COPCs are presented in Table 6-4.

6.3 Analytes Without Toxicity Values

There were a number of analytes detected in the various media that do not have toxicity factors available to evaluate the potential health effects associated with exposure (see Table 4-1 through 4-7). The majority of these analytes were analyzed in samples collected by USGS from the sediment and surface water of Ely Brook, Schoolhouse Brook, and the Ompompanoosuc River (see Tables 4-3 and 4-4). These analytes are listed below.

Bismuth [b]	Lanthanum [a, d]	Terbium [d]
Cerium [d]	Neodymium [d]	Tellurium
Cesium [d]	Niobium	Thallium
Chloride	Phosphorous [a, b]	Thulium
Dysprosium [d]	Praseodymium [d]	Thorium [a]
Erbium [d]	Rubidium [a, b, c, d]	Titanium
Europium [d]	Samarium [d]	Tungsten [a, b]
Gadolinium [d]	Scandium [c, d]	Uranium [a, b, c, d]
Gallium [a, b]	Silica	Ytterbium [d]
Germanium	Sulfate	Yttrium [d]
Holmium [d]	Sulfide	
Indium	Tantalum	

[a] Site levels similar to Schoolhouse Brook background sediment levels.
[b] Site levels similar to Ompompanoosuc River background sediment levels.
[c] Site levels similar to Schoolhouse Brook background sediment levels.
[d] Site levels similar to Ompompanoosuc River background sediment levels.

Due to the lack of toxicity values for these analytes as evidenced by the lack of RSLs for screening, risks cannot be calculated. EPA's Provisional Peer-Reviewed Toxicity Values database was searched and none of the analytes listed above had provisional toxicity values. However, to put the Site levels of these analytes into perspective, simple numerical comparisons of maximum detected site concentrations with background levels were performed, if background data were available. Because these analytes were detected in the sediment and surface water from Schoolhouse Brook and the Ompompanoosuc River, those areas were the focus of the background evaluation. Tables 4-3 and 4-4 contain a column showing the site-specific background levels. Note that background data were not available for Ely Brook sediment and surface water. The analytes marked in the table above have site concentrations that are similar to background. The background analysis shows that site levels are similar to background levels for a number of the analytes without toxicity values.

7.0 EXPOSURE ASSESSMENT

The objective of the exposure assessment is to estimate the nature, extent, and magnitude of potential exposure of humans to COPCs considering both current and future uses. The exposure assessment involves several steps, which are listed below:

- Evaluating the exposure setting, including describing current and future land and water uses and identifying potentially exposed human populations.

- Developing the conceptual site model for human exposures including sources, release mechanisms, transport and receiving media, exposure media, exposure scenarios, exposure routes, and potentially exposed populations.

- Identifying the exposure scenarios, models, and parameters with which to calculate exposure doses.

- Calculating exposure point concentrations (EPCs) for each of the exposure scenarios and routes.

The exposure assessment defines and evaluates the exposures that may be experienced by a receptor population. To have an exposure, several factors must be present: there must be a source of contamination, there must be a mechanism through which a receptor can come into contact with the COPCs in that medium, and there must actually (or potentially) be a receptor present at the point of contact.

7.1 Conceptual Site Model

This section and Figure 7-1 present the conceptual site model (CSM) for the Site as it pertains to COPC exposure and risk to human receptors. A CSM facilitates a consistent and comprehensive evaluation of the risks to human health by creating a framework for identifying the exposure routes or pathways by which human health may be impacted by COPCs predicted to exist at the Site and affected areas. A CSM depicts the relationships between the following elements necessary to construct a complete exposure pathway:

- Sources of contamination and potential COPCs;
- Contaminant release mechanisms and transport pathways;
- Exposure mechanisms and exposure routes; and
- Potentially exposed populations.

The CSM was developed to provide the basis for identifying the potential risks to human health. The model considers the current and future conditions within the Site, and the actual or potential receptors that might come into contact with the COPCs and indicates those exposure routes that are carried through the quantitative risk assessment for each receptor. An objective of developing the CSM is to focus attention on those pathways that contribute the most to the potential impacts on human health and to provide the rationale for screening out other exposure pathways that are minor components of the overall risk.

7.1.1 Sources of Contamination and Potential COPCs

Historical mining operations at the Ely Mine Copper Site have resulted in the deposition of piles of waste rock, tailings, roasted ore, and slag that are the sources of the Site contamination issues.

7.1.2 COPC Release Mechanisms and Transport Pathways

There are four primary mechanisms that can release and transport COPCs at the Site: surface water runoff, leaching into groundwater, seeps, and wind erosion. Surface water runoff occurs during precipitation events or snow melts when COPCs in the soil and waste piles are released and transported to other areas on-site and off-site via Site drainage features. Precipitation, snow melt, surface water, and groundwater which comes into contact with iron sulfide ore minerals, dominantly pyrrhotite, in the waste rock/tailings and bedrock results in weathering (oxidation) and leaching of the ore and host rock through a series of chemical reactions that define the primary mechanism by which acid drainage is generated at the Site (Seal et al., 2001 and Hammarstrom et al., 2001). The resultant low pH of drainage from these sources carries significant concentrations of elements and base metals that along with high acidity and high sulfate concentrations impact the surface waters downstream from the Site. In addition, groundwater from the underground mine pool may contribute to the release of contaminants through the discharge of acid mine drainage via surface flow from mine openings and through fractures in the bedrock. In addition, water erosion of surface waste materials may result in mass transport of potentially acid- and metal-generating materials into the stream sediment downgradient of the Site.

Due to the barren to poorly-vegetated nature of the surface waste materials, wind transport of fines also has the potential to spread these materials beyond the footprint of the piles. As a result, surface soil, subsurface soil, sediment, surface water and groundwater at the Site and proximal to the Site are potentially impacted by Site sources. In addition, surface water and sediment distal from the Site along streams downgradient are potentially affected.

7.1.3 Exposure Media and Routes of Exposure

The potentially contaminated media of concern include soil, surface water, sediment, and groundwater. The list below presents these media along with the likely routes of exposure:

- Soil – COPCs in the soil may be incidentally ingested and absorbed through the skin by exposed humans. In addition, COPCs adsorbed onto particulate released from the soil into the air would be available for inhalation. Individuals could be exposed to the surface soil (0 to 1 ft bgs) and the surface/subsurface soil combined (0 to 10 ft bgs).

- Surface Water – COPCs in the surface water may be incidentally ingested and absorbed through the skin by exposed humans.

- Sediment – COPCs in the sediment may be incidentally ingested and absorbed through the skin by exposed humans.

- Groundwater – COPCs in the groundwater may be ingested and absorbed through the skin by exposed humans while showering and bathing.

The consumption of game obtained while hunting (deer, waterfowl, etc.) in the area could be a potential pathway of exposure. However, this pathway was eliminated from consideration for the following reasons:

- Minimal potential for the metals of concern to bioaccumulate in the edible tissues of these animals. These pathways are typically of concern from potential exposure to lipophilic organic compounds like PCBs and dioxin/furans. The Site COPCs are not lipophilic and are likely to be regulated by a number of mechanisms, such as metabolism and elimination that preclude the accumulation of significant concentrations in edible tissue.

- Deer generally range across hundreds of acres and would be exposed to a wide range of habitats, most of which would likely be completely uncontaminated by the Site. In addition, the most critical exposure to deer at this Site would be incidentally ingested soil (and possibly sediment to a lesser degree) and given the nature of the typical diet – browse (leaves and shoots of woody plants), forbs (broad-leafed weeds and flowering plants), and mast (fruits and nuts) – a deer would be unlikely to consume a significant amount of incidentally ingested soil. Also, given the number of sources of surface water available, it is unlikely that this would be a significant exposure route.

- Ducks typically feed on invertebrates and aquatic vegetation, and have a limited rate of sediment consumption. As noted above, the metals of concern, while potentially high in the sediment, are not likely to bioaccumulate to a significant degree in the edible tissue of ducks or other waterfowl.

In addition, given the nature of metals in general and the potential exposure pathways at this Site, other pathways such as inhalation, incidental soil/sediment ingestion, and dermal absorption are likely to result in significantly higher exposures to both child and adult receptors than any of the above consumption of tissue pathways.

7.1.4 Potentially Exposed Populations

The HHRA focuses on those human populations likely to be exposed to the potentially contaminated Site media currently and/or in the future. There are a number of activities that may lead to contact with Site media including: riding ATVs, hunting, birding, horseback riding, spelunking, hiking, and adolescent gatherings. Of these activities, riding ATVs appears to be a common activity as indicated by the trails and tracks in and around the Site. It was also conservatively assumed that the Site could be used for residential purposes in the future. Based on the current and potential future land and water uses, four potentially exposed populations were evaluated in the HHRA. These potentially exposed populations include:

- Current/future recreational visitors (adolescent and adult) – soil exposure to the recreational visitors was based on riding ATVs because this is a common recreational activity at the Site that could result in an intensive level of soil contact. The ATV riding exposure was based on conservative assumptions that cover the potential exposure associated with other, less-intensive soil contact activities. It was assumed that the recreational visitors contact the on-site piles and the surface soil surrounding the Site. Therefore, the incidental soil ingestion, the dermal contact and absorption, and the inhalation pathways were evaluated for these receptors.

- Current/future swimmers/waders (adolescent and adult) – the swimmers/waders were assumed to contact the surface water and sediment while engaging in recreational activities in Ely Brook and the downstream water bodies. The incidental ingestion and the dermal contact and absorption pathways were evaluated for these receptors.

- Current/future residents (young child and adult) – it is possible that the nearby residents use the Site on a regular basis. This type of exposure is assumed to continue into the future. Therefore, residential exposure was evaluated for the current and future uses of the Site. The current residents were assumed to contact the surface soil and the future

residents were assumed to contact the surface and subsurface soil as a result of soil mixing during future excavation and construction activities. The incidental soil ingestion, the dermal contact and absorption, and the inhalation pathways were evaluated for residential receptors. Local area residents currently use groundwater as their source of potable water. This is expected to continue in the future. Exposure to groundwater assuming the local residents ingest the groundwater underlying the Site through the ingestion and showering/bathing exposure routes were evaluated for both current and future use scenarios.

- Future construction workers – the Site may undergo some type of construction activities at some point in the future, which may result in contact with surface and subsurface soil (top 10 feet assumed). Therefore, the incidental soil ingestion, the dermal contact and absorption, and the inhalation pathways were evaluated for these future receptors. The duration of intensive contact with the Site soil during construction activities such as excavation is expected to be short.

The generation of dust containing COPCs as a result of wind erosion, riding ATVs, and construction activities and the subsequent inhalation by exposed populations is an important route of potential exposure for the Ely Mine Site. EPA's Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites (SSG) (EPA 2002a) was used to estimate emissions. Dust emissions as a result of wind erosion were modeled to evaluate residential inhalation exposure. Emissions as a result of heavy truck traffic on unpaved roads were used to estimate inhalation exposure to recreational visitors while riding ATVs and to workers during construction activities. Appendix C presents the calculations of the particulate emission factors (PEFs) from wind erosion and truck traffic on unpaved roads. Inhalation exposure and risks were estimated using EPA's RAGS Part F guidance (EPA 2009).

7.2 Exposure Parameters

The exposure parameters that were used to calculate the exposure doses (chronic daily intakes or CDIs) for each receptor population through the applicable exposure routes are presented in Tables 7-1 through 7-4. The dermal factors are presented on Table 7-5. Two types of exposure doses were calculated depending on whether the COPC is considered to be carcinogenic. In the first model, the doses were averaged over the assumed exposure duration and were used to

evaluate the potential for non-cancer health effects (i.e. the average daily dose [ADD]). The second model, in which the doses were averaged over a 70-year lifetime, was used to evaluate potential carcinogenic risk (i.e. the lifetime average daily dose [LADD]). The exposure doses were expressed as either administered (oral, inhalation) or absorbed (dermal) doses, in milligrams of COPC per kilogram body weight per day (mg/kg-day).

To provide a range of exposure and risks, the reasonable maximum exposure (RME) and central tendency exposure (CTE) scenarios were evaluated (EPA, 1992). The RME, an estimate of the high-end exposure in a population, is based on a combination of average and high-end estimates of exposure parameters typically representing the 90th percentile or greater of actual expected exposure. The CTE represents an estimate of the average exposure in a population and is based on central estimates of exposure parameters. Both the RME and CTE were evaluated for each exposure scenario.

7.2.1 Current/Future Recreational Visitors

The recreational visitors were exposed to surface soil (0 to 1 ft bgs). Table 7-1 presents the exposure factors for the recreational visitors. The adolescent was assumed to be exposed from 10 to 18 years of age. Thus, the exposure duration (ED) for the adolescent was 8 years. For the adult, an ED of 24 years was used based on the assumption that the adult visitor is a nearby resident. The adolescent body weight (BW) was 52 kg. This value is the average body weight for males and females ages 10 to 18 (see Tables 7-6 and 7-7 of EPA, 1997a). The adult body weight will be 70 kg (EPA, 2002a). The aforementioned exposure parameters were used for the RME and CTE.

- The recreational visitors were assumed to be exposed to Site soil for 8 months of the year (April through November). The visitors are not expected to contact the soil during January, February, March, and December. For the RME, an exposure frequency (EF) of 3 days/week (assumes 4.33 weeks per month) was assumed. This equates to approximately 104 days/year. The CTE EF was 35 days/year (1 day/week).

- The incidental soil ingestion rate (IRS) was assumed to be 100 mg/day. This value represents the adult IRS conventionally used for residential exposure (EPA 2002a) and was used for the RME and CTE. The fraction ingested (FI) was 1.0 for the RME

indicating that 100 percent of the amount of ingested soil will be come from the Site. The FI was reduced to 0.5 for the CTE.

- The exposed skin surface area (SA) was assumed to consist of the head, hands, forearms, lower legs, and feet. Using the data provided in Exhibit C-1 in RAGS Part E (EPA 2004), the SAs for the adolescent and adult were 5,900 cm^2 and 6,900 cm^2, respectively. These values were for the RME. The exposed SA for the CTE consisted of the hands, forearms, and lower legs. This resulted in SAs of 3,600 cm^2 and 4,500 cm^2 for the adolescent and adult, respectively.

- The soil-to-skin adherence factor (AF) for the RME was based on the geometric mean value for the heavy equipment operators activity (0.2 mg/cm^2) (EPA 2004). The AF from this activity was selected for the recreational visitors because it is assumed to represent an upper-end activity for individuals riding ATVs. The CTE AF was based on the geometric mean value for the commercial/industrial gardening activity (0.1 mg/cm^2).

- It was assumed that the recreational visitors were at the Site for two hours per day for the RME. The exposure time (ET) for the CTE was 1 hour per day. As previously mentioned, the PEF was based on heavy truck traffic on unpaved roads according to the SSG (EPA 2002a).

7.2.2 Current/Future Swimmers/Waders

Tables 7-2 and 7-3 present the exposure factors for the swimmer/waders. The ED and BW values described in Section 7.2.1 for the recreational visitors were also used for the swimmers/waders. The swimmers/waders were assumed to be exposed to sediment and surface water for 5 months of the year (May through September) when the weather is warmer and conducive to water contact activities for 1 day/week (assumes 4.33 weeks per month). This equates to an EF of approximately 22 days/year for the RME. The EF was 11 days/year for the CTE.

- The IRS was assumed to be 100 mg/day. The FI was 1.0 for the RME and 0.5 for the CTE.

- The SAs for sediment exposure was assumed to consist of the head, hands, forearms, lower legs, and feet. Therefore, the SAs for the adolescent and adult were 5,900 cm^2 and 6,900 cm^2, respectively. These values were for the RME. The exposed SA for the CTE consisted of the hands, forearms, and lower legs. This resulted in SAs of 3,600 cm^2 and 4,500 cm^2 for the adolescent and adult, respectively

- The RME AF was based on the geometric mean value for the reed gatherers (0.32 mg/cm^2) (EPA 2004). The AF from this activity was selected for the swimmers/waders because it is assumed to represent an upper-end activity for individuals wading and contacting sediment. The CTE AF was based on the geometric mean value for the residential gardeners (0.07 mg/cm^2) (EPA 2004).

- For the RME, surface water exposure was assumed to last for 2 hours. The duration of the CTE exposure was 1 hour. The incidental surface water ingestion rate was 0.05 L/hr. While swimming, it was assumed that the individual is fully immersed. Thus, the SAs for the adolescent and adult were 14,900 cm^2 and 18,000 cm^2, respectively.

7.2.3 Current/Future Resident

Tables 7-1 and 7-4 present the exposure factors for the residents. The total ED for the RME was 30 years (6 years for young child and 24 years for adult). The CTE ED was 15 years (6 years for young child and 9 years for adult) (EPA, 1997a). The RME and CTE EF was 350 days/year.

- The IRS values were 100 mg/day and 200 mg/day for the adult and child, respectively (EPA 2002a; 2002b). The RME FI for soil was 1. The CTE FI was 0.5.

- The SA was 2,800 cm^2 for the child (head, hands, forearms, lower legs, and feet) and 5,700 cm^2 for the adult (head, hands, forearms, and lower legs) (EPA, 2004). The values were used for the RME and CTE.

- The RME AF for the child was the geometric mean value for the daycare child (0.2 mg/cm^2). The adult RME AF was the geometric mean value for the resident gardener (0.07 mg/cm^2). For the CTE, geometric mean AFs of 0.04 mg/cm^2 (children

playing in dry soil) and 0.01 mg/cm^2 (residential gardeners) were used for the child and adult, respectively (EPA 2004).

- Inhalation of dusts generated as a result of wind erosion was determined by conventional techniques presented in the SSG (EPA 2002a). The RME ET was 24 hours/day. The CTE ET was 16 hours/day.

- The groundwater ingestion rate (IRW) values were 2 L/day and 1 L/day for the adult and child, respectively (EPA 2002a; 2002b). The values were used for the RME and CTE. The RME FI was 1. The CTE FI was 0.5.

- The SAs for exposure while bathing/showering were 6,600 cm^2 and 18,000 cm^2 for the child and adult, respectively. For the RME, the child bathing time was 1 hour/event and the adult showering time was 0.58 hour/event (35 minutes) (EPA 2004). The RME bathing/showering time values were divided by 2 for the CTE.

7.2.4 Future Construction Worker

Table 7-1 presents the exposure factors for the construction worker. The adult construction worker was assumed to be exposed for 60 days/year (i.e. 5 days/week for 12 weeks) for the RME and 30 days/year for the CTE. The IRS was assumed to be 330 mg/day (EPA 2002a) for the RME. The ingestion rate for other outdoor workers (100 mg/day) was used for the CTE. The FI was assumed to be 1.

The SA was assumed to consist of the 50th percentile values for the head, hands, and forearms of males and females (i.e. 3,300 cm^2) and applied to both the RME and CTE. The RME AF was 0.24 mg/cm^2, which represents the geometric mean value for the utility workers activity. The CTE AF was 0.14 mg/cm^2, which represents the geometric mean value for the construction workers activity. The RME ET was 8 hours/day. The CTE ET was 4 hours/day. The PEF was based on heavy truck traffic on unpaved roads according to the SSG (EPA, 2002a).

7.3 Exposure Point Concentrations

According to EPA regional guidance (EPA, 1994a), risk assessments are conducted using an EPC for each COPC. The EPC represents the concentration to which a receptor is assumed to

be continuously exposed while in contact with an environmental medium. For soil, sediment, and surface water exposure, the EPC is generally defined as the 95 percent upper confidence limit on the mean (UCL) and is calculated using EPA's ProUCL software (Version 4.00.05, EPA 2010c). For groundwater, the EPC is represented by the maximum detected concentration for each COPC from the available wells. The EPCs are presented on Tables 7-6 through 7-11.

7.3.1 Soil, Sediment, and Surface Water

The following general guidelines were used to determine UCLs for the soil, sediment, and surface water. The same EPC value was used for both RME and CTE scenarios.

- If fewer than 8 samples were collected, the maximum detected concentration was selected as the EPC.

- If 8 or more samples were collected and the dataset contained more than 5 percent but less than 50 percent detects and at least 4 detects, a nonparametric-based UCL (either Kaplan-Meier (KM) or bootstrapping derived), as per ProUCL's non-parametric-based UCL recommendation, was calculated. Note that the bootstrapping method was not considered unless there were at least 10 detects.

- If 8 or more samples were collected and the dataset contained at least 50 percent detects, the appropriate distribution of the dataset was determined and UCLs were selected as guided by the ProUCL supporting documentation. Note that for datasets with censored results (i.e. non-detects), UCLs calculated using estimation procedures (e.g., KM, bootstrapping) were considered instead of employing the simple substitution method (e.g., using one-half the SQL for non-detects) for selecting appropriate UCLs as guided by the ProUCL supporting documentation.

Supporting documentation (ProUCL Outputs) for the calculation of the UCLs is presented in Appendix D. Exposure point concentrations for soil, sediment, and surface water are presented in Tables 7-6 through 7-9.

7.3.2 Groundwater

Exposure points for groundwater exposures were identified as current monitoring well locations, as each monitoring well could represent a potential future water supply well (EPA, 1994a). Ideally, the EPC for a given COPC in groundwater is represented by the maximum temporal average concentration from all of the exposure points (i.e. monitoring well locations). The use of the maximum temporal average as the EPC assumes that a sufficient number of sampling events have been conducted over a sufficient period of time so as to reliably characterize a temporal average (EPA, 1999). This was not the case for the Ely Mine Site where between one and five sampling rounds were available for the monitoring wells. As a result, the maximum detected groundwater concentrations were used as the EPCs. This will result in an over-estimation of groundwater risks. EPCs were derived for each COPC in the three water bearing units on Site (overburden, shallow bedrock, and deep bedrock). In addition, EPCs were derived for the off-site residential groundwater. The groundwater EPCs are presented on Tables 7-10 and 7-11.

8.0 RISK CHARACTERIZATION

This section provides a characterization of the potential human health risks associated with the potential exposure to COPCs in various media at the Site. Section 8.1 outlines the methods used to estimate the type and magnitude of health risks and Section 8.2 presents the risk characterization results for the current and potential future land and water use conditions at the Site.

8.1 Risk Characterization Methodology

Potential human health risks resulting from exposure to COPCs were estimated using algorithms established by EPA. The methods described by EPA are protective of human health and are likely to overestimate (rather than underestimate) risk. The methodology uses specific algorithms to calculate risk as a function of COPC concentration, human exposure parameters, and toxicity. Risks from COPCs are calculated for either carcinogenic or non-carcinogenic effects. Some carcinogenic COPCs may also exhibit non-carcinogenic effects, in which case, potential impacts are characterized for both types of health effects.

8.1.1 Carcinogens

Risks attributable to exposure to carcinogens are estimated as the probability of an individual developing cancer over a lifetime as a result of exposure to a potential carcinogen. The incremental lifetime cancer risk (ILCR) is determined as follows (EPA 1989):

$$ILCR = Intake \times CSF \text{ or } URF$$

where:
- ILCR = Incremental lifetime cancer risk, expressed as a unitless probability
- Intake = Intake (mg/kg-day or $\mu g/m^3$), a function of exposure and COPC concentration
- CSF = Cancer slope factor (mg/kg-day)-1
- URF = Unit risk factor ($\mu g/m^3$)-1

Risks below 1×10^{-6} (less than 1 in 1 million) are generally considered to be acceptable by EPA, and risks greater than 1×10^{-4} (1 in 10,000) are generally considered to be unacceptable. Risks between 1×10^{-6} and 1×10^{-4} are generally considered to be within EPA's targeted cancer risk range. Risks are estimated for all carcinogenic COPCs with available CSFs or URFs.

8.1.2 Non-Cancer Health Effects

The hazards associated with non-cancer effects of COPCs are evaluated by comparing an exposure level or intake to an RfD or RfC. The ratio of the intake to the RfD is called the hazard quotient (HQ) and is defined as follows (EPA 1989):

$$HQ = \frac{Intake}{RfD \text{ or } RfC}$$

where:
- HQ = Hazard Quotient (unitless)
- Intake = Intake (mg/kg-day or mg/m^3), a function of exposure and COPC concentration
- RfD = Reference dose (mg/kg-day)
- RfC = Reference concentration (mg/m^3)

If the ratio of the intake to the RfD or RfC exceeds 1.0, there exists a potential for non-carcinogenic (toxic) effects to occur. A Hazard Index (HI) is generated by summing the individual HQs for all COPCs. If the value of the HI exceeds 1.0, there is a potential for non-carcinogenic health effects associated with that particular COPC mixture, and therefore it is necessary to segregate the HQs by target organ effects. The HQ should not be construed as a probability, but rather as a numerical indicator of the extent to which a predicted intake exceeds or is less than an RfD or RfC.

8.1.3 Lead

Risks from lead exposure were not evaluated using the same methodology as other COPCs. The Integrated Exposure Uptake Biokinetic (IEUBK) Model and Technical Review Work Group Model for lead were used to address exposures to lead. These models estimate blood-lead concentrations. Blood-lead concentration is the most widely used index of internal lead body burdens associated with potential adverse health effects of lead. Studies indicate that infants and young children are more susceptible to adverse effects from exposure to lead than adults. Considerable behavioral and developmental impairments have been noted in children with elevated blood-lead levels. Since children are a more sensitive subpopulation than adults, exposure to lead by adults in a residential or recreational scenario is not generally evaluated and the receptor of concern for these scenarios is the young child. Evaluation of the young child in a residential or recreational scenario is considered protective of adults, including pregnant women. Exposures to lead by workers were evaluated by use of an approach that focuses on estimating fetal blood-lead concentrations in women exposed to lead-contaminated soil in non-residential scenarios. Therefore, the receptor of concern in the worker scenario is the fetus of the pregnant worker. Evaluation of the fetus of the pregnant worker is considered protective of non-pregnant workers, including those present less frequently than the typical full-time worker. The threshold for toxic effects to children from lead is believed to be in the range of 10 micrograms/deciliter (µg/dL) to 15 µg/dL. Blood-lead levels (either fetal or young child) greater than 10 µg/dL are considered to be a "concern."

Potential residential child exposure to lead in soil and drinking water was evaluated using EPA's IEUBK model for lead in children (EPA, 1994b). The IEUBK Model is designed to estimate blood levels of lead in children (under 7 years of age) based on either default or site-specific input values for air, drinking water, diet, dust, and soil exposure.

Exposures to lead in soil by non-residential adults were evaluated by use of a slope-factor approach developed by the EPA Technical Review Work Group for Lead (EPA 2003). The slope factor approach focuses on estimating fetal blood-lead concentrations in women exposed to lead-contaminated soil in non-residential scenarios. The model estimates the 95th percentile blood-lead concentration among fetuses born to women having site exposures.

Concentrations (i.e. blood-lead levels) calculated using both approaches were compared to the established blood-lead level of concern of 10 µg/dL. An additional step in the process estimates the probability that fetal blood-lead levels will exceed 10 µg/dL. EPA's stated goal for lead is that individuals exposed would have no more than a 5 percent probability of exceeding the level of concern of 10 µg/dL. Exposure concentrations, as well as default parameters for some input parameters, were used in the evaluations. The EPCs selected for use in this evaluation are the arithmetic average lead concentrations.

8.2 Risk Results

A summary of the risk results is provided in this section. The risks are presented using a series of embedded tables (Tables 8-1 through 8-6). The reader is referred to Appendix E to view the full series of RAGS Part D Tables 7, 9, and 10. Figures 8-1 through 8-6 present the risk results in graphical format. Appendix F presents example calculations. The following sections discuss risk results by affected media.

8.2.1 Soil Exposure

The following receptors were evaluated for potential contact with the Site soil:

- Current/future recreational visitor exposure to surface soil.
- Future construction worker exposure to total soil.
- Current resident exposure to surface soil.
- Future resident exposure to total soil.

Cancer Risks

The ILCRs calculated for potential exposure to Site soil are summarized below in Table 8-1. Figure 8-1 presents the ILCRs in graphical format.

Table 8-1
Soil Exposure ILCRs
Ely Copper Mine Superfund Site
Vershire, Vermont

	RME	CTE
Current/future Recreational Visitor		
Adolescent	6.7E-07	1.1E-07
Adult	1.8E-06	2.8E-07
Future Construction Worker	7.4E-07	1.4E-07
Current Resident		
Child	4.9E-06	2.3E-06
Adult	2.2E-06	3.8E-07
Future Resident		
Child	4.1E-06	2.0E-06
Adult	1.9E-06	3.2E-07

The RME and CTE cancer risk estimates for the evaluated receptors were within or less than EPA's cancer risk range.

Non-Cancer Hazards

Table 8-2 presents a summary of the HIs for the soil exposure. Figure 8-2 presents the HIs in graphical format.

Table 8-2
Soil Exposure Hazard Indices
Ely Copper Mine Superfund Site
Vershire, Vermont

	RME	CTE
Current/future Recreational Visitor		
Adolescent	0.32	0.056
Adult	0.29	0.048
Future Construction Worker	2.3	0.43
Current Resident		
Child	3.1	1.6
Adult	0.34	0.17
Future Resident		
Child	4.5	2.3
Adult	0.49	0.24

RME and CTE HIs were greater than 1.0 for current and future child residents, with cobalt (thyroid effects) and iron (gastrointestinal) as the primary contributors to the total HIs. The RME HI was greater than 1.0 for future construction workers; however, no individual COPC HI exceeded 1.0. The total HI for the nervous system was 1.1 (aluminum, manganese, and mercury). The CTE HI was less than 1.0. The HIs for the recreational visitors were less than 1.0.

Exposure to Lead

Lead was selected as a COPC for the Site surface soil and total soil. The average lead concentration in the surface soil dataset was 47 mg/kg. The average lead concentration in the total soil dataset was 40 mg/kg. For conservatism, the average lead concentration in the surface soil was used as the EPC in the lead models for soil exposure. EPA's IEUBK model estimated that the geometric mean blood-lead concentration among current and future residential children exposed to Site soil would be 1.3 µg/dL. This is less than EPA's established level of concern of 10 µg/dL. The probability that the fetal blood-lead concentration exceeds 10 µg/dL is less than 0.001 percent. EPA's target probability is 5 percent or less. The Adult Lead Model (June 2009 Version) was run for the workers. Depending on the baseline blood-lead levels assumed, the estimated geometric mean blood-lead levels ranged from 1.1 to 1.6 µg/dL. The probability that the fetal blood-lead concentration exceeds 10 µg/dL is less than 1 percent. In conclusion, exposures to lead in Site soil do not exceed EPA's target level of concern.

8.2.2 Sediment and Surface Water Exposure

Swimmers/waders were evaluated for potential contact with sediment and surface water. The following sediment and surface water exposure areas were evaluated:

- Ely Brook.
- Schoolhouse Brook.
- East Branch of the Ompompanoosuc River.

Cancer Risks

The ILCRs calculated for potential exposure to sediment and surface water are summarized in Table 8-3. Figure 8-3 presents the ILCRs in graphical format.

Table 8-3
Sediment and Surface Water Exposure ILCRs
Ely Copper Mine Superfund Site
Vershire, Vermont

	RME	CTE
Ely Brook		
Adolescent	1.2E-07	2.3E-08
Adult	2.8E-07	5.2E-08
Schoolhouse Brook		
Adolescent	4.4E-08	8.1E-09
Adult	1.0E-07	1.9E-08
East Branch of the Ompompanoosuc River		
Adolescent	1.6E-07	2.9E-08
Adult	3.7E-07	6.6E-08

The RME and CTE cancer risk estimates for the evaluated receptors were less than EPA's cancer risk range.

Non-Cancer Hazards

Hazard indices developed for the sediment and surface water exposure are shown in Table 8-4. Figure 8-4 presents the HIs in graphical format.

Table 8-4
Sediment and Surface Water Exposure Hazard Indices
Ely Copper Mine Superfund Site
Vershire, Vermont

	RME	CTE
Ely Brook		
Adolescent	0.17	0.043
Adult	0.14	0.034
Schoolhouse Brook		
Adolescent	0.028	0.0070
Adult	0.021	0.0053
East Branch of the Ompompanoosuc River		
Adolescent	0.020	0.0048
Adult	0.015	0.0036

The RME and CTE HIs for the evaluated receptors were less than the non-cancer benchmark of 1.0.

Exposure to Lead

Lead was not identified as a COPC in the sediment. However, for conservatism the average lead concentration in the Ely Brook sediment (25 mg/kg) was assumed. Lead was identified as a COPC in Schoolhouse Brook surface water. The average lead concentration in surface water was 2.3 µg/L. The average lead concentrations were used as EPCs in the lead models.

As a conservative measure, the IEUBK model was run assuming young child exposure to sediment and surface water. The model estimated that the geometric mean blood-lead concentration for child swimmers and waders exposed to sediment and surface water would be 0.94 µg/dL. This is less than EPA's established level of concern of 10 µg/dL. The probability that the fetal blood-lead concentration exceeds 10 µg/dL is less than 0.001 percent. Thus, exposures to lead in sediment and surface water do not exceed EPA's target level of concern.

8.2.3 Groundwater Exposure

Groundwater exposure was evaluated assuming residential use. Risks were calculated for the on-site groundwater by water bearing unit (i.e. overburden, shallow bedrock, deep bedrock) and the off-site groundwater collected from residential wells.

Cancer Risks

Table 8-5 presents the ILCRs calculated for potential exposure to groundwater. Figure 8-5 presents the ILCRs in graphical format.

Table 8-5
Groundwater Exposure ILCRs
Ely Copper Mine Superfund Site
Vershire, Vermont

	RME	CTE
Overburden		
Child	6.3E-05	3.1E-05
Adult	1.1E-04	2.0E-05
Shallow Bedrock		
Child	4.0E-05	2.0E-05
Adult	6.9E-05	1.3E-05
Deep Bedrock		
Child	2.2E-05	1.1E-05
Adult	3.8E-05	7.1E-06
Residential Wells		
Child	Not Calculated	
Adult	Not Calculated	

The RME cancer risk from the overburden exposure to the adult was greater than the upper end of EPA's cancer risk range. This was due to arsenic exposure. The RME and CTE cancer risk estimates for the other water bearing units were within EPA's cancer risk range. Cancer risks were not calculated for the residential well data because the COPCs identified in the residential groundwater were not carcinogenic.

Non-Cancer Hazards

Hazard indices developed for the groundwater exposure are shown in Table 8-6. Figure 8-6 presents the HIs in graphical format.

Table 8-6
Groundwater Exposure Hazard Indices
Ely Copper Mine Superfund Site
Vershire, Vermont

	RME	CTE
Overburden		
Child	274	137
Adult	117	59
Shallow Bedrock		
Child	141	70
Adult	60	30
Deep Bedrock		
Child	55	28
Adult	24	12
Residential Wells		
Child	0.32	0.16
Adult	0.13	0.067

RME and CTE HIs were greater than 1.0 for exposure to Site groundwater. In each case, cobalt (thyroid effects) was the primary contributor to the groundwater HIs followed by copper (gastrointestinal effects including indigestion, heartburn, irritable bowel syndrome, constipation, and diarrhea). The other COPCs with HIs greater than one included: manganese (nervous system), arsenic (skin), iron (gastrointestinal effects including constipation, nausea, gastric pain, and diarrhea), aluminum (nervous system), cadmium (kidney effects), and nickel (systemic toxicity). The HIs based on exposure to the residential wells were less than one.

Exposure to Lead

Lead was selected as a COPC for the overburden groundwater and the shallow bedrock groundwater. The average lead concentration in the overburden dataset was 7.3 µg/L. The average lead concentration in the shallow bedrock dataset was 7.2 µg/L. The average lead concentration in the overburden bedrock was used as the EPC in the lead model.

EPA's IEUBK model estimated that the geometric mean blood-lead concentration among future residential children exposed to Site groundwater would be 3.0 µg/dL. This is less than EPA's established level of concern of 10 µg/dL. The probability that the fetal blood-lead concentration exceeds 10 µg/dL is 0.50 percent. EPA's target probability is 5 percent or less. In conclusion, exposures to lead in Site groundwater do not exceed EPA's target level of concern.

8.2.4 Cumulative Risks

The risks presented in the previous subsections were by exposure media. It is possible that individuals could be exposed to more than one of the affected media over the duration of the potential exposure. The objective of this subsection is to present the cumulative risks associated with the Site. Table 8-7 presents the exposure scenarios that were combined to calculate the cumulative risks. For the purposes of simplifying the cumulative risk analysis, Ely Brook sediment and surface water exposures were assumed. The Site groundwater risks from the overburden were used in the cumulative risk analysis.

Table 8-7
Combined Exposure Scenarios for Cumulative Risk Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

Exposure Scenario	Soil Exposure	Sediment/Surface Water Exposure	Site Groundwater Exposure	Residential Groundwater Exposure
Current Recreational Visitor	●	●		●
Future Recreational Visitor	●	●	●	
Current Resident	●	●		●
Future Resident	●	●	●	
Future Construction Worker	●	●		●

It was assumed that current recreational visitors would contact the Site soil as well the sediment and surface water in Ely Brook while visiting the Site. These same receptors were also assumed to use the sampled residential wells as their source of groundwater. In the future, the recreational visitors are assumed to use the Site groundwater as their water source assuming the Site is developed. The same groundwater assumptions were employed for the current and future residential receptors. The future construction worker was assumed to contact the Site soil along with the sediment and surface water in Ely Brook. Tables 8-8 and 8-9 summarize the cumulative cancer risks and non-cancer HIs, respectively.

Table 8-8
Cumulative RME Cancer Risks
Ely Copper Mine Superfund Site
Vershire, Vermont

Exposure Scenario	Soil Exposure	Sediment/ Surface Water Exposure	Site Groundwater Exposure	Residential Groundwater Exposure	Total
Current Recreational Visitor - Adolescent	6.7E-07	1.2E-07	NA	NA	7.9E-07
Current Recreational Visitor - Adult	1.8E-06	2.8E-07	NA	NA	2.1E-06
Future Recreational Visitor - Adolescent	6.7E-07	1.2E-07	1.1E-04	NA	1.1E-04
Future Recreational Visitor - Adult	1.8E-06	2.8E-07	1.1E-04	NA	1.1E-04
Current Resident - Child	4.9E-06	1.2E-07	NA	NA	5.0E-06
Current Resident - Adult	2.2E-06	2.8E-07	NA	NA	2.5E-06
Future Resident - Child	4.1E-06	1.2E-07	6.3E-05	NA	6.7E-05
Future Resident - Adult	1.9E-06	2.8E-07	1.1E-04	NA	1.4E-04
Future Construction Worker	7.4E-07	2.8E-07	NA	NA	1.0E-06

Notes:

1. The current recreational visitors were assumed to be exposed to Site surface soil, sediment and surface water from Ely Brook, and groundwater from off-site residential wells (cancer risk were not calculated for the off-site wells because the COPCs are not carcinogenic).
2. The future recreational visitors were assumed to be exposed to Site surface soil, sediment, and surface water from Ely Brook, and Site overburden groundwater. Future recreational exposure to the soil is limited to the surface soil. It was assumed that any future recreational activities will occur in the undeveloped areas of the Site thereby precluding exposure to the subsurface soil. The groundwater risk to the future adult resident was used as a surrogate for the future adolescent and adult recreational visitors.
3. The current residents were assumed to be exposed to Site surface soil, sediment and surface water from Ely Brook, and groundwater from off-site residential wells (cancer risk were not calculated for the off-site wells because the COPCs are not carcinogenic).
4. The future residents were assumed to be exposed to Site total soil (0 to 10 ft bgs), sediment, and surface water from Ely Brook, and Site overburden groundwater.
5. The future construction workers were assumed to be exposed to Site total soil and sediment and surface water from Ely Brook.

As presented in Table 8-8, the cumulative risks that involved exposure to Site groundwater were greater than the EPA cancer risk range. The cancer risks associated with soil, sediment, and surface water exposure were within or less than the EPA cancer risk range.

Table 8-9
Cumulative RME Hazard Indices
Ely Copper Mine Superfund Site
Vershire, Vermont

Exposure Scenario	Soil Exposure	Sediment/ Surface Water Exposure	Site Groundwater Exposure	Residential Groundwater Exposure	Total
Current Recreational Visitor - Adolescent	0.32	0.17	NA	0.13	0.62
Current Recreational Visitor - Adult	0.29	0.14	NA	0.13	0.56
Future Recreational Visitor - Adolescent	0.32	0.17	117	NA	117
Future Recreational Visitor - Adult	0.29	0.14	117	NA	117
Current Resident - Child	3.1	0.17	NA	0.32	3.6
Current Resident - Adult	0.34	0.14	NA	0.13	0.61
Future Resident - Child	4.5	0.17	274	NA	279
Future Resident - Adult	0.49	0.14	117	NA	118
Future Construction Worker	2.3	0.14	NA	NA	2.4

Notes:

1. The current recreational visitors were assumed to be exposed to Site surface soil, sediment, and surface water from Ely Brook, and groundwater from off-site residential wells. The off-site groundwater hazard to the adult resident was used as a surrogate for the current adolescent and adult recreational visitors.
2. The future recreational visitors were assumed to be exposed to Site surface soil, sediment, and surface water from Ely Brook, and Site overburden groundwater. Future recreational exposure to the soil is limited to the surface soil. It was assumed that any future recreational activities will occur in undeveloped areas of the Site thereby precluding exposure to the subsurface soil. The groundwater hazard to the adult resident was used as a surrogate for the future adolescent and adult recreational visitors.
3. The current residents were assumed to be exposed to Site surface soil, sediment, and surface water from Ely Brook, and groundwater from off-site residential wells.
4. The future residents were assumed to be exposed to Site total soil (0 to 10 ft bgs), sediment, and surface water from Ely Brook, and Site overburden groundwater.
5. The future construction workers were assumed to be exposed to Site total soil and sediment and surface water from Ely Brook.

The cumulative HIs presented in Table 8-9 are dominated by the HIs from residential exposure to Site groundwater and soil. The recreational exposures to soil, sediment, and surface water are not a concern. Exposure to the off-site residential wells is not a concern.

It was also assumed that the future adult resident could be exposed to Site soils during routine residential activities and during recreational use in undeveloped areas of the Site. Table 8-10

presents the cumulative risks associated with this hypothetical exposure. Table 8-10 focuses on adult exposure. It is unrealistic to assume that young children would be engaged in recreational activities in the undeveloped areas of the Site. As shown, the total cancer risk was within the acceptable risk range and the non-cancer hazard was less than one.

Table 8-10
Cumulative RME Cancer Risk and Hazard Index from
Future Residential and Recreational Exposure to Soil
Ely Copper Mine Superfund Site
Vershire, Vermont

Exposure Scenario	Residential Soil Exposure	Recreational Soil Exposure	Total
Cancer Risk			
Adult	1.9E-06	1.8E-06	3.7E-06
Non-cancer Hazard			
Adult	0.49	0.29	0.78

9.0 UNCERTAINTY ANALYSIS

There are uncertainties associated with all HHRAs. This section summarizes these uncertainties, and discusses how they may affect the calculated risks discussed in Section 8.0. There is uncertainty associated with all steps of the risk assessment process. Uncertainty in the data evaluation is associated with sampling adequacy, the current status of the predictive databases for development of screening values, the procedures used to include or exclude constituents as COPCs, and the methods used and the assumptions made to determine exposure point concentrations. The selection of COPCs is based on exposure assumptions and toxicity information, which in turn have associated uncertainties. Uncertainty associated with the exposure assessment includes the values used as input variables for a given intake route and the predictions regarding future land use and population characteristics. Uncertainty in the toxicity assessment includes the quality of the existing data to support dose-response relationships and the weight-of-evidence used for determining the carcinogenicity of COPCs. Uncertainty in risk characterization includes that associated with exposure to multiple COPCs and the cumulative uncertainty from combining conservative assumptions made in earlier activities.

While there are various sources of uncertainty (as described above) throughout the risk assessment process, assumptions were made so that the calculated risks would be conservative estimates that are protective of public health. Thus, the resultant uncertainty in the numerical risk assessment is weighted toward an overestimate of risk.

Generally, risk assessments carry two types of uncertainty: measurement and informational uncertainty. Measurement uncertainty refers to the variance that can be attributed to sampling techniques and laboratory analysis of contaminants. For example, this type of uncertainty is associated with analytical data collected for each location. The risk assessment reflects the accumulated variances of the individual values used. Informational uncertainty refers to estimates of toxicity and exposure. Often this gap is significant, such as the absence of information on the effects of human exposure to low doses of a COPC, the biological mechanism of action of a COPC, or the behavior of a COPC in soil.

Once the risk assessment is complete, the results must be reviewed and evaluated to identify the type and magnitude of uncertainty involved. Reliance on results from a risk assessment without considering uncertainties, limitations, and assumptions inherent in the process can be misleading. For example, to account for uncertainties in the development of exposure assumptions, conservative estimates must be made to ensure that the particular assumptions made are protective of sensitive subpopulations or the maximum exposed individuals. If a number of conservative assumptions are combined in an exposure model, the resulting calculations can propagate the uncertainties associated with those assumptions, thereby producing a much larger uncertainty for the final results. This uncertainty is biased toward over-predicting both carcinogenic and non-carcinogenic risks. Thus, both the results of the risk assessment and the uncertainties associated with those results must be considered when making risk management decisions.

This interpretation is especially relevant when the risks exceed the point-of-departure for defining "acceptable" risk. For example, when risks calculated using a high degree of uncertainty are below an "acceptable" risk level (1×10^{-6}), the interpretation of no significant risk is straightforward. However, when risks calculated using a high degree of uncertainty are above an "acceptable" risk level (1×10^{-4}), a conclusion can be difficult unless uncertainty is considered. The risk estimates alone may indicate unacceptable risk; however, if uncertainty is

biased toward over-predicting risk to a significant degree, actual risks could fall within the acceptable range.

Uncertainties within the components of the HHRA for the Site are discussed below.

9.1 Uncertainty in Data Evaluation

9.1.1 COPC Selection

There is a minor amount of uncertainty associated with the selection of COPCs in the quantitative risk assessment. Conservative screening values were used to select COPCs; thus, it is unlikely that any contaminant that may pose a risk was eliminated from the risk assessment calculations. There were analytes detected for which EPA has little or no information regarding the analyte's toxicity. Contaminants without screening values and with no acceptable surrogate values were not evaluated as COPCs, which could underestimate risk.

A COPC was selected if the maximum detected concentration in soils, groundwater, sediments, surface water, or fish tissue exceeded its respective risk-based screening criterion. Frequency of detection was not used to eliminate COPCs. Most of the COPCs identified as driving risk in the risk estimates were detected in a majority of samples. Based on this review of the data, the absence of using frequency of detection as a COPC selection criterion is unlikely to have impacted the risk estimates.

Concentrations of some COPCs were also detected in soil, groundwater, sediment, and surface water collected from background locations. No contaminants were excluded as COPCs based on background conditions. In general, by including contaminants that are present at a site at levels consistent with background levels, the HHRA overestimates site-related risk to a minor degree. Comparisons of Site and background levels are typically performed for COPCs that resulted in elevated risk levels.

9.1.2 Background Comparison

The residential soil and groundwater exposure scenarios were the only exposures that resulted in elevated risk levels. The background dataset for groundwater consisted of a single sample. Thus, meaningful comparisons of Site and background concentrations could not be performed.

Numerical comparisons of the Site soil concentrations of cobalt, copper, and iron (the primary risk contributors) to background levels is presented below in Tables 9-1 and 9-2. In both comparisons, which include surface soil and total soil, the Site concentrations exceeded the background levels. Therefore, not including a background comparison for Site soils would overestimate risk to a small degree.

Table 9-1
Background Comparison – Surface Soil
Ely Copper Mine Superfund Site
Vershire, Vermont

COPC	Site Maximum (mg/kg)	Site Average (mg/kg)	Background Maximum (mg/kg)	Background Average (mg/kg)
Cobalt	200	18	16	6.6
Copper	23000	964	45	15
Iron	300000	53810	31000	13852

Table 9-2
Background Comparison – Total Soil
Ely Copper Mine Superfund Site
Vershire, Vermont

COPC	Site Maximum (mg/kg)	Site Average (mg/kg)	Background Maximum (mg/kg)	Background Average (mg/kg)
Cobalt	1500	28	16	6.6
Copper	36000	1200	45	15
Iron	300000	55835	31000	13852

9.1.3 XRF Data

Approximately 350 samples were analyzed using XRF techniques, of these, 45 were also analyzed at an off-site fixed laboratory (i.e. there were a total of 45 paired laboratory and XRF samples). Appendix A evaluates the XRF data and compares it with the paired samples that were submitted for laboratory analysis to determine the potential impact the non-paired XRF data might have on the HHRA results if they were used to estimate exposure point concentrations.

The results of the paired sample comparisons indicate that there is relatively good comparability between the laboratory and XRF results. The few anomalies observed in the percent difference comparison are likely a result of the analytical variability associated with both methods. As a result of these findings, there is little concern that the non-paired XRF samples, if included in exposure analysis, would significantly impact the exposure concentrations used in the HHRA.

9.2 Uncertainty in the Exposure Assessment

Uncertainty in the exposure assessment arises from the selection of receptors and selection of exposure parameters. Each is discussed below.

Exposure Routes and Receptor Identification

Selection of exposure routes and receptors may result in either under- or over-estimation of the risks, with the final result dependent on how well the receptors were defined. Given the remote location of the mine and the unlikely future residential exposure at the Site, it is anticipated that the series of exposed receptors assumed in this HHRA would likely overestimate actual risk.

Selection of Exposure Parameters

Each exposure factor selected for use in this risk assessment has some associated uncertainty. Generally, exposure factors are based on surveys of physiological and lifestyle profiles across the United States. The attributes and activities studied in these surveys generally have a broad distribution. To avoid underestimation of exposure, EPA guidelines on the RME receptor were used that generally consist of the 90^{th} percentile or greater for most parameters. Therefore, the selected values for the RME receptor represent the upper bound of the observed or expected habits of the majority of the population.

Many of the exposure parameters were determined from statistical analyses of human population characteristics. Often the database used to summarize a particular exposure parameter (i.e. body weight) is quite large. Consequently, the values chosen for such variables in the RME scenario have low uncertainty. For many parameters for which limited information exists (i.e. dermal absorption from soil), there is greater uncertainty.

Many of the quantities used to calculate exposures and risks in this report were selected from a distribution of possible values. For the RME scenario, the value representing the 95th percentile is generally selected for each parameter to ensure that the assessment bounds the actual risks from a postulated exposure. When combined, these assumptions tend to overestimate actual exposure to a significant degree.

In order to evaluate a central tendency estimate of exposure, EPA has suggested the use of the CTE receptor, whose intake variables are set at approximately the 50th percentile of the distribution. The risks for this receptor are designed to represent the range of uncertainty associated with various intake assumptions. Many of the parameters were estimated using professional judgment, although EPA Region 1 default parameters were used where available.

9.2.1 Construction Worker Exposure

It could be possible that the construction worker exposure evaluated in the HHRA does not fully cover the range of potential exposures associated with construction activities should they occur at the Site. The exposure frequency for the future construction worker exposure scenario used in the HHRA was 60 days/year. This is based on intensive soil contact activities, such as earth-moving, that are required during the construction of homes occurring 5 days per week for 12 weeks. These types of activities were assumed to occur in the warm weather months. This was considered a reasonable estimate of construction duration. However, it could be possible that construction activities last for a longer duration and that the workers involved in these activities have a higher level of soil adherence to the skin. Table 9-3 presents a matrix of varying risk levels based on increased exposure frequency and soil adherence. The EF was increased to 125 days/year, 180 days/year, and 250 days/year. The increased EFs are likely an over-estimate of the frequency of exposure as construction activities are not expected to occur into the colder months of the year when the weather is not conducive to outdoor construction activities. The soil-to-skin AF was increased from 0.24 mg/cm^2 to 0.3 mg/cm^2.

As shown on Table 9-3, the risk levels increase as the level of exposure increases. This relationship is linear and is to be expected. The EF is the driving variable in the risk increase. The original total cancer risk was less than 1E-06, the low end of EPA's acceptable risk range. The increased total cancer risk levels are slightly above the low end of the acceptable cancer risk range. The original total non-cancer HI was marginally above the non-cancer benchmark of

one with HQs less than one for all of the COPCs. The HQs for cobalt and manganese exceed one when the potential exposure increases. The aluminum HQ slightly exceeds one at the highest level of potential exposure.

Table 9-3
Impact of Increased Exposure and Soil Adherence on Construction Worker Risks
Ely Copper Mine Superfund Site
Vershire, Vermont

Contaminant of Potential Concern	Cancer Risks				Hazard Quotients			
	HHRA Result	Increased Exposure and Dermal Adherence			HHRA Result	Increased Exposure and Dermal Adherence		
		125 d/y	180 d/y	250 d/y		125 d/y	180 d/y	250 d/y
Aluminum	---	---	---	---	0.25	0.53	0.76	1.1
Antimony	---	---	---	---	0.0020	0.0043	0.0061	0.0085
Arsenic	5.6E-08	1.2E-07	1.7E-07	2.3E-07	0.022	0.047	0.067	0.093
Cadmium	4.9E-09	1.0E-08	1.5E-08	2.0E-08	0.021	0.043	0.062	0.086
Chromium	---	---	---	---	0.000025	0.000052	0.000075	0.000104
Cobalt	6.6E-07	1.4E-06	2.0E-06	2.8E-06	0.99	2.1	3.0	4.1
Copper	---	---	---	---	0.037	0.076	0.11	0.15
Iron	---	---	---	---	0.075	0.16	0.22	0.31
Manganese	---	---	---	---	0.85	1.8	2.5	3.5
Mercury	---	---	---	---	0.00037	0.00076	0.0011	0.0015
Molybdenum	---	---	---	---	0.00099	0.0021	0.0030	0.0041
Nickel	1.0E-08	2.2E-08	3.1E-08	4.4E-08	0.032	0.067	0.10	0.13
Selenium	---	---	---	---	0.0035	0.0073	0.010	0.015
Vanadium	---	---	---	---	0.0022	0.0046	0.0066	0.0092
Zinc	---	---	---	---	0.00068	0.0014	0.0020	0.0028
Total	7.4E-07	1.5E-06	2.2E-06	3.1E-06	2.3	4.8	6.9	9.5

9.3 Uncertainty in the Toxicological Evaluation

A toxicity evaluation is a COPC's hazard identification and dose-response assessment. The hazard identification deals with characterizing the nature and strength of the evidence of causation, or the likelihood that a COPC that induces adverse effects in animals will also induce adverse effects in humans. Hazard identification of carcinogenicity is an evaluation of the weight-of-evidence that a COPC causes cancer. Positive animal cancer test data suggest that humans contain tissue(s) that may also manifest a carcinogenic response; however, the animal data cannot necessarily be used to predict the target tissue in humans. In the hazard

assessment of non-cancer effects, however, positive animal data suggest the nature of the effects (the target tissues and type of effects) anticipated in humans.

The RfD for vanadium sulfate was used to quantify non-cancer hazard from vanadium exposure as opposed to using the more conservative RfD associated with metallic vanadium. Information provided by USGS indicates that vanadium, as an oxyanion, could exist in several forms at the Site, but that none would be in the elemental state. Thus, the toxicity associated with vanadium sulfate was used.

Uncertainty is associated with evaluating exposures to lead. Lead exposures were evaluated by use of the IEUBK model. This model represents a conservative approach for predicting blood lead levels in children and is designed to overestimate risks. Lead exposures by workers were evaluated by use of the Adult Lead Model (2009 Version). This approach focuses on estimating fetal blood-lead concentrations in women exposed to lead-contaminated soils in non-residential scenarios. Uncertainty is associated with estimating maternal blood-lead concentrations and with the relationship between maternal blood-lead concentrations and fetal blood-lead concentrations.

Finally, the risk characterization does not consider antagonistic or synergistic effects. Little or no information is available to determine the potential for antagonism or synergism for the COPCs. Therefore, this uncertainty cannot be evaluated for its impact on the risk assessment, since it may either underestimate or overestimate potential human health risk.

9.4 Site-Related Risk Evaluation

As presented in Section 8.2, the non-cancer HIs for residential exposure to soil exceeded the non-cancer benchmark of one. Three COPCs (cobalt, copper, and iron) were the primary contributors to the HIs that exceeded one. It is likely that the calculated HIs included a contribution from concentrations of these COPCs related to background levels. The objective of this section is to calculate HIs associated with background levels and subtract the background risks from the site risks to yield the residual site-related risks. The analysis is based on the RME.

Table 9-4 presents the maximum detected concentrations, the arithmetic means, and the 95 percent UCLs for cobalt, copper, and iron in background soil. HIs based on the background

95 percent UCL concentrations were calculated for the residential child exposure scenario (see Table 9-5). The child receptor was used because it results in a higher HI than the adult. These background HIs were subtracted from the site HIs to yield site-related HIs. The results of this evaluation are shown on Table 9-6, which presents the original site HI and the HIs associated with background levels, along with the site-related HIs that represent the original HIs minus the background HIs. The HIs decrease 23 percent and 16 percent from the estimated total Site HIs, but even with this decrease the Site-related HIs exceeded the non-cancer benchmark of one.

10.0 SUMMARY OF HUMAN HEALTH RISK ASSESSMENT

The objective of the HHRA was to evaluate the potential cancer risks and non-cancer health effects associated with exposure to Site COPCs. Recreational, worker, and residential exposure scenarios were developed to achieve this objective. The HHRA was performed using health-protective estimates of potential exposure and conservative estimates of COPC toxicity and estimates of COPC concentration levels. A qualitative evaluation of the uncertainties in the HHRA process indicates that the HHRA likely overestimates the potential human health exposure and risk associated with the Ely Copper Mine Superfund Site.

The results of the HHRA indicate the following:

- Exposure to Site groundwater results in an elevated cancer risk for the overburden and HQs/HIs greater than one for all of the water bearing units.

- Residential exposure to Site soil resulted in non-cancer HQs/HIs that were greater than one.

- Recreational exposure to Site soil, sediment, and surface water resulted in negligible levels of risk.

- The residential scenario provides an upper-bound of the potential risks associated with the Site. The predominant exposures associated with the Site (recreational) have been shown to result in risk below levels of concern.

- Exposures to lead do not exceed EPA's target level of concern.

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TABLES

Table 3-1
Summary of Total Chromium Samples and Hexavalent Chromium Samples Collected in November 2010
Ely Copper Mine Superfund Site
Vershire, Vermont

Sample ID	Total Chromium		Hexavalent Chromium	
	Sample Result	MDL	Sample Result	MDL
SED74	26	12	ND	1.2
SED75	25	12	ND	1.1
SED76	15	2	ND	0.94
SED77	20	12	ND	1.1
SS40	23	12	ND	0.93
SS41	61	12	ND	0.88
SS42	ND	16	ND	0.95
SS43	54	12	ND	0.96
SS44	14	12	ND	0.96
SS44 Dup	ND	12	ND	0.98
SS45	63	16	ND	1.1
SS46	33	12	ND	0.96
SS47	ND	12	ND	0.93
SS48	25	12	ND	0.98
SS49	33	12	ND	1
SS50	19	12	ND	0.95
SS51	14	12	ND	0.96
SS52	37	12	ND	0.94
SS53	26	12	ND	0.92

Notes:
Units in mg/kg.

Table 3-2
Summary of Soil Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Sample ID	Date	Depth Range (feet)
Background	Background	ELY-SS-BK-07A (0-3)	10/16/2007	0 - 0.25
Background	Background	ELY-SS-BK-07B (0-3)	10/16/2007	0 - 0.25
Background	Background	ELY-SS-BK-07C (0-4)	10/16/2007	0 - 0.33
Background	Background	ELY-SS-BK-08A (0-10)	10/16/2007	0 - 0.83
Background	Background	ELY-SS-BK-08B (0-7)	10/16/2007	0 - 0.58
Background	Background	ELY-SS-BK-08C (0-12)	10/16/2007	0 - 1
Background	Background	ELY-SS-BK-10A (0-6)	10/16/2007	0 - 0.5
Background	Background	ELY-SS-BK-10B (0-1)	10/16/2007	0 - 0.08
Background	Background	ELY-SS-BK-10C (0-6)	10/16/2007	0 - 0.5
Background	Background	ELY-SS-BK-10C (0-6) DUP	10/16/2007	0 - 0.5
Background	Background	ELY-SS-BK-14A (0-1)	10/16/2007	0 - 0.08
Background	Background	ELY-SS-BK-14B (0-8)	10/16/2007	0 - 0.67
Background	Background	ELY-SS-BK-14C (0-1)	10/16/2007	0 - 0.08
Background	Background	ELY-SS-BK-15A (0-2)	10/16/2007	0 - 0.17
Background	Background	ELY-SS-BK-15B (0-0.5)	10/16/2007	0 - 0.04
Background	Background	ELY-SS-BK-15C (0-2.5)	10/16/2007	0 - 0.21
Site		ELY-SB-1_2_3	12/7/2006	0 - 0
Site		ELY-SB-7_8	12/7/2006	0 - 0
Site	Lower Waste Piles and Roast Bed	SS-11X-090309AX	9/3/2009	0 - 0.5
Site	Lower Waste Piles and Roast Bed	SS-12X-090309AX	9/3/2009	0 - 0.5
Site	Lower Waste Piles and Roast Bed	SS-13X-082809AX	8/28/2009	0 - 0.3
Site	Lower Waste Piles and Roast Bed	SS-14X-083109AX	8/31/2009	0 - 0.2
Site	Lower Waste Piles and Roast Bed	SS-15X-083109AX	8/31/2009	0 - 0.2
Site	Lower Waste Piles and Roast Bed	SS-16X-090309AX	9/3/2009	0 - 0.5
Site	Lower Waste Piles and Roast Bed	SS-17X-083109AX	8/31/2009	0 - 0.3
Site	Lower Waste Piles and Roast Bed	SS-18X-082709AX	8/27/2009	0 - 1
Site	Lower Waste Piles and Roast Bed	SS-19X-082709AX	8/27/2009	0 - 1
Site	Lower Waste Piles and Roast Bed	SS-20X-083109AX	8/31/2009	0 - 0.2
Site	Lower Waste Piles and Roast Bed	SS-21X-083109AX	8/31/2009	0 - 0.2
Site	Lower Waste Piles and Roast Bed	SS-22X-082709AX	8/27/2009	0 - 1
Site	Lower Waste Piles and Roast Bed	SS-23X-082709AX	8/27/2009	0 - 1
Site	Lower Waste Piles and Roast Bed	SS-24X-083109AX	8/31/2009	0 - 0.2
Site	Lower Waste Piles and Roast Bed	SS-25X-082709AX	8/27/2009	0 - 1
Site	Lower Waste Piles and Roast Bed	SS-26X-083109AX	8/31/2009	0 - 0.3
Site	Lower Waste Piles and Roast Bed	SS-27X-082609AX	8/26/2009	0 - 1
Site	Lower Waste Piles and Roast Bed	SS-28X-083109AX	8/31/2009	0 - 1
Site	Lower Waste Piles and Roast Bed	SS-29X-083109AX	8/31/2009	0 - 0.3
Site	Lower Waste Piles and Roast Bed	TF-11X-090209AX	9/2/2009	0 - 0.5
Site	Lower Waste Piles and Roast Bed	TF-11X-090909AX	9/2/2009	0 - 0.5
Site	Lower Waste Piles and Roast Bed	TF-13X-090909BX	9/2/2009	0 - 0.5
Site	Lower Waste Piles and Roast Bed	TG-02X-082409AX	8/24/2009	0 - 1
Site	Lower Waste Piles and Roast Bed	TH-01X-082509AX	8/25/2009	0 - 1
Site	Lower Waste Piles and Roast Bed	TI-03X-082509AX	8/25/2009	0 - 1
Site	Lower Waste Piles and Roast Bed	TK-01X-082509AX	8/25/2009	0 - 1
Site	Lower Waste Piles and Roast Bed	TP-16X-080309AX	8/3/2009	0 - 1
Site	Lower Waste Piles and Roast Bed	MW-20A-072909AX	7/29/2009	2 - 2.4
Site	Lower Waste Piles and Roast Bed	MW-20A-072909BX	7/29/2009	2.4 - 4
Site	Lower Waste Piles and Roast Bed	MW-20A-072909CX	7/29/2009	4 - 5.5
Site	Lower Waste Piles and Roast Bed	MW-20A-083109AX	8/31/2009	0 - 2
Site	Lower Waste Piles and Roast Bed	MW-21A-072809AX	7/28/2009	2 - 4

Table 3-2
Summary of Soil Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Sample ID	Date	Depth Range (feet)
Site	Lower Waste Piles and Roast Bed	MW-21A-072809BX	7/28/2009	4 - 6
Site	Lower Waste Piles and Roast Bed	MW-21A-072809CD	7/28/2009	6 - 10
Site	Lower Waste Piles and Roast Bed	MW-21A-072809CX	7/28/2009	6 - 10
Site	Lower Waste Piles and Roast Bed	MW-21A-083109AX	8/31/2009	0 - 2
Site	Lower Waste Piles and Roast Bed	MW-22A-072909AX	7/29/2009	2 - 4
Site	Lower Waste Piles and Roast Bed	MW-22A-072909BX	7/29/2009	7.1 - 8.2
Site	Lower Waste Piles and Roast Bed	MW-22A-083109AX	8/31/2009	0 - 2
Site	Lower Waste Piles and Roast Bed	MW-23A-070809AX	7/8/2009	0 - 2
Site	Lower Waste Piles and Roast Bed	MW-23A-070809BX	7/8/2009	6 - 8
Site	Lower Waste Piles and Roast Bed	MW-23A-070809CX	7/8/2009	8 - 10
Site	Lower Waste Piles and Roast Bed	MW-23A-083109AX	8/31/2009	0 - 2
Site	Lower Waste Piles and Roast Bed	SB-09X-073009AX	7/30/2009	2.2 - 3
Site	Lower Waste Piles and Roast Bed	SB-09X-073009BX	7/30/2009	3 - 6
Site	Lower Waste Piles and Roast Bed	SB-09X-073009CX	7/30/2009	8 - 10
Site	Lower Waste Piles and Roast Bed	SS-20X-083109BX	8/31/2009	0.2 - 1.5
Site	Lower Waste Piles and Roast Bed	SS-27X-082609BX	8/26/2009	1 - 2
Site	Lower Waste Piles and Roast Bed	SS-30X-083109AD	8/31/2009	0 - 2
Site	Lower Waste Piles and Roast Bed	SS-30X-083109AX	8/31/2009	0 - 2
Site	Lower Waste Piles and Roast Bed	TE-09X-090109AX	9/1/2009	0 - 2
Site	Lower Waste Piles and Roast Bed	TE-12X-090109BX	9/1/2009	0 - 2.5
Site	Lower Waste Piles and Roast Bed	TE-15X-090109CX	9/2/2009	2.5 - 4
Site	Lower Waste Piles and Roast Bed	TF-02X-090109AX	9/1/2009	0 - 2
Site	Lower Waste Piles and Roast Bed	TF-13X-090209BX	9/2/2009	1 - 3.5
Site	Lower Waste Piles and Roast Bed	TF-17X-090109AX	9/1/2009	0 - 2
Site	Lower Waste Piles and Roast Bed	TP-16X-080309BX	8/3/2009	1 - 2
Site	Lower Waste Piles and Roast Bed	TP-16X-080309CX	8/3/2009	2 - 5
Site	Midslope Waste Piles	ELY-SS-NF-05 (0-3)	10/16/2007	0 - 0.25
Site	Midslope Waste Piles	ELY-SS-NF-05 (3-11)	10/16/2007	0.25 - 0.92
Site	Midslope Waste Piles	ELY-SS-NF-06 (0-3)	10/16/2007	0 - 0.25
Site	Midslope Waste Piles	ELY-SS-NF-06 (3-12)	10/16/2007	0.25 - 1
Site	Midslope Waste Piles	ELY-SS-NF-07 (0-2)	10/17/2007	0 - 0.17
Site	Midslope Waste Piles	ELY-SS-NF-07 (2-8)	10/17/2007	0.17 - 0.67
Site	Midslope Waste Piles	ELY-SS-NF-08 (0-2)	10/16/2007	0 - 0.17
Site	Midslope Waste Piles	ELY-SS-NF-08 (2-11)	10/16/2007	0.17 - 0.92
Site	Midslope Waste Piles	ELY-SS-NF-09 (0-1.5)	10/17/2007	0 - 0.13
Site	Midslope Waste Piles	ELY-SS-NF-09 (1.5-12)	10/17/2007	0.13 - 1
Site	Midslope Waste Piles	ELY-SS-NF-10 (0-4)	10/16/2007	0 - 0.33
Site	Midslope Waste Piles	ELY-SS-NF-10 (4-12)	10/16/2007	0.33 - 1
Site	Midslope Waste Piles	ELY-SS-NF-11 (0-3)	10/16/2007	0 - 0.25
Site	Midslope Waste Piles	ELY-SS-NF-11 (3-9)	10/16/2007	0.25 - 0.75
Site	Midslope Waste Piles	ELY-SS-NF-12 (0-2)	10/19/2007	0 - 0.17
Site	Midslope Waste Piles	ELY-SS-NF-12 (2-9)	10/19/2007	0.17 - 0.75
Site	Midslope Waste Piles	ELY-SS-NF-13 (0-2.5)	10/19/2007	0 - 0.21
Site	Midslope Waste Piles	ELY-SS-NF-13 (2.5-12)	10/19/2007	0.21 - 1
Site	Midslope Waste Piles	ELY-SS-SA-10 (0-6)	10/17/2007	0 - 0.5
Site	Midslope Waste Piles	ELY-SS-SA-11 (0-6)	10/17/2007	0 - 0.5
Site	Midslope Waste Piles	ELY-SS-SA-12 (0-6)	10/17/2007	0 - 0.5
Site	Midslope Waste Piles	ELY-SS-SA-12 (0-6) DUP	10/17/2007	0 - 0.5
Site	Midslope Waste Piles	ELY-SS-SA-13 (0-6)	10/17/2007	0 - 0.5
Site	Midslope Waste Piles	ELY-SS-SA-14 (0-6)	10/17/2007	0 - 0.5

Table 3-2
Summary of Soil Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Sample ID	Date	Depth Range (feet)
Site	Midslope Waste Piles	ELY-SS-SA-15 (0-6)	10/17/2007	0 - 0.5
Site	Midslope Waste Piles	ELY-SS-SA-15 (0-6) DUP	10/17/2007	0 - 0.5
Site	Midslope Waste Piles	ELY-SS-SA-16 (0-6)	10/17/2007	0 - 0.5
Site	Midslope Waste Piles	ELY-SS-SA-17 (0-6)	10/16/2007	0 - 0.5
Site	Midslope Waste Piles	ELY-SS-SA-18 (0-6)	10/17/2007	0 - 0.5
Site	Midslope Waste Piles	ELY-SS-SA-19 (0-6)	10/16/2007	0 - 0.5
Site	Midslope Waste Piles	ELY-SS-SA-20 (0-6)	10/17/2007	0 - 0.5
Site	Midslope Waste Piles	ELY-SS-SA-21 (0-6)	10/17/2007	0 - 0.5
Site	Midslope Waste Piles	ELY-SS-SA-22 (0-6)	10/17/2007	0 - 0.5
Site	Midslope Waste Piles	ELY-SS-SA-23 (0-6)	10/19/2007	0 - 0.5
Site	Midslope Waste Piles	ELY-SS-TR-03A (0.5-12)	10/17/2007	0.04 - 1
Site	Midslope Waste Piles	ELY-SS-TR-03A (0-0.5)	10/17/2007	0 - 0.04
Site	Midslope Waste Piles	ELY-SS-TR-03B (0-2)	10/17/2007	0 - 0.17
Site	Midslope Waste Piles	ELY-SS-TR-03B (2-12)	10/17/2007	0.17 - 1
Site	Midslope Waste Piles	ELY-SS-TR-03C (0-3)	10/17/2007	0 - 0.25
Site	Midslope Waste Piles	ELY-SS-TR-03C (3-12)	10/17/2007	0.25 - 1
Site	Midslope Waste Piles	ELY-SS-TR-04A (0-1)	10/17/2007	0 - 0.08
Site	Midslope Waste Piles	ELY-SS-TR-04A (1-12)	10/17/2007	0.08 - 1
Site	Midslope Waste Piles	ELY-SS-TR-04B (0-1.5)	10/17/2007	0 - 0.13
Site	Midslope Waste Piles	ELY-SS-TR-04B (1.5-12)	10/17/2007	0.13 - 1
Site	Midslope Waste Piles	ELY-SS-TR-04C (0-3)	10/17/2007	0 - 0.25
Site	Midslope Waste Piles	ELY-SS-TR-04C (3-6)	10/17/2007	0.25 - 0.5
Site	Midslope Waste Piles	ELY-SS-TR-05A (0-2.5)	10/17/2007	0 - 0.21
Site	Midslope Waste Piles	ELY-SS-TR-05A (2.5-9)	10/17/2007	0.21 - 0.75
Site	Midslope Waste Piles	ELY-SS-TR-05B (0-1)	10/17/2007	0 - 0.08
Site	Midslope Waste Piles	ELY-SS-TR-05B (1-7)	10/17/2007	0.08 - 0.58
Site	Midslope Waste Piles	ELY-SS-TR-05C (0-1)	10/17/2007	0 - 0.08
Site	Midslope Waste Piles	ELY-SS-TR-05C (1-12)	10/17/2007	0.08 - 1
Site	Midslope Waste Piles	ELY-SS-TR-05D (0-3)	10/17/2007	0 - 0.25
Site	Midslope Waste Piles	ELY-SS-TR-05D (3-12)	10/17/2007	0.25 - 1
Site	Midslope Waste Piles	ELY-SS-TR-06A (0-1)	10/19/2007	0 - 0.08
Site	Midslope Waste Piles	ELY-SS-TR-06A (1-12)	10/19/2007	0.08 - 1
Site	Midslope Waste Piles	ELY-SS-TR-06B (0-3)	10/19/2007	0 - 0.25
Site	Midslope Waste Piles	ELY-SS-TR-06B (3-7)	10/19/2007	0.25 - 0.58
Site	Midslope Waste Piles	ELY-SS-TR-06C (0-3)	10/19/2007	0 - 0.25
Site	Midslope Waste Piles	ELY-SS-TR-06C (3-12)	10/19/2007	0.25 - 1
Site	Midslope Waste Piles	ELY-SS-TZ-13 (0-4)	10/17/2007	0 - 0.33
Site	Midslope Waste Piles	ELY-SS-TZ-13 (4-12)	10/17/2007	0.33 - 1
Site	Midslope Waste Piles	ELY-SS-TZ-14 (0-4)	10/17/2007	0 - 0.33
Site	Midslope Waste Piles	ELY-SS-TZ-14 (4-12)	10/17/2007	0.33 - 1
Site	Midslope Waste Piles	ELY-SS-TZ-15 (0-3.5)	10/17/2007	0 - 0.29
Site	Midslope Waste Piles	ELY-SS-TZ-15 (3.5-12)	10/17/2007	0.29 - 1
Site	Midslope Waste Piles	ELY-SS-TZ-16 (0-4)	10/17/2007	0 - 0.33
Site	Midslope Waste Piles	ELY-SS-TZ-16 (4-12)	10/17/2007	0.33 - 1
Site	Midslope Waste Piles	ELY-SS-TZ-17 (0.5-12)	10/17/2007	0.04 - 1
Site	Midslope Waste Piles	ELY-SS-TZ-17 (0-0.5)	10/17/2007	0 - 0.04
Site	Midslope Waste Piles	ELY-SS-TZ-18 (.5-12)	10/17/2007	0.04 - 1
Site	Midslope Waste Piles	ELY-SS-TZ-18 (0-.5)	10/17/2007	0 - 0.04
Site	Midslope Waste Piles	ELY-SS-TZ-19 (.5-12)	10/17/2007	0.04 - 1
Site	Midslope Waste Piles	ELY-SS-TZ-19 (0-.5)	10/17/2007	0 - 0.04

Table 3-2
Summary of Soil Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Sample ID	Date	Depth Range (feet)
Site	Midslope Waste Piles	ELY-SS-TZ-20 (0-1)	10/17/2007	0 - 0.08
Site	Midslope Waste Piles	ELY-SS-TZ-20 (1-12)	10/17/2007	0.08 - 1
Site	Midslope Waste Piles	ELY-SS-TZ-21 (0-5)	10/17/2007	0 - 0.42
Site	Midslope Waste Piles	ELY-SS-TZ-21 (5-12)	10/17/2007	0.42 - 1
Site	Midslope Waste Piles	ELY-SS-TZ-22 (0-2)	10/17/2007	0 - 0.17
Site	Midslope Waste Piles	ELY-SS-TZ-22 (2-10)	10/17/2007	0.17 - 0.83
Site	Midslope Waste Piles	ELY-SS-TZ-23 (0-2.5)	10/17/2007	0 - 0.21
Site	Midslope Waste Piles	ELY-SS-TZ-23 (2.5-12)	10/17/2007	0.21 - 1
Site	Midslope Waste Piles	ELY-SS-TZ-24 (0-1)	10/17/2007	0 - 0.08
Site	Midslope Waste Piles	ELY-SS-TZ-24 (1-12)	10/17/2007	0.08 - 1
Site	Midslope Waste Piles	ELY-SS-TZ-25 (0-3)	10/17/2007	0 - 0.25
Site	Midslope Waste Piles	ELY-SS-TZ-25 (3-11)	10/17/2007	0.25 - 0.92
Site	Midslope Waste Piles	ELY-SS-TZ-26 (0-1.5)	10/17/2007	0 - 0.13
Site	Midslope Waste Piles	ELY-SS-TZ-26 (1.5-12)	10/17/2007	0.13 - 1
Site	Midslope Waste Piles	ELY-SS-TZ-27 (0-2.5)	10/16/2007	0 - 0.21
Site	Midslope Waste Piles	ELY-SS-TZ-27 (2.5-8)	10/16/2007	0.21 - 0.67
Site	Midslope Waste Piles	ELY-SS-TZ-28 (0.5-9)	10/19/2007	0.04 - 0.75
Site	Midslope Waste Piles	ELY-SS-TZ-28 (0-0.5)	10/19/2007	0 - 0.04
Site	Midslope Waste Piles	ELY-SS-TZ-29 (0-1)	10/19/2007	0 - 0.08
Site	Midslope Waste Piles	ELY-SS-TZ-29 (1-9)	10/19/2007	0.08 - 0.75
Site	Midslope Waste Piles	ELY-SS-TZ-30 (0.5-12)	10/19/2007	0.04 - 1
Site	Midslope Waste Piles	ELY-SS-TZ-30 (0-0.5)	10/19/2007	0 - 0.04
Site	Midslope Waste Piles	ELY-SS-TZ-31 (0-3)	10/19/2007	0 - 0.25
Site	Midslope Waste Piles	ELY-SS-TZ-31 (3-9)	10/19/2007	0.25 - 0.75
Site	Midslope Waste Piles	ELY-SS-TZ-32 (0-2)	10/19/2007	0 - 0.17
Site	Midslope Waste Piles	ELY-SS-TZ-32 (2-9)	10/19/2007	0.17 - 0.75
Site	Midslope Waste Piles	ELY-SS-TZ-33 (0-2)	10/18/2007	0 - 0.17
Site	Midslope Waste Piles	ELY-SS-TZ-33 (2-12)	10/18/2007	0.17 - 1
Site	Midslope Waste Piles	ELY-QC-1	12/3/2006	0 - 4
Site	Midslope Waste Piles	ELY-QC-2	12/3/2006	0 - 4
Site	Midslope Waste Piles	ELY-SB-1 (0-4)	12/5/2006	0 - 4
Site	Midslope Waste Piles	ELY-SB-1 (4.5-5.6)	12/5/2006	4.5 - 5.6
Site	Midslope Waste Piles	ELY-SB-2 (0-4)	12/3/2006	0 - 4
Site	Midslope Waste Piles	ELY-SB-2 (6-10)	12/3/2006	6 - 10
Site	Midslope Waste Piles	ELY-SB-3 (0-4)	12/3/2006	0 - 4
Site	Midslope Waste Piles	ELY-SB-3 (4.5-8)	12/3/2006	4.5 - 8
Site	Midslope Waste Piles	ELY-TP4A (2-3)	5/14/2007	2 - 3
Site	Midslope Waste Piles	ELY-TP6 (3-4)	5/14/2007	3 - 4
Site	Midslope Waste Piles	ELY-TP7A (4-5)	5/14/2007	4 - 5
Site	Smelter Area	ELY-SS-NF-01 (0-2)	10/16/2007	0 - 0.17
Site	Smelter Area	ELY-SS-NF-01 (2-7)	10/16/2007	0.17 - 0.58
Site	Smelter Area	ELY-SS-NF-02 (0-8)	10/16/2007	0 - 0.67
Site	Smelter Area	ELY-SS-NF-03 (0-4)	10/16/2007	0 - 0.33
Site	Smelter Area	ELY-SS-NF-03 (4-12)	10/16/2007	0.33 - 1
Site	Smelter Area	ELY-SS-SA-01 (0-2)	10/16/2007	0 - 0.17
Site	Smelter Area	ELY-SS-SA-02 (0-3)	10/16/2007	0 - 0.25
Site	Smelter Area	ELY-SS-SA-02 (0-3) DUP	10/16/2007	0 - 0.25
Site	Smelter Area	ELY-SS-SA-03 (0-6)	10/15/2007	0 - 0.5
Site	Smelter Area	ELY-SS-SA-04 (0-6)	10/15/2007	0 - 0.5
Site	Smelter Area	ELY-SS-SA-05 (0-6)	10/15/2007	0 - 0.5

Table 3-2
Summary of Soil Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Sample ID	Date	Depth Range (feet)
Site	Smelter Area	ELY-SS-SA-06 (0-6)	10/15/2007	0 - 0.5
Site	Smelter Area	ELY-SS-SA-07 (0-6)	10/15/2007	0 - 0.5
Site	Smelter Area	ELY-SS-SA-08 (0-6)	10/15/2007	0 - 0.5
Site	Smelter Area	ELY-SS-SA-09 (0-4)	10/15/2007	0 - 0.33
Site	Smelter Area	ELY-SS-TR-01A (0-1)	10/15/2007	0 - 0.08
Site	Smelter Area	ELY-SS-TR-01A (1-8)	10/15/2007	0.08 - 0.67
Site	Smelter Area	ELY-SS-TR-01B (0-1.5)	10/15/2007	0 - 0.13
Site	Smelter Area	ELY-SS-TR-01B (1.5-8)	10/15/2007	0.13 - 0.67
Site	Smelter Area	ELY-SS-TR-01C (0-2)	10/15/2007	0 - 0.17
Site	Smelter Area	ELY-SS-TR-01C (2-11)	10/15/2007	0.17 - 0.92
Site	Smelter Area	ELY-SS-TR-01D (0.5-10)	10/15/2007	0.04 - 0.83
Site	Smelter Area	ELY-SS-TR-01D (0-0.5)	10/15/2007	0 - 0.04
Site	Smelter Area	ELY-SS-TR-02A (0-1.5)	10/15/2007	0 - 0.13
Site	Smelter Area	ELY-SS-TR-02A (1.5-11)	10/15/2007	0.13 - 0.92
Site	Smelter Area	ELY-SS-TR-02B (0-4)	10/15/2007	0 - 0.33
Site	Smelter Area	ELY-SS-TR-02B (4-11)	10/15/2007	0.33 - 0.92
Site	Smelter Area	ELY-SS-TR-02C (0-3)	10/15/2007	0 - 0.25
Site	Smelter Area	ELY-SS-TR-02C (3-12)	10/15/2007	0.25 - 1
Site	Smelter Area	ELY-SS-TR-02D (0-2)	10/15/2007	0 - 0.17
Site	Smelter Area	ELY-SS-TR-02D (2-12)	10/15/2007	0.17 - 1
Site	Smelter Area	ELY-SS-TR-02D (2-12) DUP	10/15/2007	0.17 - 1
Site	Smelter Area	ELY-SS-TR-02D(0-2) DUP	10/15/2007	0 - 0.17
Site	Smelter Area	ELY-SS-TZ-01 (0-1.5)	10/16/2007	0 - 0.13
Site	Smelter Area	ELY-SS-TZ-01 (1.5-8)	10/16/2007	0.13 - 0.67
Site	Smelter Area	ELY-SS-TZ-02 (0-1.5)	10/16/2007	0 - 0.13
Site	Smelter Area	ELY-SS-TZ-02 (1.5-6)	10/16/2007	0.13 - 0.5
Site	Smelter Area	ELY-SS-TZ-04 (0-2)	10/16/2007	0 - 0.17
Site	Smelter Area	ELY-SS-TZ-04 (0-2) DUP	10/16/2007	0 - 0.17
Site	Smelter Area	ELY-SS-TZ-04 (2-6)	10/16/2007	0.17 - 0.5
Site	Smelter Area	ELY-SS-TZ-04 (2-6) DUP	10/16/2007	0.17 - 0.5
Site	Smelter Area	ELY-SS-TZ-05 (0-1)	10/16/2007	0 - 0.08
Site	Smelter Area	ELY-SS-TZ-05 (0-1)	10/23/2007	0 - 0.08
Site	Smelter Area	ELY-SS-TZ-05 (1-8)	10/16/2007	0.08 - 0.67
Site	Smelter Area	ELY-SS-TZ-05 (1-8)	10/23/2007	0.08 - 0.67
Site	Smelter Area	ELY-SS-TZ-06 (0-1)	10/16/2007	0 - 0.08
Site	Smelter Area	ELY-SS-TZ-06 (1-12)	10/16/2007	0.08 - 1
Site	Smelter Area	ELY-SS-TZ-07 (0-1.5)	10/16/2007	0 - 0.13
Site	Smelter Area	ELY-SS-TZ-07 (1.5-6.5)	10/16/2007	0.13 - 0.54
Site	Smelter Area	ELY-SS-TZ-08 (0.5-10)	10/16/2007	0.04 - 0.83
Site	Smelter Area	ELY-SS-TZ-08 (0-0.5)	10/16/2007	0 - 0.04
Site	Smelter Area	ELY-SS-TZ-09 (0.5-12)	10/18/2007	0.04 - 1
Site	Smelter Area	ELY-SS-TZ-09 (0-0.5)	10/18/2007	0 - 0.04
Site	Smelter Area	ELY-SS-TZ-10 (0-1)	10/15/2007	0 - 0.08
Site	Smelter Area	ELY-SS-TZ-10 (1-10)	10/15/2007	0.08 - 0.83
Site	Smelter Area	ELY-SS-TZ-11 (0-1)	10/15/2007	0 - 0.08
Site	Smelter Area	ELY-SS-TZ-11 (1-5.5)	10/15/2007	0.08 - 0.46
Site	Smelter Area	ELY-SS-TZ-12 (0-3)	10/18/2007	0 - 0.25
Site	Smelter Area	ELY-SS-TZ-12 (3-8)	10/18/2007	0.25 - 0.67
Site	Smelter Area	ELY-TP-104 (0-1)	10/5/2007	0 - 1
Site	Smelter Area	ELY-TP-104 (0-1) DUP	10/5/2007	0 - 1

Table 3-2
Summary of Soil Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Sample ID	Date	Depth Range (feet)
Site	Smelter Area	ELY-TP-109 (0-1)	10/5/2007	0 - 1
Site	Smelter Area	ELY-TP1 (4-5)	5/14/2007	4 - 5
Site	Smelter Area	ELY-TP-100 (4-6)	10/5/2007	4 - 6
Site	Smelter Area	ELY-TP-103 (1-2)	10/5/2007	1 - 2
Site	Smelter Area	ELY-TP-104 (2-3)	10/5/2007	2 - 3
Site	Smelter Area	ELY-TP-106 (2-3)	10/5/2007	2 - 3
Site	Smelter Area	ELY-TP-106 (2-3) DUP	10/5/2007	2 - 3
Site	Smelter Area	ELY-TP-108 (1-2)	10/5/2007	1 - 2
Site	Smelter Area	ELY-TP2B (2-3)	5/14/2007	2 - 3
Site	Smelter Area	ELY-TP3A (1.5-2.5)	5/14/2007	1.5 - 2.5
Site	Smelter Area	ELY-TP3B (2-3)	5/14/2007	2 - 3
Site	Smelter/Slag Area	SS-01X-090209AX	9/2/2009	0 - 0.5
Site	Smelter/Slag Area	SS-02X-090209AX	9/2/2009	0 - 0.5
Site	Smelter/Slag Area	SS-03X-090209AX	9/2/2009	0 - 0.5
Site	Smelter/Slag Area	SS-04X-090209AX	9/2/2009	0 - 0.5
Site	Smelter/Slag Area	SS-05X-090209AX	9/2/2009	0 - 0.3
Site	Smelter/Slag Area	SS-06X-090209AX	9/2/2009	0 - 0.5
Site	Smelter/Slag Area	SS-07X-090209AX	9/2/2009	0 - 0.5
Site	Smelter/Slag Area	SS-08X-090209AX	9/2/2009	0 - 1
Site	Smelter/Slag Area	SS-09X-090209AX	9/2/2009	0 - 1
Site	Smelter/Slag Area	SS-10X-090309AX	9/3/2009	0 - 0.5
Site	Smelter/Slag Area	TA-04X-082409AX	8/24/2009	0 - 1
Site	Smelter/Slag Area	TA-14X-082409AX	8/24/2009	0 - 1
Site	Smelter/Slag Area	TC-03X-082609AX	8/26/2009	0 - 1
Site	Smelter/Slag Area	TD-03X-082809-AX	8/28/2009	0 - 1
Site	Smelter/Slag Area	TD-05X-082809-DX	8/28/2009	0 - 1
Site	Smelter/Slag Area	SS-01X-090209BX	9/2/2009	0.5 - 2
Site	Smelter/Slag Area	SS-02X-090209BX	9/2/2009	0.5 - 1.5
Site	Smelter/Slag Area	SS-03X-090209BX	9/2/2009	0.5 - 2
Site	Smelter/Slag Area	SS-04X-090209BX	9/2/2009	0.5 - 2
Site	Smelter/Slag Area	SS-05X-090209BX	9/2/2009	0.3 - 2
Site	Smelter/Slag Area	SS-06X-090209BX	9/2/2009	0.5 - 2
Site	Smelter/Slag Area	SS-07X-090209BX	9/2/2009	0.5 - 2
Site	Smoke Flue	ELY-SS-NF-04 (0-4)	10/16/2007	0 - 0.33
Site	Smoke Flue	ELY-SS-NF-04 (0-4) DUP	10/16/2007	0 - 0.33
Site	Smoke Flue	ELY-SS-NF-04 (4-10)	10/16/2007	0.33 - 0.83
Site	Smoke Flue	ELY-SS-NF-04 (4-10) DUP	10/16/2007	0.33 - 0.83
Site	Smoke Flue	ELY-SS-TZ-42 (0-2)	10/17/2007	0 - 0.17
Site	Smoke Flue	ELY-SS-TZ-42 (2-12)	10/17/2007	0.17 - 1
Site	Smoke Flue	ELY-SS-TZ-43 (0-2.5)	10/17/2007	0 - 0.21
Site	Smoke Flue	ELY-SS-TZ-43 (2.5-11)	10/17/2007	0.21 - 0.92
Site	Smoke Flue	ELY-SS-TZ-44 (0-3)	10/17/2007	0 - 0.25
Site	Smoke Flue	ELY-SS-TZ-44 (3-12)	10/17/2007	0.25 - 1
Site	Smoke Flue	ELY-SS-TZ-45 (0-2)	10/17/2007	0 - 0.17
Site	Smoke Flue	ELY-SS-TZ-45 (2-12)	10/17/2007	0.17 - 1
Site	Upper Waste Piles	ELY-SS-NF-14 (0-3)	10/18/2007	0 - 0.25
Site	Upper Waste Piles	ELY-SS-NF-14 (3-12)	10/18/2007	0.25 - 1
Site	Upper Waste Piles	ELY-SS-NF-15 (0-2)	10/18/2007	0 - 0.17
Site	Upper Waste Piles	ELY-SS-NF-15 (2-5)	10/18/2007	0.17 - 0.42
Site	Upper Waste Piles	ELY-SS-NF-16 (0-1)	10/18/2007	0 - 0.08

Table 3-2
Summary of Soil Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Sample ID	Date	Depth Range (feet)
Site	Upper Waste Piles	ELY-SS-NF-16 (1-12)	10/18/2007	0.08 - 1
Site	Upper Waste Piles	ELY-SS-NF-17 (0-4)	10/18/2007	0 - 0.33
Site	Upper Waste Piles	ELY-SS-NF-17 (4-10)	10/18/2007	0.33 - 0.83
Site	Upper Waste Piles	ELY-SS-SA-24 (0-6)	10/18/2007	0 - 0.5
Site	Upper Waste Piles	ELY-SS-SA-24 (0-6) DUP	10/18/2007	0 - 0.5
Site	Upper Waste Piles	ELY-SS-SA-25 (0-6)	10/18/2007	0 - 0.5
Site	Upper Waste Piles	ELY-SS-SA-26 (0-6)	10/18/2007	0 - 0.5
Site	Upper Waste Piles	ELY-SS-SA-27 (0-6)	10/18/2007	0 - 0.5
Site	Upper Waste Piles	ELY-SS-SA-28 (0-6)	10/18/2007	0 - 0.5
Site	Upper Waste Piles	ELY-SS-SA-29 (0-6)	10/18/2007	0 - 0.5
Site	Upper Waste Piles	ELY-SS-SA-30 (0-6)	10/18/2007	0 - 0.5
Site	Upper Waste Piles	ELY-SS-SA-31 (0-6)	10/18/2007	0 - 0.5
Site	Upper Waste Piles	ELY-SS-TR-07A (0-1)	10/18/2007	0 - 0.08
Site	Upper Waste Piles	ELY-SS-TR-07A (1-8)	10/18/2007	0.08 - 0.67
Site	Upper Waste Piles	ELY-SS-TR-07B (0-2)	10/18/2007	0 - 0.17
Site	Upper Waste Piles	ELY-SS-TR-07B (2-8)	10/18/2007	0.17 - 0.67
Site	Upper Waste Piles	ELY-SS-TR-07C (0-2)	10/18/2007	0 - 0.17
Site	Upper Waste Piles	ELY-SS-TR-07C (2-12)	10/18/2007	0.17 - 1
Site	Upper Waste Piles	ELY-SS-TR-07D (0-2)	10/18/2007	0 - 0.17
Site	Upper Waste Piles	ELY-SS-TR-07D (2-9)	10/18/2007	0.17 - 0.75
Site	Upper Waste Piles	ELY-SS-TR-08A (0.5-12)	10/18/2007	0.04 - 1
Site	Upper Waste Piles	ELY-SS-TR-08A (0.5-12)DUP	10/18/2007	0.04 - 1
Site	Upper Waste Piles	ELY-SS-TR-08A (0-0.5)	10/18/2007	0 - 0.04
Site	Upper Waste Piles	ELY-SS-TR-08A (0-0.5) DUP	10/18/2007	0 - 0.04
Site	Upper Waste Piles	ELY-SS-TR-08B (0-3)	10/18/2007	0 - 0.25
Site	Upper Waste Piles	ELY-SS-TR-08B (0-3) DUP	10/18/2007	0 - 0.25
Site	Upper Waste Piles	ELY-SS-TR-08B (3-8)	10/18/2007	0.25 - 0.67
Site	Upper Waste Piles	ELY-SS-TR-08B (3-8) DUP	10/18/2007	0.25 - 0.67
Site	Upper Waste Piles	ELY-SS-TR-08C (0-5)	10/18/2007	0 - 0.42
Site	Upper Waste Piles	ELY-SS-TR-08C (0-5) DUP	10/18/2007	0 - 0.42
Site	Upper Waste Piles	ELY-SS-TR-08C (5-12)	10/18/2007	0.42 - 1
Site	Upper Waste Piles	ELY-SS-TR-08C (5-12) DUP	10/18/2007	0.42 - 1
Site	Upper Waste Piles	ELY-SS-TR-08D (0-2)	10/18/2007	0 - 0.17
Site	Upper Waste Piles	ELY-SS-TR-08D (0-2) DUP	10/18/2007	0 - 0.17
Site	Upper Waste Piles	ELY-SS-TR-08D (2-12)	10/18/2007	0.17 - 1
Site	Upper Waste Piles	ELY-SS-TR-08D (2-12) DUP	10/18/2007	0.17 - 1
Site	Upper Waste Piles	ELY-SS-TR-09A (0-2.5)	10/18/2007	0 - 0.21
Site	Upper Waste Piles	ELY-SS-TR-09A (2.5-12)	10/18/2007	0.21 - 1
Site	Upper Waste Piles	ELY-SS-TR-09B (0-2)	10/18/2007	0 - 0.17
Site	Upper Waste Piles	ELY-SS-TR-09B (2-5)	10/18/2007	0.17 - 0.42
Site	Upper Waste Piles	ELY-SS-TR-09C (0-1.5)	10/18/2007	0 - 0.13
Site	Upper Waste Piles	ELY-SS-TR-09C (1.5-8)	10/18/2007	0.13 - 0.67
Site	Upper Waste Piles	ELY-SS-TR-09D (0-3)	10/18/2007	0 - 0.25
Site	Upper Waste Piles	ELY-SS-TR-09D (3-12)	10/18/2007	0.25 - 1
Site	Upper Waste Piles	ELY-SS-TZ-34 (0-2.5)	10/18/2007	0 - 0.21
Site	Upper Waste Piles	ELY-SS-TZ-34 (2.5-8)	10/18/2007	0.21 - 0.67
Site	Upper Waste Piles	ELY-SS-TZ-35 (0-2)	10/18/2007	0 - 0.17
Site	Upper Waste Piles	ELY-SS-TZ-35 (2-9)	10/18/2007	0.17 - 0.75
Site	Upper Waste Piles	ELY-SS-TZ-36 (0-1.5)	10/18/2007	0 - 0.13
Site	Upper Waste Piles	ELY-SS-TZ-36 (1.5-9)	10/18/2007	0.13 - 0.75

Table 3-2
Summary of Soil Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Sample ID	Date	Depth Range (feet)
Site	Upper Waste Piles	ELY-SS-TZ-37 (0-2)	10/18/2007	0 - 0.17
Site	Upper Waste Piles	ELY-SS-TZ-37 (2-12)	10/18/2007	0.17 - 1
Site	Upper Waste Piles	ELY-SS-TZ-38 (0.5-7)	10/18/2007	0.04 - 0.58
Site	Upper Waste Piles	ELY-SS-TZ-38 (0-0.5)	10/18/2007	0 - 0.04
Site	Upper Waste Piles	ELY-SS-TZ-39 (0-3)	10/18/2007	0 - 0.25
Site	Upper Waste Piles	ELY-SS-TZ-39 (3-8)	10/18/2007	0.25 - 0.67
Site	Upper Waste Piles	ELY-SS-TZ-40 (0-2)	10/18/2007	0 - 0.17
Site	Upper Waste Piles	ELY-SS-TZ-40 (2-10)	10/18/2007	0.17 - 0.83
Site	Upper Waste Piles	ELY-SS-TZ-41 (0-1.5)	10/18/2007	0 - 0.13
Site	Upper Waste Piles	ELY-SS-TZ-41 (1.5-11)	10/18/2007	0.13 - 0.92
Site	Upper Waste Piles	TL-04X-082509AX	8/25/2009	0 - 1
Site	Upper Waste Piles	TN-02X-082509AD	8/25/2009	0 - 1
Site	Upper Waste Piles	TN-02X-082509AX	8/25/2009	0 - 1
Site	Upper Waste Piles	TN-10X-082709AX	8/27/2009	0 - 1
Site	Upper Waste Piles	TO-05X-082709AX	8/27/2009	0 - 1
Site	Upper Waste Piles	TP-15X-073009AD	7/30/2009	0 - 1
Site	Upper Waste Piles	TP-15X-073009AX	7/30/2009	0 - 1
Site	Upper Waste Piles	TQ-06X-082609AX	8/26/2009	0 - 1
Site	Upper Waste Piles	TQ-16X-082609AX	8/26/2009	0 - 1
Site	Upper Waste Piles	ELY-SB-6 (0-2)	12/2/2006	0 - 2
Site	Upper Waste Piles	ELY-SB-6 (2-4)	12/2/2006	2 - 4
Site	Upper Waste Piles	ELY-SB-8 (0-6)	12/1/2006	0 - 6
Site	Upper Waste Piles	ELY-SB-8 (6-10)	12/1/2006	6 - 10
Site	Upper Waste Piles	ELY-TP10 (7-9)	5/14/2007	7 - 9
Site	Upper Waste Piles	ELY-TP11 (4-6)	5/14/2007	4 - 6
Site	Upper Waste Piles	ELY-TP8 (2-3)	5/14/2007	2 - 3
Site	Upper Waste Piles	ELY-TP9 (3-4)	5/14/2007	3 - 4
Site	Upper Waste Piles	ELY-TPQS1	5/14/2007	2 - 3
Site	Upper Waste Piles	MW-17A-073009AX	7/30/2009	0 - 2
Site	Upper Waste Piles	MW-17A-073009BX	7/30/2009	2 - 2.6
Site	Upper Waste Piles	MW-18A-080309AX	8/3/2009	2 - 4
Site	Upper Waste Piles	MW-18A-080309BD	8/3/2009	4 - 6
Site	Upper Waste Piles	MW-18A-080309BX	8/3/2009	4 - 6
Site	Upper Waste Piles	MW-18A-080309CX	8/3/2009	4 - 6
Site	Upper Waste Piles	MW-19A-072109AX	7/21/2009	2 - 6
Site	Upper Waste Piles	MW-19A-072109BX	7/21/2009	6 - 8
Site	Upper Waste Piles	SS-32X-083109AX	8/31/2009	0 - 2
Site	Upper Waste Piles	SS-33X-083109AX	8/31/2009	0 - 2
Site	Upper Waste Piles	SS-34X-082609AX	8/26/2009	0 - 2
Site	Upper Waste Piles	SS-35X-082609AX	8/26/2009	0 - 2
Site	Upper Waste Piles	SS-36X-082609AX	8/26/2009	0 - 2
Site	Upper Waste Piles	SS-37X-082609AD	8/26/2009	0 - 2
Site	Upper Waste Piles	SS-37X-082609AX	8/26/2009	0 - 2
Site	Upper Waste Piles	SS-38X-082609AX	8/26/2009	0 - 2
Site	Upper Waste Piles	SS-39X-082609AX	8/26/2009	0 - 2
Site	Upper Waste Piles	TP-15X-073009BX	7/30/2009	1 - 2
Site	Upper Waste Piles	TP-15X-073009CX	7/30/2009	2 - 3

Table 3-3
Summary of Sediment Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Sample ID	Date
Background - East Branch Ompompanoosuc River	Background - East Branch Ompompanoosuc River	06Ely09	8/22/2006
Background - East Branch Ompompanoosuc River	Background - East Branch Ompompanoosuc River	AA62441	6/21/2006
Background - Schoolhouse Brook	Background - Schoolhouse Brook	06Ely05	8/22/2006
Background - Schoolhouse Brook	Background - Schoolhouse Brook	AA62437	6/20/2006
East Branch Ompompanoosuc River	Lower East Branch Ompompanoosuc River	06Ely10	8/22/2006
East Branch Ompompanoosuc River	Lower East Branch Ompompanoosuc River	AA62442	6/21/2006
East Branch Ompompanoosuc River	Lower East Branch Ompompanoosuc River	AA62443	6/21/2006
East Branch Ompompanoosuc River	Lower East Branch Ompompanoosuc River	AA62444	6/21/2006
Ely Brook	Beaver Pond Complex	06ElyPond1S	9/19/2006
Ely Brook	Beaver Pond Complex	06ElyPond2S	9/19/2006
Ely Brook	Beaver Pond Complex	06ElyPond3S	9/19/2006
Ely Brook	Beaver Pond Complex	06ElyPond4S	9/19/2006
Ely Brook	Beaver Pond Complex	06ElyPond5S	9/19/2006
Ely Brook	Lower Ely Brook	06Ely04	8/23/2006
Ely Brook	Lower Ely Brook	06Ely11	8/23/2006
Ely Brook	Lower Ely Brook	AA62436	6/20/2006
Ely Brook	Ely Brook - Midslope Waste Pile Drainages	ELY-SED-51C (0-0.25)	10/23/2007
Ely Brook	Ely Brook - Midslope Waste Rock Pile Drainages	06Ely03	8/23/2006
Ely Brook	Ely Brook - Midslope Waste Rock Pile Drainages	06ElyPond6S	9/19/2006
Ely Brook	Ely Brook - Midslope Waste Rock Pile Drainages	AA62435	6/20/2006
Ely Brook	Ely Brook - Upper Waste Pile Drainages	ELY-SED-17C (0-0.25)	10/23/2007
Ely Brook	Ely Brook - Upper Waste Pile Drainages	ELY-SED-40C (0-0.25)	10/23/2007
Ely Brook	Ely Brook - Upper Waste Pile Drainages	ELY-SED-53C (0-0.25)	10/23/2007
Ely Brook	Ely Brook - Upper Waste Pile Drainages	ELY-SED-54C (0-0.25)	10/23/2007
Ely Brook	Ely Brook - Upper Waste Pile Drainages	ELY-SED-55C (0-0.25)	10/23/2007
Ely Brook	Ely Brook - Upper Waste Pile Drainages	ELY-SED-55C (0-0.25) DUP	10/23/2007
Ely Brook	Ely Brook - Upper Waste Pile Drainages	ELY-SED-56C (0-0.25)	10/23/2007
Ely Brook	Ely Brook - Upper Waste Pile Drainages	ELY-SED-57C (0-0.25)	10/23/2007
Ely Brook	Ely Brook - Upper Waste Rock Pile Drainages	06Ely01	8/23/2006
Ely Brook	Ely Brook - Upper Waste Rock Pile Drainages	06Ely02	8/23/2006
Ely Brook	Ely Brook - Upper Waste Rock Pile Drainages	06Ely02-2	8/23/2006
Ely Brook	Ely Brook - Upper Waste Rock Pile Drainages	AA62433	6/20/2006
Ely Brook	Ely Brook - Upper Waste Rock Pile Drainages	AA62434	6/20/2006
Ely Brook	Lower Waste Piles and Roast Bed	SD-61X-080709AD	8/7/2009
Ely Brook	Lower Waste Piles and Roast Bed	SD-61X-080709AX	8/7/2009
Ely Brook	Lower Waste Piles and Roast Bed	SD-62X-080709AX	8/7/2009
Ely Brook	Lower Waste Piles and Roast Bed	SD-63X-080709AX	8/7/2009
Ely Brook	Lower Waste Piles and Roast Bed	SD-64X-080709AX	8/7/2009
Ely Brook	Lower Waste Piles and Roast Bed	SD-65X-080609AX	8/6/2009
Ely Brook	Lower Waste Piles and Roast Bed	SD-66X-080609AX	8/6/2009
Ely Brook	Lower Waste Piles and Roast Bed	SD-72X-080609AX	8/6/2009
Ely Brook	Lower Waste Piles and Roast Bed	SD-73X-080609AX	8/6/2009
Ely Brook	Smelter/Slag Area	SD-60X-080709AX	8/7/2009
Ely Brook	Upper Waste Piles	SD-67X-080609AX	8/6/2009
Ely Brook	Upper Waste Piles	SD-68X-080609AX	8/6/2009
Ely Brook	Upper Waste Piles	SD-69X-080709AX	8/7/2009
Ely Brook	Upper Waste Piles	SD-70X-080609AX	8/6/2009
Ely Brook	Upper Waste Piles	SD-71X-080609AX	8/6/2009

Table 3-3
Summary of Sediment Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Sample ID	Date
Schoolhouse Brook	Lower Schoolhouse Brook	06Ely06	8/22/2006
Schoolhouse Brook	Lower Schoolhouse Brook	06Ely07	8/23/2006
Schoolhouse Brook	Lower Schoolhouse Brook	06Ely08	8/22/2006
Schoolhouse Brook	Lower Schoolhouse Brook	06Ely08R	8/22/2006
Schoolhouse Brook	Lower Schoolhouse Brook	AA62439	6/20/2006
Schoolhouse Brook	Lower Schoolhouse Brook	AA62440	6/20/2006
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	06Ely12	8/23/2006
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	AA62438	6/20/2006
Schoolhouse Brook	Smelter/Slag Area	SD-58X-080709AX	8/7/2009
Schoolhouse Brook	Smelter/Slag Area	SD-59X-080709AX	8/7/2009

Table 3-4
Summary of Surface Water Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Sample ID	Date
Background - East Branch Ompompanoosuc River	Background - East Branch Ompompanoosuc River	06Ely09A RA	8/21/2006
Background - Schoolhouse Brook	Background - Schoolhouse Brook	06Ely05A RA	8/22/2006
Background - Schoolhouse Brook	Background - Schoolhouse Brook	AA62008	6/19/2006
Background - Schoolhouse Brook	Background - Schoolhouse Brook	AA62298	6/23/2006
Background - Schoolhouse Brook	Background - Schoolhouse Brook	ELY-SW-38	5/3/2007
East Branch Ompompanoosuc River	Lower East Branch Ompompanoosuc River	06Ely10A RA	8/21/2006
East Branch Ompompanoosuc River	Lower East Branch Ompompanoosuc River	06Ely20A RA	8/21/2006
East Branch Ompompanoosuc River	Lower East Branch Ompompanoosuc River	MA1PZ9	4/9/2007
East Branch Ompompanoosuc River	Lower East Branch Ompompanoosuc River	MA1Q01	4/9/2007
East Branch Ompompanoosuc River	Lower East Branch Ompompanoosuc River	MA1Q03	4/9/2007
Ely Brook	Beaver Pond Complex	06ElyPond1 FA	9/19/2006
Ely Brook	Beaver Pond Complex	06ElyPond1 RA	9/19/2006
Ely Brook	Beaver Pond Complex	06ElyPond2 FA	9/19/2006
Ely Brook	Beaver Pond Complex	06ElyPond2 RA	9/19/2006
Ely Brook	Beaver Pond Complex	06ElyPond3 FA	9/19/2006
Ely Brook	Beaver Pond Complex	06ElyPond3 RA	9/19/2006
Ely Brook	Beaver Pond Complex	06ElyPond4 FA	9/19/2006
Ely Brook	Beaver Pond Complex	06ElyPond4 RA	9/19/2006
Ely Brook	Beaver Pond Complex	06ElyPond5 FA	9/19/2006
Ely Brook	Beaver Pond Complex	06ElyPond5 RA	9/19/2006
Ely Brook	Beaver Pond Complex	AA62005	6/19/2006
Ely Brook	Beaver Pond Complex	AA62296	6/23/2006
Ely Brook	Ely Brook - Lower Ely Brook	06Ely04A RA	8/23/2006
Ely Brook	Ely Brook - Lower Ely Brook	AA62007	6/19/2006
Ely Brook	Ely Brook - Lower Ely Brook	AA62305	6/23/2006
Ely Brook	Ely Brook - Lower Ely Brook	ELY-SW-09	5/2/2007
Ely Brook	Ely Brook - Lower Ely Brook	ELY-SW-09 DUP	5/2/2007
Ely Brook	Ely Brook - Lower Ely Brook	ELY-SW-39	5/2/2007
Ely Brook	Ely Brook - Lower Ely Brook	MA1PY9	4/11/2007
Ely Brook	Ely Brook - Midslope Waste Pile Drainages	ELY-SW-12	5/2/2007
Ely Brook	Ely Brook - Midslope Waste Pile Drainages	ELY-SW-13	5/2/2007
Ely Brook	Ely Brook - Midslope Waste Pile Drainages	ELY-SW-14	5/2/2007
Ely Brook	Ely Brook - Midslope Waste Pile Drainages	ELY-SW-30	5/2/2007
Ely Brook	Ely Brook - Midslope Waste Pile Drainages	ELY-SW-32	5/2/2007
Ely Brook	Ely Brook - Midslope Waste Pile Drainages	ELY-SW-33	5/2/2007
Ely Brook	Ely Brook - Midslope Waste Pile Drainages	ELY-SW-34	5/2/2007
Ely Brook	Ely Brook - Midslope Waste Pile Drainages	ELY-SW-41	5/3/2007
Ely Brook	Ely Brook - Midslope Waste Pile Drainages	ELY-SW-51	5/2/2007
Ely Brook	Ely Brook - Midslope Waste Rock Pile Drainages	06Ely03A FA	8/23/2006
Ely Brook	Ely Brook - Midslope Waste Rock Pile Drainages	06Ely03A RA	8/23/2006
Ely Brook	Ely Brook - Midslope Waste Rock Pile Drainages	06ElyPond6 FA	9/19/2006
Ely Brook	Ely Brook - Midslope Waste Rock Pile Drainages	06ElyPond6 RA	9/19/2006
Ely Brook	Ely Brook - Midslope Waste Rock Pile Drainages	AA62006	6/19/2006
Ely Brook	Ely Brook - Midslope Waste Rock Pile Drainages	AA62297	6/23/2006
Ely Brook	Ely Brook - Midslope Waste Rock Pile Drainages	AA62304	6/23/2006
Ely Brook	Ely Brook - Upper Waste Pile Drainages	ELY-SW-17	5/2/2007
Ely Brook	Ely Brook - Upper Waste Pile Drainages	ELY-SW-29	5/2/2007
Ely Brook	Ely Brook - Upper Waste Pile Drainages	ELY-SW-40	5/2/2007
Ely Brook	Ely Brook - Upper Waste Rock Pile Drainages	06Ely01A RA	8/23/2006
Ely Brook	Ely Brook - Upper Waste Rock Pile Drainages	06Ely02A RA	8/23/2006

Table 3-4
Summary of Surface Water Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Sample ID	Date
Ely Brook	Lower Waste Piles and Roast Bed	SW-12-111809AX	11/18/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-12X-080609AX	8/6/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-13-111809AX	11/18/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-13X-080609AX	8/6/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-32-111809AX	11/18/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-32X-080609AX	8/6/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-34-111809AX	11/18/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-34X-080609AX	8/6/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-35X-080609AX	8/6/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-36-111809AX	11/18/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-41-111709AX	11/17/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-41X-080409AX	8/4/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-51-111809AX	11/18/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-51X-080409AX	8/4/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-72-111809AX	11/18/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-72X-080609AX	8/6/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-73-111809AX	11/18/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-73X-080409AX	8/4/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-82-111709AX	11/17/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-82X-080409AX	8/4/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-83-111809AD	11/18/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-83-111809AX	11/18/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-83X-080609AX	8/6/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-84-111809AX	11/18/2009
Ely Brook	Lower Waste Piles and Roast Bed	SW-84X-080609AX	8/6/2009
Ely Brook	Smelter/Slag Area	SW-09-111909AX	11/19/2009
Ely Brook	Smelter/Slag Area	SW-09X-080709AX	8/7/2009
Ely Brook	Upper Waste Piles	SW-17-111709AX	11/17/2009
Ely Brook	Upper Waste Piles	SW-17X-080409AX	8/4/2009
Ely Brook	Upper Waste Piles	SW-29-111709AX	11/17/2009
Ely Brook	Upper Waste Piles	SW-29X-080409AX	8/4/2009
Ely Brook	Upper Waste Piles	SW-40-111709AX	11/17/2009
Ely Brook	Upper Waste Piles	SW-40X-080409AX	8/4/2009
Ely Brook	Upper Waste Piles	SW-42-111709AX	11/17/2009
Ely Brook	Upper Waste Piles	SW-42X-080409AX	8/4/2009
Ely Brook	Upper Waste Piles	SW-43-120209AX	12/2/2009
Ely Brook	Upper Waste Piles	SW-43X-080509AX	8/5/2009
Ely Brook	Upper Waste Piles	SW-45-111709AD	11/17/2009
Ely Brook	Upper Waste Piles	SW-45-111709AX	11/17/2009
Ely Brook	Upper Waste Piles	SW-45X-080509AX	8/5/2009
Ely Brook	Upper Waste Piles	SW-46-111609AX	11/17/2009
Ely Brook	Upper Waste Piles	SW-46X-080509AX	8/5/2009
Ely Brook	Upper Waste Piles	SW-47-111609-AX	11/16/2009
Ely Brook	Upper Waste Piles	SW-47X-080509AX	8/5/2009
Ely Brook	Upper Waste Piles	SW-48-111609AX	11/16/2009
Ely Brook	Upper Waste Piles	SW-48X-080509AX	8/5/2009
Ely Brook	Upper Waste Piles	SW-49-111609AX	11/16/2009
Ely Brook	Upper Waste Piles	SW-49X-080509AX	8/5/2009
Ely Brook	Upper Waste Piles	SW-71-111709AX	11/17/2009
Ely Brook	Upper Waste Piles	SW-71X-080409AX	8/4/2009

Table 3-4
Summary of Surface Water Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Sample ID	Date
Ely Brook	Upper Waste Piles	SW-75-111609AX	11/16/2009
Ely Brook	Upper Waste Piles	SW-75X-080509AD	8/5/2009
Ely Brook	Upper Waste Piles	SW-75X-080509AX	8/5/2009
Ely Brook	Upper Waste Piles	SW-76X-080509AX	8/5/2009
Ely Brook	Upper Waste Piles	SW-77-111609AX	11/16/2009
Ely Brook	Upper Waste Piles	SW-77X-080509AX	8/5/2009
Ely Brook	Upper Waste Piles	SW-78-111609AX	11/16/2009
Ely Brook	Upper Waste Piles	SW-78X-080509AD	8/5/2009
Ely Brook	Upper Waste Piles	SW-78X-080509AX	8/5/2009
Ely Brook	Upper Waste Piles	SW-79-111609AX	11/16/2009
Ely Brook	Upper Waste Piles	SW-79X-080609AX	8/6/2009
Ely Brook	Upper Waste Piles	SW-80-111609AX	11/16/2009
Ely Brook	Upper Waste Piles	SW-80X-080509AX	8/5/2009
Ely Brook	Upper Waste Piles	SW-81-111709AX	11/17/2009
Ely Brook	Upper Waste Piles	SW-81X-080509AX	8/5/2009
Schoolhouse Brook	Lower Schoolhouse Brook	06Ely06A RA	8/22/2006
Schoolhouse Brook	Lower Schoolhouse Brook	06Ely07A RA	8/23/2006
Schoolhouse Brook	Lower Schoolhouse Brook	06Ely08A RA	8/21/2006
Schoolhouse Brook	Lower Schoolhouse Brook	AA62011	6/19/2006
Schoolhouse Brook	Lower Schoolhouse Brook	AA62012	6/19/2006
Schoolhouse Brook	Lower Schoolhouse Brook	AA62302	6/23/2006
Schoolhouse Brook	Lower Schoolhouse Brook	AA62303	6/23/2006
Schoolhouse Brook	Lower Schoolhouse Brook	MA1PZ7	4/11/2007
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	06Ely19A RA	8/22/2006
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	06Ely19A-R RA	8/22/2006
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	AA62009	6/19/2006
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	AA62010	6/19/2006
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	AA62013	6/19/2006
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	AA62299	6/23/2006
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	AA62300	6/23/2006
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	AA62301	6/23/2006
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	ELY-SW-04	5/2/2007
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	ELY-SW-04 DUP	5/2/2007
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	ELY-SW-06	5/2/2007
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	MA1PZ1	4/11/2007
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	MA1PZ2	4/11/2007
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	MA1PZ5	4/11/2007
Schoolhouse Brook	Schoolhouse Brook - Smelter Area	Schoolhouse Brook, Site 2A	8/22/2006
Schoolhouse Brook	Smelter/Slag Area	SW-04-111909AX	11/18/2009
Schoolhouse Brook	Smelter/Slag Area	SW-04X-080709AX	8/7/2009
Schoolhouse Brook	Smelter/Slag Area	SW-38-111909AX	11/19/2009
Schoolhouse Brook	Smelter/Slag Area	SW-38X-080709AX	8/7/2009

Table 3-5
Summary of Groundwater Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Screened Zone	Location	Sample ID	Date
Background - Overburden	Background	Shallow Overburden/Mine Waste	MW-07A	ELY-MW-07A	12/11/2006
Background - Overburden	Background	Shallow Overburden/Mine Waste	MW-07A	ELY-MW-07A	6/13/2007
Background - Overburden	Background	Shallow Overburden	MW-07A	MW-07A-081209AX	8/12/2009
Background - Overburden	Background	Shallow Overburden	MW-07A	MW-07A-112509AX	11/25/2009
Background - Shallow Bedrock	Background	Shallow Bedrock	MW-07C	ELY-MW-07C	12/11/2006
Background - Shallow Bedrock	Background	Shallow Bedrock	MW-07C	ELY-MW-07C	6/13/2007
Background - Shallow Bedrock	Background	Shallow Bedrock	MW-07C	MW-07C-081209AX	8/12/2009
Background - Shallow Bedrock	Background	Shallow Bedrock	MW-07C	MW-07C-112509AX	11/25/2009
Off-Site Residential	Off-Site	Shallow Overburden	M11LT064	M11LT064-120709AX	12/7/2009
Off-Site Residential	Off-Site	Shallow Overburden	M11LT065	M11LT065-041910AX	12/7/2009
Off-Site Residential	Off-Site	Shallow Overburden	M11LT19.0	M11LT19.0-120809AX	12/8/2009
Off-Site Residential	Off-Site	Shallow Overburden	M11LT55.1	M11LT55.1-120709AX	12/7/2009
Off-Site Residential	Off-Site	Shallow Overburden	M11LT60.0	M11LT60.0-120709AX	12/7/2009
Off-Site Residential	Off-Site	Shallow Overburden	M11LT63.0	M11LT63.0-120709AX	12/7/2009
Site - Deep Bedrock	Lower Waste Piles and Roast Bed	Deep Bedrock	MW-20D	MW-20D-091609AX	9/16/2009
Site - Deep Bedrock	Lower Waste Piles and Roast Bed	Deep Bedrock	MW-20D	MW-20D-091709AX	9/17/2009
Site - Deep Bedrock	Lower Waste Piles and Roast Bed	Deep Bedrock	MW-20D	MW-20D-120209AX	12/2/2009
Site - Deep Bedrock	Smelter/Slag Area	Deep Bedrock	MW-14D	MW-14D-091709AX	9/17/2009
Site - Deep Bedrock	Smelter/Slag Area	Deep Bedrock	MW-14D	MW-14D-091809AX	9/18/2009
Site - Deep Bedrock	Smelter/Slag Area	Deep Bedrock	MW-14D	MW-14D-091809BX	9/18/2009
Site - Deep Bedrock	Smelter/Slag Area	Deep Bedrock	MW-14D	MW-14D-120109AX	12/1/2009
Site - Deep Bedrock	Upper Waste Piles	Deep Bedrock	MW-19D	MW-19D-091509AD	9/15/2009
Site - Deep Bedrock	Upper Waste Piles	Deep Bedrock	MW-19D	MW-19D-091509AX	9/15/2009
Site - Deep Bedrock	Upper Waste Piles	Deep Bedrock	MW-19D	MW-19D-091509BX	9/15/2009
Site - Deep Bedrock	Upper Waste Piles	Deep Bedrock	MW-19D	MW-19D-120209AX	12/2/2009
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-04A	MW-04A-081209AX	8/13/2009
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-04A	MW-04A-112409AX	11/24/2009
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-15A	MW-15A-081309AX	8/13/2009
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-15A	MW-15A-112409AX	11/24/2009
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-16A	MW-16A-081309AX	8/13/2009
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-16A	MW-16A-112409AX	11/24/2009
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-20A	MW-20A-081409AX	8/14/2009
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-20A	MW-20A-120109AX	12/1/2009
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-21A	MW-21A-081309AD	8/13/2009
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-21A	MW-21A-081309AX	8/13/2009
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-21A	MW-21A-113009AD	11/30/2009

Table 3-5
Summary of Groundwater Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Screened Zone	Location	Sample ID	Date
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-21A	MW-21A-113009AX	11/30/2009
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-22A	MW-22A-081309AX	8/13/2009
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-22A	MW-22A-113009AX	11/30/2009
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-23A	MW-23A-081309AX	8/13/2009
Site - Overburden	Lower Waste Piles and Roast Bed	Shallow Overburden	MW-23A	MW-23A-112309AX	11/23/2009
Site - Overburden	Midslope Waste Piles	Shallow Overburden/Mine Waste	MW-05A	ELY-GW-MW-05A	11/7/2007
Site - Overburden	Midslope Waste Piles	Shallow Overburden/Mine Waste	MW-13A	ELY-GW-MW-13A	11/7/2007
Site - Overburden	Midslope Waste Piles	Shallow Overburden/Mine Waste	MW-14A	ELY-GW-MW-14A	11/7/2007
Site - Overburden	Midslope Waste Piles	Shallow Overburden/Mine Waste	MW-15A	ELY-GW-MW-15A	11/5/2007
Site - Overburden	Midslope Waste Piles	Shallow Overburden/Mine Waste	MW-16A	ELY-GW-MW-16A	11/5/2007
Site - Overburden	Midslope Waste Piles	Shallow Overburden/Mine Waste	MW-04A	ELY-MW-04A	12/12/2006
Site - Overburden	Midslope Waste Piles	Shallow Overburden/Mine Waste	MW-04A	ELY-MW-04A	6/13/2007
Site - Overburden	Midslope Waste Piles	Shallow Overburden/Mine Waste	MW-05A	ELY-MW-05A	12/12/2006
Site - Overburden	Midslope Waste Piles	Shallow Overburden/Mine Waste	MW-05A	ELY-MW-05A	6/13/2007
Site - Overburden	Midslope Waste Piles	Till/Deep Overburden	MW-05B	ELY-MW-05B	12/12/2006
Site - Overburden	Midslope Waste Piles	Till/Deep Overburden	MW-05B	ELY-MW-05B	6/13/2007
Site - Overburden	Midslope Waste Piles	Shallow Overburden/Mine Waste	MW-08A	ELY-MW-08A	12/12/2006
Site - Overburden	Midslope Waste Piles	Shallow Overburden/Mine Waste	MW-08A	ELY-MW-08A	6/12/2007
Site - Overburden	Smelter Area	Till/Deep Overburden	MW-02B	ELY-GW-MW-02B	11/6/2007
Site - Overburden	Smelter Area	Shallow Overburden/Mine Waste	MW-11A	ELY-GW-MW-11A	11/7/2007
Site - Overburden	Smelter Area	Shallow Overburden/Mine Waste	MW-01A	ELY-MW-01A	12/13/2006
Site - Overburden	Smelter Area	Shallow Overburden/Mine Waste	MW-01A	ELY-MW-01A	6/12/2007
Site - Overburden	Smelter Area	Till/Deep Overburden	MW-01B	ELY-MW-01B	12/13/2006
Site - Overburden	Smelter Area	Till/Deep Overburden	MW-01B	ELY-MW-01B	6/12/2007
Site - Overburden	Smelter Area	Shallow Overburden/Mine Waste	MW-02A	ELY-MW-02A	12/12/2006
Site - Overburden	Smelter Area	Shallow Overburden/Mine Waste	MW-02A	ELY-MW-02A	6/12/2007
Site - Overburden	Smelter Area	Shallow Overburden/Mine Waste	MW-02A	ELY-MW-02A DUP	6/12/2007
Site - Overburden	Smelter Area	Till/Deep Overburden	MW-02B	ELY-MW-02B	12/13/2006
Site - Overburden	Smelter Area	Till/Deep Overburden	MW-02B	ELY-MW-02B	6/12/2007
Site - Overburden	Smelter Area	Shallow Overburden/Mine Waste	MW-02A	ELY-QS-01	12/12/2006
Site - Overburden	Smelter/Slag Area	Shallow Overburden	MW-01A	MW-01A-081009AX	8/10/2009
Site - Overburden	Smelter/Slag Area	Shallow Overburden	MW-01A	MW-01A-112009AX	11/20/2009
Site - Overburden	Smelter/Slag Area	Deep Overburden	MW-01B	MW-01B-081009AX	8/10/2009
Site - Overburden	Smelter/Slag Area	Deep Overburden	MW-01B	MW-01B-112009AX	11/20/2009

Table 3-5
Summary of Groundwater Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Screened Zone	Location	Sample ID	Date
Site - Overburden	Smelter/Slag Area	Shallow Overburden	MW-02A	MW-02A-081009AX	8/10/2009
Site - Overburden	Smelter/Slag Area	Shallow Overburden	MW-02A	MW-02A-113009AX	11/30/2009
Site - Overburden	Smelter/Slag Area	Deep Overburden	MW-02B	MW-02B-081009AX	8/10/2009
Site - Overburden	Smelter/Slag Area	Deep Overburden	MW-02B	MW-02B-112009AX	11/20/2009
Site - Overburden	Smelter/Slag Area	Deep Overburden	MW-10B	MW-10B-081109AX	8/11/2009
Site - Overburden	Smelter/Slag Area	Deep Overburden	MW-10B	MW-10B-112309AX	11/23/2009
Site - Overburden	Smelter/Slag Area	Shallow Overburden	MW-11A	MW-11A-081109AX	8/11/2009
Site - Overburden	Smelter/Slag Area	Shallow Overburden	MW-11A	MW-11A-120109AX	12/1/2009
Site - Overburden	Smelter/Slag Area	Shallow Overburden	MW-14A	MW-14A-081109AX	8/11/2009
Site - Overburden	Smelter/Slag Area	Shallow Overburden	MW-14A	MW-14A-112309AX	11/23/2009
Site - Overburden	Smoke Flue	Shallow Overburden/Mine Waste	MW-10B	ELY-GW-MW-10B	11/6/2007
Site - Overburden	Upper Waste Piles	Shallow Overburden	BM-01	BM-01-120109AX	12/1/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	BM-01	BM-01X-081809AX	8/18/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden/Mine Waste	MW-09A	ELY-GW-MW-09A	11/6/2007
Site - Overburden	Upper Waste Piles	Shallow Overburden/Mine Waste	MW-06A	ELY-MW-06A	12/12/2006
Site - Overburden	Upper Waste Piles	Shallow Overburden/Mine Waste	MW-06A	ELY-MW-06A	6/12/2007
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-05A	MW-05A-081109AX	8/11/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-05A	MW-05A-120109AD	12/1/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-05A	MW-05A-120109AX	12/1/2009
Site - Overburden	Upper Waste Piles	Deep Overburden	MW-05B	MW-05B-081209AD	8/12/2009
Site - Overburden	Upper Waste Piles	Deep Overburden	MW-05B	MW-05B-081209AX	8/12/2009
Site - Overburden	Upper Waste Piles	Deep Overburden	MW-05B	MW-05B-112409AX	11/24/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-06A	MW-06A-081209AX	8/12/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-06A	MW-06A-112509AX	11/24/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-08A	MW-08A-081109AX	8/11/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-08A	MW-08A-112409AD	11/24/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-08A	MW-08A-112409AX	11/24/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-09A	MW-09A-081209AX	8/12/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-09A	MW-09A-120209AX	12/2/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-13A	MW-13A-081209AX	8/12/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-13A	MW-13A-112409AX	11/24/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-17A	MW-17A-081409AX	8/14/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-17A	MW-17A-112409AX	11/24/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-18A	MW-18A-081409AX	8/14/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-18A	MW-18A-120109AX	12/1/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-19A	MW-19A-081409AX	8/14/2009
Site - Overburden	Upper Waste Piles	Shallow Overburden	MW-19A	MW-19A-113009AX	11/30/2009
Site - Shallow Bedrock	Lower Waste Piles and Roast Bed	Shallow Bedrock	MW-03C	MW-03C-081109AX	8/11/2009
Site - Shallow Bedrock	Lower Waste Piles and Roast Bed	Shallow Bedrock	MW-03C	MW-03C-112309AX	11/23/2009
Site - Shallow Bedrock	Lower Waste Piles and Roast Bed	Shallow Bedrock	MW-04C	MW-04C-081209AX	8/12/2009
Site - Shallow Bedrock	Lower Waste Piles and Roast Bed	Shallow Bedrock	MW-04C	MW-04C-112409AX	11/24/2009
Site - Shallow Bedrock	Lower Waste Piles and Roast Bed	Shallow Bedrock	MW-20C	MW-20C-081309AX	8/13/2009
Site - Shallow Bedrock	Lower Waste Piles and Roast Bed	Shallow Bedrock	MW-20C	MW-20C-112309AX	11/23/2009

Table 3-5
Summary of Groundwater Samples Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Sample Location	Screened Zone	Location	Sample ID	Date
Site - Shallow Bedrock	Lower Waste Piles and Roast Bed	Shallow Bedrock	MW-21C	MW-21C-081409AX	8/14/2009
Site - Shallow Bedrock	Lower Waste Piles and Roast Bed	Shallow Bedrock	MW-21C	MW-21C-112409AX	11/24/2009
Site - Shallow Bedrock	Midslope Waste Piles	Shallow Bedrock	MW-12C	ELY-GW-MW-12C	11/7/2007
Site - Shallow Bedrock	Midslope Waste Piles	Shallow Bedrock	MW-03C	ELY-MW-03C	12/11/2006
Site - Shallow Bedrock	Midslope Waste Piles	Shallow Bedrock	MW-03C	ELY-MW-03C	6/12/2007
Site - Shallow Bedrock	Midslope Waste Piles	Shallow Bedrock	MW-04C	ELY-MW-04C	12/12/2006
Site - Shallow Bedrock	Midslope Waste Piles	Shallow Bedrock	MW-04C	ELY-MW-04C	6/13/2007
Site - Shallow Bedrock	Midslope Waste Piles	Shallow Bedrock	MW-05C	ELY-MW-05C	12/12/2006
Site - Shallow Bedrock	Midslope Waste Piles	Shallow Bedrock	MW-05C	ELY-MW-05C	6/13/2007
Site - Shallow Bedrock	Smelter Area	Shallow Bedrock	MW-02C	ELY-GW-MW-02C	11/6/2007
Site - Shallow Bedrock	Smelter Area	Shallow Bedrock	MW-02C	ELY-GW-MW-02C DUP	11/6/2007
Site - Shallow Bedrock	Smelter Area	Shallow Bedrock	MW-11C	ELY-GW-MW-11C	11/7/2007
Site - Shallow Bedrock	Smelter Area	Shallow Bedrock	MW-01C	ELY-MW-01C	12/13/2006
Site - Shallow Bedrock	Smelter Area	Shallow Bedrock	MW-01C	ELY-MW-01C	6/12/2007
Site - Shallow Bedrock	Smelter Area	Shallow Bedrock	MW-02C	ELY-MW-02C	12/13/2006
Site - Shallow Bedrock	Smelter Area	Shallow Bedrock	MW-02C	ELY-MW-02C	6/13/2007
Site - Shallow Bedrock	Smelter/Slag Area	Shallow Bedrock	MW-01C	MW-01C-081209AX	8/12/2009
Site - Shallow Bedrock	Smelter/Slag Area	Shallow Bedrock	MW-01C	MW-01C-112309AX	11/23/2009
Site - Shallow Bedrock	Smelter/Slag Area	Shallow Bedrock	MW-02C	MW-02C-081209AX	8/12/2009
Site - Shallow Bedrock	Smelter/Slag Area	Shallow Bedrock	MW-02C	MW-02C-112009AX	11/20/2009
Site - Shallow Bedrock	Smelter/Slag Area	Shallow Bedrock	MW-10C	MW-10C-081109AX	8/11/2009
Site - Shallow Bedrock	Smelter/Slag Area	Shallow Bedrock	MW-10C	MW-10C-112309AX	11/23/2009
Site - Shallow Bedrock	Smelter/Slag Area	Shallow Bedrock	MW-11C	MW-11C-081109AX	8/11/2009
Site - Shallow Bedrock	Smelter/Slag Area	Shallow Bedrock	MW-11C	MW-11C-112309AX	11/23/2009
Site - Shallow Bedrock	Smelter/Slag Area	Shallow Bedrock	MW-14C	MW-14C-081309AX	8/13/2009
Site - Shallow Bedrock	Smelter/Slag Area	Shallow Bedrock	MW-14C	MW-14C-112309AX	11/23/2009
Site - Shallow Bedrock	Smoke Flue	Shallow Bedrock	MW-10C	ELY-GW-MW-10C	11/6/2007
Site - Shallow Bedrock	Upper Waste Piles	Bedrock	BOM-01	ELY-BOM-1	12/11/2006
Site - Shallow Bedrock	Upper Waste Piles	Bedrock	BOM-01	ELY-BOM-1	6/14/2007
Site - Shallow Bedrock	Upper Waste Piles	Bedrock	BOM-03	ELY-GW-BOM3	11/12/2007
Site - Shallow Bedrock	Upper Waste Piles	Shallow Bedrock	MW-09C	ELY-GW-MW-09C	11/6/2007
Site - Shallow Bedrock	Upper Waste Piles	Shallow Bedrock	MW-06C	ELY-MW-06C	12/11/2006
Site - Shallow Bedrock	Upper Waste Piles	Shallow Bedrock	MW-06C	ELY-MW-06C	6/13/2007
Site - Shallow Bedrock	Upper Waste Piles	Shallow Bedrock	MW-05C	MW-05C-081309AX	8/13/2009
Site - Shallow Bedrock	Upper Waste Piles	Shallow Bedrock	MW-05C	MW-05C-112509AX	11/25/2009
Site - Shallow Bedrock	Upper Waste Piles	Shallow Bedrock	MW-06C	MW-06C-081209AX	8/12/2009
Site - Shallow Bedrock	Upper Waste Piles	Shallow Bedrock	MW-06C	MW-06C-112509AX	11/25/2009
Site - Shallow Bedrock	Upper Waste Piles	Shallow Bedrock	MW-09C	MW-09C-081309AX	8/13/2009
Site - Shallow Bedrock	Upper Waste Piles	Shallow Bedrock	MW-09C	MW-09C-112409AX	11/24/2009
Site - Shallow Bedrock	Upper Waste Piles	Shallow Bedrock	MW-12C	MW-12C-081109AX	8/11/2009
Site - Shallow Bedrock	Upper Waste Piles	Shallow Bedrock	MW-12C	MW-12C-112409AX	11/24/2009
Site - Shallow Bedrock	Upper Waste Piles	Shallow Bedrock	MW-19C	MW-19C-081409AX	8/14/2009
Site - Shallow Bedrock	Upper Waste Piles	Shallow Bedrock	MW-19C	MW-19C-112509AX	11/25/2009

Table 3-6
Summary of Fish Samples (Whole Body) Used in the HHRA
Ely Copper Mine Superfund Site
Vershire, Vermont

Data Group	Species	Sample Location	Sample ID	Date
Background - East Branch Ompompanoosuc River	Blacknose Dace	Background - East Branch Ompompanoosuc River	06Ely09F1	9/12/06
Background - East Branch Ompompanoosuc River	Blacknose Dace	Background - East Branch Ompompanoosuc River	06Ely09F2	9/12/06
Background - East Branch Ompompanoosuc River	Blacknose Dace	Background - East Branch Ompompanoosuc River	06Ely09F3	9/12/06
Background - Schoolhouse Brook	Blacknose Dace	Background - Schoolhouse Brook	06Ely05F6	9/13/06
Background - Schoolhouse Brook	Blacknose Dace	Background - Schoolhouse Brook	06Ely05F7	9/13/06
Background - Schoolhouse Brook	Brook Trout	Background - Schoolhouse Brook	06Ely05F1	9/13/06
Background - Schoolhouse Brook	Brook Trout	Background - Schoolhouse Brook	06Ely05F2	9/13/06
Background - Schoolhouse Brook	Brook Trout	Background - Schoolhouse Brook	06Ely05F3	9/13/06
Background - Schoolhouse Brook	Brook Trout	Background - Schoolhouse Brook	06Ely05F4	9/13/06
Background - Schoolhouse Brook	Brook Trout	Background - Schoolhouse Brook	06Ely05F5	9/13/06
Site - East Branch Ompompanoosuc River	Blacknose Dace	Lower East Branch Ompompanoosuc River	06Ely10F3	9/12/06
Site - East Branch Ompompanoosuc River	Blacknose Dace	Lower East Branch Ompompanoosuc River	06Ely10F4	9/12/06
Site - East Branch Ompompanoosuc River	Blacknose Dace	Lower East Branch Ompompanoosuc River	06Ely10F5	9/12/06
Site - East Branch Ompompanoosuc River	Blacknose Dace	Lower East Branch Ompompanoosuc River	06Ely20F1	9/12/06
Site - East Branch Ompompanoosuc River	Blacknose Dace	Lower East Branch Ompompanoosuc River	06Ely20F2	9/12/06
Site - East Branch Ompompanoosuc River	Blacknose Dace	Lower East Branch Ompompanoosuc River	06Ely20F3	9/12/06
Site - East Branch Ompompanoosuc River	Brook Trout	Lower East Branch Ompompanoosuc River	06Ely10F1	9/12/06
Site - East Branch Ompompanoosuc River	Brook Trout	Lower East Branch Ompompanoosuc River	06Ely10F2	9/12/06
Site - Schoolhouse Brook	Blacknose Dace	Lower Schoolhouse Brook	06Ely07F1	9/13/06
Site - Schoolhouse Brook	Blacknose Dace	Lower Schoolhouse Brook	06Ely07F2	9/13/06
Site - Schoolhouse Brook	Blacknose Dace	Lower Schoolhouse Brook	06Ely07F3	9/13/06
Site - Schoolhouse Brook	Blacknose Dace	Lower Schoolhouse Brook	06Ely08F1	9/12/06
Site - Schoolhouse Brook	Blacknose Dace	Lower Schoolhouse Brook	06Ely08F2	9/12/06
Site - Schoolhouse Brook	Blacknose Dace	Lower Schoolhouse Brook	06Ely08F3	9/12/06
Site - Schoolhouse Brook	Blacknose Dace	Lower Schoolhouse Brook	06Ely06F1	9/13/06
Site - Schoolhouse Brook	Blacknose Dace	Schoolhouse Brook - Smelter Area	06Ely19F2	9/13/06
Site - Schoolhouse Brook	Brook Trout	Schoolhouse Brook - Smelter Area	06Ely19F1	9/13/06

Table 4-1
Surface Soil - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface soil 0-1 ft bgs

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Surface Soil	7429905	Aluminum	860	100000	mg/kg	ELY-SS-NF-03 (4-12)	270/270	NA	100000	12000	7700 NC	YES	ASL
	7440360	Antimony	0.048	8.1	mg/Kg	ELY-SS-TR-08A (0.5-12)DUP	106/269	0.46 - 43	8.1	NA	3.1 NC	YES	ASL
	7440382	Arsenic	0.36	23	mg/Kg	ELY-SS-TZ-05 (0-1)	193/270	0.41 - 8.6	23	4.3	0.39 C	YES	ASL
	7440393	Barium	8.3	220	mg/kg	SS-13X-082809AX	270/270	NA	220	350	1500 NC	NO	BSL
	7440417	Beryllium	0.04	5.4	mg/Kg	ELY-SS-NF-03 (4-12)	188/270	0.035 - 4.3	5.4	1	16 NC	NO	BSL
	7440439	Cadmium	0.017	8.5	mg/Kg	ELY-SS-TR-04A (1-12)	210/269	0.2 - 10	8.5	0.52	7.0 NC	YES	ASL
	7440702	Calcium	34	42000	mg/kg	ELY-SS-TR-02A (1.5-11)	269/270	44 - 44	42000	4600	NA	NO	Essential Nutrient
	7440473	Chromium	2.5	440	mg/kg	ELY-SS-NF-03 (4-12)	270/270	NA	440	32	0.29 C	YES	ASL
	7440484	Cobalt	1.8	200	mg/kg	ELY-SS-TR-04A (1-12)	270/270	NA	200	16	2.3 NC	YES	ASL
	7440508	Copper	7.5	23000	mg/kg	ELY-SS-TR-04A (1-12)	270/270	NA	23000	45	310 NC	YES	ASL
	7439896	Iron	2400	300000	mg/kg	ELY-SS-TZ-14 (4-12)	270/270	NA	300000	31000	5500 NC	YES	ASL
	7439921	Lead	0.72	1700	mg/kg	ELY-SS-TZ-12 (0-3)	270/270	NA	1700	77	400	YES	ASL
	7439954	Magnesium	9.7	24000	mg/kg	ELY-SS-NF-03 (4-12)	270/270	NA	24000	4700	NA	NO	Essential Nutrient
	7439965	Manganese	17	8380	mg/kg	SS-13X-082809AX	270/270	NA	8380	7800	180 NC	YES	ASL
	7439976	Mercury	0.006	2.8	mg/kg	ELY-SS-TZ-39 (3-8)	252/269	0.014 - 0.1	2.8	0.29	2.3 NC	YES	ASL
	7439987	Molybdenum	0.2	69	mg/Kg	ELY-SS-SA-31 (0-6)	216/269	0.22 - 43	69	0.64	39 NC	YES	ASL
	7440020	Nickel	1.1	110	mg/kg	ELY-SS-NF-03 (4-12)	268/270	3 - 13	110	30	150 NC	NO	BSL
	7440097	Potassium	398	19000	mg/kg	ELY-SS-TZ-14 (4-12)	270/270	NA	19000	3000	NA	NO	Essential Nutrient
	7782492	Selenium	0.42	170	mg/kg	ELY-SS-SA-31 (0-6)	253/269	0.82 - 3.6	170	2.9	39 NC	YES	ASL
	7440224	Silver	0.079	23	mg/kg	ELY-SS-SA-16 (0-6)	221/270	0.11 - 2.6	23	1.4	39 NC	NO	BSL
	7440235	Sodium	23	3800	mg/Kg	ELY-SS-TR-02D (2-12) DUP	120/268	160 - 2000	3800	110	NA	NO	Essential Nutrient
	7440246	Strontium	1.2	107	mg/kg	SS-27X-082609AX	48/48	NA	107	NA	4700 NC	NO	BSL
	7440280	Thallium	0.02	16	mg/Kg	ELY-TP-109 (0-1)	138/269	0.25 - 14	16	4.3	NA	NO	NBA
	7440622	Vanadium	5.2	400	mg/kg	ELY-SS-NF-03 (4-12)	270/270	NA	400	59	39 NC	YES	ASL
	7440666	Zinc	10	1500	mg/kg	ELY-SS-TR-02D (2-12) DUP	270/270	NA	1500	86	2300 NC	NO	BSL

Notes/sources:

(1) Maximum detected concentration used for screening.
(2) Site-specific maximum background value.
(3) Risk-based residential soil concentrations obtained from the Regional Screening Level (RSL) Table (May, 2010).
(4) Hexavalent chromium RSL used for chromium.

C = cancer based screening value set at a target risk of 1E-06.
NA = not available.
NBA = no benchmark available.
NC = noncancer based screening value set at a target hazard quotient of 0.1.

ASL = above screening level.
BSL = below screening level.

Table 4-2
Total Soil - Occurrence, Distribution and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Total soil 0-10 ft bgs

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Total Soil	7429905	Aluminum	860	100000	mg/Kg	ELY-SS-NF-03 (4-12)	348/348	NA	100000	12000	7700 NC	YES	ASL
	7440360	Antimony	0.048	12	mg/Kg	ELY-TP11 (4-6)	124/348	0.46 - 43	12	NA	3.1 NC	YES	ASL
	7440382	Arsenic	0.18	23	mg/Kg	ELY-SS-TZ-05 (0-1)	225/348	0.41 - 8.6	23	4.3	0.39 C	YES	ASL
	7440393	Barium	8.3	220	mg/Kg	SS-13X-082809AX	348/348	NA	220	350	1500 NC	NO	BSL
	7440417	Beryllium	0.04	5.4	mg/Kg	ELY-SS-NF-03 (4-12)	238/348	0.035 - 4.3	5.4	1	16 NC	NO	BSL
	7440439	Cadmium	0.0066	45	mg/Kg	ELY-TP7A (4-5)	271/348	0.2 - 10	45	0.52	7 NC	YES	ASL
	7440702	Calcium	32	42000	mg/Kg	ELY-SS-TR-02A (1.5-11)	347/348	44 - 44	42000	4600	NA	NO	Essential Nutrient
	7440473	Chromium	2.5	440	mg/Kg	ELY-SS-NF-03 (4-12)	348/348	NA	440	32	0.29 C	YES	ASL
	7440484	Cobalt	1.8	1500	mg/Kg	ELY-TP7A (4-5)	348/348	NA	1500	16	2.3 NC	YES	ASL
	7440508	Copper	7.5	36000	mg/Kg	ELY-TP4A (2-3)	348/348	NA	36000	45	310 NC	YES	ASL
	7439896	Iron	2400	300000	mg/Kg	ELY-SS-TZ-14 (4-12)	348/348	NA	300000	31000	5500 NC	YES	ASL
	7439921	Lead	0.72	1700	mg/Kg	ELY-SS-TZ-12 (0-3)	346/346	NA	1700	77	400	YES	ASL
	7439954	Magnesium	9.7	24000	mg/Kg	ELY-SS-NF-03 (4-12)	348/348	NA	24000	4700	NA	NO	Essential Nutrient
	7439965	Manganese	17	8380	mg/Kg	SS-13X-082809AX	348/348	NA	8380	7800	180 NC	YES	ASL
	7439976	Mercury	0.006	2.8	mg/Kg	ELY-SS-TZ-39 (3-8)	295/348	0.006 - 0.13	2.8	0.29	2.3 NC	YES	ASL
	7439987	Molybdenum	0.19	69	mg/Kg	ELY-SS-SA-31 (0-6)	288/348	0.22 - 43	69	0.64	39 NC	YES	ASL
	7440020	Nickel	1.1	470	mg/Kg	ELY-TP7A (4-5)	346/348	3 - 13	470	30	150 NC	YES	ASL
	7440097	Potassium	398	19000	mg/Kg	ELY-SS-TZ-14 (4-12)	347/348	1740 - 1740	19000	3000	NA	NO	Essential Nutrient
	7782492	Selenium	0.24	170	mg/Kg	ELY-SS-SA-31 (0-6)	322/348	0.82 - 11	170	2.9	39 NC	YES	ASL
	7440224	Silver	0.079	23	mg/Kg	ELY-SS-SA-16 (0-6)	272/348	0.11 - 5.3	23	1.4	39 NC	NO	BSL
	7440235	Sodium	23	15000	mg/Kg	ELY-TP7A (4-5)	172/330	94 - 2000	15000	110	NA	NO	Essential Nutrient
	7440246	Strontium	1.2	107	mg/Kg	SS-25X-082709AX	100/100	NA	107	NA	4700 NC	NO	BSL
	7440280	Thallium	0.02	23	mg/Kg	ELY-TP11 (4-6)	204/348	0.25 - 14	23	4.3	NA	NO	NBA
	7440622	Vanadium	5.2	400	mg/Kg	ELY-SS-NF-03 (4-12)	348/348	NA	400	59	39 NC	YES	ASL
	7440666	Zinc	10	5700	mg/Kg	ELY-TP7A (4-5)	348/348	NA	5700	86	2300 NC	YES	ASL

Notes/sources:

(1) Maximum detected concentration used for screening.
(2) Site-specific maximum background value.
(3) Risk-based residential soil concentrations obtained from the Regional Screening Level (RSL) Table (May 2010).
(4) Hexavalent chromium RSL used for chromium.

C = cancer based screening value set at a target risk of 1E-06.
NA = not available.
NBA = no benchmark available.
NC = noncancer based screening value set at a target hazard quotient of 0.1.

ASL = above screening level.
BSL = below screening level.

Table 4-3
Sediment - Occurrence, Distribution, and Selection of Contaminants of Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Surface sediment

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Ely Brook Sediment	7429905	Aluminum	2800	111000	mg/kg	06ElyPond6S	38/38	NA	111000	NA	7700 NC	YES	ASL
	7440360	Antimony	0.06	2.03	mg/kg	06Ely04	16/38	0.05 - 49	2.03	NA	3.1 NC	NO	BSL
	7440382	Arsenic	0.63	18.5	mg/kg	SD-64X-080709AX	23/38	0.99 - 49	18.5	NA	0.39 C	YES	ASL
	7440393	Barium	27	377	mg/kg	06ElyPond3S	38/38	NA	377	NA	1500 NC	NO	BSL
	7440417	Beryllium	0.24	2.2	mg/kg	06ElyPond6S	27/35	0.32 - 2.5	2.2	NA	16 NC	NO	BSL
	7440699	Bismuth	0.17	1.74	mg/kg	06Ely11	12/12	NA	1.74	NA	NA	NO	NBA
	7440439	Cadmium	0.1	4	mg/kg	06ElyPond5S	34/38	3.1 - 7.4	4	NA	7 NC	NO	BSL
	7440702	Calcium	97.3	19300	mg/kg	06ElyPond1S	38/38	NA	19300	NA	NA	NO	Essential Nutrient
	7440451	Cerium	11.4	102	mg/kg	06ElyPond5S	12/12	NA	102	NA	NA	NO	NBA
	7440462	Cesium	45.1	68.6	mg/kg	06ElyPond3S	4/12	5 - 5	68.6	NA	NA	NO	NBA
	7440473	Chromium	17	130	mg/kg	06ElyPond2S	38/38	NA	130	NA	0.29 C	YES	ASL
	7440484	Cobalt	7.6	102	mg/kg	SD-65X-080609AX	38/38	NA	102	NA	2.3 NC	YES	ASL
	7440508	Copper	40	11000	mg/kg	SD-63X-080709AX	38/38	NA	11000	NA	310 NC	YES	ASL
	7440553	Gallium	10.4	17.4	mg/kg	06ElyPond1S	12/12	NA	17.4	NA	NA	NO	NBA
	7440746	Indium	0.04	0.26	mg/kg	06Ely04	12/12	NA	0.26	NA	NA	NO	NBA
	7439896	Iron	13700	167000	mg/kg	06Ely04	38/38	NA	167000	NA	5500 NC	YES	ASL
	7439910	Lanthanum	6	49.4	mg/kg	06ElyPond5S	12/12	NA	49.4	NA	NA	NO	NBA
	7439921	Lead	3.6	174	mg/kg	06Ely03	34/38	20 - 49	174	NA	400	NO	BSL
	7439932	Lithium	10	44	mg/kg	06ElyPond1S	12/12	NA	44	NA	16 NC	YES	ASL
	7439954	Magnesium	1270	15600	mg/kg	06ElyPond1S	38/38	NA	15600	NA	NA	NO	Essential Nutrient
	7439965	Manganese	62	3130	mg/kg	06ElyPond3S	38/38	NA	3130	NA	180 NC	YES	ASL
	7439976	Mercury	0.004	0.15	mg/kg	06ElyPond3S	24/34	0.02 - 0.1	0.15	NA	2.3 NC	NO	BSL
	7439987	Molybdenum	0.35	17.4	mg/kg	06Ely04	32/34	6.4 - 10	17.4	NA	39 NC	NO	BSL
	7440020	Nickel	6	61.1	mg/kg	06ElyPond4S	37/38	15 - 15	61.1	NA	150 NC	NO	BSL
	7440031	Niobium	3.6	10.9	mg/kg	06ElyPond1S	12/12	NA	10.9	NA	NA	NO	NBA
	7723140	Phosphorus	120	1120	mg/kg	06ElyPond3S	12/12	NA	1120	NA	NA	NO	NBA

Table 4-3
Sediment - Occurrence, Distribution, and Selection of Contaminants of Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Surface sediment

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Ely Brook Sediment (cont.)	7440097	Potassium	1500	11100	mg/kg	06ElyPond4S	34/34	NA	11100	NA	NA	NO	Essential Nutrient
	7440177	Rubidium	32.3	54.8	mg/kg	06ElyPond1S	12/12	NA	54.8	NA	NA	NO	NBA
	7440202	Scandium	10	17.5	mg/kg	06ElyPond1S	12/12	NA	17.5	NA	NA	NO	NBA
	7782492	Selenium	0.3	46	mg/kg	ELY-SED-53C (0-0.25)	36/38	10 - 11	46	NA	39 NC	YES	ASL
	7440224	Silver	0.22	9.5	mg/kg	SD-63X-080709AX	18/38	1 - 7.4	9.5	NA	39 NC	NO	BSL
	7440235	Sodium	740	17800	mg/kg	06ElyPond1S	13/20	400 - 810	17800	NA	NA	NO	Essential Nutrient
	7440246	Strontium	2.8	172	mg/kg	06ElyPond1S	26/26	NA	172	NA	4700 NC	NO	BSL
	13493809	Tellurium	0.2	1.1	mg/kg	06Ely11	4/11	0.1 - 0.1	1.1	NA	NA	NO	NBA
	7440280	Thallium	0.064	5.4	mg/kg	ELY-SED-55C (0-0.25) DUP	15/26	4 - 120	5.4	NA	NA	NO	NBA
	7440291	Thorium	2.5	8	mg/kg	06ElyPond4S	12/12	NA	8	NA	NA	NO	NBA
	7440315	Tin	1.4	3	mg/kg	06ElyPond1S	12/12	NA	3	NA	4700 NC	NO	BSL
	7440326	Titanium	0.2	5200	mg/kg	06ElyPond2S	24/24	NA	5200	NA	NA	NO	NBA
	7440337	Tungsten	0.4	0.8	mg/kg	06ElyPond2S	12/12	NA	0.8	NA	NA	NO	NBA
	7440611	Uranium	0.7	6.2	mg/kg	06ElyPond6S	12/12	NA	6.2	NA	NA	NO	NBA
	7440622	Vanadium	22	163	mg/kg	06ElyPond1S	38/38	NA	163	NA	39 NC	YES	ASL
	7440655	Yttrium	7.9	32.1	mg/kg	06ElyPond5S	12/12	NA	32.1	NA	NA	NO	NBA
	7440666	Zinc	34.9	507	mg/kg	06ElyPond5S	37/38	40 - 40	507	NA	2300 NC	NO	BSL
Schoolhouse Brook Sediment	7429905	Aluminum	4850	36900	mg/kg	06Ely08	10/10	NA	36900	31000	7700 NC	YES	ASL
	7440360	Antimony	0.65	2.39	mg/kg	06Ely12	5/10	0.62 - 22	2.39	0.15	3.1 NC	NO	BSL
	7440382	Arsenic	1	2	mg/kg	06Ely12	5/10	0.99 - 22	2	3	0.39 C	YES	ASL
	7440393	Barium	23.2	199	mg/kg	06Ely08R	10/10	NA	199	207	1500 NC	NO	BSL
	7440417	Beryllium	0.15	1.7	mg/kg	06Ely06	6/9	1 - 1.1	1.7	1.8	16 NC	NO	BSL
	7440699	Bismuth	0.18	0.56	mg/kg	06Ely12	5/5	NA	0.56	0.13	NA	NO	NBA
	7440439	Cadmium	0.075	0.3	mg/kg	06Ely12	7/10	3 - 3.3	0.3	NA	7 NC	NO	BSL
	7440702	Calcium	4200	16200	mg/kg	06Ely07	10/10	NA	16200	14200	NA	NO	Essential Nutrient

Table 4-3
Sediment - Occurrence, Distribution, and Selection of Contaminants of Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Surface sediment

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Schoolhouse Brook Sediment (cont.)	7440451	Cerium	19	24.1	mg/kg	06Ely08	5/5	NA	24.1	19.5	NA	NO	NBA
	7440473	Chromium	10.8	40	mg/kg	06Ely12	10/10	NA	40	24	0.29 C	YES	ASL
	7440484	Cobalt	3.4	13.5	mg/kg	06Ely08	10/10	NA	13.5	5	2.3 NC	YES	ASL
	7440508	Copper	160	1390	mg/kg	06Ely12	10/10	NA	1390	10.4	310 NC	YES	ASL
	7440553	Gallium	7.68	8.54	mg/kg	06Ely12	5/5	NA	8.54	7.99	NA	NO	NBA
	7440746	Indium	0.03	0.08	mg/kg	06Ely12	5/5	NA	0.08	0	NA	NO	NBA
	7439896	Iron	8760	58800	mg/kg	06Ely12	10/10	NA	58800	13900	5500 NC	YES	ASL
	7439910	Lanthanum	9.9	11.7	mg/kg	06Ely08	5/5	NA	11.7	10.5	NA	NO	NBA
	7439921	Lead	2.5	31.4	mg/kg	06Ely08	7/10	11 - 20	31.4	11.2	400	NO	BSL
	7439932	Lithium	18	24	mg/kg	06Ely08R	5/5	NA	24	24	16 NC	YES	ASL
	7439954	Magnesium	2040	7000	mg/kg	06Ely08R	10/10	NA	7000	6600	NA	NO	Essential Nutrient
	7439965	Manganese	200	1120	mg/kg	06Ely12	10/10	NA	1120	501	180 NC	YES	ASL
	7439987	Molybdenum	0.26	7.26	mg/kg	06Ely12	7/7	NA	7.26	0.28	39 NC	NO	BSL
	7440020	Nickel	7.3	15	mg/kg	AA62439	10/10	NA	15	14	150 NC	NO	BSL
	7440031	Niobium	3.3	5.1	mg/kg	06Ely08	5/5	NA	5.1	3.8	NA	NO	NBA
	7723140	Phosphorus	230	290	mg/kg	06Ely07	5/5	NA	290	260	NA	NO	NBA
	7440097	Potassium	914	9200	mg/kg	06Ely08	7/7	NA	9200	8100	NA	NO	Essential Nutrient
	7440177	Rubidium	37.7	45	mg/kg	06Ely08	5/5	NA	45	44.3	NA	NO	NBA
	7440202	Scandium	6.6	10.5	mg/kg	06Ely12	5/5	NA	10.5	5.6	NA	NO	NBA
	7782492	Selenium	0.73	9.8	mg/kg	06Ely12	7/10	10 - 11	9.8	NA	39 NC	NO	BSL
	7440235	Sodium	7600	8000	mg/kg	06Ely12	5/5	NA	8000	6800	NA	NO	Essential Nutrient
	7440246	Strontium	40.4	206	mg/kg	06Ely07	7/7	NA	206	202	4700 NC	NO	BSL
	13493809	Tellurium	0.3	0.3	mg/kg	06Ely12	1/5	0.1 - 0.1	0.3	NA	NA	NO	NBA
	7440280	Thallium	0.021	0.032	mg/kg	SD-58X-080709AX	2/5	40 - 50	0.032	NA	NA	NO	NBA
	7440291	Thorium	3.3	3.6	mg/kg	06Ely08R	5/5	NA	3.6	3.5	NA	NO	NBA
	7440315	Tin	1	2.2	mg/kg	06Ely08	5/5	NA	2.2	1.1	4700 NC	NO	BSL

Table 4-3
Sediment - Occurrence, Distribution, and Selection of Contaminants of Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Surface sediment

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Schoolhouse Brook Sediment (cont.)	7440326	Titanium	0.2	2300	mg/kg	06Ely12	9/9	NA	2300	1400	NA	NO	NBA
	7440337	Tungsten	0.4	0.6	mg/kg	06Ely12	5/5	NA	0.6	0.4	NA	NO	NBA
	7440611	Uranium	0.8	1	mg/kg	06Ely12	5/5	NA	1	0.8	NA	NO	NBA
	7440622	Vanadium	9.8	62	mg/kg	06Ely12	10/10	NA	62	40	39 NC	YES	ASL
	7440655	Yttrium	7.8	12.8	mg/kg	06Ely12	5/5	NA	12.8	8.5	NA	NO	NBA
	7440666	Zinc	21.8	93	mg/kg	06Ely12	10/10	NA	93	32	2300 NC	NO	BSL
Ompompanoosuc River Sediment	7429905	Aluminum	6900	35400	mg/kg	06Ely10	4/4	NA	35400	32700	7700 NC	YES	ASL
	7440360	Antimony	0.22	0.22	mg/kg	06Ely10	1/4	20 - 21	0.22	0.19	3.1 NC	NO	BSL
	7440382	Arsenic	5	5	mg/kg	06Ely10	1/4	20 - 21	5	3	0.39 C	YES	ASL
	7440393	Barium	25	195	mg/kg	06Ely10	4/4	NA	195	187	1500 NC	NO	BSL
	7440417	Beryllium	1.8	1.8	mg/kg	06Ely10	1/4	0.99 - 1	1.8	1.6	16 NC	NO	BSL
	7440699	Bismuth	0.07	0.07	mg/kg	06Ely10	1/1	NA	0.07	0.09	NA	NO	NBA
	7440439	Cadmium	0.1	0.1	mg/kg	06Ely10	1/4	3 - 3.1	0.1	NA	7 NC	NO	BSL
	7440702	Calcium	3100	13000	mg/kg	06Ely10	4/4	NA	13000	11300	NA	NO	Essential Nutrient
	7440451	Cerium	21.7	21.7	mg/kg	06Ely10	1/1	NA	21.7	16.5	NA	NO	NBA
	7440473	Chromium	13	31	mg/kg	AA62444	4/4	NA	31	37	0.29 C	YES	ASL
	7440484	Cobalt	6	13	mg/kg	AA62444	4/4	NA	13	5	2.3 NC	YES	ASL
	7440508	Copper	35	120	mg/kg	AA62444	4/4	NA	120	4.5	310 NC	NO	BSL
	7440553	Gallium	7.77	7.77	mg/kg	06Ely10	1/1	NA	7.77	7.68	NA	NO	NBA
	7440746	Indium	0.03	0.03	mg/kg	06Ely10	1/1	NA	0.03	NA	NA	NO	NBA
	7439896	Iron	7900	22800	mg/kg	06Ely10	4/4	NA	22800	14000	5500 NC	YES	ASL
	7439910	Lanthanum	10.9	10.9	mg/kg	06Ely10	1/1	NA	10.9	9.3	NA	NO	NBA
	7439921	Lead	10.4	10.4	mg/kg	06Ely10	1/4	20 - 21	10.4	9.6	400	NO	BSL
	7439932	Lithium	24	24	mg/kg	06Ely10	1/1	NA	24	28	16 NC	YES	ASL
	7439954	Magnesium	3100	8000	mg/kg	AA62444	4/4	NA	8000	7300	NA	NO	Essential Nutrient

Table 4-3
Sediment - Occurrence, Distribution, and Selection of Contaminants of Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Surface sediment

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Ompompanoosuc River Sediment (cont.)	7439965	Manganese	170	1120	mg/kg	06Ely10	4/4	NA	1120	475	180 NC	YES	ASL
	7439987	Molybdenum	0.5	0.5	mg/kg	06Ely10	1/1	NA	0.5	0.16	39 NC	NO	BSL
	7440020	Nickel	9.9	21	mg/kg	AA62444	4/4	NA	21	11.7	150 NC	NO	BSL
	7440031	Niobium	5.3	5.3	mg/kg	06Ely10	1/1	NA	5.3	4	NA	NO	NBA
	7723140	Phosphorus	270	270	mg/kg	06Ely10	1/1	NA	270	260	NA	NO	NBA
	7440097	Potassium	8400	8400	mg/kg	06Ely10	1/1	NA	8400	8000	NA	NO	Essential Nutrient
	7440177	Rubidium	40.5	40.5	mg/kg	06Ely10	1/1	NA	40.5	42	NA	NO	NBA
	7440202	Scandium	8.6	8.6	mg/kg	06Ely10	1/1	NA	8.6	5.6	NA	NO	NBA
	7782492	Selenium	0.3	0.3	mg/kg	06Ely10	1/4	9.9 - 10	0.3	NA	39 NC	NO	BSL
	7440235	Sodium	7600	7600	mg/kg	06Ely10	1/1	NA	7600	7200	NA	NO	Essential Nutrient
	7440246	Strontium	193	193	mg/kg	06Ely10	1/1	NA	193	198	4700 NC	NO	BSL
	7440291	Thorium	3.5	3.5	mg/kg	06Ely10	1/1	NA	3.5	2.8	NA	NO	NBA
	7440315	Tin	1.7	1.7	mg/kg	06Ely10	1/1	NA	1.7	1.1	4700 NC	NO	BSL
	7440326	Titanium	0.2	2700	mg/kg	06Ely10	2/2	NA	2700	1600	NA	NO	NBA
	7440337	Tungsten	0.4	0.4	mg/kg	06Ely10	1/1	NA	0.4	0.3	NA	NO	NBA
	7440611	Uranium	0.8	0.8	mg/kg	06Ely10	1/1	NA	0.8	0.7	NA	NO	NBA
	7440622	Vanadium	14	49	mg/kg	06Ely10	4/4	NA	49	38	39 NC	YES	ASL
	7440655	Yttrium	14	14	mg/kg	06Ely10	1/1	NA	14	8.1	NA	NO	NBA
	7440666	Zinc	28	65	mg/kg	AA62444	4/4	NA	65	33	2300 NC	NO	BSL

Notes/sources:

(1) Maximum detected concentration used for screening.

(2) Background data not available for Ely Brook. Site-specific maximum background value used for Schoolhouse Brook and Ompomanoosuc River.

(3) Risk-based residential soil concentrations obtained from the Regional Screening Level (RSL) Table (May 2010).

(4) Hexavalent chromium RSL used for chromium.

C = cancer based screening value set at a target risk of 1E-06.

NA = not available.

NBA = no benchmark available.

NC = noncancer based screening value set at a target hazard quotient of 0.1.

ASL = above screening level.

BSL = below screening level.

Table 4-4
Surface Water - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Surface Water
Exposure Medium: Surface water

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Ely Brook Surface Water	7429905	Aluminum	18	103590	µg/L	SW-43-120209AX	78/92	200 - 349	103590	NA	3700 NC	YES	ASL
	7440360	Antimony	0.34	0.67	µg/L	AA62297	2/92	0.02 - 60	0.67	NA	1.5 NC	NO	BSL
	7440382	Arsenic	0.09	0.64	µg/L	SW-72-111809AX	9/92	0.5 - 10	0.64	NA	0.045 C	YES	ASL
	7440393	Barium	4.2	46.1	µg/L	SW-48X-080509AX	92/92	NA	46.1	NA	730 NC	NO	BSL
	7440417	Beryllium	0.03	1.9	µg/L	SW-43-120209AX	42/92	0.05 - 5	1.9	NA	7.3 NC	NO	BSL
	7440439	Cadmium	0.02	55.8	µg/L	SW-43-120209AX	74/92	0.02 - 0.5	55.8	NA	1.8 NC	YES	ASL
	7440702	Calcium	3700	109890	µg/L	SW-43-120209AX	92/92	NA	109890	NA	NA	NO	Essential Nutrient
	7440451	Cerium	0.12	23.4	µg/L	06Ely04A RA	10/10	NA	23.4	NA	NA	NO	NBA
	7440462	Cesium	0.04	1.26	µg/L	06Ely04A RA	10/10	NA	1.26	NA	NA	NO	NBA
	16887006	Chloride	110	3600	µg/L	EMTT-3	65/75	200 - 390	3600	NA	NA	NO	NBA
	7440473	Chromium	0.1	125	µg/L	SW-43-120209AX	49/92	0.16 - 10	125	NA	0.043 C	YES	ASL
	7440484	Cobalt	0.16	1174	µg/L	SW-43-120209AX	84/92	50 - 50	1174	NA	1.1 NC	YES	ASL
	7440508	Copper	1.3	33178	µg/L	SW-43-120209AX	92/92	NA	33178	NA	150 NC	YES	ASL
	57125	Cyanide	3.4	3.4	µg/L	SW-29-111709AX	1/75	5 - 10	3.4	NA	73 NC	NO	BSL
	7429916	Dysprosium	0.01	1.74	µg/L	06Ely04A RA	10/10	NA	1.74	NA	NA	NO	NBA
	7440520	Erbium	0.007	0.75	µg/L	06Ely04A RA	10/10	NA	0.75	NA	NA	NO	NBA
	7440531	Europium	0.005	0.49	µg/L	06Ely04A RA	10/10	NA	0.49	NA	NA	NO	NBA
	7440542	Gadolinium	0.02	2.53	µg/L	06Ely04A RA	10/10	NA	2.53	NA	NA	NO	NBA
	7440553	Gallium	0.05	0.2	µg/L	06Ely04A FA	5/10	0.05 - 0.05	0.2	NA	NA	NO	NBA
	7440564	Germanium	0.05	0.05	µg/L	06ElyPond6 RA	2/10	0.05 - 0.05	0.05	NA	NA	NO	NBA
	7440600	Holmium	0.02	0.29	µg/L	06Ely04A RA	6/10	0.005 - 0.005	0.29	NA	NA	NO	NBA
	7439896	Iron	19.3	28300	µg/L	SW-47-111609-AX	83/92	50 - 100	28300	NA	2600 NC	YES	ASL
	7439910	Lanthanum	0.06	11.3	µg/L	06Ely04A RA	10/10	NA	11.3	NA	NA	NO	NBA
	7439921	Lead	0.03	3.9	µg/L	SW-72-111809AX	65/92	0.04 - 10	3.9	NA	15	NO	BSL
	7439932	Lithium	0.1	15.1	µg/L	06Ely04A RA	8/10	0.1 - 0.1	15.1	NA	7.3 NC	YES	ASL
	7439954	Magnesium	670	67619	µg/L	SW-43-120209AX	92/92	NA	67619	NA	NA	NO	Essential Nutrient

Table 4-4
Surface Water - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Surface Water
Exposure Medium: Surface water

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Ely Brook Surface Water (cont.)	7439965	Manganese	2.4	5230	µg/L	SW-43-120209AX	92/92	NA	5230	NA	88 NC	YES	ASL
	7439976	Mercury	0.099	0.099	µg/L	MA1PY9	1/76	0.1 - 0.2	0.099	NA	0.057 NC	YES	ASL
	7439987	Molybdenum	1.4	1.4	µg/L	SW-43-120209AX	1/91	0.06 - 10	1.4	NA	18 NC	NO	BSL
	7440008	Neodymium	0.08	12.8	µg/L	06Ely04A RA	10/10	NA	12.8	NA	NA	NO	NBA
	7440020	Nickel	0.4	308	µg/L	SW-43-120209AX	69/76	0.4 - 40	308	NA	73 NC	YES	ASL
	14797558	Nitrogen, Nitrite + Nitrate	13	170	µg/L	SW-46-111609AX	16/75	20 - 110	170	NA	5800 NC	NO	BSL
	7723140	Phosphorus	3	36	µg/L	06ElyPond1 FA	10/16	10 - 10	36	NA	NA	NO	NBA
	7440097	Potassium	506	8630	µg/L	SW-78-111609AX	86/86	NA	8630	NA	NA	NO	Essential Nutrient
	7440100	Praseodymium	0.02	3.2	µg/L	06Ely04A RA	10/10	NA	3.2	NA	NA	NO	NBA
	7440177	Rubidium	3.61	15.6	µg/L	06Ely04A RA	10/10	NA	15.6	NA	NA	NO	NBA
	7440199	Samarium	0.02	2.51	µg/L	06Ely04A RA	10/10	NA	2.51	NA	NA	NO	NBA
	7440202	Scandium	0.6	2.8	µg/L	06Ely04A RA	9/10	0.6 - 0.6	2.8	NA	NA	NO	NBA
	7782492	Selenium	0.4	9.3	µg/L	SW-43-120209AX	32/92	1 - 35	9.3	NA	18 NC	NO	BSL
	99439288	Silica	1800	33100	µg/L	06Ely04A FA	10/10	NA	33100	NA	NA	NO	NBA
	7440224	Silver	0.03	0.8	µg/L	SW-43-120209AX	22/92	0.2 - 10	0.8	NA	18 NC	NO	BSL
	7440235	Sodium	533	7970	µg/L	SW-43-120209AX	84/85	2000 - 2000	7970	NA	NA	NO	Essential Nutrient
	7440246	Strontium	19	209	µg/L	SW-78-111609AX	85/85	NA	209	NA	2200 NC	NO	BSL
	14808798	Sulfate	3700	1300000	µg/L	SW-43-120209AX	84/85	2000 - 2000	1300000	NA	NA	NO	NBA
	18496258	Sulfide	430	1300	µg/L	SW-17X-080409AX	20/75	100 - 2000	1300	NA	NA	NO	NBA
	7440257	Tantalum	0.04	0.04	µg/L	06Ely02A RA	1/10	0.02 - 0.02	0.04	NA	NA	NO	NBA
	7440279	Terbium	0.005	0.34	µg/L	06Ely04A RA	8/10	0.005 - 0.005	0.34	NA	NA	NO	NBA
	7440280	Thallium	0.024	0.31	µg/L	SW-43-120209AX	24/92	0.04 - 25	0.31	NA	NA	NO	NBA
	7440304	Thulium	0.005	0.085	µg/L	06Ely04A RA	6/10	0.005 - 0.005	0.085	NA	NA	NO	NBA

Table 4-4
Surface Water - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Surface Water
Exposure Medium: Surface water

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Ely Brook Surface Water (cont.)	7440326	Titanium	0.7	30.4	µg/L	06ElyPond1 RA	10/10	NA	30.4	NA	NA	NO	NBA
	7440611	Uranium	0.11	0.43	µg/L	06Ely04A RA	4/16	0.1 - 1	0.43	NA	NA	NO	NBA
	7440622	Vanadium	0.06	11.3	µg/L	SW-72-111809AX	62/92	0.2 - 5	11.3	NA	0.26 NC	YES	ASL
	7440644	Ytterbium	0.006	0.52	µg/L	06Ely04A RA	10/10	NA	0.52	NA	NA	NO	NBA
	7440655	Yttrium	0.07	6.16	µg/L	06ElyPond6 RA	10/10	NA	6.16	NA	NA	NO	NBA
	7440666	Zinc	0.8	8192	µg/L	SW-43-120209AX	88/92	60 - 60	8192	NA	1100 NC	YES	ASL
Schoolhouse Brook Surface Water	7429905	Aluminum	160	1500	µg/L	AA62299	18/22	200 - 394	1500	91	3700 NC	NO	BSL
	7440360	Antimony	0.63	0.7	µg/L	AA62299	3/22	0.05 - 60	0.7	NA	1.5 NC	NO	BSL
	7440393	Barium	10.6	20.6	µg/L	06Ely19A RA	22/22	NA	20.6	20.4	730 NC	NO	BSL
	7440439	Cadmium	0.07	1.2	µg/L	AA62299	8/22	0.08 - 5	1.2	NA	1.8 NC	NO	BSL
	7440702	Calcium	9200	36700	µg/L	06Ely19A RA	22/22	NA	36700	36800	NA	NO	Essential Nutrient
	7440451	Cerium	0.64	1.52	µg/L	06Ely19A-R RA	5/5	NA	1.52	0.06	NA	NO	NBA
	7440462	Cesium	0.11	0.15	µg/L	06Ely19A RA	5/5	NA	0.15	0.07	NA	NO	NBA
	16887006	Chloride	2500	5000	µg/L	06Ely06A FA	6/6	NA	5000	4200	NA	NO	NBA
	7440473	Chromium	0.44	4.5	µg/L	AA62011	12/22	0.37 - 10	4.5	3	0.043 C	YES	ASL
	7440484	Cobalt	1.4	25	µg/L	AA62299	18/22	2 - 50	25	0.1	1.1 NC	YES	ASL
	7440508	Copper	55	1100	µg/L	AA62299	20/22	2 - 2	1100	0.94	150 NC	YES	ASL
	7429916	Dysprosium	0.051	0.12	µg/L	06Ely19A-R RA	5/5	NA	0.12	NA	NA	NO	NBA
	7440520	Erbium	0.02	0.05	µg/L	06Ely19A RA	5/5	NA	0.05	NA	NA	NO	NBA
	7440531	Europium	0.02	0.04	µg/L	06Ely19A-R RA	5/5	NA	0.04	NA	NA	NO	NBA
	7440542	Gadolinium	0.073	0.18	µg/L	06Ely19A-R RA	5/5	NA	0.18	0.006	NA	NO	NBA
	7440600	Holmium	0.008	0.02	µg/L	06Ely19A RA	5/5	NA	0.02	NA	NA	NO	NBA
	7439896	Iron	35.7	1080	µg/L	MA1PZ2	21/22	105 - 105	1080	160	2600 NC	NO	BSL
	7439910	Lanthanum	0.32	0.75	µg/L	06Ely19A-R RA	5/5	NA	0.75	0.04	NA	NO	NBA
	7439921	Lead	0.036	16	µg/L	AA62303	11/22	0.1 - 10	16	NA	15	YES	ASL
	7439932	Lithium	1.6	4.2	µg/L	06Ely19A RA	5/5	NA	4.2	3.1	7.3 NC	NO	BSL
	7439954	Magnesium	1100	2340	µg/L	06Ely19A RA	22/22	NA	2340	2020	NA	NO	Essential Nutrient

Table 4-4
Surface Water - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Surface Water
Exposure Medium: Surface water

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Schoolhouse Brook Surface Water (cont.)	7439965	Manganese	8	260	µg/L	AA62299	22/22	NA	260	15	88 NC	YES	ASL
	7439976	Mercury	0.13	0.17	µg/L	MA1PZ2	3/9	0.1 - 0.2	0.17	NA	0.057 NC	YES	ASL
	7440008	Neodymium	0.36	0.86	µg/L	06Ely19A-R RA	5/5	NA	0.86	0.04	NA	NO	NBA
	7440020	Nickel	0.61	8.9	µg/L	AA62299	19/22	40 - 40	8.9	1.1	73 NC	NO	BSL
	7440031	Niobium	0.29	0.67	µg/L	06Ely08A RA	2/5	0.2 - 0.2	0.67	NA	NA	NO	NBA
	7440097	Potassium	1122	2600	µg/L	06Ely19A RA	14/14	NA	2600	2510	NA	NO	Essential Nutrient
	7440100	Praseodymium	0.08	0.21	µg/L	06Ely19A-R RA	5/5	NA	0.21	0.01	NA	NO	NBA
	7440177	Rubidium	5	5.44	µg/L	06Ely06A RA	5/5	NA	5.44	4.91	NA	NO	NBA
	7440199	Samarium	0.07	0.17	µg/L	06Ely19A-R RA	5/5	NA	0.17	0.01	NA	NO	NBA
	7440202	Scandium	0.8	0.9	µg/L	06Ely19A RA	5/5	NA	0.9	0.7	NA	NO	NBA
	7782492	Selenium	8.5	8.6	µg/L	MA1PZ2	2/22	1 - 35	8.6	NA	18 NC	NO	BSL
	99439288	Silica	6500	9100	µg/L	06Ely06A FA	5/5	NA	9100	7300	NA	NO	NBA
	7440224	Silver	0.04	0.04	µg/L	ELY-SW-06	1/22	0.2 - 10	0.04	NA	18 NC	NO	BSL
	7440235	Sodium	1700	5020	µg/L	06Ely06A RA	12/12	NA	5020	4500	NA	NO	Essential Nutrient
	7440246	Strontium	88	158	µg/L	06Ely06A RA	11/11	NA	158	166	2200 NC	NO	BSL
	14808798	Sulfate	5900	21000	µg/L	06Ely19A RA	11/11	NA	21000	10000	NA	NO	NBA
	7440257	Tantalum	0.04	0.1	µg/L	06Ely19A FA	2/5	0.02 - 0.02	0.1	NA	NA	NO	NBA
	7440279	Terbium	0.01	0.02	µg/L	06Ely19A-R RA	5/5	NA	0.02	NA	NA	NO	NBA
	7440304	Thulium	0.005	0.005	µg/L	06Ely19A-R RA	1/5	0.005 - 0.005	0.005	NA	NA	NO	NBA
	7440326	Titanium	0.9	3.4	µg/L	06Ely08A RA	5/5	NA	3.4	1.3	NA	NO	NBA
	7440337	Tungsten	0.56	0.56	µg/L	06Ely08A RA	1/5	0.5 - 0.5	0.56	NA	NA	NO	NBA
	7440611	Uranium	0.28	0.32	µg/L	06Ely19A-R RA	5/13	1 - 1	0.32	0.33	NA	NO	NBA
	7440622	Vanadium	0.3	0.97	µg/L	MA1PZ2	8/22	0.2 - 50	0.97	0.35	0.26 NC	YES	ASL
	7440644	Ytterbium	0.02	0.04	µg/L	06Ely19A RA	5/5	NA	0.04	NA	NA	NO	NBA
	7440655	Yttrium	0.22	0.44	µg/L	06Ely19A-R RA	5/5	NA	0.44	0.03	NA	NO	NBA
	7440666	Zinc	2.9	150	µg/L	AA62299	21/22	60 - 60	150	432.5	1100 NC	NO	BSL

Table 4-4
Surface Water - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Surface Water
Exposure Medium: Surface water

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Ompompanoosuc River Surface Water	7429905	Aluminum	112	149	µg/L	MA1Q03	5/5	NA	149	160	3700 NC	NO	BSL
	7440360	Antimony	0.76	0.76	µg/L	06Ely20A RA	1/5	0.3 - 60	0.76	NA	1.5 NC	NO	BSL
	7440393	Barium	10	22.6	µg/L	06Ely20A RA	5/5	NA	22.6	22.1	730 NC	NO	BSL
	7440439	Cadmium	0.02	0.02	µg/L	06Ely10A RA	1/5	0.02 - 5	0.02	NA	1.8 NC	NO	BSL
	7440702	Calcium	16500	32800	µg/L	06Ely20A RA	5/5	NA	32800	31500	NA	NO	Essential Nutrient
	7440451	Cerium	0.31	0.38	µg/L	06Ely10A RA	2/2	NA	0.38	0.46	NA	NO	NBA
	7440462	Cesium	0.1	0.11	µg/L	06Ely20A RA	2/2	NA	0.11	0.12	NA	NO	NBA
	7440484	Cobalt	0.18	0.42	µg/L	06Ely10A RA	2/5	0.56 - 50	0.42	0.11	1.1 NC	NO	BSL
	7440508	Copper	5.9	19	µg/L	MA1PZ9	5/5	NA	19	0.6	150 NC	NO	BSL
	7429916	Dysprosium	0.03	0.03	µg/L	06Ely10A RA	2/2	NA	0.03	0.03	NA	NO	NBA
	7440520	Erbium	0.01	0.02	µg/L	06Ely10A RA	2/2	NA	0.02	0.02	NA	NO	NBA
	7440531	Europium	0.009	0.01	µg/L	06Ely10A RA	2/2	NA	0.01	0.01	NA	NO	NBA
	7440542	Gadolinium	0.03	0.05	µg/L	06Ely10A RA	2/2	NA	0.05	0.04	NA	NO	NBA
	7440600	Holmium	0.005	0.006	µg/L	06Ely10A RA	2/2	NA	0.006	0.005	NA	NO	NBA
	7439896	Iron	110	185	µg/L	MA1Q03	5/5	NA	185	162	2600 NC	NO	BSL
	7439910	Lanthanum	0.17	0.21	µg/L	06Ely10A RA	2/2	NA	0.21	0.25	NA	NO	NBA
	7439921	Lead	0.1	0.1	µg/L	06Ely10A RA	2/5	10 - 10	0.1	0.2	15	NO	BSL
	7439932	Lithium	2.6	2.8	µg/L	06Ely20A RA	2/2	NA	2.8	2.8	7.3 NC	NO	BSL
	7439954	Magnesium	1130	1750	µg/L	06Ely20A RA	5/5	NA	1750	1560	NA	NO	Essential Nutrient
	7439965	Manganese	11.7	30.2	µg/L	06Ely20A RA	5/5	NA	30.2	28.2	88 NC	NO	BSL
	7439976	Mercury	0.12	0.2	µg/L	MA1PZ9	3/3	NA	0.2	NA	0.057 NC	YES	ASL
	7440008	Neodymium	0.15	0.22	µg/L	06Ely10A RA	2/2	NA	0.22	0.24	NA	NO	NBA
	7440020	Nickel	0.4	0.5	µg/L	06Ely10A RA	2/5	40 - 40	0.5	0.5	73 NC	NO	BSL
	7440097	Potassium	1350	2400	µg/L	06Ely20A RA	5/5	NA	2400	2240	NA	NO	Essential Nutrient
	7440100	Praseodymium	0.04	0.06	µg/L	06Ely10A RA	2/2	NA	0.06	0.06	NA	NO	NBA
	7440177	Rubidium	5.25	5.48	µg/L	06Ely20A RA	2/2	NA	5.48	5.53	NA	NO	NBA

Table 4-4
Surface Water - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Surface Water
Exposure Medium: Surface water

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Ompompanoosuc River Surface Water (cont.)	7440199	Samarium	0.03	0.04	µg/L	06Ely10A RA	2/2	NA	0.04	0.04	NA	NO	NBA
	7440202	Scandium	0.7	0.8	µg/L	06Ely20A RA	2/2	NA	0.8	0.7	NA	NO	NBA
	7782492	Selenium	5.8	5.8	µg/L	MA1PZ9	1/5	1 - 35	5.8	NA	18 NC	NO	BSL
	99439288	Silica	6500	7800	µg/L	06Ely10A FA	2/2	NA	7800	7200	NA	NO	NBA
	7440235	Sodium	5270	5980	µg/L	06Ely20A RA	2/2	NA	5980	5380	NA	NO	Essential Nutrient
	7440246	Strontium	141	142	µg/L	06Ely20A RA	2/2	NA	142	141	2200 NC	NO	BSL
	14808798	Sulfate	10000	10000	µg/L	06Ely10A RA	2/2	NA	10000	8000	NA	NO	NBA
	7440257	Tantalum	0.06	0.06	µg/L	06Ely20A RA	1/2	0.02 - 0.02	0.06	NA	NA	NO	NBA
	7440279	Terbium	0.006	0.006	µg/L	06Ely10A RA	1/2	0.005 - 0.005	0.006	0.006	NA	NO	NBA
	7440326	Titanium	3.8	4.3	µg/L	06Ely20A RA	2/2	NA	4.3	7.2	NA	NO	NBA
	7440611	Uranium	0.25	0.25	µg/L	06Ely10A RA	2/2	NA	0.25	0.26	NA	NO	NBA
	7440644	Ytterbium	0.01	0.01	µg/L	06Ely20A RA	2/2	NA	0.01	0.02	NA	NO	NBA
	7440655	Yttrium	0.13	0.15	µg/L	06Ely10A RA	2/2	NA	0.15	0.18	NA	NO	NBA
	7440666	Zinc	3	6.2	µg/L	MA1Q01	5/5	NA	6.2	16.3	1100 NC	NO	BSL

Notes/sources:

(1) Maximum detected concentration used for screening.

(2) Background data not available for Ely Brook. Site-specific maximum background value used for Schoolhouse Brook and Ompomanoosuc River.

(3) Risk-based tap water concentrations obtained from the Regional Screening Level (RSL) Table (May 2010).

(4) Hexavalent chromium RSL used for chromium.

C = cancer based screening value set at a target risk of 1E-06.
NA = not available.
NBA = no benchmark available.
NC = noncancer based screening value set at a target hazard quotient of 0.1.
ASL = above screening level.
BSL = below screening level.

Table 4-5
Site Groundwater - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Site Groundwater
Exposure Medium: Site Groundwater

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Overburden Groundwater	117817	Bis(2-Ethylhexyl) Phthalate	2.6	2.6	µg/L	MW-22A-113009AX	1/8	5 - 5	2.6	NA	4.8 C	NO	BSL
	7429905	Aluminum	48	150000	µg/L	ELY-MW-02B	22/24	100 - 223	150000	2000	3700 NC	YES	ASL
	7440360	Antimony	0.31	1.4	µg/L	ELY-GW-MW-15A	3/24	0.02 - 2.2	1.4	NA	1.5 NC	NO	BSL
	7440382	Arsenic	0.1	7.6	µg/L	MW-21A-113009AD	17/24	0.12 - 10	7.6	NA	0.045 C	YES	ASL
	7440393	Barium	4.1	1202	µg/L	ELY-MW-02B	24/24	NA	1202	25.2	730 NC	YES	ASL
	7440417	Beryllium	0.057	3.2	µg/L	ELY-MW-02B	13/24	0.03 - 1	3.2	0.035	7.3 NC	NO	BSL
	7440439	Cadmium	0.05	36.5	µg/L	MW-18A-120109AX	15/24	0.02 - 5	36.5	NA	1.8 NC	YES	ASL
	7440702	Calcium	4310	380000	µg/L	ELY-MW-02B	24/24	NA	380000	8400	NA	NO	Essential Nutrient
	16887006	Chloride	190	51000	µg/L	MW-01A-081009AX	24/24	420 - 440	51000	760	NA	NO	NBA
	7440473	Chromium	0.11	365.4	µg/L	ELY-MW-05A	20/24	0.23 - 10	365.4	6.1	0.043 C	YES	ASL
	7440484	Cobalt	0.6	668	µg/L	MW-18A-120109AX	21/24	0.17 - 50	668	1.4	1.1 NC	YES	ASL
	7440508	Copper	0.7	49581	µg/L	MW-18A-120109AX	22/24	0.7 - 50	49581	5.3	150 NC	YES	ASL
	57125	Cyanide	2.9	3.5	µg/L	ELY-MW-01B	4/24	5 - 10	3.5	NA	73 NC	NO	BSL
	7439896	Iron	17.9	200000	µg/L	ELY-MW-02B	23/24	100 - 100	200000	2200	2600 NC	YES	ASL
	7439921	Lead	0.17	52.2	µg/L	ELY-MW-02B	15/24	0.07 - 10	52.2	NA	15	YES	ASL
	7439954	Magnesium	639	90000	µg/L	ELY-MW-02B	24/24	NA	90000	1800	NA	NO	Essential Nutrient
	7439965	Manganese	2.1	4026	µg/L	ELY-MW-05B	24/24	NA	4026	85.6	88 NC	YES	ASL
	7439976	Mercury	0.02	0.04	µg/L	ELY-MW-02B	4/24	0.01 - 0.2	0.04	NA	0.057 NC	NO	BSL
	7439987	Molybdenum	1.2	52.4	µg/L	ELY-MW-05A	15/24	0.06 - 10	52.4	NA	18 NC	YES	ASL
	7440020	Nickel	0.54	402.1	µg/L	ELY-GW-MW-09A	23/24	40 - 40	402.1	6.1	73 NC	YES	ASL
	14797558	Nitrogen, Nitrite + Nitrate	12	2000	µg/L	ELY-MW-05B	23/24	20 - 100	2000	210	5800 NC	NO	BSL
	7440097	Potassium	915	84000	µg/L	ELY-MW-02B	24/24	NA	84000	1900	NA	NO	Essential Nutrient
	7782492	Selenium	0.3	11	µg/L	ELY-MW-02B	15/24	1 - 35	11	NA	18 NC	NO	BSL
	7440224	Silver	0.03	1.1	µg/L	MW-05B-081209AX	13/24	0.03 - 10	1.1	NA	18 NC	NO	BSL

Table 4-5
Site Groundwater - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Site Groundwater
Exposure Medium: Site Groundwater

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Overburden Groundwater (cont.)	7440235	Sodium	1000	110000	µg/L	ELY-MW-05B	24/24	NA	110000	2300	NA	NO	Essential Nutrient
	7440246	Strontium	15	1800	µg/L	ELY-MW-02B	24/24	NA	1800	32	2200 NC	NO	BSL
	14808798	Sulfate	5000	930000	µg/L	MW-18A-120109AX	24/24	NA	930000	7200	NA	NO	NBA
	18496258	Sulfide	290	1600	µg/L	MW-05B-081209AD	17/24	100 - 2000	1600	1000	NA	NO	NBA
	7440280	Thallium	0.033	3	µg/L	ELY-MW-02B	8/24	0.03 - 1	3	NA	NA	NO	NBA
	7440622	Vanadium	0.1	260.8	µg/L	ELY-MW-02B	18/24	0.11 - 50	260.8	4.4	0.26 NC	YES	ASL
	7440666	Zinc	2.9	3867	µg/L	MW-18A-120109AX	20/24	1.53 - 60	3867	175.1	1100 NC	YES	ASL
Shallow Bedrock Groundwater	7429905	Aluminum	30	360000	µg/L	ELY-GW-BOM3	15/16	200 - 200	360000	3700	3700 NC	YES	ASL
	7440360	Antimony	0.4	4.32	µg/L	ELY-GW-BOM3	2/16	0.02 - 2	4.32	NA	1.5 NC	YES	ASL
	7440382	Arsenic	0.11	4.88	µg/L	ELY-GW-BOM3	12/16	0.11 - 10	4.88	0.13	0.045 C	YES	ASL
	7440393	Barium	4.1	146	µg/L	ELY-MW-02C	16/16	NA	146	37	730 NC	NO	BSL
	7440417	Beryllium	0.06	7.3	µg/L	ELY-GW-BOM3	7/16	0.03 - 1	7.3	0.9	7.3 NC	YES	ASL
	7440439	Cadmium	0.05	13	µg/L	MW-21C-112409AX	9/16	0.02 - 5	13	NA	1.8 NC	YES	ASL
	7440702	Calcium	7912	139000	µg/L	MW-05C-081309AX	16/16	NA	139000	10000	NA	NO	Essential Nutrient
	16887006	Chloride	290	2600	µg/L	MW-14C-081309AX	16/16	370 - 370	2600	770	NA	NO	NBA
	7440473	Chromium	0.1	172	µg/L	ELY-GW-BOM3	15/16	0.4 - 10	172	4.9	0.043 C	YES	ASL
	7440484	Cobalt	0.6	267	µg/L	MW-21C-112409AX	13/16	0.14 - 50	267	5.4	1.1 NC	YES	ASL
	7440508	Copper	1.1	10600	µg/L	MW-19C-081409AX	15/16	0.9 - 25	10600	8.8	150 NC	YES	ASL
	57125	Cyanide	2.9	3.2	µg/L	ELY-GW-MW-09C	2/16	5 - 10	3.2	2.4	73 NC	NO	BSL
	7439896	Iron	20.3	310000	µg/L	ELY-GW-BOM3	16/16	100 - 100	310000	2400	2600 NC	YES	ASL
	7439921	Lead	0.04	27.2	µg/L	ELY-GW-BOM3	10/16	0.05 - 10	27.2	1.4	15	YES	ASL
	7439954	Magnesium	656	22523	µg/L	MW-21C-112409AX	16/16	NA	22523	2400	NA	NO	Essential Nutrient
	7439965	Manganese	1.1	3000	µg/L	MW-21C-081409AX	16/16	3.91 - 15	3000	123.2	88 NC	YES	ASL
	7439976	Mercury	0	0	µg/L	0	0/16	0.01 - 0.2	0	NA	0.057 NC	NO	BSL
	7439987	Molybdenum	0.88	92.7	µg/L	ELY-GW-MW-10C	11/16	0.19 - 10	92.7	NA	18 NC	YES	ASL

Table 4-5
Site Groundwater - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Site Groundwater
Exposure Medium: Site Groundwater

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Shallow Bedrock Groundwater (cont.)	7440020	Nickel	0.5	125	µg/L	ELY-GW-BOM3	16/16	40 - 40	125	5.4	73 NC	YES	ASL
	14797558	Nitrogen, Nitrite + Nitrate	14	490	µg/L	ELY-MW-02C	15/16	20 - 20	490	230	5800 NC	NO	BSL
	7440097	Potassium	1351	15000	µg/L	ELY-GW-MW-09C	16/16	NA	15000	2600	NA	NO	Essential Nutrient
	7782492	Selenium	0.4	8.9	µg/L	ELY-GW-BOM3	11/16	1 - 35	8.9	0.6	18 NC	NO	BSL
	7440224	Silver	0.33	4.15	µg/L	ELY-GW-BOM3	7/16	0.03 - 10	4.15	NA	18 NC	NO	BSL
	7440235	Sodium	1200	27700	µg/L	MW-09C-081309AX	16/16	NA	27700	6700	NA	NO	Essential Nutrient
	7440246	Strontium	45.6	461	µg/L	MW-05C-081309AX	16/16	NA	461	70	2200 NC	NO	BSL
	14808798	Sulfate	5700	600000	µg/L	MW-21C-112409AX	16/16	NA	600000	6000	NA	NO	NBA
	18496258	Sulfide	50	830	µg/L	MW-20C-081309AX	10/16	100 - 2000	830	400	NA	NO	NBA
	7440280	Thallium	0.035	2.33	µg/L	ELY-GW-BOM3	4/16	0.03 - 1	2.33	NA	NA	NO	NBA
	7440622	Vanadium	0.07	36.6	µg/L	ELY-GW-BOM3	15/16	50 - 50	36.6	2.5	0.26 NC	YES	ASL
	7440666	Zinc	2.8	1902	µg/L	MW-21C-112409AX	15/16	2.55 - 60	1902	22	1100 NC	YES	ASL
Deep Bedrock Groundwater	117817	Bis(2-Ethylhexyl) Phthalate	7.5	7.5	µg/L	MW-19D-120209AX	1/2	5 - 5	7.5	NA	4.8 C	YES	ASL
	12672296	Aroclor 1248	0.48	0.48	µg/L	MW-20D-120209AX	1/2	1 - 1	0.48	NA	0.034 C	NO	See footnote
	50293	4,4'-DDT	0.058	0.058	µg/L	MW-20D-120209AX	1/2	0.1 - 0.1	0.058	NA	0.2 C	NO	BSL
	72548	4,4'-DDD	0.03	0.03	µg/L	MW-20D-120209AX	1/2	0.1 - 0.1	0.03	NA	0.28 C	NO	BSL
	72559	4,4'-DDE	0.024	0.024	µg/L	MW-20D-120209AX	1/2	0.1 - 0.1	0.024	NA	0.2 C	NO	BSL
	7429905	Aluminum	93	6810	µg/L	MW-19D-091509BX	3/3	200 - 200	6810	NA	3700 NC	YES	ASL
	7440382	Arsenic	2.6	2.6	µg/L	MW-19D-120209AX	2/3	10 - 10	2.6	NA	0.045 C	YES	ASL
	7440393	Barium	5.4	68.1	µg/L	MW-20D-091709AX	3/3	NA	68.1	NA	730 NC	NO	BSL
	7440417	Beryllium	0.17	0.86	µg/L	MW-19D-120209AX	1/3	1 - 1	0.86	NA	7.3 NC	NO	BSL
	7440439	Cadmium	2.5	6.6	µg/L	MW-19D-091509BX	1/3	5 - 5	6.6	NA	1.8 NC	YES	ASL
	7440702	Calcium	25000	74100	µg/L	MW-19D-091509AX	3/3	NA	74100	NA	NA	NO	Essential Nutrient
	16887006	Chloride	370	470	µg/L	MW-14D-091809BX	3/3	NA	470	NA	NA	NO	NBA
	7440473	Chromium	0.7	132	µg/L	MW-14D-091809BX	3/3	NA	132	NA	0.043 C	YES	ASL

Table 4-5
Site Groundwater - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Site Groundwater
Exposure Medium: Site Groundwater

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Deep Bedrock Groundwater (cont.)	7440484	Cobalt	1.5	153	µg/L	MW-19D-091509BX	2/3	50 - 50	153	NA	1.1 NC	YES	ASL
	7440508	Copper	1.1	8910	µg/L	MW-19D-091509BX	3/3	25 - 25	8910	NA	150 NC	YES	ASL
	7439896	Iron	169	16948	µg/L	MW-19D-120209AX	3/3	NA	16948	NA	2600 NC	YES	ASL
	7439921	Lead	2.3	2.8	µg/L	MW-19D-091509BX	1/3	10 - 10	2.8	NA	15	NO	BSL
	7439954	Magnesium	2380	11500	µg/L	MW-19D-091509AX	3/3	NA	11500	NA	NA	NO	Essential Nutrient
	7439965	Manganese	23	1433	µg/L	MW-19D-120209AX	3/3	15 - 15	1433	NA	88 NC	YES	ASL
	7439987	Molybdenum	12	12	µg/L	MW-14D-091809BX	1/3	10 - 10	12	NA	18 NC	NO	BSL
	7440020	Nickel	1.2	87.4	µg/L	MW-19D-120209AX	3/3	40 - 40	87.4	NA	73 NC	YES	ASL
	14797558	Nitrogen, Nitrite + Nitrate	11	60	µg/L	MW-19D-091509BX	2/3	20 - 20	60	NA	5800 NC	NO	BSL
	7440097	Potassium	3639	7040	µg/L	MW-19D-091509AX	3/3	NA	7040	NA	NA	NO	Essential Nutrient
	7440235	Sodium	3108	5610	µg/L	MW-20D-091609AX	3/3	NA	5610	NA	NA	NO	Essential Nutrient
	7440246	Strontium	107	356	µg/L	MW-20D-091709AX	3/3	NA	356	NA	2200 NC	NO	BSL
	14808798	Sulfate	23000	220000	µg/L	MW-19D-091509BX	3/3	NA	220000	NA	NA	NO	NBA
	18496258	Sulfide	430	430	µg/L	MW-14D-091809BX	1/3	2000 - 2000	430	NA	NA	NO	NBA
	7440280	Thallium	1	1	µg/L	MW-19D-120209AX	1/3	1 - 1	1	NA	NA	NO	NBA
	7440622	Vanadium	0.77	5	µg/L	MW-19D-091509AD	3/3	50 - 50	5	NA	0.26 NC	YES	ASL
	7440666	Zinc	4.9	994	µg/L	MW-19D-091509BX	3/3	NA	994	NA	1100 NC	NO	BSL

Notes/sources:

(1) Maximum detected concentration used for screening.

(2) Maximum background value used for overburden and shallow bedrock. Background data not available foir deep bedrock.

(3) Risk-based tap water concentrations obtained from the Regional Screening Level (RSL) Table (May 2010).

(4) Hexavalent chromium RSL used for chromium.

(5) Although the maximum detected concentration of Aroclor 1248 exceeded its RSL, it was eliminated from consideration as a COPC in the HHRA. The detected sample result was considered suspect. PCBs were re-sampled in subsequent samples and were not detected. Further, the time period of mining operations pre-dates PCBs. For these reasons, PCBs were eliminated as COPCs.

C = cancer based screening value set at a target risk of 1E-06.

NA = not available.

NBA = no benchmark available.

NC = noncancer based screening value set at a target hazard quotient of 0.1.

ASL = above screening level.

BSL = below screening level.

Table 4-6
Residential Groundwater - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Residential Groundwater
Exposure Medium: Residential Groundwater

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)		COPC Flag (Y/N)	Rationale for Selection or Deletion
Residential Groundwater	7429905	Aluminum	46.8	79.3	µg/L	M11LT064-120709AX	2/6	200 - 200	79.3	NA	3700	NC	NO	BSL
	7440360	Antimony	0	0	µg/L		0/6	2 - 2	0	NA	1.5	NC	NO	BSL
	7440382	Arsenic	0	0	µg/L		0/6	10 - 10	0	NA	0.045	C	NO	BSL
	7440393	Barium	3.7	10.4	µg/L	M11LT63.0-120709AX	6/6	NA	10.4	NA	730	NC	NO	BSL
	7440417	Beryllium	0	0	µg/L		0/6	1 - 1	0	NA	7.3	NC	NO	BSL
	7440439	Cadmium	0	0	µg/L		0/6	5 - 5	0	NA	1.8	NC	NO	BSL
	7440702	Calcium	18686	33516	µg/L	M11LT60.0-120709AX	6/6	NA	33516	NA	NA		NO	Essential Nutrient
	7440473	Chromium	0	0	µg/L		0/6	10 - 10	0	NA	0.043	C	NO	BSL
	7440484	Cobalt	0	0	µg/L		0/6	50 - 50	0	NA	1.1	NC	NO	BSL
	7440508	Copper	2.4	192	µg/L	M11LT064-120709AX	6/6	NA	192	NA	150	NC	YES	ASL
	57125	Cyanide	3.2	3.8	µg/L	M11LT064-120709AX	2/5	10 - 10	3.8	NA	73	NC	NO	BSL
	7439896	Iron	62.9	452	µg/L	M11LT065041910AX	5/6	100 - 100	452	NA	2600	NC	NO	BSL
	7439921	Lead	2.5	11.3	µg/L	M11LT064-120709AX	3/6	10 - 10	11.3	NA	15		NO	BSL
	7439954	Magnesium	1110	6127	µg/L	M11LT60.0-120709AX	6/6	NA	6127	NA	NA		NO	Essential Nutrient
	7439965	Manganese	2.4	36.6	µg/L	M11LT065041910AX	4/6	15 - 15	36.6	NA	88	NC	NO	BSL
	7439976	Mercury	0	0	µg/L		0/6	0.2 - 0.2	0	NA	0.057	NC	NO	BSL
	7439987	Molybdenum	1.5	1.9	µg/L	M11LT55.1-120709AX	2/5	10 - 10	1.9	NA	18	NC	NO	BSL
	7440020	Nickel	0.87	1.1	µg/L	M11LT19.0-120809AX	2/6	40 - 40	1.1	NA	73	NC	NO	BSL
	7440097	Potassium	1300	3750	µg/L	M11LT60.0-120709AX	6/6	NA	3750	NA	NA		NO	Essential Nutrient
	7782492	Selenium	0	0	µg/L		0/6	35 - 35	0	NA	18	NC	NO	BSL
	7440224	Silver	0.51	0.63	µg/L	M11LT60.0-120709AX	2/6	10 - 10	0.63	NA	18	NC	NO	BSL
	7440235	Sodium	1450	6979	µg/L	M11LT60.0-120709AX	6/6	NA	6979	NA	NA		NO	Essential Nutrient
	7440246	Strontium	81.4	194	µg/L	M11LT60.0-120709AX	5/5	NA	194	NA	2200	NC	NO	BSL
	7440280	Thallium	0	0	µg/L		0/6	1 - 1	0	NA	NA		NO	NBA
	7440622	Vanadium	0.69	1.7	µg/L	M11LT55.1-120709AX	4/6	50 - 50	1.7	NA	0.26	NC	YES	ASL
	7440666	Zinc	9.6	153	µg/L	M11LT064-120709AX	5/6	60 - 60	153	NA	1100	NC	NO	BSL

Notes/sources:

(1) Maximum detected concentration used for screening.
(2) Background data not available.
(3) Risk-based tap water concentrations obtained from the Regional Screening Level (RSL) Table (May 2010).
(4) Hexavalent chromium RSL used for chromium.

C = cancer based screening value set at a target risk of 1E-06.
NA = not available.
NBA = no screening benchmark available.
NC = noncancer based screening value set at a target hazard quotient of 0.1.

ASL = above screening level.
BSL = below screening level.

Table 4-7
Fish - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Schoolhouse Brook Brook Trout	7429905	Aluminum	12.4	12.4	mg/kg	06Ely19F1	1/1	NA	12.4	6.9	140 NC	NO	BSL
	7440393	Barium	0.3	0.3	mg/kg	06Ely19F1	1/1	NA	0.3	0.48	27 NC	NO	BSL
	7440439	Cadmium	0.02	0.02	mg/kg	06Ely19F1	1/1	NA	0.02	0.08	0.14 NC	NO	BSL
	7440702	Calcium	2537	2537	mg/kg	06Ely19F1	1/1	NA	2537	5236.5	NA	NO	Essential Nutrient
	7440451	Cerium	0.1	0.1	mg/kg	06Ely19F1	1/1	NA	0.1	0.03	NA	NO	NBA
	7440462	Cesium	0.01	0.01	mg/kg	06Ely19F1	1/1	NA	0.01	0.03	NA	NO	NBA
	7440473	Chromium	0.3	0.3	mg/kg	06Ely19F1	1/1	NA	0.3	0.6	0.006 C	YES	ASL
	7440484	Cobalt	0.1	0.1	mg/kg	06Ely19F1	1/1	NA	0.1	0.06	0.041 NC	YES	ASL
	7440508	Copper	7.9	7.9	mg/kg	06Ely19F1	1/1	NA	7.9	1.9	5.4 NC	YES	ASL
	7429916	Dysprosium	0.008	0.008	mg/kg	06Ely19F1	1/1	NA	0.008	NA	NA	NO	NBA
	7440542	Gadolinium	0.011	0.011	mg/kg	06Ely19F1	1/1	NA	0.011	NA	NA	NO	NBA
	7440553	Gallium	0.02	0.02	mg/kg	06Ely19F1	1/1	NA	0.02	0.03	NA	NO	NBA
	7440564	Germanium	0	0	0	0	0/1	0.02 - 0.02	0	NA	NA	NO	NBA
	7440600	Holmium	0	0	0	0	0/1	0.001 - 0.001	0	NA	NA	NO	NBA
	7439896	Iron	46.9	46.9	mg/kg	06Ely19F1	1/1	NA	46.9	36	95 NC	NO	BSL
	7439910	Lanthanum	0.05	0.05	mg/kg	06Ely19F1	1/1	NA	0.05	0.02	NA	NO	NBA
	7439921	Lead	0.02	0.02	mg/kg	06Ely19F1	1/1	NA	0.02	0.06	NA	NO	NBA
	7439954	Magnesium	170.98	170.98	mg/kg	06Ely19F1	1/1	NA	170.98	310.83	NA	NO	Essential Nutrient
	7439965	Manganese	2.9	2.9	mg/kg	06Ely19F1	1/1	NA	2.9	3.68	19 NC	NO	BSL
	22967926	Mercury	0.003	0.003	mg/kg	06Ely19F1	1/1	NA	0.003	0.009	NA	NO	NBA
	7440008	Neodymium	0.05	0.05	mg/kg	06Ely19F1	1/1	NA	0.05	0.01	NA	NO	NBA
	7723140	Phosphorus	2739.87	2739.87	mg/kg	06Ely19F1	1/1	NA	2739.87	4844.46	NA	NO	NBA
	7440097	Potassium	2579.2	2579.2	mg/kg	06Ely19F1	1/1	NA	2579.2	4004.4	NA	NO	Essential Nutrient
	7440100	Praseodymium	0.01	0.01	mg/kg	06Ely19F1	1/1	NA	0.01	0.01	NA	NO	NBA
	7440177	Rubidium	3.37	3.37	mg/kg	06Ely19F1	1/1	NA	3.37	6.02	NA	NO	NBA

Table 4-7
Fish - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Schoolhouse Brook Brook Trout (cont.)	7440199	Samarium	0.01	0.01	mg/kg	06Ely19F1	1/1	NA	0.01	NA	NA	NO	NBA
	7782492	Selenium	0.3	0.3	mg/kg	06Ely19F1	1/1	NA	0.3	0.6	0.68 NC	NO	BSL
	99439288	Silica	104.9	104.9	mg/kg	06Ely19F1	1/1	NA	104.9	165.4	NA	NO	NBA
	7440235	Sodium	595.4	595.4	mg/kg	06Ely19F1	1/1	NA	595.4	870.9	NA	NO	Essential Nutrient
	7440246	Strontium	5.3	5.3	mg/kg	06Ely19F1	1/1	NA	5.3	7.8	81 NC	NO	BSL
	14808798	Sulfate	3082.4	3082.4	mg/kg	06Ely19F1	1/1	NA	3082.4	6608.6	NA	NO	NBA
	7440257	Tantalum	0.03	0.03	mg/kg	06Ely19F1	1/1	NA	0.03	0.37	NA	NO	NBA
	7440279	Terbium	0	0	0	0	0/1	0.001 - 0.001	0	NA	NA	NO	NBA
	7440280	Thallium	0	0	0	0	0/1	0.03 - 0.03	0	NA	NA	NO	NBA
	7440291	Thorium	0	0	0	0	0/1	0.1 - 0.1	0	NA	NA	NO	NBA
	7440304	Thulium	0	0	0	0	0/1	0.001 - 0.001	0	NA	NA	NO	NBA
	7440326	Titanium	0.4	0.4	mg/kg	06Ely19F1	1/1	NA	0.4	0.5	NA	NO	NBA
	7440655	Yttrium	0.03	0.03	mg/kg	06Ely19F1	1/1	NA	0.03	0.01	NA	NO	NBA
	7440666	Zinc	18.8	18.8	mg/kg	06Ely19F1	1/1	NA	18.8	23.2	41 NC	NO	BSL
Schoolhouse Brook Blacknose Dace	7429905	Aluminum	1.3	11.5	mg/kg	06Ely08F2	8/8	NA	11.5	26.5	140 NC	NO	BSL
	7440360	Antimony	0.1	0.4	mg/kg	06Ely07F3	4/8	0.1 - 0.1	0.4	NA	0.054 NC	YES	ASL
	7440393	Barium	1.44	2.31	mg/kg	06Ely19F2	8/8	NA	2.31	1.13	27 NC	NO	BSL
	7440439	Cadmium	0.03	0.07	mg/kg	06Ely07F1	8/8	NA	0.07	0.06	0.14 NC	NO	BSL
	7440702	Calcium	6193.5	7444.6	mg/kg	06Ely07F2	8/8	NA	7444.6	6847.3	NA	NO	Essential Nutrient
	7440451	Cerium	0.01	0.04	mg/kg	06Ely08F1	8/8	NA	0.04	0.09	NA	NO	NBA
	7440462	Cesium	0.02	0.03	mg/kg	06Ely07F2	8/8	NA	0.03	0.05	NA	NO	NBA
	7440473	Chromium	0.3	0.5	mg/kg	06Ely06F1	8/8	NA	0.5	0.5	0.006 C	YES	ASL
	7440484	Cobalt	0.02	0.11	mg/kg	06Ely08F2	8/8	NA	0.11	0.04	0.041 NC	YES	ASL
	7440508	Copper	1.6	5.9	mg/kg	06Ely08F3	8/8	NA	5.9	1	5.4 NC	YES	ASL
	7440542	Gadolinium	0.005	0.005	mg/kg	06Ely08F2	1/8	0.001 - 0.001	0.005	NA	NA	NO	NBA

Table 4-7
Fish - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Schoolhouse Brook Blacknose Dace (cont.)	7440553	Gallium	0.03	0.05	mg/kg	06Ely08F1	8/8	NA	0.05	0.05	NA	NO	NBA
	7439896	Iron	26.6	44.7	mg/kg	06Ely08F2	8/8	NA	44.7	60.5	95 NC	NO	BSL
	7439910	Lanthanum	0.01	0.02	mg/kg	06Ely08F1	7/8	0.01 - 0.01	0.02	0.04	NA	NO	NBA
	7439921	Lead	0.01	1.17	mg/kg	06Ely06F1	8/8	NA	1.17	0.05	NA	NO	NBA
	7439954	Magnesium	208.71	330.08	mg/kg	06Ely08F1	8/8	NA	330.08	266.71	NA	NO	Essential Nutrient
	7439965	Manganese	2.63	4.23	mg/kg	06Ely07F2	8/8	NA	4.23	6.1	19 NC	NO	BSL
	22967926	Mercury	0.008	0.021	mg/kg	06Ely19F2	8/8	NA	0.021	0.018	NA	NO	NBA
	7440008	Neodymium	0.01	0.02	mg/kg	06Ely08F1	6/8	0.01 - 0.01	0.02	0.04	NA	NO	NBA
	7440020	Nickel	0.1	0.2	mg/kg	06Ely07F1	8/8	NA	0.2	0.2	2.7 NC	NO	BSL
	7440031	Niobium	0.1	0.2	mg/kg	06Ely07F3	4/8	0.1 - 0.1	0.2	0.1	NA	NO	NBA
	7723140	Phosphorus	4202.08	5114.58	mg/kg	06Ely07F2	8/8	NA	5114.58	4846.94	NA	NO	NBA
	7440097	Potassium	1694.7	2496.3	mg/kg	06Ely07F3	8/8	NA	2496.3	2487.6	NA	NO	Essential Nutrient
	7440100	Praseodymium	0.01	0.01	mg/kg	06Ely08F1	2/8	0.01 - 0.01	0.01	0.01	NA	NO	NBA
	7440177	Rubidium	2.45	3.34	mg/kg	06Ely07F1	8/8	NA	3.34	5.44	NA	NO	NBA
	7782492	Selenium	0.4	0.5	mg/kg	06Ely06F1	8/8	NA	0.5	0.7	0.68 NC	NO	BSL
	99439288	Silica	92.1	137.1	mg/kg	06Ely08F2	8/8	NA	137.1	172.2	NA	NO	NBA
	7440235	Sodium	425.8	624.1	mg/kg	06Ely08F1	8/8	NA	624.1	582.1	NA	NO	Essential Nutrient
	7440246	Strontium	9.9	13.2	mg/kg	06Ely08F3	8/8	NA	13.2	11.4	81 NC	NO	BSL
	14808798	Sulfate	2782.8	4577.6	mg/kg	06Ely07F3	8/8	NA	4577.6	5129	NA	NO	NBA
	7440257	Tantalum	0.01	0.11	mg/kg	06Ely07F3	8/8	NA	0.11	0.19	NA	NO	NBA
	7440326	Titanium	0.1	0.8	mg/kg	06Ely08F2	8/8	NA	0.8	1.6	NA	NO	NBA
	7440622	Vanadium	0.1	0.1	mg/kg	06Ely06F1	2/8	0.2 - 0.2	0.1	0.2	0.68 NC	NO	BSL
	7440655	Yttrium	0.01	0.01	mg/kg	06Ely07F1	5/8	0.01 - 0.01	0.01	0.03	NA	NO	NBA
	7440666	Zinc	33	40.9	mg/kg	06Ely07F2	8/8	NA	40.9	33.9	41 NC	NO	BSL
	7440677	Zirconium	0.1	0.1	mg/kg	06Ely07F3	2/8	0.1 - 0.1	0.1	NA	NA	NO	NBA

Table 4-7
Fish - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Ompompanoosuc River Brook Trout	7429905	Aluminum	1.2	3.4	mg/kg	06Ely10F1	2/2	NA	3.4	NA	140 NC	NO	BSL
	7440393	Barium	0.44	0.51	mg/kg	06Ely10F1	2/2	NA	0.51	NA	27 NC	NO	BSL
	7440439	Cadmium	0.01	0.03	mg/kg	06Ely10F1	2/2	NA	0.03	NA	0.14 NC	NO	BSL
	7440702	Calcium	3668.8	4871.9	mg/kg	06Ely10F1	2/2	NA	4871.9	NA	NA	NO	Essential Nutrient
	7440451	Cerium	0.01	0.01	mg/kg	06Ely10F1	1/1	NA	0.01	NA	NA	NO	NBA
	7440462	Cesium	0.01	0.02	mg/kg	06Ely10F1	2/2	NA	0.02	NA	NA	NO	NBA
	7440473	Chromium	0.3	0.3	mg/kg	06Ely10F1	1/2	0.3 - 0.3	0.3	NA	0.006 C	YES	ASL
	7440484	Cobalt	0.02	0.06	mg/kg	06Ely10F2	2/2	NA	0.06	NA	0.041 NC	YES	ASL
	7440508	Copper	0.8	1.3	mg/kg	06Ely10F1	2/2	NA	1.3	NA	5.4 NC	NO	BSL
	7440553	Gallium	0.02	0.03	mg/kg	06Ely10F2	2/2	NA	0.03	NA	NA	NO	NBA
	7439896	Iron	21	24.6	mg/kg	06Ely10F1	2/2	NA	24.6	NA	95 NC	NO	BSL
	7439910	Lanthanum	0.01	0.01	mg/kg	06Ely10F1	1/2	0.01 - 0.01	0.01	NA	NA	NO	NBA
	7439921	Lead	0.01	0.01	mg/kg	06Ely10F1	1/2	0.01 - 0.01	0.01	NA	NA	NO	NBA
	7439954	Magnesium	212.72	214.81	mg/kg	06Ely10F2	2/2	NA	214.81	NA	NA	NO	Essential Nutrient
	7439965	Manganese	2.02	3.11	mg/kg	06Ely10F1	2/2	NA	3.11	NA	19 NC	NO	BSL
	22967926	Mercury	0.005	0.006	mg/kg	06Ely10F1	2/2	NA	0.006	NA	NA	NO	NBA
	7440020	Nickel	0.1	0.1	mg/kg	06Ely10F1	1/2	0.1 - 0.1	0.1	NA	2.7 NC	NO	BSL
	7440031	Niobium	0.1	0.1	mg/kg	06Ely10F1	1/2	0.1 - 0.1	0.1	NA	NA	NO	NBA
	7723140	Phosphorus	3323.18	3762.44	mg/kg	06Ely10F1	2/2	NA	3762.44	NA	NA	NO	NBA
	7440097	Potassium	2312.9	2639.9	mg/kg	06Ely10F2	2/2	NA	2639.9	NA	NA	NO	Essential Nutrient
	7440177	Rubidium	3.7	4.75	mg/kg	06Ely10F1	2/2	NA	4.75	NA	NA	NO	NBA
	7782492	Selenium	0.3	0.3	mg/kg	06Ely10F1	2/2	NA	0.3	NA	0.68 NC	NO	BSL
	7782492	Selenium	0.3	0.3	mg/kg	06Ely10F1	2/2	NA	0.3	NA	0.68 NC	NO	BSL
	99439288	Silica	69.4	91.4	mg/kg	06Ely10F1	2/2	NA	91.4	NA	NA	NO	NBA
	7440235	Sodium	554.7	561	mg/kg	06Ely10F2	2/2	NA	561	NA	NA	NO	Essential Nutrient

Table 4-7
Fish - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Ompompanoosuc River Brook Trout (cont.)	7440246	Strontium	7	9	mg/kg	06Ely10F1	2/2	NA	9	NA	81 NC	NO	BSL
	14808798	Sulfate	3615.6	3738.3	mg/kg	06Ely10F1	2/2	NA	3738.3	NA	NA	NO	NBA
	7440257	Tantalum	0.03	0.06	mg/kg	06Ely10F1	2/2	NA	0.06	NA	NA	NO	NBA
	7440326	Titanium	0.2	0.3	mg/kg	06Ely10F1	2/2	NA	0.3	NA	NA	NO	NBA
	7440666	Zinc	16.6	18.2	mg/kg	06Ely10F2	2/2	NA	18.2	NA	41 NC	NO	BSL
Ompompanoosuc River Blacknose Dace	7429905	Aluminum	3.8	16.8	mg/kg	06Ely20F1	6/6	NA	16.8	8.9	140 NC	NO	BSL
	7440393	Barium	1.43	2.39	mg/kg	06Ely10F4	6/6	NA	2.39	2.32	27 NC	NO	BSL
	7440439	Cadmium	0.03	0.07	mg/kg	06Ely20F2	6/6	NA	0.07	0.04	0.14 NC	NO	BSL
	7440702	Calcium	5567.6	6821.8	mg/kg	06Ely20F3	6/6	NA	6821.8	7199.7	NA	NO	Essential Nutrient
	7440451	Cerium	0.01	0.06	mg/kg	06Ely20F1	6/6	NA	0.06	0.02	NA	NO	NBA
	7440462	Cesium	0.02	0.03	mg/kg	06Ely10F3	6/6	NA	0.03	0.04	NA	NO	NBA
	7440473	Chromium	0.2	0.3	mg/kg	06Ely10F3	6/6	NA	0.3	0.4	0.006 C	YES	ASL
	7440484	Cobalt	0.06	0.09	mg/kg	06Ely10F5	6/6	NA	0.09	0.02	0.041 NC	YES	ASL
	7440508	Copper	1.8	3.5	mg/kg	06Ely20F1	6/6	NA	3.5	2.1	5.4 NC	NO	BSL
	7440553	Gallium	0.02	0.05	mg/kg	06Ely10F3	6/6	NA	0.05	0.05	NA	NO	NBA
	7439896	Iron	28.2	50.8	mg/kg	06Ely20F1	6/6	NA	50.8	33.5	95 NC	NO	BSL
	7439910	Lanthanum	0.01	0.04	mg/kg	06Ely20F1	6/6	NA	0.04	0.01	NA	NO	NBA
	7439921	Lead	0.02	0.04	mg/kg	06Ely10F5	6/6	NA	0.04	0.03	NA	NO	NBA
	7439954	Magnesium	211.4	255.29	mg/kg	06Ely20F1	6/6	NA	255.29	248.36	NA	NO	Essential Nutrient
	7439965	Manganese	3.96	6.43	mg/kg	06Ely20F1	6/6	NA	6.43	5.49	19 NC	NO	BSL
	22967926	Mercury	0.012	0.022	mg/kg	06Ely20F2	6/6	NA	0.022	0.026	NA	NO	NBA
	7440008	Neodymium	0.01	0.03	mg/kg	06Ely20F1	5/6	0.01 - 0.01	0.03	0.01	NA	NO	NBA
	7440020	Nickel	0.1	0.2	mg/kg	06Ely10F3	6/6	NA	0.2	0.2	2.7 NC	NO	BSL
	7440031	Niobium	0.1	0.1	mg/kg	06Ely10F3	2/6	0.1 - 0.1	0.1	0.1	NA	NO	NBA
	7723140	Phosphorus	3763.06	4539.78	mg/kg	06Ely20F3	6/6	NA	4539.78	4868.86	NA	NO	NBA
	7440097	Potassium	1974	2231	mg/kg	06Ely20F3	6/6	NA	2231	2462.9	NA	NO	Essential Nutrient

Table 4-7
Fish - Occurrence, Distribution, and Selection of Contaminants of Potential Concern
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish

Exposure Point	CAS Number	Contaminant	Minimum Concentration	Maximum Concentration	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening (1)	Background Value (2)	Screening Toxicity Value (N/C) (3)	COPC Flag (Y/N)	Rationale for Selection or Deletion
Ompompanoosuc River Blacknose Dace (cont.)	7440100	Praseodymium	0.01	0.01	mg/kg	06Ely10F5	2/6	0.01 - 0.01	0.01	NA	NA	NO	NBA
	7440177	Rubidium	2.82	4.13	mg/kg	06Ely20F2	6/6	NA	4.13	4.43	NA	NO	NBA
	7440199	Samarium	0.01	0.01	mg/kg	06Ely20F1	1/6	0.01 - 0.01	0.01	NA	NA	NO	NBA
	7782492	Selenium	0.3	0.5	mg/kg	06Ely20F3	6/6	NA	0.5	0.4	0.68 NC	NO	BSL
	99439288	Silica	73.4	127.4	mg/kg	06Ely20F1	6/6	NA	127.4	112.9	NA	NO	NBA
	7440235	Sodium	492.9	560.8	mg/kg	06Ely20F3	6/6	NA	560.8	600.8	NA	NO	Essential Nutrient
	7440246	Strontium	10.2	12.3	mg/kg	06Ely20F3	6/6	NA	12.3	12	81 NC	NO	BSL
	14808798	Sulfate	3080.9	3690.1	mg/kg	06Ely20F3	6/6	NA	3690.1	4299.1	NA	NO	NBA
	7440257	Tantalum	0.02	0.06	mg/kg	06Ely10F3	6/6	NA	0.06	0.09	NA	NO	NBA
	7440326	Titanium	0.4	1.1	mg/kg	06Ely20F1	6/6	NA	1.1	0.5	NA	NO	NBA
	7440655	Yttrium	0.01	0.02	mg/kg	06Ely20F1	4/6	0.01 - 0.01	0.02	0.01	NA	NO	NBA
	7440666	Zinc	33.2	41.6	mg/kg	06Ely20F1	6/6	NA	41.6	42.5	41 NC	YES	ASL

Notes/sources:
(1) Maximum detected concentration used for screening.
(2) Site-specific maximum background value. Background data not available for Ompompanoosuc River trout.
(3) Risk-based fish concentrations obtained from the Regional Screening Level (RSL) Table (May 2010).

C = cancer based screening value set at a target risk of 1E-06.
NA = not available.
NC = noncancer based screening value set at a target hazard quotient of 0.1.
ASL = above screening level.
BSL = below screening level.
NBA = no benchmark available.

Table 6-1
Non-Cancer Toxicity Data - Oral/Dermal
Ely Copper Mine Superfund Site
Vershire, Vermont

Contaminant of Potential Concern	Chronic/ Subchronic	Oral RfD		Oral Absorption Efficiency for Dermal (1)	Absorbed RfD for Dermal (1)		Primary Target Organ(s)	Combined Uncertainty/Modifying Factors	RfD: Target Organ(s)	
		Value	Units		Value	Units			Source(s)	Dates (2)
Bis(2-ethylhexyl)phthalate	Chronic	2.00E-02	(mg/kg-day)	1.0	2.00E-02	(mg/kg-day)	Liver	1,000	IRIS	9/29/2010
Aluminum	Chronic	1.00E+00	(mg/kg-day)	1.0	1.00E+00	(mg/kg-day)	Nervous system	100	PPRTV	10/23/2006
Antimony	Chronic	4.00E-04	(mg/kg-day)	0.15	6.00E-05	(mg/kg-day)	Blood	1,000	IRIS	3/3/2010
Arsenic	Chronic	3.00E-04	(mg/kg-day)	1.0	3.00E-04	(mg/kg-day)	Skin	3	IRIS	3/3/2010
Barium	Chronic	2.00E-01	(mg/kg-day)	0.07	1.40E-02	(mg/kg-day)	Kidney	300	IRIS	3/3/2010
Beryllium	Chronic	2.00E-03	(mg/kg-day)	0.007	1.40E-05	(mg/kg-day)	Gastrointestinal	300	IRIS	9/29/2010
Cadmium-diet	Chronic	1.00E-03	(mg/kg-day)	0.025	2.50E-05	(mg/kg-day)	Kidney	10	IRIS	3/3/2010
Cadmium-water	Chronic	5.00E-04	(mg/kg-day)	0.05	2.50E-05	(mg/kg-day)	Kidney	10	IRIS	3/3/2010
Chromium, Total (3)	Chronic	1.50E+00	(mg/kg-day)	0.013	1.95E-02	(mg/kg-day)	None observed	900	IRIS	3/3/2010
Cobalt	Chronic	3.00E-04	(mg/kg-day)	1.0	3.00E-04	(mg/kg-day)	Thyroid	3,000	PPRTV	8/25/2008
Copper	Chronic	4.00E-02	(mg/kg-day)	1.0	4.00E-02	(mg/kg-day)	Gastrointestinal	---	HEAST	1997
Iron	Chronic	7.00E-01	(mg/kg-day)	1.0	7.00E-01	(mg/kg-day)	Gastrointestinal	1.5	PPRTV	9/11/2006
Lead	---	NA	---	---	NA	---	---	---	---	---
Lithium	Chronic	2.00E-03	(mg/kg-day)	1.0	2.00E-03	(mg/kg-day)	No information	No information	PPRTV	May 2010 RSL Table
Manganese-diet	Chronic	7.00E-02	(mg/kg-day)	1.0	7.00E-02	(mg/kg-day)	Nervous system	1	EPA Region I	Risk Update #5, 1999
Manganese-water	Chronic	2.40E-02	(mg/kg-day)	0.040	9.60E-04	(mg/kg-day)	Nervous system	3	IRIS	3/3/2010
Mercury	Chronic	3.00E-04	(mg/kg-day)	0.07	2.10E-05	(mg/kg-day)	Nervous system	1,000	IRIS	3/3/2010
Molybdenum	Chronic	5.00E-03	(mg/kg-day)	1.00	5.00E-03	(mg/kg-day)	Increased uric acid	30	IRIS	9/29/2010
Nickel	Chronic	2.00E-02	(mg/kg-day)	0.04	8.00E-04	(mg/kg-day)	Body weight	300	IRIS	3/3/2010
Selenium	Chronic	5.00E-03	(mg/kg-day)	1.00	5.00E-03	(mg/kg-day)	Selenosis	3	IRIS	9/29/2010
Vanadium sulfate	Chronic	2.00E-02	(mg/kg-day)	0.026	5.20E-04	(mg/kg-day)	None reported	100	HEAST	1997
Zinc	Chronic	3.00E-01	(mg/kg-day)	1.0	3.00E-01	(mg/kg-day)	Blood	3	IRIS	3/3/2010

Notes:

(1) Source: RAGS Part E Guidance

(2) Represents date source was searched.

(3) Chromium III.

Definitions: IRIS=Integrated Risk Information Sys NA=Not available
HEAST = Health Effects Assessment Summary Table
OEHHA=California Office of Environmental Health Hazard Assessment
PPRTV = Provisional Peer-Reviewed Toxicity Value

Table 6-2
Cancer Toxicity Data - Oral/Dermal
Ely Copper Mine Superfund Site
Vershire, Vermont

Contaminant of Potential Concern	Oral Cancer Slope Factor		Oral Absorption Efficiency for Dermal (1)	Absorbed Cancer Slope Factor for Dermal (1)		Weight of Evidence/ Cancer Guideline Description	Oral CSF	
	Value	Units		Value	Units		Source(s)	Dates (2)
Bis(2-ethylhexyl)phthalate	1.40E-02	$(mg/kg\text{-}day)^{-1}$	1.0	1.40E-02	$(mg/kg\text{-}day)^{-1}$	B2	IRIS	9/29/2010
Aluminum	NA	---	---	NA	---	No information	---	---
Antimony	NA	---	---	NA	---	No information	---	---
Arsenic	1.50E+00	$(mg/kg\text{-}day)^{-1}$	1.0	1.50E+00	$(mg/kg\text{-}day)^{-1}$	A	IRIS	3/3/2010
Barium	NA	---	---	NA	---	D	IRIS	3/3/2010
Beryllium	NA	---	---	NA	---	Cannot be determined	IRIS	9/29/2010
Cadmium	NA	---	---	NA	---	B1	IRIS	3/3/2010
Chromium, Total (3)	NA	---	---	NA	---	D	IRIS	11/10/2010
Cobalt	NA	---	---	NA	---	No information	---	---
Copper	NA	---	---	NA	---	D	IRIS	9/29/2010
Iron	NA	---	---	NA	---	No information	---	---
Lead	NA	---	---	NA	---	B2	IRIS	3/3/2010
Lithium	NA	---	---	NA	---	No information	---	---
Manganese	NA	---	---	NA	---	D	IRIS	3/3/2010
Mercury	NA	---	---	NA	---	D	IRIS	3/3/2010
Molybdenum	NA	---	---	NA	---	Not assessed	IRIS	9/29/2010
Nickel	NA	---	---	NA	---	No information	---	---
Selenium	NA	---	---	NA	---	D	IRIS	9/29/2010
Vanadium	NA	---	---	NA	---	No information	---	---
Zinc	NA	---	---	NA	---	D	IRIS	3/3/2010

Notes:

(1) Source: RAGS Part E Guidance

(2) Represents date source was searched.

(3) Chromium III.

Definitions: IRIS = Integrated Risk Information System

NJDEP=New Jersey Department of Environmental Protection

NA = Not available.

A - Human carcinogen.

B1 - Probable human carcinogen - indicates that limited human data are available.

B2 - Probable human carcinogen - indicates sufficient evidence in animals and inadequate or no evidence in humans.

C - Possible human carcinogen.

D - Not classifiable as a human carcinogen.

Table 6-3
Non-Cancer Toxicity Data - Inhalation
Ely Copper Mine Superfund Site
Vershire, Vermont

Contaminant of Potential Concern	Chronic/ Subchronic	Inhalation RfC		Primary Target Organ(s)	Combined Uncertainty/Modifying Factors	RfC: Target Organ(s)	
		Value	Units			Source(s)	Dates (1)
Bis(2-ethylhexyl)phthalate	---	NA	---	---	---	---	---
Aluminum	Chronic	5.00E-03	mg/m^3	Nervous system	300	PPRTV	10/23/2006
Antimony	---	NA	---	---	---	---	---
Arsenic	Chronic	1.50E-05	mg/m^3	Developmental, Cardiovascular system, Nervous system, Lung, Skin	30	CalEPA-OEHHA	May 2010 RSL Table
Barium	Chronic	5.00E-04	mg/m^3	Developmental	1,000	HEAST	July, 1997
Beryllium	Chronic	2.00E-05	mg/m^3	Respiratory tract	10	IRIS	9/29/2010
Cadmium	Chronic	1.00E-05	mg/m^3	Kidney	9	ATSDR	12/1/2009
Chromium, Total (2)	---	NA	---	---	---	---	---
Cobalt	Chronic	6.00E-06	mg/m^3	Respiratory tract	300	PPRTV	8/25/2008
Copper	---	NA	---	---	---	---	---
Iron	---	NA	---	---	---	---	---
Lead	---	NA	---	---	---	---	---
Lithium	---	NA	---	---	---	---	---
Manganese	Chronic	5.00E-05	mg/m^3	Nervous system	1,000	IRIS	3/3/2010
Mercury	Chronic	3.00E-04	mg/m^3	Nervous system	30	IRIS	3/3/2010
Molybdenum	---	NA	---	---	---	---	---
Nickel	Chronic	9.00E-05	mg/m^3	Respiratory tract	30	ATSDR	12/1/2009
Selenium	Chronic	2.00E-02	mg/m^3	No information	No information	CalEPA-OEHHA	May 2010 RSL Table
Vanadium sulfate	---	NA	---	---	---	---	---
Zinc	Chronic	NA	---	---	---	---	---

Notes:

(1) Represents date source was searched.

(2) Chromium III.

Definitions: ATSDR=Agency for Toxic Substances and Disease Registry
CalEPA=California Environmental Protection Agency
HEAST=Health Effects Assessment Summary Tables (EPA, 1997b)
IRIS=Integrated Risk Information System
NA=Not available
PPRTV = Provisional Peer-Reviewed Toxicity Value (EPA, various dates)
OEHHA=California Office of Environmental Health Hazard Assessment

Table 6-4
Cancer Toxicity Data - Inhalation
Ely Copper Mine Superfund Site
Vershire, Vermont

Contaminant of Potential Concern	Unit Risk		Weight of Evidence/ Cancer Guideline Description	Unit Risk: Inhalation CSF	
	Value	Units		Source(s)	Dates (1)
Bis(2-ethylhexyl)phthalate	2.40E-06	$(\mu g/m^3)^{-1}$	No information	CalEPA-OEHHA	May 2010 RSL Table
Aluminum	NA	---	No information	---	---
Antimony	NA	---	No information	---	---
Arsenic	4.30E-03	$(\mu g/m^3)^{-1}$	A	IRIS	3/3/2010
Barium	NA	---	D	IRIS	3/3/2010
Beryllium	2.40E-03	$(\mu g/m^3)^{-1}$	B1	IRIS	9/29/2010
Cadmium	1.80E-03	$(\mu g/m^3)^{-1}$	B1	IRIS	3/3/2010
Chromium, Total (2)	NA	---	D	IRIS	11/10/2010
Cobalt	9.00E-03	$(\mu g/m^3)^{-1}$	No information	PPRTV	8/25/2008
Copper	NA	---	D	IRIS	9/29/2010
Iron	NA	---	No information	---	---
Lead	NA	---	B2	IRIS	3/3/2010
Lithium	NA	---	No information	---	---
Manganese	NA	---	D	IRIS	3/3/2010
Mercury	NA	---	D	IRIS	3/3/2010
Molybdenum	NA	---	Not assessed	IRIS	9/29/2010
Nickel	2.60E-04	$(\mu g/m^3)^{-1}$	No information	CalEPA-OEHHA	3/3/2010
Selenium	NA	---	D	IRIS	3/3/2010
Vanadium	NA	---	No information	---	---
Zinc	NA	---	D	IRIS	3/3/2010

Notes:

(1) Represents date source was searched.

(2) Chromium III.

Definitions: CalEPA=California Environmental Protection Agency
IRIS = Integrated Risk Information System.
OEHHA=California Office of Environmental Health Hazard Assessment
NA = Not available.
PPRTV = Provisional Peer-Reviewed Toxicity Value
A - Human carcinogen.
B1 - Probable human carcinogen - indicates that limited human data are available.
B2 - Probable human carcinogen - indicates sufficient evidence in animals and inadequate or no evidence in humans.
C - Possible human carcinogen.
D - Not classifiable as a human carcinogen.

Table 7-1
Soil Exposure Parameters
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Soil

Exposure Point	Receptor Population	Exposure Route	Receptor Age	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CTE Value	CTE Rationale/ Reference	Intake Equation/ Model Name
Soil	Recreational Visitor	Ingestion	Adolescent (10 to 18 years)	EPC	Exposure Point Concentration	mg/kg	Calculated	See Table 7-6	Calculated	See Table 7-6	Chronic daily intake (mg/kg-day) = EPC x IRS x FI x EF x ED x CF x 1/BW x 1/AT
				IRS	Ingestion Rate of Soil	mg/day	100	EPA, 2002a	100	EPA, 2002a	
				FI	Fraction Ingested	unitless	1	Professional judgment	0.5	Professional judgment	
				EF	Exposure Frequency	days/year	104	Professional judgment	35	Professional judgment	
				ED	Exposure Duration	years	8	Estimated	8	Estimated	
				CF	Conversion factor	kg/mg	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	52	Tables 7-6 and 7-7 of EPA, 1997a	52	Tables 7-6 and 7-7 of EPA, 1997a	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	2,920	Calculated (ED x 365 days/year)	2,920	Calculated (ED x 365 days/year)	
			Adult	EPC	Exposure Point Concentration	mg/kg	Calculated	See Table 7-6	Calculated	See Table 7-6	Chronic daily intake (mg/kg-day) = EPC x IRS x FI x EF x ED x CF x 1/BW x 1/AT
				IRS	Ingestion Rate of Soil	mg/day	100	EPA, 2002a	100	EPA, 2002a	
				FI	Fraction Ingested	unitless	1	Professional judgment	0.5	Professional judgment	
				EF	Exposure Frequency	days/year	104	Professional judgment	35	Professional judgment	
				ED	Exposure Duration	years	24	Professional judgment	24	Professional judgment	
				CF	Conversion factor	kg/mg	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	70	EPA, 2002a	70	EPA, 2002a	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	8,760	Calculated (ED x 365 days/year)	8,760	Calculated (ED x 365 days/year)	
		Dermal Contact	Adolescent (10 to 18 years)	EPC	Exposure Point Concentration	mg/kg	Calculated	See Table 7-6	Calculated	See Table 7-6	Dermally Absorbed Dose (mg/kg-day) = EPC x SA x AF x ABS x EF x ED x CF x 1/BW x 1/AT
				SA	Exposed Skin Surface Area	cm^2/day	5,900	EPA, 2004	3,600	EPA, 2004	
				AF	Soil to Skin Adherence Factor	mg/cm^2	0.2	EPA, 2004	0.1	EPA, 2004	
				ABS	Dermal Absorption Factor	unitless	COPC specific	EPA, 2004, see Table 7-5	COPC specific	EPA, 2004, see Table 7-5	
				EF	Exposure Frequency	days/year	104	Professional judgment	35	Professional judgment	
				ED	Exposure Duration	years	8	Estimated	8	Estimated	
				CF	Conversion factor	kg/mg	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	52	Tables 7-6 and 7-7 of EPA, 1997a	52	Tables 7-6 and 7-7 of EPA, 1997a	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	2,920	Calculated (ED x 365 days/year)	2,920	Calculated (ED x 365 days/year)	
			Adult	EPC	Exposure Point Concentration	mg/kg	Calculated	See Table 7-6	Calculated	See Table 7-6	Dermally Absorbed Dose (mg/kg-day) = EPC x SA x AF x ABS x EF x ED x CF x 1/BW x 1/AT
				SA	Exposed Skin Surface Area	cm^2/day	6,900	EPA, 2004	4,500	EPA, 2004	
				AF	Soil to Skin Adherence Factor	mg/cm^2	0.2	EPA, 2004	0.1	EPA, 2004	
				ABS	Dermal Absorption Factor	unitless	COPC specific	EPA, 2004, see Table 7-5	COPC specific	EPA, 2004, see Table 7-5	
				EF	Exposure Frequency	days/year	104	Professional judgment	35	Professional judgment	
				ED	Exposure Duration	years	24	Professional judgment	24	Professional judgment	
				CF	Conversion factor	kg/mg	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	70	EPA, 2002a	70	EPA, 2002a	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	8,760	Calculated (ED x 365 days/year)	8,760	Calculated (ED x 365 days/year)	

Table 7-1
Soil Exposure Parameters
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Soil

Exposure Point	Receptor Population	Exposure Route	Receptor Age	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CTE Value	CTE Rationale/ Reference	Intake Equation/ Model Name
Soil (cont.)	Recreational Visitor (cont.)	Inhalation	Adolescent (10 to 18 years)	EPC	Exposure Point Concentration	mg/kg	Calculated	See Table 7-6	Calculated	See Table 7-6	EC ($\mu g/m^3$) = (CA x ET x EF x ED x CF2)/AT where: CA ($\mu g/m^3$) = (EPC/PEF) x CF3
				EC	Exposure Concentration	$\mu g/m^3$	Calculated	EPA, 2009a	Calculated	EPA, 2009a	
				CA	Chemical Concentration in Air	$\mu g/m^3$	Calculated	EPA, 2009a	Calculated	EPA, 2009a	
				ET	Exposure Time	hours/day	2	Professional judgment	1	Professional judgment	
				EF	Exposure Frequency	days/year	104	Professional judgment	35	Professional judgment	
				ED	Exposure Duration	years	8	Estimated	8	Estimated	
				CF2	Conversion Factor 2	days/hour	0.042	Units conversion	0.042	Units conversion	
				CF3	Conversion Factor 3	µg/mg	1000	Units conversion	1000	Units conversion	
				BW	Body Weight	kg	52	Tables 7-6 and 7-7 of EPA, 1997a	52	Tables 7-6 and 7-7 of EPA, 1997a	
				PEF	Particulate Emission Factor	m^3/kg	2.0E+06	See Appendix C	1.9E+06	See Appendix C	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	2,920	Calculated (ED x 365 days/year)	2,920	Calculated (ED x 365 days/year)	
			Adult	EPC	Exposure Point Concentration	mg/kg	Calculated	See Table 7-6	Calculated	See Table 7-6	EC ($\mu g/m^3$) = (CA x ET x EF x ED x CF2)/AT where: CA ($\mu g/m^3$) = (EPC/PEF) x CF3
				EC	Exposure Concentration	$\mu g/m^3$	Calculated	EPA, 2009a	Calculated	EPA, 2009a	
				CA	Chemical Concentration in Air	$\mu g/m^3$	Calculated	EPA, 2009a	Calculated	EPA, 2009a	
				ET	Exposure Time	hours/day	2	Professional judgment	1	Professional judgment	
				EF	Exposure Frequency	days/year	104	Professional judgment	35	Professional judgment	
				ED	Exposure Duration	years	24	Professional judgment	24	Professional judgment	
				CF2	Conversion Factor 2	days/hour	0.042	Units conversion	0.042	Units conversion	
				CF3	Conversion Factor 3	µg/mg	1000	Units conversion	1000	Units conversion	
				BW	Body Weight	kg	70	EPA, 2002a	70	EPA, 2002a	
				PEF	Particulate Emission Factor	m^3/kg	2.0E+06	See Appendix C	2.0E+06	See Appendix C	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	8,760	Calculated (ED x 365 days/year)	8,760	Calculated (ED x 365 days/year)	
	Resident	Ingestion	Child (1 to 6 years)	EPC	Exposure Point Concentration	mg/kg	Calculated	See Tables 7-6, 7-7	Calculated	See Tables 7-6, 7-7	Chronic daily intake (mg/kg-day) = EPC x IRS x FI x EF x ED x CF x 1/BW x 1/AT
				IRS	Ingestion Rate of Soil	mg/day	200	EPA, 2002b	200	EPA, 2002b	
				FI	Fraction Ingested	unitless	1	Professional judgment	0.5	Professional judgment	
				EF	Exposure Frequency	days/year	350	EPA, 2002a	350	EPA, 2002a	
				ED	Exposure Duration	years	6	EPA, 2002a	6	EPA, 2002a	
				CF	Conversion factor	kg/mg	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	15	EPA, 2002b	15	EPA, 2002b	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	2,190	Calculated (ED x 365 days/year)	2,190	Calculated (ED x 365 days/year)	
			Adult	EPC	Exposure Point Concentration	mg/kg	Calculated	See Tables 7-6, 7-7	Calculated	See Tables 7-6, 7-7	Chronic daily intake (mg/kg-day) = EPC x IRS x FI x EF x ED x CF x 1/BW x 1/AT
				IRS	Ingestion Rate of Soil	mg/day	100	EPA, 2002a	100	EPA, 2002a	
				FI	Fraction Ingested	unitless	1	Professional judgment	0.5	Professional judgment	
				EF	Exposure Frequency	days/year	350	EPA, 2002a	350	EPA, 2002a	
				ED	Exposure Duration	years	24	EPA, 2002a	9	EPA, 1997a	
				CF	Conversion factor	kg/mg	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	70	EPA, 2002a	70	EPA, 2002a	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	8,760	Calculated (ED x 365 days/year)	3,285	Calculated (ED x 365 days/year)	

Table 7-1
Soil Exposure Parameters
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Soil

Exposure Point	Receptor Population	Exposure Route	Receptor Age	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CTE Value	CTE Rationale/ Reference	Intake Equation/ Model Name
Soil (cont.)	Resident (cont.)	Dermal Contact	Child (1 to 6 years)	EPC	Exposure Point Concentration	mg/kg	Calculated	See Tables 7-6, 7-7	Calculated	See Tables 7-6, 7-7	Dermally Absorbed Dose (mg/kg-day) = EPC x SA x AF x ABS x EF x ED x CF x 1/BW x 1/AT
				SA	Exposed Skin Surface Area	cm^2/day	2,800	EPA, 2004	2,800	EPA, 2004	
				AF	Soil to Skin Adherence Factor	mg/cm^2	0.2	EPA, 2004	0.04	EPA, 2004	
				ABS	Dermal Absorption Factor	unitless	COPC specific	EPA, 2004, see Table 7-5	COPC specific	EPA, 2004, see Table 7-5	
				EF	Exposure Frequency	days/year	350	EPA, 2002a	350	EPA, 2002a	
				ED	Exposure Duration	years	6	EPA, 2002a	6	EPA, 2002a	
				CF	Conversion factor	kg/mg	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	15	EPA, 2002b	15	EPA, 2002b	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	2,190	Calculated (ED x 365 days/year)	2,190	Calculated (ED x 365 days/year)	
			Adult	EPC	Exposure Point Concentration	mg/kg	Calculated	See Tables 7-6, 7-7	Calculated	See Tables 7-6, 7-7	Dermally Absorbed Dose (mg/kg-day) = EPC x SA x AF x ABS x EF x ED x CF x 1/BW x 1/AT
				SA	Exposed Skin Surface Area	cm^2/day	5,700	EPA, 2004	5,700	EPA, 2004	
				AF	Soil to Skin Adherence Factor	mg/cm^2	0.07	EPA, 2004	0.01	EPA, 2004	
				ABS	Dermal Absorption Factor	unitless	COPC specific	EPA, 2004, see Table 7-5	COPC specific	EPA, 2004, see Table 7-5	
				EF	Exposure Frequency	days/year	350	EPA, 2002a	350	EPA, 2002a	
				ED	Exposure Duration	years	24	EPA, 2002a	9	EPA, 1997a	
				CF	Conversion factor	kg/mg	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	70	EPA, 2002a	70	EPA, 2002a	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	8,760	Calculated (ED x 365 days/year)	3,285	Calculated (ED x 365 days/year)	
		Inhalation	Child (1 to 6 years)	EPC	Exposure Point Concentration	mg/kg	Calculated	See Tables 7-6, 7-7	Calculated	See Tables 7-6, 7-7	EC ($\mu g/m^3$) = (CA x ET x EF x ED x CF2)/AT where: CA ($\mu g/m^3$) = (EPC/PEF) x CF3
				EC	Exposure Concentration	$\mu g/m^3$	Calculated	EPA, 2009a	Calculated	EPA, 2009a	
				CA	Chemical Concentration in Air	$\mu g/m^3$	Calculated	EPA, 2009a	Calculated	EPA, 2009a	
				ET	Exposure Time	hours/day	24	Professional judgment	16	Professional judgment	
				EF	Exposure Frequency	days/year	350	EPA, 2002a	350	EPA, 2002a	
				ED	Exposure Duration	years	6	EPA, 2002a	6	EPA, 2002a	
				CF2	Conversion Factor 2	days/hour	0.042	Units conversion	0.042	Units conversion	
				CF3	Conversion Factor 3	$\mu g/mg$	1000	Units conversion	1000	Units conversion	
				PEF	Particulate Emission Factor	m^3/kg	6.3E+09	See Appendix C	6.3E+09	See Appendix C	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	2,190	Calculated (ED x 365 days/year)	2,190	Calculated (ED x 365 days/year)	
			Adult	EPC	Exposure Point Concentration	mg/kg	Calculated	See Tables 7-6, 7-7	Calculated	See Tables 7-6, 7-7	EC ($\mu g/m^3$) = (CA x ET x EF x ED x CF2)/AT where: CA ($\mu g/m^3$) = (EPC/PEF) x CF3
				EC	Exposure Concentration	$\mu g/m^3$	Calculated	EPA, 2009a	Calculated	EPA, 2009a	
				CA	Chemical Concentration in Air	$\mu g/m^3$	Calculated	EPA, 2009a	Calculated	EPA, 2009a	
				ET	Exposure Time	hours/day	24	Professional judgment	16	Professional judgment	
				EF	Exposure Frequency	days/year	350	EPA, 2002a	350	EPA, 2002a	
				ED	Exposure Duration	years	24	Professional judgment	9	EPA, 1997a	
				CF2	Conversion Factor 2	days/hour	0.042	Units conversion	0.042	Units conversion	
				CF3	Conversion Factor 3	$\mu g/mg$	1000	Units conversion	1000	Units conversion	
				PEF	Particulate Emission Factor	m^3/kg	6.3E+09	See Appendix C	6.3E+09	See Appendix C	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	8,760	Calculated (ED x 365 days/year)	3,285	Calculated (ED x 365 days/year)	

Table 7-1
Soil Exposure Parameters
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Soil

Exposure Point	Receptor Population	Exposure Route	Receptor Age	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CTE Value	CTE Rationale/ Reference	Intake Equation/ Model Name
Soil (cont.)	Construction Worker	Ingestion	Adult	EPC	Exposure Point Concentration	mg/kg	Calculated	See Table 7-7	Calculated	See Table 7-7	Chronic daily intake (mg/kg-day) = EPC x IRS x FI x EF x ED x CF x 1/BW x 1/AT
				IRS	Ingestion Rate of Soil	mg/day	330	EPA, 2002a	100	EPA, 2002a	
				FI	Fraction Ingested	unitless	1	Professional judgment	1	Professional judgment	
				EF	Exposure Frequency	days/year	60	Professional judgment	30	Professional judgment	
				ED	Exposure Duration	years	1	Professional judgment	1	Professional judgment	
				BW	Body Weight	kg	70	EPA, 2002a	70	EPA, 2002a	
				CF	Conversion factor	kg/mg	1.00E-06	Units conversion	1.00E-06	Units conversion	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	365	Calculated (ED x 365 days/year)	365	Calculated (ED x 365 days/year)	
		Dermal Contact	Adult	EPC	Exposure Point Concentration	mg/kg	Calculated	See Table 7-7	Calculated	See Table 7-7	Dermally Absorbed Dose (mg/kg-day) = EPC x SA x AF x ABS x EF x ED x CF x 1/BW x 1/AT
				SA	Exposed Skin Surface Area	cm^2/day	3,300	EPA, 2004	3,300	EPA, 2004	
				AF	Soil to Skin Adherence Factor	mg/cm^2	0.24	EPA, 2004	0.14	EPA, 2004	
				ABS	Dermal Absorption Factor	unitless	COPC specific	EPA, 2004, see Table 7-5	COPC specific	EPA, 2004, see Table 7-5	
				EF	Exposure Frequency	days/year	60	Professional judgment	30	Professional judgment	
				ED	Exposure Duration	years	1	Professional judgment	1	Professional judgment	
				BW	Body Weight	kg	70	EPA, 2002a	70	EPA, 2002a	
				CF	Conversion factor	kg/mg	1.00E-06	Units conversion	1.00E-06	Units conversion	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	365	Calculated (ED x 365 days/year)	365	Calculated (ED x 365 days/year)	
		Inhalation	Adult	EPC	Exposure Point Concentration	mg/kg	Calculated	See Table 7-7	Calculated	See Table 7-7	EC (µg/m^3) = (CA x ET x EF x ED x CF2)/AT where: CA (µg/m^3) = (EPC/PEF) x CF3
				EC	Exposure Concentration	µg/m^3	Calculated	EPA, 2009a	Calculated	EPA, 2009a	
				CA	Chemical Concentration in Air	µg/m^3	Calculated	EPA, 2009a	Calculated	EPA, 2009a	
				ET	Exposure Time	hours/day	8	Professional judgment	4	Professional judgment	
				EF	Exposure Frequency	days/year	60	Professional judgment	30	Professional judgment	
				ED	Exposure Duration	years	1	Professional judgment	1	Professional judgment	
				CF2	Conversion Factor 2	days/hour	0.042	Units conversion	0.042	Units conversion	
				CF3	Conversion Factor 3	µg/mg	1000	Units conversion	1000	Units conversion	
				BW	Body Weight	kg	70	EPA, 2002a	70	EPA, 2002a	
				PEF	Particulate Emission Factor	m^3/kg	5.4E+05	See Appendix C	6.9E+05	See Appendix C	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	365	Calculated (ED x 365 days/year)	365	Calculated (ED x 365 days/year)	

Table 7-2
Sediment Exposure Parameters
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment

Exposure Point	Receptor Population	Exposure Route	Receptor Age	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CTE Value	CTE Rationale/ Reference	Intake Equation/ Model Name
Sediment	Swimmer/Wader	Ingestion	Adolescent (10 to 18 years)	EPC	Exposure Point Concentration	mg/kg	Calculated	See Table 7-8	Calculated	See Table 7-8	Chronic daily intake (mg/kg-day) = EPC x IRSED x FI x EF x ED x CF x 1/BW x 1/AT
				IRSED	Incidental Sediment Ingestion Rate	mg/day	100	EPA, 2002a	100	EPA, 2002a	
				FI	Fraction Ingested	unitless	1	Professional judgment	0.5	Professional judgment	
				EF	Exposure Frequency	days/year	22	Professional judgment	11	Professional judgment	
				ED	Exposure Duration	years	8	Estimated	8	Estimated	
				CF	Conversion factor	kg/mg	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	52	Tables 7-6 and 7-7 of EPA, 1997a	52	Tables 7-6 and 7-7 of EPA, 1997a	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	2,920	Calculated (ED x 365 days/year)	2,920	Calculated (ED x 365 days/year)	
			Adult	EPC	Exposure Point Concentration	mg/kg	Calculated	See Table 7-8	Calculated	See Table 7-8	Chronic daily intake (mg/kg-day) = EPC x IRSED x FI x EF x ED x CF x 1/BW x 1/AT
				IRSED	Incidental Sediment Ingestion Rate	mg/day	100	EPA, 2002a	100	EPA, 2002a	
				FI	Fraction Ingested	unitless	1	Professional judgment	0.5	Professional judgment	
				EF	Exposure Frequency	days/year	22	Professional judgment	11	Professional judgment	
				ED	Exposure Duration	years	24	Professional judgment	24	Professional judgment	
				CF	Conversion factor	kg/mg	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	70	EPA, 2002a	70	EPA, 2002a	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	8,760	Calculated (ED x 365 days/year)	8,760	Calculated (ED x 365 days/year)	
		Dermal Contact	Adolescent (10 to 18 years)	EPC	Exposure Point Concentration	mg/kg	Calculated	See Table 7-8	Calculated	See Table 7-8	Dermally Absorbed Dose (mg/kg-day) = EPC x SA x AF x ABS x EF x ED x CF x 1/BW x 1/AT
				SA	Exposed Skin Surface Area	cm^2/day	5,900	EPA, 2004	3,600	EPA, 2004	
				AF	Soil to Skin Adherence Factor	mg/cm^2	0.32	EPA, 2004	0.07	EPA, 2004	
				ABS	Dermal Absorption Factor	unitless	COPC specific	EPA, 2004, see Table 7-5	COPC specific	EPA, 2004, see Table 7-5	
				EF	Exposure Frequency	days/year	22	Professional judgment	11	Professional judgment	
				ED	Exposure Duration	years	8	Estimated	8	Estimated	
				CF	Conversion factor	kg/mg	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	52	Tables 7-6 and 7-7 of EPA, 1997a	52	Tables 7-6 and 7-7 of EPA, 1997a	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	2,920	Calculated (ED x 365 days/year)	2,920	Calculated (ED x 365 days/year)	
			Adult	EPC	Exposure Point Concentration	mg/kg	Calculated	See Table 7-8	Calculated	See Table 7-8	Dermally Absorbed Dose (mg/kg-day) = EPC x SA x AF x ABS x EF x ED x CF x 1/BW x 1/AT
				SA	Exposed Skin Surface Area	cm^2/day	6,900	EPA, 2004	4,500	EPA, 2004	
				AF	Soil to Skin Adherence Factor	mg/cm^2	0.32	EPA, 2004	0.07	EPA, 2004	
				ABS	Dermal Absorption Factor	unitless	COPC specific	EPA, 2004, see Table 7-5	COPC specific	EPA, 2004, see Table 7-5	
				EF	Exposure Frequency	days/year	22	Professional judgment	11	Professional judgment	
				ED	Exposure Duration	years	24	Professional judgment	24	Professional judgment	
				CF	Conversion factor	kg/mg	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	70	EPA, 2002a	70	EPA, 2002a	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	8,760	Calculated (ED x 365 days/year)	8,760	Calculated (ED x 365 days/year)	

Table 7-3
Surface Water Exposure Parameters
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Surface Water
Exposure Medium: Surface Water

Exposure Point	Receptor Population	Exposure Route	Receptor Age	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CTE Value	CTE Rationale/ Reference	Intake Equation/ Model Name
Surface Water	Swimmer/Wader	Ingestion	Adolescent (10 to 18 years)	EPC	Exposure Point Concentration	µg/L	Calculated	See Table 7-9	Calculated	See Table 7-9	Chronic daily intake (mg/kg-day) = EPC x IRW x FI x ET x EF x ED x CF x 1/BW x 1/AT
				IRW	Incidental Surface Water Ingestion Rate	L/hr	0.05	EPA, 1989	0.05	EPA, 1989	
				FI	Fraction Ingested	unitless	1	Professional judgment	1	Professional judgment	
				ET	Exposure Time	hours/day	2	Professional judgment	1	Professional judgment	
				EF	Exposure Frequency	days/year	22	Professional judgment	11	Professional judgment	
				ED	Exposure Duration	years	8	Estimated	8	Estimated	
				BW	Body Weight	kg	52	Tables 7-6 and 7-7 of EPA, 1997a	52	Tables 7-6 and 7-7 of EPA, 1997a	
				CF	Conversion factor	mg/µg	1.00E-03	Units conversion	1.00E-03	Units conversion	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	2,920	Calculated (ED x 365 days/year)	2,920	Calculated (ED x 365 days/year)	
			Adult	EPC	Exposure Point Concentration	µg/L	Calculated	See Table 7-9	Calculated	See Table 7-9	Chronic daily intake (mg/kg-day) = EPC x IRW x FI x ET x EF x ED x CF x 1/BW x 1/AT
				IRW	Incidental Surface Water Ingestion Rate	L/hr	0.05	EPA, 1989	0.05	EPA, 1989	
				FI	Fraction Ingested	unitless	1	Professional judgment	1	Professional judgment	
				ET	Exposure Time	hours/day	2	Professional judgment	1	Professional judgment	
				EF	Exposure Frequency	days/year	22	Professional judgment	11	Professional judgment	
				ED	Exposure Duration	years	24	Professional judgment	24	Professional judgment	
				BW	Body Weight	kg	70	EPA, 2002a	70	EPA, 2002a	
				CF	Conversion factor	mg/µg	1.00E-03	Units conversion	1.00E-03	Units conversion	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	8,760	Calculated (ED x 365 days/year)	8,760	Calculated (ED x 365 days/year)	
		Dermal Contact	Adolescent (10 to 18 years)	EPC	Exposure Point Concentration	µg/L	Calculated	See Table 7-9	Calculated	See Table 7-9	Dermally Absorbed Dose (mg/kg-day) = EPC x Kp x t_{event} x SA x EV x EF x ED x CF x 1/BW x 1/AT
				Kp	Dermal Permeability Coefficient	cm/hour	COPC specific	EPA, 2004; See Table 7-5	COPC specific	EPA, 2004; See Table 7-5	
				t_{event}	Event Duration	hour/event	2	Professional judgment	1	Professional judgment	
				SA	Exposed Skin Surface Area	cm^2/day	14,900	EPA, 2004	14,900	EPA, 2004	
				EV	Event Frequency	event/day	1	EPA, 2004	1	EPA, 2004	
				EF	Exposure Frequency	days/year	22	Professional judgment	11	Professional judgment	
				ED	Exposure Duration	years	8	Estimated	8	Estimated	
				CF	Conversion factor	mg-L/µg-cm^3	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	52	Tables 7-6 and 7-7 of EPA, 1997a	52	Tables 7-6 and 7-7 of EPA, 1997a	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	2,920	Calculated (ED x 365 days/year)	2,920	Calculated (ED x 365 days/year)	
			Adult	EPC	Exposure Point Concentration	µg/L	Calculated	See Table 7-9	Calculated	See Table 7-9	Dermally Absorbed Dose (mg/kg-day) = EPC x Kp x t_{event} x SA x EV x EF x ED x CF x 1/BW x 1/AT
				Kp	Dermal Permeability Coefficient	cm/hour	COPC specific	EPA, 2004; See Table 7-5	COPC specific	EPA, 2004; See Table 7-5	
				t_{event}	Event Duration	hour/event	2	Professional judgment	1	Professional judgment	
				SA	Exposed Skin Surface Area	cm^2/day	18,000	EPA, 2004	18,000	EPA, 2004	
				EV	Event Frequency	event/day	1	EPA, 2004	1	EPA, 2004	
				EF	Exposure Frequency	days/year	22	Professional judgment	11	Professional judgment	
				ED	Exposure Duration	years	24	Professional judgment	24	Professional judgment	
				CF	Conversion factor	mg-L/µg-cm^3	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	70	EPA, 2002a	70	EPA, 2002a	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	8,760	Calculated (ED x 365 days/year)	8,760	Calculated (ED x 365 days/year)	

Table 7-4
Groundwater Exposure Parameters
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Groundwater
Exposure Medium: Groundwater

Exposure Point	Receptor Population	Exposure Route	Receptor Age	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CTE Value	CTE Rationale/ Reference	Intake Equation/ Model Name
Groundwater	Resident	Ingestion	Child (1 to 6 years)	EPC	Exposure Point Concentration	µg/L	Calculated	See Tables 7-10, 7-11	Calculated	See Tables 7-10, 7-11	Chronic daily intake (mg/kg-day) = EPC x IRW x FI x EF x ED x CF x 1/BW x 1/AT
				IRW	Water Ingestion Rate	L/day	1	EPA, 2002b	1	EPA, 2002b	
				FI	Fraction Ingested	unitless	1	Professional judgment	0.5	Professional judgment	
				EF	Exposure Frequency	days/year	350	EPA, 2002a	350	EPA, 2002a	
				ED	Exposure Duration	years	6	EPA, 2002a	6	EPA, 2002a	
				BW	Body Weight	kg	15	EPA, 2002a	15	EPA, 2002a	
				CF	Conversion factor	mg/µg	1.00E-03	Units conversion	1.00E-03	Units conversion	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	2,190	Calculated (ED x 365 days/year)	2,190	Calculated (ED x 365 days/year)	
			Adult	EPC	Exposure Point Concentration	µg/L	Calculated	See Tables 7-10, 7-11	Calculated	See Tables 7-10, 7-11	Chronic daily intake (mg/kg-day) = EPC x IRW x FI x EF x ED x CF x 1/BW x 1/AT
				IRW	Water Ingestion Rate	L/day	2	EPA, 2002a	2	EPA, 2002a	
				FI	Fraction Ingested	unitless	1	Professional judgment	0.5	Professional judgment	
				EF	Exposure Frequency	days/year	350	EPA, 2002a	350	EPA, 2002a	
				ED	Exposure Duration	years	24	EPA, 2002a	9	EPA, 1997a	
				BW	Body Weight	kg	70	EPA, 2002a	70	EPA, 2002a	
				CF	Conversion factor	mg/µg	1.00E-03	Units conversion	1.00E-03	Units conversion	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	8,760	Calculated (ED x 365 days/year)	3,285	Calculated (ED x 365 days/year)	
		Dermal Contact	Child (1 to 6 years)	EPC	Exposure Point Concentration	µg/L	Calculated	See Tables 7-10, 7-11	Calculated	See Tables 7-10, 7-11	Dermally Absorbed Dose (mg/kg-day) = EPC x Kp x t_{event} x SA x EV x EF x ED x CF x 1/BW x 1/AT
				Kp	Dermal Permeability Coefficient	cm/hour	COPC specific	EPA, 2004; See Table 7-5	COPC specific	EPA, 2004; See Table 7-5	
				t_{event}	Event Duration	hour/event	1	EPA, 2004	0.5	Professional judgment	
				SA	Exposed Skin Surface Area	cm^2/day	6,600	EPA, 2004	6,600	EPA, 2004	
				EV	Event Frequency	event/day	1	EPA, 2004	1	EPA, 2004	
				EF	Exposure Frequency	days/year	350	EPA, 2002a	350	EPA, 2002a	
				ED	Exposure Duration	years	6	EPA, 2002a	6	EPA, 2002a	
				CF	Conversion factor	mg-L/µg-cm^3	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	15	EPA, 2002a	15	EPA, 2002a	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	2,190	Calculated (ED x 365 days/year)	2,190	Calculated (ED x 365 days/year)	
			Adult	EPC	Exposure Point Concentration	µg/L	Calculated	See Tables 7-10, 7-11	Calculated	See Tables 7-10, 7-11	Dermally Absorbed Dose (mg/kg-day) = EPC x Kp x t_{event} x SA x EV x EF x ED x CF x 1/BW x 1/AT
				Kp	Dermal Permeability Coefficient	cm/hour	COPC specific	EPA, 2004; See Table 7-5	COPC specific	EPA, 2004; See Table 7-5	
				t_{event}	Event Duration	hour/event	0.58	EPA, 2004	0.29	Professional judgment	
				SA	Exposed Skin Surface Area	cm^2/day	18,000	EPA, 2004	18,000	EPA, 2004	
				EV	Event Frequency	event/day	1	EPA, 2004	1	EPA, 2004	
				EF	Exposure Frequency	days/year	350	EPA, 2002a	350	EPA, 2002a	
				ED	Exposure Duration	years	24	EPA, 2002a	9	EPA, 1997a	
				CF	Conversion factor	mg-L/µg-cm^3	1.00E-06	Units conversion	1.00E-06	Units conversion	
				BW	Body Weight	kg	70	EPA, 2002a	70	EPA, 2002a	
				AT-C	Averaging Time (Cancer)	days	25,550	EPA, 1989	25,550	EPA, 1989	
				AT-NC	Averaging Time (Non-Cancer)	days	8,760	Calculated (ED x 365 days/year)	3,285	Calculated (ED x 365 days/year)	

Table 7-5
Dermal Absorption Factors
Ely Copper Mine Superfund Site
Vershire, Vermont

COPC	Dermal Absorption Factor for Soil [a] (unitless)	Dermal Permeability Coefficient [b] (cm/hour)
Bis(2-Ethylhexyl)Phthalate	0.1	NA
Aluminum	NA	NA
Antimony	NA	0.001
Arsenic	0.03	0.001
Barium	NA	0.001
Beryllium	NA	0.001
Cadmium	0.001	0.001
Chromium	NA	0.002
Cobalt	NA	NA
Copper	NA	0.001
Iron	NA	NA
Lead	NA	NA
Lithium	NA	NA
Manganese	NA	0.001
Mercury	NA	0.001
Molybdenum	NA	NA
Nickel	NA	0.0002
Selenium	NA	0.001
Vanadium	NA	0.001
Zinc	NA	0.0006

Notes:

[a] Exhibit 3-4, Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual, Part E – Supplemental Guidance for Dermal Risk Assessment (EPA, 2004).

[b] Exhibit B-4, Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual, Part E – Supplemental Guidance for Dermal Risk Assessment (EPA, 2004).

NA = Not available

Table 7-6
Surface Soil - Exposure Point Concentration Summary
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Soil
Exposure Medium: Surface soil 0-1 ft bgs

Exposure Point	Contaminant of Potential Concern	Units	Arithmetic Mean	95% UCL (Distribution)	Maximum Concentration	Exposure Point Concentration			
						Value	Units	Statistic	Rationale
Surface soil	Aluminum	mg/kg	10083	10820 (G)	100000	10820	mg/kg	95% Approximate Gamma UCL	ProUCL Recommendation
	Antimony	mg/kg	1.1	1.276 (G)	8.1	1.3	mg/kg	95% KM (t) UCL	ProUCL Recommendation
	Arsenic	mg/kg	2.4	2.725 (ND)	23	2.7	mg/kg	95% KM (BCA) UCL	ProUCL Recommendation
	Cadmium	mg/kg	0.87	0.987 (L)	8.5	0.99	mg/kg	95% KM (BCA) UCL	ProUCL Recommendation
	Chromium	mg/kg	35	43.71 (ND)	440	44	mg/kg	95% Chebyshev (Mean, Sd) UCL	ProUCL Recommendation
	Cobalt	mg/kg	18	24.58 (ND)	200	25	mg/kg	95% Chebyshev (Mean, Sd) UCL	ProUCL Recommendation
	Copper	mg/kg	964	1086 approx. (G)	23000	1086	mg/kg	95% Approximate Gamma UCL	ProUCL Recommendation
	Iron	mg/kg	53810	66914 (ND)	300000	66914	mg/kg	95% Chebyshev (Mean, Sd) UCL	ProUCL Recommendation
	Manganese	mg/kg	296	448.9 (ND)	8380	449	mg/kg	95% Chebyshev (Mean, Sd) UCL	ProUCL Recommendation
	Mercury	mg/kg	0.12	0.148 (L)	2.8	0.15	mg/kg	95% KM (BCA) UCL	ProUCL Recommendation
	Molybdenum	mg/kg	4.9	6.865 (ND)	69	6.9	mg/kg	95% KM (Chebyshev) UCL	ProUCL Recommendation
	Selenium	mg/kg	16	21.07 (ND)	170	21	mg/kg	95% KM (Chebyshev) UCL	ProUCL Recommendation
	Vanadium	mg/kg	47	56.35 (ND)	400	56	mg/kg	95% Chebyshev (Mean, Sd) UCL	ProUCL Recommendation

Notes:
Maximum detected concentration used as the EPC in instances where n < 8 or detects < 4.

G = Gamma distribution.
L = Lognormal distribution.
ND = Data distribution is not discernable.

Table 7-7
Total Soil - Exposure Point Concentration Summary
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Total soil 0-10 ft bgs

Exposure Point	Contaminant of Potential Concern	Units	Arithmetic Mean	95% UCL (Distribution)	Maximum Concentration	Exposure Point Concentration			
						Value	Units	Statistic	Rationale
Total Soil	Aluminum	mg/kg	11114	11814 (G)	100000	11814	mg/kg	95% Approximate Gamma UCL	ProUCL Recommendation
	Antimony	mg/kg	0.90	1.054 approx. (G)	12	1.1	mg/kg	95% KM (t) UCL	ProUCL Recommendation
	Arsenic	mg/kg	2.1	2.323 (ND)	23	2.3	mg/kg	95% KM (BCA) UCL	ProUCL Recommendation
	Cadmium	mg/kg	1.1	1.859 (ND)	45	1.9	mg/kg	95% KM (Chebyshev) UCL	ProUCL Recommendation
	Chromium	mg/kg	40	48.14 (ND)	440	48	mg/kg	95% Chebyshev (Mean, Sd) UCL	ProUCL Recommendation
	Cobalt	mg/kg	28	50.24 (ND)	1500	50	mg/kg	95% Chebyshev (Mean, Sd) UCL	ProUCL Recommendation
	Copper	mg/kg	1200	1887 (ND)	36000	1887	mg/kg	95% Chebyshev (Mean, Sd) UCL	ProUCL Recommendation
	Iron	mg/kg	55835	67413 (ND)	300000	67413	mg/kg	95% Chebyshev (Mean, Sd) UCL	ProUCL Recommendation
	Manganese	mg/kg	290	410.9 (ND)	8380	411	mg/kg	95% Chebyshev (Mean, Sd) UCL	ProUCL Recommendation
	Mercury	mg/kg	0.11	0.125 (L)	2.8	0.13	mg/kg	95% KM (BCA) UCL	ProUCL Recommendation
	Molybdenum	mg/kg	4.7	6.391 (ND)	69	6.4	mg/kg	95% KM (Chebyshev) UCL	ProUCL Recommendation
	Nickel	mg/kg	24	27.33 (ND)	470	27	mg/kg	95% KM (BCA) UCL	ProUCL Recommendation
	Selenium	mg/kg	15	21.76 (ND)	170	22	mg/kg	97.5% KM (Chebyshev) UCL	ProUCL Recommendation
	Vanadium	mg/kg	50	56.92 (ND)	400	57	mg/kg	95% Chebyshev (Mean, Sd) UCL	ProUCL Recommendation
	Zinc	mg/kg	162	261.8 (ND)	5700	262	mg/kg	95% Chebyshev (Mean, Sd) UCL	ProUCL Recommendation

Notes:
Maximum detected concentration used as the EPC in instances where n < 8 or detects < 4.

G = Gamma distribution.
L = Lognormal distribution.
ND = Data distribution is not discernable.

Table 7-8
Sediment - Exposure Point Concentration Summary
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Surface sediment

Exposure Point	Contaminant of Potential Concern	Units	Arithmetic Mean	95% UCL (Distribution)	Maximum Concentration	Exposure Point Concentration			
						Value	Units	Statistic	Rationale
Ely Brook Sediment	Aluminum	mg/kg	27044	34526 (G)	111000	34526	mg/kg	95% Approximate Gamma UCL	ProUCL Recommendation
	Arsenic	mg/kg	2.7	3.663 (L)	18.5	3.7	mg/kg	95% KM (Percentile Bootstrap) UCL	ProUCL Recommendation
	Chromium	mg/kg	47	53.93 (G)	130	54	mg/kg	95% Approximate Gamma UCL	ProUCL Recommendation
	Cobalt	mg/kg	29	45.08 (ND)	102	45	mg/kg	95% Chebyshev (Mean, Sd) UCL	ProUCL Recommendation
	Copper	mg/kg	2193	3118 (G)	11000	3118	mg/kg	95% Approximate Gamma UCL	ProUCL Recommendation
	Iron	mg/kg	61572	74219 (G)	167000	74219	mg/kg	95% Approximate Gamma UCL	ProUCL Recommendation
	Lithium	mg/kg	28	34.09 (N)	44	34	mg/kg	95% Student's-t UCL	ProUCL Recommendation
	Manganese	mg/kg	728	973 (G)	3130	973	mg/kg	95% Approximate Gamma UCL	ProUCL Recommendation
	Selenium	mg/kg	12	35.03 (ND)	46	35	mg/kg	99% KM (Chebyshev) UCL	ProUCL Recommendation
	Vanadium	mg/kg	65	74.77 approx. (G)	163	75	mg/kg	95% Approximate Gamma UCL	ProUCL Recommendation
Schoolhouse Brook Sediment	Aluminum	mg/kg	21361	42733 (ND)	36900	42733	mg/kg	95% Chebyshev (Mean, Sd)	ProUCL Recommendation
	Arsenic	mg/kg	1.1	1.414 (ND)	2.0	1.4	mg/kg	95% KM (t) UCL	ProUCL Recommendation
	Chromium	mg/kg	22	29.18 (N)	40	29	mg/kg	95% Student's t-UCL	ProUCL Recommendation
	Cobalt	mg/kg	8.8	10.68 (N)	14	11	mg/kg	95% Student's t-UCL	ProUCL Recommendation
	Copper	mg/kg	339	852.8 (ND)	1390	853	mg/kg	95% Chebyshev (Mean, Sd)	ProUCL Recommendation
	Iron	mg/kg	22836	32270 (G)	58800	32270	mg/kg	95% Approximate Gamma	ProUCL Recommendation
	Lithium	mg/kg	21	Not Determined	24	24	mg/kg	Maximum	See footnote.
	Manganese	mg/kg	545	739.2 (N)	1120	739	mg/kg	95% Student's t-UCL	ProUCL Recommendation
	Vanadium	mg/kg	33	44.58 (N)	62	44.6	mg/kg	95% Student's t-UCL	ProUCL Recommendation
Ompompanoosuc River Sediment	Aluminum	mg/kg	16100	Not Determined	35400	35400	mg/kg	Maximum	See footnote.
	Arsenic	mg/kg	17	Not Determined	5	5	mg/kg	Maximum	See footnote.
	Chromium	mg/kg	22	Not Determined	31	31	mg/kg	Maximum	See footnote.
	Cobalt	mg/kg	8.4	Not Determined	13	13	mg/kg	Maximum	See footnote.
	Iron	mg/kg	14375	Not Determined	22800	22800	mg/kg	Maximum	See footnote.
	Lithium	mg/kg	24	Not Determined	24	24	mg/kg	Maximum	See footnote.
	Manganese	mg/kg	493	Not Determined	1120	1120	mg/kg	Maximum	See footnote.
	Vanadium	mg/kg	28	Not Determined	49	49	mg/kg	Maximum	See footnote.

Notes:
Maximum detected concentration used as the EPC in instances where n < 8 or detects < 4.
G = Gamma distribution.
N = Normal distribution.
ND = Data distribution is not discernable.

Table 7-9
Surface Water - Exposure Point Concentration Summary
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Surface Water
Exposure Medium: Surface Water

Exposure Point	Contaminant of Potential Concern	Units	Arithmetic Mean	95% UCL (Distribution)	Maximum Concentration	Exposure Point Concentration			
						Value	Units	Statistic	Rationale
Ely Brook Surface Water	Aluminum	µg/L	7061	12885 approx. (G)	103590	12885	µg/L	95% KM (Chebyshev) UCL	ProUCL Recommendation
	Arsenic	µg/L	0.17	0.246 (ND)	0.64	0.25	µg/L	95% KM (t) UCL	ProUCL Recommendation
	Cadmium	µg/L	3.3	7.55 (ND)	56	7.6	µg/L	97.5% KM (Chebyshev) UCL	ProUCL Recommendation
	Chromium	µg/L	4.3	10.35 (L)	125	10	µg/L	95% KM (Chebyshev) UCL	ProUCL Recommendation
	Cobalt	µg/L	86	153.2 approx. (G)	1174	153	µg/L	95% KM (Chebyshev) UCL	ProUCL Recommendation
	Copper	µg/L	3230	4391 approx. (G)	33178	4391	µg/L	95% Adjusted Gamma UCL	ProUCL Recommendation
	Iron	µg/L	2786	6354 (ND)	28300	6354	µg/L	97.5% KM (Chebyshev) UCL	ProUCL Recommendation
	Lithium	µg/L	3.5	6.299 (N)	15	6.3	µg/L	95% KM (t) UCL	ProUCL Recommendation
	Manganese	µg/L	614	776.6 approx. (G)	5230	777	µg/L	95% Approximate Gamma UCL	ProUCL Recommendation
	Mercury	µg/L	0.12	Not Determined	0.099	0.099	µg/L	Maximum	See footnote.
	Nickel	µg/L	26	57.27 (ND)	308	57	µg/L	97.5% KM (Chebyshev) UCL	ProUCL Recommendation
	Vanadium	µg/L	0.83	1.545 (ND)	11	1.5	µg/L	95% KM (Chebyshev) UCL	ProUCL Recommendation
	Zinc	µg/L	458	1073 (ND)	8192	1073	µg/L	95% KM (Chebyshev) UCL	ProUCL Recommendation
Schoolhouse Brook Surface Water	Chromium	µg/L	2.0	2.585 (N)	4.5	2.6	µg/L	95% KM (t) UCL	ProUCL Recommendation
	Cobalt	µg/L	4.0	8.939 (ND)	25	8.9	µg/L	95% KM (Chebyshev) UCL	ProUCL Recommendation
	Copper	µg/L	129	336.8 (ND)	1100	337	µg/L	95% KM (Chebyshev) UCL	ProUCL Recommendation
	Manganese	µg/L	43	56.46 (L)	260	56	µg/L	95% H-UCL	ProUCL Recommendation
	Mercury	µg/L	0.14	Not Determined	0.17	0.17	µg/L	Maximum	See footnote.
	Vanadium	µg/L	0.4	0.471 approx. (G)	0.97	0.47	µg/L	95% KM (t) UCL	ProUCL Recommendation
Ompompanoosuc River Surface Water	Mercury	µg/L	0.16	Not Determined	0.2	0.2	µg/L	Maximum	See footnote.

Notes:
Maximum detected concentration used as the EPC in instances where n < 8 or detects < 4.

G = Gamma distribution.
L = Lognormal distribution.
N = Normal distribution.
ND = Data distribution is not discernable.

Table 7-10
Groundwater - Exposure Point Concentration Summary
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Site Groundwater
Exposure Medium: Groundwater

Exposure Point	Contaminant of Potential Concern	Units	Arithmetic Mean (1)	95% UCL (Distribution)	Maximum Concentration	Exposure Point Concentration			
						Value	Units	Statistic	Rationale (2)
Overburden Groundwater	Aluminum	µg/L	9867	Not Determined	150000	150000	µg/L	Maximum detection	See footnote.
	Arsenic	µg/L	5.5	Not Determined	7.6	7.6	µg/L	Maximum detection	See footnote.
	Barium	µg/L	43	Not Determined	1202	1202	µg/L	Maximum detection	See footnote.
	Cadmium	µg/L	6.0	Not Determined	37	37	µg/L	Maximum detection	See footnote.
	Chromium	µg/L	17	Not Determined	365	365	µg/L	Maximum detection	See footnote.
	Cobalt	µg/L	117	Not Determined	668	668	µg/L	Maximum detection	See footnote.
	Copper	µg/L	4652	Not Determined	49581	49581	µg/L	Maximum detection	See footnote.
	Iron	µg/L	8651	Not Determined	200000	200000	µg/L	Maximum detection	See footnote.
	Lead	µg/L	7.3	Not Determined	52	52	µg/L	Maximum detection	See footnote.
	Manganese	µg/L	1000	Not Determined	4026	4026	µg/L	Maximum detection	See footnote.
	Molybdenum	µg/L	8.9	Not Determined	52	52	µg/L	Maximum detection	See footnote.
	Nickel	µg/L	57	Not Determined	402	402	µg/L	Maximum detection	See footnote.
	Vanadium	µg/L	30	Not Determined	261	261	µg/L	Maximum detection	See footnote.
	Zinc	µg/L	625	Not Determined	3867	3867	µg/L	Maximum detection	See footnote.
Shallow Bedrock Groundwater	Aluminum	µg/L	9333	Not Determined	360000	360000	µg/L	Maximum detection	See footnote.
	Antimony	µg/L	1.3	Not Determined	4.3	4.3	µg/L	Maximum detection	See footnote.
	Arsenic	µg/L	5.5	Not Determined	4.9	4.9	µg/L	Maximum detection	See footnote.
	Beryllium	µg/L	0.89	Not Determined	7.3	7.3	µg/L	Maximum detection	See footnote.
	Cadmium	µg/L	3.3	Not Determined	13	13	µg/L	Maximum detection	See footnote.
	Chromium	µg/L	11	Not Determined	172	172	µg/L	Maximum detection	See footnote.
	Cobalt	µg/L	46	Not Determined	267	267	µg/L	Maximum detection	See footnote.
	Copper	µg/L	1037	Not Determined	10600	10600	µg/L	Maximum detection	See footnote.
	Iron	µg/L	10750	Not Determined	310000	310000	µg/L	Maximum detection	See footnote.
	Lead	µg/L	7.2	Not Determined	27	27	µg/L	Maximum detection	See footnote.
	Manganese	µg/L	298	Not Determined	3000	3000	µg/L	Maximum detection	See footnote.
	Molybdenum	µg/L	12	Not Determined	93	93	µg/L	Maximum detection	See footnote.
	Nickel	µg/L	26	Not Determined	125	125	µg/L	Maximum detection	See footnote.
	Vanadium	µg/L	20	Not Determined	37	37	µg/L	Maximum detection	See footnote.
	Zinc	µg/L	191	Not Determined	1902	1902	µg/L	Maximum detection	See footnote.

Table 7-10
Groundwater - Exposure Point Concentration Summary
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Site Groundwater
Exposure Medium: Groundwater

Exposure Point	Contaminant of Potential Concern	Units	Arithmetic Mean (1)	95% UCL (Distribution)	Maximum Concentration	Exposure Point Concentration			
						Value	Units	Statistic	Rationale (2)
Deep Bedrock Groundwater	Bis(2-Ethylhexyl)Phthalate	µg/L	6.3	Not Determined	7.5	7.5	µg/L	Maximum detection	See footnote.
	Aluminum	µg/L	1718	Not Determined	6810	6810	µg/L	Maximum detection	See footnote.
	Arsenic	µg/L	8.5	Not Determined	2.6	2.6	µg/L	Maximum detection	See footnote.
	Cadmium	µg/L	4.9	Not Determined	6.6	6.6	µg/L	Maximum detection	See footnote.
	Chromium	µg/L	15	Not Determined	132	132	µg/L	Maximum detection	See footnote.
	Cobalt	µg/L	60	Not Determined	153	153	µg/L	Maximum detection	See footnote.
	Copper	µg/L	1788	Not Determined	8910	8910	µg/L	Maximum detection	See footnote.
	Iron	µg/L	3171	Not Determined	16948	16948	µg/L	Maximum detection	See footnote.
	Manganese	µg/L	389	Not Determined	1433	1433	µg/L	Maximum detection	See footnote.
	Nickel	µg/L	42	Not Determined	87	87	µg/L	Maximum detection	See footnote.
	Vanadium	µg/L	31	Not Determined	5.0	5.0	µg/L	Maximum detection	See footnote.

Notes:

(1) This value is the average concentration from all of the sampled wells.

(2) The maximum detected concentration was used as the EPC. It was determined that the number a groundwater sampling rounds was not enough to calculate temporal average concentrations.

Table 7-11
Residential Groundwater - Exposure Point Concentration Summary
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario Timeframe: Current/Future
Medium: Residential Groundwater
Exposure Medium: Residential Groundwater

Exposure Point	Contaminant of Potential Concern	Units	Arithmetic Mean (1)	95% UCL (Distribution)	Maximum Concentration	Exposure Point Concentration			
						Value	Units	Statistic	Rationale (2)
Groundwater	Copper	µg/L	46	Not Determined	192	192	µg/L	Maximum detection	See footnote.
	Vanadium	µg/L	17	Not Determined	1.7	1.7	µg/L	Maximum detection	See footnote.

Notes:

(1) This value is the average concentration from all of the sampled wells.

(2) The maximum detected concentration was used as the EPC. It was determined that the number a groundwater sampling rounds was not enough to calculate temporal average concentrations.

Table 9-4
Summary of Background Levels in Soil
Ely Copper Mine Supefund Site
Vershire, Vermont

COPC	Maximum Concentration	Arithmetic Mean	95% UCL
Cobalt	16	6.6	8.6
Copper	45	15.3	20.2
Iron	31000	13852	17730

Note:
Units in mg/kg.

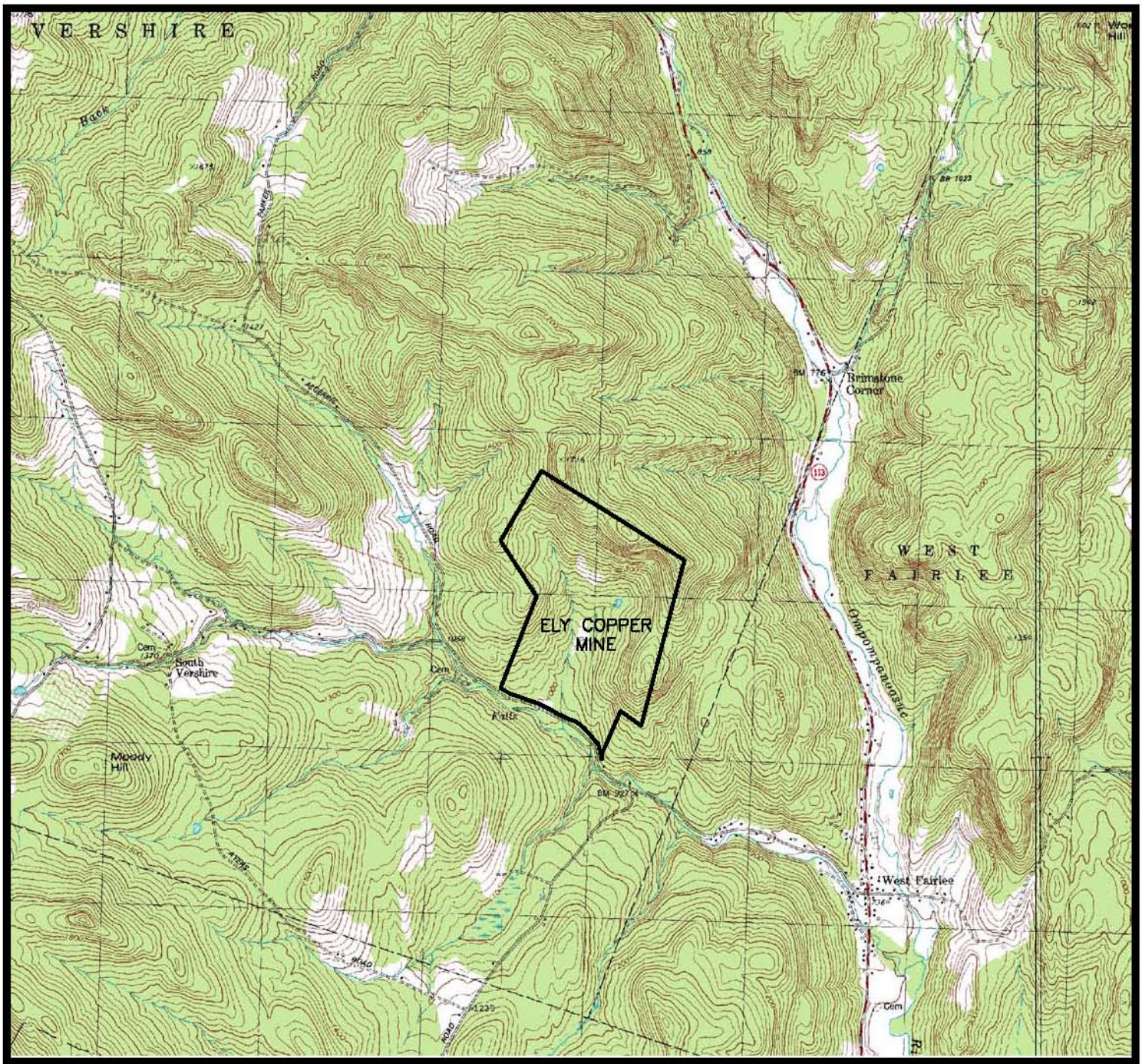
Table 9-5
Residential Exposure HIs Based on Background Concentrations
Ely Copper Mine Superfund Site
Vershire, Vermont

COPC	95% UCL (mg/kg)	Residential HI
Cobalt	8.6	0.37
Copper	20.2	0.0065
Iron	17730	0.32
	Total:	0.70

Table 9-6
Summary of Total Site-Related HIs from Residential Exposure
Ely Copper Mine Superfund Site
Vershire, Vermont

Scenario	Estimated Total Site HI	Estimated Site HI from Cobalt, Copper, and Iron	Background HI from Cobalt, Copper, and Iron	Total Site-Related HI	Percent Decrease
Current Residential Exposure	3.1	2.6	0.7	2.4	-23%
Future Residential Exposure	4.5	4.0	0.7	3.8	-16%

FIGURES



NORTH

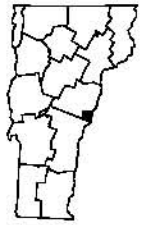
USGS TOPOGRAPHIC MAP

VERSHIRE, VERMONT

1983

APPROXIMATE SCALE
1 INCH = 3,000 FEET

Nobis Engineering, Inc.
18 Chenell Dr.
Concord, NH 03302-2090
Tel (603) 224-4182
Fax (603) 224-2507
www.nobisengineering.com



QUADRANGLE LOCATION

FIGURE 2-1

LOCUS PLAN
ELY COPPER MINE
SUPERFUND SITE
VERSHIRE, VERMONT

PROJECT: 80024.00

AUGUST 2008

N
W
E
S

NF-17
SS-39
SS-37
Underground Mine Workings
1350
SS-38
SA-31
1300
TP-08
SS-36
TP-10
TZ-38
NF-16
TP-09
SS-35
1250
TP-11
TZ-37
MW-19A
SS-34
SA-30
SB-08
TR-08D
SA-29
NF-15
1300
TZ-39
TR-08C
SA-28
TZ-40
TR-09A
TR-08B
TR-09B
TR-08A
SA-25
TR-09C
SA-27
SA-26
TR-09D
TZ-36
TZ-41
SB-06
SS-33
TP-15
MW-18A
SA-24
Upper Waste Pile Area
TQ-06
TP-01
TR-07A - D
TZ-35
TQ-16
Upper Waste Pile Area
NF-14
TP-06
TN-02
TZ-34
TP-08 - 11
TP-15
TN-10
SS-32
TZ-33
TL-04
POND 1
TO-05
NF-13
POND 2
POND 4
TZ-31
TZ-32
TK-01
TZ-30
SS-29
SS-30
POND 3
SS-28
TR-06A - C
NF-12
SA-23
POND 5
TZ-28
SB-09
TZ-29
MW-17A
SS-26
TP-16
SA-22
SA-21
MW-22A
TR-05A - D
TP-07A
SS-27
Former Flotation Mill
NF-11
SS-24
TI-03
SS-25
Ely Brook
TZ-27
SA-19
SA-20
MW-21A
TR-04B
TP-06
TR-04C
Lower Waste / Roast Bed Area
TR-04A
TZ-26
Lower Waste Pile Area
SA-16
SS-23
NF-10
SA-17
SS-21
TP-04A
TH-01
TZ-25
SS-22
SA-18
TZ-22
TZ-23
SA-15
SS-20
Ore Roast Bed Area
MW-20D
TZ-24
SB-03
TZ-21
SA-14
SS-19
NF-09
TG-02
SS-16
TF-17
TF-13
SS-12
SS-17
SB-02
MW-23A
TF-11
TF-02
TZ-19
TZ-20
TZ-18
SS-18
NF-08
TZ-17
SB-01
SS-15
SA-13
SS-14
SS-11
TE-09
TE-15
TE-12
TZ-16
SA-12
SS-13
NF-07
TZ-15
SA-11
TZ-14
TR-03B
TD-05
NF-06
TD-03
TR-03C
TR-03A
SS-10
SA-10
TZ-42
SS-04
TZ-13
TA-14
TP-103
TA-04
TP-106
NF-04
NF-05
SS-09
TP-104
TP-109
TP-108
SS-01
SS-03
TR-02A - D
TP-02B
TP-100
SA-08
SA-07
TZ-12
SS-02
TZ-11
SA-05
SA-06
SA-09
TZ-10
SA-04
TZ-09
TP-01
TZ-06
NF-03
TR-01A - D
SA-03
TZ-08
TZ-05
SA-02
NF-02
TP-03B
TP-03A
TZ-07
TZ-02
SA-01
SS-08
TZ-04
NF-01
TZ-01
Schoolhouse Brook
South Vershire Road
Smelter / Slag Area
SS-07
TZ-45
TZ-44
TC-03
SS-06
SS-05
TZ-43

1200
1150
1100
1050
1000
950

LEGEND

Sample Locations

NF-04 — Proposed Soil Sample Location

Mining Materials

- Underground Mine Workings
- Waste Rock Areas
- Smelter Area
- Slag Pile Area

Drawn By: JDF	Checked By: AB
Filename: HHRA_soil_locs.mxd	
Date: 12/15/10	Revision No. 00

APPROXIMATE SCALE
140 70 0 140 280
Feet

- Shaft
- Adit
- Prospect Tunnel
- Drill Hole
- Smoke Flue
- Treeline
- Paved Road
- Gravel Road
- Trail
- Parcel Boundary
- 1050 Topographic Contour (ft-msl)
- Stream
- Railroad

FIGURE 3-1

SOIL SAMPLE LOCATIONS
ELY COPPER MINE
VERSHIRE, VERMONT

Nobis Engineering, Inc.
18 Chenell Drive
Concord, NH
tel (603) 224-4182
fax (603) 224-2507
www.nobisengineering.com

N
W
E
S

Underground Mine Workings
Upper Waste Pile Area
Upper Waste Pile Area
Former Flotation Mill
Lower Waste Pile / Roast Bed Area
Ore Roast Bed Area
Smelter / Slag Area
Lower Waste Pile Area
Ely Brook
South Vershire Road
Schoolhouse Brook
POND 1
POND 2
POND 3
POND 4
POND 5
EB-1080M
SW-77
SW-78
SW-48
SW-79
SW-76
SW-48
SW-47
SW-75
SW-43
SW-46
SW-81
SED-55C
SED-57C
SW-80
SED-54C
SED-56C
SW-45
EM-POND2
EB-865M
SED-17C/ SW-17
SW-29
SED-53C
SD/SW-71
EM-POND3
EB-770M
SD-68
EM-POND4
SED-40C/SW-40
SW-82
SW-42
SD-70
EM-POND5
SD-69
SD-67
SW-41
SD-65
EBT2-245M/ SW-66
SW-83
EM-POND6
SED-51C/ SW-51
SD/SW-73
SW-30
SD/SW-72
EB-600M
SW-14
SD-64
SW-13
SW-32
SW-12
EB-515M
SD-63
SW-33
SW-36
SW-84
SD-62
SW-34
SW-39
SD-61
SW-35
SD-60
EB-190M
EB-90M
SW-09
EB-20M
1350
1300
1250
1200
1150
1100
1050
1000
950

LEDGEND

Sample Locations

- SW — Proposed Surface Water Sample Location
- SED — Proposed Sediment Sample Location
- SED/SW — Proposed Sediment & Surface Water Sample Location

Mining Materials

- Underground Mine Workings
- Waste Rock Areas
- Smelter Area
- Slag Pile Area

Drawn By: JDF	Checked By: AB
Filename: HHRA_EB_locs.mxd	
Date: 12/15/10	Revision No. 00

APPROXIMATE SCALE
140 70 0 140 280
Feet

- Shaft
- Adit
- Prospect Tunnel
- Drill Hole
- Smoke Flue
- Treeline
- Paved Road
- Gravel Road
- Trail
- Parcel Boundary
- 1050 Topographic Contour (ft-msl)
- Stream
- Railroad

FIGURE 3-2

ELY BROOK
SAMPLE LOCATIONS
ELY COPPER MINE
VERSHIRE, VERMONT

Nobis Engineering, Inc.
18 Chenell Drive
Concord, NH
tel (603) 224-4182
fax (603) 224-2507
www.nobisengineering.com

N
W
E
S

Former Flotation Mill

Ely Brook

Lower Waste Pile Area

Lower Waste Pile / Roast Bed Area

Ore Roast Bed Area

Smelter / Slag Area

SB-3670M (SW, SD, & Fish)

South Vershire Road

SB-3255M (SW)

SB-3245M

SB-3260M

SW-38

SD-59

SW-06

Schoolhouse Brook

SD-58

SB-3100M

SB-3125M (SW, SD, & Fish)

SW-04

SB-2860M

SB-2400M (SW, SD, & Fish)

LEDGEND

Sample Locations

SW
Proposed Surface Water Sample Location

SED
Proposed Sediment Sample Location

Fish
Fish Sample Location

Mining Materials

Underground Mine Workings
Waste Rock Areas
Smelter Area
Slag Pile Area

Shaft
Adit
Prospect Tunnel
Drill Hole

Smoke Flue
Treeline
Paved Road
Gravel Road
Trail

1050 Parcel Boundary
Topographic Contour (ft-msl)
Stream
Railroad

Drawn By: JDF | Checked By: AB
Filename: HHRA_SHB_locs.mxd
Date: 12/15/10 | Revision No. 00
APPROXIMATE SCALE
140 70 0 140 280
Feet

FIGURE 3-3
SCHOOLHOUSE BROOK
SAMPLE LOCATIONS
ELY COPPER MINE
VERSHIRE, VERMONT

Nobis Engineering, Inc.
18 Chenell Drive
Concord, NH
tel (603) 224-4182
fax (603) 224-2507
www.nobisengineering.com

Upper Waste Pile

Lower Waste Pile / Roast Bed

Smelter / Slag

Ely Brook

South Vershire Road

OR-24050M
(SW, SD, & Fish)

OR-23630M
(SW & Fish)

OR-23200M
(SW, SD, & Fish)

OR-22390M

OR-22320M

OR-20200M

OR-19560M

OR-11800M

N

W

E

S

LEDGEND

Sample Locations

SW
Proposed Surface Water Sample Location

SED
Proposed Sediment Sample Location

Fish
Fish Sample Location

Mining Materials

Underground Mine Workings
Waste Rock Areas
Smelter Area
Slag Pile Area

Shaft
Adit
Prospect Tunnel
Drill Hole

Smoke Flue
Treeline
Paved Road
Gravel Road
Trail

Parcel Boundary
1050 Topographic Contour (ft-msl)
Stream
Railroad

Drawn By: JDF | Checked By: AB
Filename: Ompo Sample Locs.mxd
Date: 12/15/10 | Revision No. 00

APPROXIMATE SCALE
1,350 675 0 1,350 2,700
Feet

FIGURE 3-4
OMPOMPANOOSUC RIVER
SAMPLE LOCATIONS
ELY COPPER MINE
VERSHIRE, VERMONT

Nobis Engineering, Inc.
18 Chenell Drive
Concord, NH
tel (603) 224-4182
fax (603) 224-2507
www.nobisengineering.com

MW-07A,C

BOM-01

Underground Mine Workings

MW-19A,C,D

MW-18A

Upper Waste Pile Area

Upper Waste Pile Area

MW-06A,C

MW-09A,C

BOM-03

MW-05A,B,C

MW-13A

POND 1

POND 2

POND 4

MW-12C

POND 3

MW-08A

POND 5

MW-17A

MW-22A

Former Flotation Mill

Ely Brook

MW-21A,C

Lower Waste Pile Area

Lower Waste Pile / Roast Bed Area

MW-04A,C

MW-15A

MW-20A,C,D

Ore Roast Bed Area

MW-23A

Smelter / Slag Area

MW-03C

MW-16A

MW-14A,C,D

MW-10B,C

MW-11A,C

South Vershire Road

MW-02A,B,C

Schoolhouse Brook

MW-01A,B,C

1350 1300 1250 1200 1150 1100 1050 1000 950

N W E S

APPROXIMATE SCALE
140 70 0 140 280 Feet

Shaft
Adit
Prospect Tunnel
Drill Hole

Smoke Flue
Treeline
Paved Road
Gravel Road
Trail

Parcel Boundary
1050 Topographic Contour (ft-msl)
Stream
Railroad

LEGEND

Sample Locations

MW Monitoring Well Location
A = Shallow Overburden
B = Deep Overburden
C = Shallow Bedrock
D = Deep Bedrock

Mining Materials

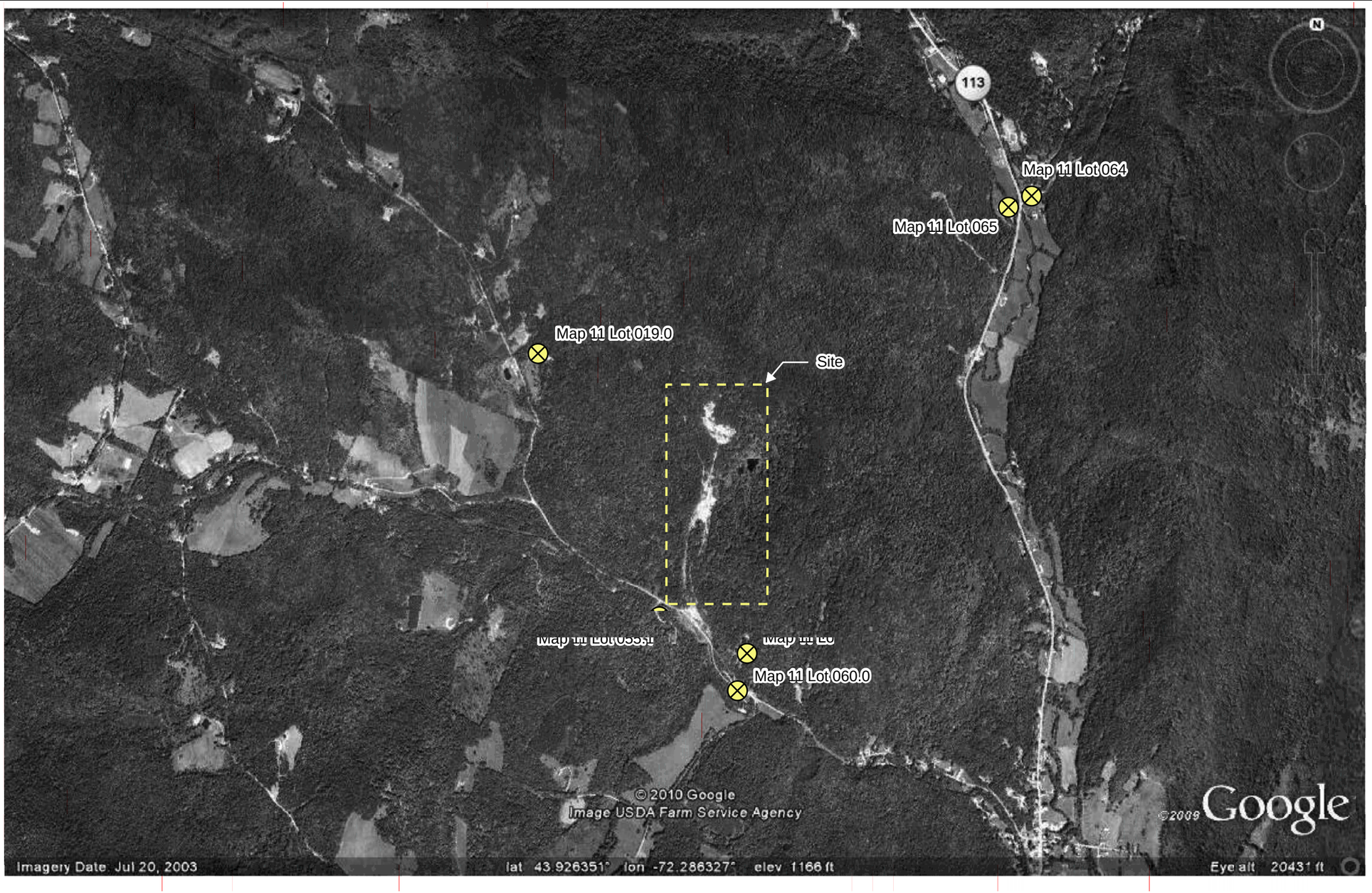
- Underground Mine Workings
- Waste Rock Areas
- Smelter Area
- Slag Pile Area

Drawn By: JDF	Checked By: AB
Filename: HHRA_MW_locs.mxd	
Date: 12/15/10	Revision No. 00

FIGURE 3-5

GROUNDWATER SAMPLE LOCATIONS
ELY COPPER MINE
VERSHIRE, VERMONT

Nobis Engineering, Inc.
18 Chenell Drive
Concord, NH
tel (603) 224-4182
fax (603) 224-2507
www.nobisengineering.com



N

Legend

Residential Well Sample Location

APPROXIMATE SCALE

2,170 1,085 0 2,170 Feet

Notes:

1. This map is based on a Google Earth image.

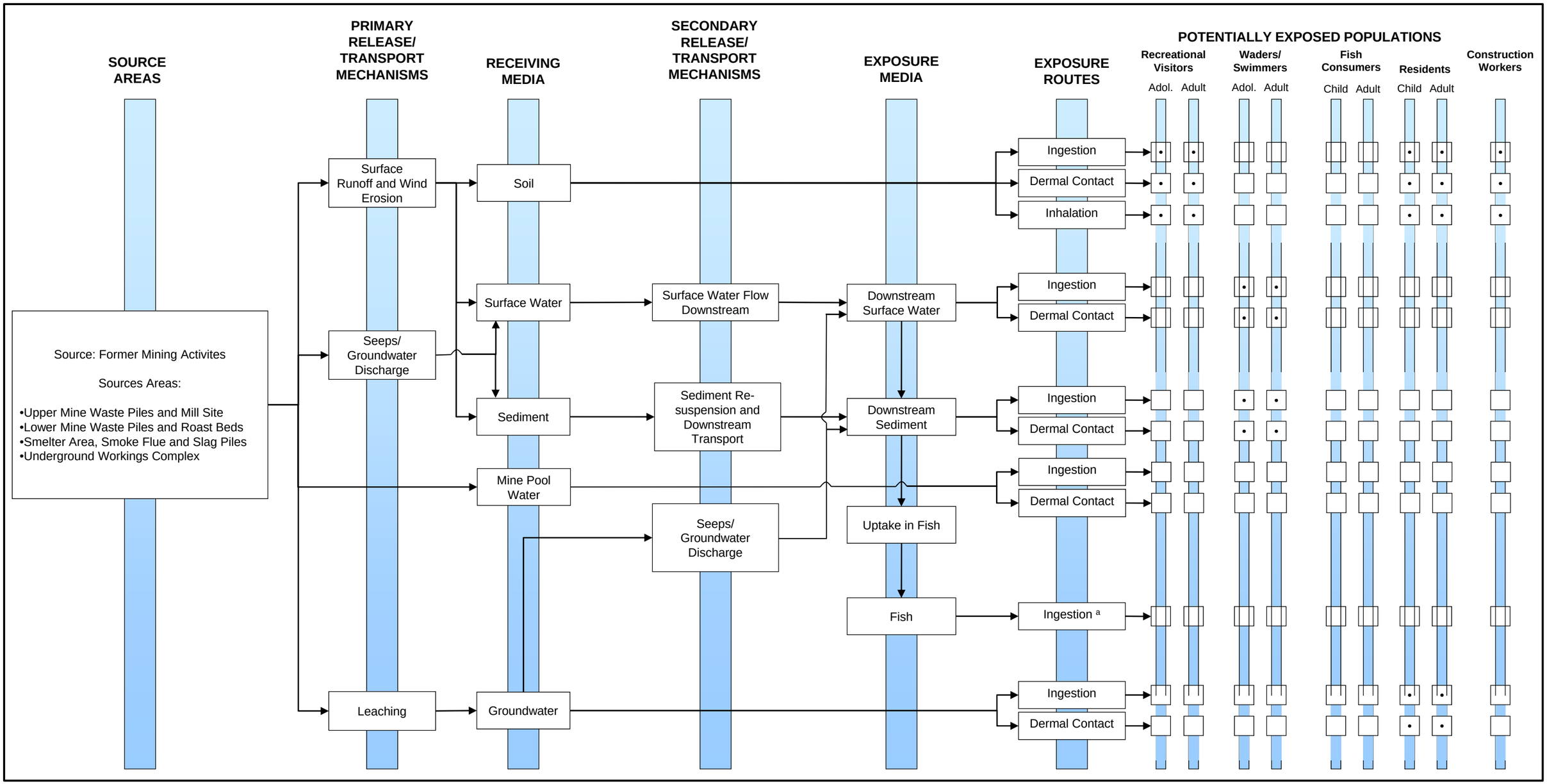
Nobis Engineering, Inc.
585 Middlesex Street
Lowell, MA 01851
(978) 683-0891
www.nobisengineering.com

FIGURE 3-6

RESIDENTIAL WELL SAMPLE LOCATIONS
ELY COPPER MINE
VERSHIRE, VERMONT

PREPARED BY: JF	CHECKED BY: AB
PROJECT NO. 80024	DATE: 12/15/10 Rev 00

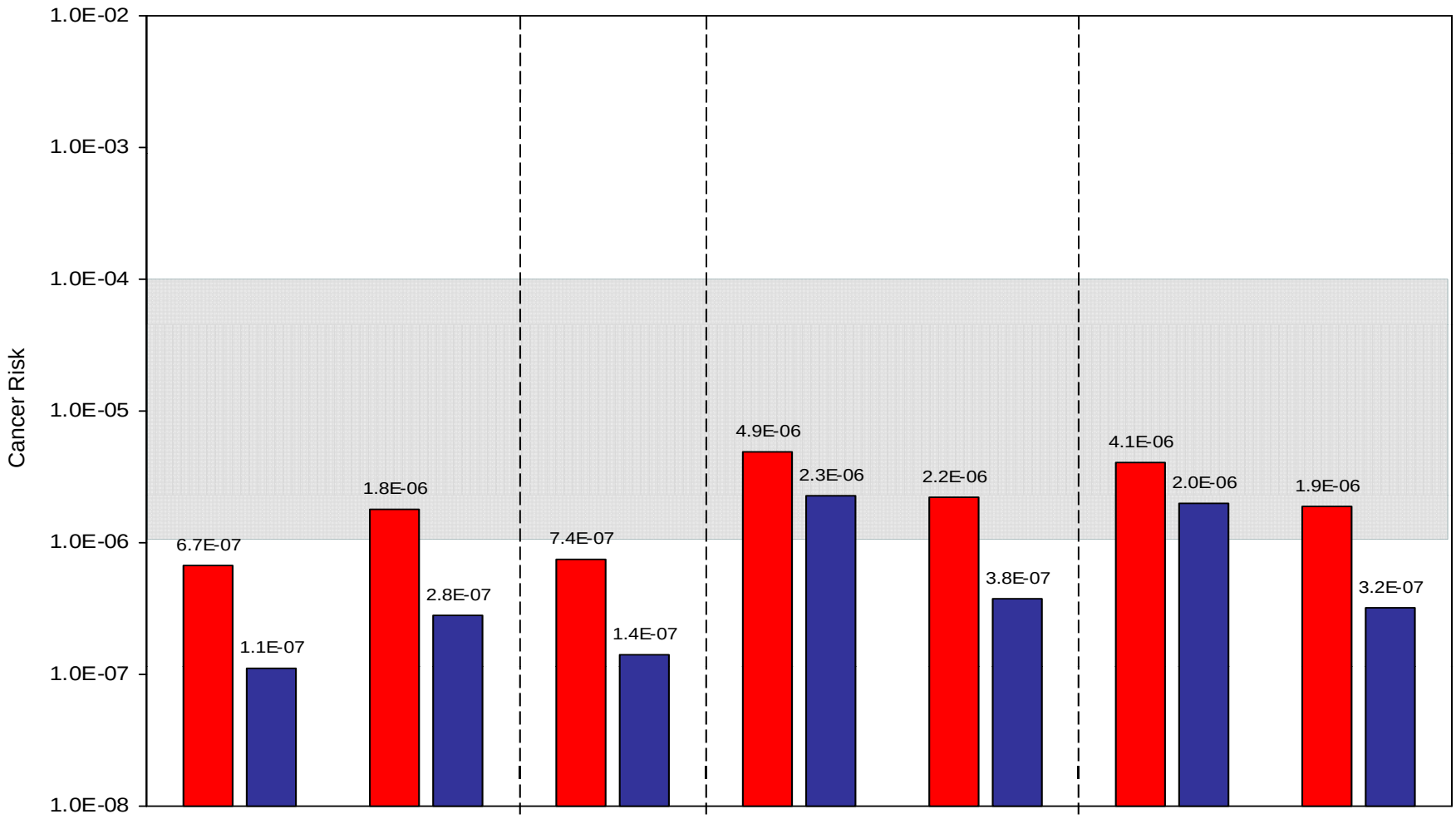
FIGURE 7-1
HUMAN HEALTH RISK ASSESSMENT - CONCEPTUAL SITE MODEL
ELY COPPER MINE SUPERFUND SITE



[a] The ingestion of fish pathway was eliminated from consideration in the HHRA in the COPC selection process (see Section 5.5).

• = Complete exposure pathway

= Incomplete exposure pathway



Legend:

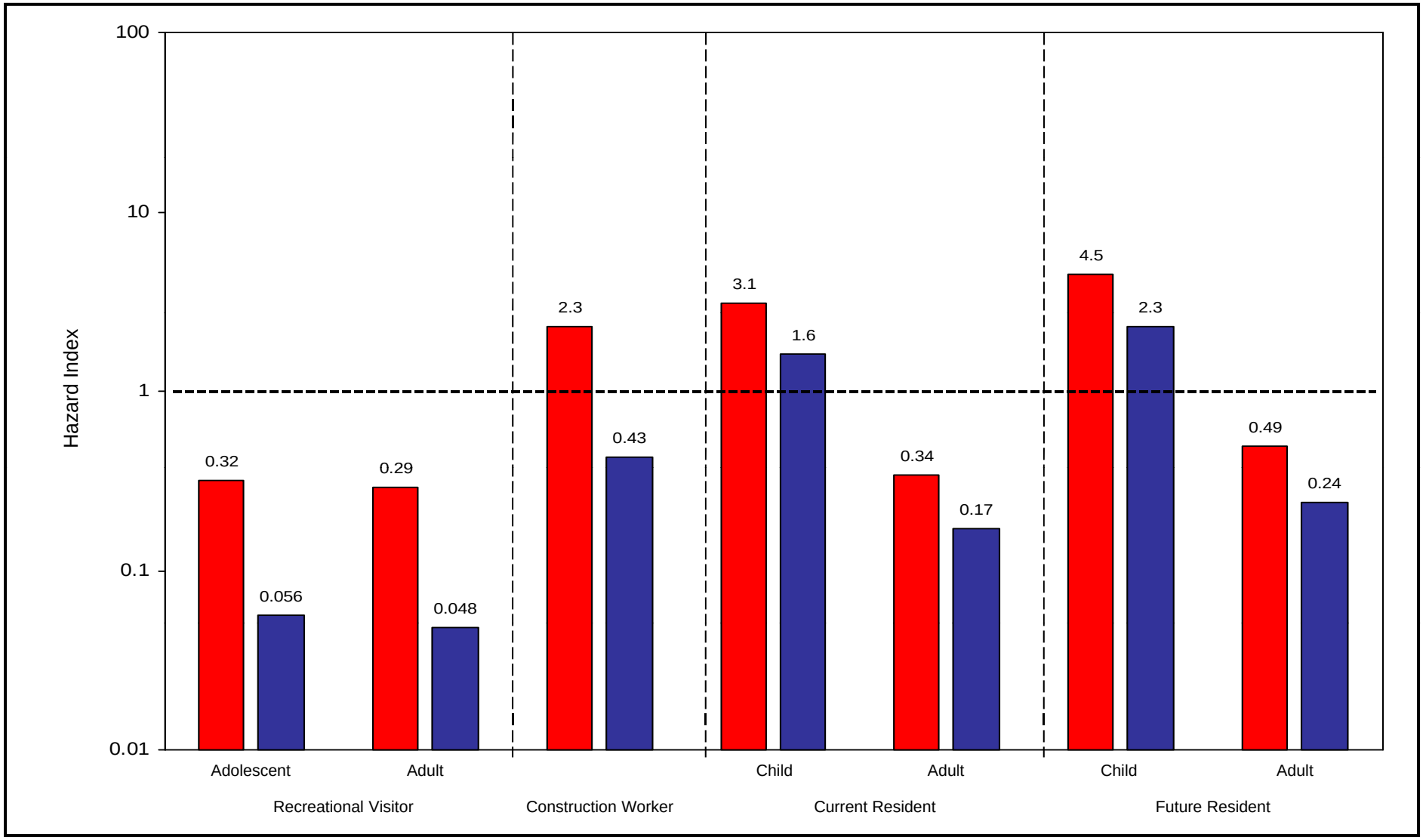
= RME cancer risk.

= CTE cancer risk.

Gray shaded area represents EPA's cancer risk range (1E-06 to 1E-04).

FIGURE 8-1
SUMMARY OF CANCER RISKS FROM SOIL EXPOSURE

ELY COPPER MINE SUPERFUND SITE



Legend:

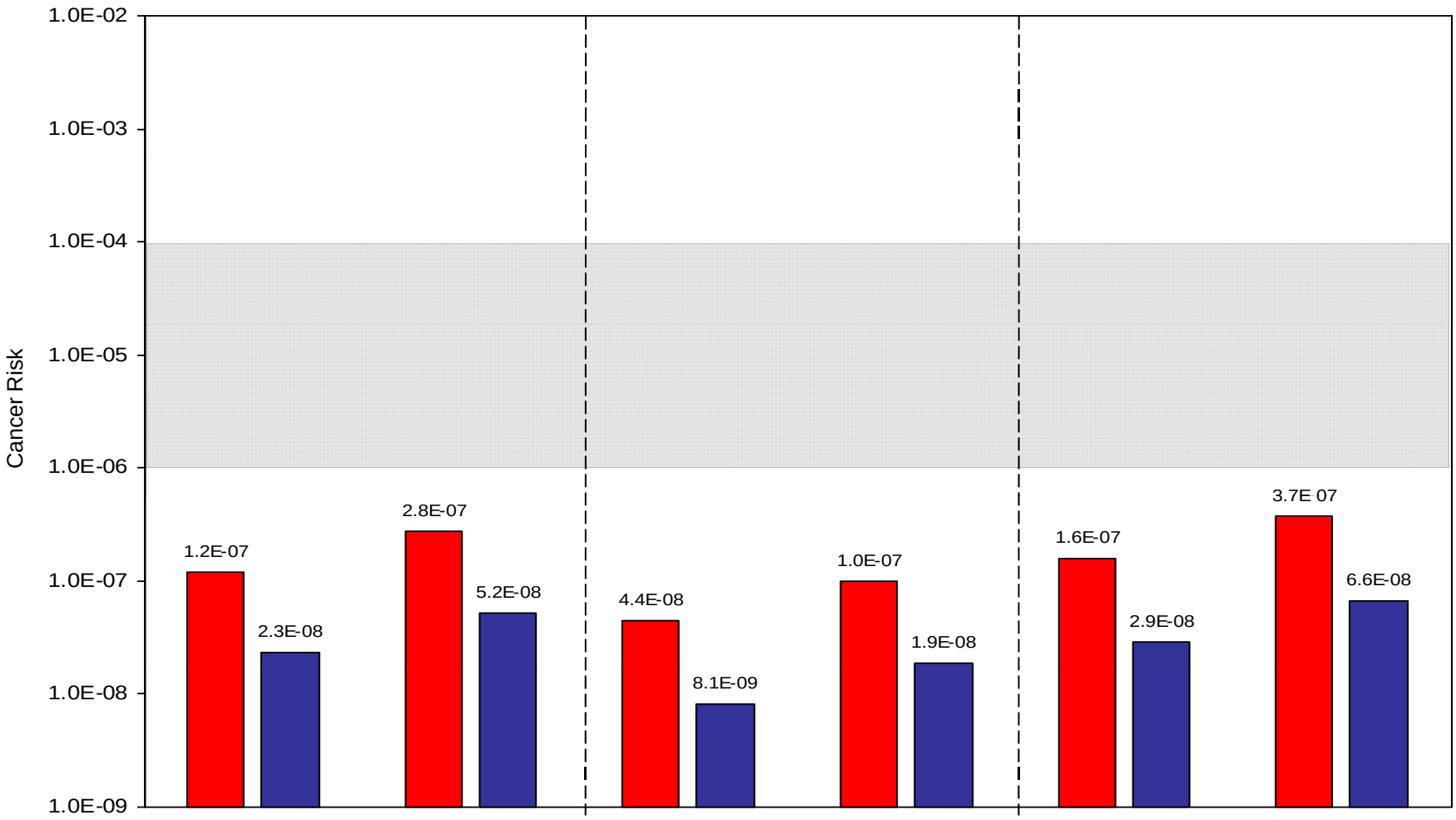
= RME hazard index.

= CTE hazard index.

* Horizontal line represents the noncancer benchmark of one.

FIGURE 8-2
SUMMARY OF HAZARD INDICES FROM SOIL EXPOSURE

ELY COPPER MINE SUPERFUND SITE



Legend:

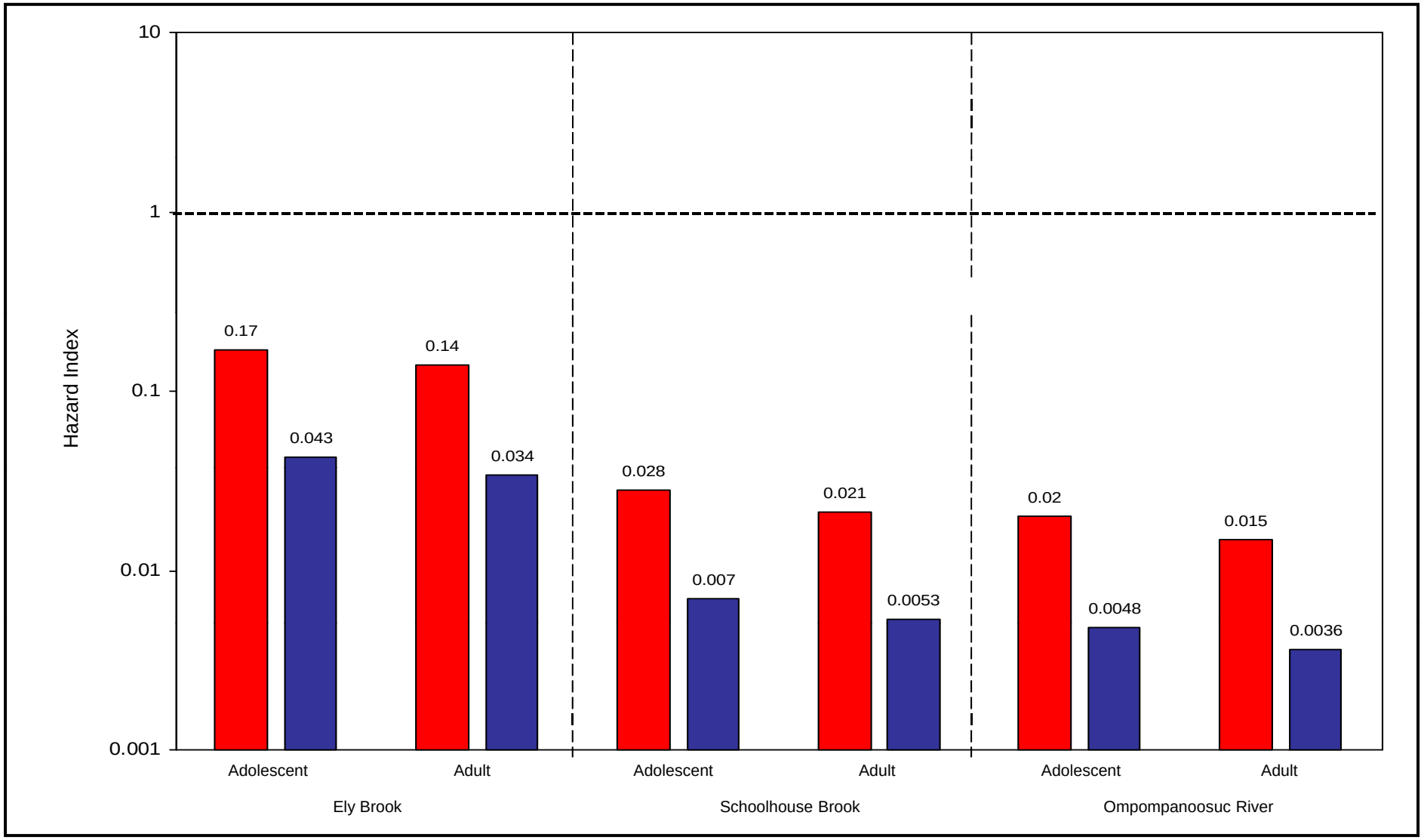
= RME cancer risk.

= CTE cancer risk.

Gray shaded area represents EPA's cancer risk range (1E-06 to 1E-04).

FIGURE 8-3
SUMMARY OF CANCER RISKS FROM SEDIMENT AND SURFACE WATER EXPOSURE

ELY COPPER MINE SUPERFUND SITE



Legend:

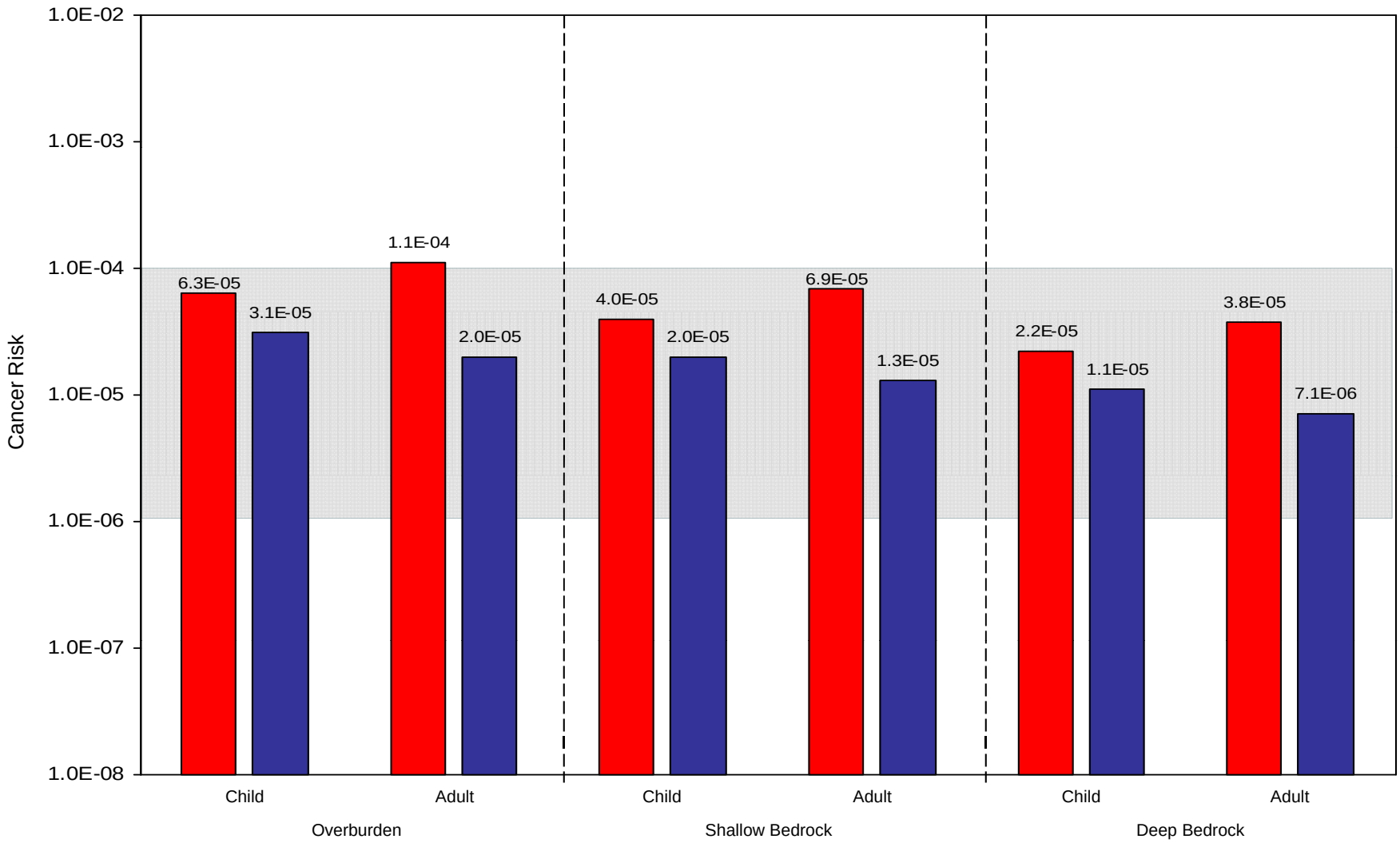
= RME hazard index.

= CTE hazard index.

* Horizontal line represents the noncancer benchmark of one.

FIGURE 8-4
SUMMARY OF HAZARD INDICES FROM SEDIMENT AND SURFACE WATER EXPOSURE

ELY COPPER MINE SUPERFUND SITE



Legend:

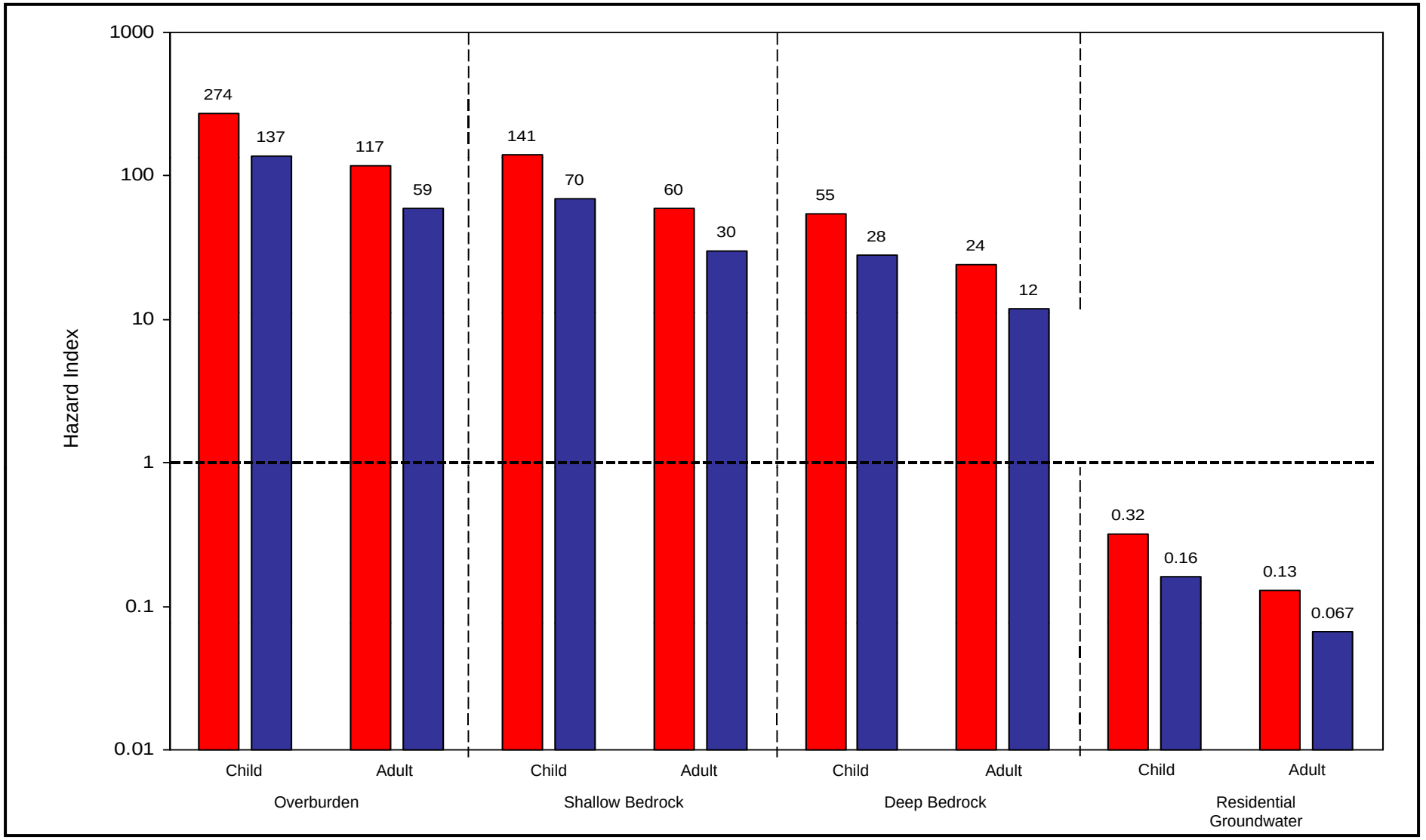
= RME cancer risk.

= CTE cancer risk.

Gray shaded area represents EPA's cancer risk range (1E-06 to 1E-04).

FIGURE 8-5
SUMMARY OF CANCER RISKS FROM GROUNDWATER EXPOSURE

ELY COPPER MINE SUPERFUND SITE



Legend:

= RME hazard index.

= CTE hazard index.

* Horizontal line represents the noncancer benchmark of one.

FIGURE 8-6
SUMMARY OF HAZARD INDICES FROM GROUNDWATER EXPOSURE

ELY COPPER MINE SUPERFUND SITE

APPENDIX A

APPENDIX A

Comparison of Laboratory Results and XRF Results

Nobis conducted extensive field sampling investigations in 2009 in support of the current RI/FS. Soil sampling was conducted during several phases of Site investigation activities including surface soil transects for x-ray fluorescence (XRF) field screening and laboratory analysis to assess the limits of waste areas. The XRF screening focused on five metals: copper, iron, lead, selenium, and zinc. Approximately 350 samples were analyzed using XRF techniques, of these, 45 were also analyzed at an off-site fixed laboratory (i.e., there were a total of 45 paired laboratory and XRF samples). The objective of this appendix is to evaluate the XRF data and compare it with the paired samples that were submitted for laboratory analysis and use these findings to determine the potential impact the non-paired XRF data might have on the HHRA results if it were used to estimate exposure point concentrations.

Table A-1 presents the results of the paired XRF and laboratory samples. This table also shows the percent difference between the laboratory and XRF results for each paired sample location. Table A-2 summarizes the results of the comparisons. In general, there was relatively good agreement between the laboratory results and the XRF results. The exception to this was for selenium that had a low frequency of detection (12 out of 45) for the XRF samples. As a result of this low detection frequency, it is difficult to make quantitative comparisons between the paired laboratory and XRF results. Therefore, selenium was not considered for evaluation. For the limited paired data with detections of selenium, the XRF data were consistently greater than the laboratory results.

On average, the XRF results for copper were about 30 percent less than the laboratory results and the laboratory and XRF results for lead were essentially equal. The XRF results were slightly greater than the laboratory results for zinc (6 percent), again on an average basis. The XRF results were 23 percent greater than the laboratory results for iron. There was a significant range of differences for copper, iron, lead, and zinc as shown on Table A-2. For example, the copper comparisons ranged from the XRF results about 250 percent less than the laboratory results to XRF results 68 percent greater than the laboratory results. A similar range of differences was observed for the other metals. For each metal, the magnitude of percent difference for the XRF results that were less than the laboratory results was greater than when XRF results were greater than the laboratory results.

Conclusion

The results of the paired sample comparisons indicate that there is relatively good comparability between the laboratory and XRF results. The few anomalies observed in the percent difference comparison are likely a result of the analytical variability associated with both methods. As a result of these findings, there is little concern that the non-paired XRF samples, if included in exposure analysis, would significantly impact the exposure concentrations used in the HHRA.

APPENDIX A

Table A-1
Comparison of Paired Laboratory Results and XRF Results
Ely Copper Mine Superfund Site
Vershire, Vermont
Page 1 of 4

Sample Location:	SS-01	SS-01	SS-02	SS-02	SS-03	SS-03	SS-04	SS-04	SS-05	SS-05	SS-06	SS-06
Station ID:	SS-01X-090209AX	SS-01X-090209BX	SS-02X-090209AX	SS-02X-090209BX	SS-03X-090209AX	SS-03X-090209BX	SS-04X-090109AX	SS-04X-090209BX	SS-05X-090209AX	SS-05X-090209BX	SS-06X-090209AX	SS-06X-090209BX
Sample Date:	9/2/2009	9/2/2009	9/2/2009	9/2/2009	9/2/2009	9/2/2009	9/1/2009	9/2/2009	9/2/2009	9/2/2009	9/2/2009	9/2/2009
Laboratory Results (mg/kg)												
Copper	362	172	494	418	188	283	122	62.1	31.8	28.8	44.7	33.2
Iron	31000	17100	34000	27700	25300	29600	21000	29500	23500	29000	23700	28800
Lead	295	6.6	29.7	11.6	17.9	10.4	15.3	9.9	17.8	9	37.1	11.8
Selenium	3.3	1.6	4.2	1.1	0.57	1.2	0.62	0.36	0.97	0.5	0.95	0.43
Zinc	108	65	73.5	58.6	55.8	72.2	47.1	64.2	41.6	62.5	56.8	75.7
XRF Results (mg/kg)												
Copper	275	138	423	332	167	217	107	47	27	30	33	23
Iron	39188	26306	52941	35901	39351	35673	31479	36873	35523	37525	31297	36984
Lead	168	14	28	12	17	12	22	13	22	15	31	10
Selenium	3 U	3 U	3 U	3 U	3 U	3 U	3 U	2 U	3 U	3 U	3 U	3 U
Zinc	107	62	87	54	70	73	49	58	47	68	65	101
Percent Difference Between Laboratory Result and XRF Result												
Copper	-32%	-25%	-17%	-26%	-13%	-30%	-14%	-32%	-18%	4%	-35%	-44%
Iron	21%	35%	36%	23%	36%	17%	33%	20%	34%	23%	24%	22%
Lead	-76%	53%	-6%	3%	-5%	13%	30%	24%	19%	40%	-20%	-18%
Selenium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Zinc	-1%	-5%	16%	-9%	20%	1%	4%	-11%	11%	8%	13%	25%

Notes:

NA = Not applicable, the percent difference was not calculated because the XRF results was not detected.

* If the percent difference is a negative value, the XRF results was less than the laboratory result.

APPENDIX A

Table A-1
Comparison of Paired Laboratory Results and XRF Results
Ely Copper Mine Superfund Site
Vershire, Vermont
Page 2 of 4

Sample Location:	SS-07	SS-07	SS-10	SS-11	SS-12	SS-16	SS-20	SS-27	SS-30	SS-30	TA	TA
Station ID:	SS-07X-090209AX	SS-07X-090209BX	SS-10X-090309AX	SS-11X-090309AX	SS-12X-090309AX	SS-16X-090309AX	SS-20X-083109BX	SS-27X-082609BX	SS-30X-083109AD	SS-30X-083109AX	TA-04X-082409AX	TA-14X-082409AX
Sample Date:	9/2/2009	9/2/2009	9/3/2009	9/3/2009	9/3/2009	9/3/2009	8/31/2009	8/26/2009	8/31/2009	8/31/2009	8/24/2009	8/24/2009
Laboratory Results (mg/kg)												
Copper	26.4	23.5	605	897	149	80.1	192	1180	1390	1400	365	632
Iron	22800	31200	20800	78700	9760	26300	33500	36800	181000	166000	20900	23300
Lead	34.9	8.8	93.9	74.9	19.5	18.7	12.2	10.2	33.2	36.2	27.6	116
Selenium	0.66	0.48	2	3.3	0.8	0.47	1	2	16.4	15.6	1	1.4
Zinc	55.6	72.4	129	164	37.2	67.5	65.4	85.7	114	85	113	136
XRF Results (mg/kg)												
Copper	27	27	618	261	248	55	172	1347	403	403	309	483
Iron	35595	44193	34692	39269	25839	29945	39607	46502	100000	100000	26114	29374
Lead	34	13	96	35	36	17	13	19	72	72	27	88
Selenium	3 U	3 U	3 U	3 U	3	3 U	2 U	4	28	28	2 U	3 U
Zinc	66	79	170	77	69	68	54	91	72	72	83	104
Percent Difference Between Laboratory Result and XRF Result												
Copper	2%	13%	2%	-244%	40%	-46%	-12%	12%	-245%	-247%	-18%	-31%
Iron	36%	29%	40%	-100%	62%	12%	15%	21%	-81%	-66%	20%	21%
Lead	-3%	32%	2%	-114%	46%	-10%	6%	46%	54%	50%	-2%	-32%
Selenium	NA	NA	NA	NA	73%	NA	NA	50%	41%	44%	NA	NA
Zinc	16%	8%	24%	-113%	46%	1%	-21%	6%	-58%	-18%	-36%	-31%

Notes:

NA = Not applicable, the percent differ

* If the percent difference is a negative

APPENDIX A

Table A-1
Comparison of Paired Laboratory Results and XRF Results
Ely Copper Mine Superfund Site
Vershire, Vermont

Sample Location:	TC	TD	TD	TE	TE	TE	TF	TF	TF	TF	TG	TH
Station ID:	TC-03X-082609AX	TD-03X-082809-AX	TD-05X-082809-DX	TE-09X-090109AX	TE-12X-090109BX	TE-15X-090109CX	TF-02X-090109AX	TF-11X-0-90909AX	TF-13X-0-90909BX	TF-17X-090109AX	TG-02X-082409AX	TH-01X-082509AX
Sample Date:	8/26/2009	8/28/2009	8/28/2009	9/1/2009	9/1/2009	9/2/2009	9/1/2009	9/2/2009	9/2/2009	9/1/2009	8/24/2009	8/25/2009
Laboratory Results (mg/kg)												
Copper	19.3	420	995	469	185	268	1350	5610	7850	81.7	182	1920
Iron	21300	18800	11800	78200	21700	29600	89000	135000	30700	25000	19800	96600
Lead	16.7	9.5	118	32.3	3.4	9.2	29.5	46.1	4.8	9.6	7.7	29.5
Selenium	0.63	3.8	0.58	22.8	1.2	2.2	17.6	33.2	3.7	0.64	0.85	18.2
Zinc	43.8	70.5	49.2	69.3	51.9	116	90.2	140	107	53.1	30.3	107
XRF Results (mg/kg)												
Copper	19	1318	422	336	176	133	1099	4216	18554	76	180	1045
Iron	35477	100000	36456	98293	43811	31894	100000	100000	77677	47969	62422	99183
Lead	27	57	13	56	10	7	47	72	26	12	12	44
Selenium	3 U	34	3 U	25	4	3 U	31	43	13	3 U	3 U	21
Zinc	54	135	83	174	64	93	227	129	287	82	86	90
Percent Difference Between Laboratory Result and XRF Result												
Copper	-2%	68%	-136%	-40%	-5%	-102%	-23%	-33%	58%	-8%	-1%	-84%
Iron	40%	81%	68%	20%	50%	7%	11%	-35%	60%	48%	68%	3%
Lead	38%	83%	-808%	42%	66%	-31%	37%	36%	82%	20%	36%	33%
Selenium	NA	89%	NA	9%	70%	NA	43%	23%	72%	NA	NA	13%
Zinc	19%	48%	41%	60%	19%	-25%	60%	-9%	63%	35%	65%	-19%

Notes:

NA = Not applicable, the percent differ

* If the percent difference is a negative

APPENDIX A

Table A-1
Comparison of Paired Laboratory Results and XRF Results
Ely Copper Mine Superfund Site
Vershire, Vermont

Sample Location:	TI	TK	TL	TN	TN	TO	TP	TQ	TQ
Station ID:	TI-03X-082509AX	TK-01X-082509AX	TL-04X-082509AX	TN-02X-082509AX	TN-02X-082509AD	TO-05X-082709AX	TP-10X-082709AX	TQ-06X-082609AX	TQ-16X-082609AX
Sample Date:	8/25/2009	8/25/2009	8/25/2009	8/25/2009	8/25/2009	8/27/2009	8/27/2009	8/26/2009	8/26/2009
Laboratory Results (mg/kg)									
Copper	61.8	110	68.6	61	78.3	1100	135	288	109
Iron	23600	17400	20800	20300	25300	74000	24900	73700	26100
Lead	22.7	26.8	15	12.1	14.6	17.9	15.9	6.1	8.5
Selenium	0.58	0.42	0.69	0.62	0.68	7.7	0.76	2.7	0.46 J
Zinc	77.5	75.1	55.2	58.6	72	85.7	76.1	38	76
XRF Results (mg/kg)									
Copper	77	247	66	56	56	1080	121	227	93
Iron	36291	45528	30601	29697	29697	99912	34966	94500	31648
Lead	28	27	15	13	13	37	22	15	13
Selenium	3 U	3 U	3 U	3 U	3 U	14	3 U	3 U	3 U
Zinc	71	92	64	69	69	84	80	31	73
Percent Difference Between Laboratory Result and XRF Result									
Copper	20%	55%	-4%	-9%	-40%	-2%	-12%	-27%	-17%
Iron	35%	62%	32%	32%	15%	26%	29%	22%	18%
Lead	19%	1%	0%	7%	-12%	52%	28%	59%	35%
Selenium	NA	NA	NA	NA	NA	45%	NA	NA	NA
Zinc	-9%	18%	14%	15%	-4%	-2%	5%	-23%	-4%

Notes:

NA = Not applicable, the percent differ

* If the percent difference is a negative

APPENDIX A

Table A-2
Summary of the Comparison of the Paired Laboratory Results and XRF Results
Ely Copper Mine Superfund Site
Vershire, Vermont

Metal	Average Difference	Notes	Minimum Difference	Maximum Difference
Copper	-31%	XRF less than lab	-247%	68%
Iron	23%	XRF greater than lab	-100%	81%
Lead	-1%	XRF and lab essentially equal	-808%	83%
Selenium	Comparisons not summarized			
Zinc	6%	XRF slightly greater than lab	-113%	65%

APPENDIX B

10-23-2006

Provisional Peer Reviewed Toxicity Values for

Aluminum
(CASRN 7429-90-5)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
i.v.	intravenous
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR ALUMINUM (CASRN 7429-90-5)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).

2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.

3. Other (peer-reviewed) toxicity values, including:

   - Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
   - California Environmental Protection Agency (CalEPA) values, and
   - EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions or the EPA Headquarters Superfund Program sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

This document has passed the STSC quality review and peer review evaluation indicating that the quality is consistent with the SOPs and standards of the STSC and is suitable for use by registered users of the PPRTV system.

INTRODUCTION

Verified toxicity values for aluminum (Al) and its compounds are unavailable on IRIS or HEAST (U.S. EPA, 2006, 1997), except for a chronic oral RfD of 4E-4 mg/kg-day for aluminum phosphide. However, occupational guidelines and standards have been established for a number of chemical and physical forms of Al, including, from ACGIH, 8-hour TWA-TLVs of 10 mg/m^3 for the compound as a metal dust or oxide, 5 mg/m^3 as "pyro" powders or welding fumes, and 2 mg/m^3 for soluble salts or organic forms of the metal (ACGIH, 1998). From NIOSH, 10-hour TWA-RELs of 10 mg/m^3 are specified for "total" Al dust versus 5 mg/m^3 for the respirable portion (NIOSH, 1994). NIOSH covers all other forms of the metal by identical values to those specified by ACGIH (ACGIH, 1998). OSHA PELs for Al include an 8-hour TWA value of 15 mg/m^3 for "total" metal dust, versus 5 mg/m^3 for the respirable portion (NIOSH, 1994). The U.S. EPA's CARA list (U.S. EPA, 1994) cites a HEA for Al (U.S. EPA, 1987), and ATSDR has updated its toxicological profile of the element (ATSDR, 1998).

The U.S. FDA (2000) has specified a maximum aluminum concentration of 25 mcg/L in large-volume parenterals (LVP) used in total parenteral nutrition (TPN). The FDA regulation applies to all LVPs used in TPN, including but not limited to parenteral amino acid solutions, highly concentrated dextrose solutions, parenteral lipid emulsions, sodium chloride and electrolyte solutions, and sterile water for injection.

Research papers pertinent to the potential toxicological and carcinogenic effects of Al were sought through computer searches of the HSDB, RTECS, MEDLINE and TOXLINE (and its subfiles) databases, covering the time period 1995-1999. The literature searches were conducted in June, 1999.

REVIEW OF PERTINENT DATA

The review by Stokinger (1981) gives an account of Al as an all-pervasive component of products that are central to the daily lives of most Americans. For example, the metal is a crucial part of manufactured products for the building, automobile and container industries, while Al as powder or flake is a component in a number of consumer products, such as paints, fireworks, etc. Al complexes and minerals are used in the brewing and paper industries, and as coagulants for water purification. Aluminum oxide finds application in abrasives, as a catalyst or absorbent, and as a component in fillers. Aluminum chloride is included in cosmetic formulations such as deodorants.

Human exposure to Al arises principally from food and water, through its widespread use in food additives, packaging and cooking utensils and Al-containing medications, particularly antacid, buffered aspirin, anti-ulcer and anti-diarrheal formulations (Marquis, 1989; Lione, 1985). Pennington and Schoen (1995) estimated daily Al intakes of 0.1-0.3 mg/kg-day for infants and children 6 months-6 years of age and 0.1-0.18 mg/kg-day for older children and adults, based on the FDA Total Diet Study (1993) and the U.S. Department of Agriculture Nationwide Food Consumption Survey (1987-1988). These data are in broad agreement with those of Wilhelm et al. (1995) who reported the dietary intake of Al in German children (living in the Duisberg area) as ranging from 0.008 to 0.11 mg Al/kg-day. In addition, these values are consistent with a range of 1-20 mg/day (0.014-0.3 mg/kg -day) for normal oral daily Al intake from food and water reported by other investigators (Ganrot, 1986; Iyengar et al., 1987; Wilhelm et al., 1990). However, users of Al-containing medications can ingest much larger amounts of the element, possibly as high as 840-5000 mg/day (12-71 mg/kg-day) from antacids, 126-728 mg/day (1.8-10.4 mg/kg-day) from buffered aspirins and 828 mg/day (11.8 mg/kg-day) from anti-ulcer compounds when taken at recommended dosages (Lione, 1985).

Toxicokinetics of Aluminum

There is a large amount of information available on the absorption, transfer from tissue to tissue and elimination of Al from the body, including data that have been amassed from studies on either human volunteers or laboratory animals. In general, the chemical appears to be poorly absorbed from the gastrointestinal tract, though the portion of the load that is retained will vary depending on the concentration, the chemical species administered, the fasting or fed state of the

host, gastrointestinal pH, animal model, etc. For example, Yokel and McNamara (1988) administered single oral doses of a number of Al compounds (both water soluble and insoluble) to New Zealand white rabbits and obtained absorbed proportions of the load ranging from 0.27% to 27%. Fractional uptake of Al in humans under normal conditions (i.e., with no intake of large quantities of Al from medicine) was estimated to be 0.1-0.3% assuming an intake of 20 mg Al/day (0.3 mg Al/kg-day) and urinary excretion of 20-50 µg Al/day (0.3-0.7 µg Al/kg-day) (Ganrot, 1986). However, little information is available on the actual mechanism by which the element and its compounds are transported across the brush border. (Wilhelm et al., 1990; Lione, 1985).

Although the overall extent of Al absorption is poor following oral exposure, there may be significant intake of the compound by those taking large amounts of Al compounds in patented remedies. As stated, absorption of Al is influenced by gastrointestinal conditions and content because Al can form various complexes with different solubilities and oxidation states depending on pH and interactions with dietary constituents. At low pH (3-5) in aqueous solutions, the soluble (ionic) forms of the Al prevail (Al^{3+}); at high pH (>8), Al in the form of soluble aluminum oxide is present; and at pH 5-8, the element is predominantly in the form of aluminum hydroxide, which is insoluble (van der Voet and de Wolff, 1986; Wilhelm et al., 1990). Ingested constituents that can influence absorption by forming complexes with Al include phosphate, fluoride, calcium, citrate and lactate. For example, Al is used to bind dietary phosphorus and decrease its absorption as a control for hyperphosphatemia, and citrate and lactate are complexing agents that can significantly increase Al absorption (Slanina et al., 1984, 1985, 1986; Partridge et al., 1989; Domingo et al., 1991; Ittel et al., 1991; Lione, 1985; Wihelm et al., 1990).

A number of recent reports of studies on the gastrointestinal absorption of Al have examined the influence of organic anions such as citrate. In general, the presence of such components appears to enhance the absorption of Al, within narrow limits. For example, Deng et al. (1998) administered a single oral dose of either distilled water, 2 mmoles/L aluminum chloride or 2 mmoles/L aluminum chloride plus 2 mmoles/L sodium citrate to six male Wistar rats/group. Animals were bled at 1, 2 and 4 hours after dosing, then terminated after 6 hours. Inductively coupled plasma (ICP) was used to measure Al concentrations in blood, bone (tibia), kidney, liver and the intestinal wall. Irrespective of treatment, the appearance of Al in the blood of dosed groups peaked after 1 hour, with the concentration of the element at higher levels in those animals receiving citrate in addition to aluminum chloride. In those animals receiving aluminum chloride alone, significant tissue concentrations of the element were restricted to the gastrointestinal wall. Those receiving citrate displayed measurable quantities of the element in several of the other monitored tissues, including bone.

Sutherland and Greger (1998a) used a similar dosing regimen to examine the kinetics of absorption and elimination of Al in male Sprague-Dawley rats that had received a single oral dose of 0, 0.25, 0.5 or 1 mmoles/L/kg body weight aluminum lactate in 1 mL of 16% citrate. Concentrations of Al in serum, liver, kidney or bone (tibia) were measured at various post-dosing time intervals up to 6 hours. Depending on the dose, absorption factors for Al of up to 4.2% of the administered dose were observed, with the greater proportion retained in bone. The authors reported a slower rate of absorption in those animals receiving Al at the higher doses, an

observation potentially indicating reduced gut motility and/or saturation of the transcellular absorption processes at the higher concentrations. Aluminum deposited in kidney and bone appeared to turn-over at a slower rate than in the liver.

The influence of citrate on the gastrointestinal absorption of Al in man was examined directly by Taylor et al. (1998) who administered a drink containing Al and citrate to three volunteers. Aluminum and citrate concentrations were monitored in serial blood and urine samples for up to 24 hours. The kinetics of citrate and Al differed markedly, the former peaking in plasma after 32 minutes, versus 87 minutes for Al. This suggests that Al probably does not cross the gastrointestinal barrier as the citrate. Furthermore, the authors reported that the overall extent of Al absorption had probably not exceeded 1% in their experiment, a finding that contrasts with the higher values reported by Sutherland and Greger (1998a) in Sprague-Dawley rats and by Deng et al. (1998) in Wistar rats.

As discussed in a report by Glynn et al. (1999), gastrointestinal absorption of Al from aqueous media will be almost impossible to predict, because of the likelihood that the element will become absorbed to food particles in the intestinal lumen. Accordingly, depending on the dose, mode of delivery and caloric state of the experimental animal (fed/fasted), significant amounts of aqueous forms of Al will be absorbed only when available binding sites on food have become saturated. This presents an inherently complex overall picture of the element's absorption since, additionally, the normal dietary content of Al will be substantial. Thus, it may be assumed that some sequestered Al will be absorbed along with non-sequestered water soluble forms of the element, while the rest will be retained within the gastrointestinal tract.

Sutherland and Greger (1998b) used their aluminum lactate in 16% citrate dosing regimen to examine the comparative importance of biliary versus urinary excretion of Al. Five to seven male Sprague-Dawley rats/group who had previously received an implanted bile cannula were treated by gavage. Another similarly-treated cohort of five animals/group were housed in metabolic cages immediately after dosing to provide 0- to 3-hour and 3- to 6-hour urine specimens. At termination, all animals were sacrificed and exsanguinated, and tissue, bile and urine samples were measured by graphite furnace atomic absorption spectroscopy. Among the key findings to emerge from this study was the incremental appearance of Al in bile as early as 15 minutes after dosing. However, overall amounts of Al were greater in the 3-hour urine samples than those that had accumulated in bile samples collected within a similar time frame. The fact that control rats excreted 3 times more Al in bile than in urine during the first 3 hours after dosing led the authors to conclude that, at low exposure to Al (in controls receiving Al solely from food), the liver is capable of excreting the element to the bile, a mechanism that becomes saturated as the level of Al administration becomes increased. Thereafter, urinary excretion becomes the primary route of elimination in circumstances of Al overload.

Aluminum can also be absorbed by inhalation as indicated by age-related deposition in the lungs of the general population and exposure-related increased blood and urine concentrations in workers exposed to Al (Bast-Pettersen et al., 1994; Sjogren et al., 1996; Hosovski et al., 1990; Wilhelm et al., 1990; U.S. EPA, 1987). Aluminum occurs primarily in particulate form in the ambient atmosphere and as various dusts and fumes during its production and use. Common forms of inhaled Al include aluminum oxide (alumina; Al_2O_3), pyro powders

(powder and flake Al-treated to reduce surface oxidation), Al welding fume and soluble salts (e.g., aluminum chloride and sulfate) (ACGIH, 1998).

Neurotoxicity as a Primary Toxicological Effect of Aluminum

One of the greatest health concerns regarding Al is its neurological effects. The first evidence for Al-induced neurotoxicity in humans was seen in patients who, as a result of receiving long-term hemodialysis for chronic renal failure, developed a degenerative neurological syndrome (dialysis dementia) characterized by the gradual loss of motor, speech and cognitive functions (Alfrey, 1993). This dementia, attributable to Al in the dialysate, is usually fatal within 6-9 months after the first clinical signs appear. In addition, many patients received high oral doses of Al to act as phosphate binders. Autopsies of these patients revealed increased concentrations of Al in the gray matter and cerebral spinal fluid (CSF) but no evidence of neurofibrillary degeneration (NFD) despite the elevated Al levels. Once the connection between Al and dialysis dementia was established, Al was removed from dialysis fluid and the incidence of dementia rapidly declined, thereby strengthening the argument that Al was a causal agent in dialysis dementia (Ganrot, 1986).

Amyotrophic Lateral Sclerosis (ALS) and Parkinson's Disease (PD) are other neurological diseases which have been associated with Al exposure. ALS is a progressive disease of the Central Nervous System (CNS) that is characterized by an accumulation of neurofibrillary tangles. In Guam, southern West New Guinea and parts of Japan, there is an unusually high prevalence of ALS and PD. This may be related to the natural abundance of Al coupled with the virtual lack of magnesium and calcium in the drinking water supplies and soil of these areas. In a study designed to evaluate effects of high Al and low calcium levels in the diet, much like the conditions associated with Guam and other similar areas, cynomolgus monkeys were placed on a low calcium diet either with or without supplemental Al and manganese (Garruto et al., 1989). Chronic calcium deficiency alone produced neurodegenerative effects, although neurofibrillary changes were most frequently seen in the monkeys on a low calcium diet supplemented with Al and manganese.

Though a cause and effect relationship between Al and three forms of chronic encephalopathy in humans: senile dementia of the Alzheimer type (SDAT, Alzheimer's Disease), endemic Amyotrophic Lateral Sclerosis (ALS) and endemic Parkinsonism-dementia (PD, a mixture of Parkinsonism and senile dementia) has been suggested, there is no firm evidence that it plays a causal role in the development of these diseases (Ganrot, 1986; Lione, 1985). The condition is degenerative and characterized by the progressive loss of speech, motor and cognitive functions, with death typically occurring within 1-6 months. Autopsies of patients revealed increased concentrations of Al in the gray matter and cerebral spinal fluid (CSF), though with no conclusive evidence of NFD or other neuropathological changes despite the elevated Al levels.

The neurotoxicity of Al is well documented in certain animal species. Aluminum induces a spectrum of behavioral abnormalities and brain neurofibrillary degenerative changes in rabbits and cats when injected intracranially or parenterally in high doses, though hamsters and monkeys are less sensitive (Ganrot, 1986; Lione, 1985). Such studies have been designed as models for

the possible neurotoxicological effects of Al in humans. However, it should be noted that the neurofibrillary changes in affected animals differ in morphological detail from those associated with SDAT. As discussed further in the Oral Toxicity section, oral doses of Al can also induce neurobehavioral effects in adult mice and rats and in their developing offspring. In general, such neurotoxic effects of Al appear to be more subtle than those induced through routes of administration that by-pass the gastrointestinal tract, perhaps reflecting the lower doses of Al reaching the brain.

Recent reports of studies on the effects of Al on neurotoxicity in animals have sought to define the biochemical mechanisms that are impaired when Al crosses the blood-brain barrier. However, a unifying concept has yet to emerge, though the passage of the element into various regions of the brain has been clearly demonstrated (Deloncle et al., 1995). Among the many biochemical functions and processes that appear to be perturbed by the presence of Al in the brain are the peroxidation status of biological membranes (Katyal et al., 1997; Deloncle et al., 1999), inhibition of the neuronal glutamate-nitric oxide-cyclic GMP pathway (Cucarella et al., 1998), and the marked reduction of protein- and non-protein-bound thiols and the specific activity of Na^{+}/K^{+} and Mg^{++} ATPases (Katyal et al., 1997). The relative importance of each of these mechanisms and how (or whether) they interact to bring about the observed physiological changes remains unclear.

Other Effects of Aluminum

Osteomalacia was frequently observed among long-term dialysis patients with neurological signs and is commonly attributed to Al overload (Ganrot, 1986; Lione, 1985). This bone condition is characterized by widened osteoid (unmineralized bone matrix) with no fibrosis, reduced mineralization rate, skeletal pain and a strong tendency for fractures, lack of response to vitamin D therapy and increased Al concentration in bone. Effects on bone histology and elevated bone Al levels have also been observed in patients with normal renal function who received total parenteral nutrition with Al-contaminated casein as a protein source, and in parenteral Al loading induced osteomalacia in rats and dogs (Lione, 1985).

There are a number of published reports of studies in which the carcinogenicity of aluminum compounds has been evaluated. These include oral exposure studies in which the compounds were made available to experimental animals in the drinking water or diet (Schroeder and Mitchener, 1975a,b: Oneda et al., 1994), and inhalation epidemiological studies, in which the incidence of tumor formation in persons exposed to aluminum-containing dusts and fumes in an occupational setting was compared to unexposed individuals (Spinelli et al., 1991; Thériault et al., 1984, 1990; Armstrong et al., 1986; Tremblay et al., 1995; Selden et al., 1997; Cullen et al., 1996; Dufresne et al., 1996; Ronneberg and Langmark, 1992). However, it has been generally concluded that the inferential association between exposure to Al and marginally increased incidences of tumors of the bladder and/or lung are confounded because of the co-exposure of subjects in such settings to other harmful and potentially carcinogenic substances, such as polycyclic aromatic hydrocarbons (PAHs and coal tar pitch volatiles (CTPV) (Ronneberg and Langmark, 1992). Therefore, the issue of the potential carcinogenicity of Al compounds remains uncertain.

Human Studies

Oral Exposure

Few reports have been identified that address the toxicological effects of Al in humans exposed orally. Furthermore, in a review, Reiber et al. (1995) pointed to the conflicting findings that have been reported when the incidence of neurological symptoms has been assessed in relation to Al exposure in either cross-sectional, ecological or case-control epidemiological studies. Among the more recent studies that have used this approach, Martyn et al. (1997) discussed the findings of a case-control study involving 441 men in England and Wales who were afflicted with either Alzheimer's disease, brain cancer, dementia or other neurological conditions. Assessing the historical exposure of these subjects failed to establish a link between Al in drinking water at the prevailing concentrations (below 0.2 mg/L) and the incidence of one or more of the conditions under investigation. No data were located regarding the oral carcinogenicity of aluminum compounds in humans.

Inhalation Exposure

Neurobehavioral effects were evaluated in a group of 87 Al foundry workers who were occupationally exposed to 4.6-11.5 mg/m^3 Al fumes and dust for a mean of 12.0 years [standard deviation (SD) 4.5 years, shortest exposure 6 years] compared to an unexposed control group (n=60) who were matched for age, job seniority and social status to exposed subjects (Hosovski et al., 1990). It is reported that environmental Al concentrations were measured for each worker separately during the winter and summer, implying that personal sampling may have been used and that the contributing concentrations are time-weighted averages. In certain places, the number of particles ranged as high as 329-1020/cm^2 air, and dust particle sizes were ≤1, 1-5 and ≤5 microns in 65.6, 26.6 and 7.6% of the samples, respectively. Tests of psychomotor ability (simple and complex reaction time, oculomotor coordination), intellectual ability (Wechsler intelligence, performance intelligence and verbal intelligence quotients and Wechsler subtests on information processing, memory, understanding, calculation, coding, picture completion, picture grouping, object assembling, assembling of cubes and common concepts) and cerebral damage (Bender visual motor test) were conducted. Performance of the exposed workers was found to be significantly ($p<0.02$) impaired on the complex reaction time, oculomotor coordination, memory, coding, picture completion and object assembling tests. However, the investigators noted that the performance deficits had no clinical manifestations, and that additional studies were probably needed to confirm the possibility of cerebral damage. The study yielded a lowest available non-duration adjusted LOAEL of 4.6 mg Al/m^3 for psychomotor and cognitive impairment during repeated 8-hour occupational exposures (Hosovski et al., 1990), that could be corrected for discontinuous exposure (10 m^3/20 m^3 and 5 days/7 days) to yield a $LOAEL_{HEC}$ of 1.64 mg/m^3 Al.

Aluminum oxide powders were administered to Canadian miners (mainly underground gold and uranium miners) in known exposures as a means of prophylaxis against silicosis (Stokinger, 1981; Rifat et al., 1990). Data in which more than 42 million Al treatments (≈150,000 man-years) had been given over a period of 27 years ending in 1971 were reviewed

by Stokinger (1981). The effectiveness of this treatment is uncertain but no lung damage or other ill effects (not specified) were observed. The powders (McIntyre powder) were prepared by grinding Al pellets so that 96% of the particles were ≤1.2 μm in diameter. During this process most of the particles became oxidized to aluminum oxide; the powder contained 85% aluminum oxide and 15% elemental Al. According to Stokinger (1981), recommended exposure concentrations were 30,000 particles of respirable size per cubic centimeter (ppcc) for 10 minutes/day or 10,000-20,000 ppcc for 20 minutes/day (total treatment days not indicated). Rifat et al. (1990) stated that the recommended exposure was to an Al dust concentration of 20,000-34,000 parts per ml air in the miners' changing rooms before each shift for 10 minutes. Stokinger (1981) reported that the 30,000 ppcc concentration corresponds to ≈350 mg/m^3, which is equivalent to an 8-hour average concentration of 2 mg/m^3. Based on the Stokinger (1981) data and the fact that one unspecified study used levels 30 times higher than advised, the TLV of 10 mg/m^3 is recommended for Al dust (ACGIH, 1998).

The increasing awareness of the potential neurotoxicity of Al has resulted in a number of investigations of the incidence of neurotoxicological symptoms in Al workers. Although treatment with McIntyre powder had not produced apparent adverse effects, a neurobehavioral evaluation of male miners (261 exposed to McIntyre powder, 346 unexposed) who started working between 1940 and 1979 (additional duration data not reported) was performed in 1988-1989 (Rifat et al., 1990). There were no significant differences between exposed and unexposed miners in reported diagnoses of neurological disorder. Results of cognitive testing (Mini-Mental State Examination for general cognitive function, Ravens colored progressive matrices test for reasoning and Symbol Digit Modalities Test for spatial perceptual accuracy and information processing), however, showed that the exposed group had significantly ($p \leq 0.001$) impaired performance on at least one test, and when all test scores were summed. Also, the likelihood of scores in the impaired range increased with duration of exposure.

A neurologic syndrome was described in Al smelting plant potroom workers (White et al., 1992). Twenty-five men were evaluated for suspected work-related neurologic illness based on findings in three patients studied previously. The average duration of employment was 18.7 years (SD, 3.6; range, 12-23 years), 15 of the patients were working at the time of evaluation, and 10 had taken early retirement or medical leave due to workplace-related symptoms (mean length of time since exposure was 1.3 years ranging from 0.2-5 years). Quantitative exposure level data were not reported, but 21 of the workers had been employed in the potroom prior to installation of fume hoods for a mean duration of 5.3 years (range 3-7 years). Symptoms most often reported by the patients were frequent loss of balance (88%), memory loss (84%) and joint pain (84%); other symptoms included dizziness (80%), numbness (80%), parasthesias (72%) and tremor (68%). Neurologic examinations showed mild to moderate signs of lack of coordination (tremor, dyssynergy of upper extremity limb movement or ataxia) in 84% of the patients. Neuropsychologic effects were evaluated in 21 of the patients using the Wechsler Adult Intelligence Scale-Revised (intellectual functioning), Wide Range Achievement Test-Revised (academic functioning), Halstead-Reitan Neuropsychological Test Battery (neuropsychological assessment) and Minnesota Multiphasic Personality Inventory (personality functioning). Memory function was assessed with the Wechsler Memory Scale (14 patients) and Wechsler Memory Scale-Revised (8 patients). The memory function evaluation showed mild to moderate impairment on subtests of immediate recall for verbal or visual information (70-75% of the

tested patients) and delayed verbal or visual recall (50-70%). Other effects included mild or moderate impairment on Halstead-Reitan tests of abstract reasoning and flexible thinking (42% of the tested patients), memory for tactile information (53%) and sustained attention and discrimination of tonal and speech patterns (44 and 64%, respectively). On the Wechsler memory and Halstead-Reitan tests, mild and moderate impairment was defined as scores 1.5-2 and ≥2 standard deviations below the mean of the normal population, respectively. Most (89%) of the patients tested with the Minnesota Multiphasic Personality Inventory had abnormally elevated scores (≥2 SDs above the population mean) indicative of clinical depression. Significant positive correlations were found between severity of incoordination (signs and symptoms) and degree of exposure (qualitative) before the introduction of the ventilation hoods.

White et al. (1992) noted two other studies that described neurologic problems among Al smelter workers. Thus, an evaluation of 444 electrolysis workers found neuropsychiatric changes in 123 (28%), "neurotic syndromes" in 89 (20%) and "slight pyramidal and cerebellar changes" in 39 (9%) (Langauer-Lewowicka and Braszczynska, 1983). In the second study, symptoms including mental confusion, concentration and memory problems were described in six potroom workers (Cawthon, 1988).

In another study of Al production workers, neuropsychological effects were assessed in 38 elderly men who had been exposed for at least 10 years exclusively in the potroom (n=14), foundry (n=8) or other manual labor departments of the same plant (n=16, control group) (Bast-Pettersen et al., 1994). The mean ages and employment durations of the groups were in the ranges of 62.5-63.5 and 19.2-19.6 years, respectively. The men were examined soon after or just before retirement in 1991. Limited environmental monitoring data indicates that the degree of Al exposure varied between the subgroups and over the years. Average annual total dust concentrations in the potroom were reduced significantly from 9.5 mg/m^3 in 1977 to 3.0 mg/m^3 in 1990. Aluminum levels were not specifically reported, but the average Al content in the total potroom dust was approximately 20% by weight; other constituents of the dust included fluoride and coal tar pitch components. Data from an Al uptake/excretion study of workers from the same plant indicated that the level of Al exposure was approximately 8 times higher in the potroom than in the foundry (0.48 and 0.06 mg/m^3, respectively) (Drablos et al., 1992). Medical examinations (including lung function, standard laboratory tests and serum and urine Al concentrations) and a neuropsychological test battery were performed. The battery assessed six mental functions (neuropsychiatric symptoms, motoric/sensoric, reaction time, psychomotor speed/efficiency, memory/learning and intelligence) using a questionnaire and 15 different objective tests. Some subtle deficits were found in potroom workers that were not considered to be indicative of a significant neurological syndrome. The findings in potroom workers included a subclinical tremor as indicated by results of a static steadiness test [time scores on one of two test indices were significantly worse in comparison with the control group (84% slower, p=0.03)], and possible tendencies (i.e., test results that were about 1 SD below normal mean values but not statistically significant) for increased risk of impaired visuospatial organization (Block Design subtest of the Wechsler Adult Intelligence Scale) and psychomotor tempo (one Halstead ReitanTrail Making test). Although these findings were not considered to be indicative of a neurologic syndrome, it was suggested that they may be early signs of CNS impairment. Additionally, the finding of a subclinical tremor seems to be consistent with the tremor and other

signs of incoordination observed in 84% of the patients in the White et al. (1992) study summarized above.

Studies of Al welders are consistent with those of Al smelter workers in indicating that occupational exposure to Al can be neurotoxic. CNS function was evaluated in 17 welders who had an average of 15 years (range 5-27 years) experience, with the last 4 years exclusively with Al (Hanninen et al., 1994). Most of the welders had equipment that ventilated the welding masks but the respiratory protection was not always used. The assessment included measurements of serum and urinary Al, neuropsychological tests (simple reaction time, three tests for psychomotor speed, two tests for visual and spatial ability, four memory tests and two verbal ability tests), a symptom questionnaire and neurological interview, quantitative electroencephalography (QEEG) and P-300 event-related auditory-evoked responses. Serum and urine Al levels were 3.5 and 8.5 times higher, respectively, than an unexposed reference population. The welders performed normally on the neuropsychological tests, although correlation analysis of test scores and exposure parameters showed weak negative associations between the four memory tests and urinary Al level and a positive association between the variability (standard deviation) of visual reaction times and serum Al levels. Analysis of the QEEG data showed that serum Al levels were positively correlated with the amount of delta and theta activity in the brain frontal region and negatively correlated with the amount of alpha activity in the frontal region. Results of this study (disturbances of memory and attention, QEEG changes similar to those in patients with Al encephalopathy) were interpreted as consistent with known CNS effects of Al, but insufficient for establishing a definite relationship between Al exposure and effects.

In another study of Al welders, CNS evaluations were performed on 38 men who had at least 5 years exposure (mean 17.1 years) and a control group of 44 railway track welders exposed to metal fumes other than Al (mean 13.8 years) (Sjogren et al., 1996). Limited monitoring data indicated that the median exposure to welding fumes was 10 mg/m^3 and that the Al content was 40% of the total fumes. Symptom questionnaires, psychological tests (simple reaction time, finger tapping speed and endurance, digit span, vocabulary, tracking, symbol digit coding, cylinders, olfactory threshold and Luria-Nebraska motor scale), neurophysiological indices [electroencephalography, P-300 auditory-evoked responses, brain-stem auditory evoked responses and diadochokinesis (ability to perform rapidly alternating movements with one limb)] and blood and urine Al levels were assessed. The blood and urine Al concentrations were approximately 3 and 7 times higher in the Al welders than in the controls, but there were no clear correlations between duration of exposure to Al and concentration of Al in blood or urine. The Al welders reported more acute CNS symptoms (e.g., concentration difficulties) and had decreased motor function in five tests (finger tapping in non-dominant hand, two tasks from the Luria-Nebraska motor scale, pegboard peg movement with dominant hand, amplitude of diadochokinesis in dominant hand) when compared to the control group. Urinary Al concentration was significantly correlated with acute CNS symptoms, but not with any of the performance measures. To further study possible dose-effect relationships of Al exposure, the Al welders were combined with the control group and divided into three exposure categories according to urinary Al levels, using the 50^{th} and 75^{th} percentiles as category dividers. The group with the highest mean urinary Al level had significantly more acute CNS symptoms and significantly reduced performance on one of the motor function tests (a Luria-Nebraska motor

scale task) when compared to the group with the lowest Al level. In an earlier study of 65 welders with ≥10 years of exposure to Al fumes, the highest exposure category (based on exposure duration) was 2.8 times more likely than unexposed workers to have three or more neuropsychiatric symptoms (Sjogren et al., 1990).

A body of epidemiological evidence has pointed to an increased incidence of cancers of various kinds in workers employed in the aluminum production industry. However, as discussed in a review by Ronneberg and Langmark (1992), the concern about potential cancer hazards in the aluminum industry has primarily arisen because of exposures to polycyclic aromatic hydrocarbons (PAHs) and coal tar pitch volatiles (CTPVs) rather than to Al *per se*. Thus, while a number of studies have provided inferential data linking occupationally exposed aluminum workers with an increased risk of developing tumors of the bladder or lung (Gibbs, 1985; Thériault et al., 1984, 1990; Armstrong et al., 1986; Spinelli et al., 1991; Pearson et al., 1993; Tremblay et al., 1995), it would be unwise to ascribe any excess tumor formation to the effects of Al in view of the concurrent exposure to well-documented carcinogenic PAHs such as benzo(a)pyrene. The issue is further complicated by the likely exposure of production workers to other substances such as fluorides, sulfur dioxide, aromatic amines and asbestos (Ronneberg and Langmark, 1992; Tremblay et al., 1995; Dufresne et al., 1996), and to the possible effects of cigarette smoking in affected individuals. Consequently, these studies have failed to provide direct evidence for the carcinogenicity of Al fumes and dusts.

Animal Studies

Oral Exposure

Numerous subchronic animal studies were located in the biomedical/toxicological literature but only those that define the threshold region of the oral dose-response relationship are summarized in this paper. A major limitation of many of the studies of Al toxicity is the lack of complete information on total dietary (e.g., food and drinking water) intake of Al and of other elements that are known to effect Al biokinetics and toxicity (e.g., calcium and magnesium). Estimated or reported dosages used in studies in which Al content of the basal diets are not reported must be assumed to underestimate the actual experimental dosages. The magnitude of the underestimate may be considerable. For example, a range of Al contents of 200-1200 mg Al/kg for commercial grain-based diets (Golub et al., 1992b) would provide 30-200 mg Al/kg bw-day in a subchronic or chronic mouse bioassay [based on U.S. EPA (1988) default values for body weight and food intake]. On this basis, studies in which complete dietary Al intakes were not reported or could not be estimated may provide some information about the hazards of oral exposure to Al but are inappropriate for establishing NOAELs or LOAELs for the critical effect of Al. NOAELs and LOAELs from studies that provide estimates of total Al dosages, or otherwise provide information relevant to determining the NOAEL/LOAEL boundary for the critical effect of Al are presented in Table 1 and are summarized below.

Systemic toxicity

Groups of 10 female Sprague-Dawley rats were administered aluminum nitrate nonahydrate in sugar-containing drinking water at doses of 360, 720 and 3600 mg/kg-day (26, 52

and 259 mg Al/kg bw-day, respectively) for 100 days (Domingo et al., 1987). A control group received sugar-containing distilled water only. Sugar had been added to the drinking water of all groups to reduce the taste-aversive effects of Al. The level of Al in the diet was not reported. Animals were housed in metabolic cages to facilitate the collection of fecal and urine samples. Food and water consumption were measured daily, body weights were noted weekly and blood samples were taken at monthly intervals and at termination to monitor clinical chemistry and hematological parameters. At termination, all animals were necropsied, and the weights of major organs (brain, heart, lungs, kidneys, liver and spleen) were monitored. Aluminum concentrations were measured in various tissues, pieces of which were processed for histopathological examination. A significant decrease ($p<0.05$) in body weight gain was observed in the 259 mg Al/kg-day group, attributed by the authors to decreased food intake. Overall, no consistent variations in hematological (hemoglobin, hematocrit) or clinical chemistry (SGOT, SGPT, alkaline phosphatase, urea, creatinine, total protein, cholesterol, glucose) parameters were observed. No histopathological alterations in the heart, liver, kidney, spleen, brain and cerebellum were observed. Interpretation of these data was complicated by the concurrent exposure of the rats to high doses of nitrate of up to 475 times the RfD for nitrate (1.6 mg nitrate-nitrogen/kg-day) which is based on methemoglobinemia in humans (U.S. EPA, 1999). Therefore, because of nitrate co-exposure, the absence from the study design of a food-restricted control group and uncertainty surrounding the contribution of Al in food, the apparent effect of Al on body weight gain cannot be conclusively attributed to Al alone.

Some recent studies have identified a number of potential toxicological responses in laboratory animals exposed orally to Al compounds in a subchronic or chronic dosing regimen. In most cases, however, only one dose level was employed in the study compared to controls, and since the amount of Al in the diet was not given, the resulting dose level represents an incremental dose of Al compared to that of controls as baseline. However, while these studies may offer inadequate quantitative dosimetric information for NOAEL/LOAEL identification and consequent RfD development, they provide an qualitative indication of a range of potential toxicological responses that might be induced in humans exposed to the element. For example, Garbossa et al. (1998) studied the potential for water-soluble Al to affect the erythropoietic integrity of late erythroid progenitor cells in the bone marrow. Three groups of five male Wistar rats/group were either (1) gavaged with citrate at a dose of 1.0 μm Al/g-day (27 mg/kg-day), 5 days/week, for 15 weeks, (2) had drinking water containing 100 mmol Al/L made available to them as the citrate for the same length of time or (3) maintained as controls. As calculated by the authors, the dose associated with the applied concentration of Al in drinking water approximated to 14-17 μmol/g-day (420 mg/kg-day). Rats had access to a standard chow diet, though with no indication of the baseline concentration of Al provided therein. At the end of the in-life phase of the study, all rats were sacrificed, and samples of blood were obtained for hematological investigation. Femoral bone marrow cells were flushed with physiological medium, stimulated with recombinant human erythropoietin, then monitored for the comparative incidence of colony-forming units-erythroid (CFU-E). Further tests were carried out to monitor the osmotic fragility and average life-span of erythrocytes from each test group. The animals in the group receiving Al at the higher dose showed decreased hematocrit, hemoglobin concentration, median osmotic fragility and erythrocyte life-span values compared to controls. The content of Al increased in the serum and bone of both exposed groups, the distribution of concentrations in bone correlating inversely with the extent of an animal's CFU-E development.

That Al in drinking water may have the ability to cause histopathological changes and altered hepatic enzyme activities was suggested by Basu et al. (1997) who made available aluminum chloride in drinking water to groups of eight male Sprague-Dawley rats at a dose of 50 mg/kg-day (10.1 mg Al/kg-day) for 40 days. Additionally, other groups of similarly-treated rats received drinking water containing either 0, 50, 100, 200 or 400 ppm (mg/L) added calcium (Ca), as the chloride. The authors reported increased specific activities of acid and alkaline phosphatases in liver 10,000 x g supernatants from Al-receiving animals versus controls, and in alkaline phosphatase activity in equivalent kidney preparations. The presence of Ca in the drinking water appeared to reverse these changes, plus the accompanying histopathological features associated with them.

Konishi et al. (1996) examined the ability of Al and Ca to cause opposite and potentially harmful effects in laboratory animals, in relation to the well-documented association between Al and the onset of osteomalacia. Male STD Wistar rats were divided into four groups (n=4), receiving either (1) a normal diet (Group I), (2) a normal diet supplemented with Al (Group II), (3) a Ca-deficient diet (Group III) or (4) a Ca-deficient diet with supplemental Al (Group IV), for 10 weeks. Blood samples were taken at termination, and then animals were perfused with paraformaldehyde/glutaraldehyde fixative. Levels of Ca, iron (Fe) and Al in serum and bone were measured by atomic absorption spectrophotometry, and sections of the resected right tibia were prepared for histopathological examination after decalcification in 5% formic acid in 10% formalin.

There were statistically-significant changes in body weight gain when those of groups 3 and 4 were compared to animals from groups 1 and 2, the values for the latter groups remaining constant from about 4 weeks of dosing. In discussing their histopathological findings, the authors described no decrease in the thickness of cortical bone in Group II compared to control, while bone specimen from Groups III and IV showed "an increase in osteoid as well as osteoblasts and osteoclasts", in addition to other disturbances of ossification. Such effects were considered to suggest bone fragility, with changes being more marked in Group IV compared to III. The amount of Al in the tibia of exposed rats was significantly greater in Group II than in Group I, whereas the average levels in Groups III and IV showed a further increase in Al deposition, most notably in group IV. There were also differences among the groups in the concentration of Fe in bone (tibia), and in the concentrations of Al, Ca, Fe and the levels of parathyroid hormone in blood. The authors concluded that Ca deficiency appeared to potentiate the deposition of orally administered Al in bone, and the attendant inhibition of ossification. Iron deposition was also thought to play a role in the osteogenic disturbance, where Ca is deficient.

A histopathological investigation indicated profound changes in the cerebrovascular and neuronal integrity when male Long-Evans rats (n=9) were exposed for 52 weeks to 0.5 ppm aluminum fluoride in drinking water (Varner et al., 1998). This corresponded to an Al dose of 0.019 mg/kg-day, based on a default drinking water consumption of 0.057 L/day, and a default body weight of 0.472 kg for male Long-Evans rats (U.S. EPA, 1988). Duel control groups received either NaF (fluoride controls) or double distilled deionized water. Tissue levels of Al were measured in brain, liver and kidney by the use of a direct current plasma technique.

Animals receiving aluminum fluoride showed poor survival compared to the other groups, with 6/9 having died by week 48. The tissue concentrations of Al were increased in the brain and kidney compared to both the control groups, with Al-fluorescence being used to demonstrate that Al deposition was mostly in the vasculature. Morphological and histopathological changes due to treatment were apparent in the liver, kidney and spleen. Some changes in neuronal integrity were also evident in the hippocampus and neocortex. Other cytological changes in the brain were associated with chromatid clumping, pyknosis and vacuolation.

A report by Somova et al. (1997) describes a study in which 10 male Wistar rats/group received either 0, 5 or 20 mg/kg-day aluminum chloride by gavage in water for 6 months. At termination, all animals were exsanguinated, then subjected to a necropsy in which excised pieces of liver, kidney and cardiac and skeletal muscle were taken for histopathological examination. Pieces of brain were examined by electron as well as light microscopy, and all tissues were monitored for Al concentration by atomic absorption spectrophotometry. As tabulated by the authors, Al in plasma and all of the listed tissues was dose-dependently increased to levels that were statistically significantly greater than controls. However, though described in qualitative terms and illustrated photographically, the Al-induced lesions did not receive a quantitative treatment in the report. Thus, while at least some of the low dose rats displayed NFD (neuro fibrillar degeneration) of the hippocampal region of the brain, insufficient data are provided in the report to apply this observation to the identification of a NOAEL or LOAEL.

Dietary experiments

Six Beagle dogs/sex/group were fed a diet providing either, in males, 0, 118, 317 or 1034 mg/kg-day sodium aluminum phosphate (0, 3.4, 9.0 or 29.4 mg Al/kg-day, respectively) or, in females, 0, 112, 361 or 1087 mg/kg-day sodium aluminum phosphate (0, 3.2, 10.3 or 30.9 mg Al/kg bw-day, respectively), for 6 months (Katz et al., 1984). No information was available on the level of Al in the diet, and no compound-related effects on body weight gain, hematological and clinical chemistry parameters (parameters not specified) or histopathological endpoints (major organs and tissues examined) were observed. A highest NOEL of 30.9 mg Al/kg-day could be tentatively identified in this study, but this would not include the contribution of Al from the basal diet, nor reflect the identification of any toxicological effects, since the NOEL occurred at the upper limit of the dose-response curve.

Neurotoxicity

A number of studies have been reported in which neurotoxicological/neurobehavioral effects have been explicitly evaluated. In others, the effects of Al on neurological developmental have been addressed. For example, Golub et al. (1989) fed diets containing Al as the lactate at 25 (controls), 500 or 1000 mg Al/kg diet (3.3, 65 or 130 mg Al/kg-day) to groups of 15 female Swiss-Webster mice for 6 weeks (Golub et al., 1989). No mice were exposed to lactate alone. While no statistically significant differences in food intake or body weight gain were observed, mice fed the highest Al concentration gained less weight than the controls or low-dose group. As reported by the authors, a significant decrease (20%) in spontaneous motor activity (i.e., total, vertical and horizontal movement) was observed in the 130 mg Al/kg-day group. Activity in the

65 mg Al/kg-day group was not significantly different than the controls. Thus, the highest NOAEL is 65 mg Al/kg-day and the LOAEL is 130 mg Al/kg-day.

Neurobehavioral effects of aluminum lactate were evaluated in groups of 12 female N:NIH Swiss-Webster mice (4.5-5.5 weeks old) that were fed 25 (controls) or 1000 mg Al/g diet for 90 days (Golub et al., 1992a). Based on a food factor of 0.19 kg diet/kg body weight/day calculated using an algorithm relating food consumption to body weight (U.S. EPA, 1988) and reported body weight data (the time-weighted average weight is 25.4 g), the dosage in the treated mice is estimated to be 190 mg Al/kg bw-day. No mice were exposed to lactate alone. A neurobehavioral test battery used by Donald et al. (1989) was administered at the beginning of the experiment (day 0) and after 45 and 90 (±3) days, with motor activity evaluated at the latter two time points. Aluminum levels were measured in brain, femur and liver at the end of the exposure period.

Body weight was significantly increased in the treated mice but no exposure-related changes in food intake or overt signs of neurotoxicity were observed. Results of the neurobehavioral tests showed significantly decreased hindlimb grip strength at 90 days, decreased air puff startle response at 90 days and decreased auditory startle response at 45 days in the treated mice. Spontaneous motor activity was reduced at 90 days as indicated by decreased total activity counts, horizontal activity counts and percentage of intervals with high activity counts. Aluminum concentrations in the brain and liver were increased approximately 3-fold in the treated mice, but brain and liver lipid peroxidation indices were not altered.

Male Wistar rats (6-8 per group) were exposed continuously for 6 months to food containing 1.52 mg Al/kg (normal diet) or 1000 mg Al/kg as aluminum chloride with citrate (Florence et al., 1994). The average daily Al intake was estimated to be 0.13 or 84 mg Al/kg bw-day, assuming a body weight of 0.305 kg (arithmetic mean of default mature weight of male Wistar rats and the starting weight in this study of 0.11 kg) and a food intake of 0.026 kg food/kg bw-day, calculated using an algorithm relating food intake to body weight (U.S. EPA, 1988). The citrate content of the diet was in a 1:1 stoichiometric proportion to Al, therefore, the estimated daily intake was 598 mg/kg-day. Rats exposed to Al developed histopathological abnormalities in brain tissue, not specific to any brain region, characterized by extensive cytoplasmic vacuolization in astrocytes, swelling of astrocytic processes, particularly of astrocyte end-feet abutting blood vessels. Neurons also exhibited vacuolization and nuclear inclusions. Although no specific behavioral assays were reported, the investigators noted that "no significant behavioral changes were observed". Accordingly, the functional significance of the histopathological lesions is uncertain. The lesions appear to differ from the NFD observed with parenteral Al exposures (Kowall et al., 1989; Wakayama et al., 1993); or from exposures to Al in combination with calcium deprivation (Garruto et al., 1989; Kihira et al., 1995; Mitani, 1992). The LOAEL for histopathological changes in the brain was 84 mg Al/kg-day.

Male Sprague-Dawley rats (40 per group) were exposed in drinking water to 0, 50 or 100 mg Al/kg bw-day as aluminum nitrate with citric acid for 6.5 months beginning at 21 days of age, 8 months of age or 16 months of age (Domingo et al., 1996). The citric acid dosage was 355 or 710 mg/kg-day in the 50 or 100 mg Al/kg bw-day groups, respectively. Controls did not receive citric acid. Dietary Al intake was not reported; the rats were maintained on Panlab rat

chow. Animals from control and exposed groups were subjected to a number of neurobehavioral tests, and at termination, Al levels were measured in various excised regions of the brain. The authors observed the highest Al levels in the olfactory bulb and rhachidical bulb, while the cortex and thalamus were the regions showing the lowest Al content. However, compared to controls, there were no significant effects ($p>0.05$) of Al (with citric acid) on spontaneous motor activity (open-field) or passive avoidance operant training or performance (grid floor shock, light/dark shuttle box). Thus, the NOAEL was 100 mg Al/kg-day with citric acid; although this does not include the Al contribution from food. This study is listed on Table 1 because the NOAEL, although probably underestimated because of unreported Al intake from food, is still lower than the LOAELs from other studies.

Groups of six male albino rats were administered 0 or 25 mg Al/kg bw-day as aluminum nitrate in normal saline by gavage, 10% ethanol in drinking water, or 25 mg Al/kg bw-day by gavage combined with 10% ethanol in drinking water, 6 days/week for 6 weeks (Flora et al., 1991). The level of Al in the diet was not reported. Urinary Δ-aminolevulinic acid (ALA), blood ALA-dehydratase (ALAD), blood zinc protoporphyrin (ZPP), glutamic oxaloacetic transaminase (GOT) and glutamic pyruvic transaminase (GPT) in serum and liver and brain biogenic amines and their metabolites [dopamine (DA), norepinephrine (NE), 5-hydroxytryptamine (5-HT), homovanillic acid (HVA) and 5-hydroxyindolacetic acid (5-HIAA)] were evaluated at the end of the treatment period. Treatment with Al alone caused significantly increased blood ALAD ($p<0.01$), decreased liver GPT ($p<0.05$), decreased brain DA ($p<0.01$), increased brain NE ($p<0.05$) and decreased brain 5-HT ($p<0.05$). Compared to treatment with Al alone, concurrent exposure to ethanol and Al produced significantly decreased ALAD, increased ALA, increased ZPP, increased liver GPT, increased serum GOT and increased brain HVA. Significant changes found only in the combined Al and ethanol group included increased serum GPT, increased brain NE and decreased brain 5-HT. Treatment with ethanol alone only inhibited blood ALAD. The rats were co-exposed to relatively high levels of nitrate [comparable to those in the Domingo et al. (1987) subchronic study], but it seems likely that some of the changes (i.e., effects on brain chemicals) are related to aluminum which is known to be neurotoxic. Because the toxicological significance of the changes is unclear due to lack of evaluation of neurobehavioral performance and other endpoints, there is uncertainty whether the 25 mg Al/kg-day dose is a NOAEL or a LOAEL, an uncertainty compounded by the absence of information about the level of Al in the basal diet.

Reproductive/developmental toxicity

A number of studies have been carried out to examine the effects of Al compounds on developmental toxicity, particularly their effects on postnatal neurobehavioral development. For example, Bernuzzi et al. (1989) exposed groups of 6-12 pregnant Wistar rats to aluminum chloride or aluminum lactate in the diet on gestational days 1 through 21. The rats received nominal daily doses of 0, 100, 300, 400 mg Al/kg as aluminum chloride or 0, 100, 200 or 400 mg Al/kg as aluminum lactate. No rats were exposed to lactate alone, and information regarding level of Al in the basal diet was not reported. On the average, there was a less than 10% decrease in maternal body weight gain and no effect on food or water intake. No significant difference in litter size was observed. However, postnatal mortality increased 55% and 26% in offspring of the rats exposed to 300 or 400 mg Al/kg-day, respectively. The offspring of dams

fed ≥300 mg Al/kg-day weighed significantly less than controls on postnatal day 1. Decreased body weight was also observed on postnatal days (PD) 4 and 14 in the offspring of rats fed 400 mg Al/kg-day as aluminum lactate. The following tests were used to assess neuromotor development (maturation): righting reflex, grasping reflex, negative geotaxis, suspension test and locomotor coordination. The tests were performed on PDs 4, 6, 9, 12 and 20, respectively. Impairment of neuromotor development (righting and grasping reflexes) was observed in the pups exposed to ≥200 mg Al/kg-day. Impaired grasping reflex was also observed in the 100 mg/kg-day aluminum lactate group. Offspring of rats fed 400 mg/kg-day also exhibited altered performance on the locomotor coordination test.

A follow-up study by the same research group found that ingestion of 400 mg Al/kg bw-day as aluminum lactate had no effect on postnatal mortality, body weight and righting and grasping reflex tests (Muller et al., 1990), although significant differences between control and exposure groups were noted in locomotor coordination and operant conditioning tests. Significant differences between controls and exposed groups in the negative geotaxis test were limited to those pups of dams treated during the second and third weeks of gestation, a finding interpreted by the authors to indicate the possibility of long-term effects on the central nervous system of trans-placenta exposure to Al during a later organogenic phase. According to Muller et al. (1990), the contradictions between this and their earlier study (Bernuzzi et al., 1989) could be related to environmental modifications. In particular, the mothers and pups were much more protected in the Muller et al. (1990) study than in the previous one because they were housed in plastic cages instead of wire mesh cages and received cotton to build nests. Body temperature of the pups, therefore, may have been more adequately maintained in the Muller et al. (1990) study. As discussed in this study, toxicity in pups can be confounded by insufficient body temperature, and delayed pup weight gain could explain the differences in neuromotor performance.

Muller et al. (1990) administered diets supplemented with 0 or 400 mg Al/kg bw-day as aluminum lactate to groups of 6-9 pregnant Wistar rats on days 1-7, 1-14 or 1-21 of gestation. No rats were exposed to lactate alone, and information regarding level of Al in the basal diet was not reported. Neuromotor development was assessed on postnatal days 4, 6, 9, 12 and 20 using tests of righting reflex, grasping reflex, negative geotaxis, suspension and locomotor coordination, respectively. Learning ability was also tested on PD 65 using operant conditioning. No effects on maternal body weight or food intake were observed in dams exposed on gestational days 1-7 or 1-14. In the dams exposed on gestational days (GD) 1-21, a significant decrease in maternal body weight (26 and 35%, respectively) was observed on days 16 and 19 of gestation. Decreased food intake was also observed on day 19 of gestation. No effects on litter size, postnatal mortality or postnatal body weight were observed. Impairment of neuromotor development ($p<0.05$) was observed in two of the five tests (negative geotaxis and locomotor coordination); no differences between the three treated groups were observed. For the operant conditioning test, there were significant differences ($p<0.05$) between the treated and control young rats. No differences between the three treated groups were observed. The LOAEL for developmental toxicity is 400 mg Al/kg-day, but this does not include the contribution of Al from the basal diet.

Groups of 10 pregnant Sprague Dawley rats were administered 180, 360 or 720 mg/kg-day aluminum nitrate nonahydrate by gavage (13, 26, 52 mg Al/kg bw-day) on GDs 6-14

(Paternain et al., 1988). A vehicle (water) only control group was used. The level of Al in the diet was not reported. Aluminum exposed dams gained significantly less weight than the controls. No significant effects on the numbers of litters, corpora lutea, total implants, live fetuses, resorptions or runt fetuses were observed. Significant decreases in fetal body weight and tail length were observed at all three Al doses; decreased fetal body length was also observed at the 52 mg Al/kg-day dose level. No dose-related external or visceral malformations were observed in the offspring. However, a significant increase in the incidence of skeletal malformations (delayed ossification, hypoplastic deformed ribs) was observed at all three treatment levels. In addition, the incidence of hematomas was significantly increased at the high dose. Because the rats were co-exposed to relatively high levels of nitrate [comparable to those in the Domingo et al. (1987) subchronic study], the effects of treatment cannot be conclusively attributed to Al alone, in the absence of a nitrate-exposed control group.

By contrast to the striking findings of potentially teratogenic effects of aluminum nitrate in Sprague-Dawley rats, as described above (Paternain et al., 1988), equivalent experiments by Domingo et al. (1989) in Swiss mice did not reveal any reproductive, developmental or teratogenic effects of Al, when administered to dams as the hydroxide. Domingo et al. (1989) administered by gavage 0, 66.5, 133 or 266 mg/kg-day aluminum hydroxide (0, 23.9, 47.8 or 95.5 mg Al/kg bw-day) to groups of 20 pregnant Swiss mice on GD 6-15. The level of Al in the diet was not reported. The dams were killed on GD 18. No compound-related effects were observed on maternal mortality, clinical signs, body weight, food intake or absolute or relative heart, lung, spleen, liver, kidney and brain weights. In addition, no compound-related effects were observed on numbers of implantations, resorptions, live and dead fetuses, sex ratio and the incidences of external malformations, internal soft-tissue defects or skeletal abnormalities. Therefore, this study identifies a NOEL of 95.5 mg Al/kg-day by default for reproductive, developmental and teratogenic toxicity in mice. However, neuromotor development was not assessed and the contribution of Al from the basal diet was not stated in the report.

A number of studies have been designed to evaluate the influence of citrate or lactate on the potential developmental toxicity of Al. For example, Gomez et al. (1991) exposed groups of 15-19 pregnant Sprague-Dawley rats to either distilled water (controls) or 133 mg Al/kg bw-day in the form of either aluminum hydroxide (384 mg/kg-day), aluminum citrate (1064 mg/kg-day) or aluminum hydroxide (384 mg/kg-day) concurrent with citric acid (62 mg/kg-day) by gavage on GD 6-15. The level of Al in the diet was not reported and no rats were exposed to citric acid alone. Terminations were performed on GD 20. Maternal and fetal evaluations showed exposure-related effects only in the group exposed to aluminum hydroxide and citric acid concurrently. Significant changes included reduced maternal body weight gain on GDs 6-20 (but not at sacrifice on day 20), reduced fetal body weight and some skeletal variations (increased delayed occipital and sternebrae ossification and increased absence of xiphoides). No effects were seen on maternal food consumption or clinical signs, maternal absolute or relative liver, kidney or brain weights, gravid uterine weight, corpora lutea/dam, implantations/litter, pre- or postimplantation loss/litter, viable or nonviable implants/litter, fetal sex ratio or fetal malformations (external, visceral or skeletal). This study identified a stand alone minimum LOAEL of 133 mg Al/kg-day for non-neurobehavioral developmental toxicity of aluminum hydroxide and aluminum citrate in rats. Although confidence in this LOAEL is low (because aluminum hydroxide administered concurrently with citric acid induced did developmental

effects and because the dose does not include a contribution of Al from the basal diet) the value is consistent with the developmental NOAEL of 95.5 mg Al/kg-day for aluminum hydroxide in mice (Domingo et al., 1989).

In a similar experimental protocol, groups of 11-13 pregnant female Swiss albino (CD-1) mice were administered 57.5 mg Al/kg bw-day as either aluminum hydroxide (166 mg/kg-day), aluminum lactate (627 mg/kg-day) or aluminum hydroxide (166 mg/kg-day) concurrent with lactic acid (570 mg/kg-day) by gavage on gestation days 6-15 (Colomina et al., 1992). Other groups were treated with lactic acid alone (570 mg/kg-day, equivalent to the amount in 627 mg/kg of aluminum lactate) or distilled water (controls). The level of Al in the diet was not reported. Fetal evaluations were performed on GD 18, including examinations for skeletal and visceral abnormalities in approximately two-thirds and one-third of the pups, respectively. The investigators noted that the dose of Al (57.5 mg/kg-day) is equivalent to ingestion of 3.5 g Al/day by a 60 kg person, which is higher than the usual quantities of Al ingested therapeutically for peptic disorders. Maternal body weight gain was significantly lower than control values in the aluminum lactate-treated mice when evaluated over GDs 6-9 (92%), 6-12 (55.6%) and 0-18 (38.5%) and in the mice treated with combined aluminum hydroxide and lactic acid evaluated over GDs 6-12 (37.8%), 6-15 (42.7%) and 0-18 (15.7%). The decreased maternal weight gain in the aluminum lactate group was accompanied by significantly reduced food consumption during gestation days 6-18. Significant developmental and/or teratological effects in the aluminum lactate group included 16% reduced fetal body weight ($p<0.01$) and increased incidences of cleft palate (13.2%, $p<0.05$), dorsal hyperkyphosis (i.e., excessive flexion of spine) (13.5%, $p<0.05$) and delayed parietal ossification (15.4%, $p<0.01$). These developmental effects were not observed in any of the control or aluminum hydroxide exposed pups, and the only other significant changes in the other groups were decreased maternal relative liver weight and delayed fetal parietal ossification in the lactic acid only exposure group. Other types of internal or skeletal malformations or variations were not found in any of the fetuses. Additionally, no effects were seen on maternal absolute or relative kidney weight, gravid uterine weight, numbers of implantation sites/litter, live or dead fetuses, resorptions, postimplantation loss/litter, litters with dead fetuses or fetal sex ratio in any of the groups. By analogy to the findings of the Domingo et al. (1989) and Gomez et al. (1991) studies, the lack of developmental effects of aluminum hydroxide at the tested dose could be related to low solubility and absorption.

In a more recent study, pregnant Swiss mice were administered gavage doses of 0 or 104 mg Al/kg bw-day as aluminum hydroxide on days 6-15 of gestation (Colomina et al., 1994). Dietary Al intake was not reported; the mice were maintained on Panlab rodent chow. Compared to controls, there were no effects ($p>0.05$) of Al on maternal body or organ weight, number of implantations per litter, number of resorptions per litter, number of dead fetuses per litter, percentage of positive post-implantation loss, sex ratio or fetal body weight per litter. Gross external, visceral or skeletal examination of fetuses revealed no abnormalities or developmental variations. Thus, the NOAEL for development effects from this study is 104 mg Al/kg-day, however, this does not include the Al contribution from food. Thus, based on this study and the previous study (Colomina et al., 1992), aluminum lactate appears to be more potent as a developmental toxicant in mice than the less water soluble aluminum hydroxide.

Groups of 16 pregnant Swiss-Webster mice were fed 25 (control group), 500 or 1000 mg Al/kg diet as aluminum lactate throughout gestation and lactation (Donald et al., 1989). The control diet was fed to pups that were selected for post-weaning neurobehavioral assessment. Reported maternal doses were 5, 100 and 200 mg Al/kg bw-day at the beginning of pregnancy and 10.5, 210 and 420 mg Al/kg bw-day near the end of lactation. No mice were exposed to lactate alone. There were no treatment-related changes in maternal survival, body weight (measured on GD 0 and 16 and PDs 0, 5, 10, 15 and 20), food intake, toxic signs or neurobehavior (evaluated after pups were weaned at PD 21 using the same test battery used for the pups and described below), or on litter size or postnatal growth and development in pups as assessed by body weight, toxic signs on PDs 0-55, and by crown-rump length on PDs 0 and 20. Neurobehavioral maturation was tested in two pups per litter on PDs 8-18 with a 12-item test battery (fore- and hindlimb grasp, fore- and hindpaw placement on sticks of 2 widths, vibrissa placing, visual placing, auditory and air puff startle, eye opening and screen grasp, cling and climb). A neurobehavioral test battery was administered to six pups per litter at age 25 days (4 days postweaning) or 39 days (fore- and hindlimb grip strengths, temperature sensitivity of tail, negative geotaxis, startle reflex to air puff and auditory stimuli) or age 21 and 35 days (foot splay). The pre-weaning neurobehavioral testing showed that a significant ($p=0.007$) number of pups in the high dose group had impaired vertical screen climb performance. The postweaning neurobehavioral assessment showed significantly ($p<0.05$) altered performance on several tests. These included decreased forelimb grip strength at age 39 days in the low dose group, increased hindlimb grip strength at age 25 days in both low and high dose groups, increased foot splay distance at age 21 days in both low and high dose groups and at age 35 days in the low dose group, and increased forelimb grip strength at age 25 days and decreased thermal sensitivity at age 25 and 39 days in the high dose group. There were no treatment-related changes in concentrations of Al in pup liver or bone (brain tissue was not analyzed).

In a more recent study of similar design by the same group of investigators, groups of 14 and 9 female Swiss Webster mice (6-8 weeks old) were fed 25 (control) or 1000 mg Al/g diet as aluminum lactate, respectively, during gestation and lactation (Golub et al., 1992b). The 1000 mg/g concentration was selected based on the demonstration of neurobehavioral effects in weanlings at this level (Donald et al., 1989). No mice were exposed to lactate alone. Using food intake and body weight values estimated from reported data, maternal doses are estimated to be approximately 4.3 and 174 mg Al/kg bw-day at the beginning of gestation and 4.8 and 607 at the end of the lactation period. At birth, litters were fostered either within or between groups to provide four groups of offspring that were exposed to excess Al via maternal diet during gestation, lactation, both or neither (i.e., 25 ppm during gestation and lactation, 1000 ppm during gestation and 25 ppm during lactation, 25 ppm during gestation and 1000 ppm during lactation, and 1000 ppm during gestation and lactation). Maternal effects included significantly ($p\leq0.015$) reduced (10-12%) body weight gain and food intake in the treated group during late pregnancy and lactation, and signs of neurotoxicity (hindlimb splaying and dragging) in one treated dam at postnatal day 21 (weaning); this dam had seizures and died 4 days later. No treatment-related effects on litter size, birth weight, crown-rump length, righting ability at birth, sex ratio or postnatal survival were observed. Both gestation-only and lactation-only exposure caused significantly ($p<0.05$) decreased body weight gain in the treated pups beginning on postnatal day 10; combined gestation and lactation exposure produced the greatest decrease (approximately 24% at weaning). Neurobehavioral testing using the same battery as Donald et al. (1989) was

performed at weaning on the dams and on a total of 12, 16, 12 and 6 pups (1 male and 1 female pup per litter) from the control, gestation-only, lactation-only and combined gestation and lactation groups, respectively. Results of this testing showed effects only in pups, including significantly decreased forelimb grip strength after gestation-only exposure, increased hindlimb grip strength after both gestation and lactation exposure, decreased temperature sensitivity after lactation-only exposure, and longer negative geotaxis latency after lactation-only exposure. In general, the findings of this study are consistent with those of Donald et al. (1989) in showing neurodevelopmental effects at the 1000 mg/kg dietary concentration, although intake dosages are dissimilar at the end of lactation. Using the dosage at the beginning of gestation, this study defines a LOAEL of 174 mg/kg-day for developmental effects.

The Donald et al. (1989) study differs from that of Golub et al. (1992b) in that offspring were not fostered, were tested at a later age (25 vs. 21 days), were allowed 4 days of recovery from the treated diet prior to testing, participated in other behavioral tests currently, and experienced no growth retardation. The effects found only in the cross-fostered groups in the Golub et al. (1992b) study (lower forelimb strength after gestation exposure and altered negative geotaxis latencies after lactation only exposure) were not observed by Donald et al. (1989). Increased footsplay was observed by Donald et al. (1989) but not by Golub et al. (1992b), perhaps due to an opposing effect of smaller pup body size in this study. Neither gestation or lactation exposure affected pup brain or liver Al concentrations, but lactation exposure caused significantly lower manganese and iron concentrations in liver and manganese concentrations in brain.

In a further extension of the two previous studies (Donald et al., 1989; Golub et al., 1992b), pregnant female Swiss-Webster mice were exposed continuously to a semi-purified diet containing 7 (control), 500 or 1000 mg Al/kg from the time of conception, through pregnancy and lactation (Golub et al., 1995). At weaning, pups were exposed to the same Al diet as their mothers (500 or 1000 mg Al/kg) until they were 150-170 days of age or were switched to the control diet (7 mg Al/kg) for the same time period. Based on reported dosages in previous studies by the same investigators, estimated daily dosages for mice exposed to 1000 mg Al/kg diet were as follows: 200 mg/kg bw-day in pregnant mice, 420 mg/kg-day in lactating mice and 130 mg/kg-day in offspring (Golub et al., 1994); doses for the mice exposed to 500 mg Al/kg diet were assumed to be approximately half of that of mice fed 1000 mg Al/kg, or 100 mg/kg-day in pregnant mice, 210 mg/kg-day in lactating mice and 65 mg/kg-day in offspring. Compared to the control diet, the Al diet had no effect on dam weight, gestation length, litter size, pup weight, offspring growth or organ weights. Operant conditioning (nose poke) of offspring for delayed spatial alternation or discrimination reversal tasks was initiated at 50 days of age and continued 5 days/week for a total of 35 sessions. A neurobehavioral test battery was conducted when the offspring were 150-170 days of age (forelimb and hindlimb grip strength, temperature sensitivity, negative geotaxis, air puff and auditory startle response). Maternal and pre-weaning exposure to 500 mg Al/kg significantly affected ($p<0.05$) operant training in the offspring, but not performance after training in delayed spatial alternation or discrimination reversal tasks (i.e., decreased number of training sessions to achieve the training criteria). This exposure also significantly decreased forelimb and hindlimb grip strength and puff startle response ($p<0.05$). Pre-weaning and combined pre- and post-weaning exposure to 1000 mg Al/kg significantly increased ($p<0.05$) incidence of cagemate aggression at the time behavioral

testing. No effects were observed on auditory startle response, temperature sensitivity or negative geotaxis in offspring. Histopathological examination of the brain and spinal cord revealed no treatment-related changes. Thus, the LOAEL for combined maternal and pre-weaning exposure on neurobehavioral effects in mice would approximate to 100 mg Al/kg-day (estimated daily maternal dosage).

Pregnant Charles River CD rats were administered gavage doses of 0, 250, 500 or 1000 mg Al/kg bw-day ("experiment A") or 0, 5, 25, 50, 250 or 500 mg Al/kg bw-day ("experiment B") as aluminum lactate in distilled water on GDs 5-15 (Agarwal et al., 1996). Dietary Al intake was not reported. Offspring were examined for body weight, anogenital distance, oestrus cycle regularity (after puberty), duration of pseudopregnancy induced by mechanical stimulation of the cervix, oocyte production induced by an injection of human chorionic gonadotropin, and male and female gonad weights. Aluminum had no effect on litter size and no consistent effects on birth weight were observed. For example, birth weights were decreased in male offspring from dams that received 250 mg Al/kg-day, but not at higher dosages, and the effect was observed only in experiment A. Female offspring birth weights decreased at certain dosage levels in experiment A and increased at these same dosage levels in experiment B. Similar inconsistencies between experiment A and B were observed for gonadal weights, anogenital distance, time to puberty (vaginal opening), duration of pseudopregnancy or numbers of superovulated oocytes. A significantly increased ($p<0.05$) number of abnormal oestrus cycle lengths (defined as less than 4 days or greater than 5 days) occurred in offspring from dams that received 250 mg Al/kg-day (in experiment A, the endpoint was not measured in experiment B). However, the effect was most pronounced in the first three oestrus cycles (of five observed) and not detected by the 5th cycle. Thus, the NOAEL for temporary disturbance of the oestrus cycle in offspring of dams administered Al is 250 mg Al/kg-day. NOAELs for all other reproductive endpoints in this study were 1000 mg Al/kg-day. These NOAELs do not include the contribution of Al in food.

In a three-generation study, Ondreicka et al. (1966) exposed initial groups of seven female and three male Dobra Voda mice to either 0 or 19.3 mg Al/kg bw-day as aluminum chloride in drinking water. The diet also contained 160 to 180 ppm Al, giving an estimated intake of 27-31 mg/kg-day based on default values for food consumption and body weight for chronic exposure of mice (U.S. EPA, 1988). Using this estimate, the total Al intakes (drinking water and food) were 27 mg/kg-day (controls) and 46.3 mg/kg-day (exposed group). The P_0 group produced three litters (designated F_{1a}, F_{1b} and F_{1c}) and the F_{1a} group produced two litters (designated F_{2a} and F_{2b}) from which the weanlings were exposed to Al in the drinking water starting at 4 weeks of age. There was no difference in body weight gain among the groups in the P_0 generation, a result that contrasted with the striking decrease in this parameter in the treated F_{1b}, F_{1c}, F_{2a} and F_{2b} groups. Though no effects on erythrocyte count, hemoglobin levels or histopathology of the liver, spleen and kidneys were observed in the P_0, F_1 or F_2 generations at the end of the study and no significant differences were seen in the number of litters or offspring between the exposed and control groups, the study identified a LOAEL of 46.3 mg Al/kg-day, based on the observed changes in body weight gain.

Other toxicological effects of aluminum

In a study designed to determine the effects of oral Al exposure on susceptibility to bacterial infection, female Swiss-Webster mice (13-14 per group) were exposed to a diet containing 25 (control), 500 or 1000 mg Al/kg as aluminum lactate during pregnancy, through lactation and for 10 days following weaning of the pups (Yoshida et al., 1989). Based on reported dosages in previous studies by the same investigators, estimated daily dosages for mice exposed to 1000 mg Al/kg diet are as follows: 200 mg/kg-day during pregnancy and 420 mg/kg-day during lactation; doses for the mice exposed to 500 mg Al/kg diet are assumed to be approximately half of that of mice fed 1000 mg Al/kg, or 100 mg/kg-day in pregnant mice and 210 mg/kg-day in lactating mice (Golub et al., 1994). At weaning, dams and pups were inoculated with a tail vein injection of *Listeria monocytogenes* and monitored for mortality for 10 days. In a separate experiment, female mice, 6 weeks of age, were exposed to the same dietary Al levels for 6 weeks and then inoculated with *L. monocytogenes*. Estimated Al dosages were 5, 98 or 195 mg Al/kg bw-day for the 25, 500 or 1000 mg Al/kg dietary levels, respectively, based on a default food factor of 0.195 kg diet/kg bw-day assuming a reference "subchronic" food intake and body weight for female B6C3F1 mice over the period from weaning to 90 days (U.S. EPA, 1988). Inoculation resulted in significantly greater ($p<0.025$) mortality in dams exposed to 500 or 1000 mg Al/kg diet compared to controls. There were no differences in mortality between the groups of inoculated pups or between groups of inoculated adult mice exposed to Al for 6 weeks. The LOAEL for pregnant mice was 100 mg Al/kg bw-day and the NOAEL for adult, non-pregnant mice was 195 mg Al/kg bw-day. Although the exposure duration in this study was only 7 weeks, it is included in Table 1 because it provides the only dose-response data on the effects of Al on resistance to pathogens.

Carcinogenicity studies

Schroeder and Mitchener (1975a) exposed 52 Long-Evans rats/sex/group to 0 or 5 ppm Al as potassium aluminum sulfate in drinking water for life. Based on default values for drinking water consumption and body weight for this strain of rat in a chronic study (U.S. EPA, 1988), these values are equivalent to Al doses of 0.472 and 0.67 mg/kg-day, for males and females, respectively. Study endpoints included body and heart weight; serum glucose, cholesterol and uric acid; and urinary protein, glucose and pH. All animals were necropsied at the time of natural death, and histological examinations were carried out on heart, lung, kidney, liver, spleen and gross tumors, for approximately 50% of the animals in the group. The only remarkable finding was a significant increase ($p<0.005$) in gross tumor incidence in exposed male rats [13/25 (52%) compared to 4/26 (15%) in controls], although the tumor sites were not reported. Six of the tumors in the exposed males (46% of total) were considered malignant compared to two malignant tumors (50% of total) in the male controls. There were no significant differences in tumor incidences between exposed and control females.

In another study by the same investigators, 54 Swiss mice/sex/group were exposed to drinking water containing 0 or 5 ppm Al as aluminum potassium sulfate for life (Schroeder and Mitchener, 1975b). Based on default values for drinking water consumption and body weight for B6C3F1 mice in a chronic study (U.S. EPA, 1988), these values approximate to Al doses of 1.2 mg/kg-day in both males and females. Study endpoints included body weight, gross pathology,

and some limited histology of the heart, lung, liver, kidney and spleen. The incidences of gross tumors were 15/41 (36.6%) and 11/38 (28.9%) in exposed and control males, respectively, and 19/41 (46.3%) and 14/47 (29.8%) in exposed and control females, respectively, differences that did not achieve statistical significance by Fisher's exact test, although incidences of multiple tumors and lymphoma leukemia were considered by the authors to be significantly increased in females ($p<0.025$ and $p<0.05$, respectively). However, a definitive assessment of aluminum carcinogenicity in both this and the rat study (Schroeder and Mitchener, 1975a) is precluded by the limitations of the pathology examinations and reporting.

In a more recent study, the tumorigenic potential of aluminum potassium sulfate was assessed in B6C3F1 mice chronically exposed in the diet (Oneda et al., 1994). Sixty animals/sex/group were fed a diet containing 0, 1.0, 2.5, 5.0 or 10.0% (w/w) for 20 months. These concentrations of aluminum potassium sulfate (as the dodecahydrate) are equivalent to 0, 569, 1422, 2844 and 5687 ppm Al. Using food factors calculated with an algorithm relating food consumption to body weight (U.S. EPA, 1988) and body weight data estimated from growth curves reported by the investigators, the dosages of aluminum are estimated to be 0, 95, 237, 483 or 1024 mg Al/kg-day in males and 0, 97, 242, 512 or 1110 mg Al/kg-day in females. Clinical signs, food consumption, and body weight were evaluated weekly. Hematology, clinical chemistry or urine endpoints were not assessed. Necropsies that included organ weight measurements and comprehensive histological examinations (including brain) were performed on all animals, including those that died during the course of the study. Survival rates were higher than control values in all treated male and female groups, ranging from 86.7-95.0% compared to 73.3% in males and 86.7-91.7% compared to 78.3% in females. No changes in food consumption were observed, but body weight gain was increased in both sexes at 95-97 and 237-242 mg Al/kg-day (weights were 10-23% higher than controls at end of study), was similar to controls in both sexes at 483-512 mg Al/kg-day, and decreased in both sexes at 1024-1110 mg Al/kg-day (11-16% lower than controls at end of study). There were no exposure-related increased incidences of tumors, other proliferative lesions or non-neoplastic lesions. In fact, the incidence of spontaneous hepatocellular carcinomas was significantly decreased in males at 1024 mg Al/kg-day (5.5% compared to 20.5% in controls, $p<0.01$).

Inhalation Exposure

Groups of 20 weanling Fischer 344 rats/sex and 20 weanling Hartley guinea pigs/sex were exposed to 0, 0.25, 2.5 or 25 mg/m^3 aluminum chlorhydrate [$Al_2(OH)_5Cl{\geq}x(H_2O)$] for 6 hours/day, 5 days/week for 6 months (Steinhagen et al., 1978). Analysis of the aluminum chlorhydrate by the investigators showed it to contain 24.5% Al, indicating that the animals were exposed to 0, 0.061, 0.61 and 6.1 mg Al/m^3. Body weights were measured weekly for the first 8 weeks and biweekly thereafter. At the end of the exposure period, 10 animals (5/sex) of each species were sacrificed for organ weight measurements (heart, lung, liver, kidney, spleen and brain) and histological examination of the lungs, liver and kidney. In addition, comprehensive histological examinations were performed on animals in the control and 6.1 mg AL/m^3 groups. The remainder of the animals was used for hematology evaluation (RBC, WBC, hematocrit and hemoglobin) and Al measurements in blood and tissues. Apparent effects of Al included multifocal granulomatous pneumonia in both species at ≥0.61 mg Al/m^3, significantly increased absolute and relative lung weights in both species, and decreased body weight gain in rats and

minimal lung edema in guinea pigs at 6.1 mg Al/m^3. The granulomatous reaction was characterized by foci of giant vacuoled particle-containing macrophages in the lungs and macrophages that did not appear to contain vacuoles or other evidence of phagocytized material in the peribronchial lymph nodes. There was a significant dose-related accumulation of Al in the lungs of both species at ≥0.061 mg Al/m^3. However, a NOAEL of 0.061 mg/m^3 could be identified for the onset of compound-induced histopathological effects.

In other studies, groups of 14-30 guinea pigs, rats and hamsters were exposed to fine metallic Al powders (pyro, atomized and flaked) at concentrations of 15, 30, 50 or 100 mg powder/m^3 air for 6 hours/day, 5 days/week for 6 months (Gross et al., 1973). Alveolar proteinosis occurred in exposed animals of all three species after 2 months of exposure, but fibrosis or other pulmonary changes did not develop. Similarly, groups of 23 or 46 rats and 48 hamsters were exposed to undetermined concentrations of Al fumes or Al powder (20% Al, 80% $Al(OH)_3$) for morning hours only or morning and afternoon for up to 20 months (Christie et al., 1963). Effects were similar for both forms of Al in both species, including initial increased alveolar macrophage proliferation followed by nodular hyalinized areas, with development of pneumonia but no fibrosis.

Exposure to 2.18 mg Al fibers/m^3 for 6 hours/day, 5 days/week for up to 86 weeks produced slightly increased alveolar macrophages and some irritation of the nasal passages in a group of 50 Alderly Park rats (Pigott et al., 1981). Finally, a study by Drew et al. (1974) observed the development of granulomatous nodules also developed in male hamsters that were exposed to 8 mg Al/m^3 of *Alchlor* (a propylene glycol complex of aluminum-chloride-hydroxide) for 6 hours/day, 5 days/week for 20 or 30 exposures. The alterations persisted at the longest post treatment observation (6 weeks) and consistently developed at the bifurcation of the bronchioloalveolar ducts, which is a likely site of particulate deposition.

DERIVATION OF A PROVISIONAL CHRONIC RfD FOR ALUMINUM

This survey of the toxicological effects of Al in rodents suggests that neurotoxicological and developmental (including neurodevelopmental) endpoints are among the most sensitive indicators of Al toxicity. However, as vehicles for the development of toxicity values such as a provisional chronic RfD, the latter group of studies are considered to be more appropriate, since the level of exposure to Al appears to be better characterized. In fact, neurobehavioral deficits have been observed in mice and rats exposed during various stages of development and in subchronic studies (Bernuzzi et al., 1989; Donald et al., 1989; Golub et al., 1989, 1992a, b, 1995; Muller et al., 1990), as described above. These deficits include impaired operant learning, changes in grip strength, altered startle response and impaired motor coordination. In addition, several studies have shown that oral Al can produce histopathological changes in the CNS, although the histopathological lesions have yet to be causally related to the neurobehavioral deficits. Thus, Florence et al. (1994) reported histopathological changes in the brain of rats exposed to dietary Al for 6 months, the changes including the appearance of vacuolation of the cell body and cell processes of astrocytes in the brain and swelling of astrocytic processes. In addition, more localized vacuolization of neurons in the brain also was observed. These changes

were observed in rats exposed to elevated Al in the diet and are distinct from the NFD that has been observed in rats, rabbits and monkeys maintained on elevated dietary Al in combination with reduced dietary calcium (Garruto et al., 1989; Kihira et al., 1994; Mitani, 1992; Yano et al., 1989; Yoshida et al., 1990) or in rabbits administered intracisternal or intraventricular injections of Al (Kowall et al., 1989; Wakayama et al., 1993). Interpretation of the low-calcium studies is complicated by the observation that NFD was observed in animals maintained on low-calcium diets without excess Al and was enhanced by the addition of excess Al to these diets (Garruto et al., 1989; Kihira et al., 1994). Furthermore, Al has been shown to inhibit the gastrointestinal absorption of calcium (Orihuela et al., 1996), an effect that may exacerbate the calcium deprivation induced by low calcium diets. Thus, it is not clear whether calcium deprivation enhances the neurotoxicity of Al or Al exacerbates the adverse effects of calcium deprivation.

Donald et al. (1989) and Golub et al. (1995) are co-principal studies that identify a LOAEL of 100 mg Al/kg-day for minimal neurotoxicity in the offspring of mice exposed to dietary aluminum lactate (soluble aluminum) during gestation and lactation. The neurotoxicity associated with this LOAEL is consistent with LOAELs from other developmental and subchronic neurobehavioral studies in mice and rats which used higher dietary dosages of aluminum lactate or aluminum chloride (Golub et al., 1989, 1992a,b; Bernuzzi et al., 1989; Muller et al., 1990). Of the above, Golub et al., (1995) is the only study in which a histopathological examination of the brain and spinal cord was conducted and no abnormalities were reported. The Florence et al. (1994) study indicates that histopathological abnormalities of the CNS can occur in rats exposed subchronically to 84 mg/kg-day; although this is lower than the LOAEL for neurobehavioral effects, it was not chosen as the principal study because the functional significance of the histopathological lesions are uncertain.

A number of studies were identified that, at face value, appeared to indicate LOAELs at lower doses than the 100 mg Al/kg-day value selected herein, for example, Paternain et al. (1988) and Colomina et al. (1992). However, in these as in many of the studies under consideration, insufficient information on dietary Al (Al content and/or feed type) was reported to permit a reliable estimation of the overall dose level to which the animals were subjected.

Other developmental studies with aluminum hydroxide and/or citrate in mice and rats identified a NOAEL which are equivalent (95.5 mg Al/kg-day), or a minimum LOAEL that was greater (133 mg Al/kg-day) than the 100 mg Al/kg-day critical LOAEL (Domingo et al., 1989; Gomez et al., 1991), an overlap potentially related to differences in effective doses due to variations in unreported Al dietary content and factors affecting absorption such as chemical form (e.g., the use of less absorbable aluminum hydroxide). In addition, the LOAEL of 43.3 mg Al/kg-day for decreased body weight gain in mice exposed to aluminum chloride for 180-390 days (Ondreicka et al., 1966) was thought be inappropriate for risk assessment due to the small sample size and to the poor reporting of study details. Aluminum nitrate caused alterations in levels of brain biogenic amines and hepatic and hematological indices in rats exposed to 21.4 mg Al/kg-day for 6 weeks (Flora et al., 1991). This dose is not a LOAEL because insufficient information is available to determine if the effects are adverse.

Therefore, the LOAEL of 100 mg Al/kg-day for minimal neurotoxicity in the offspring of mice (Donald et al., 1989, Golub et al., 1995) is selected as the basis for the provisional chronic

RfD. The LOAEL is considered minimal because the results of the postweaning neurobehavioral test battery indicate that performance deficits may be marginal. In particular, of the three observed effects (decreased forelimb and increased hindlimb grip strengths, increased hindlimb foot splay distance), one effect (increased grip strength) has unclear toxicological significance and two effects (increased grip strength and foot splay distance) did not persist after 2 weeks of no further exposure.

Application of an uncertainty factor (UF) of 100 (3 for use of a minimal LOAEL, 10 for interspecies extrapolation and 3 for intrahuman variability where the critical effects have been observed in a sensitive sub-group) results in a provisional RfD of

p-RfD = 1E-0 mg Al/kg-day.

The provisional RfD of **1E-0** mg Al/kg-day is approximately 3-fold higher than estimated normal daily Al intake of approximately 0.2-0.3 mg/kg-day (Iyengar et al., 1987; Ganrot, 1986; Wilhelm et al., 1990). Chronic users of medications such as antacids, buffered aspirins and antiulceratives would be expected to ingest much larger amounts of Al, possibly as high as 10-70 mg/kg-day. However, these subjects would not represent the most sensitive population (developing infants), as indicated by the animal data.

Low confidence is placed in the co-critical studies, because they only identify a LOAEL for a sensitive effect and evaluated comparatively small numbers of animals. Confidence in the data base is low because the most reliable supporting data for neurotoxicity of Al in humans are of limited general relevance (e.g., dialysis encephalopathy is manifested in patients with impaired renal function and excessive Al uptake from intravenous exposure). In fact, neurotoxicity remains to be assessed in animals chronically exposed to Al, and developmental morphology has not been adequately investigated in two animal species. These limitations in the Al data base do not increase uncertainty in the RfD; therefore, a data base uncertainty factor was not used. However, reflecting the low confidence in the co-critical studies, there is low overall confidence in the RfD.

DERIVATION OF A PROVISIONAL CHRONIC RfC FOR ALUMINUM

Al seems to be the most likely cause for the generally and consistently reported psychomotor and cognitive effects (particularly signs of impaired coordination) in Al production workers and welders (Bast-Pettersen et al., 1994; Rifat et al., 1990; Hosovski et al., 1990; White et al., 1992; Hanninen et al., 1994; Sjogren et al., 1990, 1996). In addition, there is strong evidence that Al is neurotoxic by other routes of exposure. Thus, a degenerative neurological syndrome (dialysis dementia) has been documented in humans with chronic renal failure, apparently due to an increased exposure to Al from dialysis treatment and/or ingestion of phosphate binding agents which contain Al (Alfrey, 1993). This syndrome is characterized by gradual loss of motor, speech and cognitive functions. Neurotoxicity, particularly neuromuscular effects such as decreased motor activity, startle responsiveness and grip strength, has also been observed in mice following subchronic oral exposure and in the offspring of mice and rats exposed orally during gestation and/or lactation. Based on this information, as well as evidence

that Al is absorbed by Al production workers and welders, the hypothesis that the occupational studies are indicative of a neurotoxic effect of Al appears to be justified. However, the only occupational study that has yielded suitable monitoring data is that of Hosovski et al. (1990), in which workers were exposed to presumed time-weighted average (TWA) concentrations of 4.6-11.5 mg Al/m^3 magnitude for an average of 12 years. Using 4.6 mg Al/m^3 as the LOAEL for psychomotor and cognitive impairment for an 8-hour occupational exposure (Hosovski et al., 1990) and corrections for discontinuous exposure (10 m^3/20 m^3 and 5 days/7 days), the $LOAEL_{HEC}$ is 1.64 mg/m^3. Applying an uncertainty factor of 300 for intrahuman variability (10), use of a LOAEL (10) and an incomplete database (3) yields a provisional RfC of

p-RfC = 1.64 mg/m^3/300 = 5E-3 mg/m^3.

The lack of inhalation developmental studies may increase uncertainty in the database because oral data in animals indicate that neurotoxic and morphological developmental effects may occur at lower doses than neurotoxicity in adults. Additionally, there is uncertainty related to the lack of corroborating data on air concentrations associated with neurotoxicity. Confidence in the critical study is low to medium because only a LOAEL was identified. Confidence in the database is medium because (1) there are no corroborating data on effect levels (NOAELs and additional LOAELs), (2) no data are available for developmental neurotoxicity by the inhalation route and (3) a well-designed two-generation reproduction study is lacking. Reflecting the low to medium confidence in the critical study and database, there is low to medium confidence in the provisional RfC.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR ALUMINUM

Weight-Of-Evidence Classification

A considerable number of epidemiological studies have examined the incidence of excess tumor formation in persons occupationally exposed to Al in the form of dusts or fumes. In general, a body of inferential evidence exists for an increase in cancer of the bladder and lung through such occupational exposure to Al, although conclusions linking these responses to the effects of Al are confounded by attendant co-exposure to other harmful emissions such as PAHs and by cigarette smoking. A 20-month exposure of B6C3F1 mice to Al potassium sulfate dodecahydrate in the diet at concentrations up to 10% w/w displayed no indication of compound-related carcinogenicity and, in general, no indication of adverse toxicological effects of any kind (Oneda et al., 1994). Similarly, the life-time exposure of Swiss mice and Long-Evans rats to 5 ppm Al as aluminum potassium sulfate in drinking water provided no convincing evidence for the carcinogenicity of Al compounds (Schroeder and Mitchener, 1975a,b). Gene reversion experiments on Al compounds resulted in negative results in *S. typhimurium* (Ahn and Jeffrey, 1994). Taking all of the evidence of Al carcinogenicity together, and in accordance with the U.S. EPA (2005) cancer guidelines, aluminum is classified as *inadequate information to assess carcinogenic potential*. The basis for this classification is insufficient evidence in epidemiological/occupational studies, lack of demonstrated carcinogenicity or mutagenicity in

available animal studies, lack of positive evidence of non-carcinogenicity and lack of mode of action data for aluminum.

Quantitative Estimates of Carcinogenic Risk

Due to insufficient data, a provisional oral slope factor and inhalation unit risk could not be developed.

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Table 1. Summary of oral toxicity data for aluminum[a]

Study	Type	Species	Al	Exposure Concentration (ppm)	Exposure Dosage (mg Al/kg-day)	Exposure Frequency and Duration	Critical Effect	NOAEL (mg Al/kg-day)	LOAEL (mg Al/kg-day)	FEL (mg Al/kg-day)
Ondreicka et al., 1966	Subchronic 3-gen dietary	Dobra Voda mice	chloride	--	27 (control), 46	Continuous, 180-390 days	Decreased body weight gain in F1 and F2.	--	46	--
Golub et al., 1989	Subchronic dietary	S-W mice	lactate	25 (control), 500,1000	3.3 (control), 65,130	Continuous, 6 weeks	Decreased spontaneous motor activity; decreased weight gain.	65	130	--
Golub et al., 1992a	Subchronic dietary	S-W mice	lactate	25 (control), 1000	190	Continuous, 90 days	Decreased hindlimb grip, decreased spontaneous motor activity, decreased startle response.	--	190	--
Florence et al., 1994	Subchronic dietary	Wistar rat	chloride (with citric acid)	1.52 (control), 1000	0.13 (control), 84	Continuous, 6 months	Histopathological changes in brain astrocytes and neurons.	--	84	--
Domingo et al., 1996	Subchronic drinking water	Sprague Dawley rats	nitrate (with citric acid)	--	0, 50, 100 (plus unreported dietary Al)	Continuous, 6.5 months	Operant conditioning and performance	100	--	--
Yoshida et al., 1989	Subchronic dietary	S-W mice	lactate	25 (control), 500, 1000	5 (control), 98, 195	Continuous, 7 weeks	Increased mortality from *L. monocytogenes* inoculation	195	--	--
Donald et al., 1989	Developmental dietary	S-W mice	lactate	25 (control), 500, 1000	5 (control), 100, 200	Continuous, gestation and lactation	Neurobehavioral effects.	--	100	--
Golub et al., 1992b	Developmental dietary	S-W mice	lactate	25 (control), 1000	4 (control), 174	Continuous, gestation and lactation	Neurobehavioral effects.	--	174	--
Golub et al., 1995	Developmental dietary	S-W mice	lactate	7, 500, 1000	1 (control), 100, 200	Continuous, gestation, lactation to maturity	Neurobehavioral effects.	--	100	--

Table 1. Summary of oral toxicity data for aluminum[a]

Study	Type	Species	Al	Exposure Concentration (ppm)	Exposure Dosage (mg Al/kg-day)	Exposure Frequency and Duration	Critical Effect	NOAEL (mg Al/kg-day)	LOAEL (mg Al/kg-day)	FEL (mg Al/kg-day)
Yoshida et al., 1989	Developmental dietary	S-W mice	lactate	25 (control), 500, 1000	4 (control), 100, 200	Continuous, gestation and lactation	Increased mortality of dams from *L. monocytogenes* inoculation	--	100	--

[a]Studies for which total dosages were reported or could be estimated (unless otherwise noted).

8-25-2008

Provisional Peer Reviewed Toxicity Values for

Cobalt
(CASRN 7440-48-4)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR COBALT (CASRN 7440-48-4)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).

2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.

3. Other (peer-reviewed) toxicity values, including:

   - Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
   - California Environmental Protection Agency (CalEPA) values, and
   - EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because new information becomes available and scientific methods improve over time, PPRTVs are reviewed on a five-year basis and updated into the active database. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically

updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

The Integrated Risk Information System (IRIS) does not report a Reference Dose (RfD) for cobalt (U.S. EPA, 2007). The Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1997a) and Drinking Water Standards and Health Advisories list (U.S. EPA, 2004) likewise do not contain an RfD for cobalt. The Chemical Assessments and Related Activities (CARA) lists (U.S. EPA, 1991, 1994a) report a Health Effect Assessment (HEA) for cobalt (U.S. EPA, 1987). The 1987 HEA derived a chronic RfD of 0.005 mg cobalt/kg-day based on a no-observed-adverse-effect level (NOAEL) of 5 mg cobalt/kg-day for testicular effects in a subchronic rat study (Nation et al., 1983). The Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological Profile for cobalt and its compounds reports an oral Minimal Risk Level (MRL) for intermediate exposure of $1x10^{-2}$ mg/kg-day (ATSDR, 2004), based on a lowest-observed-adverse-effect level (LOAEL) of approximately 1 mg cobalt/kg-day for polycythemia in humans (Davis and Fields, 1958). ATSDR (2004) did not derive an oral MRL for chronic exposure. This MRL for intermediate exposure was based on the polycythemic effect of cobalt exposure (1 mg cobalt/kg-day, Davis and Fields, 1958) by application of an UF of 10 for a LOAEL and an UF of 10 for human variability. The World Health Organization (WHO, 2005) has not published an Environmental Health Criteria (EHC) document on cobalt. An International Agency for Research on Cancer (IARC) Monograph on cobalt and its compounds (IARC, 2006) and the National Toxicology Program (NTP) Status Reports (NTP, 2005) were searched for relevant information.

IRIS (U.S. EPA, 2007) does not report a Reference Concentration (RfC) for cobalt. The HEAST (U.S. EPA, 1997a) likewise does not list an RfC for cobalt. The cobalt HEA (U.S. EPA, 1987) derived a subchronic inhalation RfC of $9x10^{-5}$ mg/m^3 based on a LOAEL of 0.1 mg/m^3 for

respiratory effects in a 3-month study in swine (Kerfoot et al., 1975). A chronic inhalation RfC of $9x10^{-6}$ mg/m^3 was derived from the same study. The ATSDR Toxicological Profile for cobalt and its compounds reports an inhalation MRL for chronic exposure of $1x10^{-4}$ mg/m^3 (ATSDR, 2004), based on a NOAEL of 0.0053 mg cobalt/m^3 for decreased pulmonary function in humans (Nemery et al., 1992). The American Conference of Governmental Industrial Hygienists (ACGIH, 2004) has set a Threshold Limit Value-Time-Weighted Average (TLV-TWA) of 0.02 mg/m^3 for cobalt and inorganic cobalt compounds, expressed as cobalt, based on respiratory and cardiovascular effects. The National Institute for Occupational Safety and Health (NIOSH, 2005) Recommended Exposure Limit (REL) TWA for cobalt is 0.05 mg/m^3, based on effects in the respiratory system. The Occupational Safety and Health Administration (OSHA, 2005) Permissible Exposure Limit (PEL) is 0.1 mg/m^3.

IRIS (U.S. EPA, 2007) does not report a cancer classification, slope factor or unit risk for cobalt. The HEAST (U.S. EPA, 1997a) and Drinking Water Standards and Health Advisories list (U.S. EPA, 2004) likewise do not report carcinogenicity assessments for cobalt. The CARA lists (U.S. EPA, 1991, 1994a) do not report a cancer classification or an estimate of the carcinogenic potency of stable cobalt compounds due to a lack of pertinent data. An IARC Monograph on cobalt and its compounds (IARC, 2006) classified cobalt sulfate and other soluble cobalt (II) salts as "possibly carcinogenic to humans." ACGIH (2004) has classified cobalt in category A3 – confirmed animal carcinogen with unknown relevance to humans.

Literature searches for studies relevant to the derivation of provisional toxicity values for cobalt were conducted initially through 2000 in TOXLINE (supplemented with BIOSIS and NTIS updates), MEDLINE, TSCATS, RTECS, CCRIS, DART, EMIC/EMICBACK, HSDB, GENETOX and CANCERLIT and subsequently from 2000 to August 2005 in MEDLINE, TOXLINE (NTIS subfile), TOXCENTER, TSCATS, CCRIS, DART/ETIC, GENETOX, HSDB, RTECS and Current Contents. An updated literature search was performed in MEDLINE from 2005 to June 2008.

REVIEW OF PERTINENT DATA

Human Studies

Overview

Indicators of adverse health effects in humans following oral exposure to cobalt include increased erythrocyte number and hemoglobin (Taylor et al., 1977; Duckham and Lee, 1976; Davis and Fields, 1958), cardiomyopathy (Morin et al., 1971; Alexander, 1969, 1972) and decreased iodine uptake by the thyroid (Roche and Layrisse, 1956). Cardiomyopathy is an endpoint of concern for cobalt in humans; however, it is highly likely that alcohol consumed in "beer-cobalt cardiomyopathy," as well as other factors, such as smoking, played a role in the effects that were observed. Cobalt is a sensitizer in humans by any route of exposure. Sensitized individuals may react to inhalation of cobalt by developing asthma; ingestion or dermal contact with cobalt may result in development of dermatitis. Several studies have suggested that cross-

sensitization may occur between cobalt and nickel (Shirakawa et al., 1990; Lammintausta et al., 1985; Bencko et al., 1983; Rystedt and Fisher, 1983).

Respiratory effects, including respiratory irritation, wheezing, asthma, pneumonia and fibrosis, have been widely reported in humans exposed to cobalt by inhalation (for review, see Barceloux, 1999; Lison, 1996). Epidemiology studies show decreased pulmonary function in workers exposed to inhaled cobalt (Nemery et al., 1992; Gennart and Lauwerys, 1990). Results of studies investigating cancer incidence in workers exposed to inhaled cobalt are suggestive of a possible association between exposure to cobalt and respiratory tumors (Tuchsen et al., 1996; Mur et al., 1987; Morgan, 1983).

Oral Exposure

In humans, cobalt stimulates production of red blood cells through increased production of the hormone erythropoietin and has been explored for use in the treatment of anemia (Smith and Fisher, 1973; Duckham and Lee, 1976). Increases in red blood cell counts and blood hemoglobin have been reported in non-anemic volunteers (Davis and Fields, 1958) and in anephric anemic patients (Taylor et al., 1977; Duckham and Lee, 1976).

Reversible polycythemia (increase in blood cell number) was reported (see Table 1) in six healthy adult males following treatment with 150 mg cobalt chloride per day for 22 days (Davis and Fields, 1958). Five subjects received 150 mg cobalt chloride/day for the entire exposure period and a sixth subject initially received 120 mg cobalt chloride/day, which was later increased (time not specified) to 150 mg/day. Cobalt chloride was administered as a 2% solution diluted in either water or milk. Assuming an average body weight of 70 kg, 150 mg cobalt chloride/day corresponds to approximately 1 mg cobalt/kg-day. Outcomes assessed in this study were red blood cell count, hemoglobin percentage, leukocyte count, reticulocyte percentage and thrombocyte count. Polycythemia was observed in all six patients within 7 to 22 days of treatment as demonstrated by increases in red blood cell counts ranging from 0.5 to 1.19

Table 1. Hematopoietic, Thyroid and Developmental Effects of Cobalt (Co) via Oral Route

Target Organ	Species	Effect	Dosage (mg Co/kg-day)
Hematopoietic Effects	Human Human Rat	Reversible Effect (Polycythemia) ↑ Hemoglobin and ↑ RBC Hematopoietic effect	1.0 0.16 – 0.32* 0.5 – 32.0
Thyroid	Human Mice	↓ Iodine uptake Histopathological changes in thyroid	1.0 48.0
Fetus	Rat	Developmental toxicity	5.2 – 21.0
Heart	Rat	↓ Myocardial function	8.0

*Therapeutic doses for anemic patients

million (approximately 16-20% increase above pre-treatment levels) and increases in hemoglobin levels ranging from 6 to 11% above pretreatment values. In five of the six subjects, reticulocyte levels were elevated, reaching at least twice the pre-experiment values. Thrombocyte and total leukocyte counts were not significantly different from pretreatment values. Erythrocyte counts returned to pre-treatment levels within 9 to 15 days after cobalt administration was discontinued. The fact that leucocyte counts remained relatively constant throughout the experiment supports the concept that this is a true polycythemia. As such, based on the results of this study, 1 mg cobalt/kg-day was identified as a LOAEL for cobalt-induced polycythemia in humans.

Duckham and Lee (1976) treated 12 anephric patients on dialysis with 25 to 50 mg cobalt chloride daily for approximately 12 weeks. Assuming an average body weight of 70 kg, doses of 25 and 50 mg cobalt chloride/day are equivalent to 0.16 and 0.32 mg cobalt/kg-day, respectively. During the exposure period, patients also received daily treatment with 100 mg ferrous sulfate and 50 mg ascorbic acid. Within approximately 2 months of initiation of treatment with cobalt, an increase in hemoglobin of 26-70% was observed in patients treated with 0.32 mg cobalt/kg-day. Serum cobalt levels appeared to reach steady state within 2 months of exposure (approximately 40-100 µg cobalt/100 mL). In a subgroup of three patients, continuation of treatment with 0.16 mg cobalt/kg-day for approximately 3 months maintained elevated hemoglobin levels. Hemoglobin levels decreased rapidly when cobalt therapy was discontinued. The authors did not report whether therapy with ferrous sulfate and ascorbic acid was discontinued at the same time. Results of this study are difficult to interpret because patients were anephric and on dialysis, which may have altered cobalt pharmacokinetics and dose-effect relationships. Furthermore, since it is well established that treatment with ferrous sulfate alone increases hemoglobin concentration (Hillman, 2001), concomitant therapy with iron is a confounding factor. Since this study did not evaluate the response of patients treated with ferrous sulfate alone, it is not possible to determine the relative contributions of iron and cobalt to the observed increases in hemoglobin. Thus, adverse effect levels cannot be confidently determined for cobalt. In a separate study, a group of eight anephric patients with refractory anemia were treated with 25 to 50 mg cobalt chloride daily for 12 to 36 weeks (Taylor et al., 1977). Increased hemoglobin concentration and decreased requirement for blood transfusions were observed (Taylor et al., 1977). Data on hemoglobin concentrations (or other indicators of polycythemia) were not reported.

Pregnant women given 75 to 100 mg cobalt chloride/day with no other treatment for 90 days to 6 months did not experience pregnancy-induced reductions in hematocrit and hemoglobin levels, compared to untreated controls (Holly, 1955). However, daily treatment with 1 g ferrous sulfate alone or combined daily treatment with 60 to 90 mg cobalt chloride and 0.8 to 1.2 g ferrous sulfate prevented pregnancy-related decreases in hematocrit and hemoglobin levels. The response to combined cobalt chloride and iron therapy was more pronounced than the response to iron therapy alone. In patients treated with iron only, decreases in hemoglobin and hematocrit were prevented in approximately 80% of patients, compared to 100% of patients treated with combined cobalt chloride and iron.

Cardiomyopathy has been observed in association with consumption of large quantities of beer containing cobalt chloride (introduced into the beer to stabilize the foam) (Alexander,

1969, 1972; Morin et al., 1971). Exposure estimates in reported cases range from 0.04 to 0.14 mg cobalt/kg-day (corresponding to approximately 8-30 pints of beer daily) over a period of years (Alexander, 1969, 1972; Morin et al., 1971). The cardiomyopathy in the beer drinkers, referred to in the literature as "beer-cobalt cardiomyopathy," was fatal to 43% of the subjects within several years, with approximately 18% of these deaths occurring within the first several days following diagnosis. Beer-cobalt cardiomyopathy appeared to be similar to alcoholic cardiomyopathy and beriberi; however, the onset of the beer-cobalt cardiomyopathy was much more abrupt. The practice of adding cobalt to beer to stabilize the foam has been discontinued. It should be noted, however, that the cardiomyopathy may also have been due to the fact that the beer drinkers had protein-poor diets and may have had prior or concurrent cardiac and hepatic damage from alcohol abuse. Due to the potential adverse effects of poor nutrition and/or chronic ethanol exposure on cardiovascular health, it is difficult to delineate the contribution of oral cobalt exposure to the observed cardiomyopathy. As such, no adverse effects levels can be determined for cobalt-induced cardiotoxicity.

The thyroid also appears to be a target organ for cobalt (see Table 1). Treatment of 12 euthyroid (normal thyroid) patients with 150 mg cobalt chloride/day (equivalent to 1 mg cobalt/kg-day, assuming a body weight of 70 kg) for 2 weeks resulted in a greatly reduced uptake of 48-hour radioactive iodine by the thyroid when measured after 1 week of exposure to cobalt, with uptake nearly abolished completely by the second week of exposure to cobalt (Roche and Layrisse, 1956). It should be noted that when cobalt treatment was discontinued, iodine uptake returned to pre-treatment reported values. No other clinical details were provided for the human subjects. Therefore, based on the results of this study, a LOAEL of 1 mg cobalt/kg-day was identified for decreased radioactive iodine uptake in human thyroid following oral cobalt exposure. In another small clinical study (Paley et al., 1958), decreased radioactive iodine uptake was reported in two of four (3 males, 1 female) euthyroid patients orally administered 37.5 mg cobalt/day as cobalt chloride (equivalent to 0.54 mg cobalt/kg-day, assuming a body weight of 70 kg) for 10 to 14 days. One of the two subjects with reported decreased iodine uptake had received i.v. cobalt in addition to oral cobalt intake, and had been previously diagnosed with hyperthyroidism (although was clinically euthyroid at the time of study). The i.v. dosing may have raised the internal cobalt concentration to a level greater than the reported 0.54 mg dosage based upon oral dosing of 37.5 mg/day in other subjects that did not receive i.v. cobalt. Of the remaining three subjects, 24-hour iodine uptake was not significantly decreased following oral cobalt exposure compared to corresponding pre-treatment values (based on pairwise t-test). The oral cobalt dose of 0.54 mg cobalt/kg-day represents a NOAEL for thyroid effects in humans. It should be noted that the Roche and Layrisse (1956) and Paley et al. (1958) studies lack details pertinent to other clinical conditions (e.g. including effects on thyroid stimulating hormone [TSH]) of these patients; thus the mechanism for the effect of cobalt on thyroidal iodine uptake cannot be ascertained. However, cobalt appears to increase thiocyanate-induced release of radioiodine from the thyroid, suggesting a possible effect on binding of iodine (e.g., iodination of thyroglobulin) in the thyroid gland.

Cobalt has been found to be a sensitizer in humans. Individuals are sensitized following dermal or inhalation exposure, but flares of dermatitis may be triggered following cobalt ingestion. In a small clinical study, several patients with eczema of the hands were challenged orally with 1 mg cobalt sulfate (0.005 mg cobalt/kg-day, assuming a body weight of 70 kg) in

tablet form once per week for 3 weeks; this translates to an estimated average daily dose of 0.0007 mg cobalt/kg-day (1 day a week/7 days a week × 0.005 mg cobalt/kg-day). 28/47 patients had a flare of dermatitis following the oral challenge (Veien et al., 1987). All 47 patients had positive dermal patch tests to cobalt (13 to cobalt alone and 34 to nickel and cobalt) and 7 of the 13 patients who had patch-tested positive to cobalt alone reacted to the oral challenge. These results suggest that cobalt allergy can be induced from oral ingestion exposures to cobalt. Although the exposure levels associated with sensitization to cobalt following inhalation or dermal exposure have not been established, interrelationships have been found to exist between cobalt and nickel sensitization (Bencko et al., 1983; Rystedt and Fisher, 1983; Veien et al., 1987). In guinea pigs, nickel and cobalt sensitization appear to be interrelated and mutually enhancing (Lammintausta et al., 1985). Therefore, it is possible that in people sensitized by nickel, exposure to cobalt may result in an allergic reaction.

Inhalation Exposure

Numerous studies have investigated health effects in workers occupationally exposed to cobalt-bearing dust (Linna et al., 2003; Swennen et al., 1993; Auchincloss et al., 1992; Cugell, 1992; Nemery et al., 1992; Prescott et al., 1992; Gennart and Lauwerys, 1990; Meyer-Bisch et al., 1989; Raffn et al., 1988; Shirakawa et al., 1988, 1989; Sprince et al., 1988; Kusaka et al., 1986a,b; Demedts et al., 1984; Davison et al., 1983). However, many of these studies are of limited utility for risk assessment due to inadequate characterization of exposure and/or effects. Four studies were considered to be potentially suitable for RfC derivation. Two of these focused exclusively on respiratory effects (Nemery et al., 1992; Gennart and Lauwerys, 1990); one studied only thyroid effects (Prescott et al., 1992) and one considered multiple endpoints (Swennen et al., 1993). The populations studied included diamond-cobalt saw manufacturers, diamond polishers, plate painters and cobalt production workers. All four studies were cross-sectional design.

Several studies have examined the effects of hard metal, a mixture containing approximately 20% cobalt with the remainder being primarily tungsten carbide. Exposure of humans to hard metal has been shown to result in an increase in cancer mortality (Moulin et al., 1998; Lasfargues et al., 1994) as well as a number of other diseases, including asthma and pulmonary fibrosis (for reviews, see Barceloux, 1999; Lison, 1996). There is substantial evidence from animal studies that tungsten, although it acts as an inert dust by itself, can potentiate the effects of cobalt on the respiratory tract (Lasfargues et al., 1995; Lison et al., 1995, 1996; Swennen et al., 1993). For this reason, studies of hard metal were not given further consideration.

Gennart and Lauwerys (1990) studied ventilatory function in workers at a plant producing diamond-cobalt circular saws. The form of cobalt used in diamond polishing is primarily metallic cobalt powder; specific cobalt species contained in this powder were not identified. The exposed population consisted of 48 workers (34 males and 14 females) who agreed to participate in the study (an additional 27 workers declined). Exposure duration for these workers ranged from 0.1 to 32 years, with an average of approximately 6 years. The work involved weighing and mixing cobalt powder and microdiamond particles (and possibly small amounts of other undisclosed substances), cold pressing, heating and hot pressing. After

sintering, the pieces were welded onto steel disks. These operations were performed in two rooms called the mixing room and the oven room, where all the examined workers spent most of their time. Controls consisted of 23 workers (11 males and 12 females) from other factories in the same area who were not exposed to known pneumotoxic chemicals. Personal air samples were collected at different workplaces during half a workshift. Subjects filled out a questionnaire regarding occupational and medical histories, smoking habits and pulmonary symptoms; gave a urine sample for cobalt determination; and participated in lung function tests. Cobalt concentrations varied from 9.4 to 2875 $\mu g/m^3$ in the mixing room (geometric mean=135.5 $\mu g/m^3$) and from 6.2 to 51.2 $\mu g/m^3$ in the oven room (geometric mean=15.2 $\mu g/m^3$). The prevalence of respiratory symptoms, such as cough, sputum and dyspnea, were significantly increased in the exposed workers compared to the control group (numeric data not reported). Mean predicted values of FEV_1 (forced expiratory volume in 1 second adjusted for body size) and FVC (forced vital capacity) were significantly lower, and the prevalence of abnormal values was higher in the cobalt exposed workers (both smokers and non-smokers) compared to the control group. In controls, FEV_1 and FVC were 95.4 and 101.6 percent of predicted values, respectively. Mean percent predicted FEV_1 and FVC in exposed non-smokers were 87.1 and 92.3, respectively, and in exposed smokers were 83.9 and 93.4, respectively. Among non-smokers, all measures of pulmonary function were lower in workers exposed for 5 years or more than in those exposed to cobalt for a shorter period of time.

Nemery et al. (1992) conducted a cross-sectional study of cobalt exposure and respiratory effects in diamond polishers who were primarily exposed to metallic cobalt-containing dust; species of cobalt in the dust samples were not identified. The study group was composed of 194 polishers working in 10 different workshops. In two of these workshops (#1, 2), the workers used cast iron polishing disks almost exclusively, and in the others, they primarily used cobalt-containing disks. The number of subjects from each workshop varied from 6 to 28 and the participation rate varied from 56 to 100%. The low participation in some workshops reflects the fact that only workers who used cobalt disks were initially asked to be in the study; low participation is not due to a high refusal rate (only eight refusals were documented). More than a year after the polishing workshops were studied, an additional three workshops with workers engaged in sawing diamonds, cleaving diamonds or drawing jewelry were studied as an unexposed control group (n=59 workers). Subjects were asked to fill out a questionnaire regarding employment history, working conditions, medical history, respiratory symptoms and smoking habits; to give a urine sample for cobalt determination; and to undergo a clinical examination and lung function tests. Both area air samples and personal air samples were collected (always on a Thursday). Sampling for area air determinations started 2 hours after work began and continued until 1 hour before the end of the work day. Personal air samples were collected from the breathing zone of a few workers per workshop for four successive 1-hour periods. Air samples were analyzed for cobalt and iron. In addition, personal air samplers were used to sample the air 1 cm above the polishing disks. These samples were analyzed for the entire spectrum of mineral and metallic compounds. Air samples were not obtained at one of the polishing workshops (#4); however, this workshop was reported to be almost identical to an adjoining workshop (#3) for which samples were obtained. Urinary cobalt levels were similar between workers in these two workshops, so exposure was considered to be similar as well.

Results of area and personal air sampling were strongly correlated (R=0.92), with area air sampling reporting lower concentrations than personal air samples in all workshops except one (#9) (Nemery et al., 1992). In this workshop, personal air samples appeared to be artificially low in comparison to area air samples and urinary cobalt levels of the workers. When this workshop was excluded, a strong correlation (R=0.85-0.88) between urinary cobalt and cobalt in the air was observed. Based on urinary cobalt levels, the predicted concentration of cobalt expected in personal air samples from workshop #9 was approximately 45 $\mu g/m^3$ (the mean value actually reported was 6 $\mu g/m^3$). The polishing workshops were divided into two groups: those with low exposure to cobalt (#1-5, n=102) and those with high exposure to cobalt (#6-10, n=91). Mean cobalt exposure concentrations were 0.4, 1.6 and 10.2 $\mu g/m^3$ by area air sampling and 0.4, 5.3 and 15.1 $\mu g/m^3$ by personal air sampling in the control, low-exposure and high-exposure groups, respectively. The inclusion of the apparently biased personal air samples from workshop #9 means that the reported mean cobalt exposure in the high-exposure group obtained by personal air sampling (15.1 $\mu g/m^3$) may be lower than the true value. Air concentrations of iron were highest in the two polishing workshops that used iron disks and the sawing workshop (highest value=62 $\mu g/m^3$), and were not correlated with cobalt levels. Analysis of samples taken near the disks showed the presence of cobalt, with occasional traces of copper, zinc, titanium, manganese, chromium, silicates and silicon dioxide. No tungsten was detected. Some workers may have previously been exposed to asbestos since pastes containing asbestos had been used in the past to glue the diamonds onto holders. However, since the asbestos was in its non-friable form, exposure was insufficient to produce functional impairment. Smoking habits were similar in workers from the high-exposure, low-exposure and control groups. Duration of exposure was not discussed.

Workers in the high-exposure group were more likely than those in the other groups to complain about respiratory symptoms; the prevalences of eye, nose and throat irritation and cough, and the fraction of these symptoms related to work, were significantly increased in the high-exposure group (Nemery et al., 1992). Workers in the high-exposure group also had significantly lower lung function compared to controls and low-exposure group workers, as assessed by FVC, FEV_1, MMEF (forced expiratory flow between 25 and 75% of the FVC) and mean PEF (peak expiratory flow rate), although the prevalence of abnormal values did not differ significantly between exposure categories. In controls, FVC, FEV_1 and MMEF were approximately 110, 107 and 94 percent of predicted values, respectively, compared to approximately 105, 104 and 87 percent of predicted values, respectively, in the high-exposure group workers. Results in the low-exposure group did not differ from controls. The effect on spirometric parameters in the high exposure group was present in both men and women. Women seemed to be affected more than men; however, the interaction between exposure and sex was not significant (two-way analysis of variance). Smoking was found to exert a strong effect on lung function; however, lung function level remained negatively correlated with exposure to cobalt, independent of smoking.

A cobalt dose-effect relationship is evident from the Nemery et al. (1992) study, based on a multivariate regression analysis of urinary cobalt and lung function measurements. Increasing urinary cobalt concentration (approximate range <1-70 µg cobalt/g creatinine) was significantly (p<0.05) associated with co-variate-adjusted decreasing forced expiratory volume (FEV1%) and forced vital capacity (FVC%). Significant co-variates retained in the regression analysis

included gender and smoking. The model predicted 3% and 4% decreases in FEV1% and FVC%, respectively, in association with a 10-fold increase in urinary cobalt concentration. The approximate mean urinary cobalt levels of the control and high exposure groups were 2 and 20 μg cobalt/g creatinine, respectively. The magnitude of the cobalt effect was similar to the predicted effect of smoking, approximately 3-4% decrease in FEV1% and FVC%. Cobalt concentration determined from personal air sampling may be more representative of airborne cobalt exposure than area sampling. As such, 5.3 $\mu g/m^3$ and 15.1 $\mu g/m^3$ represent a NOAEL and LOAEL, respectively, for decreased pulmonary function and increased symptoms of airway irritation.

Swennen et al. (1993) conducted a cross-sectional study of workers exposed to metallic cobalt and various inorganic cobalt salts and oxides (specific species not identified) at a cobalt plant producing these materials from cobalt metal cathodes and scrap metal. The study group included 82 male workers from the cobalt plant who had no history of lung disease prior to employment and who had never been exposed to other pneumotoxic chemicals. Methods for selection or exclusion of subjects in constructing the cohort and participation were not reported. The control group comprised 82 age-matched workers from the mechanical workshop of a nearby plant owned by the same company. Workers filled out a questionnaire regarding occupational history, respiratory complaints and smoking habits; received a routine clinical examination; participated in lung function tests; had a chest radiograph taken; and gave blood and urine samples (before and after working on Monday and Friday of one week) for determination of cobalt content as well as hematological and serum chemistry analyses. Exposure was monitored by personal air samplers worn by each cobalt worker for 6 hours on both Monday and Friday.

Workers in the cobalt plant were exposed to cobalt concentrations ranging from 1 to 7772 $\mu g/m^3$ (Swennen et al., 1993). The geometric mean exposure concentration was 125 $\mu g/m^3$. Exposure duration ranged from 0.3 to 39.4 years, with an average exposure of 8.0 years. A significantly higher number of exposed workers reported dyspnea than did controls. The increase occurred primarily among smokers although no significant interaction was found between smoking and exposure to cobalt. Based on a logistic regression model, the probability of dyspnea during exercise was significantly associated with increasing cobalt concentration in the air or urine. The parameters of the model were not reported. The clinical examinations detected significantly increased prevalence of skin disorders (eczema, erythema) (51 vs. 25%) and wheezing (16 vs. 6%) in the exposed group compared to controls. Lung function tests did not differ between the two groups; however, a few significant trends were noted: the FEV_1/VC (forced expiratory volume in one second/vital capacity) ratio decreased with increasing concentration of cobalt in the air and urine, and the RV (residual volume) and TLC (total lung capacity) increased with increasing duration of exposure. No lung abnormalities were found by chest radiographs in either group. Blood analyses did not show polycythemia, and in fact, there were slight, but significant, decreases in red blood cell count, hemoglobin and hematocrit in the exposed workers. White blood cell counts were significantly increased. Serum levels of the thyroid hormone T3 (triiodothyronine) were slightly (7%), but significantly, decreased in the exposed group, while T4 (thyroxine) and TSH (thyrotropin) were not affected. Serum markers for cardiomyopathy (i.e., myocardial creatine kinase) were unchanged.

Prescott et al. (1992) conducted a cross-sectional study to investigate the effects of cobalt exposure on thyroid volume in female plate painters. The test group included 61 female plate painters exposed to cobalt blue dyes in two porcelain factories. The control group consisted of 48 unexposed women working at the same factories. The dyes used in the two factories differed; factory I (36 workers) used cobalt aluminate, which is insoluble, and factory II (25 workers) used cobalt-zinc silicate, which was reported to be "semi-soluble." Workers were exposed to cobalt during the painting procedure when the plates were spray-painted (under a fume hood) two or three times with the water-based cobalt blue underglaze and when the excess color was removed with a brush after drying. Cobalt concentrations were reported to be approximately 0.05 mg/m^3 in the workplaces (no further details on air levels were reported). The average duration of exposure was 14.6 years in group I workers and 16.2 years in group II workers. Subjects filled out a questionnaire regarding health, use of medicines, day of menstrual cycle, employment information and smoking habits and agreed to give blood and urine samples for determination of thyroid hormone levels (e.g. thyroxine (T4), triiodothyronine (T3), and thyroid stimulating hormone) and cobalt concentration, respectively, and to undergo ultrasonography to determine volume of the thyroid gland.

Urinary cobalt levels were similar in group I exposed workers and controls (Prescott et al., 1992). Group II workers exposed to semi-soluble cobalt-zinc silicate had urinary cobalt levels that were approximately 10-fold higher than controls. Group I workers did not differ from controls for any of the thyroid parameters measured; however, Group II workers had a significant 22% increase in serum T4 (thyroxine) levels. Mean thyroid volume was lower in this group as well, although the difference from controls (16.1 mL in group II vs. 19.2 mL in controls and 18.7 mL in group I) was not statistically significant. The occurrence of respiratory effects in these workers was not reported.

Results of three studies investigating cancer incidence in workers exposed to cobalt by the inhalation route (Tuchsen et al., 1996; Mur et al., 1987; Morgan, 1983) are suggestive of a possible association between exposure to cobalt and respiratory tumors. Morgan (1983) investigated the health and causes of death of 49 men occupationally exposed to cobalt salts and oxides (specific species not identified) in a manufacturing plant in South Wales. During the study period, 33 men died (five with lung cancer and three with cancer at other sites). The expected number of deaths was 3.0 for lung cancer and 4.1 for cancers at other sites, based on national statistics, resulting in mortality ratios of 1.7 and 0.73, respectively (statistical analysis of data not reported).

Mur et al. (1987) analyzed the mortality of a cohort of 1143 workers in a plant that refined and processed cobalt and sodium. The plant workers may have been involved in multiple processing applications utilizing different forms of cobalt including cobalt chloride, oxides and other salts (specific species not identified). An increase in deaths [Standard Mortality Ratio (SMR) = 4.66; 95% confidence interval (CI) = 1.46-10.64] resulting from lung cancer was observed in workers based on four cases observed in the exposed group and one case expected based on French national statistics. In a study within the cohort that controlled for age and smoking habits, 44% (four workers) in the exposed group and 17% (three workers) in the control group died of lung cancer. The authors indicated that the differences were not statistically

significant and that the workers were exposed to arsenic and nickel in addition to cobalt. The exposure levels of cobalt were not reported.

Tuchsen et al. (1996) analyzed the cancer incidence of a cohort of 874 women who worked in one of two factories (382 from one factory, 492 from a second factory) applying a cobalt-based (cobalt-aluminate spinel) plate underglaze. From unexposed areas of factory I , 520 referents were selected. Both groups were compared to statistics for all Danish women in the same calendar year. During the 5-year follow-up period, the overall cancer incidence was only slightly elevated in exposed workers, while the incidence of lung cancers was significantly increased [Standard Incidence Ratio (SIR) = 2.35; 95% CI = 1.01-4.6]. The incidence of lung cancers in the referents (not exposed to cobalt) was greater than that of all Danish women, but the difference was not statistically significant. Exposure characterization prior to 1980 was not described, while exposures after 1980 were variable and reported as a mean concentration for a given year. Exposures were generally in the range of 0-1 mg cobalt/m^3 except for 2 years, during which they were greater.

Animal Studies

Overview

Studies in animals show that oral exposure to cobalt produces effects similar to those observed in humans, including increases in red blood cells and hemoglobin (Domingo et al., 1984; Krasovskii and Fridlyand, 1971; Murdock, 1959; Holly, 1955; Stanley et al., 1947), thyroid effects (Shrivastava et al., 1996) and cardiac effects (Haga et al., 1996; Pehrsson et al., 1991; Mohiuddin et al., 1970). Other findings in animals not reported in humans include neurobehavioral changes (Singh and Junnarkar, 1991; Bourg et al., 1985; Krasovskii and Fridlyand, 1971) and testicular toxicity (Anderson et al., 1992, 1993; Pedigo et al., 1988; Corrier et al., 1985; Mollenhauer et al., 1985; Domingo et al., 1984; Nation et al., 1983). Developmental toxicity studies in rats and mice provide evidence that high oral doses of cobalt may produce developmental effects in animals, in some cases in the absence of overt maternal toxicity (Szakmary et al., 2001; Paternain et al., 1988; Domingo et al., 1985).

Animal data support the conclusion that the respiratory tract is the critical target for inhaled cobalt (NTP, 1991; Bucher et al., 1990; Wehner et al., 1977). Subchronic inhalation exposure to cobalt resulted in cytotoxicity and reparative proliferation in all regions of the respiratory tract in rats and mice (NTP, 1991; Bucher et al., 1990). Available chronic animal studies have demonstrated the carcinogenic potential of inhaled cobalt in male and female rats and mice, with alveolar and bronchiolar tumors being the most prevalent (Bucher et al., 1999; NTP, 1998).

Oral Exposure

Studies in rats show that subchronic oral exposure to cobalt chloride increases red blood cell counts and hemoglobin levels with NOAELs ranging from 0.05 to 0.62 mg cobalt/kg-day (Krasovskii and Fridlyand, 1971; Stanley et al., 1947) and LOAELs ranging from 0.5 to 32 mg cobalt/kg-day (Domingo et al., 1984; Krasovskii and Fridlyand, 1971; Murdock, 1959; Holly,

1955; Stanley et al., 1947). In general, effects in animal studies were observed at higher exposure levels than those reported in humans.

Effects of cobalt on red blood cells and hemoglobin were investigated in Sprague-Dawley rats treated with 2.5, 10, and 40 mg cobalt chloride hexahydrate/kg-day (equivalent to 0.62, 2.5, and 9.9 mg cobalt/kg-day, respectively) for 8 weeks (Stanley et al., 1947). After 8 weeks of exposure, increases in hemoglobin and red blood cell number were observed in the 2.5 and 9.9 mg cobalt/kg-day treatment groups. Statistical significance was not reported.

Hemoglobin and hematocrit were significantly increased in male Sprague-Dawley rats exposed to 500 ppm cobalt chloride in drinking water, equivalent to approximately 32 mg cobalt/kg-day (assuming a water intake of 0.139 L/kg-day for male Sprague-Dawley rats; U.S. EPA, 1988), for 3 months (Domingo et al., 1984). Compared to controls, hematocrit and hemoglobin were both increased by approximately 30% at the end of the 3-month exposure period, with increases observed within the first 2 weeks of exposure (numeric data not presented). Following the 3-month exposure period, histopathological examination showed no treatment-related morphological or ultrastructural changes to any organ. Increased tissue weights were observed for spleen, heart and lungs, and testicular weight was decreased compared to controls. Based on the results of this study, 32 mg cobalt/kg-day was identified as a subchronic LOAEL for increased hematocrit and hemoglobin and decreased testicular weight in rats.

In rats exposed to 40 mg cobalt chloride/kg-day (equivalent to 18 mg cobalt/kg-day) for 4 months, hemoglobin and red blood cell count were increased by 37 and 21%, respectively, compared to controls (Holly, 1955). Similar effects were observed following concomitant administration of 40 mg cobalt chloride/kg-day and 200 mg ferrous sulfate, with increases of 30% for hemoglobin and 32% for red blood cell count, compared to controls. Statistical significance was not reported.

Oral exposure of rats to 10 mg cobalt/kg-day (as cobalt chloride) for 5 months resulted in increases in hemoglobin, hematocrit and red blood cell count compared to untreated controls, with effects reaching a plateau after approximately 60 days of exposure (Murdock, 1959). Statistical significance was not reported. No changes were observed for mean corpuscular hemoglobin concentration and mean cell volume compared to untreated controls, indicating that stimulation of erythropoiesis by cobalt did not result in the production of abnormal red blood cells.

The effects of exposure to 0.05, 0.5, and 2.5 mg cobalt/kg-day (as cobalt chloride) for 7 months were examined in rats (Krasovskii and Fridlyand, 1971). Treatment with 0.5 and 2.5 mg cobalt/kg-day, but not 0.05 mg cobalt/kg-day, for 7 months increased red blood cells and hemoglobin. Stimulation of hematopoiesis was more pronounced in the 2.5 mg cobalt/kg-day group than in the 0.5 mg cobalt/kg-day group, with polycythemia in the 0.5 mg cobalt/kg-day group described as mild and transient. Results of this study are difficult to evaluate since numeric data and statistical analyses were not reported.

Studies in animals have noted cardiac effects following cobalt (cobalt sulfate) exposure (Haga et al., 1996; Pehrsson et al., 1991; Mohiuddin et al., 1970), although at higher exposure levels than observed in human studies. The effect of cobalt on myocardial function was examined in rats exposed to 8.4 mg cobalt/kg-day for 16 or 24 weeks (Haga et al. 1996). After 24 weeks of exposure, decreased left ventricular systolic and diastolic function was observed. An increase in the ventricular weight to body weight ratio indicates that left ventricular hypertrophy is a contributory factor in cobalt-induced myocardial dysfunction although a mechanism was not identified. Significant effects on cardiac function were not observed following 16 weeks of exposure. In guinea pigs, exposure to 20 mg cobalt/kg-day as cobalt sulfate in the diet for 5 weeks resulted in decreased absolute and relative heart weights and a greater incidence of abnormal electrocardiograms compared to animals fed on diets not supplemented with cobalt (Mohiuddin et al., 1970). Cardiac arrhythmias, including bradycardia, and repolarization abnormalities, were observed in 65% of cobalt-treated animals compared to 5% of control animals. Cellular alterations, observed at the light and electron microscopic levels, in cardiac tissues included pericardial thickening and inflammation, myocardial degeneration and vacuolization, endocardial thickening and myofibrillar damage. In contrast, no effects on cardiac function were observed in male rats (12/group) exposed to protein-restricted diets containing 8.4 mg cobalt/kg-day for 8 weeks (Pehrsson et al., 1991). Treated rats showed a significant decrease in body weight but no differences in left ventricular function relative to animals treated with protein-restricted diets without added cobalt. Although the results from the Pehrsson et al. (1991) and Haga et al. (1996) rat studies conflict, it appears that oral cobalt-induced myocardial injury/dysfunction may have a significant time-dependence. Oral cobalt (as cobalt sulfate) at the same dose level (8.4 mg cobalt/kg-day) did not appear to alter cardiac structure or function following exposure for up to 16 weeks (Pehrsson et al., 1991; Haga et al., 1996). However, ventricular hypertrophy with a concomitant decrease in left ventricular systolic and diastolic function was observed in rats after 24 weeks of oral cobalt (Haga et al., 1996). Thus, based on the results of this study, 8.4 mg cobalt/kg-day represents a subchronic LOAEL for myocardial toxicity in rats; Based on the results of the Mohiuddin et al. (1970) study, a LOAEL of 20 mg cobalt/kg-day was identified for myocardial toxicity in guinea pigs.

Histopathological changes in the thyroid gland have been observed following exposure of female mice to 400 ppm cobalt chloride (~48 mg cobalt/kg-day, assuming an average water intake of 0.265 L/kg-day for female mice; U.S. EPA, 1988) in drinking water for 15 to 45 days (Shrivastava et al., 1996). The severity of effect increased with exposure duration. After 15 days of exposure, a reduction in thyroid epithelial cell height with degenerated nuclei and reduced amount of colloid with peripheral resorption vacuoles was observed, with more pronounced effects after 30 days of exposure. More significant degenerative changes were observed after 45 days of exposure, including necrotic epithelial cells, reduced connective tissue between follicles, lymphocytic infiltrate and larger amounts of colloid within the lumen. Based upon significant thyroid toxicity observed in this study, a LOAEL of 48 mg cobalt/kg-day was identified in mice.

Developmental effects of orally administered cobalt have been studied in rats, rabbits and mice (Szakmary et al., 2001; Pedigo and Vernon, 1993; Paternain et al., 1988; Seidenberg et al., 1986; Domingo et al., 1985; Elbetieha et al., 2008). Szakmary et al. (2001) evaluated the developmental effects of oral cobalt sulfate exposure in rats, mice and rabbits. Exposure of

pregnant rats to 5.2-21.0 mg cobalt/kg-day (oral gavage) decreased perinatal growth and survival, retarded skeletal development and produced skeletal and urogenital malformations, with a LOAEL of 5.2 mg cobalt/kg-day. Maternal toxicity (increased relative liver, adrenal, spleen weights; increased BUN, serum creatinine) was only observed at the highest dose (21.0 mg cobalt/kg-day). Thus, embryotoxicity in rats was observed at exposure levels below the LOAEL for maternal toxicity. In pregnant mice exposed to 10.5 mg cobalt/kg-day, retarded skeletal development and malformations of the eye, kidney and skeleton were observed in the absence of maternal toxicity. In pregnant rabbits exposed to 4.2 mg cobalt/kg-day, 20% mortality was observed in dams. Fetal resorptions were observed in 30% of surviving dams. Results of the studies in rats and mice provide evidence that adverse developmental effects can occur in the absence of maternal toxicity, and that rabbits are more sensitive to oral cobalt.

Domingo et al. (1985) treated pregnant female rats (15 animals/group) with 5.4 to 21.8 mg cobalt/kg-day as cobalt chloride from gestation day 14 through lactation day 21. Offspring were examined for mortality, body weight, body and tail length and general signs of toxicity after 1, 4 and 21 days of nursing. In contrast to the study by Szakmary et al. (2001), results of the Domingo et al. (1985) study reported maternal toxicity at all doses that produced adverse developmental effects (specific maternal effects observed were not reported). Fetal effects at 5.4 mg cobalt/kg-day included stunted growth of the pups of both sexes, decreased body length and tail length in male offspring and decreased spleen and liver weight in female offspring. Effects at the 10.9 mg cobalt/kg-day dose included decreased body weight in female pups, while at 21.8 mg cobalt/kg-day, decreased number of living young and decreased survival were seen. Blood parameters (liver enzymes, bilirubin, total protein, uric acid, urea, creatinine, hemoglobin and hematocrit) in pups did not show any treatment-related changes. No signs of toxicity were observed in surviving pups in any of the cobalt exposure groups.

No significant effects on fetal growth or survival were found in rats exposed to 6.2 to 24.8 mg cobalt/kg-day as cobalt chloride (oral gavage) during gestation days 6-15 (Paternain et al., 1988). The incidence of stunted fetuses was higher in the animals treated with 12.4 or 24.8 mg cobalt/kg-day (0.3 stunted fetuses per litter in the 12.4 mg cobalt/kg-day group; 1.0 stunted fetuses per litter in the 24.8 mg cobalt/kg-day group) compared to the control group (0 stunted fetuses per litter); however, the differences were not statistically significant. No treatment-related effects were observed for the number of corpora lutea, total implants, resorptions, the number of dead and live fetuses or fetal size parameters. No gross external abnormalities, skeletal malformations or other signs of fetal toxicity were observed. Maternal effects, including reduced body weight gain and food consumption and altered hematological parameters (increased hematocrit, hemoglobin and reticulocytes), were reported at all exposure levels. No fetal effects were reported in mice exposed to 81.7 mg cobalt/kg-day (oral gavage) during gestation days 8-12 (Seidenberg et al., 1986), but a significant ($p<0.05$) decrease in maternal weight was found. Additional details were not reported.

Pedigo and Vernon (1993) exposed male B6C3F1 mice to 400 ppm cobalt chloride (~45 mg cobalt/kg-day, assuming a water intake of 0.247 L/kg-day for male B6C3F1 mice; U.S. EPA, 1988) in the drinking water for 10 weeks, after which the males were mated with control females to examine for dominant lethal effects. Relative to the control group, the cobalt treatment group had a lower percentage of pregnant females (control, 29/32; cobalt, 18/31), lower number of

implantations per female (control, 8.3; cobalt, 6.5) and higher preimplantation losses (control, 0.43; cobalt, 2.4). At the end of the 10-week treatment period, sperm concentration was decreased to 15.3% and motility decreased to 18.3% of controls. Several measures of sperm velocity were also depressed relative to controls. All sperm parameters, except sperm concentration, returned to control levels 8 weeks after the cobalt exposure was terminated. The increase in preimplantation losses in the dominant lethal assay appears related to adverse effects on spermatogenesis rather than to effects on preimplantation development of embryos.

Several studies reported testicular degeneration and atrophy in rats exposed to 11.7 to 46.9 mg cobalt/kg-day as cobalt chloride for 2-3 months in the diet or in the drinking water (Anderson et al., 1992, 1993; Pedigo et al., 1988; Corrier et al., 1985; Mollenhauer et al., 1985; Domingo et al., 1984; Nation et al., 1983). Pedigo et al. (1988) exposed male CD-1 mice to 100, 200 or 400 ppm of cobalt chloride (~11.7, 23.4 or 46.9 mg cobalt/kg-day, respectively, assuming an average water intake of 0.258 L/kg-day for male mice; U.S. EPA, 1988) in the drinking water for 13 weeks. High-dose animals showed a significantly decreased testicular weight beginning at week 9 of treatment and a decreased epididymal sperm concentration by week 11 of treatment. All dose groups showed significantly decreased testicular weight and epididymal sperm concentration and increased serum testosterone levels by week 12 of exposure, with the magnitude increasing with dose. Effects on serum testosterone levels may be secondary to effects on spermatogenesis and related to inhibition of local inhibitory feed-back mechanisms. Based on the results of this study, 11.7 mg cobalt/kg-day was identified as a subchronic LOAEL for decreased testicular weight and epididymal sperm concentration in male rats.

Anderson et al. (1992, 1993) exposed groups of male CD-1 mice to 400 ppm of cobalt chloride (~46.9 mg cobalt/kg-day, assuming an average water intake of 0.258 L/kg-day for male mice; U.S. EPA, 1988) in the drinking water for up to 13 weeks. A decrease in testicular weight and a progressive degeneration of the seminiferous tubules were seen beginning at 9 weeks of exposure. Initial changes were vacuolization of Sertoli cells and abnormal spermatid nuclei, followed by sloughing of cells, shrinkage of tubules and thickened endothelium. No recovery was reported after a 20-week non-exposure recovery period. Co-administration of 800 ppm of zinc chloride provided a partial protection against the effects of cobalt. Based on the results of this study, 46.9 mg cobalt/kg-day was identified as a subchronic LOAEL for decreased testicular weight and degeneration of seminiferous tubules in male mice. Similar histology (degeneration of the testes, particularly the seminiferous tubules) was noted in Sprague-Dawley rats exposed to 20 mg cobalt/kg-day in the diet for up to 98 days (Corrier et al., 1985; Mollenhauer et al., 1985). Decreased testicular weight was seen in Sprague-Dawley rats exposed to 500 ppm cobalt chloride (~32 mg cobalt/kg-day, assuming a water intake of 0.139 L/kg-day for male Sprague-Dawley rats; U.S. EPA, 1988) for 3 months (Domingo et al., 1984).

Elbetieha et al. (2008) examined the potential effects of cobalt on male fertility in forty adult (60 day-old) male Swiss mice exposed to cobalt chloride hexahydrate via drinking water at concentrations of 200, 400, or 800 ppm for 12 weeks. Based on daily water intake reported in the study, daily average doses of cobalt chloride were estimated at 26, 47, or 93 mg/kg-day (equivalent to 6.5, 11.7, or 23 mg cobalt/kg-day); control animals received untreated tap water. Mice were observed daily for signs of clinical toxicity during the exposure period. At the end of the 12-week cobalt exposure period, male mice were separated into individual cages containing

two virgin Swiss female mice and given *ad libitum* access to food and untreated tap water. Mice were cohabitated for 10 days during which it was estimated that the females completed two estrus cycles. Male control and cobalt-treated mice were necropsied after day 10 of cohabitation and testes, seminal vesicles, epididymides and preputial glands were harvested, weighed, and prepared for analysis. The left testis and epididymis from each male mouse was processed for determination of sperm count, while the right testis was processed for histopathology. Ten days later, female mice were necropsied and examined for number of pregnancies, number of implantation sites, number of viable fetuses, total number of resorptions, and incidence rate of resorptions.

Ingestion of cobalt chloride was associated with 1/10 and 2/10 deaths in the mid- and high-dose treatment groups, respectively, during week 10 of exposure. Average body weight gain was significantly reduced in all cobalt treatment groups ($p < 0.01$). No other signs of clinical toxicity were observed in surviving male mice. Relative to the control group, the number of pregnant females mated with male mice from the mid- and high-dose groups was significantly ($p < 0.05$) reduced (control, 19/20; mid-dose, 12/18; high-dose, 7/16). The number of implantation sites was significantly ($p < 0.01$) reduced in females mated with low- and mid-dose males (control, 7.89; low-dose, 5.67; mid-dose, 5.42), and the number of viable fetuses was significantly ($p < 0.05$) reduced in females mated with males from all cobalt treatment groups (control, 7.74; low-dose, 5.0; mid-dose, 4.67; high-dose, 5.83). In addition, the total number of resorptions (control, 3/150; low-dose, 9/81; mid-dose, 9/65; high-dose, 10/45) and the number of animals with resorptions (control, 3/19; low-dose, 10/15; mid-dose, 10/16; high-dose, 5/7) were significantly ($p < 0.05$) increased in females mated with males from all three cobalt-treatment groups. Analysis of male reproductive organs revealed a significant ($p < 0.005$) decrease in absolute epididymal weight in mice of the high-dose treatment group. Testes weights were significantly ($p < 0.01$) reduced in males at all doses of cobalt, and a significant ($p < 0.005$) increase in the absolute weight of seminal vesicles of the mid- and high-dose males only. Compared to controls, testicular sperm counts and daily sperm production were decreased in the mid- and high-dose males, but not in the low-dose animals. Epididymal sperm counts were decreased in male mice from all three cobalt treatment groups. Histopathological examination of testis tissue from males of the mid- and high-dose revealed a number of abnormalities including necrosis of the seminiferous tubules and interstitium, congested blood vessels, hypertrophy of the interstitial Leydig cells, and degeneration of the spermatogonial cells; incidence rate of these observations was not reported. These testicular histopathologies were not observed in the testes of control and low-dose treated males. Based on the results of this study, a LOAEL of 6.5 mg/kg-day was identified for decreased testicular weight, epididymal sperm counts, and associated reproductive abnormalities in pregnant females. A NOAEL was not identified.

Nation et al. (1983) exposed groups (n=6) of male Sprague-Dawley rats (weighing 200-210 g) to diets containing 0, 5 or 20 mg cobalt/kg-day as cobalt chloride for a total of 69 days. Following 14 days of exposure, animals were trained for scheduled (operant) or conditioned suppression neurobehavioral tests. Other than two seizures in the same high-dose animal, no overt signs of neurotoxicity were reported at any exposure level. A trend toward a decreased response rate in the schedule training behavior was observed in both the exposed groups but only attained statistical significance in the high-dose animals near the end of the operant testing period (sessions 28-35, on exposure days 44-51). A trend toward decreased

conditioned suppression behavior did not attain statistical significance in either group. Animals exposed to 20 mg cobalt/kg-day, but not 5 mg cobalt/kg-day, showed a significantly decreased weight of the testes following 69 days of exposure. Based on the results of this study, a NOAEL of 5 mg cobalt/kg-day and a LOAEL of 20 mg cobalt/kg-day was identified for decreased testicular weight and changes in operant behavior in male Sprague-Dawley rats.

Several other studies have examined the effects of cobalt on neurobehavioral parameters (Singh and Junnarkar, 1991; Krasovskii and Fridlyand, 1971; Bourg et al., 1985). In groups of male Sprague-Dawley rats (n=8) exposed to 20 mg cobalt/kg-day as cobalt chloride for 57 days in the drinking water, cobalt enhanced behavioral reactivity to stress (the animals were less likely to descend from a safe platform to an electrified grid) (Bourg et al., 1985). Singh and Junnarkar (1991) reported a moderate reduction in spontaneous activity and mild hypothermia in rats exposed orally to cobalt chloride (approximately 8 mg cobalt/kg-day) or cobalt sulfate (approximately 35 mg cobalt/kg-day). Krasovskii and Fridlyand (1971) exposed groups of rats (number and sex not specified) to 0.05, 0.5 or 2.5 mg cobalt/kg-day as cobalt chloride for up to 7 months. Neurobehavioral tests showed that treatment with cobalt resulted in a significant (p<0.05) increase in the latent reflex period at 0.5 mg cobalt/kg and above, and a pronounced neurotropic effect (disturbed conditioned reflexes) at 2.5 mg cobalt/kg.

Inhalation Exposure

In a subchronic inhalation study, groups of 10 F344/N rats and 10 B6C3F1 mice of each sex were exposed to cobalt sulfate hexahydrate aerosol (MMAD=0.83-1.10 μm; σ_g not reported) at concentrations of 0, 0.3, 1, 3, 10 or 30 mg/m^3 (equivalent to 0, 0.067, 0.22, 0.67, 2.2 or 6.7 mg cobalt/m^3) 6 hours/day, 5 days/week for 13 weeks (Bucher et al., 1990; NTP, 1991). Although this report indicates that exposure was to cobalt sulfate heptahydrate aerosol, detailed analysis of the cobalt aerosol in the 2-year continuation study (Bucher et al., 1999; NTP, 1998) reports that the aerosol was actually composed of cobalt sulfate hexahydrate; thus, exposure to the hexahydrate form is assumed for the 13-week study. Animals were monitored for body weight and observed for clinical signs during the exposure period. Urine samples for urinalysis and cobalt determination were collected from rats prior to sacrifice. Following termination of exposure, all animals were sacrificed and necropsied. Blood samples were collected and analyzed for hematological parameters (rats and mice) and serum chemistry and thyroid function parameters (rats only). The major organs were weighed. Animals from the control and high-dose groups received comprehensive histopathological examinations, while those from the lower dose groups received more limited examinations focused on the respiratory tissues.

All rats survived until scheduled necropsy (NTP, 1991; Bucher et al., 1990). Gross evidence of toxicity was noted only in rats exposed to 6.7 mg cobalt/m^3, and they displayed clinical signs of toxicity (ruffled fur, hunched posture) and reduced body weights. Polycythemia, indicated by significant increases in red blood cell count, hemoglobin and hematocrit, was noted in males exposed to ≥0.67 mg cobalt/m^3 and females exposed to ≥2.2 mg cobalt/m^3. In addition, platelets were significantly reduced in rats of both sexes at ≥2.2 mg cobalt/m^3 and reticulocytes were increased in females at 6.7 mg cobalt/m^3. Leukocyte counts and differentials were unaffected. Serum cholesterol was significantly reduced in males at ≥2.2 mg cobalt/m^3 and females at 6.7 mg cobalt/m^3. No other serum chemistry parameters were affected, including

creatine kinase isozymes indicative of damage to cardiac muscle cells. Among the thyroid hormones, T3 (triiodothyronine) was significantly reduced in females at 2.2 mg cobalt/m^3 (83% of control) and males at 6.7 mg cobalt/m^3 (62% of control) and TSH (thyrotropin) was significantly reduced in males at 6.7 mg cobalt/m^3 (30% of control), but T4 (thyroxine) was not affected in either sex at any dose and the researchers concluded that thyroid function was not consistently affected in this study. Urinalysis revealed a dose-related increase in the number of epithelial cells and granular casts in the urine of many exposed male rats (3-7 per group exposed to $\geq$0.67 mg cobalt/m^3) but not in the urine of control male rats. The researchers interpreted this finding as indicating minimal nephropathy in exposed male rats although histopathological lesions were not detected in the kidney. No effects on sperm counts, sperm motility or the incidence of abnormal sperm were noted. Average estrus cycle of females exposed to 6.7 mg cobalt/m^3 was slightly longer than controls, but the difference was not significant. Absolute and relative lung weights were significantly increased in both male and female rats at $\geq$0.22 mg cobalt/m^3. Other organ weights were not affected by treatment. Compound-related lesions were found only in the respiratory tissues of exposed rats. Degenerative, inflammatory and regenerative lesions were found throughout the respiratory tract (see Table 2). Incidence and severity of lesions were similar in males and females. The most sensitive tissue was the larynx, with squamous metaplasia present at all exposure levels.

Among mice, 2/10 males exposed to 6.7 mg cobalt/m^3 died during the study (NTP, 1991; Bucher et al., 1990). The only clinical signs of toxicity observed were rapid breathing and skin discoloration in one of the mice that died. Body weights were reduced throughout the study in both males and females exposed to 6.7 mg cobalt/m^3. No dose-related hematological effects were found. Absolute and relative lung weights were significantly increased in male and female mice exposed to $\geq$2.2 mg cobalt/m^3. Respiratory lesions were similar to those observed in rats. As with rats, the most sensitive tissue was the larynx, with squamous metaplasia present at all exposure levels. Reproductive system effects were more prominent in mice than rats. Males had significantly decreased testicular weight (48% compared to control), decreased epididymal weight (81% compared to control), testicular atrophy consisting of loss of germinal epithelium in the seminiferous tubules and foci of mineralization and an increased percentage of abnormal sperm at 6.7 mg cobalt/m^3 (295% compared to control). Significant reductions in sperm motility of 90, 87 and 54% were observed in the 0.67, 2.2 and 6.7 mg cobalt/m^3 exposure groups, respectively (lower doses were not tested). Females had a significantly increased length of the estrus cycle at 6.7 mg cobalt/m^3 (119% longer compared to control).

Other studies in animals have also reported respiratory lesions and altered respiratory function following inhalation exposure to cobalt. Kyono et al. (1992) observed mild pulmonary lesions in rats exposed to 2.12 mg/m^3 of cobalt aerosols (generated from an aqueous suspension of ultrafine metallic cobalt particles) 5 hours/day for 4 days. Lesions were characterized by focal hypertrophy of the epithelium, abnormal macrophages, vacuolization of type I epithelial cells and proliferation of type II epithelial cells, which are indicative of an initial inflammatory response. Kerfoot et al. (1975) exposed groups of five miniature swine to 0, 0.1 or 1.0 mg/m^3 of pure cobalt metal powder for 6 hours/day, 5 days/week for 3 months. Wheezing was observed in animals from both cobalt groups after 4 weeks of exposure (numeric data not reported). Tidal volume was decreased to 73% and 64% of controls in the low and high dose groups, respectively, and total respiratory compliance was decreased relative to controls (low dose, 66%

Table 2. Rats with Selected Lesions in the 13-Week Cobalt Sulfate Inhalation Study[a]

Site	Lesion	Exposure Group (mg Cobalt (Co) per m^3)					
		Control	0.067 mg Co/m^3	0.22 mg Co/m^3	0.67 mg Co/m^3	2.2 mg Co/m^3	6.7 mg Co/m^3
Larynx	Inflammation	M: 0 F: 1	M: 2 F: 2	M: 8 [c] F: 7 [c]	M: 9 [c] F: 10 [c]	M: 9 [c] F: 10 [c]	M: 9 [c] F: 10 [c]
	Squamous metaplasia	M: 0 F: 1	M: 9 [c] F: 7 [c]	M: 10 [c] F: 10 [c]	M: 10 [c] F: 10 [c]	M: 10 [c] F: 10 [c]	M: 10 [c] F: 10 [c]
Lung	Inflammation	M: 0 F: 0	M: 0 F: 0	M: 6 [b] F: 2	M: 10 [c] F: 9 [c]	M: 10 [c] F: 10 [c]	M: 10 [c] F: 10 [c]
	Fibrosis	M: 0 F: 0	M: 0 F: 0	M: 0 F: 0	M: 0 F: 1	M: 1 F: 4 [b]	M: 10 [c] F: 5 [b]
	Bronchiolar epithelium regeneration	M: 0 F: 0	M: 0 F: 0	M: 0 F: 0	M: 0 F: 0	M: 0 F: 0	M: 7 [c] F: 5 [b]

M: number of males with lesions out of 50 animals.
F: number of females with lesions out of 50 animals.
[a] NTP, 1991; Bucher et al., 1990
[b] $p<0.05$ vs controls by Fisher exact test
[c] $p<0.01$ vs controls by fisher exact test

of control; high dose, 56% of control). Statistical significance was not reported. Examination of lung tissue by electron microscopy revealed septa thickened by collagen, elastic tissue and fibroblasts in both exposure groups, with more pronounced effects in the high dose group. Johansson et al. (1987) exposed rabbits (8/group) to 0.4 or 2 mg cobalt/m^3 as cobalt chloride, 6 hours/day, 5 days/week for 14-16 weeks. Nodular accumulation of alveolar type II cells (8/8 rabbits in both cobalt groups), abnormal accumulation of enlarged, vaculolated alveolar macrophages (5/8 in the low dose group and 8/8 in the high dose group) and interstitial inflammation (4/8 rabbits in the low dose group and 8/8 rabbits in the high dose group) were observed, with more pronounced effects in the high dose group.

The carcinogenicity of inhaled cobalt was investigated in groups of 50 F344/N rats and 50 B6C3F1 mice of each sex exposed to cobalt sulfate hexahydrate aerosol (MMAD=1.4-1.6 μm; σ_g=2.1-2.2) at concentrations of 0, 0.3, 1 or 3 mg/m^3 (equivalent to 0, 0.067, 0.22 or 0.67 mg cobalt/m^3) 6 hours/day, 5 days/week for 105 weeks (Bucher et al., 1999; NTP, 1998). Animals were monitored for body weight and observed for clinical signs during the exposure period. Following termination of exposure, all animals were sacrificed and necropsied. At necropsy, all organs and tissues were examined for gross lesions, trimmed and examined histologically.

In F344 rats, there were no changes in survival or mean body weights in males or females of any exposure group (Bucher et al., 1999; NTP, 1998). Irregular breathing was noticed more frequently in female rats exposed to 0.67 mg cobalt/m^3 than in controls or other treatment groups; no changes in clinical signs were noted in any of the treated male rats. Incidence of selected neoplasms and nonneoplastic lesions of the lung in rats is summarized in Table 3. Both male and female rats in all exposure groups showed a high incidence (94% or greater) of squamous metaplasia of the alveolar epithelium, fibrosis of the pulmonary interstitium and granulomatous inflammation, with all lesions increasing in severity with increasing exposure level. Significant increases in alveolar/bronchiolar adenomas or carcinomas were seen in high-dose male rats, while significant increases in alveolar/bronchiolar adenomas or carcinomas were

Table 3. Incidence of Selected Neoplasms and Nonneoplastic Lesions in the Respiratory Tract of Rats in the 2-Year Inhalation Study of Cobalt Sulfate[a]

Site	Lesion Type	Exposure Group (mg Cobalt (Co) per m^3)			
		Control	0.067 mg Co/m^3	0.22 mg Co/m^3	0.67 mg Co/m^3
Lung	Alveolar epithelium hyperplasia	M: 9 F: 15	M: 20 [b] F: 7	M: 20 [b] F: 20	M: 23 [c] F: 33 [c]
	Alveolar epithelium metaplasia	M: 0 F: 2	M: 50 [c] F: 47 [c]	M: 48 [c] F: 50 [c]	M: 49 [c] F: 49 [c]
	Inflammation granulomatous	M: 2 F: 9	M: 50 [c] F: 47 [c]	M: 48 [c] F: 50 [c]	M: 50 [c] F: 49 [c]
	Alveolar/bronchiolar adenoma	M: 1 F: 0	M: 4 F: 1	M: 1 F: 10 [c]	M: 6 F: 9 [c]
	Alveolar/bronchiolar carcinoma	M: 0 F: 0	M: 0 F: 2	M: 3 F: 6 [b]	M: 1 F: 6 [b]
	A/B adenoma or carcinoma	M: 1 F: 0	M: 4 F: 3	M: 4 F: 15 [c]	M: 7 [b] F: 15 [c]
	Squamous cell carcinoma	M: 0 F: 0	M: 0 F: 0	M: 0 F: 1	M: 0 F: 1
Nose	Lateral wall hyperplasia	M: 2 F: 1	M: 14 [c] F: 8 [b]	M: 21 [c] F: 26 [c]	M: 21 [c] F: 38 [c]
	Olfactory epithelium atrophy	M: 8 F: 5	M: 24 [c] F: 29 [c]	M: 42 [c] F: 46 [c]	M: 48 [c] F: 47 [c]
Larynx	Squamous metaplasia	M: 0 F: 1	M: 10 [c] F: 22 [c]	M: 37 [c] F: 39 [c]	M: 50 [c] F: 48 [c]

M: Incidence of lesions in male rats out of 50 animals.
F: Incidence of lesions in female rats out of 50 animals.
[a] Bucher et al., 1999; NTP, 1998
[b] $p<0.05$ compared to control by logistic regression test
[c] $p<0.01$ compared to control by logistic regression test

seen in the mid- and high-dose female rats. The combined incidence of alveolar/bronchiolar neoplasms (adenoma and carcinoma) in male rats and female rats was significantly greater than that in control animals, and a significant linear trend occurred in both sexes. Rats of both sexes showed treatment-related increases in hyperplasia of the lateral nasal wall, atrophy of the olfactory epithelium and squamous metaplasia of the larynx. A significant increase in the incidence of pheochromocytoma in 0.67 mg cobalt/m^3 dosed females was also noted (2/48, 1/49, 4/50 and 10/50 in control, 0.067, 0.22 and 0.67 mg cobalt/m^3 groups, respectively). A marginally increased incidence of pheochromocytoma in males exposed to 0.22 mg cobalt/m^3, but not in those exposed to 0.67 mg cobalt/m^3, was considered by the study authors not to be related to treatment.

In B6C3F1 mice, no changes in survival were observed in any exposure group (Bucher et al., 1999; NTP, 1998). Male mice exposed to 0.67 mg cobalt/m^3 showed a decreased mean body weight relative to controls from week 96 through the end of the study (105 weeks). Mean body weights of exposed female mice were generally greater than those of controls throughout the study. Irregular breathing was noted slightly more frequently in female mice exposed to 0.22 mg cobalt/m^3 than in controls or other exposed groups. Incidence of selected neoplasms and nonneoplastic lesions of the lung in mice is summarized in Table 4. A dose-related increase in

Table 4. Incidence of Selected Neoplasms and Nonneoplastic Lesions in the Respiratory Tract of Mice in the 2-Year Inhalation Study of Cobalt Sulfate[a]

Site	Lesion Type	Exposure Group (mg cobalt (Co) per cubic meter)			
		Control	0.067 mg Co/m^3	0.22 mg Co/m^3	0.67 mg Co/m^3
	Bronchus cytoplasmic vacuolization	M: 0 F: 0	M: 18 [c] F: 6 [b]	M: 34 [c] F: 31 [c]	M: 38 [c] F: 43 [c]
	Alveolar/bronchiolar adenoma	M: 9 F: 3	M: 12 F: 6	M: 13 F: 9	M: 18 [b] F: 10 [b]
	Alveolar/bronchiolar carcinoma	M: 4 F: 1	M: 12 F: 1	M: 13 F: 4	M: 18 [b] F: 9 [c]
	A/B adenoma or carcinoma	M: 11 F: 4	M: 14 F: 7	M: 19 F: 13 [c]	M: 28 [c] F: 18 [c]
Nose	Olfactory epithelium atrophy	M: 0 F: 0	M: 0 F: 2	M: 28 [c] F: 12 [c]	M: 48 [c] F: 46 [c]
	Hyperplasia	M: 0 F: 0	M: 0 F: 0	M: 0 F: 0	M: 10 [c] F: 30 [c]
Larynx	Squamous metaplasia	M: 0 F: 0	M: 37 [c] F: 45 [c]	M: 48 [c] F: 40 [c]	M: 44 [c] F: 50 [c]

M: Incidence of lesions in male mice out of 50 animals.
F: Incidence of lesions in female mice out of 50 animals.
[a] Bucher et al., 1999; NTP, 1998
[b] $p<0.05$ compared to control by logistic regression test
[c] $p<0.01$ compared to control by logistic regression test

the occurrence of cytoplasmic vacuolization of the bronchus was seen in both sexes of mice, with incidences at all exposure levels being significantly different from controls. As in rats, both sexes of mice showed a significant linear trend toward increased alveolar/bronchiolar tumors, with the 0.67 mg cobalt/m^3 male and the 0.22- and 0.67 mg cobalt/m^3 female groups attaining statistical significance. Mice of both sexes showed significantly increased incidences of squamous metaplasia of the larynx ($p<0.05$) at all exposure levels examined. In male mice, but not in females, the incidence of hemangiosarcoma was significantly elevated in animals exposed to 0.22 mg cobalt/m^3, but not in other exposure groups (2/50, 4/50, 8/50 and 7/50 in the control, 0.067, 0.22 and 0.67 mg cobalt/m^3 groups, respectively).

Wehner et al. (1977, 1979) exposed 2-month-old male Syrian golden hamsters to inhaled cobalt oxide at 0 or 10 mg/m^3 (51 animals/group), 7 hours/day, 5 days/week for approximately 15 months. The incidence of tumors in treated hamsters was not statistically different from controls. There was "limited" histopathologic and ultrastructural examination in the study. No developmental toxicity studies were located following inhalation exposure to cobalt.

Other Studies

Parenteral Administration

Heath (1956) injected groups of 10 male and 20 female rats with a single intramuscular 28 mg dose of powdered cobalt in the thigh. Injection-site sarcomas appeared in 18 (60%) of the

treated rats within 5-12 months. Similar results were observed in Wistar rats by Gilman (1962) and Gilman and Ruckerbauer (1962), with single intramuscular doses of 20 mg of cobalt oxide and cobalt sulfide. Cobalt oxide and cobalt sulfide given intramuscularly at doses twice those used in rats did not induce sarcomas in mice (Gilman and Ruckerbauer, 1962). Shabaan et al. (1977) observed a high incidence of fibrosarcomas in rats given subcutaneous injections of cobalt chloride at 40 mg/kg-day for 10 days. Tumors developed in 8-12 months. Stoner et al. (1976) tested cobalt acetate in the strain A mouse pulmonary tumor test. Groups of 20 mice/sex received three times per week intraperitoneal injections for a total of 19 cumulative doses of 0, 95, 237 or 475 mg/kg. Survival was high over the 30-week observation period, and the incidence of lung tumors in treated mice was not statistically different from controls.

Genotoxicity Studies

The genetic toxicity of cobalt was reviewed by Beyersman and Hartwig (1992) and more recently by De Boeck et al. (2003b), Hartwig and Schwerdtle (2002) and Lison et al. (2001). Cobalt compounds have generally tested negative in bacterial mutagenicity assays, with occasional positive results occurring with the addition of an exogenous metabolic system. In contrast, cobalt compounds have generally tested positive in yeast and plant cells. In mammalian cell systems, cobalt has been shown to induce DNA strand breaks, sister-chromatid exchanges and morphological cell transformation.

Results of *in vitro* studies using human peripheral blood mononucleated cells show that cobalt metal and cobalt chloride induced DNA strand breaks at non-cytotoxic concentrations (De Boeck et al., 1998, 2003a). Evidence demonstrating mutagenic activity of cobalt *in vivo* in humans is lacking. No significant change in DNA strand breaks were observed in lymphocytes from nonsmoking workers who had been occupationally exposed to cobalt or hard metal dust although a positive association was observed between DNA strand breaks and smoking (De Boeck et al., 2000).

Experimental data in animals provide evidence of genotoxicity following *in vivo* exposure to cobalt. Single oral exposure of male Swiss mice to 0, 4.96, 9.92 or 19.8 mg cobalt/kg-day, as cobalt chloride, resulted in significantly increased percentages of both chromosomal breaks and chromosomal aberrations in bone marrow cells, with significant linear trends toward increasing aberrations with increased exposure (Palit et al., 1991a,b,c,d). Thirty hours following single intraperitoneal injection of cobalt chloride at doses of 6.19, 12.4, or 22.3 mg cobalt/kg in BALB/c mice, an increase in micronucleus formation was seen in the mid- and high-dose mice but not in low-dose mice (Suzuki et al., 1993). Single injection of 12.4 mg cobalt/kg resulted in significantly increased micronucleus formation at 24 hours post-injection but not at 12, 48, 72 or 96 hours. Pedigo and Vernon (1993) reported that treatment with 400 ppm cobalt chloride (~45 mg cobalt/kg-day, assuming a water intake of 0.247 L/kg-day for male B6C3F1 mice; U.S. EPA, 1988) in the drinking water of male B6C3F1 mice for 10 weeks resulted in an increase in dominant lethal effects as indicated by changes in the number of pregnant females, percentage of live embryos and number of pre-implantation losses per female.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC ORAL RfD VALUES FOR COBALT

Indicators of human health effects following oral exposure to cobalt (Co) include increased erythrocyte production and hemogloblin levels, decreased iodine uptake by the thyroid gland, elicitation of dermatitis in sensitized individuals and cardiomyopathy. Observations in humans for effects on the heart, blood and the thyroid gland are supported by results of studies in animals. Other effects, including neurobehavioral, developmental and testicular toxicity were observed in animals and at relatively high doses; these endpoints were not considered further for the development of the subchronic or chronic provional RfD (p-RfD).

Cardiomyopathy was considered as an endpoint of concern for cobalt exposure in humans; however, it is probable that alcohol consumed in "beer-cobalt cardiomyopathy," as well as other associated factors such as nutritional deficiency, played a role in the cardiotoxic effects observed. Therefore, a dose-response relationship could not be determined for cobalt exposure from these studies. Studies in animals have noted cardiac effects following cobalt exposure at higher exposure levels than observed in human studies of "beer-cobalt cardiomyopathy." On this basis, cardiomyopathy was not selected as the critical endpoint for p-RfD derivation.

Allergic response in cobalt-sensitized workers was considered as a potential critical endpoint for the derivation of an oral p-RfD. However, the available data provide no information on the dose-response relationship of cobalt sensitization, nor is a no observable adverse effect level (NOAEL) for the elicitation of an allergic response in humans defined. Interrelationships also exist between cobalt and nickel (Ni) sensitization so that people sensitized by (Ni) may have an allergic reaction following cobalt exposure. Allergic response was, therefore, not chosen as the critical effect for p-RfD derivation.

Cobalt has been shown to induce polycythemia which is characterized by an increase in erythrocyte number and hemoglobin levels through stimulation of erythropoietin, a hormone produced primarily in the kidney. The hematological effects of cobalt treatment have been reported in healthy, non-anemic adults (Davis and Fields, 1958) and in anephric anemic dialysis patients (Taylor et al., 1977; Duckham and Lee, 1976). However, the effects observed in healthy adults were reversible and erythrocyte counts returned to pre-treatment levels within 9 to 15 days after cobalt administration was discontinued. In anephric dialysis patients, treatment with cobalt resulted in an increase in hemoglobin from levels clinically described as "anemic" to levels at or near "normal." Thus, the effect of cobalt administration in these patients was clinically beneficial. Furthermore, the results of this study are difficult to interpret due to confounding factors, including the anephric status of patients and the concomitant administration of iron. Hematologic effects of cobalt were also found in several studies in rats (Domingo et al., 1984; Krasovskii and Fridlyand, 1971; Murdock, 1959; Holly, 1955, Stanley et al., 1947), supporting the plausibility for the effects observed in humans. However, the effects in animals were generally observed at higher doses than that used in the Davis and Fields (1958) human study. It is not known whether cobalt exposure in humans at higher dose levels would increase erythrocytes sufficiently above normal physiological levels to significantly increase the risk of cardiovascular effects. Therefore, polycythemia was not chosen as the critical effect for p-RfD derivation.

Effects of cobalt on thyroidal iodine uptake were identified as an endpoint of concern in humans, based on a preliminary report by Roche and Layrisse (1956). This report showed that oral exposure to cobalt (1 mg cobalt/kg-day) for 2 weeks markedly inhibited radioactive iodine uptake in the human thyroid. In a smaller human clinical study, reduced iodine uptake was reported in 2 of 4 euthyroid patients exposed to 0.54 mg cobalt/kg-day by the oral route for up to 14 days (Paley et al., 1958). A confounding factor in this study is that one of the two subjects reported to have reduced iodine uptake had received intravenous (i.v.) cobalt in addition to oral cobalt intake. The i.v. loading dose regimen may have raised the internal concentration of cobalt to a level greater than the estimated 0.54 mg cobalt/kg-day based on oral intake alone, rendering the Paley et al. (1958) study inappropriate for consideration. Importantly, long-term cobalt exposure (up to 7 months) at 2-4 mg/kg-day in anemic children has been reported to cause goiter (Gross et al., 1954; Kriss et al., 1955; Little and Sunico, 1958). Therefore, while reduced iodine uptake is reported in humans following short-term exposures at low doses (Roche and Layrisse, 1956; Paley et al., 1958), potentially more severe thyroid lesions may occur as a function of increased duration or dose. Based on observations from rodent models of cobalt exposure, the severity of thyroid toxicity appears to be related to duration of exposure. Indeed, necrosis and inflammation of the thyroid has been reported in mice exposed to approximately 48 mg cobalt/kg-day with an increase in severity over a period of 15-45 days (Shrivastava et al., 1996).

Subchronic provisional RfD

Although cobalt exposure induces decreased radioactive iodine uptake in the thyroid (Roche and Layrisse, 1956), and polycythemia (Davis and Fields, 1958) in humans at similar daily exposure levels (1 mg/kg-day and 0.97 mg/kg-day, respectively), thyroid toxicity is chosen as the critical effect for derivation of provisional oral reference values. Cobalt-induced polycythemia and decreased iodine uptake by the thyroid were reversible following relatively short-term exposure in humans, however supporting studies indicate the potential for more severe thyroid effects (e.g., Kriss et al., 1955). The point of departure (POD) of 1 mg cobalt/kg-day for decreased iodine uptake in human thyroid is the LOAEL; dividing this POD by a composite uncertainty (UF) of 300 yields a **subchronic p-RfD of 3E-3 mg/kg-day** as follows:

Subchronic p-RfD = LOAEL ÷ UF
= 1 mg/kg-d ÷ 300
= **0.003 or 3E-3 mg/kg-day**

The composite UF of 300 is composed of three uncertainty factors: An UF of 10 for LOAEL to NOAEL extrapolation was applied because the POD is based on a LOAEL. An UF of 10 was applied due to the lack of data regarding inter-individual human variability or information on sensitive subpopulations. Specifically, because the critical study (Roche and Layrisse, 1956) for oral cobalt was based on healthy (euthyroid) adults, an UF of 10 was applied to protect sensitive human populations. The available database includes several short-term human studies, multiple developmental studies in animals and animal studies investigating hematological, cardiac, neurological, neurobehavioral, and thyroid endpoints. The lack of a multi-generation reproductive toxicity study is of particular concern because the database includes several animal studies indicating effects on sperm function and testicular degeneration which raises concerns

that cobalt exposure may affect reproductive capability. Therefore, an UF of 3 was applied to account for lack of a multi-generation toxicity study.

Chronic provisional RfD

Using the same LOAEL of 1 mg/kg-day for decreased iodine uptake in humans, and an additional UF of 10 for extrapolating from subchronic to chronic duration (composite UF of 3000), a **chronic p-RfD of 3E-4 mg/kg-day** is derived as follows:

p-RfD = LOAEL ÷ UF
= 1 mg/kg-d ÷ 1000
= **0.0003 or 3E-4 mg/kg-day**

An UF of 10 for extrapolation from subchronic to chronic duration was applied because the critical effect was chosen from a principal study of a relatively short duration (2 weeks) of oral exposure in humans. The temporal relationship between cobalt-induced decreased radioactive iodine uptake and more severe thyroid toxicity should be considered carefully. One postulated temporal relationship is that chronic exposure may have no greater effect than that resulting from short-term exposure, because if the precursor event of inhibition of iodine uptake does not occur, then there may be no change in thyroid function in the short- or long-term. Prolonged cobalt exposure could have less of an effect because of the compensatory response of the pituitary-thyroid axis to iodine deficiency, via increasing iodine uptake. However, although plausible, there are no data to suggest that this postulated temporal relationship exist for cobalt-induced thyroid toxicity. Indeed, a limited number of clinical observations primarily in children exposed to oral cobalt at doses of 2-4 mg/kg-day for up to 7 months suggest the potential for more severe thyroid toxicity (e.g., Kriss et al., 1955). In addition, cobalt may not be readily eliminated from the body; for example, the biological half-life of cobalt chloride in rats is 25 hours (Rosenberg, 1993). Therefore, an UF of 10 for extrapolation from subchronic to chronic duration was applied.

Confidence in the principal study is low-to-medium. Roche and Layrisse (1956) examined twelve subjects over a two-week exposure period. Since only a single dose level was evaluated, a NOAEL for decreased iodine uptake was not identified. Other human and animal studies support the plausibility of cobalt producing thyroid toxicity (Paley et al., 1958; Prescott et al., 1992; Shirivistava et al., 1996). Confidence in the database is low-to-medium. Although some studies (Gross et al., 1954; Kriss et al., 1955; Little and Sunico, 1957) of longer duration reported increased severity of thyroid effects (e.g., goiter) in children exposed to cobalt at higher doses (2-4 mg cobalt/kg-day), critical details of these studies are unavailable for assessment. Therefore, a temporal relationship between prolonged oral cobalt exposure and increased severity of thyroid effects in humans (or experimental animals) is not clear, based upon available data. As such, a low confidence in the provisional subchronic and chronic RfDs results.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC INHALATION RfC VALUES FOR COBALT

The human and animal database indicates that respiratory effects are sensitive endpoints of inhaled cobalt. Symptoms of respiratory tract irritation and altered pulmonary function have been widely reported in workers exposed to cobalt-containing airborne media. Of the four human epidemiology studies discussed above, the study by Nemery et al. (1992) provides the strongest basis for derivation of a provisional RfC (p-RfC). Workers in this study were exposed to lower air concentrations of metallic cobalt dust than in the studies by Gennart and Lauwerys (1990), Prescott et al. (1992) and Swennen et al. (1993). The values obtained from personal air samples from the Nemery et al. (1992) study, indicate a NOAEL of 5.3 $\mu g/m^3$ and a LOAEL of 15.1 $\mu g/m^3$. Furthermore, the Nemery et al. (1992) study demonstrated a dose-effect relationship on lung function which correlated with urinary cobalt-levels, after adjusting for effects of smoking and gender.

Animal data support the conclusion that the respiratory tract is the critical target for inhaled cobalt (NTP, 1991; Bucher et al., 1990; Wehner et al., 1977). Subchronic and chronic inhalation exposure to cobalt resulted in inflammation, fibrosis, and bronchiolar regeneration in all regions of the respiratory tract in both rats and mice (NTP, 1991, 1998; Bucher et al., 1990, 1999) at doses higher than those identified in the Nemery et al (1992) study. The NTP (1991) study further demonstrated that cobalt can produce testicular effects in male mice following inhalation exposure, but the effects were produced only at high dose levels. Oral studies have also identified the testes as a target for cobalt toxicity. Multi-generation reproduction studies following inhalation or oral exposure to cobalt are not available. Although developmental toxicity studies following inhalation exposure to cobalt are not available, oral studies provide evidence that high oral doses of cobalt may produce developmental effects in animals (Szakmary et al., 2001; Paternain et al., 1988; Domingo et al., 1985).

Decreased pulmonary function and respiratory tract irritation were identified as the co-critical effects for derivation of the subchronic and chronic p-RfCs. Assuming the personal air samples to be more representative of worker exposure than the area air samples, the study by Nemery et al. (1992) identified a NOAEL of 5.3 $\mu g/m^3$ and a LOAEL of 15.1 $\mu g/m^3$ for metallic cobalt for effects on pulmonary function (e.g. forced expiratory volume (FEV), forced vital capacity (FVC) and forced expiratory flow [referred to as MMEF]) and an increased prevalence of symptoms of respiratory tract irritation (e.g. nose/throat irritation, cough, phlegm, dyspnea). Although the LOAEL may be biased low due to inclusion of data from workshop #9, this does not affect the p-RfC derivation. A NOAEL/LOAEL approach is taken for the derivation of inhalation RfC values because the critical effect data are not amenable to benchmark dose modeling. For example, workers in the low cobalt exposure group experienced a slight but non-statistically significant increase in ventilatory function compared to controls, whereas a significant decrease in ventilatory function was observed in the high cobalt exposure group compared to both the control and the low cobalt exposure groups. The NOAEL for occupational exposure was adjusted to continuous exposure as follows:

$$5.3\ \mu g/m^3\ (10\ m^3/day\ /\ 20\ m^3/day)\ (5\ days\ /\ 7\ days) = 1.9\ \mu g/m^3$$

Using the $NOAEL_{ADJ}$ of 1.9 $\mu g/m^3$ as the POD, the subchronic p-RfC and chronic p-RfC for cobalt was derived as shown below.

Subchronic p-RfC

Dividing the $NOAEL_{ADJ}$ of 1.9 $\mu g/m^3$ by a composite UF of 100 yields a **subchronic p-RfC of 2E-5 mg/m^3** for metallic cobalt as follows:

$$\begin{aligned} \text{Subchronic p-RfC} &= NOAEL_{ADJ} \div UF \\ &= 1.9\ \mu g/m^3 \div 100 \\ &= \mathbf{0.00002\ or\ 2E\text{-}5\ mg/m^3} \end{aligned}$$

The composite UF of 100 is composed of two uncertainty factors: 10 for database insufficiencies and 10 for inter-individual variability. Nemery et al. (1992) did not report exposure duration for any worker in this study; an assumption is made that worker exposure was at least of subchronic duration. A factor of 10 was applied to account for database insufficiencies due to the lack of inhalation developmental toxicity studies and a multi-generation reproduction study. A factor of 10 was applied to account for human variability, including sensitive subgroups. Individuals with underlying respiratory diseases (asthma, chronic obstructive pulmonary disease) may be more sensitive to the respiratory effects of inhaled cobalt. This subchronic p-RfC may not be protective for people with hypersensitivity to cobalt.

Chronic p-RfC

Dividing the $NOAEL_{ADJ}$ of 1.9 $\mu g/m^3$ by a composite UF of 300 yields a **chronic p-RfC of 6E-6 mg/m^3** for metallic cobalt as follows:

$$\begin{aligned} \text{Chronic p-RfC} &= NOAEL_{ADJ} \div UF \\ &= 1.9\ \mu g/m^3 \div 300 \\ &= \mathbf{0.000006\ or\ 6E\text{-}6\ mg/m^3} \end{aligned}$$

The composite UF of 300 is composed of three uncertainty factors: 3 to account for extrapolating from an assumed subchronic exposure duration to a chronic exposure duration, 10 for database insufficiencies and 10 for human inter-individual variability. A factor of 3 is applied to account for extrapolating from an assumed subchronic to chronic exposure duration. Since Nemery et al. (1992) did not report duration for any worker in this study, it is possible that exposure duration may have been subchronic or longer for some workers. A factor of 10 is applied to account for database insufficiencies due to the lack of inhalation developmental toxicity studies and a multi-generation reproduction study. A factor of 10 is applied to account for human variability, including sensitive subgroups. Individuals with underlying respiratory diseases (asthma, chronic obstructive pulmonary disease) may be more sensitive to the respiratory effects of inhaled cobalt. This chronic p-RfC may not be protective for people with hypersensitivity to cobalt.

Confidence in the key study (Nemery et al., 1992) is low because this cross-sectional study:

- looked at only respiratory endpoints;
- included a control group that was studied more than 1 year after the exposed population;
- included a study group exposed to iron and diamond dust in addition to cobalt (and possibly to asbestos in the past);
- did not report duration of exposure; and
- encountered a number of procedural difficulties during its course (e.g., construction of control group).

Confidence in the database is medium. The choice of the critical endpoint is well supported by other studies in humans and animals. Subchronic exposure studies in rats and mice (NTP, 1991) found histopathological changes in the upper respiratory tract. Other studies in animals support these findings. Reproductive and developmental effects have not been adequately studied. Furthermore, oral studies reported large doses were required to produce reproductive or developmental effects. It would be difficult to get a large enough internal dose via inhalation to produce these effects. For these reasons, there is medium-to-low confidence in the subchronic and chronic p-RfCs.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR COBALT

Weight-of-Evidence Descriptor

Under the 2005 Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005a), cobalt sulfate (soluble) is described as "likely to be carcinogenic to humans by the inhalation route," based on both the limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in animals as shown by a statistically significant increased incidence of alveolar/bronchiolar tumors in both sexes of rats and mice, pheochromocytomas in female rats, and hemangiosarcomas in male mice (Bucher et al., 1999). While available studies in humans have suggested a possible association between exposure to cobalt and respiratory tumors in cobalt workers (Tuchsen et al., 1996; Mur et al., 1987; Morgan et al., 1983), limitations within these studies, including small numbers of subjects, inadequate exposure assessment and potential exposure to other chemicals make them inadequate for assessing the carcinogenic potential of cobalt. Studies for evaluation of the oral carcinogenic potential for cobalt were not located.

Mode-of-Action Discussion

The U.S. EPA (2005a) Guidelines for Carcinogen Risk Assessment defines mode of action as "a sequence of key events and processes, starting with the interaction of an agent with a cell, proceeding through operational and anatomical changes, and resulting in cancer formation." Examples of possible modes of carcinogenic action, in general, include mutagenic, mitogenic, anti-apoptotic (inhibition of programmed cell death), cytotoxic with reparative cell proliferation, and immunologic suppression.

While the mode of action of cobalt-induced carcinogenicity has not been determined, data suggests a number of potential biological events that might be involved including non-mutagenic genotoxicity (e.g. clastogenicity). A recent review by Lison et al. (2001) of *in vitro* and *in vivo* experiments in animal models indicates that two different mechanisms of genotoxicity may contribute to the carcinogenic potential of cobalt compounds: DNA strand breakage and inhibition of DNA repair. DNA strand breaks have been reported at non-cytotoxic concentrations in human peripheral blood monocytes (De Boeck et al., 1998, 2003a). Furthermore, oral exposure of mice to cobalt chloride resulted in significantly increased percentages of both chromosomal breaks and chromosomal aberrations in bone marrow cells (Palit et al., 1991a,b,c,d). Mechanistic studies suggest cobalt-induced oxidative stress may be involved. Exposure to cobalt compounds increases indices of oxidative stress, including diminished levels of reduced glutathione, increased levels of oxidized glutathione, increased levels of oxygen radicals and increased free-radical-induced DNA damage (Kawanishi et al., 1994; Lewis et al., 1991; Kadiiska et al., 1989; Zhang et al., 1998; Moorehouse et al., 1985). To compound the potential DNA strand breaking effects of cobalt, it appears that cobalt may also inhibit the repair of such genetic damage. A review by Hartwig and Schwerdtle (2002) concluded that cobalt may specifically target zinc finger structures in DNA repair proteins, interfering with base and nucleotide excision repair. Collectively, while data indicate that cobalt induces DNA damage and repair inhibition, there is weak evidence to suggest direct or indirect mutagenicity in bacterial or mammalian systems.

Potential for a Mutagenic Mode of Action

Key events

The precise mechanism of cobalt-induced carcinogenicity has not been fully determined. There is evidence that cobalt is capable of eliciting genotoxic effects. While evaluations for mutagenic effects in bacteria have generally yielded negative results, results in several mammalian cell systems have suggested that cobalt is genotoxic in mammalian cells. Limited data from *in vivo* animal studies show that cobalt induces genotoxic effects, including chromosomal breaks, chromosomal aberrations and micronucleus formation. The most likely mechanisms for the genotoxic effects of cobalt are DNA strand breakage and the inhibition of DNA repair.

Strength, consistency, specificity of association

Although the carcinogenic potential of inhaled cobalt has been demonstrated in rats and mice by increased incidence of alveolar/bronchiolar tumors (Bucher et al., 1999; NTP, 1998), direct evidence demonstrating that cobalt can induce mutagenic changes in cells of the respiratory tract is lacking. *In vivo* exposure to hard metal dust containing 6.3% cobalt, 84% tungsten and 5.4% carbon induced DNA strand breaks in rat type II epithelial lung cells (De Boeck et al., 2003c). Chromosome/genome mutations were observed within 12 hours of exposure to a single intratracheal instillation of 16.6 mg hard metal dust/kg body weight. Since the mutagenic potential of cobalt alone was not evaluated in this study, a causal relationship between type II epithelial cell mutations and cobalt exposure could not be established. Potential mutagenic changes in respiratory tract cells could also be mediated through activated oxygen

species released by inflammatory cells (e.g., macrophages, polymorpohnuclear neutrophils), rather than directly by cobalt (Lison et al., 2001).

Dose-response concordance

A dose-response concordance has not been established between the development of bronchoalveolar tumors and mutagenesis following inhalation exposure to cobalt. Dose-response information on mutagenicity is available for acute oral and parenteral exposure to cobalt in mice (Suzuki et al., 1993; Palit et al., 1991a,b,c,d). No carcinogenicity data are available for the oral or parenteral routes upon which to base a dose-response concordance. Furthermore, no data are available on the mutagenic potential of cobalt in respiratory tract cells following *in vitro* or *in vivo* exposure.

Temporal relationships

In vivo studies in animals show that acute oral and parenteral exposure to cobalt produces genotoxicity to bone marrow cells (Suzuki et al., 1993; Palit et al., 1991a,b,c,d). Due to the lack of data on the mutagenic potential of cobalt in respiratory tract cells, the temporal relationship between potential mutagenic mechanisms and the development of bronchoalveolar tumors cannot be assessed. Development of lung tumors in animals exposed to cobalt occurred following chronic exposure (NTP, 1998).

Biological plausibility and coherence

In vivo mutagenicity studies in mice show that oral and intraperitoneal exposure to single doses of cobalt chloride induced mutagenic changes in bone marrow cells (Suzuki et al., 1993; Palit et al., 1991a,b,c,d). Although it has been hypothesized that the bronchoalveolar tumors are the result of genotoxicity (De Boeck et al., 2003b; Hartwig and Schwerdtle, 2002; Lison et al., 2001), no direct evidence is available linking cobalt-induced mutagenesis to the development of cancer. Carcinogenicity through an indirect mutagenic mode of action may be mediated by activated inflammatory cells (macrophages, polymorpohnuclear neutrophils) (Lison et al., 2001).

Other Potential Mode(s) of Action: Cytotoxicity and Cellular Regeneration

Subchronic and chronic inhalation studies (Bucher et al., 1990, 1999; NTP, 1991, 1998) in rodents provide some evidence that cobalt causes cell injury with subsequent reparative cell proliferation, which may be involved in the development of bronchoalveolar tumors. Following inhalation exposure to cobalt sulfate hexahydrate aerosol at concentrations of 0.3 to 30 mg/m^3 (equivalent to 0.067 to 6.7 mg cobalt/m^3) for 3 months, rats and mice developed several lesions indicative of cell damage and proliferation throughout the entire respiratory tract, including nasal epithelial degeneration and metaplasia, laryngeal inflammation and metaplasia, bronchiolar epithelial regeneration and ectasia, alveolar hyperplasia and lung fibrosis (NTP, 1991; Bucher et al., 1990). Squamous hyperplasia of the larynx was the most sensitive effect (LOAEL=0.067 mg cobalt/m^3). The results of the 2-year carcinogenesis study (Bucher et al., 1999; NTP, 1998) in rats and mice revealed a statistically significant increase in combined alveolar/bronchiolar adenomas and carcinomas in the 0.67 mg cobalt/m^3 group, but not in the 0.067 and 0.22 mg

cobalt/m^3 groups for male rats and mice. In female rats and mice, a statistically significant increase in combined alveolar/bronchiolar adenomas and carcinomas was observed in the 0.22 and 0.67 mg cobalt/m^3 groups, but not in the 0.067 mg cobalt/m^3 group. In this same study, granulomatous inflammation of the lung was observed at all exposure levels (0.067, 0.22 and 0.67 mg cobalt/m^3) in rats. Other markers of cell damage and proliferation, including hyperplasia, metaplasia and fibrosis, were observed in the 0.22 and 0.67 mg cobalt/m^3 exposure groups. Compared to rats, mice appeared to be less sensitive to cobalt-induced cytotoxic changes. Results of this study show that bronchoalveolar tumors develop at exposure levels that also produce cell damage and reparative proliferation, although cell damage and repair are also observed at lower exposure levels than tumorigenesis. These observations suggest the possibility that cell injury in the respiratory tract may have preceded the development of cancers although direct evidence for this assertion is lacking.

Although limited evidence of carcinogenicity in humans is available, results of several epidemioloic studies suggest a possible association between exposure to cobalt and respiratory tumors (Tuchsen et al., 1996; Mur et al., 1987; Morgan, 1983). Subchronic exposure studies in cobalt workers show an association between cobalt exposure and diminished pulmonary function (Nemery et al., 1992; Gennart and Lauwerys, 1990). Taken together, results of studies in rodents and humans suggest that inhaled cobalt may produce a cytotoxic response in the respiratory tract that may contribute to decreases in pulmonary function and the development of bronchoalveolar tumors.

Sustained cell proliferation, in response to cytotoxicity, can be a significant risk factor for cancer (Correa, 1996). Sustained cytotoxicity and regenerative cell proliferation may result in the perpetuation of mutations (spontaneous or directly or indirectly induced by the chemical), resulting in uncontrolled growth. It is also possible that continuous proliferation may increase the probability that damaged DNA will not be repaired. No data on cobalt are available to directly evaluate the relationship between cell damage and reparative proliferation and the development of bronchoalveolar tumors.

Conclusions Regarding Cancer Mode of Action

Limited evidence supports genotoxicity and cytotoxicity followed by cellular regeneration as potential modes of action for cobalt tumorigenicity. *In vitro* and *in vivo* studies provide evidence that cobalt is capable of eliciting genotoxic effects in mammalian cells; however, two key uncertainties remain:

(1) No direct evidence linking cobalt-induced mutagenesis to the development of cancer is available and (2) the mutagenic potential of cobalt in respiratory cells has not been evaluated.

Results of the 3-month and 2-year inhalation studies in rats and mice (Bucher et al., 1990, 1999; NTP, 1991, 1998) are also consistent with the hypothesis that cobalt acts through a mode of action involving cytotoxicity and cellular regeneration, based on the observations that these effects occur following subchronic exposure and bronchoalveolar tumors develop at exposure levels that produce cytotoxicity and reparative proliferation. These observations suggest the possibility that cell injury in the respiratory tract may have preceded the development of cancers

although direct evidence for this assertion is lacking. No mode of action data are available to explain the statistically significant increases in the incidences of pheochromocytomas and hemangiosarcomas that were observed in female rats and male mice, respectively.

Because a mutagenic mode of action is plausible, but cannot be clearly established for carcinogenicity of inhaled cobalt, it is recommended that an age-dependent adjustment factor not be applied to the unit risk to account for possible age-dependence of carcinogenic potency as described in U.S. EPA (2005b).

Quantitative Estimates of Carcinogenic Risk

Oral Exposure

Human or animal studies examining the carcinogenicity of cobalt following oral exposure were not located. Therefore, derivation of an oral slope factor is precluded.

Inhalation Exposure

As available human inhalation studies were not sufficiently detailed, particularly with regards to analysis of exposure, the NTP (1998; Bucher et al., 1999) 2-year carcinogenicity study in rats and mice was chosen as the principal study for the derivation of an inhalation unit risk, based on the dose-response relationship for statistically significant increased incidences of alveolar/bronchiolar (A/B) neoplasms (adenoma and carcinoma). Although statistically significant increases in the incidences of pheochromocytomas and hemangiosarcomas were observed in female rats and male mice, respectively, these tumors were not considered for the derivation of the inhalation unit risk because a higher and more consistent response across species was observed for alveolar/bronchiolar tumors. The exposure concentrations in this study were adjusted to continuous exposure as follows:

$$Conc_{[ADJ]} = Conc \times \frac{5\ days/\ week}{7\ days/\ week} \times \frac{6\ hours/\ day}{24\ hours/\ day}$$

This adjustment resulted in duration-adjusted concentrations of 0, 0.012, 0.040 and 0.120 mg cobalt/m^3, respectively, for exposure to cobalt sulfate hexahydrate at 0.0, 0.3, 1.0 and 3.0 mg/m^3 exposure levels. Using the RDDR computer program, as specified in the RfC guidelines (U.S. EPA, 1994b), human equivalent concentrations (HECs, in mg cobalt/m^3) were calculated at each exposure level for each species and sex using body weight default values (U.S. EPA, 1994b), assuming exposure to particulates (MMAD=1.5 μm, σ_g=2.2) with effects occurring in the thoracic region of the respiratory tract. Table 5 shows the resulting HECs.

Table 5. Human Equivalent Concentrations (mg Cobalt/m^3) Corresponding to Exposure Concentrations in the NTP (1998; Bucher et al., 1999) Chronic Cancer Bioassay

Study	Male Rat	Female Rat	Male Mouse	Female Mouse
RDDR Multiplier	0.83	0.79	1.48	1.44
Control	0	0	0	0
Low	0.010	0.0095	0.018	0.017
Medium	0.033	0.032	0.059	0.058
High	0.10	0.095	0.18	0.17

All models for quantal data in the U.S. EPA Benchmark Dose (BMD) software (version 1.3.2) were fit to incidence for tumors (combined A/B adenomas and carcinomas), in rats and mice; males and females were modeled separately. All data sets modeled showed a statistical trend for increased tumor incidence with increasing exposure concentration. In accordance with the U.S. EPA (2000) BMD methodology, the default benchmark response (BMR) of 10% increase in extra risk was used as the basis for the BMD, with the BMDL represented by the 95% lower confidence limit on the BMD. Models were run using the default restrictions on parameters built into the BMD software. Table 6 shows the exposure concentration and incidence data that were modeled.

Table 7 summarizes the BMD modeling results. BMDLs shown were derived from acceptable model fits (p>0.5). As is shown in Table 7, BMDLs were similar across study groups (range: 0.011-0.035 mg/m^3). Lung tumors in female rats were chosen as the endpoint for use as a point of departure for derivation of the inhalation unit risk. The BMDL for this endpoint was the lowest for all study groups (i.e., male and female rats and mice) and was based on a model that showed a good fit to the data (p=0.84), as reflected in the proximity of the BMDL to the BMD, after dropping the high exposure group. Dropping the high exposure group is recommended according to U.S. EPA (2000) procedure when no models achieve adequate fit using all exposure levels. Although this left only two exposure levels (in addition to the control), these exposure levels are in the low-dose portion of the curve within the region of the dose-response relationship in which response is increasing with exposure level (i.e., the region of interest for deriving the point of departure) and bracket the derived BMD. Appendix A presents the results from all model runs used to support this toxicity assessment.

Table 6. Neoplasm Incidence Observed in the NTP (1998; Bucher et al., 1999) Chronic Cancer Bioassay

Animal/Strain/Site	Incidence of Neoplasms			
	Human Equivalent Concentration of Cobalt (mg/m^3)			
F-344 Rats (male)	**0**	**0.010**	**0.033**	**0.10**
Lung: A/B adenoma or carcinoma	1/50	4/50	4/48	7/50
	Human Equivalent Concentration of Cobalt (mg/m^3)			
F-344 Rats (female)	**0**	**0.0095**	**0.032**	**0.095**
Lung: A/B adenoma or carcinoma	0/50	3/49	15/50	15/50
	Human Equivalent Concentration of Cobalt (mg/m^3)			
B6C3F1 Mice (male)	**0**	**0.018**	**0.059**	**0.18**
Lung: A/B adenoma or carcinoma	11/50	14/50	19/50	28/50
	Human Equivalent Concentration of Cobalt (mg/m^3)			
B6C3F1 Mice (female)	**0**	**0.017**	**0.058**	**0.17**
Lung: A/B adenoma or carcinoma	4/50	7/50	13/50	18/50

Table 7. Summary of BMD Modeling Results for Cobalt Cancer Data

Tumor	Species	Sex	BMD (mg/m^3)	BMDL (mg/m^3)
Lung: A/B adenoma or carcinoma	rat	male	0.085	0.035
Lung: A/B adenoma or carcinoma	rat	female	0.014[a]	0.011[a]
Lung: A/B adenoma or carcinoma	mouse	male	0.026	0.015
Lung: A/B adenoma or carcinoma	mouse	female	0.038	0.023

[a] Based on control, low and middle exposure levels; high exposure level was dropped due to failure of models to achieve adequate fit using all exposure levels.

In the absence of mode of action data to inform the low dose extrapolation for cobalt, an inhalation cancer unit risk was calculated by linear extrapolation of the BMDL to zero exposure level (U.S. EPA, 2005a). The provisional **inhalation unit risk of 9 $(mg/m^3)^{-1}$** for cobalt sulfate (soluble) was calculated as follows:

Provisional Unit Risk = BMR / BMDL
= 0.1 / 0.011
= **9 $(mg/m^3)^{-1}$**

Table 8 shows continuous life-time exposure concentrations that correspond with specified risk levels (i.e., $1x10^{-4}$, $1x10^{-5}$, $1x10^{-6}$).

Table 8. Continuous Life-time Exposure Concentrations Corresponding to Specified Cancer Risk

Exposure Concentration at $1x10^{-4}$ Risk	$1.1x10^{-5}$ mg/m^3
Exposure Concentration at $1x10^{-5}$ risk	$1.1x10^{-6}$ mg/m^3
Exposure Concentration at $1x10^{-6}$ Risk	$1.1x10^{-7}$ mg/m^3

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APPENDIX A: SUMMARY OF BMD MODELING OF TUMOR INCIDENCE DATA IN MALE AND FEMALE RATS AND MICE (NTP, 1998; BUCHER ET AL., 1999)

Male rat – A/B adenoma or carcinoma:
All models show acceptable fit ($p > 0.1$)
Log-logistic model yielded best fit (highest p-value and lowest AIC)
Best estimate of BMDL = 0.035 mg/m^3

Model	p	AIC	BMD mg/m^3	BMDL mg/m^3
gamma (power ≥1)	0.502	111.12	0.087	0.043
logistic	0.446	111.52	0.099	0.066
log logistic (slope ≥1)	0.510	111.07	0.085	**0.035**
2 degree polynomial (pos betas)	0.502	111.12	0.087	0.043
1 degree polynomial (pos betas)	0.502	111.12	0.087	0.043
probit	0.453	111.47	0.098	0.063
log probit (slope ≥1)	0.357	112.11	0.104	0.064
quantal linear	0.502	111.12	0.087	0.043
quantal quadratic	0.373	111.99	0.010	0.069
weibull (power ≥1)	0.502	111.12	0.087	1.043

Output from BMD v1.3.2 is shown below:

```
==============================================================================
        Logistic Model $Revision: 2.1 $ $Date: 2000/02/26 03:38:20 $
        Input Data File: C:\PROJECTS\COBALT\BMDS\RAMALULOG.(D)
        Gnuplot Plotting File:  C:\PROJECTS\COBALT\BMDS\RAMALULOG.plt
                                          Fri Sep 09 11:46:38 2005
==============================================================================

BMDS MODEL RUN
~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~

  The form of the probability function is

  P[response] = background+(1-background)/[1+EXP(-intercept-slope*Log(dose))]

  Dependent variable = INRM
  Independent variable = ECRM
  Slope parameter is restricted as slope ≥ 1

  Total number of observations = 4
  Total number of records with missing values = 0
  Maximum number of iterations = 250
  Relative Function Convergence has been set to 1e-008
  Parameter Convergence has been set to 1e-008
```

User has chosen the log transformed model

Default Initial Parameter Values
background = 0.02
intercept = 0.683504
slope = 1

Asymptotic Correlation Matrix of Parameter Estimates

(*** The model parameter(s) -slope
have been estimated at a boundary point or have been specified by the user
and do not appear in the correlation matrix)

	background	intercept
background	1	-0.63
intercept	-0.63	1

Parameter Estimates

Variable	Estimate	Std. Err.
background	0.0398603	0.0231667
intercept	0.272287	0.592931
slope	1	NA

NA - Indicates that this parameter has hit a bound
implied by some inequality constraint and thus
has no standard error.

Analysis of Deviance Table

Model	Log(likelihood)	Deviance	Test DF	p-value
Full model	-52.8567			
Fitted model	-53.5353	1.35715	2	0.5073
Reduced model	-55.5862	5.45902	3	0.1411

AIC: 111.071

Goodness of Fit

Dose	Est._Prob.	Expected	Observed	Scaled Size	Residual
0.0000	0.0399	1.993	1	50	-0.7178
0.0100	0.0523	2.615	4	50	0.8797
0.0330	0.0797	3.827	4	48	0.09207
0.1000	0.1513	7.565	7	50	-0.2228

Chi-square = 1.35 DF = 2 p-value = 0.5099

Benchmark Dose Computation

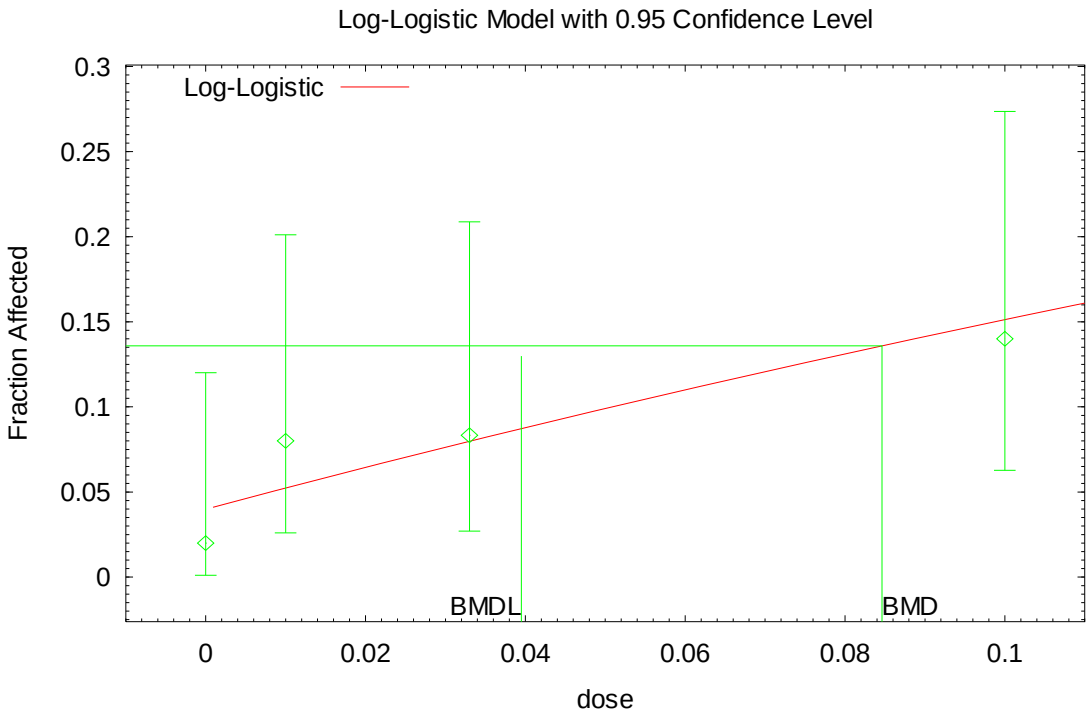
Specified effect = 0.1

Risk Type = Extra risk

Confidence level = 0.95

BMD = 0.0846262

BMDL = 0.0394914


Log-Logistic Model with 0.95 Confidence Level
Log-Logistic
Fraction Affected
0.3
0.25
0.2
0.15
0.1
0.05
0
BMDL
BMD
0
0.02
0.04
0.06
0.08
0.1
dose
09:11 09/10 2005

Female rat – A/B adenoma or carcinoma:
Most models showed poor fit ($p < 0.05$) with highest exposure level included (no increase in incidence at the highest exposure level.
The log-logistic model showed the best fit (*p*=0.11, lowest AIC)

Model	*p*	AIC	BMD mg/m^3	BMDL mg/m^3
gamma (power ≥1)	0.025	155.21	0.018	0.043
logistic	0.000	167.02	0.045	0.036
log logistic (slope ≥1)	0.090	152.86	0.015	**0.011**
2 degree polynomial (pos betas)	0.025	155.21	0.018	0.014
1 degree polynomial (pos betas)	0.025	155.21	0.018	0.014
probit	0.000	166.25	0.042	0.033
log probit (slope ≥1)	0.000	166.51	0.032	0.023
quantal linear	0.025	155.21	0.018	0.014
quantal quadratic	0.000	170.16	0.052	0.040
weibull (power ≥1)	0.025	155.21	0.018	0.014

Omitting the data from the highest exposure level improved fit of all models ($p > 0.1$)
Log-probit model yielded best fit (highest p-value and lowest AIC)
Best estimate of BMDL=0.011 mg/m^3

Model	p	AIC	BMD mg/m^3	BMDL mg/m^3
gamma (power ≥1)	1.000	87.66	0.013	0.0077
logistic	0.242	89.69	0.020	0.0164
log logistic (slope ≥1)	1.000	87.66	0.013	0.0071
2 degree polynomial (pos betas)	0.710	87.66	0.014	0.0077
1 degree polynomial (pos betas)	1.000	86.40	0.011	0.0073
probit	0.289	89.31	0.019	0.0152
log probit (slope ≥1)	0.843	85.98	0.014	**0.0110**
quantal linear	0.710	86.40	0.011	0.0073
quantal quadratic	0.535	86.70	0.017	0.0139
weibull (power ≥1)	1.000	87.66	0.014	0.0077

Output from BMD v1.3.2 (all data included) is shown below:

```
==========================================================================
        Logistic Model $Revision: 2.1 $ $Date: 2000/02/26 03:38:20 $
        Input Data File: C:\PROJECTS\COBALT\BMDS\RAFELU\RAFELULOGLOG.(D)
        Gnuplot Plotting File:
C:\\PROJECTS\COBALT\BMDS\RAFELU\RAFELULOGLOG.plt
```

Fri Sep 09 16:34:38 2005

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BMDS MODEL RUN

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The form of the probability function is

P[response] = background + (1-background)/[1+EXP(-intercept-slope*Log(dose))]

Dependent variable = INRF
Independent variable = ECRF
Slope parameter is restricted as slope $\geq$ 1

Total number of observations = 4
Total number of records with missing values = 0
Maximum number of iterations = 250
Relative Function Convergence has been set to 1e-008
Parameter Convergence has been set to 1e-008

User has chosen the log transformed model

Default Initial Parameter Values
background = 0
intercept = 1.93572
slope = 1

Asymptotic Correlation Matrix of Parameter Estimates

(*** The model parameter(s) -background -slope
have been estimated at a boundary point, or have been specified by the user,
and do not appear in the correlation matrix)

intercept

intercept 1

Parameter Estimates
~~~~~~~~~~

Variable	Estimate	Std. Err.
background	0	NA
intercept	1.98253	0.20995
slope	1	NA

NA - Indicates that this parameter has hit a bound implied by some inequality constraint and thus has no standard error.

Analysis of Deviance Table

Model	Log(likelihood)	Deviance	Test DF	P-value
Full model	-72.3723			
Fitted model	-75.4299	6.1152	3	0.1061
Reduced model	-89.3929	34.0413	3	<.0001

AIC: 152.86

Goodness of Fit

Dose	Est._Prob.	Expected	Scaled Observed	Size	Residual
0.0000	0.0000	0.000	0	50	0
0.0095	0.0645	3.162	3	49	-0.09415
0.0320	0.1885	9.427	15	50	2.015
0.0950	0.4082	20.411	15	50	-1.557

Chi-square = 6.49 DF = 3 p-value = 0.0900

Benchmark Dose Computation

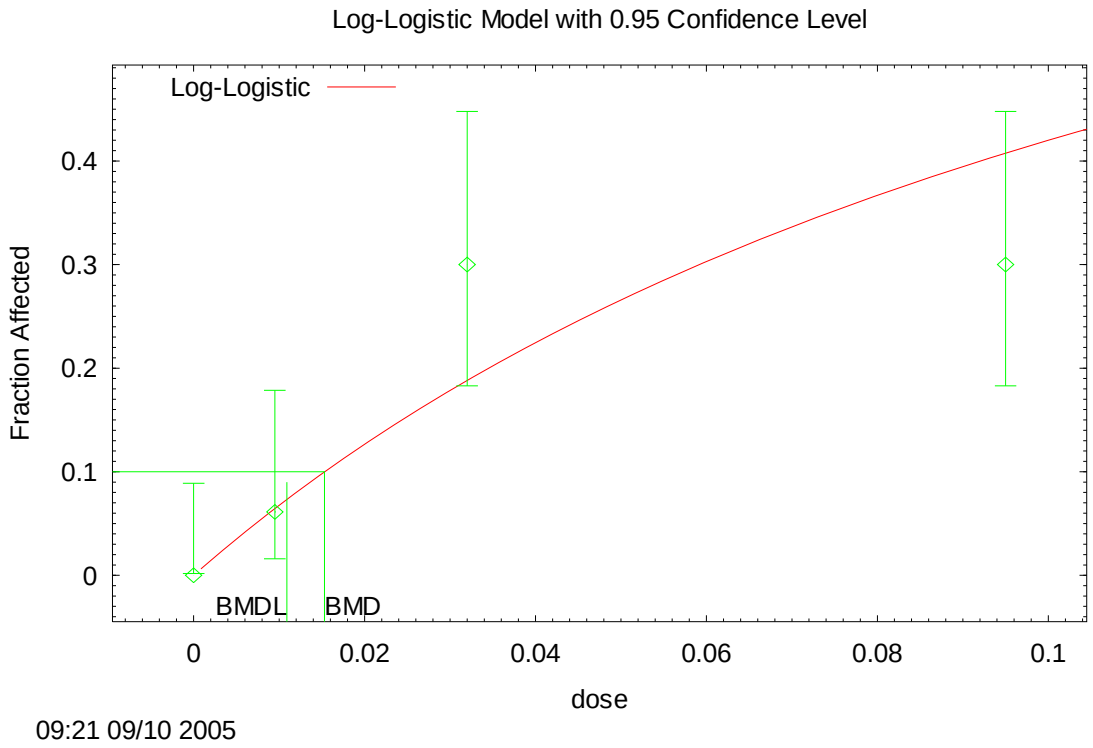
Specified effect = 0.1

Risk Type = Extra risk

Confidence level = 0.95

BMD = 0.0153022

BMDL = 0.0109172



Output from BMD v1.3.2 (highest exposure level excluded) is shown below:

```
================================================================================
        Probit Model $Revision: 2.1 $ $Date: 2000/02/26 03:38:53 $
        Input Data File:
C:\PROJECTS\COBALT\BMDS\RAFELUSE\RAFELUSEPROLOG.(D)
        Gnuplot Plotting File:
C:\PROJECTS\COBALT\BMDS\RAFELUSE\RAFELUSEPROLOG.plt
                                                  Fri Sep 09 16:41:28 2005
================================================================================

BMDS MODEL RUN
~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~

  The form of the probability function is:

  P[response] = Background
        + (1-Background) * CumNorm(Intercept+Slope*Log(Dose)),

  where CumNorm(.) is the cumulative normal distribution function
```

Dependent variable = INRF
Independent variable = ECRF
Slope parameter is restricted as slope $\geq$ 1

Total number of observations = 4
Total number of records with missing values = 1
Maximum number of iterations = 250
Relative Function Convergence has been set to 1e-008
Parameter Convergence has been set to 1e-008

User has chosen the log transformed model

Default Initial (and Specified) Parameter Values
background = 0
intercept = 3.0285
slope = 1

Asymptotic Correlation Matrix of Parameter Estimates

(*** The model parameter(s) -background -slope
have been estimated at a boundary point, or have been specified by the user,
and do not appear in the correlation matrix)

	intercept
intercept	1

Parameter Estimates

Variable	Estimate	Std. Err.
background	0	NA
intercept	2.97347	0.157916
slope	1	NA

NA - Indicates that this parameter has hit a bound
implied by some inequality constraint and thus
has no standard error.

Analysis of Deviance Table

Model	Log(likelihood)	Deviance	Test DF	P-value
Full model	-41.8291			
Fitted model	-41.9887	0.319256	2	0.8525
Reduced model	-54.9105	26.1628	2	<.0001

AIC: 85.9774

Goodness of Fit

Dose	Est._Prob.	Expected	Observed	Scaled Size	Residual
0.0000	0.0000	0.000	0	50	0
0.0095	0.0462	2.263	3	49	0.5015
0.0320	0.3197	15.985	15	50	-0.2987

Chi-square = 0.34 DF = 2 p-value = 0.8434

Benchmark Dose Computation

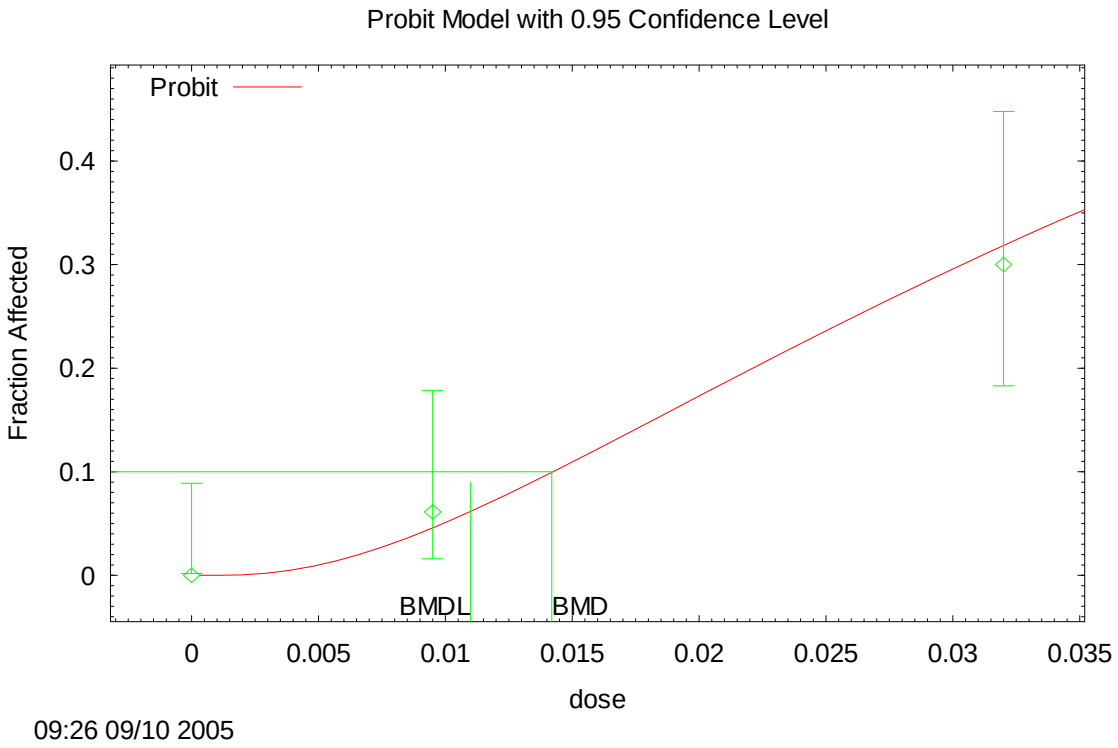
Specified effect = 0.1

Risk Type = Extra risk

Confidence level = 0.95

BMD = 0.0141927

BMDL = 0.0109984


Probit Model with 0.95 Confidence Level
Probit
Fraction Affected
0.4
0.3
0.2
0.1
0
BMDL
BMD
0
0.005
0.01
0.015
0.02
0.025
0.03
0.035
dose
09:26 09/10 2005

Male mouse – A/B adenoma or carcinoma:
All models show acceptable fit (p > 0.1)
Log-logistic model yielded best fit (highest p-value and lowest AIC)
Best estimate of BMDL = 0.015 mg/m^3

Model	p	AIC	BMD mg/m^3	BMDL mg/m^3
gamma (power ≥1)	0.944	251.10	0.033	0.0215
logistic	0.759	251.54	0.048	0.0359
log logistic (slope ≥1)	0.999	250.99	0.026	**0.0150**
2 degree polynomial (pos betas)	0.944	251.10	0.033	0.0215
1 degree polynomial (pos betas)	0.944	251.10	0.033	0.0215
probit	0.775	251.50	0.046	0.0349
log probit (slope ≥1)	0.594	252.03	0.059	0.0397
quantal linear	0.944	251.10	0.033	0.0215
quantal quadratic	0.412	252.76	0.080	0.0633
weibull (power ≥1)	0.944	251.10	0.033	0.0215

Output from BMD v1.3.2 is shown below:

```
==============================================================================
        Logistic Model $Revision: 2.1 $ $Date: 2000/02/26 03:38:20 $
        Input Data File: C:\ROJECTS\COBALT\BMDS\MOMALU\MOMALULOGLOG.(D)
        Gnuplot Plotting File:
C:\PROJECTS\COBALT\BMDS\MOMALU\MOMALULOGLOG.plt
                                        Fri Sep 09 16:57:38 2005
==============================================================================

BMDS MODEL RUN
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  The form of the probability function is

  P[response] = background+(1-background)/[1+EXP(-intercept-slope*Log(dose))]


  Dependent variable = INMM
  Independent variable = ECMM
  Slope parameter is restricted as slope ≥ 1

  Total number of observations = 4
  Total number of records with missing values = 0
  Maximum number of iterations = 250
  Relative Function Convergence has been set to 1e-008
  Parameter Convergence has been set to 1e-008
```

User has chosen the log transformed model

Default Initial Parameter Values
background = 0.22
intercept = 1.47367
slope = 1

Asymptotic Correlation Matrix of Parameter Estimates

(*** The model parameter(s) -slope
have been estimated at a boundary point, or have been specified by the user,
and do not appear in the correlation matrix)

	background	intercept
background	1	-0.62
intercept	-0.62	1

Parameter Estimates

Variable	Estimate	Std. Err.
background	0.22179	0.0478621
intercept	1.45848	0.375385
slope	1	NA

NA - Indicates that this parameter has hit a bound
implied by some inequality constraint and thus
has no standard error.

Analysis of Deviance Table

Model	Log(likelihood)	Deviance	Test DF	p-value
Full model	-123.493			
Fitted model	-123.494	0.00271986	2	0.9986
Reduced model	-130.684	14.3818	3	0.002429

AIC: 250.988

Goodness of Fit

Dose	Est._Prob.	Expected	Observed	Scaled Size	Residual
0.0000	0.2218	11.090	11	50	-0.03047
0.0180	0.2777	13.884	14	50	0.03649
0.0590	0.3793	18.963	19	50	0.0109
0.1800	0.5613	28.065	28	50	-0.0185

Chi-square = 0.00 DF = 2 p-value = 0.9986

Benchmark Dose Computation

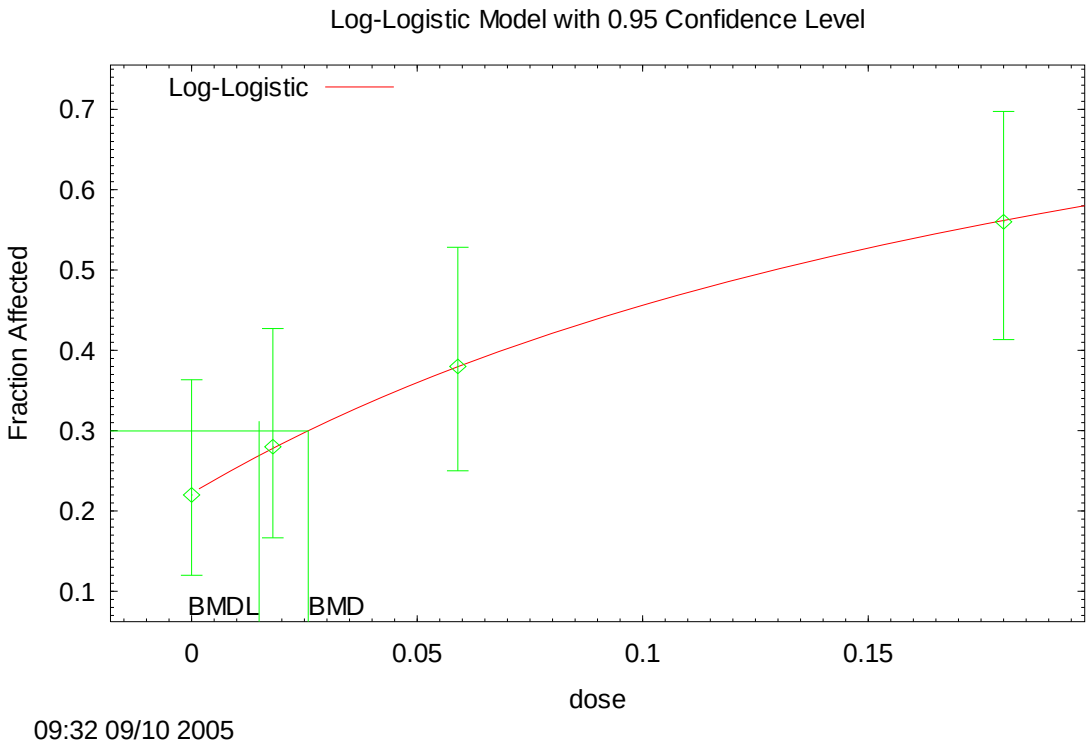
Specified effect = 0.1

Risk Type = Extra risk

Confidence level = 0.95

BMD = 0.0258434

BMDL = 0.0149697


Log-Logistic Model with 0.95 Confidence Level
Log-Logistic
Fraction Affected
0.7
0.6
0.5
0.4
0.3
0.2
0.1
BMDL
BMD
0
0.05
0.1
0.15
dose
09:32 09/10 2005

Male mouse – A/B adenoma or carcinoma:
All models show acceptable fit ($p > 0.1$)
Log-logistic model yielded best fit (highest p-value and lowest AIC)
Best estimate of BMDL = 0.023 mg/m^3

Model	p	AIC	BMD mg/m^3	BMDL mg/m^3
gamma (power ≥1)	0.571	196.12	0.0455	0.0296
logistic	0.273	197.61	0.0735	0.0562
log logistic (slope ≥1)	0.700	195.72	0.0384	**0.0231**
2 degree polynomial (pos betas)	0.571	196.12	0.0455	0.0296
1 degree polynomial (pos betas)	0.571	196.12	0.0455	0.0296
probit	0.300	197.42	0.0697	0.0528
log probit (slope ≥1)	0.167	198.57	0.0768	0.0524
quantal linear	0.571	196.12	0.0455	0.0296
quantal quadratic	0.117	199.26	0.0959	0.0739
weibull (power ≥1)	0.571	196.12	0.0455	0.0296

Output from BMD v1.3.2 is shown below:

```
================================================================================
        Logistic Model $Revision: 2.1 $ $Date: 2000/02/26 03:38:20 $
        Input Data File: C:\PROJECTS\COBALT\BMDS\MOFELU\MOFELULOGLOG.(D)
        Gnuplot Plotting File:
C:\PROJECTS\COBALT\BMDS\MOFELU\MOFELULOGLOG.plt
                                        Fri Sep 09 17:05:08 2005
================================================================================

BMDS MODEL RUN
~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~

  The form of the probability function is

  P[response] = background+(1-background)/[1+EXP(-intercept-slope*Log(dose))]


  Dependent variable = INMF
  Independent variable = ECMF
  Slope parameter is restricted as slope ≥ 1

  Total number of observations = 4
  Total number of records with missing values = 0
  Maximum number of iterations = 250
  Relative Function Convergence has been set to 1e-008
  Parameter Convergence has been set to 1e-008
```

User has chosen the log transformed model

Default Initial Parameter Values
background = 0.08
intercept = 1.17812
slope = 1

Asymptotic Correlation Matrix of Parameter Estimates

(*** The model parameter(s) -slope
have been estimated at a boundary point, or have been specified by the user,
and do not appear in the correlation matrix)

	background	intercept
background	1	-0.6
intercept	-0.6	1

Parameter Estimates

Variable	Estimate	Std. Err.
background	0.0920048	0.035283
intercept	1.06119	0.354864
slope	1	NA

NA - Indicates that this parameter has hit a bound
implied by some inequality constraint and thus
has no standard error.

Analysis of Deviance Table

Model	Log(likelihood)	Deviance	Test DF	p-value
Full model	-95.5104			
Fitted model	-95.8619	0.702985	2	0.7036
Reduced model	-102.791	14.5619	3	0.002232

AIC: 195.724

Goodness of Fit

Dose	Est._Prob.	Expected	Scaled Observed	Size	Residual
0.0000	0.0920	4.600	4	50	-0.2937
0.0170	0.1345	6.726	7	50	0.1135
0.0580	0.2223	11.117	13	50	0.6403
0.1700	0.3911	19.556	18	50	-0.451

Chi-square = 0.71 DF = 2 p-value = 0.7003

Benchmark Dose Computation

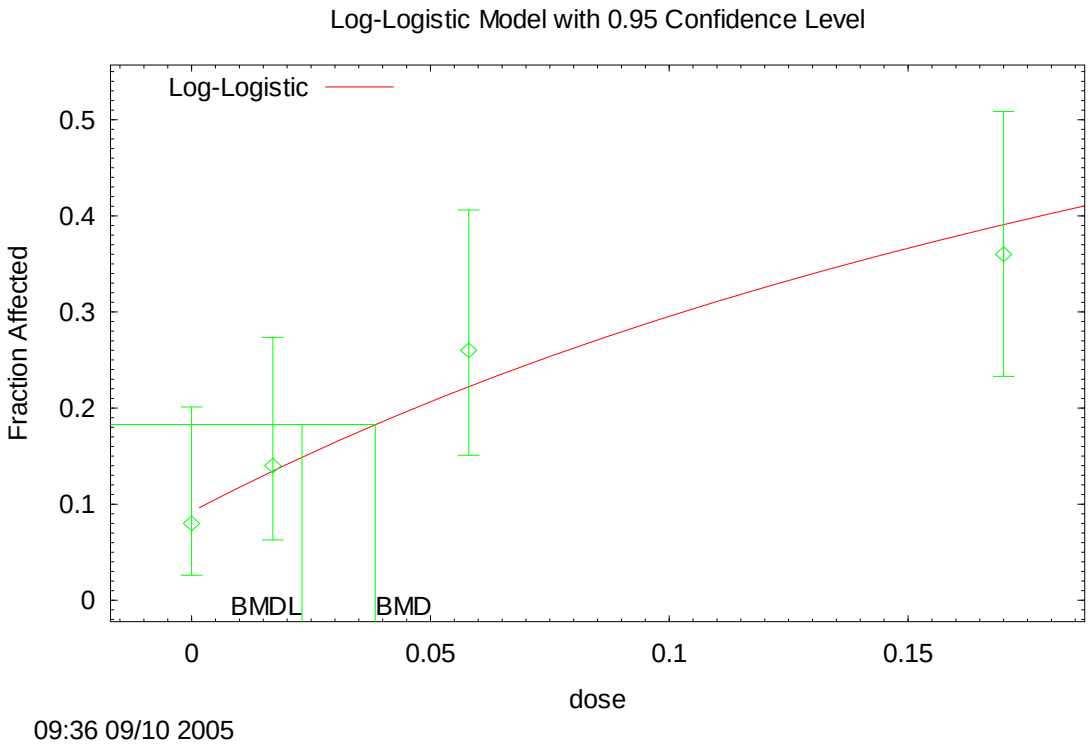
Specified effect = 0.1

Risk Type = Extra risk

Confidence level = 0.95

BMD = 0.0384492

BMDL = 0.0231


Log-Logistic Model with 0.95 Confidence Level
Log-Logistic
Fraction Affected
0.5
0.4
0.3
0.2
0.1
0
BMDL
BMD
0
0.05
0.1
0.15
dose
09:36 09/10 2005

9-11-2006

Provisional Peer Reviewed Toxicity Values for

Iron and Compounds
(CASRN 7439-89-6)

Derivation of Subchronic and Chronic Oral RfDs

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISONAL PEER REVIEWED TOXICITY INFORMATION FOR IRON (CASRN 7439-89-6) AND COMPOUNDS
Derivation of Subchronic and Chronic Oral RfDs

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).

2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.

3. Other (peer-reviewed) toxicity values, including:

   - Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
   - California Environmental Protection Agency (CalEPA) values, and
   - EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions or the EPA Headquarters Superfund Program sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI

INTRODUCTION

A reference dose (RfD) for iron is not available on the Integrated Risk Information System (IRIS) (U.S. EPA, 2006) or the Drinking Water Standards and Health Advisories list (U.S. EPA, 2005). The Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1997) reported that data regarding iron were inadequate for quantitative risk assessment. The Chemical Assessment and Related Activities (CARA) list (1991, 1994) includes a Health Effects Assessment (HEA) for Iron and Compounds (U.S. EPA, 1984) that found no reliable quantitative oral toxicity data. Iron has not been the subject of a toxicological review by the Agency for Toxic Substances Disease Registry (ATSDR) (2005) or the World Health Organization (WHO) (2005). Monographs by the International Agency for Research on Cancer (IARC) (1972, 1987), toxicity reviews by Jacobs (1977), Bothwell et al. (1979), Lauffer (1991) and Grimsley (2001), a review on dietary iron by the National Academy of Sciences (NAS) (2001), and the National Toxicology Program (NTP) (2001, 2005) management status report and chemical repository summary were consulted for relevant information. The NAS (2001) derived a Tolerable Upper Intake (TUI) level of 45 mg iron/day. The TUI is based on a minimal LOAEL of 70 mg/day (60 mg iron as ferrous fumerate plus 11 mg/day of dietary iron) identified by Frykman et al. (1994) for gastrointestinal effects and an uncertainty factor of 1.5 for use of a minimal LOAEL; a higher

uncertainty factor was not used since the nature of the observed gastrointestinal effects was considered to be self-limiting. The U.S. Food and Drug Administration (FDA) promulgated a Rule in 1997 for labeling of iron-containing dietary supplements for the prevention of accidental poisoning in children (U.S. FDA, 1997). The Rule, as modified in 2003, does not contain specific exposure limits (U.S. FDA, 2003). In general, the FDA follows the NAS guidance on exposure limits for toxicity of essential elements, such as iron. Previous literature searches were conducted through September, 2001 as follows: TOXLINE (oral and inhalation toxicity and cancer from 1983 - September, 2001); CANCERLIT (1990 - September, 2001); MEDLINE (1991 - September, 2001); TSCATS, RTECS, DART/ETICBACK, EMIC/EMICBACK, HSDB, GENETOX, and CCRIS. Update literature searches were performed in October, 2005 in MEDLINE, TOXLINE (NTIS subfile), TOXCENTER, TSCATS, CCRIS, DART/ETIC, GENETOX, HSDB, RTECS and Current Contents.

REVIEW OF PERTINENT LITERATURE

Iron is an essential element and deriving a risk assessment value for such chemicals poses a special problem in that the dose-adversity curve is "U-shaped". Thus, the risk value must be protective against deficiency as well as toxicity. The NAS (2001) has established guidelines for iron intake that take into account physiological differences during different life stages. For non-breast-fed infants aged 0-6 months, the NAS (2001) established a daily adequate intake (AI) for iron of 0.27 mg/day (0.04 mg/kg-day for infants 2-6 months old) based on the daily amount of iron secreted in human milk; breast-fed infants typically receive only 0.15 to 0.3 mg Fe/day. The NAS (2001) Dietary Reference Intakes (DRIs) for children are as follows: 11 mg/day (1.2 mg/kg-day) for infants between the ages of 7 and 12 months, 7 mg/day (0.54 mg/kg-day) for children aged 1-3 years, 10 mg/day (0.45 mg/kg-day) for ages 4-8 years, 8 mg/day (0.2 mg/kg-day) for ages 9-13 years and 11 mg/day (0.17 mg/kg-day) for boys and 15 mg/day (0.26 mg/kg-day) for girls aged 14-18 years. The DRI for men aged 19 years and above is 8 mg/day (0.11 mg/kg-day). The DRI for non-pregnant women is 18 mg/day (0.29 mg/kg-day) for ages between 19 and 50 years and 8 mg/day (0.13 mg/kg-day) for ages 51 years and older. The DRI for pregnant women is 27 mg/day (0.37 mg/kg-day for those aged 14-18 years and 0.35 mg/kg-day for those aged 19-50 years). The DRI during lactation is 10 mg/day (0.18 mg/kg-day) for women aged 14-18 years and 9 mg/day (0.15 mg/kg-day) for women aged 19-50 years.

According to the Centers for Disease Control and Prevention (CDC, 1998; CDC, 2005), iron deficiency is one of the most common known forms of nutritional deficiency. Its prevalence is highest among young children and women of childbearing age, particularly pregnant women. In children, iron deficiency causes developmental delays and behavioral disturbances, and in pregnant women, it increases the risk for a preterm delivery and delivering a low-birthweight baby. Young children are at great risk of iron deficiency because of rapid growth and increased iron requirements. Iron deficiency can occur due to lack of iron in the diet. If this continues, anemia results. Anemia is a manifestation of iron deficiency when it is relatively severe. Iron deficiency anemia significantly impairs mental and psychomotor development in infants and children. Although iron deficiency can be reversed with treatment, the reversibility of the mental and psychomotor impairment is not yet clearly understood. Thus, prevention and treatment need to be emphasized more than detection. In addition, iron deficiency increases a child's

susceptibility to lead toxicity. Lead replaces iron in the absorptive pathway when iron is unavailable.

In humans and other animals, levels in the body are regulated primarily through changes in the amount of iron absorbed by the gastrointestinal mucosa. The absorption of dietary iron is influenced by body stores, by the amount and chemical nature of iron in ingested food and by a variety of dietary factors that increase or decrease the availability of iron for absorption (Hillman, 2001; Santi and Masters, 2001). Iron contained in meat protein (hemoglobin and myoglobin) is absorbed intact without first being broken down to elemental iron. Non-heme iron must first be reduced to ferrous iron (Fe^{2+}) before it can be absorbed. Ferrous iron is transported across intestinal mucosal cells by active transport with the rate of transport inversely related to body iron stores. Depending upon the iron status of the body, iron is stored bound to ferritin within mucosal cells and macrophages in the liver, spleen and bone, or is transported in the plasma bound to transferrin. Serum levels of ferritin and transferrin, along with several red blood cell parameters, can be used clinically to evaluate iron balance. Although iron absorption is regulated, excessive accumulation of iron in the body resulting from chronic ingestion of high levels of iron cannot be prevented by intestinal regulation and humans do not have a mechanism to increase excretion of absorbed iron in response to elevated body levels (NAS, 1989, 2001).

Human Studies

Acute Exposure

Information on acute oral toxic doses of iron in humans is available from numerous case reports of ingestion by children, but values vary because it is difficult to obtain accurate estimates of the amount taken in most overdose situations. Reviews of these case reports indicate that doses in the range of 200-300 mg iron/kg are generally considered lethal (Arena, 1970; Krenzelok and Hoff, 1979; NRC, 1979; Engle et al., 1987; Mann et al., 1989; Klein-Schwartz et al., 1990).

Therapeutic Studies

Ferrous salts are administered orally for the therapeutic treatment of iron deficiency. The oral absorption of ferrous iron supplements is considered to be essentially the same for all ferrous salts (e.g., sulfate, fumarate, succinate and gluconate) and is approximately three times greater than that of ferric (Fe^{3+}) salts (Hillman, 2001); thus, ferric iron is not used therapeutically. Constipation and other gastrointestinal effects, including nausea, vomiting, diarrhea and gastrointestinal pain are commonly associated with administration of oral ferrous salt supplements (Hillman, 2001; Santi and Masters, 2001). Severity of effects is variable, ranging from mild to severe, and depends upon dose and individual susceptibility. The onset of symptoms typically occurs at the initiation of treatment and continues throughout the duration of treatment. Although there is no indication that the severity of gastrointestinal effects varies over the course of treatment, severity is decreased in some patients when iron supplements are administered with food (Hillman, 2001; Santi and Masters, 2001). For most patients, iron deficiency is reversed within six months of treatment, thus limiting the duration of exposure.

The mechanism of iron-induced gastrointestinal toxicity is not established, although it is postulated that adverse effects are due to irritant effects of the free iron ion on the gastric muscosa (Liguori, 1993). The role of absorbed iron in the development of gastrointestinal adverse effects is unknown. The adverse effects of exposure to oral iron supplements has been investigated in several studies (Blot et al., 1981; Brock et al., 1985; Coplin et al., 1991; Fryklman et al., 1994; Hallberg et al., 1966; Liguori, 1993).

Frykman et al. (1994) evaluated the adverse effects of daily oral therapy with iron fumarate in a double-blind, crossover, placebo-controlled study in Swedish male [n=25; mean age 45 years (range 40-52)] and female [n=23; mean age 41 years (range 34-45)] adult blood donors. Study subjects were administered 60 mg elemental iron as a daily dose of iron fumarate for one month, with each study subject serving as their own placebo control. Compared to the placebo treatment period, the percentage of subjects reporting constipation (placebo 20%, ferrous fumarate 35%, $p<0.05$) and total gastrointestinal symptoms (nausea, obstipation, gastric pain and diarrhea (placebo 14%, ferrous fumarate 25%, $p<0.01$) was significantly increased during ferrous fumarate treatment. Although the severity of gastrointestinal effects was graded as minor in most study subjects, four subjects withdrew from the study due to severe gastrointestinal symptoms associated with iron fumarate. In a matched group of 49 adults taking a daily combination supplement of porcine-derived heme-iron and iron fumarate containing a total daily supplement of 18 mg iron/per day, the frequency of gastrointestinal symptoms was not increased compared to placebo. No differences in therapeutic efficacy, as measured by serum ferritin and hemoglobin levels, were observed between the non-heme iron and heme-iron treatment groups.

Adverse effects of four oral iron preparations were evaluated in 1496 male and female adult blood donors in a series of double-blind, placebo controlled trials (Hallberg et al., 1966). The following treatment groups were compared: (1) placebo (195 subjects) and ferrous sulfate (198 subjects; 222 mg elemental iron/day); (2) placebo (199 subjects), ferrous sulfate (120 subjects; 222 mg elemental iron/day), ferrous fumarate (118 subjects, 222 mg elemental iron/day), and ferrous gluconate (120 subjects; 222 mg elemental iron/day); and (3) placebo (200 subjects), ferrous sulfate (195 subjects; 180 mg elemental iron/day), ferrous glycine sulfate (200 subjects; 180 mg elemental iron/day), and ferrous gluconate (196 subjects; 180 mg elemental iron/day). Treatments were administered for two weeks. For all iron treatments, the frequency of adverse gastrointestinal effects was significantly increased compared to the matched placebo group ($p<0.05$). Adverse effects reported include constipation, diarrhea, heartburn, nausea and epigastric pain. No statistically significant differences in the frequency of adverse effects were observed between iron treatments for subjects receiving 222 mg elemental iron/day or between iron treatments for subjects receiving 180 mg elemental iron/day. In the seven iron treatment groups, the percentage of subjects reporting gastrointestinal effects ranged from 22.9% in the 222 mg ferrous sulfate group to 31.5% in the 222 mg ferrous gluconate group. In the three placebo treatment groups, the percentage of subjects reporting gastrointestinal effects ranged from 12.4 to 13.6%. Although statistical comparisons were not made between the 180 and 222 mg iron/day treatments, the frequency of adverse effects was similar for all iron treatment groups.

Gastrointestinal symptoms were reported in pregnant women treated daily with oral iron supplements containing 105 mg elemental iron and 500 mg ascorbic acid (55 women) or 105 mg

elemental iron, 500 mg ascorbic acid and 350 mg folic acid (54 women) during the third trimester of pregnancy (Blot et al., 1981). The form of iron was not reported. No placebo control group was included. Gastrointestinal adverse effects reported include nausea, diarrhea, constipation and epigastric pain. Approximately 16% of all patients reported minor gastrointestinal symptoms, 14% reported severe effects and 6% stopped treatment due to adverse effects. Adverse effects occurred with approximately the same frequency in the two treatment group, although data were not reported.

The tolerability of iron protein succinylate and ferrous sulfate were compared in a double-blind clinical trial in 1095 patients with iron deficiency (Liguori, 1993). Patients received daily treatment with a controlled-release formulation of ferrous sulfate containing 105 mg elemental iron (64 males and 485 females) or iron protein succinylate containing 120 mg elemental iron (55 males and 491 females) for 60 days. No placebo control group was included. In the ferrous sulfate group, 26.3% of patients reported adverse gastrointestinal effects (heartburn, epigastric pain, constipation and abdominal pain), compared to 11.5% of patients treated with iron protein succinylate ($p<0.05$).

The adverse effects of oral treatment with a conventional ferrous sulfate tablet were compared to a ferrous sulfate wax-matrix tablet in a single-blind, parallel group study in 543 subjects (Brock et al., 1985). No placebo control group was included. Subjects were administered a conventional ferrous sulfate table containing 50 mg elemental iron/day (272 subjects) or a sulfate wax-matrix tablet containing 50 mg elemental iron/day (271 subjects) for 56 days. Approximately 45% of subjects treated with conventional ferrous sulfate reported moderate-to-severe gastrointestinal effects, including abdominal discomfort, nausea, vomiting, constipation and diarrhea, compared to approximately 17% of subjects treated with the ferrous sulfate wax-matrix preparation, a statistically significant difference ($p<0.001$).

The tolerability of ferrous sulfate (50 mg elemental iron/day) and bis-glycino iron II (50 mg elemental iron/day) was compared in a double-blind, crossover trial in 42 women (Coplin et al., 1991). The treatment period for each iron supplement was two weeks. No placebo treatment period was included. The frequency of adverse gastrointestinal effects (abdominal pain, bloating, constipation, diarrhea and nausea) was similar for the two treatments, with 54% and 59% of subjects reporting gastrointestinal symptoms during treatment with bis-glycino iron II and ferrous sulfate, respectively. The difference between treatments was not statistically significant.

Effects of iron therapy on the upper gastrointestinal tract were evaluated in 14 healthy volunteers [13 women, 1 man; mean age 29 years (range: 24-48 years)] who were instructed to ingest 325 mg tablets of ferrous sulfate (119.5 mg elemental iron) three times/day before meals (358.5 mg elemental iron/day) for 2 weeks (Laine et al., 1988). Evaluation consisted of a gastrointestinal symptom survey, qualitative (Hemoccult) and quantitative (HemoQuant; mg mercury/g stool) testing for fecal blood loss, endoscopy of the upper gastrointestinal tract and histological examination of pinch biopsies of the gastric body, antrum and duodenum. Based on actual average ingestion of 2.5 tablets/day (2-week study) and 2.6 tablets/day (1-week study) and a reference human body weight of 70 kg (U.S. EPA, 1987), the estimated doses consumed by the subjects were 4.3 and 4.4 mg iron/kg-day, respectively, in addition to dietary iron. Compared to

baseline measurements in the two weeks prior to treatment, all subjects had significantly increased ($p<0.05$) dark brown-black stools and symptoms of nausea and vomiting during the treatment period, but not abdominal pain. Hemoglobin levels in stool did not change significantly after iron treatment. Endoscopic examination showed a significant ($p=0.003$) increase in abnormalities in the stomach, but not duodenum, after therapy. These changes consisted of erythema, small areas of subepithelial hemorrhage and solitary antral erosions in nine, six and two subjects, respectively, and were considered only minimally abnormal. No treatment-related histological changes were observed. Although it was speculated that the changes in the stomach could represent a mild form of iron poisoning, the investigators concluded that the treatment caused mild endoscopic abnormalities of uncertain clinical significance in the stomach. Evidence for iron overload (tissue biopsies or hematologic iron status indices) was not examined. Considering additional dietary exposure, an exposure level of about 4.3 mg/kg-day represents, at worst, a minimal LOAEL.

Adverse developmental effects in humans have not been associated with the ingestion of supplemental iron during pregnancy. As indicated above, NAS (2001) recommended that pregnant women supplement their diets with 27 mg iron/day (0.35 mg/kg-day). McElhatton et al. (1991) reported on 49 women who took an overdose of a simple iron preparation (53%) or iron with folate preparation (47%). In 48 of the women, the amount of iron ingested was known; 28 took > 1.2 g and the remainder took $\leq$ 1.2 g. There were 25 women who received chelation treatment with desferrioxamine (DFO) and 12 who received an emetic. Maternal toxicity, consisting of nausea, vomiting, hematoemesis, abdominal pain and diarrhea, was observed in 35 of the women. Two spontaneous abortions occurred and there were three premature deliveries. One of the spontaneous abortions and the premature deliveries were not related to the iron overdose. It is not known if the other spontaneous abortion occurring at 22 weeks (3 weeks after the overdose) was caused by the iron overdose. No conclusions on the developmental toxicity of iron can be made.

Chronic Exposure

While chronic iron toxicity occurs in people with genetic metabolic disorders resulting in excessive iron absorption or abnormal hemoglobin synthesis, or who receive frequent blood transfusions (Jacobs, 1977; Bothwell et al., 1979), there is a long-standing controversy as to whether a chronic overload due to oral intake is possible in individuals with a normal ability to control iron absorption (Hillman and Finch, 1985). Nevertheless, "the cumulative experience in human subjects suffering from iron overload of various etiologies strongly suggests that iron is noxious to tissues [when]...present in parenchymal cells...for a sufficiently long period of time" (Bothwell et al., 1979).

Looker et al. (1988) made comparisons of dietary iron intake and biochemical indices of iron status based on values taken from the second National Health and Nutrition Examination Survey (NHANES II) data base[1]. NHANES II was a probability sample of the noninstitutionalized U.S. population aged 6 months to 74 years, conducted between 1976 and

[1] The latest version of this data base, NHANES III (1984-1988) evaluated 30,000 subjects aged 2 months and above (NAS, 2001). Despite minor differences in the data sets, the conclusions drawn by Looker et al. (1988) based on NHANES II appear to be valid for the NHANES III data.

1980 by the National Center for Health Statistics. These data suggest that normal intake of iron by men 16-74 years old exceeds the DRI, and that iron intake is somewhat lower than the DRI for women younger than 51 years. Concomitant with the study of dietary intake, the NHANES II measured the iron status of these populations. The percent serum transferrin saturation, a measure of the residual capacity of the iron transport system to process potential variations in iron from dietary intake or catabolized body stores, ranged from 24% saturation for pre- and post-menopausal women not using iron supplements to 29% saturation for adult male supplement users. These values are within the normal range (20-40%). The Looker et al. (1988) evaluation of the NHANES II iron status data concerned iron deficiencies, only, and did not address iron overload directly. However, iron overload conditions would likely be evidenced by increased saturation of serum transferrin and increased serum ferritin concentrations, which were also within the normal range. Therefore, the corresponding dietary intakes are presumed to represent chronic NOAELs. Looker et al. (1988) estimated daily iron intakes ranging from 10.0 for elderly women to 18.7 mg/day for young adult men in the study population. These daily intakes correspond to a range of about 0.15 to 0.27 mg/kg-day, depending on assumptions of average body weight. Taking the highest intake level of 18.7 mg/day and a body weight of 70 kg, a NOAEL of 0.27 is established for chronic iron toxicity.

Hemosiderosis (or siderosis) and iron overload are increases in tissue iron or a general increase in iron stores without associated tissue damage (Bothwell et al., 1979; Jacobs, 1977). Hemochromatosis describes massive iron overload (15 g of body iron stores or greater) together with cirrhosis and/or other tissue damage attributable to iron. Although focal deposits of iron may occur in any part of the body where red cells are extravasated, the clinical syndrome of hemochromatosis typically involves damage to the hepatic parenchyma (particularly fibrosis), heart (cardiac dysfunction including failure) and endocrine glands (particularly hypogonadism). Pancreatic iron deposition is common and massive deposits may be associated with fibrosis and diabetes. A number of studies involving chronic oral administration of iron to animals have been designed in an attempt to identify an animal model for hemochromatosis. Most of these studies have been negative (Bothwell et al., 1979; NRC, 1979). Animal studies involving parenteral administration of iron have been generally negative as well, even though parenteral routes bypass the mechanisms that regulate absorption of iron from the gastrointestinal tract.

Chronic iron toxicity has been observed in people with idiopathic hemochromatosis (a genetic metabolic disorder resulting in excessive iron absorption), abnormalities of hemoglobin synthesis (e.g., thalassemia) or various anemic states (e.g., sideroblastic anemia), frequent blood transfusions or a combination of these conditions (Jacobs, 1977; Bothwell et al., 1979). Chronic hemochromatosis has also occurred among the South African Bantu population from an excessive intake of absorbable iron in an alcoholic beverage.

Habitual excessive intake of iron by the Bantus is attributed to consumption of home-brewed Kaffir beer, which was contaminated by iron vessels during brewing (Bothwell and Bradlow, 1960; Bothwell et al., 1964). The beer's high acidity (pH 3-3.5) enhanced iron leaching from the vessels. The iron in the beer is readily assimilable (i.e., ionizable) due to the acidity and presence of iron-complexing ligands such as fructose, and is absorbed to approximately the same degree as ferric chloride. The alcohol content of the beer is also believed to contribute to the bioavailability of the iron (Jacobs, 1977; Finch and Monsen, 1972). Based

primarily on drinking habits and analyses of beer samples, the estimated average dietary iron intake of the Bantu men ranged from 50-100 mg/day from beer alone (Bothwell et al., 1964). Using a reference body weight of 70 kg (U.S. EPA, 1987), this range corresponds to 0.7-1.4 mg/kg-day. Histological examinations of the liver of 147 Bantus (129 male, 18 female) ranging in age from 11-70 years (most were between 20 and 50 years old) that died from acute traumatic causes were performed (Bothwell and Bradlow, 1960). Varying degrees of hepatic siderosis were observed in 89% of the cases; the degree tended to increase with age 40-50 years or less. The siderosis was mild in 59% and severe in 19% of the cases, respectively. There was a close correlation between hepatic iron concentration and portal fibrosis and cirrhosis. Although the overall prevalence was low (15.6% fibrosis and 1.4% cirrhosis), all 11 subjects with the highest iron concentrations (>2.0% dry weight of liver) showed either fibrosis or cirrhosis. Histological examination of the spleen (50 subjects) also showed siderosis and unspecified histological changes. Malnutrition and alcoholism could have played a role in the etiology of the hepatic and splenic siderosis in the Bantus. A NOAEL in the range of 0.7 - 1.4 mg/kg-day is indicated but may be low given the likely higher bioavailability of iron in the beer than for normal dietary exposure. Given the generally poor nutritional health status of this population, the relevance of this study for application to the U.S. population is questionable.

Ethiopia reportedly has the highest per capita iron intake in the world, with an average daily intake of 471 mg iron/day (range 98-1418 mg/day; 1.4-20.3 mg iron/kg-day assuming 70 kg body weight) (Roe, 1966; Hofvander, 1968). Increased stored iron in the liver and adverse health effects have not been observed due to low bioavailability of the iron in Ethiopian food.

A few studies have suggested that high iron intake may be a risk factor for myocardial infarction (Salonen et al., 1992; Lauffer, 1991; Sullivan, 1992). Five other large studies found no association between serum ferritin levels and coronary heart disease (NAS, 2001). Various other measures of iron status (serum transferrin saturation, serum iron concentration and total iron-binding capacity) have been examined for a possible link to cardiovascular disease in prospective cohort studies, but results overall have been characterized as contradictory (Meyers, 1996; NAS, 2001). The NAS (2001) concluded that the available evidence "does not provide convincing support for a causal relationship" between the level of dietary iron intake and the risk for coronary heart disease, although iron cannot be definitively excluded as a risk factor.

Animal Studies

Repeated-dose oral studies in experimental animals found no significant effect of treatment with inorganic iron compounds. No treatment-related adverse changes in clinical signs, body or organ weights, food consumption or histopathology were observed in male Sprague-Dawley rats that had daily dietary intakes of 35, 70 or 140 mg of iron (as $FeSO_4$ or FeEDTA) per kg for up to 61 days (Appel et al., 2001). In male and female F344 rats that were exposed to drinking water containing 0.25 or 0.5% ferric chloride ($FeCl_3 \cdot 6H_2O$) for 104 weeks, there were no dose-related effects other than reduced water intake (possibly affected by palatability) and body weight gain (Sato et al., 1992). In the latter study, the iron intakes were 58 or 110 mg/kg-day in males and 65 or 116 mg/kg-day in females.

No treatment-related teratogenic or embryotoxic effects were observed in rats given 2.7 mg iron/kg-day as ferric chloride on gestational days 6-15 (Nolen et al., 1972), or in rats and mice given 24-76 mg iron/kg-day as ferrous sulfate for 6 days during gestation (days unspecified) (Tadokoro et al., 1979). Some embryonic mortality (numbers and species not reported) occurred in the latter study at 240 mg iron/kg-day.

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC RfDs FOR IRON

Iron is an essential element, as such, the RfD must be protective against both toxicity and deficiency. Using the values for dietary intake and iron status indices taken from the second National Health and Nutrition Examination Survey (NHANES II) data base, it is possible to establish a NOAEL for chronic toxicity. Looker et al. (1988) made comparisons of dietary iron intake and biochemical indices of iron status using data from NHANES II. The average intakes of iron ranged from 0.15 to 0.27 mg/kg-day. The serum ferritin levels and percent serum transferrin saturation were within the normal range. Thus, intake levels of 0.15-0.27 mg/kg-day are sufficient to protect against iron deficiency. However, the NHANES II data do not provide information to identify daily dietary iron intakes associated with toxicity. Therefore, daily dietary iron intakes were not considered as the basis for the p-RfD.

Most of the quantitative chronic oral toxicity data for iron have been obtained from studies of the Bantu population of South Africa. These data indicate that intakes in the range of 0.7-1.4 mg iron/kg-day in home-brewed beer are associated with hemosiderosis and liver cirrhosis (Bothwell and Bradlow, 1960; Bothwell et al., 1964). However, confounding factors such as malnutrition and unusually high iron bioavailability due to the high acidity and ethanol in the beer preclude use of these data for risk assessment. Much higher dietary intakes (average 6.7 mg/kg-day) of less soluble forms of iron are tolerated in non-western diets as indicated by studies of populations in Ethiopia. Thus, although toxicity associated with iron overload due to chronic oral intake can be demonstrated qualitatively or even semiquantitatively, assignment of a precise LOAEL for normal individuals consuming western diets is compromised by studies containing confounding factors.

Gastrointestinal toxicity, which is commonly associated with the therapeutic use of iron supplements, was identified as the critical effect for the basis of the provisional subchronic and chronic RfDs. The most frequently reported symptoms include epigastric pain, nausea, vomiting, constipation and diarrhea. Several prospective clinical trials in healthy subjects and iron-deficient patients identify a LOAEL for gastrointestinal toxicity of 50 to 180 mg elemental iron/day; NOAELs were not established (Blot et al., 1981; Brock et al., 1985; Coplin et al., 1991; Frykman et al., 1994; Hallberg et al., 1966; Liguori, 1993). The treatment durations in these studies range from 2 weeks to approximately 3 months. Although no chronic exposure studies reporting gastrointestinal toxicity were identified, clinical experience with iron supplements indicates that gastrointestinal effects are associated with oral iron therapy, regardless of the duration of treatment and that symptom intensity does not change over the course of treatment (Hillman, 2001; Santi and Masters, 2001). This observation suggests that the response is related to the concentration of iron in the intestinal tract and not to the time-integrated dose. Therefore,

gastrointestinal toxicity is considered as the critical effect for both the subchronic and chronic p-RfDs.

The lowest LOAEL of 50 mg elemental iron/day for gastrointestinal toxicity associated with iron supplements was reported in two studies that did not use a placebo-controlled design (Brock et al., 1985; Coplin et al., 1991); therefore, data were not considered suitable for derivation of the p-RfD. The placebo-controlled, cross-over design study by Frykman et al. (1994) reporting a LOAEL of 60 mg/day in Swedish men and women was identified as the critical study. Results of this study show that daily treatment with ferrous fumarate (60 mg elemental iron/day) for one month produced a statistically significant increase in gastrointestinal effects compared to placebo. To determine the LOAEL for total daily iron intake, the LOAEL for daily supplementation with ferrous fumarate of 60 mg elemental iron/day was added to the estimated mean dietary intake for six European countries of 11 mg elemental iron/day (NAS, 2001) for a total daily iron intake of 71 mg elemental iron/day. Based on a reference body weight of 70 kg (U.S. EPA, 1987), the LOAEL for gastrointestinal effects for total daily iron intake is 1 mg elemental iron/kg-day. This LOAEL is considered to be a minimal LOAEL because gastrointestinal effects were characterized by most study participants as minor in severity.

The provisional subchronic and chronic RfD for iron was derived from the LOAEL of 1 mg/kg-day for total daily iron intake for adverse gastrointestinal effects as follows:

$$\begin{aligned} \textbf{p-RfD (subchronic and chronic)} &= \textbf{LOAEL} \div \textbf{UF} \\ &= \textbf{1 mg/kg-day} \div \textbf{1.5} \\ &= \textbf{0.7 mg/kg-day} \end{aligned}$$

Dividing the LOAEL of 1 mg/kg-day by an uncertainty factor of 1.5 yields a subchronic and chronic p-RfD of 0.7 mg/mg-day. The uncertainty factor of 1.5 includes the individual uncertainty factors of 1.5 for use of a minimal LOAEL, 1 for sensitive individuals, 1 for less than lifetime exposure, and 1 for an adequate data base. An uncertainty factor of 1.5 was applied to account for extrapolation from a minimal LOAEL to a NOAEL for a non-serious effect. A higher uncertainty factor for use of a minimal LOAEL was not used since the observed gastrointestinal effects are not considered serious and are reversible when exposure is discontinued. Furthermore, gastrointestinal symptoms are not associated with dietary intake of similar levels of iron (NAS, 2001). Because individuals sensitive to gastrointestinal symptoms are considered to be included in the studies investigating effects of therapeutic iron; an uncertainty factor of 1 for sensitive individuals results. An uncertainty factor of 1 was used to account for less than lifetime exposure. Although exposure duration in the Frykman et al. (1994) study was only one month, there is no evidence to suggest that symptoms increase with longer exposure periods. An uncertainty factor of 1 was used to reflect an adequate database in humans, due to the extensive use of therapeutic iron.

Except for individuals with disorders of iron metabolism, little information is available on the long-term systemic toxicity of orally ingested iron. This assessment, therefore, focuses more on what is known to be a safe oral intake of iron for the general human population (i.e., apparently healthy normal individuals). The provisional reference dose is estimated to be an

intake for the general population that is adequately protective from adverse health effects. Further, it is also important to note that individual requirements for, as well as adverse reactions to, iron may be highly variable. Some individuals may, in fact, consume a diet that contributes more than the provisional reference dose, without any cause for concern. In addition, specific population subgroups may have higher nutritional requirements than the provisional RfD would provide. The p-RfD may not be protective of individuals with inherited disorders of iron metabolism or other conditions which affect iron homeostasis.

This assessment is essentially the same as that proposed by Stifelman et al. (2005).

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6-16-05

Provisional Peer Reviewed Toxicity Values for

Iron and Compounds
(CASRN 7439-89-6)

Derivation of a Chronic Inhalation RfC

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level

MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	**provisional inhalation unit risk**
p-OSF	**provisional oral slope factor**
p-RfC	**provisional inhalation reference concentration**
p-RfD	**provisional oral reference dose**
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	**Provisional Peer Reviewed Toxicity Value**
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

6-16-05

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR IRON (CASRN 7439-89-6) AND COMPOUNDS
Derivation of an Inhalation RfC

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).

2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.

3. Other (peer-reviewed) toxicity values, including:

   - Minimal Risk Levels produced by the Agency for Toxic Substances and Disease Registry (ATSDR),
   - California Environmental Protection Agency (CalEPA) values, and
   - EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions (or the EPA HQ Superfund Program) sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

Disclaimers

Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

An RfC for iron is not listed on IRIS (U.S. EPA, 2001) and was not considered by the RfD/RfC Work Group (U.S. EPA, 1995). The HEAST (U.S. EPA, 1997) reported that data regarding iron were inadequate for quantitative risk assessment. The CARA list (1991, 1994a) includes a Health Effects Assessment for Iron and Compounds (U.S. EPA, 1984) that reported negative epidemiological studies (no association between excess mortality or respiratory diseases and occupational exposure to iron oxide dusts) and no available subchronic or chronic inhalation studies in animals. In March, 2004, a literature search was also conducted using TOXLINE, MEDLINE, Chemical Abstracts and Biological Abstracts data bases.

Occupational exposure limits have been established for soluble iron salts and iron oxide, as well as for organic iron compounds not covered in this issue paper. The ACGIH (1991a,

2001) has adopted a TLV-TWA, NIOSH (2001a) has established a REL-TWA, and OSHA (2001a, 2001b) has adopted a construction industry PEL-TWA of 1 mg/m^3, as Fe, to reduce the likelihood of irritation to eyes, skin, and respiratory tract from exposure to aerosols or mists of soluble iron salts (ferrous and ferric sulfates and chlorides, and ferric nitrate). The ACGIH (1991b, 2001) has adopted a TLV-TWA and NIOSH (2001b) has established a REL-TWA of 5 mg/m^3, as Fe, for dust and fume of ferric oxide (Fe_2O_3) to protect against siderosis, a benign pneumoconiosis. OSHA (2001c) has adopted a PEL-TWA of 10 mg/m^3 for ferric oxide fume, to protect against accumulation of iron dust in the lungs.

Iron has not been the subject of a toxicological profile by ATSDR (2001) or the WHO (2001). Monographs by IARC (1972, 1984, 1987), a toxicity review on iron (Grimsley, 2001), and the NTP (2001a, 2001b) management status report and chemical repository summary were consulted for information relevant to inhalation toxicity of iron and inorganic iron compounds. The following computer searches, performed in April, 1993, were screened to identify additional pertinent studies not discussed in review documents: TOXLINE (1983-April, 1993), CANCERLIT (1990 - April, 1993), MEDLINE (1991 - April, 1993), TSCATS, RTECS, and HSDB. Update literature searches were conducted in September, 2001 in TOXLINE (1992-September, 2001), CANCERLIT (1992- September, 2001), MEDLINE (1992-September, 2001), TSCATS, RTECS, DART/ETICBACK, EMIC/EMICBACK, HSDB, GENETOX, and CCRIS.

REVIEW OF PERTINENT LITERATURE

Human Studies

A number of studies have examined the relationship between respiratory disease and inhalation exposure to iron compounds for workers employed in hematite mining or other iron-related occupations, such as welding or steel-making (U.S. EPA, 1984; IARC, 1972, 1984; Grimsley, 2001). However, since these studies involved concurrent exposure to silica and other metals, they are not suitable for the health risk assessment of iron or iron compounds. The literature search did not discover any studies that examined subchronic or chronic inhalation exposures of humans to quantified levels of iron or iron compounds alone.

In a case-control study of cancer incidence, a Swedish male worker population (1958-1971) was reported to have had a high exposure to iron oxides from the production of sulfuric acid from pyrite (FeS_2) (Axelson and Sjöberg, 1979). The workers were exposed to iron oxide (Fe_2O_3) along with 1-2% copper, 0.01-0.1% arsenic, nickel and cobalt as impurities. Exposure in the workroom was estimated as approximately 50-100 mg/m^3, and the particle size as 25% below 10 μm and 5-10% below 5 μm. However, there were no measurements of exposure levels or particle size, and exposure durations were not reported. No cases of siderosis were known from the plant.

Animal Studies

Inhalation studies for iron compounds in animals include a chronic study of hamsters exposed to ferric oxide (Fe_2O_3) dust (Nettesheim et al., 1975) and a 2-month study in rabbits exposed to aerosols of ferric chloride (Johansson et al., 1992).

In a cancer study, groups of male Syrian hamsters (132 per group) were exposed to filtered air or Fe_2O_3 (analytic grade) dust at a concentration of 40 mg/m^3, 6 hours/day, 5 days/week for life (Nettesheim et al., 1975). The particle size had a geometric mean diameter of 0.11 μm. In addition, two satellite groups (15 hamsters per treatment) were sacrificed, three animals at a time, at 2, 4, 8, 12, and 104 weeks, so that the accumulation of iron in the lung from inhaled Fe_2O_3 could be compared to background iron concentrations in heme. The animals were examined daily, before and after each exposure, for clinical signs, and body weights were recorded monthly. All animals except those cannibalized (<2%) were necropsied. Histological analyses were performed on the major organs, including heart, trachea, lungs, and nasal cavities. Examination of the satellite groups demonstrated the gradual increase in iron accumulation in the lung, reaching a total of 10 mg per lung at 104 weeks. Histological examination revealed iron deposits in the lungs and tracheal and bronchial lymph nodes of all exposed animals. Diffuse and focal alveolar fibrosis was also frequently observed in the lungs of treated animals. Results for the histological endpoints were not reported quantitatively. In this study, 40 mg/m^3 is a LOAEL for respiratory effects (alveolar fibrosis) in hamsters exposed to Fe_2O_3 dust.

Groups of 8 male rabbits (strain not reported) were exposed to aerosols of 0, 1.4, or 3.1 mg/m^3 of iron as $FeCl_3$ 6 hours/day, 5 days/week for 2 months (Johansson et al., 1992). At termination, the upper left lung lobe was examined by light microscopy, pieces of the lower left lung were analyzed by electron microscopy or used for phospholipid analysis, and the right lung was lavaged to obtain macrophages for morphological and functional analyses. The mass median aerodynamic diameter of the aerosols was ~1 μm as measured with an impactor. Treatment had no effect on survival. Lungs were spotted with black in 7/8 high-iron rabbits, in 2/8 low-iron rabbits, and in 0/8 controls. The absolute weight of the left lower lobe of the lung was significantly elevated compared to controls in the high-iron group. Exposure-related histopathology was observed in the lungs. In the high-exposure group, the lungs contained naked granulomas [large nodules (≥ 1 mm) of densely packed granular macrophages], accumulations of granular macrophages in terminal bronchioles, and foci of interstitial lymphocytic inflammatory reaction. Small granulomas were observed in one low-iron and one control rabbit. Accumulations of normal and granular macrophages were observed in the alveoli of exposed rabbits. In the control group, normal lung tissue contained some small accumulations of macrophages with occasional small inflammatory reaction. The high exposure group had a significantly higher density of alveolar type II cells than the controls. Ultrastructural analysis of macrophages showed a significantly higher number of abnormal cells, cells with enlarged lysosomes, and black inclusions in cells in both exposed groups; the high-iron group had higher

percentages of cells with laminar inclusions or with smooth cell surfaces. In functional tests, macrophages from the high-exposure group showed significantly elevated phagocytic activity, but no significant increase in oxidative metabolic activity (superoxide generation). Total phospholipids were elevated in the high-exposure group, but, as indicated by the lack of increase in phosphatidyl cholines or the percentage of 1,2-dipalmitoylphosphatidylcholine, the amount of surfactant was unchanged. In this study, the low concentration of 1.4 mg/m^3 is a NOAEL and the high concentration of 3.1 mg/m^3 is a LOAEL for adverse lung effects (nodular granulomas ≥ 1 mm in diameter, abnormal macrophages) in rabbits exposed to ferric chloride aerosols. Because of its focus on alveolar macrophage effects, this study provided no information regarding clinical signs of toxicity, body weight changes, clinical biochemistry, nasopharyngeal effects or histology of any other tissue besides the lung.

Other Studies

In a cancer study, groups of Syrian golden hamsters (24 per sex per group) received intratracheal instillations of 0 or "a maximum dose"[1] of 3 mg of Fe_2O_3 dust in 0.2 ml of saline once a week for 15 weeks, and then were observed up to week 120 (Stenbäck et al., 1976). Analysis by the sedimentation method demonstrated that 98% of the particles were less than 10 µm in diameter. Animals were weighed weekly and autopsied. Organs with gross lesions and the larynx, trachea, bronchi, and lungs were examined histologically. Treatment with ferric oxide had no effect on survival and no effect on body weight except during the final weeks of survival (data not shown). Deposited iron oxide was grossly visible as dark patches on the lung surface. Histologically, dust accumulations surrounded by cellular infiltrates were observed in the peribronchial region. Interstitial fibrosis was observed occasionally, but distinct inflammatory changes were rare. Results for the nonneoplastic endpoints were not reported quantitatively.

FEASIBILITY OF DERIVING A PROVISIONAL RfC FOR IRON

No adequate human or animal inhalation data are available for exposure to iron or inorganic iron compounds. The epidemiological study of Axelson and Sjöberg (1979) did not provide quantitative measures of exposure and did not characterize noncancer endpoints. Although Nettesheim et al. (1975) reported diffuse and focal alveolar fibrosis in the lungs of hamsters chronically exposed to iron oxide by inhalation at a concentration of 40 mg/m^3, the lack of incidence data prevents an evaluation of the significance of these findings. The subchronic study of Johansson et al. (1992), in which rabbits were exposed to aerosols of ferric chloride for

[1]The authors provided no further information regarding dosage. It is not clear whether animals were given amounts lower than 3 mg on some occasions.

2 months, demonstrated a NOAEL of 1.4 mg/m^3 and a LOAEL of 3.1 mg/m^3 for respiratory effects (granuloma nodules greater than 1 mm diameter in the lungs). However, this study does not meet the minimum standards for an inhalation bioassay as stipulated by the U.S. EPA (1994b) guidelines for derivation of an inhalation reference concentration. Inadequacies of the study include relatively small group sizes, relatively short study duration, and the failure to examine a sufficient array of endpoints. Thus this study is inadequate for the purposes of deriving a p-RfC for iron. Consequently, the available data are insufficient for derivation of a p-RfC.

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7-29-05

Provisional Peer Reviewed Toxicity Values for

Iron and Compounds

(CASRN 7439-89-6)

Derivation of a Carcinogenicity Assessment

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
i.v.	intravenous
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level

MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	**provisional inhalation unit risk**
p-OSF	**provisional oral slope factor**
p-RfC	**provisional inhalation reference concentration**
p-RfD	**provisional oral reference dose**
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	**Provisional Peer Reviewed Toxicity Value**
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

7-29-05

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR IRON (CASRN 7439-89-6) AND COMPOUNDS
Derivation of a Carcinogenicity Assessment

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

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   - California Environmental Protection Agency (CalEPA) values, and
   - EPA Health Effects Assessment Summary Table (HEAST) values.

A PPRTV is defined as a toxicity value derived for use in the Superfund Program when such a value is not available in EPA's Integrated Risk Information System (IRIS). PPRTVs are developed according to a Standard Operating Procedure (SOP) and are derived after a review of the relevant scientific literature using the same methods, sources of data, and Agency guidance for value derivation generally used by the EPA IRIS Program. All provisional toxicity values receive internal review by two EPA scientists and external peer review by three independently selected scientific experts. PPRTVs differ from IRIS values in that PPRTVs do not receive the multi-program consensus review provided for IRIS values. This is because IRIS values are generally intended to be used in all EPA programs, while PPRTVs are developed specifically for the Superfund Program.

Because science and available information evolve, PPRTVs are initially derived with a three-year life-cycle. However, EPA Regions (or the EPA HQ Superfund Program) sometimes request that a frequently used PPRTV be reassessed. Once an IRIS value for a specific chemical becomes available for Agency review, the analogous PPRTV for that same chemical is retired. It should also be noted that some PPRTV manuscripts conclude that a PPRTV cannot be derived based on inadequate data.

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Users of this document should first check to see if any IRIS values exist for the chemical of concern before proceeding to use a PPRTV. If no IRIS value is available, staff in the regional Superfund and RCRA program offices are advised to carefully review the information provided in this document to ensure that the PPRTVs used are appropriate for the types of exposures and circumstances at the Superfund site or RCRA facility in question. PPRTVs are periodically updated; therefore, users should ensure that the values contained in the PPRTV are current at the time of use.

It is important to remember that a provisional value alone tells very little about the adverse effects of a chemical or the quality of evidence on which the value is based. Therefore, users are strongly encouraged to read the entire PPRTV manuscript and understand the strengths and limitations of the derived provisional values. PPRTVs are developed by the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center for OSRTI. Other EPA programs or external parties who may choose of their own initiative to use these PPRTVs are advised that Superfund resources will not generally be used to respond to challenges of PPRTVs used in a context outside of the Superfund Program.

Questions Regarding PPRTVs

Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

A cancer assessment for iron is not listed on IRIS (U.S. EPA, 2005a), the HEAST (U.S. EPA, 1997), or the Drinking Water Standards and Health Advisories list (U.S. EPA, 2000), and was not considered by the CRAVE Work Group (U.S. EPA, 1995). The CARA list (1991, 1994) includes a Health Effects Assessment for Iron and Compounds (U.S. EPA, 1984) that assigned iron and its compounds to weight-of-evidence Group C, possible human carcinogen. This assessment was based on conflicting evidence of lung tumors following occupational inhalation exposure to ferric oxide (mixed exposure), and injection-site tumors in one patient and in mice treated with iron-dextran. IARC (1972, 1987) assigned ferric oxide to Group 3, not classifiable as to its carcinogenicity to humans based on inadequate data in humans (increased incidence of lung cancer following occupational exposure to iron dusts in mixtures) and apparently negative

evidence for carcinogenicity in mice, hamsters and guinea pigs exposed by inhalation or intratracheal instillation. For ferric oxide dust and fume, the ACGIH (1991, 2001) lists an A4 notation, not classifiable as a human carcinogen; this is based on mixed exposure studies in humans and primarily negative studies in animals. In March, 2004, a literature search was also conducted using TOXLINE, MEDLINE, Chemical Abstracts and Biological Abstracts data bases.

Iron has not been the subject of a toxicological review by ATSDR (2001) or the WHO (2001). Monographs by IARC (1972, 1984, 1987), a toxicity review on iron (Grimsley, 2001), and the NTP (2001a, 2001b) management status report and chemical repository summary were consulted for information relevant to the carcinogenicity of iron and inorganic iron compounds. The following computer searches, performed in April, 1993, were screened to identify additional pertinent studies not discussed in review documents: TOXLINE (1983-April, 1993), CANCERLIT (1990 - April, 1993), MEDLINE (1991 - April, 1993), TSCATS, RTECS, and HSDB. Update literature searches were conducted in September, 2001 in TOXLINE (1992-September, 2001), CANCERLIT (1992- September, 2001), MEDLINE (1992-September, 2001), TSCATS, RTECS, DART/ETICBACK, EMIC/EMICBACK, HSDB, GENETOX, and CCRIS.

REVIEW OF PERTINENT LITERATURE

Human Studies

Oral Exposure

Because iron is an essential element, the NAS (2001) has established guidelines for daily dietary intakes, based on gender, age, and physiological status, that are designed to avoid adverse effects of deficiency and excess. Individuals of northern European descent who are affected by hereditary hemochromatosis, an autosomal, recessive disorder, are not protected by these guidelines. These individuals exhibit excessive absorption of dietary iron, which results in abnormally high accumulations of iron in liver and brain tissues. When the liver consequently develops cirrhosis, the risk of developing primary hepatocellular carcinoma increases significantly. It is not clear whether these findings are relevant to excess iron intake by the general population.

Bird et al. (1996) investigated the association between plasma ferritin and iron intake and the development of adenomatous polyps, which are intermediate markers for colorectal cancer. The study population consisted of men and women between the ages of 50 and 75 years old who underwent routine screening by flexible sigmoidoscopy at one of two medical centers during 1991-1993. Individuals with cancer, inflammatory bowel disease, or familial polyposis were excluded. Cases (300 men and 167 women) were subjects diagnosed for the first time with one

or more histologically confirmed adenomatous polyps. Controls (331 men and 167 women) had no history of polyps and none discovered at sigmoidoscopy. Cases and controls were matched by sex, age (± 5 years), date of sigmoidoscopy (± 3 months), and medical center. Plasma ferritin levels, hematocrit, and certain nutritional indicators (carotenoids, ascobate, folate) were measured in blood samples drawn 6 months after examination. Iron intakes for the year preceding sigmoidoscopy were estimated by means of a semiquantitative food frequency questionnaire. After controlling for possible confounding factors, subjects with high plasma ferritin levels (>289 μg/L) had a multivariate-adjusted odds ratio for colorectal polyps of 1.5 (95% confidence interval (C.I. = 1.0-2.3) compared to subjects with low/normal levels (73-141 μg/L). The pattern for iron intake was U-shaped. Compared with subjects consuming an adequate amount of iron (11.6-13.6 mg/day), multivariate-adjusted odds ratios for colorectal polyps in men were 1.6 (95% C.I. = 1.1-2.4) for intakes below 11.6 mg/day and 1.4 (95% C.I.= 0.9-2.0) for intakes above 27.3 mg/day. The highest odds ratio of 2.1 (95% C.I. = 1.3-3.5) was found after further adjustment for smoking for men at the lowest level of iron intake. The association between iron intake and colorectal polyps disappeared when exposure group class of reaction was based on dietary intake alone (i.e., high iron supplementation ignored). The authors concluded that there was a weak positive association between iron exposure and colorectal polyps that may increase the risk of colorectal cancer but note that some factor in supplementation may have been responsible for the effect.

Inhalation Exposure

Most studies of cancer incidence following occupational exposure to iron dust are excluded from consideration because of confounding exposures to silica, radon daughters, soot, asbestos, or other types of metals in the study populations (U.S. EPA, 1984; IARC, 1972, 1984, 1987).

A case-control study examined cancer incidence in a Swedish male worker population (1958-1971) with a high exposure to iron oxides from the production of sulfuric acid from pyrite (FeS_2) (Axelson and Sjöberg, 1979). The workers were exposed to iron oxide (Fe_2O_3) along with 1-2% copper, 0.01-0.1% arsenic, nickel and cobalt as impurities. Exposure in the workroom was estimated as approximately 50-100 mg/m^3, and the particle size as 25% below 10 μm and 5-10% below 5 μm. No cases of siderosis were known from the plant. The Swedish National Cancer Register was consulted for locating cases of cancer that could have been caused by environmental exposure; the study examined cancers of the stomach, liver, lung, kidney, and bladder, and hematological malignancies. Each cancer case was matched with two controls from the local population register by matching for sex, age, and residency in the same or adjacent neighborhood block. Company files were searched to determine the length of exposure; those with less than 5 months of exposure were considered to be nonexposed. The study found no association between exposure to iron oxides and any of the selected types of cancer.

Animal Studies

Oral Exposure

Groups of F344 rats (50 per sex per group) were given ferric chloride ($FeCl_3 \cdot 6H_2O$) in drinking water at concentrations of 0, 0.25, or 0.5% (weight/volume) for 104 weeks, and then given distilled water for an 8 week recovery period (Sato et al., 1992). The intake of ferric chloride was reported to be 0, 169.7, or 319.7 mg/kg-day for males and 0, 187.9, or 336.0 mg/kg-day for females. The iron intakes were 0, 58.4, or 110 mg/kg-day in males and 0, 64.6, or 115.6 mg/kg-day in females. Rats were observed daily for clinical signs and mortality. Body weights were measured once a week for 13 weeks and every fourth week thereafter. All rats dying prematurely and survivors at week 112 were examined for gross and microscopic neoplastic and non-neoplastic lesions. There were dose-related decreases in drinking water intake and terminal body weight in both sexes. These may have been related to reduced palatability. Survival in both sexes was not significantly affected by exposure to ferric chloride. No increases in tumor incidence were observed in rats exposed to ferric chloride for two years.

Inhalation Exposure

Groups of male Syrian hamsters (132 per group) were exposed to filtered air or Fe_2O_3 (analytic grade) dust at a concentration of 40 mg/m^3, 6 hours/day, 5 days/week for life (Nettesheim et al., 1975). The particle size had a geometric mean diameter of 0.11 μm. In addition, two satellite groups (15 hamsters per treatment) were sacrificed, three animals at a time, at 2, 4, 8, 12, and 104 weeks, so that the accumulation of iron in the lung from inhaled Fe_2O_3 could be compared to background iron concentrations in heme. The animals were examined daily, before and after each exposure, for clinical signs; body weights were recorded monthly. All animals except those cannibalized (<2%) were necropsied. Histological analyses were performed for the major organs, including heart, trachea, lungs, and nasal cavities. Examination of the satellite groups demonstrated a gradual increase in iron accumulation in the lung, reaching a total of 10 mg per lung at 104 weeks. Exposure to Fe_2O_3 had no effect on survival or body weight gain and did not increase the incidence of tumors. The authors concluded that inhalation of Fe_2O_3 was not carcinogenic to hamsters.

Groups of Syrian golden hamsters (24 per sex per group) received intratracheal instillations of 0 or 3 mg[1] of Fe_2O_3 dust in 0.2 ml of saline once a week for 15 weeks, and then were observed up to week 120 (Stenbäck et al., 1976). Analysis by the sedimentation method demonstrated that 98% of the particles were less than 10 μm in diameter. Animals were weighed

[1]The authors characterized the treatment as a 'maximum dose of 3 mg'. It is not clear whether the hamsters received lower doses on some occasions.

weekly and autopsied. Organs with gross lesions and the larynx, trachea, bronchi, and lungs were examined histologically. Treatment with ferric oxide had no effect on survival and did not affect body weight except during the final weeks of survival (data not shown). Treatment did not induce tumors of the respiratory tract and the incidence of forestomach papillomas in the treatment group was less than in the control group.

Other Studies

Genotoxicity

Genotoxicity assays of inorganic iron salts were primarily negative in bacteria, but were more often positive in mammalian systems. Iron did not induce reverse mutations in *Salmonella typhimurium* strains TA98, TA102, TA1535, or TA1537, with or without activation (Wong, 1988). Ferric chloride and ferrous sulfate tested negative in strains TA98, TA100, TA1535, TA1537, and TA1538 with or without metabolic activation (Shimizu et al., 1985; Dunkel et al., 1999). Ferrous sulfate also tested negative in strains TA97 and TA102, with or without activation (Fujita et al., 1994), but positive in TA1537 and TA1538 (U.S. EPA, 1984). Ferrous and ferric chloride did not induce DNA repair in *Bacillus subtilis* (rec assay) (Leifer et al., 1981). Ferrous sulfate increased the frequency of mutations at the TK locus of mouse L5178Y lymphoma cells, with or without metabolic activation, but only at high concentrations that were likely to be cytotoxic; ferric chloride only increased the frequency of TK mutations when tested with metabolic activation (Dunkel et al., 1999). Ferrous sulfate did not induce sister chromatid exchanges *in vitro* (Ohno et al., 1982). DNA-protein cross-links were generated in mammalian cells cultured in the presence of ferrous iron (Altman et al., 1995). Single- and double-strand DNA breaks were produced in supercoiled plasmid DNA (Toyokuni and Sagripanti, 1992) and in isolated rat liver nuclei (U.S. EPA, 1984) treated with ferrous or ferric chloride. No breakage was detected electrophoretically in Chinese hamster ovary cell DNA treated with ferrous chloride (U.S. EPA, 1984). In a model of oxidative damage within cells, ferrous sulfate, in the presence of hydrogen peroxide, was demonstrated to induce double-strand breaks and intra-strand cross-links in DNA *in vitro* (Lloyd and Phillips, 1999).

Cell transformation

Iron compounds have yielded variable results in studies of cell transformation *in vitro*. Particles of magnetite (Fe_3O_4) induced transformation of cultured a Chinese hamster lung cell line (V_{79}), but only at cytotoxic concentrations (Elias et al., 1995). Ferrous chloride and ferrous sulfate induced cell transformation in viral-enhanced Syrian hamster embryo (SA7/SHE) cells (U.S. EPA, 1984).

Mechanistic Studies

Adverse effects of iron are thought to be related to the formation of reactive oxygen species via the Fenton reaction (Henle and Linn, 1997). Hydrogen peroxide can react with ferrous ion, resulting in the conversion to ferric ion and the production of hydroxyl radicals. Ferric ion can also react with hydrogen peroxide, producing superoxide radical. Reactive oxygen species may react with DNA. However, because of the complex homeostatic mechanisms involved in iron transport and metabolism, unbound ferrous iron is not likely to be present except in conditions of excessive iron intake.

PROVISIONAL WEIGHT-OF-EVIDENCE CLASSIFICATION

U.S. EPA (1984) classified iron and its compounds, including ferric dextran, as possible human carcinogens (Group C). This assessment was based on reports associating an increased incidence of lung cancer with exposure to hematite dust (confounded by coincident exposures to tobacco, alcohol, silica, soot, and fumes of other metals), inconsistent reports of lung tumors in animals exposed by inhalation or tracheal instillation to ferric oxide, and reports of injection site tumors in one patient injected with iron dextran and in mice injected with iron dextran or saccharated iron oxide. The current PPRTV assessment excludes organic forms of iron and studies in which the levels of impurities are significant.

Results of the case-control study by Bird et al. (1996) provide evidence of a weak association between elevated iron intake or high plasma ferritin (a measure of body stores) and the prevalence of adenomatous colorectal polyps, a possible precursor to colorectal cancer. Weaknesses of this study include the 6-month period between examination and ferritin measurements, and the possible recall errors affecting the dietary questionnaire for the previous year. In addition, the association between iron intake and colorectal polyps was stronger at low iron intake and not related to dietary (i.e., environmental) intake. Although the association between cirrhotic hereditary hemochromatosis and hepatocellular carcinoma is well established, the evidence for dietary iron intake and hepatic cancer in the general population was characterized by the NAS (2001) as inconclusive. In a chronic rat assay, Sato et al. (1992) found no evidence of carcinogenicity of ferric chloride ingested in drinking water at concentrations up to 0.5%. In summary, the evidence for carcinogenicity of ingested inorganic iron compounds in humans and animals is inadequate.

Evidence from the case-control study of Axelson and Sjöberg (1979) suggests that inhaled iron oxide may not be carcinogenic to humans. However, uncertainty remains because levels of exposure were not measured, the durations of exposure were not reported, and individuals exposed for up to 5 months were categorized as 'nonexposed.' In addition, the lack of reported cases of siderosis in the workplace suggests that the exposure levels may have been

lower than estimated. Thus, the evidence for carcinogenicity of inhaled iron oxide in humans is considered inadequate. Results of the study of Nettesheim et al. (1975) indicate that chronic inhalation exposure to iron oxide at a concentration of 40 mg/m^3 is not carcinogenic to hamsters. This finding is supported by the negative results for carcinogenicity of iron oxide administered by intratracheal instillation to hamsters for 15 weeks (Stenbäck et al., 1976). However, as both hamster studies used single exposure concentrations, the possibility of carcinogenicity at higher exposure levels cannot be disregarded.

Following the U.S. EPA (2005b) guidelines for carcinogen risk assessment, the available data are inadequate for an assessment of the human carcinogenic potential of inhaled iron oxide or ingested iron chloride.

QUANTITATIVE ESTIMATES OF CARCINOGENIC RISK

Derivation of quantitative estimates of cancer risk for ingested or inhaled iron or iron oxide is precluded by the absence of adequate data demonstrating carcinogenicity.

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6-12-2008

Provisional Peer Reviewed Toxicity Values for

Lithium
(CASRN 7439-93-2)

Superfund Health Risk Technical Support Center
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Cincinnati, OH 45268

Acronyms and Abbreviations

bw	body weight
cc	cubic centimeters
CD	Caesarean Delivered
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CNS	central nervous system
cu.m	cubic meter
DWEL	Drinking Water Equivalent Level
FEL	frank-effect level
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
g	grams
GI	gastrointestinal
HEC	human equivalent concentration
Hgb	hemoglobin
i.m.	intramuscular
i.p.	intraperitoneal
IRIS	Integrated Risk Information System
IUR	inhalation unit risk
i.v.	intravenous
kg	kilogram
L	liter
LEL	lowest-effect level
LOAEL	lowest-observed-adverse-effect level
LOAEL(ADJ)	LOAEL adjusted to continuous exposure duration
LOAEL(HEC)	LOAEL adjusted for dosimetric differences across species to a human
m	meter
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	modifying factor
mg	milligram
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	minimal risk level
MTD	maximum tolerated dose
MTL	median threshold limit
NAAQS	National Ambient Air Quality Standards
NOAEL	no-observed-adverse-effect level
NOAEL(ADJ)	NOAEL adjusted to continuous exposure duration
NOAEL(HEC)	NOAEL adjusted for dosimetric differences across species to a human
NOEL	no-observed-effect level
OSF	oral slope factor
p-IUR	provisional inhalation unit risk
p-OSF	provisional oral slope factor
p-RfC	provisional inhalation reference concentration

p-RfD	provisional oral reference dose
PBPK	physiologically based pharmacokinetic
ppb	parts per billion
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
RBC	red blood cell(s)
RCRA	Resource Conservation and Recovery Act
RDDR	Regional deposited dose ratio (for the indicated lung region)
REL	relative exposure level
RfC	inhalation reference concentration
RfD	oral reference dose
RGDR	Regional gas dose ratio (for the indicated lung region)
s.c.	subcutaneous
SCE	sister chromatid exchange
SDWA	Safe Drinking Water Act
sq.cm.	square centimeters
TSCA	Toxic Substances Control Act
UF	uncertainty factor
µg	microgram
µmol	micromoles
VOC	volatile organic compound

PROVISIONAL PEER REVIEWED TOXICITY VALUES FOR LITHIUM (CASRN 7439-93-2)

Background

On December 5, 2003, the U.S. Environmental Protection Agency's (EPA's) Office of Superfund Remediation and Technology Innovation (OSRTI) revised its hierarchy of human health toxicity values for Superfund risk assessments, establishing the following three tiers as the new hierarchy:

1. EPA's Integrated Risk Information System (IRIS).

2. Provisional Peer-Reviewed Toxicity Values (PPRTV) used in EPA's Superfund Program.

3. Other (peer-reviewed) toxicity values, including:

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Questions regarding the contents of the PPRTVs and their appropriate use (e.g., on chemicals not covered, or whether chemicals have pending IRIS toxicity values) may be directed to the EPA Office of Research and Development's National Center for Environmental Assessment, Superfund Health Risk Technical Support Center (513-569-7300), or OSRTI.

INTRODUCTION

Lithium (Li), an alkali metal, exists in two isotopic forms (^{7}Li and ^{6}Li) and is naturally present in soil and water. Lithium has numerous industrial and commercial uses including as a cell additive in electrolytic aluminum production, a catalyst of chemical reactors, a component of fluxes and brazing alloys, a component of batteries, specialized glass and ceramics, and a sanitizing agent for swimming pools, hot tubs and spas (Leonard et al., 1995; Moore, 1995). Lithium carbonate and lithium citrate are also used for the therapeutic treatment of psychiatric disorders, primarily in the acute and long-term maintenance treatment of bipolar mood disorders.

A reference dose (RfD) or reference concentration (RfC) for lithium are not available on the Integrated Risk Information System (IRIS) (U.S. EPA, 2007), the Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1997), or the Drinking Water Standards and Health Advisories list (U.S. EPA, 2004). The U.S. Environmental Protection Agency's (EPA) Chemical Assessments and Related Activities (CARA) (U.S. EPA, 1994) lists only Reportable Quantity (RQ) documents for lithium chromate and lithium hydride; the RQ documents (U.S. EPA, 1983, 1988) for these two compounds state that the data are not sufficient for derivation of an RQ as there are no subchronic or chronic studies. Neither the Agency for Toxic Substances Disease and Registry (ATSDR) (2006), the National Toxicology Program (NTP) (2006), the International Agency for Research on Cancer (IARC) (2006), nor the World Health Organization (WHO) (2006) has produced documents regarding lithium. The following sources were also consulted: Chemical Hazard Information Profiles (U.S. EPA, 1980), National Occupational Health Survey of Mining (NIOSH, 1990) and Information Profiles on Potential Occupational Hazards - Classes (NIOSH, 1978). Literature searches were conducted from 1965 to August 2006 in TOXLINE

(including NTIS and BIOSIS updates), CANCERLIT, MEDLINE, CCRIS, GENETOX, HSDB, EMIC/EMICBACK, DART/ETICBACK, RTECS and TSCATS.

REVIEW OF PERTINENT LITERATURE

Human Studies

Oral Exposure

Overview of the Therapeutic Use of Lithium – Lithium carbonate, and more recently, lithium citrate have been used since 1949 in the treatment of bipolar affective (manic-depressive) disorder; thus, extensive clinical literature on the beneficial and adverse effects of lithium is available. Lithium is therapeutically used in the treatment of bipolar affective disorder as a sole therapy or in combination with other antidepressant drugs and in the treatment of schizophrenia in combination with anti-psychotic drugs. Although lithium is effective in the treatment of bipolar affective disorders, adverse effects are associated with therapeutic dose levels, resulting in a low therapeutic index (e.g., ratio of dose associated with therapeutic efficacy to dose associated with adverse effects). Thus, lithium is not simply prescribed by dose, but is monitored based on serum concentrations. For the treatment of bipolar disorder, the desired therapeutic serum concentrations range from 0.6 to 1.4 mmol Li/L, although concentrations of 0.8-1.0 mmol Li/L are generally accepted as providing optimal therapeutic effects (Physicians Desk Reference, 2006; Baldessarini and Tarazi, 2001).

Although the precise mechanism of action has not been established, it is unlikely that a single mechanism of action is responsible for the therapeutic and adverse effects of lithium. Several mechanisms for the therapeutic effects of lithium have been proposed. Since the chemical properties of lithium are similar to those of sodium, lithium can be substituted for sodium in generating action potentials and in some sodium transport processes across membranes. Lithium also appears to alter neurotransmitters, enhances some actions of serotonin, has variable effects on norepinephrine, augments the synthesis of acetylcholine and increases norepinephrine and dopamine turnover. Lithium also alters brain inositol phosphate levels, affecting second messenger responses for α-adrenergic and muscarinic transmission. A decrease in functioning brain protein kinases has also been identified as a consistent effect of lithium. Lithium also interacts with nuclear regulatory factors that affect gene expression (Baldessarini and Tarazi, 2001).

The potential for lithium to cause toxicity has been of significant concern due to its use on a maintenance basis for a lifelong disorder; thus, a large body of clinical literature on lithium-induced toxicity exists, including several reviews (Gitlin, 1999; Berk and Berk, 2003; Markowitz et al., 2000; Moore, 1995; McIntyre et al., 2001; Awad et al., 2002; Presne et al., 2003; Jefferson, 1998). Effects that are associated with therapeutic use of lithium include: neurological and psychiatric effects (tremor, choreoathetosis, motor hyperactivity, ataxia, aphasia, fatigue, cognitive impairment); decreased thyroid function; hyperparathyroidism; renal effects (nephrogenic diabetes insipidus, nephritis, chronic progressive renal disease); edema (related to sodium retention); cardiovascular effects (T wave flattening); acniform skin eruptions;

benign leukocytosis; and gastrointestinal effects (nausea, vomiting, abdominal pain, diarrhea). Since all therapeutic serum concentrations are associated with adverse effects, long-term treatment strategies for individual patients must balance the beneficial effects of lithium therapy with the risks and severity of toxicity. Although available data are not sufficient to define dose-response relationships, it is generally accepted that severity of adverse effects is related to serum lithium levels.

Adverse renal effects associated with lithium therapy have received extensive focus due to their serious nature and frequency of occurrence. Thus, lithium-induced renal toxicity has been the subject of numerous clinical and animal studies. The most common adverse renal effect is nephrogenic diabetes insipidus (NDI), which occurs over the range of therapeutic serum lithium concentrations (e.g., 0.6-1.4 mmol Li/L). The development of NDI involves lithium-induced down-regulation of the vasopressin-regulated water channel aquaporin-2, expressed on the apical plasma membrane of principal cells of the collecting duct (Markowitz et al., 2000). The consequence of this effect is to reduce the capacity of the kidneys to preserve free water, resulting in impaired renal concentrating ability and the production of excessively dilute urine. Clinically, this manifests as polyuria, with secondary thirst, and volume depletion. Although other mechanisms may also contribute to polyuria, interference with vasopressin-induced antidiuresis is considered the most important cause (Gitlin, 1999). It has been estimated that renal concentrating ability is impaired in at least 50% of patients undergoing lithium treatment, with polyuria and polydipsia in approximately 20% of patients (Presne et al., 2003; Gitlin, 1999; McIntyre et al., 2001). In a review of data from several studies published between 1979 and 1986, impairment of concentrating ability was seen in at least 54% of 1105 patients on chronic lithium therapy, with polyuria observed at serum lithium concentrations ranging from 0.6 to 1.2 mmol/L (Boton et al., 1987). Thus, periodic measurement of serum creatinine, creatinine clearance, 24-hour urine volume and urine protein has become integral to the management patients on long-term lithium therapy (Jefferson, 1998).

NDI appears to be reversible early in treatment, but may be progressive during the first decade, leading to irreversible damage over time (Gitlin, 1999). A small percentage of patients show progressive renal failure indicated by a pronounced decrease in glomerular filtration rate (GFR) and renal insufficiency, with little or no proteinuria (Markowitz et al., 2000). Severe decreases in GFR have resulted in the need for maintenance hemodiaylsis, typically after 10 or more years of lithium therapy. Results of renal biopsy on patients with chronic renal effects showed interstitial fibrosis, tubular atrophy, focal sclerosis, acquired renal cystic disease and cytoplasmic swelling with glycogen deposits in the distal convoluted tubules and collecting ducts (Markowitz et al., 2000; Gitlin, 1999). Confounding factors (other medical disorders such as hypertension, heart disease) may contribute to susceptibility and severity of irreversible damage (Gitlin, 1999).

Studies on Adverse Effects in Patients Treated with Lithium: Renal Effects – The results of retrospective and prospective studies and findings of case reports summarized below focus on adverse effects observed in patients maintained on chronic lithium therapy for the treatment of affective mood disorders. As expected based on the established pharmacological profile of therapeutic lithium, decreased renal concentrating ability is the most frequently reported adverse effect, although neurological, dermal, cardiovascular and endocrine effects are

also observed. Interpretation of results from clinical studies is difficult due to many factors, including the lack of baseline data prior to lithium use, absence of control groups, presence of pre-existing renal and other diseases and use of concomitant medications. Furthermore, since clinicians rely upon serum lithium concentrations, rather than daily doses, to evaluate the dose-response relationships between lithium treatment, efficacy and adverse effects, daily lithium doses often are not reported nor were the results of male vs. female dosing reported separately. However, the clinical literature provides consistent evidence that the kidney is a primary target organ for lithium in men and women, and supports that adverse renal effects occur over the range of desired therapeutic serum concentrations (0.6-1.4 mmol Li/L).

In a prospective study, a cohort of 373 patients who started receiving lithium therapy at various times between 1979 and 1987 were given pre-treatment examinations to establish baseline levels for renal parameters (Schou and Vestergaard, 1988; Vestergaard and Schou, 1988). Patients were examined once before and on the average 3.3 times during lithium therapy. Patients who had been treated with lithium prior to entry into the cohort and patients taking neuroleptic agents during lithium therapy were excluded from analysis. On examination days, urine was collected every 24 hours and data were disregarded if less than 75% of the daily lithium dose was recovered. The desamino-8-D-arginine vasopressin (DDAVP) test was used to determine renal concentrating ability. The mean lithium dose was 23.2 mmol Li/day and the mean lithium serum concentration was 0.68 mmol/L. Because of a high drop-out rate (for various reasons), especially among men, and because the dosing durations of the full cohort ranged from 5 months to 7 years, data from a subcohort of 39 patients who received lithium therapy continuously for 4 years were compared with the data for the whole cohort to guard against errors due to selective sample attrition. The ratio of men to women in the whole cohort and in the subcohort remained constant.

Patients in the whole cohort developed a moderate rise in urine volume and a moderate fall in renal concentrating ability (Schou and Vestergaard, 1988; Vestergaard and Schou, 1988). Urine volume increased by 7% (not statistically significant) for the whole cohort and 23% for the subcohort ($p=0.05$). For the whole cohort, urine volume was positively correlated with lithium dosage ($r=0.29$, $p<0.001$). Renal concentrating ability fell by 7% ($p<0.01$) for the whole cohort and by 10% for the 4-year subcohort ($p<0.01$). Changes in renal concentrating ability took place within the first 1-2 years of lithium therapy for members of the whole cohort, with no additional changes in renal function when treatment duration was extended more than 2 years. There was no correlation between concentrating ability and lithium dosage in the whole cohort. Glomerular function, as determined by measurement of serum creatinine concentrations and urine creatinine concentrations, was not affected by lithium therapy in the whole cohort. In addition, there was no change in the incidence of proteinuria associated with lithium treatment. Complaints of increased thirst, frequent urination and nocturia were made more often during lithium therapy than before lithium therapy in both the cohort and the subcohort. Therefore, the results from the subcohort support the results from the entire cohort. Assuming that the average body weight was 70 kg, patients were exposed to 2.32 mg Li/kg-day. This study identifies a lowest-observed-adverse-effect level (LOAEL) of 2.32 mg Li/kg-day for increased urine volume and decreased urine concentrating ability.

A group of 53 patients (20 men and 33 women) was examined prior to starting long-term lithium therapy and again after 4 and 12 months on lithium (Smigan et al., 1984). Twenty-five patients of this cohort had previously received lithium, but treatment had been withdrawn for 27 months before the start of the present treatment. Over the course of the study, 13-28% of the 53 patients received neuroleptics. Lithium carbonate dosages were not provided, but serum lithium levels were maintained at approximately 0.6 mmol/L. A clinically significant change in renal concentrating ability (defined as having a urine osmolality below 600 mOsm/kg water) was observed in a small group (n=6) of patients after 12 months on lithium treatment. Of these six patients, five had been treated previously with lithium and had some signs of impaired renal concentrating ability at the start of the present treatment. Multiple regression analysis determined that concurrent treatment with neuroleptics did not contribute to the decline of renal function.

Polyuria and/or decreased urine concentrating ability were found among 112 women and 125 men (average weight, 76.2 kg) exposed to 12-57 mmol/day of lithium (mean dose of 32.6 mmol/day of lithium or 3.0 mg Li/kg-day) for 0.5-17 years (mean duration, 5.2 years) in a retrospective study by Vestergaard et al. (1979). Serum lithium concentration ranged from 0.2 to 2.0 mmol/L. Baseline renal function prior to lithium therapy was not assessed, since all patients were on maintenance lithium therapy prior to the start of the study. The majority of patients were also receiving concomitant therapy with other medications, such as neuroleptics and/or antidepressants, and 37% the patients received concomitant therapy with hypnotics or anxiolytics. In a follow-up study, 184 of the original 237 patients were re-examined 2 years later (Vestergaard and Amdisen, 1981). The 184 patients were divided into two groups; those patients who continued with lithium (147) and those who discontinued (37). Lithium-treated patients were compared with 68 manic-depressive patients that were about to receive lithium. Glomerular function did not change over the 2-year period. In patients who had discontinued lithium treatment, there was an improvement in renal concentrating ability when compared with the patients who continued with lithium therapy. However, maximal urine osmolality did not reach the level found in the control (pre-lithium treatment) group, although the urine volume approached levels in the control group. There was a further increase in urine volume and decreased in urine osmolarity for those patients that continued with lithium therapy.

In a study involving 116 men and 152 women who took an average of 1322 mg/day of lithium carbonate (3.57 mg Li/kg-day) for an average period of 37.6 months, maximum concentrating ability was lower in all patients receiving lithium than in 59 control patients not receiving lithium (Gelenberg et al., 1987). However, differences did not achieve statistical significance. A major limitation of this study is that baseline data were not available.

Results of a biopsy study in patients receiving lithium maintenance therapy provide evidence of lithium-induced histopathological changes to the kidney (Hestbech et al., 1977). Fourteen manic-depressive patients received an average of 42 mmol Li/day as lithium carbonate (4.2 mg Li/kg-day) for 1.5-15 years. Serum lithium concentrations ranged from 0.6 to 1.3 mmol/L. Thirteen age-matched patients without renal disease served as the source of kidney biopsy specimens for control observations. Impaired urine concentrating ability and polyuria was observed in lithium patients. Histopathological examination of the kidney biopsy samples revealed a pronounced degree of focal nephron atrophy and/or interstitial fibrosis in 13 of the

14 patients examined. Semiquantitative assessment of renal lesions revealed significantly greater degrees of focal cortical fibrosis, diffuse medullary fibrosis, mononuclear cell infiltrates and distal tubular dilatation in the patients than in the controls. Quantitative assessment revealed significantly greater percentages of totally sclerotic glomeruli, fibrous cortical tissue and unidentifiable and atrophic renal tubules in patients than in controls.

Hansen et al. (1979) also reported impaired renal concentrating ability in 14 patients (7 men and 7 women) treated with 36 mmol/day of lithium (3.6 mg Li/kg-day) for 1.3 to 12 years. Serum lithium concentrations ranged from 1.75 to 4.50 mmol/L. Results of renal biopsy showed interstitial fibrosis and tubular atrophy. Baseline levels were not available and controls were not used. There was a significant negative correlation between the degree of tubular atrophy and renal concentrating ability.

Walker et al. (1982) examined renal function and biopsy samples in 47 patients (18 men and 29 women) who were receiving an average of 1250 mg/day of lithium carbonate (3.38 mg Li/kg-day) for an average of 5 years. The median serum lithium concentration was 0.84 mmol/L. Thirty-two patients not receiving lithium therapy were used as the controls. Decreased urine concentrating ability and impaired urinary acidification, indicative of distal nephron dysfunction, were observed in patients receiving lithium relative to controls. Lithium-treated patients also exhibited decreased glomerular filtration rate, as measured by significantly increased serum creatinine, increased β_2-microglobulins, and decreased Cr-EDTA clearance, compared to controls. Histological examination of kidney biopsy samples did not reveal abnormalities.

Hansen and Amdisen (1978) reported effects on the kidneys in a case study of 23 patients who were exposed to therapeutic doses of 24-56 mmol Li/day (2.4-5.6 mg Li/kg-day) for 6.1-8.5 years. Patients were hospitalized due to severe lithium intoxication. Pre-exposure baseline levels were not available. Impaired renal concentrating ability was a consistent finding. Abnormal electroencephalography (EEG) was also reported. There was no relationship between the severity of symptoms of lithium intoxication and the serum lithium concentration on admission to the hospital. Many of the patients (22 out of 23) included in the Hansen and Amdisen (1978) study experienced frank adverse effects, including renal insufficiency in 17 patients, mental and neurological symptoms (decreased alertness or slight apathy) in18 patients, muscular rigidity and/or muscular fasciculations in 14 patients, slight ataxia in 6 patients, and stupor and latent convulsive movements in 14 patients. Severely abnormal electroencephalograms were observed in 19 patients. Two patients died and two patients developed persisting neurological sequelae.

Studies on Adverse Effects in Patients Treated with Lithium: Other (Non-Renal) Adverse Effects – Neurological effects, including tremors, are commonly reported in patients treated with lithium. Neurological effects of lithium were evaluated in 28 patients (15 men and 13 women) with bipolar affective disorder receiving 1012 mg/day of lithium carbonate (2.74 mg/kg-day) for 4.1 years. The mean serum lithium level was 0.68 mmol/L. Although patients did not develop overt neurological effects, nerve conduction velocities were prolonged (Chang et al., 1990). Electrodiagnostic tests revealed a slowing of motor and sensory nerve conduction velocities and prolonged central neural conduction times obtained from somatosensory and brainstem auditory evoked potentials that correlated with serum lithium levels. Another patient

developed polyneuropathy after being exposed to 1.62 mg Li/kg-day for an unspecified period (years) (Tomasina et al., 1990). The patient had a sensorimotor peripheral neuropathy with mostly axonal degeneration. The patient improved after the lithium therapy was discontinued. Levine and Puchalski (1990) have also described two cases of pseudotumor cerebri syndrome in patients that were exposed to 900-1200 mg/day of lithium carbonate (2.43-3.24 mg Li/kg-day) for 4-8 years. The serum lithium concentration did not exceed 1.0 mmol/L. This syndrome is characterized by chronic headaches, bilateral papilledema and increased intracranial pressure in the absence of any localized neurological signs or symptoms.

Hagino et al. (1995) observed adverse effects on 20 children aged four through six exposed to oral lithium for the treatment of aggressive and/or mood-disordered children. All children were hospitalized during the course of the study as part of the medical intervention program. Daily lithium doses were adjusted to maintain serum lithium concentrations between 0.6 and 1.2 mmol/L and ranged from 12.2 to 48.9 mg/kg-day. Patients remained on lithium therapy for up to 37 days. Adverse effects to the central nervous system (tremor, drowsiness, ataxia, confusion) were the most commonly observed effects, reported in approximately 60% of patients. Other effects observed included gastrointestinal effects (nausea, vomiting, abdominal discomfort) in 25% of patients, renal effects (polyuria) in 10% of patients and blurred vision in 10% of patients. No adverse effects were observed in the cardiovascular, pulmonary, autonomic, hematological or integumental systems. The potential contribution of concomitant medications was not ruled out by the study authors. Sixteen of the 20 children also received one or more psychoactive medications and six children received antibiotics for infections.

Adverse effects on thyroid function, primarily asymptomatic hypothyroidism, have been reported in patients treated with lithium. Thyroid effects may be secondary to altered renal clearance of iodine, rather than to direct effects of lithium on the thyroid (Moore, 1995). A retrospective study was conducted involving 129 patients (46 men, 83 women) who were on lithium therapy for 2-180 months and 21 patients who served as controls (Bocchetta et al., 1991). Most of the patients receiving lithium had previously received or were receiving medication (antipsychotics and antidepressants) other than lithium. Serum lithium concentrations ranged between 0.5 and 1.0 mmol/L. Palpable and/or visible goiter was found in 51% of the patients receiving lithium ($p<0.01$), compared with 9.5% occurrence in the control group. Based on elevated thyroid stimulating hormone (TSH) levels, subclinical hypothyroidism was diagnosed in 19% of the patients on lithium compared with 9.5% in the control group. There were no differences in thyroid function tests between patients receiving lithium alone or receiving additional medication. The researchers noted that lithium-induced subclinical hypothyroidism may be transient and recommended that repeated determinations of TSH is required. Joffe et al. (1988) reported that 20% of the 42 patients receiving lithium carbonate therapy for 3 months required thyroid replacement or had evidence of subclinical hypothyroidism. Cowdry et al. (1983) also reported that 12 of 24 patients who were on lithium therapy for 12 months developed hypothyroidism. Only those patients with a median serum lithium concentration of 0.6 mmol/L were used in the study. When 22 women received lithium therapeutically for more than 2 years, there was evidence of subclinical hypothyroidism in 32% of these patients (Bartalena et al., 1990).

Hyperparathyroidism with progressive hypercalcemia was reported to occur in a patient who received lithium for 6 years (Graze, 1981). The daily dosage was not reported by the author. The patient had progressive hypercalcemia for the duration of the therapy and a parathyroid adenoma.

Raoof et al. (1989) demonstrated a concentration-dependent inhibition by lithium chloride on human sperm motility *in vitro* at semen concentrations that would be expected from therapeutic doses of lithium. However, Raboch et al. (1981) found no abnormality in sperm count, motility or morphology in semen samples obtained from 14 patients that were using lithium for an average of 4.1 years. The mean serum lithium level was 0.64 mmol/L and the mean lithium level in the semen was 1.48 mmol/L.

A group of 16 men and 4 women were treated with 1008 mg/day of lithium carbonate (2.72 mg Li/kg-day) as either a once-daily dose or as a divided twice-daily dose for an average of 4.4 years (Abraham et al., 1992). An elevated white cell count, increased serum phosphate, and elevated serum ionized calcium were observed in the study group receiving the once-daily lithium treatment. These effects were not seen in the study group that received lithium twice a day. No evidence of polyuria was observed in either group and no significant differences were observed between the two treatment regimens with respect to mental status, serum lithium or electrocardiograms.

Adverse effects on cardiac conductivity, including sinoatrial block, sinus bradycardia and junctional escape rhythm, have been reported in patients taking therapeutic lithium (Moore, 1995). A patient who received 600 mg/day of lithium carbonate (1.62 mg Li/kg-day) for 4 months (serum lithium concentration 1.3-2.0 mmol/L) developed symptomatic sinus node dysfunction, which disappeared after discontinuation of lithium therapy (Riccioni et al., 1983). Roose et al. (1979) also reported cardiac sinus node dysfunction during lithium treatment in several patients exposed to at least 8.6 mg/kg-day of lithium for 10 years.

Studies on Developmental Effects of Lithium Treatment During Pregnancy – The potential for lithium-induced developmental effects was the subject of an assessment conducted by Moore and an Institute for Evaluating Health Risks (IEHR) Expert Scientific Committee (Moore, 1995). Data from 139 references, including registries, prospective studies and case histories were reviewed. This assessment determined that sufficient evidence is available to conclude that therapeutic use of lithium causes developmental effects in offspring when maternal serum lithium concentrations are within the therapeutic range. Review of developmental effects reported in birth registries revealed reports of cardiovascular defects associated with lithium treatment. Reports of Ebstein's anomaly (a structural defect in which there is a downward displacement of the trisucpid valve into the right ventricle, and valvular redundancy with adherence of some cusps to the right ventricular wall; affected individuals may have right ventricular failure or conduction abnormalities), in particular, were in "substantial excess among all malformations." Although the magnitude of the increase could not be determined from birth registry reports, data indicate that first-trimester lithium exposure increases the risk of cardiac malformations. Other studies reviewed by Moore (1995) also report an association between maternal lithium treatment and cardiovascular defects in offspring. The literature reviewed also suggests a possible association between maternal lithium treatment and neonatal mortality.

There are also reports that newborn infants of mothers on lithium therapy may exhibit symptoms of acute lithium toxicity such as cyanosis, hypotonia and cardiac toxicity. However, the available data regarding developmental effects in humans are limited by insufficient dose-response information.

Jacobson et al. (1992) prospectively recruited and followed 148 women using lithium during the first trimester of pregnancy. Each study patient was matched with a woman (control) of similar age (within 2 years). The mean lithium dose was 927 mg/day of lithium carbonate (2.5 mg Li/kg-day); the authors did not report serum lithium concentrations. No significant differences between the exposed group and the controls were observed for congenital defects (3% in lithium patients and 2% in controls) and spontaneous abortions (9% in lithium patients and 8% in controls). Kallen and Tandberg (1983) identified a cohort of 350 mothers who were treated with lithium during their pregnancy. The authors reported that the total delivery outcome was poorer than expected, with high perinatal death and malformation rates compared to the national average expected rates in Sweden. Congenital heart defects occurred in 6 cases compared with the national expected number of 2.1 cases ($p<0.05$). However, the sample size was relatively small and the difference between delivery outcome in women on lithium and in women on other psychotropic drugs was not statistically significant. Weinstein and Goldfield (1975) reviewed 143 cases of lithium use during pregnancy collected by the Register of Lithium Babies. There were 13 malformed infants (9.1%) among the 143 in the register. Of these 13 malformed infants, 11 were born with significant malformations of the cardiovascular system. As was the case with the previous study, the daily lithium dosage was not reported and at least 6 of the 13 mothers who delivered malformed babies were exposed to other medications in addition to lithium. Krause et al. (1990) reported a case of severe polyhydramnios that developed from the 26th week of gestation. Except for weeks 6-13 of gestation, the mother was maintained on lithium (serum level, 0.7 mmol/L) prior to the diagnosis. Ang et al. (1990) described a similar case report in a woman who was exposed to lithium during pregnancy. The infant displayed symptoms of lithium toxicity, including polyuria.

Studies on the Carcinogenic Potential of Therapeutic Lithium – Controlled studies on the potential of therapeutic lithium to induce cancer have not been reported. Although a few case studies have reported associations between lithium therapy and recurrence of cancer, data are inadequate to establish any association between lithium and the development or recurrence of cancer in humans. Furthermore, given that the widespread clinical use of lithium as a long-term maintenance treatment in patients with affective mood disorders has not revealed an increased incidence or recurrence of cancer, it is unlikely that lithium is carcinogenic in humans. Nonetheless, the few studies examining potential carcinogenic effects of lithium are briefly reviewed below.

Several case reports that suggest an association of lithium-induced leukocytosis with induction or reinduction of acute and chronic leukemia. Orr and McKerna (1979) reported the recurrence of acute monocytic leukemia in a 64-year-old woman who was previously in remission. This patient received 600 mg/day of lithium carbonate for 7 weeks before the leukemic relapse occurred. Nielsen (1980) reported the development of acute myeloid leukemia in one male and one female patient administered lithium for a duration of 1 and 12 years, respectively. Jim (1980) reported the occurrence of chronic monocytic leukemia in a patient

who received 900 mg/day of lithium carbonate for 11 months prior to the diagnosis of leukemia. A 37-year-old woman developed chronic granulocytic leukemia after receiving 600 mg of lithium carbonate 3 times/day for 5 years (Schottlander et al., 1980).

Contrary to anecdotal reports that attempt to associate an increased risk of leukemia with lithium intake, the limited epidemiological information suggests no increased risk. Resek and Olivieri (1983) examined the relationship between leukemia and chronic lithium therapy during 1971-1980 by examining hospital records of 187 leukemia patients to determine whether these patients were receiving lithium medication prior to the their illness. Only 7% of these patients had received psychiatric services and in all cases, these patients were not treated with lithium. The authors reported that there was no association between lithium therapy and leukemia. In a 14-year ecological study, one human population in El Paso exposed to lithium via drinking water (66 μg/L) was compared with another human population in Dallas-Fort Worth that was not exposed to lithium in drinking water (Frenkel and Herbert, 1974). There was no difference in the incidence of chronic or acute granulocytic leukemia in the two populations.

Only two separate cases have been reported in the literature of the possible association of lithium with cancers other than leukemia. Brownlie et al. (1980) reported the occurrence of papillary cell carcinoma of the thyroid in a 55-year-old woman after 3.5 years of lithium therapy. McHenry et al. (1990) also reported three cases of thyroid carcinoma occurring in association with chronic (9 years) lithium therapy.

Studies of Adverse Effects of Lithium in Healthy Volunteers – The effect of lithium therapy on short- and long-term memory was assessed in healthy volunteers exposed to daily oral lithium for 3 weeks (Stip et al., 2000). Groups of 15 healthy men and women were randomized into placebo or lithium treatment groups. Subjects in the lithium group were administered lithium twice daily at doses ranging from 1050 to 1950 mg/day (197 to 366 mg Li/day) in order to achieve a mean serum lithium concentration of 0.8 mmol/L. The form of the lithium was unstated but the dose is consistent with lithium carbonate. Actual serum lithium concentrations were not reported. Cognitive performance (attention and memory) was assessed in each subject at 3 times during the study: baseline, after 3 weeks of treatment and 2 weeks after discontinuation of treatment. After 3 weeks of treatment, performance scores for short-term memory tasks (assessed using an auditory digit span) for subjects taking lithium were significantly lower ($p<0.03$) compared to placebo. Results of long-term memory assessments (using recall tests) showed adverse effects in lithium-treated subjects compared to controls. Performance on short- and long-term memory tests improved 2 weeks after discontinuation of treatment. Results indicate that lithium produces effects in the central nervous system in healthy subjects at exposure levels corresponding to the target therapeutic serum concentrations. The mean dose, 1569 mg/day (295 mg Li/day) was a LOAEL in this study.

Effects on the hemopoeitic system and clotting have also been reported in healthy volunteers exposed to lithium (Stein et al., 1981). Groups of at least five non-psychiatric volunteers received 900 mg/day lithium carbonate (2.43 mg Li/kg-day). Granulocyte count, expressed as a percent of baseline, was significantly increased by 25% ($p<0.05$), 32% ($p<0.001$), and 42% ($p<0.001$) after 1, 2 and 3 weeks of exposure. Volunteers administered 0, 300, 600, 900, 1200 or 1500 mg/day of lithium carbonate (0, 0.8, 1.62, 2.43, 3.24 or 4.05 mg Li/kg-day)

orally for 1 week developed increased granulocytosis at doses ≥ 2.43 mg Li/kg-day. Granulocyte count was increased by 26, 55 and 43% of baseline values in the 2.43, 3.24 and 4.05 mg Li/kg-day groups, respectively. Decreased bleeding times were also observed in the 3.24 and 4.05 mg Li/kg-day dose groups, although there was no apparent treatment effect on platelet count. Serum lithium concentrations were not reported.

The use of lithium as a therapeutic agent to reverse chemotherapy-induced neutropenia and thrombocytopenia has been explored in humans, with studies providing conflicting results. Richmon et al. (1984) reported an increase in neutrophil and thrombocyte production in five cancer patients who received 900 mg/day of lithium carbonate (169 mg Li/day) for an unspecified duration. Twenty-two patients with oligoblastic leukemia receiving 900 mg/day of lithium carbonate for an unspecified time remained cytopenic without evidence of lithium-induced bone marrow proliferation (Barlogie et al., 1984). In another study, Friedenberg and Marx (1980) reported that lithium increased the granulocyte count in eight healthy volunteers who had received 900 mg/day of lithium carbonate for 1 week. Despite the observed increase in granulocyte number, there was a reduction in bactericidal capacity (function) of granulocytes in these individuals.

Inhalation Exposure

No studies on the effects of inhaled lithium in humans were identified.

Animal Studies

Oral Exposure

Subchronic and chronic oral exposure studies evaluating comprehensive toxicity endpoints in laboratory animals are not available. Few animal studies have investigated the adverse effects of chronic oral exposure to lithium. The primary purpose of animal studies has been to evaluate specific adverse effects associated with the therapeutic serum lithium concentration range, with most studies focusing on lithium-induced renal toxicity. The available data in animals provide supporting evidence that subchronic and chronic oral exposure to lithium induces similar adverse effects as those associated with the therapeutic use of lithium in patients. However, insufficient data are available to determine dose-response relationships for adverse effects.

Cancer Bioassays – No long-term animal bioassays examining the carcinogenicity of lithium were identified. An abstract by Prolov and Pliss (1991) reported that lithium carbonate promoted bladder carcinogenesis in rats previously exposed to N-buty-N-(4-hydroxybutyl)-nitrosamine; no additional publications of this finding were identified. Although Hori and Oka (1979) stimulated cell multiplication of mammary gland explants with lithium in C3H/HeN virgin female mice, Ziche et al. (1980) were unable to demonstrate any growth promoting effect of lithium on primary carcinomas induced by two chemical carcinogens (7,12-dimethylbenz[α]anthracene and N-nitrosomethylurea) in Sprague-Dawley and Buffalo/N female rats.

Adverse Renal Effects – Chronic renal failure was induced in male and female Wistar rats fed diets containing 0 or 40 mmol lithium chloride/kg diet (0 or 3.58 mg Li/kg-day) from birth for 55 to 56 weeks (Christensen and Ottosen, 1986). Plasma lithium concentration ranged from 0.6 to 0.7 mmol/L after 16 weeks and from 1.0 to 1.1 mmol/L after 48 weeks of treatment. Mortality was 51% in lithium-treated rats compared to only 6% in control rats. Mean plasma urea concentration was elevated by 74% after 16 weeks and 175% after 48 weeks, compared to controls. After 55 weeks of treatment, inulin clearance was reduced by 62% and lithium clearance was reduced by 39% compared to controls. Lithium-treated rats also had polyuria and diminished renal concentrating ability (assessed by failure to respond to exogenous vasopressin). No treatment-related effects on systolic or diastolic blood pressure were observed. Morphological examination of the kidneys of lithium-treated rats revealed large cortical cysts, dilated distal tubules and collecting ducts, and widespread interstitial fibrosis. Glomerular volume and proximal tubular mass were significantly reduced. Comprehensive toxicity endpoints were not examined in this study. A LOAEL of 3.58 mg Li/kg-day for adverse renal effects was identified; a NOAEL was not established.

Two groups of six male Wistar SPF rats were exposed to a diet containing 0 or 40 mmol LiCl/kg diet (0 or 4.5 mg Li/kg-day) for 3 weeks and then 0 or 60 mmol LiCl/kg (0 or 6.7 mg Li/kg-day) for an additional 18 weeks (Christensen et al., 1982). The time-weighted average daily dose was 6.4 mg Li/kg-day, with mean serum lithium concentrations of 0.7-0.8 mmol/L. Rats exposed to lithium developed polyuria and lowered renal concentrating ability after 2 weeks of exposure to diets containing lithium. Focal microscopic changes in distal convoluted tubules and collecting ducts were also observed. Focal basal vacuolization of the cytoplasm was observed after 2-4 weeks of lithium exposure. After 8 weeks of treatment, all rats had severe nuclear polymorphism, nuclear hyperchromasia and cellular polymorphism with tubular giant cells. In addition to these cellular changes, dilatations of the tubular lumen and focal atrophy of the tubular cells were observed in rats exposed to lithium for 21 weeks. Renal concentrating ability was significantly decreased after 2 weeks of dietary exposure. After lithium was withdrawn for 8 weeks, structural changes persisted, but concentrating ability was normalized. Based on their experimental findings, the authors concluded that the use of urinary concentrating ability as an index of lithium-induced structural damage may underestimate lithium-induced effects on the kidney. A LOAEL of 4.5 mg Li/kg-day for adverse renal effects observed after 2 weeks of treatment was identified; a NOAEL was not established.

Polyuria and vasopressin-resistant diabetes insipidus developed within 3 weeks of exposure to dietary lithium (Kling et al., 1984). Two groups of 12 male Wistar rats were exposed to 0 or 90 mmol/kg diet of lithium carbonate (11.6 mg Li/kg-day) for 126 days. Serum lithium concentration was maintained at 0.8 mmol/L. Early lesions were associated with the cortical collecting tubules and distal tubules, extending into the medullary collecting tubules by week 3 of treatment. There were alterations in nuclear size and shape and cytoplasmic basophilia of tubular cells, focal dilation and thinning of the tubular epithelium, and occasional sloughing of cells into the tubular lumen. In this study, early tubular lesions correlated with the polyuria. However, polyuria remained constant while morphological changes deteriorated for several more weeks of lithium exposure. A LOAEL of 11.6 mg Li/kg-day for adverse renal effects was identified; a NOAEL was not established.

Two groups of seven male Wistar rats were exposed to a control diets or a diet containing lithium for 112 days (Marcussen et al., 1969). Due to poor reporting of methods and results, the concentration of lithium in the diet or daily dose of lithium could not be determined. Rats exposed to lithium developed uremia and reduced body weight. All kidneys were polycystic in the cortical areas, and distal tubules and cortical collecting ducts were dilated. Severe fibrosis was observed in the interstitial space. Tubular glomeruli (67%) and some hypertrophic glomeruli were also observed. The hypertrophic glomeruli did not compensate adequately for other impaired glomerular function, as indicated by an overall decrease in GFR.

Other (Non-Renal) Adverse Effects – Reductions in body weights were observed in rats exposed to dietary lithium (Ehlers and Koob, 1985). Twenty-nine male Wistar rats were exposed to 0, 30 or 40 mmol Li/kg diet (0, 19 or 26 mg Li/kg-day) for 56 days. In the 40 mmol Li/kg diet group, body weight was reduced by 37% ($p<0.001$). A significant increase ($p<0.05$) in brain theta wave activity in the 6-8 Hz range in all lithium-treated animals was also observed. A LOAEL of 19 mg Li/kg-day for adverse effects to the central nervous system was identified; a NOAEL was not established.

The effect of dietary lithium on thyroid function was examined in two groups of 10 male Wistar rats exposed to 0 or 1100 mg lithium carbonate/kg diet (0 or 20 mg Li/kg-day) for 120 days (Dhawan et al., 1985). Serum lithium levels ranged from 0.44 to 0.65 mmol/L. There was a significant decrease ($p<0.01$) in circulating levels of T_4 and T_3 after 1 month of exposure to lithium. There was also a marked decrease ($p<0.001$) in thyroid hormone levels after 4 months of lithium treatment. A LOAEL of 20 mg Li/kg-day for adverse thyroid effects was identified; a NOAEL was not established. Etling et al. (1987) investigated the effect of lithium on thyroid hormone levels in rats exposed to 0, 300 or 600 mg lithium carbonate/L in drinking water (0, 12.8 or 25.6 mg Li/kg-day) for 5 weeks. Decreases in serum T_3 and T_4 were observed only in the 600 mg/L group. A NOAEL and LOAEL of 12.8 and 25.6 mg Li/kg-day, respectively, for adverse thyroid effects were identified.

The effect of 90-day dietary exposure to lithium on male reproductive organs was evaluated by Thakur et al. (2003). Groups of 20 sexually mature Wistar rats were exposed to lithium carbonate at dietary concentrations of 0, 500, 800 or 1100 mg/kg diet for 90 days (equivalent to 0, 6.6, 10.6 or 14.2 mg Li/kg body weight-day). Serum lithium concentrations were not reported. Assessments included weight of reproductive organs, histopathology of testis, epididymis, seminal vesicle, and prostate, testicular interstitial fluid volume, testosterone level, sperm morphology and fertility index. Weights of the testes and epididymis, sperm number from cauda epididymis, daily sperm production, serum testosterone and interstitial fluid volume were significantly reduced in the mid- and high-dose groups, compared to controls. Seminal vesicle and prostate secretions were completely blocked in the mid- and high-dose groups. The percentage of abnormal sperm was significantly increased in all lithium treatment groups. Histopathological assessments revealed degeneration of spermatogenic cells and vacuolization of Sertoli cell cytoplasm in the high-dose treatment groups. A LOAEL of 6.6 mg Li/kg body weight-day for increased percentage of abnormal sperm was identified; a NOAEL was not established.

Sharma and Iqbal (2005) evaluated the effects of oral exposure of male Wistar rats to lithium nitrate for 7 weeks. Groups of 12 rats were exposed to 0 or 20 mg Li/kg body weight by gavage on alternate days (10 mg Li/kg-day) for 7 weeks and examined for effects on blood chemistry and hematology at the end of the treatment period. Serum lithium concentration was not reported. Numerous blood chemistry and hematology parameters were significantly different from controls: decreased hemoglobin and erythrocyte count, elevated white cell count, elevated erythrocyte sedimentation rate, elevated glucose, decreased protein and elevated blood urea nitrogen (BUN), calcium and phosphorous. Histopathological effects observed in the kidney included ruptured epithelial lining of the proximal and distal tubules of the medulla, renal tubular necrosis, thickened capsular wall of the glomerulus and cytoplasmic vacuolization in the corticomedullary region. Comprehensive toxicity endpoints were not examined in this study. A LOAEL of 10 mg Li/kg body weight for hematological and renal effects was identified; a NOAEL was not established.

Developmental Effects – The review by Moore and an IEHR Expert Scientific Committee (Moore, 1995) on lithium-induced developmental effects included available data from studies in animals using a variety of experimental designs. The data in animals are of limited usefulness to providing a comprehensive picture of lithium-induced developmental effects because of limitations of available studies. Issues with some of the studies include small number of animals, inability to ascertain litter incidence, inadequate reporting, administration of only a single dose, and failure to report or describe chemical characteristics of test materials. Despite these limitations, sufficient data are available to suggest that prenatal developmental toxicity can occur in studies with rats and mice in which lithium is administered during pregnancy and fetuses are examined just before birth. Doses associated with adverse developmental effects ranged from 2.71 to 12.67 mmol/kg body weight-day. Evidence of maternal toxicity was often present. Specific cardiac developmental effects have not been reported in animal studies; however, it does not appear that rigorous assessments of cardiac morphology have been conducted. Results of selected animal developmental studies are briefly summarized below.

Hoberman et al. (1990) evaluated the developmental effects of lithium hypochlorite administered by gavage once daily to groups of 25 Sprague-Dawley rats on days 6 through 15 of gestation at dosages 0 (vehicle), 10, 50, 100 or 500 mg/kg-day (0, 0.4, 2.1, 4.2 or 21 mg Li/kg-day). Six of the 25 rats in the 500 mg/kg-day group died between days 12 and 20 of gestation. Decreased fetal body weight, wavy ribs and delayed ossification of the thoracic vertebrae (bifid centra), forepaw and hindpaw phalanges, and metatarsal and metacarpal bones were observed in the offspring of the highest exposure group. Maternal NOAEL and the developmental NOAEL for lithium hypochlorite were determined by the authors to be 100 mg/kg-day of lithium hypochlorite (4.2 mg Li/kg-day). The LOAEL for developmental toxicity is 500 mg/kg-day (21 mg Li/kg-day) and the maternal frank effect level (FEL) is 500 mg/kg-day (21 mg/kg-day).

Twenty albino Sprague-Dawley rats were exposed to 0 or 100 mg/kg-day (0 or 18.8 mg Li/kg-day) lithium carbonate on days 16 through 20 of gestation (Fritz, 1988). Signs of maternal toxicity, including reduced weight gain and feed consumption, polyuria and polydipsia, were observed. Enlarged renal pelvises were observed in 50% of the fetuses in the lithium-exposed

group. Exposure of rats to a lower daily dose of lithium carbonate (11.3 mg Li/kg-day) on gestational days 16 through 20 induced similar maternal effects and some prenatal mortality; however, no signs of impaired renal development for the young offspring that survived were observed. A LOAEL of 11.3 mg Li/kg body weight for maternal effects was identified; a NOAEL was not established. For fetal effects, NOAEL and LOAEL values were identified as 11.3 and 18.8 mg Li/kg-day, respectively.

Developmental toxicity was evaluated in the offspring of 44 pregnant Wistar rats exposed to 0, 50 or 100 mg/kg-day of lithium carbonate (equivalent to 0, 9.5 or 19 mg Li/kg-day) by oral gavage on gestational days 6 through 15 (Marethe and Thomas, 1986). Reduction in the number of implantations, number of live fetuses and fetal body weights and a higher number of resorptions were reported in the 100 mg/kg-day group. A developmental NOAEL for lithium carbonate is 50 mg/kg-day (9.5 mg Li/kg-day) and a LOAEL of 100 mg/kg-day (19 mg Li/kg-day) was determined.

Statistically significant reductions in total body weights of the fetus and the fetal length were observed in the offspring of albino rats exposed to 7 mg/kg-day of lithium carbonate (1.3 mg Li/kg-day) for the first 10 days of gestation (Sharma and Rawat, 1986). The authors did not report the number of animals used in the study, but rather the number of abnormal developmental observations and expressed these as a percentage of control. The authors also reported a high incidence of cleft palate abnormalities (46%), fetal brain liquification (46%), hepatomegaly (46%) and non-ossification of upper and lower digits (30 and 37%, respectively). Lower incidences of cardiomegaly (3%) and hydronephrosis (3%) were observed. A LOAEL of 1.3 mg Li/kg body weight for adverse developmental effects was identified; a NOAEL was not established.

Groups of 12 female Sprague-Dawley rats were fed 0 or 1000 ppm lithium carbonate (0 or 18.5 mg Li/kg-day) in the diet throughout gestation (Ibrahim and Canolty, 1990). Following parturition, the dams were exposed to the same concentration of lithium and were also allowed to nurse the pups for an additional 21 days. Dietary lithium resulted in decreased growth in both the dams and the offspring, as well as increased mortality of the offspring. Litter size was decreased 25% and mean pup weight was decreased by 10%. The highest mortality was observed in the group of pups that were exposed to lithium during both gestation and lactation. Gross malformations were not observed in the newborn animals. A LOAEL of 18.5 mg Li/kg body weight for adverse developmental effects was identified; a NOAEL was not established.

Groups of three to six mice (HmM/ICR strain) were exposed to 0, 200 or 465 mg/kg-day of lithium carbonate (0, 37.8 or 87.9 mg Li/kg-day) on gestational days 6 through 15 (Szabo, 1970). The human equivalent dose for mice, based on lithium plasma levels of 0.6-1.6 mmol/L, was calculated by the authors as 465 mg/kg-day. The highest dose, 465 mg/kg-day, caused an increased incidence of maternal (37%) and fetal (32%) deaths. Nineteen percent of the surviving fetuses had cleft palate. The 200 mg/kg-day dose (37 mg Li/kg-day) did not cause maternal or fetal deaths; the incidence of cleft palate in fetuses was 0.4%, which was not statistically significantly elevated relative to controls. Cleft palate was not observed in any of the 181 fetuses in the control group. NOAEL and LOAEL values for maternal effects and developmental effects were identified as 37.8 and 87.9 mg Li/kg-day, respectively.

Inhalation Exposure

Only three studies that evaluated the effect of inhaled lithium in animals were identified (Johansson et al., 1988; Greenspan et al., 1986; Rebar et al., 1986). Greenspan et al. (1986) exposed groups of eight male and eight female F344/Lov rats to an aerosol mixture containing lithium carbonate (80%) and lithium hydroxide (20%) at concentrations of 0, 620, 1400, 2300 or 2600 mg Li/m^3 once for 4 hours (Greenspan et al., 1986). The 14-day LC_{50} values were estimated to be 1700 mg Li/m^3 for the males and 2000 mg Li/m^3 for the females from the single exposure. No clinical signs of toxicity were observed in animals exposed to 620 mg Li/m^3. At concentrations $\geq$ 1400 mg/m^3, signs of acute effects on the respiratory system included respiratory distress and bronchospasms. At necropsy, severe congestion of the lungs was observed in 14 out of the 16 animals in the two highest exposure groups. Three of 16 animals at 1400 mg Li/m^3 showed congestion in the lungs. Histopathologic lesions in the respiratory tract were seen in the $\geq$ 1400 mg/m^3 groups, and were found only in animals dying within 12 days of exposure. Lesions were characterized as necrotizing rhinitis, necrotizing laryngitis and secondary suppurative bronchiolitis and bronchopneumonia. There was congestion of the thymus and tracheobronchial lymph nodes in almost half of the animals exposed to lithium aerosols. Similar observations were reported by Rebar et al. (1986) in groups of eight male and eight female rats that were exposed to an aerosol mixture of lithium monoxide and lithium hydroxide at concentrations of 0, 570, 840, 1200 or 1500 mg Li/m^3 for 4 hours. The 14-day LC_{50} value was 940 mg/m^3. In this same study, the 14-day LC_{50} value for a 4-hour exposure to an aerosol containing only lithium hydroxide was 960 mg Li/m^3. Clinical signs and pathological changes were similar to those described in the Greenspan et al. (1986) study. Exposure to the lithium hydroxide/lithium monoxide aerosol mixture at concentrations of 570 Li mg/m^3 and greater resulted in upper respiratory tract and pulmonary lesions.

No adverse respiratory effect were observed in groups of eight male rabbits exposed to aerosols of 0, 0.1 or 0.32 mg Li/m^3 as lithium chloride for 6 hours/day, 5 days/week for 4-8 weeks (Johansson et al., 1988). Inhalation of lithium chloride produced no adverse effects on lung morphology or phospholipid content. The number of alveolar macrophages in lithium-treated animals was not different compared to controls.

Supporting Studies

Toxicokinetic

The clinical pharmacokinetics of lithium has been extensively studied. Reviews and clinical pharmacology text books provide summaries of the pharmacokinetic profile of therapeutic lithium (Ward et al., 1994; Baldessarini and Tarazi, 2001; Potter and Hollister, 2001). The bioavailability of oral lithium preparations ranges from 80 to 100%, although it is generally accepted that the oral bioavailability of lithium is 100%. Peak plasma concentrations are typically reached within 2 hours of administration of lithium. Lithium does not bind to plasma proteins. The volume of distribution of lithium is calculated as 0.66 L/kg. Although lithium is distributed into total body water, lithium distribution is not uniform in all tissue compartments. As an element, lithium is not metabolized and is excreted intact, primarily by the kidney; elimination through sweat, saliva and feces is negligible. Approximately 80% of the filtered load

of lithium is reabsorbed by the kidney and elimination correlates with renal function. Excretion follows first-order kinetics, with an average half-life of 22 hours and an average clearance of 0.35 mL/min-kg (0.5 L/day-kg).

Genotoxicity

Moore (1995) reviewed genotoxicity data as part of their assessment of the developmental effects of lithium and concluded that there is no evidence demonstrating genetic toxicity of lithium in bacterial or *in vitro* mammalian test systems. Garson et al. (1981) reported no increase in the occurrence of chromosome breaks in 23 human subjects under continuous lithium treatment for a period of 1-8 years when compared with 19 healthy age-matched controls. Lithium hypochlorite was not mutagenic in the Ames test nor did it induce DNA damage in the unscheduled DNA synthesis assay using rat primary hepatocytes or increase chromosome aberrations when tested orally in rats at maximally tolerated doses (Weiner et al., 1990).

DERIVATION OF PROVISIONAL SUBCHRONIC AND CHRONIC RfD VALUES FOR LITHIUM

The use of lithium as a long-term maintenance therapy in the treatment of bipolar affective disorders has led to an extensive body of literature on the adverse effects associated with oral lithium therapy. Adverse effects, which are observed in several organs and systems, are associated with the entire target therapeutic serum lithium concentration range, leading to treatment strategies based on a risk-benefit assessment for individual patients. The available clinical data identify the lower bound of the therapeutic serum lithium concentration range (0.6 mmol/L) as a LOAEL; the clinical literature does not identify a NOAEL for adverse effects associated with therapeutic lithium. Data reported in humans studies are not sufficient to define the relationship between serum lithium concentrations and the development or severity of adverse effects, although it is generally accepted that the severity of adverse effects is related to serum lithium levels. Given the lack of adequate dose-response data, a single critical effect cannot be identified for lithium. Occupational and environmental oral exposure studies in humans are not available.

Adverse renal effects associated with lithium therapy have received extensive focus due to their serious nature and frequency of occurrence. The most common adverse renal effect is impaired renal concentrating ability and the production of excessively dilute urine. Clinically, this manifests as polyuria, with secondary thirst, and volume depletion. The onset of impaired renal concentrating capacity typically is within the first 2 years of treatment. Although altered renal function appears to be reversible early in treatment, it may be progressive during the first decade of lithium treatment, leading to irreversible damage over time.

Lithium therapy produces side effects in a number of organs and systems other than the kidney. The most frequent neurologic side effects are lethargy, fatigue, weakness, tremor and cognitive impairment. Endocrine glands such as the thyroid and parathyroid can also be affected. Although serious cardiovascular effects are rare, they do occur, the most common

being changes in the EKG. Gastrointestinal side effects include nausea, vomiting, diarrhea and abdominal cramping. The most frequently observed hematological reaction is a benign leukocytosis. Developmental effects, primarily involving the heart, undoubtedly represent the most serious type of unwanted effects.

The available animal data provide supportive evidence that lithium produces adverse effects in several organs and systems at exposure levels that result in serum lithium concentrations in same range as that targeted for therapeutic use in humans. However, available studies do not evaluate comprehensive toxicity endpoints or identify a NOAEL for adverse effects. Thus, although results of toxicity studies in animals are consistent with the adverse effects profile in humans exposed to therapeutic lithium, data are not suitable as the basis for the provisional subchronic and chronic RfD.

The lower bound of the therapeutic serum lithium concentration range of 0.6 mmol/L is selected as the basis for derivation for the provisional RfD and subchronic RfD (p-RfD; p-sRfD). Given that the adverse effects profile of therapeutic lithium is similar for patients on short- and long-term lithium therapy, an RfD based on the LOAEL of 0.6 mmol Li/L is applicable for subchronic and chronic exposures. Based on the pharmacokinetic considerations detailed below, to achieve a serum lithium concentration of 0.6 mmol Li/L, the daily ingestion of lithium by a 70-kg individual is calculated as approximately 1.8 mg Li/kg-day.

At steady state,

$$D = \frac{C_P \cdot Cl}{f}$$

where D is the dose (mg/kg-day), C_p is the plasma concentration (mg/L), Cl is the plasma clearance (L/kg-day) and f is the fraction of the dose absorbed. Assuming values of 0.5 L/kg-day for Cl and 1 for f (Baldessarini and Tarazi, 2001), a steady-state plasma concentration of 0.6 mmol/L (4.2 mg Li/L) corresponds to a daily dose of 2.1 mg Li/kg-day.

The provisional subchronic and chronic RfD for lithium was derived from the LOAEL of 2.1 mg/kg-day for adverse effects in several organs and systems. Dividing the LOAEL of 2.1 mg/kg-day by an uncertainty factor of 1000 yields a subchronic and chronic p-**RfD of 0.002 mg/kg-day or 2 µg/kg-day**.

p-RfD = LOAEL ÷ UF
= 2.1 mg/kg-day ÷ 1000
= 0.002 mg/kg-day or 2 µg/kg-day

The composite uncertainty (UF) of 1000 includes a factor of 10 to extrapolate from a LOAEL to a NOAEL, a factor of 10 to protect susceptible individuals and a factor of 10 to account for database insufficiencies as follows:

- A default 10-fold UF for extrapolation from a LOAEL to a NOAEL was used because the lower bound of the therapeutic serum lithium range is associated with the development of adverse effects in several organs and systems; a NOAEL for adverse effects of therapeutic lithium has not been established in the clinical or animal literature.
- A default 10-fold UF was used to account for potentially susceptible individuals in the absence of quantitative information on the variability of response to lithium in the human population. Since lithium adversely affects several organs and systems, numerous pre-existing disease states (e.g., renal disease, cardiovascular disease, endocrine disease) may increase susceptibility to lithium.
- A UF of 10 was applied for database uncertainties. The renal effects of lithium have been extensively studied in humans and animals. However, much less information is available on the effects of lithium in other systems, including the cardiovascular, neurological and endocrine systems, and subchronic and chronic exposure studies in animals assessing comprehensive endpoints are not available. Furthermore, although lithium appears to produce developmental effects in humans, the database lacks well-controlled epidemiology studies and multi-generation reproduction studies in animals.

A wide range of estimates for daily dietary intake of lithium has been reported. Several authors report estimates for the average daily dietary intake of lithium, ranging from 0.24 to 1.5 μg/kg-day (Noel et al., 2003; Clarke et al., 1987; Hamilton and Minski, 1973; Evans et al., 1985; Clark and Gibson, 1988). A much higher estimate for daily intake from food and municipal drinking water ranging from 33 to 80 μg Li/kg-day was reported by Moore (1995). The source of the discrepancy between these estimates is unknown. The p-RfD of 2 μg/kg-day is above most estimates of daily dietary intake, but below the range estimated by Moore (1995).

Confidence in the LOAEL value is low-to-medium. Since the clinical literature has focused on the therapeutic treatment of patients, information on effects observed below the minimally effective dose is lacking. Confidence in the database is also low-to-medium. Although there is an extensive database demonstrating the adverse effects of chronic exposure to therapeutic lithium, information regarding the dose-response relationship of lithium to the development of adverse effects is lacking. Thus, the relative sensitivity of the different target organs cannot be identified based on human data. Furthermore, since most animals studies have been designed to evaluate specific adverse effects associated with the therapeutic serum lithium concentration range, NOAEL and LOAEL values have not been established in animals studied for comprehensive toxicity endpoints. The database also lacks well-controlled epidemiology studies and multi-generation reproduction studies in animals, even though there is evidence of developmental effects in lithium patients. Low-to-medium confidence in the p-RfD is the result.

FEASIBILITY FOR DERIVING A PROVISIONAL SUBCHRONIC RfC FOR LITHIUM

No studies investigating the effects of acute, subchronic or chronic inhalation exposure to lithium in humans were identified. The available studies in animals did not evaluate comprehensive histopathological, biochemical and clinical endpoints of inhalation exposure.

Thus, due to the lack of data, derivation of a provisional subchronic or chronic RfC for lithium is precluded.

PROVISIONAL CARCINOGENICITY ASSESSMENT FOR LITHIUM

Weight of Evidence Descriptor

Cancer studies in humans and cancer bioassays in animals exposed to lithium by the oral or inhaled routes were not found. Results of *in vitro* and *in vivo* studies in bacterial and mammalian systems indicate that lithium is not genotoxic. Under EPA's *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005), the hazard descriptor, "data are inadequate for an assessment of human carcinogenic potential," is appropriate for lithium.

Quantitative Estimates of Carcinogenic Risk

Due to the lack of data, derivation of an oral cancer slope factor and an inhalation cancer unit risk are precluded.

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APPENDIX C

APPENDIX C

Particulate Emission Factor Calculations

Table C-1
Calculation of the Particulate Emission Factor Based on Wind Erosion

$$PEF\ (m^3/kg) = Q/C \times \frac{3{,}600\ sec/hour}{0.036 \times (1 - V) \times (U_m/U_t)^3 \times F(x)}$$

Where:		**Value**
PEF =	Particulate emission factor (m^3/kg).	6.31E+09 (calculated)
Q/C_{wind} =	Inverse of the ratio of the geometric mean air concentration to the emission flux at the center of a square source (g/m^2-s per kg/m^3).	50.03511 (Based on 5 acres)
V =	Fraction of vegetative cover (unitless).	0.5 (default value)
U_m =	Mean annual wind speed (m/sec).	3.89 (default value)
U_t =	Equivalent threshold value of wind speed at 7m (m/sec).	11.32 (default value)
F(x) =	Function dependent on U_m/U_t derived using Cowherd et al. (1985).	0.0391 (default value)

- Based on Portland, ME climate. Closest to Ely Copper Mine Site.

Table C-2
Calculation of the Particulate Emission Factor Based on Truck Traffic on Unpaved Roads

$$PEF\ (m^3/kg) = Q/C_{sr}\ x\ \frac{1}{F_D}\ x\ \left[\frac{T\ x\ A_R}{\frac{2.6\ x\ (s/12)^{0.8}\ x\ (W/3)^{0.4}}{(M_{dry}/0.2)^{0.3}}\ x\ \frac{(365\ d/yr - p)}{365 d/yr}\ x\ 281.9\ g/km\ x\ \sum VKT}\right]$$

Where:

$$Q/C_{sr}\ (g/m^2 - s\ per\ kg/m^3) = A\ x\ exp\left[\frac{(\ln A_s - B)^2}{C}\right]$$

$$F_D = 0.1852 + \frac{5.3537}{t_c} + \frac{-9.6318}{t_c^2}$$

$$A_R\ (m^2) = (L_R\ x\ W_R)$$

Where:		**Recreational Visitor**		**Construction Worker**	
		RME	**CTE**	**RME**	**CTE**
PEF =	Particulate emission factor from truck traffic on unpaved roads (m^3/kg).	Adol: 2.0E+06 Adult:2.0E+06	Adol: 1.9E+06 Adult:2.0E+06	5.37E+05	6.89E+05
Q/C_{sr} =	Inverse of 1-h average air concentration along a straight road segment bisecting a 350-acre square site (g/m^2-s per kg/m^3).	12.9		12.9	
F_D =	Dispersion correction factor (unitless).	Adol: 0.19 Adult:0.19	Adol: 0.20 Adult:0.19	0.20	0.23
T =	Total time over which exposure occurs (s).	Adol: 5.99E+06 Adult:1.80E+07	Adol: 1.01E+06 Adult:3.02E+06	1.73E+06	4.32E+05

Table C-2 continued Where:	Recreational Visitor		Construction Worker	
	RME	CTE	RME	CTE
t_c = Total time over which exposure occurs (hr)[a].	Adol: 1,664 Adult:4,992	Adol: 280 Adult:840	480	120
A_R = Surface area of contaminated road segment (m^2).	7,140		7,140	
s = Road surface silt content (%).	8.5 (default)		8.5 (default)	
W = Mean vehicle weight (tons).	0.4 (estimate for ATVs)		20 tons (estimate for dump trucks)	
M_{dry} = Road surface moisture content (%).	0.2 (default)		0.2 (default)	
p = Number of days with at least 0.01 inches of precipitation (days/yr).	150 (area-specific)		150 (area-specific)	
VKT = Sum of fleet vehicle kilometers traveled during the exposure duration (km)[b].	Adol: 9,984 Adult:29,952	Adol: 1,680 Adult:5,040	2,160	360
A = Constant (unitless). Default value (EPA, 2002a).	12.9351		12.9351	
A_s = Areal extent of site soil contamination (acres).	350 (size of site)		350 (size of site)	
B = Constant (unitless). Default value (EPA, 2002a).	5.7383		5.7383	
C = Constant (unitless). Default value (EPA, 2002a).	71.7711		71.7711	
L_R = Length of road segment (m).	1,190[c]		1,190[c]	
W_R = Width of road segment (m). Default value (EPA, 2002a).	6		6	

[a] Assumes the following:

Recreational visitor adolescent: RME 2 hours/day for 104 days/year for 8 years; CTE 1 hour/day for 35 days/year for 8 years.
Recreational visitor adult: RME 2 hours/day for 104 days/year for 24 years; CTE 1 hour/day for 35 days/year for 24 years.
Construction worker: RME 60 days/year for 8 hours/day for 1 year; CTE 30 days/year for 4 hours per day for 1 year.

[b] VKT assumes the following:

Recreational visitor adolescent: RME = 10 vehicles x 1.2 km/day (see L_r) x104 days/year x 8 years. CTE= 5 vehicles x 1.2 km/day x 35 days/year x 8 years.
Recreational visitor adult: RME = 10 vehicles x 1.2 km/day (see L_r) x104 days/year x 24 years. CTE= 5 vehicles x 1.2 km/day x 35 days/year x 24 years.
Construction worker: RME = 30 vehicles x 1.2 km/day (see L_r) x 60 days/year x 1 year. CTE= 10 vehicles x 1.2 km/day x 30 days/year x 1 years.

[c] Calculated based on the area of the site (approximately 350 acres). Assumes site is roughly configured as a square with the unpaved road transecting the area evenly. Value is equal to the square root of the area where 350 acres equals 1,416,400m2.

APPENDIX D

Appendix D
Surface Soil

Aluminum

General Statistics

Number of Valid Observations	270	Number of Distinct Observations	137

Raw Statistics		Log-transformed Statistics	
Minimum	860	Minimum of Log Data	6.757
Maximum	100000	Maximum of Log Data	11.51
Mean	10083	Mean of log Data	8.964
Median	8530	SD of log Data	0.751
SD	8317		
Coefficient of Variation	0.825		
Skewness	5.193		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Lilliefors Test Statistic	0.134	Lilliefors Test Statistic	0.0716
Lilliefors Critical Value	0.0539	Lilliefors Critical Value	0.0539
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	10918	95% H-UCL	11332
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	12678
95% Adjusted-CLT UCL (Chen-1995)	11086	97.5% Chebyshev (MVUE) UCL	13685
95% Modified-t UCL (Johnson-1978)	10945	99% Chebyshev (MVUE) UCL	15663

Gamma Distribution Test		Data Distribution	
k star (bias corrected)	2.093	Data appear Gamma Distributed at 5% Significance Level	
Theta Star	4816		
MLE of Mean	10083		
MLE of Standard Deviation	6969		
nu star	1130		
Approximate Chi Square Value (.05)	1053	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0491	95% CLT UCL	10915
Adjusted Chi Square Value	1053	95% Jackknife UCL	10918
		95% Standard Bootstrap UCL	10913
Anderson-Darling Test Statistic	0.586	95% Bootstrap-t UCL	11159
Anderson-Darling 5% Critical Value	0.765	95% Hall's Bootstrap UCL	11333
Kolmogorov-Smirnov Test Statistic	0.0374	95% Percentile Bootstrap UCL	10977
Kolmogorov-Smirnov 5% Critical Value	0.0562	95% BCA Bootstrap UCL	11090
Data appear Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	12289
		97.5% Chebyshev(Mean, Sd) UCL	13244
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	15119
95% Approximate Gamma UCL	10820		
95% Adjusted Gamma UCL	10824		

Potential UCL to Use		Use 95% Approximate Gamma UCL	10820

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Surface Soil

Antimony

General Statistics

Number of Valid Data	269	Number of Detected Data	106
Number of Distinct Detected Data	71	Number of Non-Detect Data	163
		Percent Non-Detects	60.59%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.048	Minimum Detected	-3.037
Maximum Detected	8.1	Maximum Detected	2.092
Mean of Detected	1.267	Mean of Detected	-0.43
SD of Detected	1.433	SD of Detected	1.291
Minimum Non-Detect	0.46	Minimum Non-Detect	-0.777
Maximum Non-Detect	43	Maximum Non-Detect	3.761

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	269
Number treated as Detected	0
Single DL Non-Detect Percentage	100.00%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Lilliefors Test Statistic	0.197	Lilliefors Test Statistic	0.121
5% Lilliefors Critical Value	0.0861	5% Lilliefors Critical Value	0.0861
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	3.892	Mean	0.682
SD	3.783	SD	1.409
95% DL/2 (t) UCL	4.273	95% H-Stat (DL/2) UCL	6.615
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	-0.574
		SD in Log Scale	1.022
		Mean in Original Scale	0.928
		SD in Original Scale	1.078
		95% t UCL	1.036
		95% Percentile Bootstrap UCL	1.034
		95% BCA Bootstrap UCL	1.046

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only
k star (bias corrected)	0.861	Data appear Gamma Distributed at 5% Significance Level
Theta Star	1.472	
nu star	182.6	
A-D Test Statistic	0.654	
5% A-D Critical Value	0.789	
K-S Test Statistic	0.789	
5% K-S Critical Value	0.0909	
Data appear Gamma Distributed at 5% Significance Level		

Assuming Gamma Distribution		Nonparametric Statistics	
Gamma ROS Statistics using Extrapolated Data		Kaplan-Meier (KM) Method	
Minimum	1E-12	Mean	1.097
Maximum	8.1	SD	1.27
Mean	1.347	SE of Mean	0.109
Median	1.2	95% KM (t) UCL	1.276
SD	1.076	95% KM (z) UCL	1.275
k star	1.151	95% KM (jackknife) UCL	1.276
Theta star	1.17	95% KM (bootstrap t) UCL	1.297
Nu star	619.3	95% KM (BCA) UCL	1.279
AppChi2	562.6	95% KM (Percentile Bootstrap) UCL	1.27
95% Gamma Approximate UCL	1.482	95% KM (Chebyshev) UCL	1.57
95% Adjusted Gamma UCL	1.483	97.5% KM (Chebyshev) UCL	1.775
		99% KM (Chebyshev) UCL	2.177
		Potential UCLs to Use	
		95% KM (t) UCL	1.276

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Soil

Arsenic

General Statistics

Number of Valid Data	270	Number of Detected Data	193
Number of Distinct Detected Data	70	Number of Non-Detect Data	77
		Percent Non-Detects	28.52%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.36	Minimum Detected	-1.022
Maximum Detected	23	Maximum Detected	3.135
Mean of Detected	2.883	Mean of Detected	0.758
SD of Detected	3.119	SD of Detected	0.716
Minimum Non-Detect	0.41	Minimum Non-Detect	-0.892
Maximum Non-Detect	8.6	Maximum Non-Detect	2.152

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	260
Number treated as Detected	10
Single DL Non-Detect Percentage	96.30%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Lilliefors Test Statistic	0.256	Lilliefors Test Statistic	0.0912
5% Lilliefors Critical Value	0.0638	5% Lilliefors Critical Value	0.0638
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	2.446	Mean	0.567
SD	2.764	SD	0.767
95% DL/2 (t) UCL	2.724	95% H-Stat (DL/2) UCL	2.592
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE yields a negative mean		Mean in Log Scale	0.557
		SD in Log Scale	0.731
		Mean in Original Scale	2.396
		SD in Original Scale	2.761
		95% t UCL	2.674
		95% Percentile Bootstrap UCL	2.681
		95% BCA Bootstrap UCL	2.746

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only
k star (bias corrected)	1.784	Data do not follow a Discernable Distribution (0.05)
Theta Star	1.616	
nu star	688.7	
A-D Test Statistic	6.344	
5% A-D Critical Value	0.768	
K-S Test Statistic	0.768	
5% K-S Critical Value	0.0665	
Data not Gamma Distributed at 5% Significance Level		

Assuming Gamma Distribution		Nonparametric Statistics	
Gamma ROS Statistics using Extrapolated Data		Kaplan-Meier (KM) Method	
Minimum	1E-12	Mean	2.416
Maximum	23	SD	2.765
Mean	2.729	SE of Mean	0.17
Median	2.2	95% KM (t) UCL	2.697
SD	2.725	95% KM (z) UCL	2.696
k star	1.421	95% KM (jackknife) UCL	2.696
Theta star	1.921	95% KM (bootstrap t) UCL	2.779
Nu star	767.1	95% KM (BCA) UCL	2.725
AppChi2	703.8	95% KM (Percentile Bootstrap) UCL	2.705
95% Gamma Approximate UCL	2.974	95% KM (Chebyshev) UCL	3.159
95% Adjusted Gamma UCL	2.975	97.5% KM (Chebyshev) UCL	3.48
		99% KM (Chebyshev) UCL	4.111
		Potential UCLs to Use	
		95% KM (BCA) UCL	2.725

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Soil

Cadmium

General Statistics

Number of Valid Data	269	Number of Detected Data	210
Number of Distinct Detected Data	99	Number of Non-Detect Data	59
		Percent Non-Detects	21.93%

Raw Statistics

Minimum Detected	0.017
Maximum Detected	8.5
Mean of Detected	0.95
SD of Detected	1.086
Minimum Non-Detect	0.2
Maximum Non-Detect	10

Log-transformed Statistics

Minimum Detected	-4.075
Maximum Detected	2.14
Mean of Detected	-0.483
SD of Detected	0.918
Minimum Non-Detect	-1.609
Maximum Non-Detect	2.303

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	269
Number treated as Detected	0
Single DL Non-Detect Percentage	100.00%

UCL Statistics

Normal Distribution Test with Detected Values Only

Lilliefors Test Statistic	0.229
5% Lilliefors Critical Value	0.0611

Data not Normal at 5% Significance Level

Lognormal Distribution Test with Detected Values Only

Lilliefors Test Statistic	0.0534
5% Lilliefors Critical Value	0.0611

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

DL/2 Substitution Method

Mean	1.041
SD	1.078
95% DL/2 (t) UCL	1.149

Maximum Likelihood Estimate(MLE) Method N/A

MLE method failed to converge properly

Assuming Lognormal Distribution

DL/2 Substitution Method

Mean	-0.387
SD	0.947
95% H-Stat (DL/2) UCL	1.2

Log ROS Method

Mean in Log Scale	-0.561
SD in Log Scale	0.857
Mean in Original Scale	0.848
SD in Original Scale	0.984
95% t UCL	0.947
95% Percentile Bootstrap UCL	0.954
95% BCA Bootstrap UCL	0.958

Gamma Distribution Test with Detected Values Only

k star (bias corrected)	1.284
Theta Star	0.74
nu star	539.5
A-D Test Statistic	4.455
5% A-D Critical Value	0.776
K-S Test Statistic	0.776
5% K-S Critical Value	0.0639

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data

Minimum	1E-12
Maximum	8.5
Mean	0.93
Median	0.66
SD	0.983
k star	0.777
Theta star	1.197
Nu star	417.9
AppChi2	371.5
95% Gamma Approximate UCL	1.046
95% Adjusted Gamma UCL	1.047

Note: DL/2 is not a recommended method.

Data Distribution Test with Detected Values Only

Data appear Lognormal at 5% Significance Level

Nonparametric Statistics

Kaplan-Meier (KM) Method

Mean	0.873
SD	1.007
SE of Mean	0.0638
95% KM (t) UCL	0.978
95% KM (z) UCL	0.978
95% KM (jackknife) UCL	0.978
95% KM (bootstrap t) UCL	1.003
95% KM (BCA) UCL	0.987
95% KM (Percentile Bootstrap) UCL	0.982
95% KM (Chebyshev) UCL	1.151
97.5% KM (Chebyshev) UCL	1.271
99% KM (Chebyshev) UCL	1.508

Potential UCLs to Use

95% KM (BCA) UCL	0.987

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Soil

Chromium

General Statistics

Number of Valid Observations	270	Number of Distinct Observations	130

Raw Statistics		Log-transformed Statistics	
Minimum	2.5	Minimum of Log Data	0.916
Maximum	440	Maximum of Log Data	6.087
Mean	35.26	Mean of log Data	3.327
Median	32	SD of log Data	0.721
SD	31.85		
Coefficient of Variation	0.903		
Skewness	7.969		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Lilliefors Test Statistic	0.158	Lilliefors Test Statistic	0.0992
Lilliefors Critical Value	0.0539	Lilliefors Critical Value	0.0539
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	38.46	95% H-UCL	39.31
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	43.78
95% Adjusted-CLT UCL (Chen-1995)	39.45	97.5% Chebyshev (MVUE) UCL	47.12
95% Modified-t UCL (Johnson-1978)	38.62	99% Chebyshev (MVUE) UCL	53.68

Gamma Distribution Test		Data Distribution
k star (bias corrected)	2.25	Data do not follow a Discernable Distribution (0.05)
Theta Star	15.67	
MLE of Mean	35.26	
MLE of Standard Deviation	23.5	
nu star	1215	
Approximate Chi Square Value (.05)	1135	
Adjusted Level of Significance	0.0491	
Adjusted Chi Square Value	1135	
Anderson-Darling Test Statistic	1.672	
Anderson-Darling 5% Critical Value	0.764	
Kolmogorov-Smirnov Test Statistic	0.0653	
Kolmogorov-Smirnov 5% Critical Value	0.0562	
Data not Gamma Distributed at 5% Significance Level		

Assuming Gamma Distribution	
95% Approximate Gamma UCL	37.74
95% Adjusted Gamma UCL	37.76

Nonparametric Statistics	
95% CLT UCL	38.45
95% Jackknife UCL	38.46
95% Standard Bootstrap UCL	38.39
95% Bootstrap-t UCL	40.02
95% Hall's Bootstrap UCL	53.92
95% Percentile Bootstrap UCL	38.5
95% BCA Bootstrap UCL	39.74
95% Chebyshev(Mean, Sd) UCL	43.71
97.5% Chebyshev(Mean, Sd) UCL	47.37
99% Chebyshev(Mean, Sd) UCL	54.55

Potential UCL to Use	
Use 95% Chebyshev (Mean, Sd) UCL	43.71

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Surface Soil

Cobalt

General Statistics

Number of Valid Observations	270	Number of Distinct Observations	128

Raw Statistics		Log-transformed Statistics	
Minimum	1.8	Minimum of Log Data	0.588
Maximum	200	Maximum of Log Data	5.298
Mean	18.2	Mean of log Data	2.521
Median	11	SD of log Data	0.773
SD	24.09		
Coefficient of Variation	1.324		
Skewness	4.397		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Lilliefors Test Statistic	0.279	Lilliefors Test Statistic	0.113
Lilliefors Critical Value	0.0539	Lilliefors Critical Value	0.0539
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	20.61	95% H-UCL	18.41
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	20.66
95% Adjusted-CLT UCL (Chen-1995)	21.03	97.5% Chebyshev (MVUE) UCL	22.35
95% Modified-t UCL (Johnson-1978)	20.68	99% Chebyshev (MVUE) UCL	25.66

Gamma Distribution Test		Data Distribution	
k star (bias corrected)	1.446	Data do not follow a Discernable Distribution (0.05)	
Theta Star	12.58		
MLE of Mean	18.2		
MLE of Standard Deviation	15.13		
nu star	781		
Approximate Chi Square Value (.05)	717.1	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0491	95% CLT UCL	20.61
Adjusted Chi Square Value	716.8	95% Jackknife UCL	20.61
		95% Standard Bootstrap UCL	20.53
Anderson-Darling Test Statistic	13.26	95% Bootstrap-t UCL	21.22
Anderson-Darling 5% Critical Value	0.773	95% Hall's Bootstrap UCL	21.33
Kolmogorov-Smirnov Test Statistic	0.182	95% Percentile Bootstrap UCL	20.92
Kolmogorov-Smirnov 5% Critical Value	0.0567	95% BCA Bootstrap UCL	20.88
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	24.58
		97.5% Chebyshev(Mean, Sd) UCL	27.35
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	32.78
95% Approximate Gamma UCL	19.82		
95% Adjusted Gamma UCL	19.82		

Potential UCL to Use			
		Use 95% Chebyshev (Mean, Sd) UCL	24.58

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Surface Soil

Copper

General Statistics

Number of Valid Observations	270	Number of Distinct Observations	169

Raw Statistics		**Log-transformed Statistics**	
Minimum	7.5	Minimum of Log Data	2.015
Maximum	23000	Maximum of Log Data	10.04
Mean	963.5	Mean of log Data	6.083
Median	550	SD of log Data	1.396
SD	1695		
Coefficient of Variation	1.759		
Skewness	8.762		

Relevant UCL Statistics

Normal Distribution Test		**Lognormal Distribution Test**	
Lilliefors Test Statistic	0.286	Lilliefors Test Statistic	0.103
Lilliefors Critical Value	0.0539	Lilliefors Critical Value	0.0539
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		**Assuming Lognormal Distribution**	
95% Student's-t UCL	1134	95% H-UCL	1436
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	1744
95% Adjusted-CLT UCL (Chen-1995)	1192	97.5% Chebyshev (MVUE) UCL	2000
95% Modified-t UCL (Johnson-1978)	1143	99% Chebyshev (MVUE) UCL	2501

Gamma Distribution Test		**Data Distribution**
k star (bias corrected)	0.753	Data Follow Appr. Gamma Distribution at 5% Significance Level
Theta Star	1279	
MLE of Mean	963.5	
MLE of Standard Deviation	1110	
nu star	406.7	
Approximate Chi Square Value (.05)	360.9	**Nonparametric Statistics**
Adjusted Level of Significance	0.0491	95% CLT UCL 1133
Adjusted Chi Square Value	360.7	95% Jackknife UCL 1134
		95% Standard Bootstrap UCL 1131
Anderson-Darling Test Statistic	1.133	95% Bootstrap-t UCL 1243
Anderson-Darling 5% Critical Value	0.796	95% Hall's Bootstrap UCL 1912
Kolmogorov-Smirnov Test Statistic	0.05	95% Percentile Bootstrap UCL 1141
Kolmogorov-Smirnov 5% Critical Value	0.0578	95% BCA Bootstrap UCL 1215
Data follow Appr. Gamma Distribution at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL 1413
		97.5% Chebyshev(Mean, Sd) UCL 1608
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL 1990
95% Approximate Gamma UCL	1086	
95% Adjusted Gamma UCL	1086	

Potential UCL to Use	Use 95% Approximate Gamma UCL 1086

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Surface Soil

Iron

General Statistics

Number of Valid Observations	270	Number of Distinct Observations	138

Raw Statistics		Log-transformed Statistics	
Minimum	2400	Minimum of Log Data	7.783
Maximum	300000	Maximum of Log Data	12.61
Mean	53810	Mean of log Data	10.48
Median	31000	SD of log Data	0.956
SD	49398		
Coefficient of Variation	0.918		
Skewness	1.489		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Lilliefors Test Statistic	0.189	Lilliefors Test Statistic	0.0629
Lilliefors Critical Value	0.0539	Lilliefors Critical Value	0.0539
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	58772	95% H-UCL	63342
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	72912
95% Adjusted-CLT UCL (Chen-1995)	59046	97.5% Chebyshev (MVUE) UCL	80265
95% Modified-t UCL (Johnson-1978)	58818	99% Chebyshev (MVUE) UCL	94708

Gamma Distribution Test		Data Distribution	
k star (bias corrected)	1.331	Data do not follow a Discernable Distribution (0.05)	
Theta Star	40414		
MLE of Mean	53810		
MLE of Standard Deviation	46633		
nu star	719		
Approximate Chi Square Value (.05)	657.8	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0491	95% CLT UCL	58755
Adjusted Chi Square Value	657.5	95% Jackknife UCL	58772
		95% Standard Bootstrap UCL	58827
Anderson-Darling Test Statistic	3.756	95% Bootstrap-t UCL	59030
Anderson-Darling 5% Critical Value	0.776	95% Hall's Bootstrap UCL	58965
Kolmogorov-Smirnov Test Statistic	0.116	95% Percentile Bootstrap UCL	58729
Kolmogorov-Smirnov 5% Critical Value	0.0568	95% BCA Bootstrap UCL	59056
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	66914
		97.5% Chebyshev(Mean, Sd) UCL	72584
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	83722
95% Approximate Gamma UCL	58818		
95% Adjusted Gamma UCL	58846		

Potential UCL to Use		Use 95% Chebyshev (Mean, Sd) UCL	66914

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Surface Soil

Manganese

General Statistics

Number of Valid Observations	270	Number of Distinct Observations	138

Raw Statistics

Minimum	17
Maximum	8380
Mean	296.4
Median	165.5
SD	574.6
Coefficient of Variation	1.938
Skewness	10.78

Log-transformed Statistics

Minimum of Log Data	2.833
Maximum of Log Data	9.034
Mean of log Data	5.19
SD of log Data	0.915

Relevant UCL Statistics

Normal Distribution Test

Lilliefors Test Statistic	0.313
Lilliefors Critical Value	0.0539

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Lilliefors Test Statistic	0.0554
Lilliefors Critical Value	0.0539

Data not Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	354.2

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995)	378.5
95% Modified-t UCL (Johnson-1978)	358

Assuming Lognormal Distribution

95% H-UCL	306.1
95% Chebyshev (MVUE) UCL	350.3
97.5% Chebyshev (MVUE) UCL	384.1
99% Chebyshev (MVUE) UCL	450.6

Gamma Distribution Test

k star (bias corrected)	1.125
Theta Star	263.6
MLE of Mean	296.4
MLE of Standard Deviation	279.5
nu star	607.3
Approximate Chi Square Value (.05)	551.1
Adjusted Level of Significance	0.0491
Adjusted Chi Square Value	550.8
Anderson-Darling Test Statistic	7.283
Anderson-Darling 5% Critical Value	0.781
Kolmogorov-Smirnov Test Statistic	0.113
Kolmogorov-Smirnov 5% Critical Value	0.0571

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	326.7
95% Adjusted Gamma UCL	326.8

Data Distribution

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

95% CLT UCL	354
95% Jackknife UCL	354.2
95% Standard Bootstrap UCL	353.4
95% Bootstrap-t UCL	410.9
95% Hall's Bootstrap UCL	603.1
95% Percentile Bootstrap UCL	359.6
95% BCA Bootstrap UCL	389.3
95% Chebyshev(Mean, Sd) UCL	448.9
97.5% Chebyshev(Mean, Sd) UCL	514.8
99% Chebyshev(Mean, Sd) UCL	644.4

Potential UCL to Use

Use 95% Chebyshev (Mean, Sd) UCL 448.9

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Surface Soil

Mercury

General Statistics

Number of Valid Data	269	Number of Detected Data	252
Number of Distinct Detected Data	112	Number of Non-Detect Data	17
		Percent Non-Detects	6.32%

Raw Statistics

Minimum Detected	0.006
Maximum Detected	2.8
Mean of Detected	0.13
SD of Detected	0.207
Minimum Non-Detect	0.014
Maximum Non-Detect	0.1

Log-transformed Statistics

Minimum Detected	-5.116
Maximum Detected	1.03
Mean of Detected	-2.511
SD of Detected	0.94
Minimum Non-Detect	-4.269
Maximum Non-Detect	-2.303

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	163
Number treated as Detected	106
Single DL Non-Detect Percentage	60.59%

UCL Statistics

Normal Distribution Test with Detected Values Only

Lilliefors Test Statistic	0.274
5% Lilliefors Critical Value	0.0558

Data not Normal at 5% Significance Level

Lognormal Distribution Test with Detected Values Only

Lilliefors Test Statistic	0.0276
5% Lilliefors Critical Value	0.0558

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

DL/2 Substitution Method

Mean	0.124
SD	0.202
95% DL/2 (t) UCL	0.144

Maximum Likelihood Estimate(MLE) Method N/A

MLE yields a negative mean

Assuming Lognormal Distribution

DL/2 Substitution Method

Mean	-2.577
SD	0.958
95% H-Stat (DL/2) UCL	0.136

Log ROS Method

Mean in Log Scale	-2.572
SD in Log Scale	0.948
Mean in Original Scale	0.124
SD in Original Scale	0.202
95% t UCL	0.144
95% Percentile Bootstrap UCL	0.146
95% BCA Bootstrap UCL	0.153

Gamma Distribution Test with Detected Values Only

k star (bias corrected)	1.191
Theta Star	0.109
nu star	600.2
A-D Test Statistic	3.462
5% A-D Critical Value	0.779
K-S Test Statistic	0.779
5% K-S Critical Value	0.0591

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data

Minimum	1E-12
Maximum	2.8
Mean	0.124
Median	0.074
SD	0.202
k star	0.566
Theta star	0.219
Nu star	304.5
AppChi2	265.1
95% Gamma Approximate UCL	0.143
95% Adjusted Gamma UCL	0.143

Note: DL/2 is not a recommended method.

Data Distribution Test with Detected Values Only

Data appear Lognormal at 5% Significance Level

Nonparametric Statistics

Kaplan-Meier (KM) Method

Mean	0.124
SD	0.201
SE of Mean	0.0123
95% KM (t) UCL	0.144
95% KM (z) UCL	0.144
95% KM (jackknife) UCL	0.144
95% KM (bootstrap t) UCL	0.159
95% KM (BCA) UCL	0.148
95% KM (Percentile Bootstrap) UCL	0.145
95% KM (Chebyshev) UCL	0.178
97.5% KM (Chebyshev) UCL	0.201
99% KM (Chebyshev) UCL	0.246

Potential UCLs to Use

95% KM (BCA) UCL	0.148

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Soil

Molybdenum

General Statistics

Number of Valid Data	269	Number of Detected Data	216
Number of Distinct Detected Data	118	Number of Non-Detect Data	53
		Percent Non-Detects	19.70%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.2	Minimum Detected	-1.609
Maximum Detected	69	Maximum Detected	4.234
Mean of Detected	5.611	Mean of Detected	0.947
SD of Detected	8.101	SD of Detected	1.271
Minimum Non-Detect	0.22	Minimum Non-Detect	-1.514
Maximum Non-Detect	43	Maximum Non-Detect	3.761

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	268
Number treated as Detected	1
Single DL Non-Detect Percentage	99.63%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Lilliefors Test Statistic	0.252	Lilliefors Test Statistic	0.0728
5% Lilliefors Critical Value	0.0603	5% Lilliefors Critical Value	0.0603
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	5.256	Mean	0.894
SD	7.479	SD	1.293
95% DL/2 (t) UCL	6.008	95% H-Stat (DL/2) UCL	6.81
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	0.747
		SD in Log Scale	1.277
		Mean in Original Scale	4.772
		SD in Original Scale	7.47
		95% t UCL	5.523
		95% Percentile Bootstrap UCL	5.595
		95% BCA Bootstrap UCL	5.662

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	0.76	Data do not follow a Discernable Distribution (0.05)	
Theta Star	7.388		
nu star	328.1		
A-D Test Statistic	5.076	Nonparametric Statistics	
5% A-D Critical Value	0.795	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.795	Mean	4.851
5% K-S Critical Value	0.0642	SD	7.486
Data not Gamma Distributed at 5% Significance Level		SE of Mean	0.462
		95% KM (t) UCL	5.613
Assuming Gamma Distribution		95% KM (z) UCL	5.611
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	5.611
Minimum	1E-12	95% KM (bootstrap t) UCL	5.735
Maximum	69	95% KM (BCA) UCL	5.624
Mean	5.174	95% KM (Percentile Bootstrap) UCL	5.639
Median	2.4	95% KM (Chebyshev) UCL	6.865
SD	7.468	97.5% KM (Chebyshev) UCL	7.736
k star	0.316	99% KM (Chebyshev) UCL	9.447
Theta star	16.35		
Nu star	170.2	Potential UCLs to Use	
AppChi2	141.1	95% KM (Chebyshev) UCL	6.865
95% Gamma Approximate UCL	6.244		
95% Adjusted Gamma UCL	6.25		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Soil

Selenium

General Statistics

Number of Valid Data	269	Number of Detected Data	253
Number of Distinct Detected Data	142	Number of Non-Detect Data	16
		Percent Non-Detects	5.95%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.42	Minimum Detected	-0.868
Maximum Detected	170	Maximum Detected	5.136
Mean of Detected	16.41	Mean of Detected	1.97
SD of Detected	21.28	SD of Detected	1.384
Minimum Non-Detect	0.82	Minimum Non-Detect	-0.198
Maximum Non-Detect	3.6	Maximum Non-Detect	1.281

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	108
Number treated as Detected	161
Single DL Non-Detect Percentage	40.15%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Lilliefors Test Statistic	0.226	Lilliefors Test Statistic	0.0814
5% Lilliefors Critical Value	0.0557	5% Lilliefors Critical Value	0.0557
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	15.49	Mean	1.848
SD	20.96	SD	1.43
95% DL/2 (t) UCL	17.6	95% H-Stat (DL/2) UCL	21.99
Maximum Likelihood Estimate(MLE) Method		Log ROS Method	
Mean	7.193	Mean in Log Scale	1.855
SD	29.81	SD in Log Scale	1.421
95% MLE (t) UCL	10.19	Mean in Original Scale	15.5
95% MLE (Tiku) UCL	10.57	SD in Original Scale	20.95
		95% t UCL	17.61
		95% Percentile Bootstrap UCL	17.58
		95% BCA Bootstrap UCL	17.79

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	0.72	Data do not follow a Discernable Distribution (0.05)	
Theta Star	22.78		
nu star	364.5		
A-D Test Statistic	4.446	Nonparametric Statistics	
5% A-D Critical Value	0.799	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.799	Mean	15.5
5% K-S Critical Value	0.0599	SD	20.91
Data not Gamma Distributed at 5% Significance Level		SE of Mean	1.278
		95% KM (t) UCL	17.61
Assuming Gamma Distribution		95% KM (z) UCL	17.6
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	17.61
Minimum	1E-12	95% KM (bootstrap t) UCL	17.83
Maximum	170	95% KM (BCA) UCL	17.62
Mean	15.43	95% KM (Percentile Bootstrap) UCL	17.61
Median	5.9	95% KM (Chebyshev) UCL	21.07
SD	21	97.5% KM (Chebyshev) UCL	23.48
k star	0.276	99% KM (Chebyshev) UCL	28.21
Theta star	55.94		
Nu star	148.4	Potential UCLs to Use	
AppChi2	121.3	95% KM (Chebyshev) UCL	21.07
95% Gamma Approximate UCL	18.89		
95% Adjusted Gamma UCL	18.91		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Soil

Vanadium

General Statistics

Number of Valid Observations	270	Number of Distinct Observations	134

Raw Statistics		Log-transformed Statistics	
Minimum	5.2	Minimum of Log Data	1.649
Maximum	400	Maximum of Log Data	5.991
Mean	47.34	Mean of log Data	3.667
Median	43	SD of log Data	0.638
SD	33.97		
Coefficient of Variation	0.717		
Skewness	4.726		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Lilliefors Test Statistic	0.147	Lilliefors Test Statistic	0.0892
Lilliefors Critical Value	0.0539	Lilliefors Critical Value	0.0539
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	50.75	95% H-UCL	51.58
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	56.78
95% Adjusted-CLT UCL (Chen-1995)	51.38	97.5% Chebyshev (MVUE) UCL	60.62
95% Modified-t UCL (Johnson-1978)	50.85	99% Chebyshev (MVUE) UCL	68.15

Gamma Distribution Test		Data Distribution	
k star (bias corrected)	2.75	Data do not follow a Discernable Distribution (0.05)	
Theta Star	17.21		
MLE of Mean	47.34		
MLE of Standard Deviation	28.55		
nu star	1485		
Approximate Chi Square Value (.05)	1397	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0491	95% CLT UCL	50.74
Adjusted Chi Square Value	1396	95% Jackknife UCL	50.75
		95% Standard Bootstrap UCL	50.76
Anderson-Darling Test Statistic	1.152	95% Bootstrap-t UCL	51.63
Anderson-Darling 5% Critical Value	0.761	95% Hall's Bootstrap UCL	52.37
Kolmogorov-Smirnov Test Statistic	0.064	95% Percentile Bootstrap UCL	50.77
Kolmogorov-Smirnov 5% Critical Value	0.056	95% BCA Bootstrap UCL	51.56
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	56.35
		97.5% Chebyshev(Mean, Sd) UCL	60.25
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	67.91
95% Approximate Gamma UCL	50.34		
95% Adjusted Gamma UCL	50.36		

Potential UCL to Use			
		Use 95% Chebyshev (Mean, Sd) UCL	56.35

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Total Soil

Aluminum

General Statistics

Number of Valid Observations	348	Number of Distinct Observations	178

Raw Statistics

Minimum	860
Maximum	100000
Mean	11114
Median	9450
SD	8343
Coefficient of Variation	0.751
Skewness	3.988

Log-transformed Statistics

Minimum of Log Data	6.757
Maximum of Log Data	11.51
Mean of log Data	9.069
SD of log Data	0.751

Relevant UCL Statistics

Normal Distribution Test

Lilliefors Test Statistic	0.11
Lilliefors Critical Value	0.0475

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Lilliefors Test Statistic	0.0725
Lilliefors Critical Value	0.0475

Data not Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	11852

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995)	11952
95% Modified-t UCL (Johnson-1978)	11868

Assuming Lognormal Distribution

95% H-UCL	12443
95% Chebyshev (MVUE) UCL	13771
97.5% Chebyshev (MVUE) UCL	14756
99% Chebyshev (MVUE) UCL	16691

Gamma Distribution Test

k star (bias corrected)	2.158
Theta Star	5151
MLE of Mean	11114
MLE of Standard Deviation	7566
nu star	1502
Approximate Chi Square Value (.05)	1413
Adjusted Level of Significance	0.0493
Adjusted Chi Square Value	1412
Anderson-Darling Test Statistic	0.549
Anderson-Darling 5% Critical Value	0.765
Kolmogorov-Smirnov Test Statistic	0.0459
Kolmogorov-Smirnov 5% Critical Value	0.0493

Data appear Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	11814
95% Adjusted Gamma UCL	11817

Data Distribution

Data appear Gamma Distributed at 5% Significance Level

Nonparametric Statistics

95% CLT UCL	11850
95% Jackknife UCL	11852
95% Standard Bootstrap UCL	11845
95% Bootstrap-t UCL	11994
95% Hall's Bootstrap UCL	12050
95% Percentile Bootstrap UCL	11848
95% BCA Bootstrap UCL	11937
95% Chebyshev(Mean, Sd) UCL	13064
97.5% Chebyshev(Mean, Sd) UCL	13907
99% Chebyshev(Mean, Sd) UCL	15564

Potential UCL to Use

Use 95% Approximate Gamma UCL 11814

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Total Soil

Antimony

General Statistics

Number of Valid Data	348	Number of Detected Data	124
Number of Distinct Detected Data	78	Number of Non-Detect Data	224
		Percent Non-Detects	64.37%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.048	Minimum Detected	-3.037
Maximum Detected	12	Maximum Detected	2.485
Mean of Detected	1.33	Mean of Detected	-0.487
SD of Detected	1.694	SD of Detected	1.393
Minimum Non-Detect	0.46	Minimum Non-Detect	-0.777
Maximum Non-Detect	43	Maximum Non-Detect	3.761

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

		Number treated as Non-Detect	348
		Number treated as Detected	0
		Single DL Non-Detect Percentage	100.00%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Lilliefors Test Statistic	0.225	Lilliefors Test Statistic	0.129
5% Lilliefors Critical Value	0.0796	5% Lilliefors Critical Value	0.0796
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	3.443	Mean	0.372
SD	3.878	SD	1.536
95% DL/2 (t) UCL	3.786	95% H-Stat (DL/2) UCL	5.849
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	-0.93
		SD in Log Scale	1.15
		Mean in Original Scale	0.771
		SD in Original Scale	1.163
		95% t UCL	0.874
		95% Percentile Bootstrap UCL	0.879
		95% BCA Bootstrap UCL	0.892

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only
k star (bias corrected)	0.759	Data Follow Appr. Gamma Distribution at 5% Significance Level
Theta Star	1.752	
nu star	188.2	
A-D Test Statistic	1.164	
5% A-D Critical Value	0.794	
K-S Test Statistic	0.794	
5% K-S Critical Value	0.0865	
Data follow Appr. Gamma Distribution at 5% Significance Level		

Assuming Gamma Distribution		Nonparametric Statistics	
Gamma ROS Statistics using Extrapolated Data		Kaplan-Meier (KM) Method	
Minimum	1E-12	Mean	0.903
Maximum	12	SD	1.343
Mean	1.634	SE of Mean	0.0913
Median	1.394	95% KM (t) UCL	1.054
SD	1.454	95% KM (z) UCL	1.053
k star	0.416	95% KM (jackknife) UCL	1.054
Theta star	3.932	95% KM (bootstrap t) UCL	1.074
Nu star	289.2	95% KM (BCA) UCL	1.053
AppChi2	250.8	95% KM (Percentile Bootstrap) UCL	1.057
95% Gamma Approximate UCL	1.884	95% KM (Chebyshev) UCL	1.301
95% Adjusted Gamma UCL	1.885	97.5% KM (Chebyshev) UCL	1.473
		99% KM (Chebyshev) UCL	1.812
		Potential UCLs to Use	
		95% KM (t) UCL	1.054

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Total Soil

Arsenic

General Statistics

Number of Valid Data	348	Number of Detected Data	225
Number of Distinct Detected Data	83	Number of Non-Detect Data	123
		Percent Non-Detects	35.34%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.18	Minimum Detected	-1.715
Maximum Detected	23	Maximum Detected	3.135
Mean of Detected	2.659	Mean of Detected	0.642
SD of Detected	2.977	SD of Detected	0.778
Minimum Non-Detect	0.41	Minimum Non-Detect	-0.892
Maximum Non-Detect	8.6	Maximum Non-Detect	2.152

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

		Number treated as Non-Detect	338
		Number treated as Detected	10
		Single DL Non-Detect Percentage	97.13%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Lilliefors Test Statistic	0.243	Lilliefors Test Statistic	0.0726
5% Lilliefors Critical Value	0.0591	5% Lilliefors Critical Value	0.0591
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	2.142	Mean	0.391
SD	2.545	SD	0.825
95% DL/2 (t) UCL	2.367	95% H-Stat (DL/2) UCL	2.269
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE yields a negative mean		Mean in Log Scale	0.35
		SD in Log Scale	0.799
		Mean in Original Scale	2.052
		SD in Original Scale	2.545
		95% t UCL	2.277
		95% Percentile Bootstrap UCL	2.287
		95% BCA Bootstrap UCL	2.311

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	1.618	Data do not follow a Discernable Distribution (0.05)	
Theta Star	1.643		
nu star	728.1		
A-D Test Statistic	5.191	Nonparametric Statistics	
5% A-D Critical Value	0.77	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.77	Mean	2.075
5% K-S Critical Value	0.0618	SD	2.553
Data not Gamma Distributed at 5% Significance Level		SE of Mean	0.139
		95% KM (t) UCL	2.305
Assuming Gamma Distribution		95% KM (z) UCL	2.304
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	2.305
Minimum	1E-12	95% KM (bootstrap t) UCL	2.344
Maximum	23	95% KM (BCA) UCL	2.323
Mean	2.486	95% KM (Percentile Bootstrap) UCL	2.31
Median	2.024	95% KM (Chebyshev) UCL	2.681
SD	2.526	97.5% KM (Chebyshev) UCL	2.943
k star	0.712	99% KM (Chebyshev) UCL	3.458
Theta star	3.494		
Nu star	495.3	Potential UCLs to Use	
AppChi2	444.7	95% KM (BCA) UCL	2.323
95% Gamma Approximate UCL	2.769		
95% Adjusted Gamma UCL	2.77		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Total Soil

Cadmium

General Statistics

Number of Valid Data	348	Number of Detected Data	271
Number of Distinct Detected Data	118	Number of Non-Detect Data	77
		Percent Non-Detects	22.13%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.013	Minimum Detected	-4.343
Maximum Detected	45	Maximum Detected	3.807
Mean of Detected	1.251	Mean of Detected	-0.611
SD of Detected	3.687	SD of Detected	1.161
Minimum Non-Detect	0.2	Minimum Non-Detect	-1.609
Maximum Non-Detect	10	Maximum Non-Detect	2.303

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

		Number treated as Non-Detect	345
		Number treated as Detected	3
		Single DL Non-Detect Percentage	99.14%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Lilliefors Test Statistic	0.368	Lilliefors Test Statistic	0.0601
5% Lilliefors Critical Value	0.0538	5% Lilliefors Critical Value	0.0538
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	1.275	Mean	-0.507
SD	3.287	SD	1.165
95% DL/2 (t) UCL	1.566	95% H-Stat (DL/2) UCL	1.368
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE yields a negative mean		Mean in Log Scale	-0.723
		SD in Log Scale	1.091
		Mean in Original Scale	1.064
		SD in Original Scale	3.273
		95% t UCL	1.353
		95% Percentile Bootstrap UCL	1.38
		95% BCA Bootstrap UCL	1.466

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	0.715	Data do not follow a Discernable Distribution (0.05)	
Theta Star	1.751		
nu star	387.4		
A-D Test Statistic	12.29	Nonparametric Statistics	
5% A-D Critical Value	0.8	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.8	Mean	1.09
5% K-S Critical Value	0.0578	SD	3.274
Data not Gamma Distributed at 5% Significance Level		SE of Mean	0.176
		95% KM (t) UCL	1.381
Assuming Gamma Distribution		95% KM (z) UCL	1.381
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	1.381
Minimum	1E-12	95% KM (bootstrap t) UCL	1.687
Maximum	45	95% KM (BCA) UCL	1.434
Mean	1.18	95% KM (Percentile Bootstrap) UCL	1.391
Median	0.535	95% KM (Chebyshev) UCL	1.859
SD	3.283	97.5% KM (Chebyshev) UCL	2.192
k star	0.314	99% KM (Chebyshev) UCL	2.846
Theta star	3.764		
Nu star	218.3	Potential UCLs to Use	
AppChi2	185.1	95% KM (Chebyshev) UCL	1.859
95% Gamma Approximate UCL	1.392		
95% Adjusted Gamma UCL	1.393		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Total Soil

Chromium

General Statistics

Number of Valid Observations	348	Number of Distinct Observations	179

Raw Statistics		Log-transformed Statistics	
Minimum	2.5	Minimum of Log Data	0.916
Maximum	440	Maximum of Log Data	6.087
Mean	40.17	Mean of log Data	3.459
Median	35	SD of log Data	0.719
SD	34.1		
Coefficient of Variation	0.849		
Skewness	5.932		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Lilliefors Test Statistic	0.154	Lilliefors Test Statistic	0.0988
Lilliefors Critical Value	0.0475	Lilliefors Critical Value	0.0475
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	43.18	95% H-UCL	44.31
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	48.84
95% Adjusted-CLT UCL (Chen-1995)	43.8	97.5% Chebyshev (MVUE) UCL	52.18
95% Modified-t UCL (Johnson-1978)	43.28	99% Chebyshev (MVUE) UCL	58.75

Gamma Distribution Test		Data Distribution	
k star (bias corrected)	2.267	Data do not follow a Discernable Distribution (0.05)	
Theta Star	17.72		
MLE of Mean	40.17		
MLE of Standard Deviation	26.68		
nu star	1578		
Approximate Chi Square Value (.05)	1487	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0493	95% CLT UCL	43.18
Adjusted Chi Square Value	1486	95% Jackknife UCL	43.18
		95% Standard Bootstrap UCL	43.1
Anderson-Darling Test Statistic	2.672	95% Bootstrap-t UCL	43.97
Anderson-Darling 5% Critical Value	0.764	95% Hall's Bootstrap UCL	44.49
Kolmogorov-Smirnov Test Statistic	0.0681	95% Percentile Bootstrap UCL	43.19
Kolmogorov-Smirnov 5% Critical Value	0.0493	95% BCA Bootstrap UCL	44.03
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	48.14
		97.5% Chebyshev(Mean, Sd) UCL	51.58
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	58.36
95% Approximate Gamma UCL	42.63		
95% Adjusted Gamma UCL	42.64		

Potential UCL to Use			
		Use 95% Chebyshev (Mean, Sd) UCL	48.14

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Total Soil

Cobalt

General Statistics

Number of Valid Observations	348	Number of Distinct Observations	164

Raw Statistics		Log-transformed Statistics	
Minimum	1.8	Minimum of Log Data	0.588
Maximum	1500	Maximum of Log Data	7.313
Mean	28.39	Mean of log Data	2.645
Median	12	SD of log Data	0.899
SD	93.49		
Coefficient of Variation	3.293		
Skewness	12.36		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Lilliefors Test Statistic	0.388	Lilliefors Test Statistic	0.121
Lilliefors Critical Value	0.0475	Lilliefors Critical Value	0.0475
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	36.66	95% H-UCL	23.29
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	26.29
95% Adjusted-CLT UCL (Chen-1995)	40.18	97.5% Chebyshev (MVUE) UCL	28.54
95% Modified-t UCL (Johnson-1978)	37.21	99% Chebyshev (MVUE) UCL	32.98

Gamma Distribution Test		Data Distribution	
k star (bias corrected)	0.836	Data do not follow a Discernable Distribution (0.05)	
Theta Star	33.96		
MLE of Mean	28.39		
MLE of Standard Deviation	31.05		
nu star	581.8		
Approximate Chi Square Value (.05)	526.8	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0493	95% CLT UCL	36.63
Adjusted Chi Square Value	526.6	95% Jackknife UCL	36.66
		95% Standard Bootstrap UCL	36.47
Anderson-Darling Test Statistic	2.874E+28	95% Bootstrap-t UCL	46.69
Anderson-Darling 5% Critical Value	0.793	95% Hall's Bootstrap UCL	69.6
Kolmogorov-Smirnov Test Statistic	0.238	95% Percentile Bootstrap UCL	37.4
Kolmogorov-Smirnov 5% Critical Value	0.0506	95% BCA Bootstrap UCL	41.58
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	50.24
		97.5% Chebyshev(Mean, Sd) UCL	59.69
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	78.25
95% Approximate Gamma UCL	31.35		
95% Adjusted Gamma UCL	31.36		

Potential UCL to Use		Use 95% Chebyshev (Mean, Sd) UCL	50.24

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Total Soil

Copper

General Statistics

Number of Valid Observations	348	Number of Distinct Observations	228

Raw Statistics		Log-transformed Statistics	
Minimum	7.5	Minimum of Log Data	2.015
Maximum	36000	Maximum of Log Data	10.49
Mean	1200	Mean of log Data	6.148
Median	530	SD of log Data	1.412
SD	2938		
Coefficient of Variation	2.448		
Skewness	8.135		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Lilliefors Test Statistic	0.342	Lilliefors Test Statistic	0.0749
Lilliefors Critical Value	0.0475	Lilliefors Critical Value	0.0475
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	1460	95% H-UCL	1531
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	1840
95% Adjusted-CLT UCL (Chen-1995)	1533	97.5% Chebyshev (MVUE) UCL	2091
95% Modified-t UCL (Johnson-1978)	1472	99% Chebyshev (MVUE) UCL	2584

Gamma Distribution Test		Data Distribution	
k star (bias corrected)	0.645	Data do not follow a Discernable Distribution (0.05)	
Theta Star	1862		
MLE of Mean	1200		
MLE of Standard Deviation	1495		
nu star	448.6		
Approximate Chi Square Value (.05)	400.5	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0493	95% CLT UCL	1459
Adjusted Chi Square Value	400.3	95% Jackknife UCL	1460
		95% Standard Bootstrap UCL	1466
Anderson-Darling Test Statistic	5.583	95% Bootstrap-t UCL	1638
Anderson-Darling 5% Critical Value	0.807	95% Hall's Bootstrap UCL	1566
Kolmogorov-Smirnov Test Statistic	0.0936	95% Percentile Bootstrap UCL	1487
Kolmogorov-Smirnov 5% Critical Value	0.0511	95% BCA Bootstrap UCL	1531
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	1887
		97.5% Chebyshev(Mean, Sd) UCL	2184
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	2767
95% Approximate Gamma UCL	1345		
95% Adjusted Gamma UCL	1345		

Potential UCL to Use		Use 95% Chebyshev (Mean, Sd) UCL	1887

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Total Soil

Iron

General Statistics

Number of Valid Observations	348	Number of Distinct Observations	182

Raw Statistics

Minimum	2400
Maximum	300000
Mean	55835
Median	33250
SD	49550
Coefficient of Variation	0.887
Skewness	1.387

Log-transformed Statistics

Minimum of Log Data	7.783
Maximum of Log Data	12.61
Mean of log Data	10.54
SD of log Data	0.92

Relevant UCL Statistics

Normal Distribution Test

Lilliefors Test Statistic	0.197
Lilliefors Critical Value	0.0475

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Lilliefors Test Statistic	0.0688
Lilliefors Critical Value	0.0475

Data not Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	60215

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995)	60415
95% Modified-t UCL (Johnson-1978)	60248

Assuming Lognormal Distribution

95% H-UCL	63972
95% Chebyshev (MVUE) UCL	72393
97.5% Chebyshev (MVUE) UCL	78759
99% Chebyshev (MVUE) UCL	91262

Gamma Distribution Test

k star (bias corrected)	1.42
Theta Star	39330
MLE of Mean	55835
MLE of Standard Deviation	46861
nu star	988.1
Approximate Chi Square Value (.05)	916.1
Adjusted Level of Significance	0.0493
Adjusted Chi Square Value	915.8
Anderson-Darling Test Statistic	5.599
Anderson-Darling 5% Critical Value	0.774
Kolmogorov-Smirnov Test Statistic	0.119
Kolmogorov-Smirnov 5% Critical Value	0.0498

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	60221
95% Adjusted Gamma UCL	60239

Data Distribution

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

95% CLT UCL	60204
95% Jackknife UCL	60215
95% Standard Bootstrap UCL	60061
95% Bootstrap-t UCL	60408
95% Hall's Bootstrap UCL	60442
95% Percentile Bootstrap UCL	60232
95% BCA Bootstrap UCL	60128
95% Chebyshev(Mean, Sd) UCL	67413
97.5% Chebyshev(Mean, Sd) UCL	72422
99% Chebyshev(Mean, Sd) UCL	82263

Potential UCL to Use

Use 95% Chebyshev (Mean, Sd) UCL 67413

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Total Soil

Manganese

General Statistics

Number of Valid Observations	348	Number of Distinct Observations	181

Raw Statistics

Minimum	17
Maximum	8380
Mean	290.2
Median	175.5
SD	516.5
Coefficient of Variation	1.78
Skewness	11.55

Log-transformed Statistics

Minimum of Log Data	2.833
Maximum of Log Data	9.034
Mean of log Data	5.215
SD of log Data	0.887

Relevant UCL Statistics

Normal Distribution Test

Lilliefors Test Statistic	0.299
Lilliefors Critical Value	0.0475

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Lilliefors Test Statistic	0.0501
Lilliefors Critical Value	0.0475

Data not Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	335.8

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995)	354
95% Modified-t UCL (Johnson-1978)	338.7

Assuming Lognormal Distribution

95% H-UCL	300.6
95% Chebyshev (MVUE) UCL	338.7
97.5% Chebyshev (MVUE) UCL	367.4
99% Chebyshev (MVUE) UCL	423.8

Gamma Distribution Test

k star (bias corrected)	1.229
Theta Star	236.2
MLE of Mean	290.2
MLE of Standard Deviation	261.8
nu star	855.2
Approximate Chi Square Value (.05)	788.3
Adjusted Level of Significance	0.0493
Adjusted Chi Square Value	788
Anderson-Darling Test Statistic	7.29
Anderson-Darling 5% Critical Value	0.779
Kolmogorov-Smirnov Test Statistic	0.111
Kolmogorov-Smirnov 5% Critical Value	0.05

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	314.8
95% Adjusted Gamma UCL	314.9

Data Distribution

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

95% CLT UCL	335.7
95% Jackknife UCL	335.8
95% Standard Bootstrap UCL	335.6
95% Bootstrap-t UCL	369.8
95% Hall's Bootstrap UCL	537.3
95% Percentile Bootstrap UCL	341.8
95% BCA Bootstrap UCL	364.5
95% Chebyshev(Mean, Sd) UCL	410.9
97.5% Chebyshev(Mean, Sd) UCL	463.1
99% Chebyshev(Mean, Sd) UCL	565.7

Potential UCL to Use

Use 95% Chebyshev (Mean, Sd) UCL 410.9

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Total Soil

Mercury

General Statistics

Number of Valid Data	348	Number of Detected Data	295
Number of Distinct Detected Data	118	Number of Non-Detect Data	53
		Percent Non-Detects	15.23%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.006	Minimum Detected	-5.116
Maximum Detected	2.8	Maximum Detected	1.03
Mean of Detected	0.12	Mean of Detected	-2.618
SD of Detected	0.195	SD of Detected	0.963
Minimum Non-Detect	0.009	Minimum Non-Detect	-4.711
Maximum Non-Detect	0.13	Maximum Non-Detect	-2.04

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	264
Number treated as Detected	84
Single DL Non-Detect Percentage	75.86%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Lilliefors Test Statistic	0.28	Lilliefors Test Statistic	0.0357
5% Lilliefors Critical Value	0.0516	5% Lilliefors Critical Value	0.0516
Data not Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	0.107	Mean	-2.745
SD	0.182	SD	0.973
95% DL/2 (t) UCL	0.123	95% H-Stat (DL/2) UCL	0.115
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE yields a negative mean		Mean in Log Scale	-2.756
		SD in Log Scale	0.972
		Mean in Original Scale	0.107
		SD in Original Scale	0.182
		95% t UCL	0.123
		95% Percentile Bootstrap UCL	0.124
		95% BCA Bootstrap UCL	0.129

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	1.14	Data appear Lognormal at 5% Significance Level	
Theta Star	0.105		
nu star	672.5		
A-D Test Statistic	4.714	Nonparametric Statistics	
5% A-D Critical Value	0.78	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.78	Mean	0.107
5% K-S Critical Value	0.0541	SD	0.182
Data not Gamma Distributed at 5% Significance Level		SE of Mean	0.00979
		95% KM (t) UCL	0.123
Assuming Gamma Distribution		95% KM (z) UCL	0.123
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	0.123
Minimum	1E-12	95% KM (bootstrap t) UCL	0.134
Maximum	2.8	95% KM (BCA) UCL	0.125
Mean	0.111	95% KM (Percentile Bootstrap) UCL	0.124
Median	0.0685	95% KM (Chebyshev) UCL	0.149
SD	0.182	97.5% KM (Chebyshev) UCL	0.168
k star	0.462	99% KM (Chebyshev) UCL	0.204
Theta star	0.24		
Nu star	321.2	Potential UCLs to Use	
AppChi2	280.7	95% KM (BCA) UCL	0.125
95% Gamma Approximate UCL	0.127		
95% Adjusted Gamma UCL	0.127		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Total Soil

Molybdenum

General Statistics

Number of Valid Data	348	Number of Detected Data	288
Number of Distinct Detected Data	148	Number of Non-Detect Data	60
		Percent Non-Detects	17.24%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.19	Minimum Detected	-1.661
Maximum Detected	69	Maximum Detected	4.234
Mean of Detected	5.295	Mean of Detected	0.845
SD of Detected	7.68	SD of Detected	1.308
Minimum Non-Detect	0.22	Minimum Non-Detect	-1.514
Maximum Non-Detect	43	Maximum Non-Detect	3.761

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	347
Number treated as Detected	1
Single DL Non-Detect Percentage	99.71%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Lilliefors Test Statistic	0.253	Lilliefors Test Statistic	0.102
5% Lilliefors Critical Value	0.0522	5% Lilliefors Critical Value	0.0522
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	5.108	Mean	0.82
SD	7.214	SD	1.333
95% DL/2 (t) UCL	5.745	95% H-Stat (DL/2) UCL	6.559
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	0.678
		SD in Log Scale	1.305
		Mean in Original Scale	4.605
		SD in Original Scale	7.162
		95% t UCL	5.238
		95% Percentile Bootstrap UCL	5.287
		95% BCA Bootstrap UCL	5.314

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	0.726	Data do not follow a Discernable Distribution (0.05)	
Theta Star	7.296		
nu star	418		
A-D Test Statistic	8.038	Nonparametric Statistics	
5% A-D Critical Value	0.799	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.799	Mean	4.689
5% K-S Critical Value	0.0557	SD	7.193
Data not Gamma Distributed at 5% Significance Level		SE of Mean	0.391
		95% KM (t) UCL	5.333
Assuming Gamma Distribution		95% KM (z) UCL	5.331
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	5.332
Minimum	1E-12	95% KM (bootstrap t) UCL	5.425
Maximum	69	95% KM (BCA) UCL	5.419
Mean	4.958	95% KM (Percentile Bootstrap) UCL	5.352
Median	2.1	95% KM (Chebyshev) UCL	6.391
SD	7.132	97.5% KM (Chebyshev) UCL	7.127
k star	0.402	99% KM (Chebyshev) UCL	8.574
Theta star	12.34		
Nu star	279.6	Potential UCLs to Use	
AppChi2	241.8	95% KM (Chebyshev) UCL	6.391
95% Gamma Approximate UCL	5.731		
95% Adjusted Gamma UCL	5.734		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Total Soil

Nickel

General Statistics

Number of Valid Data	348	Number of Detected Data	346
Number of Distinct Detected Data	167	Number of Non-Detect Data	2
		Percent Non-Detects	0.57%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	1.1	Minimum Detected	0.0953
Maximum Detected	470	Maximum Detected	6.153
Mean of Detected	24.39	Mean of Detected	2.907
SD of Detected	29.01	SD of Detected	0.76
Minimum Non-Detect	3	Minimum Non-Detect	1.099
Maximum Non-Detect	13	Maximum Non-Detect	2.565

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	98
Number treated as Detected	250
Single DL Non-Detect Percentage	28.16%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Lilliefors Test Statistic	0.215	Lilliefors Test Statistic	0.067
5% Lilliefors Critical Value	0.0476	5% Lilliefors Critical Value	0.0476
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	24.27	Mean	2.897
SD	28.97	SD	0.771
95% DL/2 (t) UCL	26.83	95% H-Stat (DL/2) UCL	26.45
Maximum Likelihood Estimate(MLE) Method		Log ROS Method	
Mean	18.37	Mean in Log Scale	2.9
SD	35.11	SD in Log Scale	0.765
95% MLE (t) UCL	21.47	Mean in Original Scale	24.28
95% MLE (Tiku) UCL	21.55	SD in Original Scale	28.96
		95% t UCL	26.84
		95% Percentile Bootstrap UCL	26.99
		95% BCA Bootstrap UCL	27.89

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	1.878	Data do not follow a Discernable Distribution (0.05)	
Theta Star	12.99		
nu star	1300		
A-D Test Statistic	1.768	Nonparametric Statistics	
5% A-D Critical Value	0.767	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.767	Mean	24.28
5% K-S Critical Value	0.0496	SD	28.92
Data not Gamma Distributed at 5% Significance Level		SE of Mean	1.553
		95% KM (t) UCL	26.84
Assuming Gamma Distribution		95% KM (z) UCL	26.83
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	26.84
Minimum	1E-12	95% KM (bootstrap t) UCL	28.48
Maximum	470	95% KM (BCA) UCL	27.33
Mean	24.27	95% KM (Percentile Bootstrap) UCL	26.93
Median	19	95% KM (Chebyshev) UCL	31.05
SD	28.97	97.5% KM (Chebyshev) UCL	33.97
k star	1.474	99% KM (Chebyshev) UCL	39.73
Theta star	16.46		
Nu star	1026	Potential UCLs to Use	
AppChi2	952.5	95% KM (BCA) UCL	27.33
95% Gamma Approximate UCL	26.13		
95% Adjusted Gamma UCL	26.14		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Total Soil

Selenium

General Statistics

Number of Valid Data	348	Number of Detected Data	322
Number of Distinct Detected Data	175	Number of Non-Detect Data	26
		Percent Non-Detects	7.47%

Raw Statistics		**Log-transformed Statistics**	
Minimum Detected	0.24	Minimum Detected	-1.427
Maximum Detected	170	Maximum Detected	5.136
Mean of Detected	15.84	Mean of Detected	1.817
SD of Detected	21.43	SD of Detected	1.514
Minimum Non-Detect	0.82	Minimum Non-Detect	-0.198
Maximum Non-Detect	11	Maximum Non-Detect	2.398

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	217
Number treated as Detected	131
Single DL Non-Detect Percentage	62.36%

UCL Statistics

Normal Distribution Test with Detected Values Only		**Lognormal Distribution Test with Detected Values Only**	
Lilliefors Test Statistic	0.233	Lilliefors Test Statistic	0.0682
5% Lilliefors Critical Value	0.0494	5% Lilliefors Critical Value	0.0494
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	14.77	Mean	1.683
SD	20.96	SD	1.545
95% DL/2 (t) UCL	16.62	95% H-Stat (DL/2) UCL	22.06
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE yields a negative mean		Mean in Log Scale	1.679
		SD in Log Scale	1.541
		Mean in Original Scale	14.74
		SD in Original Scale	20.97
		95% t UCL	16.6
		95% Percentile Bootstrap UCL	16.51
		95% BCA Bootstrap UCL	16.86

Gamma Distribution Test with Detected Values Only		**Data Distribution Test with Detected Values Only**	
k star (bias corrected)	0.642	Data do not follow a Discernable Distribution (0.05)	
Theta Star	24.67		
nu star	413.6		
A-D Test Statistic	4.714	**Nonparametric Statistics**	
5% A-D Critical Value	0.807	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.807	Mean	14.74
5% K-S Critical Value	0.053	SD	20.94
Data not Gamma Distributed at 5% Significance Level		SE of Mean	1.124
		95% KM (t) UCL	16.6
Assuming Gamma Distribution		95% KM (z) UCL	16.59
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	16.6
Minimum	1E-12	95% KM (bootstrap t) UCL	16.83
Maximum	170	95% KM (BCA) UCL	16.61
Mean	14.69	95% KM (Percentile Bootstrap) UCL	16.59
Median	4.65	95% KM (Chebyshev) UCL	19.64
SD	21.01	97.5% KM (Chebyshev) UCL	21.76
k star	0.258	99% KM (Chebyshev) UCL	25.93
Theta star	57.05		
Nu star	179.2	**Potential UCLs to Use**	
AppChi2	149.3	97.5% KM (Chebyshev) UCL	21.76
95% Gamma Approximate UCL	17.64		
95% Adjusted Gamma UCL	17.65		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Total Soil

Vanadium

General Statistics

Number of Valid Observations	348	Number of Distinct Observations	180

Raw Statistics

Minimum	5.2
Maximum	400
Mean	49.51
Median	46
SD	31.68
Coefficient of Variation	0.64
Skewness	4.443

Log-transformed Statistics

Minimum of Log Data	1.649
Maximum of Log Data	5.991
Mean of log Data	3.737
SD of log Data	0.601

Relevant UCL Statistics

Normal Distribution Test

Lilliefors Test Statistic	0.126
Lilliefors Critical Value	0.0475

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Lilliefors Test Statistic	0.0933
Lilliefors Critical Value	0.0475

Data not Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	52.31

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995)	52.74
95% Modified-t UCL (Johnson-1978)	52.38

Assuming Lognormal Distribution

95% H-UCL	53.38
95% Chebyshev (MVUE) UCL	57.91
97.5% Chebyshev (MVUE) UCL	61.22
99% Chebyshev (MVUE) UCL	67.73

Gamma Distribution Test

k star (bias corrected)	3.163
Theta Star	15.65
MLE of Mean	49.51
MLE of Standard Deviation	27.84
nu star	2201
Approximate Chi Square Value (.05)	2093
Adjusted Level of Significance	0.0493
Adjusted Chi Square Value	2093
Anderson-Darling Test Statistic	1.495
Anderson-Darling 5% Critical Value	0.76
Kolmogorov-Smirnov Test Statistic	0.0563
Kolmogorov-Smirnov 5% Critical Value	0.0491

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	52.07
95% Adjusted Gamma UCL	52.08

Data Distribution

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

95% CLT UCL	52.31
95% Jackknife UCL	52.31
95% Standard Bootstrap UCL	52.25
95% Bootstrap-t UCL	52.84
95% Hall's Bootstrap UCL	53.2
95% Percentile Bootstrap UCL	52.44
95% BCA Bootstrap UCL	52.65
95% Chebyshev(Mean, Sd) UCL	56.92
97.5% Chebyshev(Mean, Sd) UCL	60.12
99% Chebyshev(Mean, Sd) UCL	66.41

Potential UCL to Use

Use 95% Chebyshev (Mean, Sd) UCL 56.92

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Total Soil

Zinc

General Statistics

Number of Valid Observations	348	Number of Distinct Observations	189

Raw Statistics

Minimum	10
Maximum	5700
Mean	162
Median	85.35
SD	426.9
Coefficient of Variation	2.634
Skewness	10.83

Log-transformed Statistics

Minimum of Log Data	2.303
Maximum of Log Data	8.648
Mean of log Data	4.567
SD of log Data	0.801

Relevant UCL Statistics

Normal Distribution Test

Lilliefors Test Statistic	0.369
Lilliefors Critical Value	0.0475

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Lilliefors Test Statistic	0.107
Lilliefors Critical Value	0.0475

Data not Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	199.8

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995)	213.9
95% Modified-t UCL (Johnson-1978)	202

Assuming Lognormal Distribution

95% H-UCL	144.5
95% Chebyshev (MVUE) UCL	161
97.5% Chebyshev (MVUE) UCL	173.3
99% Chebyshev (MVUE) UCL	197.4

Gamma Distribution Test

k star (bias corrected)	1.09
Theta Star	148.7
MLE of Mean	162
MLE of Standard Deviation	155.2
nu star	758.5
Approximate Chi Square Value (.05)	695.6
Adjusted Level of Significance	0.0493
Adjusted Chi Square Value	695.4
Anderson-Darling Test Statistic	2.874E+28
Anderson-Darling 5% Critical Value	0.782
Kolmogorov-Smirnov Test Statistic	0.211
Kolmogorov-Smirnov 5% Critical Value	0.0501

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	176.7
95% Adjusted Gamma UCL	176.8

Data Distribution

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

95% CLT UCL	199.7
95% Jackknife UCL	199.8
95% Standard Bootstrap UCL	199.2
95% Bootstrap-t UCL	257.5
95% Hall's Bootstrap UCL	391.2
95% Percentile Bootstrap UCL	202.8
95% BCA Bootstrap UCL	214.4
95% Chebyshev(Mean, Sd) UCL	261.8
97.5% Chebyshev(Mean, Sd) UCL	304.9
99% Chebyshev(Mean, Sd) UCL	389.7

Potential UCL to Use

Use 95% Chebyshev (Mean, Sd) UCL 261.8

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Ely Brook

Aluminum

General Statistics

Number of Valid Observations	38	Number of Distinct Observations	33

Raw Statistics

Minimum	2800
Maximum	111000
Mean	27044
Median	17800
SD	23819
Coefficient of Variation	0.881
Skewness	1.51

Log-transformed Statistics

Minimum of Log Data	7.937
Maximum of Log Data	11.62
Mean of log Data	9.827
SD of log Data	0.919

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.839
Shapiro Wilk Critical Value	0.938

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.969
Shapiro Wilk Critical Value	0.938

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	33563

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995)	34411
95% Modified-t UCL (Johnson-1978)	33721

Assuming Lognormal Distribution

95% H-UCL	40008
95% Chebyshev (MVUE) UCL	48452
97.5% Chebyshev (MVUE) UCL	57379
99% Chebyshev (MVUE) UCL	74915

Gamma Distribution Test

k star (bias corrected)	1.368
Theta Star	19775
MLE of Mean	27044
MLE of Standard Deviation	23125
nu star	103.9
Approximate Chi Square Value (.05)	81.41
Adjusted Level of Significance	0.0434
Adjusted Chi Square Value	80.59
Anderson-Darling Test Statistic	0.56
Anderson-Darling 5% Critical Value	0.767
Kolmogorov-Smirnov Test Statistic	0.105
Kolmogorov-Smirnov 5% Critical Value	0.146

Data appear Gamma Distributed at 5% Significance Level

Data Distribution

Data appear Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	34526
95% Adjusted Gamma UCL	34880

Nonparametric Statistics

95% CLT UCL	33400
95% Jackknife UCL	33563
95% Standard Bootstrap UCL	33408
95% Bootstrap-t UCL	34829
95% Hall's Bootstrap UCL	34766
95% Percentile Bootstrap UCL	33382
95% BCA Bootstrap UCL	34016
95% Chebyshev(Mean, Sd) UCL	43887
97.5% Chebyshev(Mean, Sd) UCL	51174
99% Chebyshev(Mean, Sd) UCL	65490

Potential UCL to Use

Use 95% Approximate Gamma UCL 34526

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002) and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Ely Brook

Antimony

General Statistics

Number of Valid Data	38	Number of Detected Data	16
Number of Distinct Detected Data	14	Number of Non-Detect Data	22
		Percent Non-Detects	57.89%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.06	Minimum Detected	-2.813
Maximum Detected	2.03	Maximum Detected	0.708
Mean of Detected	0.546	Mean of Detected	-1.203
SD of Detected	0.603	SD of Detected	1.145
Minimum Non-Detect	0.05	Minimum Non-Detect	-2.996
Maximum Non-Detect	49	Maximum Non-Detect	3.892

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	38
Number treated as Detected	0
Single DL Non-Detect Percentage	100.00%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.778	Shapiro Wilk Test Statistic	0.914
5% Shapiro Wilk Critical Value	0.887	5% Shapiro Wilk Critical Value	0.887
Data not Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	3.671	Mean	-0.175
SD	5.887	SD	1.929
95% DL/2 (t) UCL	5.282	95% H-Stat (DL/2) UCL	17.11
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	-1.645
		SD in Log Scale	1.012
		Mean in Original Scale	0.333
		SD in Original Scale	0.436
		95% t UCL	0.453
		95% Percentile Bootstrap UCL	0.455
		95% BCA Bootstrap UCL	0.478

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only
k star (bias corrected)	0.831	Data Follow Appr. Gamma Distribution at 5% Significance Level
Theta Star	0.657	
nu star	26.58	
A-D Test Statistic	0.855	
5% A-D Critical Value	0.765	
K-S Test Statistic	0.765	
5% K-S Critical Value	0.221	
Data follow Appr. Gamma Distribution at 5% Significance Level		

Assuming Gamma Distribution		Nonparametric Statistics	
Gamma ROS Statistics using Extrapolated Data		Kaplan-Meier (KM) Method	
Minimum	0.06	Mean	0.401
Maximum	2.03	SD	0.51
Mean	0.565	SE of Mean	0.107
Median	0.532	95% KM (t) UCL	0.581
SD	0.419	95% KM (z) UCL	0.577
k star	1.677	95% KM (jackknife) UCL	0.579
Theta star	0.337	95% KM (bootstrap t) UCL	0.642
Nu star	127.5	95% KM (BCA) UCL	0.612
AppChi2	102.4	95% KM (Percentile Bootstrap) UCL	0.589
95% Gamma Approximate UCL	0.704	95% KM (Chebyshev) UCL	0.866
95% Adjusted Gamma UCL	0.71	97.5% KM (Chebyshev) UCL	1.067
		99% KM (Chebyshev) UCL	1.461
		Potential UCLs to Use	
		95% KM (t) UCL	0.581

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Ely Brook

Arsenic

General Statistics

Number of Valid Data	38	Number of Detected Data	23
Number of Distinct Detected Data	17	Number of Non-Detect Data	15
		Percent Non-Detects	39.47%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.63	Minimum Detected	-0.462
Maximum Detected	18.5	Maximum Detected	2.918
Mean of Detected	3.49	Mean of Detected	0.972
SD of Detected	3.591	SD of Detected	0.712
Minimum Non-Detect	0.995	Minimum Non-Detect	-0.00501
Maximum Non-Detect	49	Maximum Non-Detect	3.892

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	38
Number treated as Detected	0
Single DL Non-Detect Percentage	100.00%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.588	Shapiro Wilk Test Statistic	0.962
5% Shapiro Wilk Critical Value	0.914	5% Shapiro Wilk Critical Value	0.914
Data not Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	4.086	Mean	0.748
SD	5.724	SD	1.132
95% DL/2 (t) UCL	5.652	95% H-Stat (DL/2) UCL	6.443
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	0.588
		SD in Log Scale	0.799
		Mean in Original Scale	2.558
		SD in Original Scale	3.025
		95% t UCL	3.385
		95% Percentile Bootstrap UCL	3.399
		95% BCA Bootstrap UCL	3.724

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only
k star (bias corrected)	1.725	Data Follow Appr. Gamma Distribution at 5% Significance Level
Theta Star	2.022	
nu star	79.37	
A-D Test Statistic	0.765	
5% A-D Critical Value	0.755	
K-S Test Statistic	0.755	
5% K-S Critical Value	0.184	
Data follow Appr. Gamma Distribution at 5% Significance Level		

Assuming Gamma Distribution		Nonparametric Statistics	
Gamma ROS Statistics using Extrapolated Data		Kaplan-Meier (KM) Method	
Minimum	1E-12	Mean	2.652
Maximum	18.5	SD	3.148
Mean	3.218	SE of Mean	0.554
Median	2.767	95% KM (t) UCL	3.587
SD	3.03	95% KM (z) UCL	3.564
k star	0.567	95% KM (jackknife) UCL	3.584
Theta star	5.673	95% KM (bootstrap t) UCL	4.512
Nu star	43.11	95% KM (BCA) UCL	3.752
AppChi2	29.06	95% KM (Percentile Bootstrap) UCL	3.685
95% Gamma Approximate UCL	4.775	95% KM (Chebyshev) UCL	5.068
95% Adjusted Gamma UCL	4.855	97.5% KM (Chebyshev) UCL	6.113
		99% KM (Chebyshev) UCL	8.166
		Potential UCLs to Use	
		95% KM (Percentile Bootstrap) UCL	3.685

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Ely Brook

Chromium

General Statistics

Number of Valid Observations	38	Number of Distinct Observations	34

Raw Statistics		**Log-transformed Statistics**	
Minimum	18	Minimum of Log Data	2.89
Maximum	130	Maximum of Log Data	4.868
Mean	46.89	Mean of log Data	3.726
Median	41.15	SD of log Data	0.496
SD	24.66		
Coefficient of Variation	0.526		
Skewness	1.328		

Relevant UCL Statistics

Normal Distribution Test		**Lognormal Distribution Test**	
Shapiro Wilk Test Statistic	0.883	Shapiro Wilk Test Statistic	0.963
Shapiro Wilk Critical Value	0.938	Shapiro Wilk Critical Value	0.938
Data not Normal at 5% Significance Level		**Data appear Lognormal at 5% Significance Level**	

Assuming Normal Distribution		**Assuming Lognormal Distribution**	
95% Student's-t UCL	53.64	95% H-UCL	54.88
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	63.96
95% Adjusted-CLT UCL (Chen-1995)	54.4	97.5% Chebyshev (MVUE) UCL	71.39
95% Modified-t UCL (Johnson-1978)	53.79	99% Chebyshev (MVUE) UCL	85.99

Gamma Distribution Test		**Data Distribution**	
k star (bias corrected)	3.941	**Data appear Gamma Distributed at 5% Significance Level**	
Theta Star	11.9		
MLE of Mean	46.89		
MLE of Standard Deviation	23.62		
nu star	299.5		
Approximate Chi Square Value (.05)	260.5	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0434	95% CLT UCL	53.47
Adjusted Chi Square Value	258.9	95% Jackknife UCL	53.64
		95% Standard Bootstrap UCL	53.38
Anderson-Darling Test Statistic	0.571	95% Bootstrap-t UCL	55.2
Anderson-Darling 5% Critical Value	0.752	95% Hall's Bootstrap UCL	54.86
Kolmogorov-Smirnov Test Statistic	0.105	95% Percentile Bootstrap UCL	53.51
Kolmogorov-Smirnov 5% Critical Value	0.144	95% BCA Bootstrap UCL	54.12
Data appear Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	64.33
		97.5% Chebyshev(Mean, Sd) UCL	71.88
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	86.7
95% Approximate Gamma UCL	53.93		
95% Adjusted Gamma UCL	54.25		

Potential UCL to Use		Use 95% Approximate Gamma UCL	53.93

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002) and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Ely Brook

Cobalt

General Statistics

Number of Valid Observations	38	Number of Distinct Observations	31

Raw Statistics		Log-transformed Statistics	
Minimum	9.5	Minimum of Log Data	2.251
Maximum	102	Maximum of Log Data	4.625
Mean	28.98	Mean of log Data	3.128
Median	19.4	SD of log Data	0.668
SD	22.77		
Coefficient of Variation	0.786		
Skewness	1.587		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Shapiro Wilk Test Statistic	0.779	Shapiro Wilk Test Statistic	0.913
Shapiro Wilk Critical Value	0.938	Shapiro Wilk Critical Value	0.938
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	35.21	95% H-UCL	35.72
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	42.76
95% Adjusted-CLT UCL (Chen-1995)	36.07	97.5% Chebyshev (MVUE) UCL	49.01
95% Modified-t UCL (Johnson-1978)	35.37	99% Chebyshev (MVUE) UCL	61.29

Gamma Distribution Test		Data Distribution	
k star (bias corrected)	2.086	Data do not follow a Discernable Distribution (0.05)	
Theta Star	13.9		
MLE of Mean	28.98		
MLE of Standard Deviation	20.07		
nu star	158.5		
Approximate Chi Square Value (.05)	130.4	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0434	95% CLT UCL	35.05
Adjusted Chi Square Value	129.3	95% Jackknife UCL	35.21
		95% Standard Bootstrap UCL	34.97
Anderson-Darling Test Statistic	1.677	95% Bootstrap-t UCL	37.19
Anderson-Darling 5% Critical Value	0.758	95% Hall's Bootstrap UCL	35.65
Kolmogorov-Smirnov Test Statistic	0.184	95% Percentile Bootstrap UCL	35.25
Kolmogorov-Smirnov 5% Critical Value	0.145	95% BCA Bootstrap UCL	35.97
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	45.08
		97.5% Chebyshev(Mean, Sd) UCL	52.04
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	65.72
95% Approximate Gamma UCL	35.23		
95% Adjusted Gamma UCL	35.51		

Potential UCL to Use			
		Use 95% Chebyshev (Mean, Sd) UCL	45.08

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Ely Brook

Copper

General Statistics

Number of Valid Observations	38	Number of Distinct Observations	38

Raw Statistics		**Log-transformed Statistics**	
Minimum	40	Minimum of Log Data	3.689
Maximum	11000	Maximum of Log Data	9.306
Mean	2193	Mean of log Data	6.88
Median	1685	SD of log Data	1.583
SD	2327		
Coefficient of Variation	1.061		
Skewness	1.794		

Relevant UCL Statistics

Normal Distribution Test		**Lognormal Distribution Test**	
Shapiro Wilk Test Statistic	0.826	Shapiro Wilk Test Statistic	0.899
Shapiro Wilk Critical Value	0.938	Shapiro Wilk Critical Value	0.938
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		**Assuming Lognormal Distribution**	
95% Student's-t UCL	2829	95% H-UCL	7718
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	7862
95% Adjusted-CLT UCL (Chen-1995)	2931	97.5% Chebyshev (MVUE) UCL	9888
95% Modified-t UCL (Johnson-1978)	2848	99% Chebyshev (MVUE) UCL	13867

Gamma Distribution Test		**Data Distribution**
k star (bias corrected)	0.697	Data appear Gamma Distributed at 5% Significance Level
Theta Star	3145	
MLE of Mean	2193	
MLE of Standard Deviation	2626	
nu star	52.98	
Approximate Chi Square Value (.05)	37.26	
Adjusted Level of Significance	0.0434	
Adjusted Chi Square Value	36.72	
Anderson-Darling Test Statistic	0.656	
Anderson-Darling 5% Critical Value	0.79	
Kolmogorov-Smirnov Test Statistic	0.128	
Kolmogorov-Smirnov 5% Critical Value	0.149	
Data appear Gamma Distributed at 5% Significance Level		

Assuming Gamma Distribution	
95% Approximate Gamma UCL	3118
95% Adjusted Gamma UCL	3164

Nonparametric Statistics	
95% CLT UCL	2813
95% Jackknife UCL	2829
95% Standard Bootstrap UCL	2809
95% Bootstrap-t UCL	3047
95% Hall's Bootstrap UCL	3120
95% Percentile Bootstrap UCL	2829
95% BCA Bootstrap UCL	2924
95% Chebyshev(Mean, Sd) UCL	3838
97.5% Chebyshev(Mean, Sd) UCL	4550
99% Chebyshev(Mean, Sd) UCL	5948

Potential UCL to Use	
Use 95% Approximate Gamma UCL	3118

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Ely Brook

IRON

General Statistics

Number of Valid Observations	38	Number of Distinct Observations	36

Raw Statistics		**Log-transformed Statistics**	
Minimum	13700	Minimum of Log Data	9.525
Maximum	167000	Maximum of Log Data	12.03
Mean	61572	Mean of log Data	10.81
Median	47450	SD of log Data	0.686
SD	40649		
Coefficient of Variation	0.66		
Skewness	0.918		

Relevant UCL Statistics

Normal Distribution Test		**Lognormal Distribution Test**	
Shapiro Wilk Test Statistic	0.895	Shapiro Wilk Test Statistic	0.959
Shapiro Wilk Critical Value	0.938	Shapiro Wilk Critical Value	0.938
Data not Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	

Assuming Normal Distribution		**Assuming Lognormal Distribution**	
95% Student's-t UCL	72697	95% H-UCL	79097
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	94875
95% Adjusted-CLT UCL (Chen-1995)	73468	97.5% Chebyshev (MVUE) UCL	109034
95% Modified-t UCL (Johnson-1978)	72861	99% Chebyshev (MVUE) UCL	136847

Gamma Distribution Test		**Data Distribution**
k star (bias corrected)	2.266	Data appear Gamma Distributed at 5% Significance Level
Theta Star	27167	
MLE of Mean	61572	
MLE of Standard Deviation	40899	
nu star	172.2	
Approximate Chi Square Value (.05)	142.9	**Nonparametric Statistics**
Adjusted Level of Significance	0.0434	95% CLT UCL 72419
Adjusted Chi Square Value	141.8	95% Jackknife UCL 72697
		95% Standard Bootstrap UCL 72185
Anderson-Darling Test Statistic	0.477	95% Bootstrap-t UCL 74393
Anderson-Darling 5% Critical Value	0.757	95% Hall's Bootstrap UCL 73503
Kolmogorov-Smirnov Test Statistic	0.104	95% Percentile Bootstrap UCL 72225
Kolmogorov-Smirnov 5% Critical Value	0.145	95% BCA Bootstrap UCL 73764
Data appear Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL 90315
		97.5% Chebyshev(Mean, Sd) UCL 102752
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL 127183
95% Approximate Gamma UCL	74219	
95% Adjusted Gamma UCL	74798	

Potential UCL to Use		
		Use 95% Approximate Gamma UCL 74219

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Ely Brook

Lithium

General Statistics

Number of Valid Observations	12	Number of Distinct Observations	10

Raw Statistics		**Log-transformed Statistics**	
Minimum	10	Minimum of Log Data	2.303
Maximum	44	Maximum of Log Data	3.784
Mean	28	Mean of log Data	3.239
Median	23.5	SD of log Data	0.474
SD	11.75		
Coefficient of Variation	0.42		
Skewness	0.115		

Relevant UCL Statistics

Normal Distribution Test		**Lognormal Distribution Test**	
Shapiro Wilk Test Statistic	0.91	Shapiro Wilk Test Statistic	0.907
Shapiro Wilk Critical Value	0.859	Shapiro Wilk Critical Value	0.859
Data appear Normal at 5% Significance Level		**Data appear Lognormal at 5% Significance Level**	

Assuming Normal Distribution		**Assuming Lognormal Distribution**	
95% Student's-t UCL	34.09	95% H-UCL	38.55
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	45.43
95% Adjusted-CLT UCL (Chen-1995)	33.7	97.5% Chebyshev (MVUE) UCL	52.86
95% Modified-t UCL (Johnson-1978)	34.11	99% Chebyshev (MVUE) UCL	67.47

Gamma Distribution Test		**Data Distribution**	
k star (bias corrected)	4.183	**Data appear Normal at 5% Significance Level**	
Theta Star	6.693		
MLE of Mean	28		
MLE of Standard Deviation	13.69		
nu star	100.4		
Approximate Chi Square Value (.05)	78.28	**Nonparametric Statistics**	
Adjusted Level of Significance	0.029	95% CLT UCL	33.58
Adjusted Chi Square Value	75.31	95% Jackknife UCL	34.09
		95% Standard Bootstrap UCL	33.44
Anderson-Darling Test Statistic	0.453	95% Bootstrap-t UCL	34.72
Anderson-Darling 5% Critical Value	0.732	95% Hall's Bootstrap UCL	32.9
Kolmogorov-Smirnov Test Statistic	0.166	95% Percentile Bootstrap UCL	33.25
Kolmogorov-Smirnov 5% Critical Value	0.246	95% BCA Bootstrap UCL	33.83
Data appear Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	42.78
		97.5% Chebyshev(Mean, Sd) UCL	49.18
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	61.74
95% Approximate Gamma UCL	35.91		
95% Adjusted Gamma UCL	37.33		

Potential UCL to Use		Use 95% Student's-t UCL	34.09

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002) and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Ely Brook

Manganese

General Statistics

Number of Valid Observations	38	Number of Distinct Observations	35

Raw Statistics

Minimum	62
Maximum	3130
Mean	728.2
Median	451.5
SD	775.5
Coefficient of Variation	1.065
Skewness	1.608

Log-transformed Statistics

Minimum of Log Data	4.127
Maximum of Log Data	8.049
Mean of log Data	6.051
SD of log Data	1.107

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.774
Shapiro Wilk Critical Value	0.938

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.95
Shapiro Wilk Critical Value	0.938

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	940.5

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995)	970.3
95% Modified-t UCL (Johnson-1978)	946

Assuming Lognormal Distribution

95% H-UCL	1240
95% Chebyshev (MVUE) UCL	1478
97.5% Chebyshev (MVUE) UCL	1786
99% Chebyshev (MVUE) UCL	2392

Gamma Distribution Test

k star (bias corrected)	0.996
Theta Star	731.4
MLE of Mean	728.2
MLE of Standard Deviation	729.8
nu star	75.67
Approximate Chi Square Value (.05)	56.64
Adjusted Level of Significance	0.0434
Adjusted Chi Square Value	55.96
Anderson-Darling Test Statistic	0.693
Anderson-Darling 5% Critical Value	0.776
Kolmogorov-Smirnov Test Statistic	0.142
Kolmogorov-Smirnov 5% Critical Value	0.147

Data appear Gamma Distributed at 5% Significance Level

Data Distribution

Data appear Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	973
95% Adjusted Gamma UCL	984.9

Nonparametric Statistics

95% CLT UCL	935.2
95% Jackknife UCL	940.5
95% Standard Bootstrap UCL	934
95% Bootstrap-t UCL	993.2
95% Hall's Bootstrap UCL	973.4
95% Percentile Bootstrap UCL	947.5
95% BCA Bootstrap UCL	962.4
95% Chebyshev(Mean, Sd) UCL	1277
97.5% Chebyshev(Mean, Sd) UCL	1514
99% Chebyshev(Mean, Sd) UCL	1980

Potential UCL to Use

Use 95% Approximate Gamma UCL 973

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Ely Brook

Selenium

General Statistics

Number of Valid Data	38	Number of Detected Data	36
Number of Distinct Detected Data	32	Number of Non-Detect Data	2
		Percent Non-Detects	5.26%

Raw Statistics		**Log-transformed Statistics**	
Minimum Detected	0.3	Minimum Detected	-1.204
Maximum Detected	46	Maximum Detected	3.829
Mean of Detected	12.26	Mean of Detected	1.57
SD of Detected	14.61	SD of Detected	1.553
Minimum Non-Detect	10	Minimum Non-Detect	2.303
Maximum Non-Detect	11	Maximum Non-Detect	2.398

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	26
Number treated as Detected	12
Single DL Non-Detect Percentage	68.42%

UCL Statistics

Normal Distribution Test with Detected Values Only		**Lognormal Distribution Test with Detected Values Only**	
Shapiro Wilk Test Statistic	0.772	Shapiro Wilk Test Statistic	0.93
5% Shapiro Wilk Critical Value	0.935	5% Shapiro Wilk Critical Value	0.935
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	
Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	11.89	Mean	1.575
SD	14.3	SD	1.511
95% DL/2 (t) UCL	15.8	95% H-Stat (DL/2) UCL	32.21
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE yields a negative mean		Mean in Log Scale	1.533
		SD in Log Scale	1.519
		Mean in Original Scale	11.74
		SD in Original Scale	14.38
		95% t UCL	15.68
		95% Percentile Bootstrap UCL	15.45
		95% BCA Bootstrap UCL	16.06

Gamma Distribution Test with Detected Values Only		**Data Distribution Test with Detected Values Only**	
k star (bias corrected)	0.616	Data do not follow a Discernable Distribution (0.05)	
Theta Star	19.89		
nu star	44.37		
A-D Test Statistic	0.918	**Nonparametric Statistics**	
5% A-D Critical Value	0.798	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.798	Mean	11.79
5% K-S Critical Value	0.154	SD	14.18
Data not Gamma Distributed at 5% Significance Level		SE of Mean	2.336
		95% KM (t) UCL	15.73
Assuming Gamma Distribution		95% KM (z) UCL	15.63
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	15.73
Minimum	0.3	95% KM (bootstrap t) UCL	16.53
Maximum	46	95% KM (BCA) UCL	15.42
Mean	11.82	95% KM (Percentile Bootstrap) UCL	15.69
Median	4	95% KM (Chebyshev) UCL	21.97
SD	14.34	97.5% KM (Chebyshev) UCL	26.38
k star	0.633	99% KM (Chebyshev) UCL	35.03
Theta star	18.68		
Nu star	48.08	**Potential UCLs to Use**	
AppChi2	33.16	99% KM (Chebyshev) UCL	35.03
95% Gamma Approximate UCL	17.13		
95% Adjusted Gamma UCL	17.4		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Ely Brook

Vanadium

General Statistics

Number of Valid Observations	38	Number of Distinct Observations	33

Raw Statistics		Log-transformed Statistics	
Minimum	22	Minimum of Log Data	3.091
Maximum	163	Maximum of Log Data	5.094
Mean	64.88	Mean of log Data	4.047
Median	52	SD of log Data	0.503
SD	34.48		
Coefficient of Variation	0.531		
Skewness	1.151		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Shapiro Wilk Test Statistic	0.888	Shapiro Wilk Test Statistic	0.97
Shapiro Wilk Critical Value	0.938	Shapiro Wilk Critical Value	0.938
Data not Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	74.31	95% H-UCL	76.11
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	88.82
95% Adjusted-CLT UCL (Chen-1995)	75.19	97.5% Chebyshev (MVUE) UCL	99.26
95% Modified-t UCL (Johnson-1978)	74.49	99% Chebyshev (MVUE) UCL	119.8

Gamma Distribution Test		Data Distribution
k star (bias corrected)	3.831	Data Follow Appr. Gamma Distribution at 5% Significance Level
Theta Star	16.94	
MLE of Mean	64.88	
MLE of Standard Deviation	33.15	
nu star	291.1	
Approximate Chi Square Value (.05)	252.6	
Adjusted Level of Significance	0.0434	
Adjusted Chi Square Value	251.1	
Anderson-Darling Test Statistic	0.566	
Anderson-Darling 5% Critical Value	0.752	
Kolmogorov-Smirnov Test Statistic	0.144	
Kolmogorov-Smirnov 5% Critical Value	0.144	
Data follow Appr. Gamma Distribution at 5% Significance Level		

Assuming Gamma Distribution		Nonparametric Statistics	
95% Approximate Gamma UCL	74.77	95% CLT UCL	74.08
95% Adjusted Gamma UCL	75.21	95% Jackknife UCL	74.31
		95% Standard Bootstrap UCL	74.21
		95% Bootstrap-t UCL	75.68
		95% Hall's Bootstrap UCL	75.94
		95% Percentile Bootstrap UCL	74.12
		95% BCA Bootstrap UCL	74.67
		95% Chebyshev(Mean, Sd) UCL	89.26
		97.5% Chebyshev(Mean, Sd) UCL	99.81
		99% Chebyshev(Mean, Sd) UCL	120.5

Potential UCL to Use			
		Use 95% Approximate Gamma UCL	74.77

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Schoolhouse Brook

Aluminum

General Statistics

Number of Valid Observations	10	Number of Distinct Observations	10

Raw Statistics		Log-transformed Statistics	
Minimum	4850	Minimum of Log Data	8.487
Maximum	36900	Maximum of Log Data	10.52
Mean	21361	Mean of log Data	9.633
Median	22000	SD of log Data	0.928
SD	15505		
Coefficient of Variation	0.726		
Skewness	-0.0244		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Shapiro Wilk Test Statistic	0.725	Shapiro Wilk Test Statistic	0.766
Shapiro Wilk Critical Value	0.842	Shapiro Wilk Critical Value	0.842
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	30349	95% H-UCL	58615
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	51876
95% Adjusted-CLT UCL (Chen-1995)	29386	97.5% Chebyshev (MVUE) UCL	64718
95% Modified-t UCL (Johnson-1978)	30343	99% Chebyshev (MVUE) UCL	89943

Gamma Distribution Test		Data Distribution	
k star (bias corrected)	1.209	Data do not follow a Discernable Distribution (0.05)	
Theta Star	17670		
MLE of Mean	21361		
MLE of Standard Deviation	19428		
nu star	24.18		
Approximate Chi Square Value (.05)	13.98	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0267	95% CLT UCL	29426
Adjusted Chi Square Value	12.66	95% Jackknife UCL	30349
		95% Standard Bootstrap UCL	28966
Anderson-Darling Test Statistic	1.22	95% Bootstrap-t UCL	31975
Anderson-Darling 5% Critical Value	0.738	95% Hall's Bootstrap UCL	27185
Kolmogorov-Smirnov Test Statistic	0.324	95% Percentile Bootstrap UCL	29620
Kolmogorov-Smirnov 5% Critical Value	0.271	95% BCA Bootstrap UCL	29906
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	42733
		97.5% Chebyshev(Mean, Sd) UCL	51981
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	70147
95% Approximate Gamma UCL	36932		
95% Adjusted Gamma UCL	40811		

Potential UCL to Use Use 95% Chebyshev (Mean, Sd) UCL 42733

Recommended UCL exceeds the maximum observation

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Schoolhouse Brook

Arsenic

General Statistics

Number of Valid Data	10	Number of Detected Data	5
Number of Distinct Detected Data	2	Number of Non-Detect Data	5
		Percent Non-Detects	50.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	1	Minimum Detected	0
Maximum Detected	2	Maximum Detected	0.693
Mean of Detected	1.2	Mean of Detected	0.139
SD of Detected	0.447	SD of Detected	0.31
Minimum Non-Detect	0.99	Minimum Non-Detect	-0.0101
Maximum Non-Detect	22	Maximum Non-Detect	3.091

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	10
Number treated as Detected	0
Single DL Non-Detect Percentage	100.00%

Warning: Data set has only 2 Distinct Detected Values.
This may not be adequate enough to compute meaningful and reliable test statistics and estimates.
The Project Team may decide to use alternative site specific values to estimate environmental parameters (e.g., EPC, BTV).

Unless Data Quality Objectives (DQOs) have been met, it is suggested to collect additional observations.

The number of detected data may not be adequate enough to perform GOF tests, bootstrap, and ROS methods.
Those methods will return a 'N/A' value on your output display!

It is necessary to have 4 or more Distinct Values for bootstrap methods.
However, results obtained using 4 to 9 distinct values may not be reliable.
It is recommended to have 10 to 15 or more observations for accurate and meaningful results and estimates.

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.552	Shapiro Wilk Test Statistic	0.552
5% Shapiro Wilk Critical Value	0.762	5% Shapiro Wilk Critical Value	0.762
Data not Normal at 5% Significance Level		**Data not Lognormal at 5% Significance Level**	
Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	3.799	Mean	0.629
SD	4.536	SD	1.241
95% DL/2 (t) UCL	6.428	95% H-Stat (DL/2) UCL	18.12
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	-0.0446
		SD in Log Scale	0.345
		Mean in Original Scale	1.012
		SD in Original Scale	0.392
		95% t UCL	1.24
		95% Percentile Bootstrap UCL	1.223
		95% BCA Bootstrap UCL	1.288

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	4.776	**Data do not follow a Discernable Distribution (0.05)**	
Theta Star	0.251		
nu star	47.76		
A-D Test Statistic	1.334	**Nonparametric Statistics**	
5% A-D Critical Value	0.679	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.679	Mean	1.143
5% K-S Critical Value	0.358	SD	0.35
Data not Gamma Distributed at 5% Significance Level		SE of Mean	0.148
		95% KM (t) UCL	1.414
Assuming Gamma Distribution		95% KM (z) UCL	1.386
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	1.411
Minimum	0.841	95% KM (bootstrap t) UCL	N/A
Maximum	2	95% KM (BCA) UCL	1.333
Mean	1.192	95% KM (Percentile Bootstrap) UCL	1.4
Median	1.011	95% KM (Chebyshev) UCL	1.787

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Sediment - Schoolhouse Brook

SD	0.345	97.5% KM (Chebyshev) UCL	2.066
k star	11.09	99% KM (Chebyshev) UCL	2.614
Theta star	0.108		
Nu star	221.8	**Potential UCLs to Use**	
AppChi2	188.4	95% KM (t) UCL	1.414
95% Gamma Approximate UCL	1.404	95% KM (% Bootstrap) UCL	1.4
95% Adjusted Gamma UCL	1.446		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Schoolhouse Brook

Chromium

General Statistics

Number of Valid Observations	10	Number of Distinct Observations	9

Raw Statistics		Log-transformed Statistics	
Minimum	10.8	Minimum of Log Data	2.38
Maximum	40	Maximum of Log Data	3.689
Mean	22.46	Mean of log Data	3.017
Median	22	SD of log Data	0.472
SD	9.864		
Coefficient of Variation	0.439		
Skewness	0.334		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Shapiro Wilk Test Statistic	0.939	Shapiro Wilk Test Statistic	0.918
Shapiro Wilk Critical Value	0.842	Shapiro Wilk Critical Value	0.842
Data appear Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	28.18	95% H-UCL	32.16
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	37.46
95% Adjusted-CLT UCL (Chen-1995)	27.94	97.5% Chebyshev (MVUE) UCL	43.9
95% Modified-t UCL (Johnson-1978)	28.23	99% Chebyshev (MVUE) UCL	56.57

Gamma Distribution Test		Data Distribution	
k star (bias corrected)	3.868	Data appear Normal at 5% Significance Level	
Theta Star	5.806		
MLE of Mean	22.46		
MLE of Standard Deviation	11.42		
nu star	77.36		
Approximate Chi Square Value (.05)	58.1	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0267	95% CLT UCL	27.59
Adjusted Chi Square Value	55.21	95% Jackknife UCL	28.18
		95% Standard Bootstrap UCL	27.31
Anderson-Darling Test Statistic	0.332	95% Bootstrap-t UCL	28.76
Anderson-Darling 5% Critical Value	0.729	95% Hall's Bootstrap UCL	27.89
Kolmogorov-Smirnov Test Statistic	0.18	95% Percentile Bootstrap UCL	27.56
Kolmogorov-Smirnov 5% Critical Value	0.267	95% BCA Bootstrap UCL	27.9
Data appear Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	36.06
		97.5% Chebyshev(Mean, Sd) UCL	41.94
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	53.5
95% Approximate Gamma UCL	29.91		
95% Adjusted Gamma UCL	31.47		

Potential UCL to Use			
		Use 95% Student's-t UCL	28.18

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

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Sediment - Schoolhouse Brook

Cobalt

General Statistics

Number of Valid Observations	10	Number of Distinct Observations	9

Raw Statistics		Log-transformed Statistics	
Minimum	3.4	Minimum of Log Data	1.224
Maximum	13.5	Maximum of Log Data	2.603
Mean	8.76	Mean of log Data	2.087
Median	8.95	SD of log Data	0.463
SD	3.321		
Coefficient of Variation	0.379		
Skewness	-0.413		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Shapiro Wilk Test Statistic	0.948	Shapiro Wilk Test Statistic	0.872
Shapiro Wilk Critical Value	0.842	Shapiro Wilk Critical Value	0.842
Data appear Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	10.68	95% H-UCL	12.53
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	14.61
95% Adjusted-CLT UCL (Chen-1995)	10.34	97.5% Chebyshev (MVUE) UCL	17.09
95% Modified-t UCL (Johnson-1978)	10.66	99% Chebyshev (MVUE) UCL	21.97

Gamma Distribution Test		Data Distribution
k star (bias corrected)	4.366	Data appear Normal at 5% Significance Level
Theta Star	2.006	
MLE of Mean	8.76	
MLE of Standard Deviation	4.192	
nu star	87.33	
Approximate Chi Square Value (.05)	66.78	**Nonparametric Statistics**
Adjusted Level of Significance	0.0267	95% CLT UCL 10.49
Adjusted Chi Square Value	63.67	95% Jackknife UCL 10.68
		95% Standard Bootstrap UCL 10.4
Anderson-Darling Test Statistic	0.47	95% Bootstrap-t UCL 10.45
Anderson-Darling 5% Critical Value	0.728	95% Hall's Bootstrap UCL 10.3
Kolmogorov-Smirnov Test Statistic	0.206	95% Percentile Bootstrap UCL 10.28
Kolmogorov-Smirnov 5% Critical Value	0.267	95% BCA Bootstrap UCL 10.23
Data appear Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL 13.34
		97.5% Chebyshev(Mean, Sd) UCL 15.32
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL 19.21
95% Approximate Gamma UCL	11.45	
95% Adjusted Gamma UCL	12.01	

Potential UCL to Use			
		Use 95% Student's-t UCL	10.68

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

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Sediment - Schoolhouse Brook

Copper

General Statistics

Number of Valid Observations	10	Number of Distinct Observations	10

Raw Statistics		Log-transformed Statistics	
Minimum	160	Minimum of Log Data	5.075
Maximum	1390	Maximum of Log Data	7.237
Mean	338.8	Mean of log Data	5.562
Median	225	SD of log Data	0.632
SD	372.9		
Coefficient of Variation	1.101		
Skewness	3.051		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Shapiro Wilk Test Statistic	0.493	Shapiro Wilk Test Statistic	0.705
Shapiro Wilk Critical Value	0.842	Shapiro Wilk Critical Value	0.842
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	555	95% H-UCL	529.1
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	588.6
95% Adjusted-CLT UCL (Chen-1995)	654.4	97.5% Chebyshev (MVUE) UCL	708.8
95% Modified-t UCL (Johnson-1978)	573.9	99% Chebyshev (MVUE) UCL	944.9

Gamma Distribution Test		Data Distribution	
k star (bias corrected)	1.502	Data do not follow a Discernable Distribution (0.05)	
Theta Star	225.5		
MLE of Mean	338.8		
MLE of Standard Deviation	276.4		
nu star	30.05		
Approximate Chi Square Value (.05)	18.53	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0267	95% CLT UCL	532.8
Adjusted Chi Square Value	16.97	95% Jackknife UCL	555
		95% Standard Bootstrap UCL	521.4
Anderson-Darling Test Statistic	1.613	95% Bootstrap-t UCL	1501
Anderson-Darling 5% Critical Value	0.735	95% Hall's Bootstrap UCL	1384
Kolmogorov-Smirnov Test Statistic	0.374	95% Percentile Bootstrap UCL	570.4
Kolmogorov-Smirnov 5% Critical Value	0.27	95% BCA Bootstrap UCL	689.3
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	852.8
		97.5% Chebyshev(Mean, Sd) UCL	1075
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	1512
95% Approximate Gamma UCL	549.4		
95% Adjusted Gamma UCL	599.7		

Potential UCL to Use			
		Use 95% Chebyshev (Mean, Sd) UCL	852.8

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

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Sediment - Schoolhouse Brook

Iron

General Statistics

Number of Valid Observations	10	Number of Distinct Observations	10

Raw Statistics		Log-transformed Statistics	
Minimum	8760	Minimum of Log Data	9.078
Maximum	58800	Maximum of Log Data	10.98
Mean	22836	Mean of log Data	9.899
Median	20650	SD of log Data	0.533
SD	14134		
Coefficient of Variation	0.619		
Skewness	2.034		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Shapiro Wilk Test Statistic	0.794	Shapiro Wilk Test Statistic	0.964
Shapiro Wilk Critical Value	0.842	Shapiro Wilk Critical Value	0.842
Data not Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	31029	95% H-UCL	34345
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	39539
95% Adjusted-CLT UCL (Chen-1995)	33260	97.5% Chebyshev (MVUE) UCL	46868
95% Modified-t UCL (Johnson-1978)	31509	99% Chebyshev (MVUE) UCL	61266

Gamma Distribution Test		Data Distribution	
k star (bias corrected)	2.736	Data appear Gamma Distributed at 5% Significance Level	
Theta Star	8346		
MLE of Mean	22836		
MLE of Standard Deviation	13806		
nu star	54.72		
Approximate Chi Square Value (.05)	38.72	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0267	95% CLT UCL	30188
Adjusted Chi Square Value	36.4	95% Jackknife UCL	31029
		95% Standard Bootstrap UCL	29818
Anderson-Darling Test Statistic	0.34	95% Bootstrap-t UCL	37183
Anderson-Darling 5% Critical Value	0.73	95% Hall's Bootstrap UCL	62725
Kolmogorov-Smirnov Test Statistic	0.186	95% Percentile Bootstrap UCL	30426
Kolmogorov-Smirnov 5% Critical Value	0.268	95% BCA Bootstrap UCL	33530
Data appear Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	42319
		97.5% Chebyshev(Mean, Sd) UCL	50749
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	67309
95% Approximate Gamma UCL	32270		
95% Adjusted Gamma UCL	34334		

Potential UCL to Use		Use 95% Approximate Gamma UCL	32270

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Schoolhouse Brook

Lithium

General Statistics

Number of Valid Observations	5	Number of Distinct Observations	4

Raw Statistics

Minimum	18
Maximum	24
Mean	21.4
Median	22
SD	2.793
Coefficient of Variation	0.131
Skewness	-0.34

Log-transformed Statistics

Minimum of Log Data	2.89
Maximum of Log Data	3.178
Mean of log Data	3.056
SD of log Data	0.133

Warning: There are only 4 Distinct Values in this data
There are insufficient Distinct Values to perform some GOF tests and bootstrap methods.
Those methods will return a 'N/A' value on your output display!

It is necessary to have 4 or more Distinct Values to compute bootstrap methods.
However, results obtained using 4 to 9 distinct values may not be reliable.
It is recommended to have 10-15 or more observations for accurate and meaningful bootstrap results.

Warning: A sample size of 'n' = 5 may not adequate enough to compute meaningful and reliable test statistics and estimates!

It is suggested to collect at least 8 to 10 observations using these statistical methods!
If possible compute and collect Data Quality Objectives (DQO) based sample size and analytical results.

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.865
Shapiro Wilk Critical Value	0.762

Data appear Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.864
Shapiro Wilk Critical Value	0.762

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	24.06

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995)	23.25
95% Modified-t UCL (Johnson-1978)	24.03

Assuming Lognormal Distribution

95% H-UCL	24.63
95% Chebyshev (MVUE) UCL	26.95
97.5% Chebyshev (MVUE) UCL	29.35
99% Chebyshev (MVUE) UCL	34.07

Gamma Distribution Test

k star (bias corrected)	28.78
Theta Star	0.744
MLE of Mean	21.4
MLE of Standard Deviation	3.989
nu star	287.8
Approximate Chi Square Value (.05)	249.5
Adjusted Level of Significance	0.0086
Adjusted Chi Square Value	233.7
Anderson-Darling Test Statistic	0.443
Anderson-Darling 5% Critical Value	0.678
Kolmogorov-Smirnov Test Statistic	0.248
Kolmogorov-Smirnov 5% Critical Value	0.357

Data appear Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	24.68
95% Adjusted Gamma UCL	26.35

Data Distribution

Data appear Normal at 5% Significance Level

Nonparametric Statistics

95% CLT UCL	23.45
95% Jackknife UCL	24.06
95% Standard Bootstrap UCL	23.15
95% Bootstrap-t UCL	23.58
95% Hall's Bootstrap UCL	22.73
95% Percentile Bootstrap UCL	23.2
95% BCA Bootstrap UCL	22.8
95% Chebyshev(Mean, Sd) UCL	26.84
97.5% Chebyshev(Mean, Sd) UCL	29.2
99% Chebyshev(Mean, Sd) UCL	33.83

Potential UCL to Use Use 95% Student's-t UCL 24.06

Recommended UCL exceeds the maximum observation

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Schoolhouse Brook

Manganese

General Statistics

Number of Valid Observations	10	Number of Distinct Observations	10

Raw Statistics

Minimum	200
Maximum	1120
Mean	544.7
Median	422
SD	335.5
Coefficient of Variation	0.616
Skewness	0.534

Log-transformed Statistics

Minimum of Log Data	5.298
Maximum of Log Data	7.021
Mean of log Data	6.116
SD of log Data	0.651

Relevant UCL Statistics

Normal Distribution Test

Shapiro Wilk Test Statistic	0.873
Shapiro Wilk Critical Value	0.842

Data appear Normal at 5% Significance Level

Lognormal Distribution Test

Shapiro Wilk Test Statistic	0.897
Shapiro Wilk Critical Value	0.842

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	739.2

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995)	738.3
95% Modified-t UCL (Johnson-1978)	742.1

Assuming Lognormal Distribution

95% H-UCL	952.5
95% Chebyshev (MVUE) UCL	1050
97.5% Chebyshev (MVUE) UCL	1268
99% Chebyshev (MVUE) UCL	1696

Gamma Distribution Test

k star (bias corrected)	2.076
Theta Star	262.4
MLE of Mean	544.7
MLE of Standard Deviation	378.1
nu star	41.52
Approximate Chi Square Value (.05)	27.75
Adjusted Level of Significance	0.0267
Adjusted Chi Square Value	25.81
Anderson-Darling Test Statistic	0.532
Anderson-Darling 5% Critical Value	0.733
Kolmogorov-Smirnov Test Statistic	0.204
Kolmogorov-Smirnov 5% Critical Value	0.269

Data appear Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	815
95% Adjusted Gamma UCL	876.3

Data Distribution

Data appear Normal at 5% Significance Level

Nonparametric Statistics

95% CLT UCL	719.2
95% Jackknife UCL	739.2
95% Standard Bootstrap UCL	710.2
95% Bootstrap-t UCL	773.9
95% Hall's Bootstrap UCL	701.7
95% Percentile Bootstrap UCL	715.9
95% BCA Bootstrap UCL	729.7
95% Chebyshev(Mean, Sd) UCL	1007
97.5% Chebyshev(Mean, Sd) UCL	1207
99% Chebyshev(Mean, Sd) UCL	1600

Potential UCL to Use

Use 95% Student's-t UCL 739.2

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Sediment - Schoolhouse Brook

Vanadium

General Statistics

Number of Valid Observations	10	Number of Distinct Observations	10

Raw Statistics		Log-transformed Statistics	
Minimum	9.8	Minimum of Log Data	2.282
Maximum	62	Maximum of Log Data	4.127
Mean	33.17	Mean of log Data	3.305
Median	33	SD of log Data	0.697
SD	19.68		
Coefficient of Variation	0.593		
Skewness	0.103		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Shapiro Wilk Test Statistic	0.878	Shapiro Wilk Test Statistic	0.881
Shapiro Wilk Critical Value	0.842	Shapiro Wilk Critical Value	0.842
Data appear Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	44.58	95% H-UCL	62.42
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	67.14
95% Adjusted-CLT UCL (Chen-1995)	43.62	97.5% Chebyshev (MVUE) UCL	81.59
95% Modified-t UCL (Johnson-1978)	44.61	99% Chebyshev (MVUE) UCL	110

Gamma Distribution Test		Data Distribution	
k star (bias corrected)	1.952	Data appear Normal at 5% Significance Level	
Theta Star	16.99		
MLE of Mean	33.17		
MLE of Standard Deviation	23.74		
nu star	39.04		
Approximate Chi Square Value (.05)	25.73	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0267	95% CLT UCL	43.41
Adjusted Chi Square Value	23.86	95% Jackknife UCL	44.58
		95% Standard Bootstrap UCL	43.06
Anderson-Darling Test Statistic	0.601	95% Bootstrap-t UCL	44.98
Anderson-Darling 5% Critical Value	0.733	95% Hall's Bootstrap UCL	42.09
Kolmogorov-Smirnov Test Statistic	0.241	95% Percentile Bootstrap UCL	42.9
Kolmogorov-Smirnov 5% Critical Value	0.269	95% BCA Bootstrap UCL	42.48
Data appear Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	60.29
		97.5% Chebyshev(Mean, Sd) UCL	72.03
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	95.09
95% Approximate Gamma UCL	50.33		
95% Adjusted Gamma UCL	54.26		

Potential UCL to Use			
		Use 95% Student's-t UCL	44.58

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Ely Brook

ALUMINUM

General Statistics

Number of Valid Data	92	Number of Detected Data	78
Number of Distinct Detected Data	77	Number of Non-Detect Data	14
		Percent Non-Detects	15.22%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	18	Minimum Detected	2.89
Maximum Detected	103590	Maximum Detected	11.55
Mean of Detected	8321	Mean of Detected	7.82
SD of Detected	13533	SD of Detected	2.005
Minimum Non-Detect	200	Minimum Non-Detect	5.298
Maximum Non-Detect	349	Maximum Non-Detect	5.855

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	24
Number treated as Detected	68
Single DL Non-Detect Percentage	26.09%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Lilliefors Test Statistic	0.27	Lilliefors Test Statistic	0.131
5% Lilliefors Critical Value	0.1	5% Lilliefors Critical Value	0.1
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	7072	Mean	7.345
SD	12797	SD	2.162
95% DL/2 (t) UCL	9289	95% H-Stat (DL/2) UCL	36289
Maximum Likelihood Estimate(MLE) Method		Log ROS Method	
Mean	4225	Mean in Log Scale	7.333
SD	15641	SD in Log Scale	2.197
95% MLE (t) UCL	6935	Mean in Original Scale	7074
95% MLE (Tiku) UCL	6979	SD in Original Scale	12795
		95% t UCL	9291
		95% Percentile Bootstrap UCL	9326
		95% BCA Bootstrap UCL	10130

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only
k star (bias corrected)	0.511	Data Follow Appr. Gamma Distribution at 5% Significance Level
Theta Star	16278	
nu star	79.74	
A-D Test Statistic	0.888	
5% A-D Critical Value	0.816	
K-S Test Statistic	0.816	
5% K-S Critical Value	0.107	
Data follow Appr. Gamma Distribution at 5% Significance Level		

Assuming Gamma Distribution		Nonparametric Statistics	
Gamma ROS Statistics using Extrapolated Data		Kaplan-Meier (KM) Method	
Minimum	1E-12	Mean	7061
Maximum	103590	SD	12733
Mean	7055	SE of Mean	1336
Median	2610	95% KM (t) UCL	9281
SD	12806	95% KM (z) UCL	9258
k star	0.126	95% KM (jackknife) UCL	9279
Theta star	55963	95% KM (bootstrap t) UCL	10579
Nu star	23.2	95% KM (BCA) UCL	9474
AppChi2	13.24	95% KM (Percentile Bootstrap) UCL	9366
95% Gamma Approximate UCL	12361	95% KM (Chebyshev) UCL	12885
95% Adjusted Gamma UCL	12474	97.5% KM (Chebyshev) UCL	15405
		99% KM (Chebyshev) UCL	20355
		Potential UCLs to Use	
		95% KM (Chebyshev) UCL	12885

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Ely Brook

ARSENIC

General Statistics

Number of Valid Data	92	Number of Detected Data	9
Number of Distinct Detected Data	5	Number of Non-Detect Data	83
		Percent Non-Detects	90.22%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.09	Minimum Detected	-2.408
Maximum Detected	0.64	Maximum Detected	-0.446
Mean of Detected	0.191	Mean of Detected	-1.972
SD of Detected	0.196	SD of Detected	0.751
Minimum Non-Detect	0.5	Minimum Non-Detect	-0.693
Maximum Non-Detect	10	Maximum Non-Detect	2.303

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	92
Number treated as Detected	0
Single DL Non-Detect Percentage	100.00%

Warning: There are only 9 Detected Values in this data
Note: It should be noted that even though bootstrap may be performed on this data set
the resulting calculations may not be reliable enough to draw conclusions

It is recommended to have 10-15 or more distinct observations for accurate and meaningful results.

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.609	Shapiro Wilk Test Statistic	0.658
5% Shapiro Wilk Critical Value	0.829	5% Shapiro Wilk Critical Value	0.829
Data not Normal at 5% Significance Level		**Data not Lognormal at 5% Significance Level**	
Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	0.5	Mean	-0.846
SD	0.49	SD	0.532
95% DL/2 (t) UCL	0.585	95% H-Stat (DL/2) UCL	0.549
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	-2.047
		SD in Log Scale	0.635
		Mean in Original Scale	0.159
		SD in Original Scale	0.114
		95% t UCL	0.178
		95% Percentile Bootstrap UCL	0.179
		95% BCA Bootstrap UCL	0.179

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	1.225	**Data do not follow a Discernable Distribution (0.05)**	
Theta Star	0.156		
nu star	22.05		
A-D Test Statistic	1.666	**Nonparametric Statistics**	
5% A-D Critical Value	0.732	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.732	Mean	0.167
5% K-S Critical Value	0.283	SD	0.157
Data not Gamma Distributed at 5% Significance Level		SE of Mean	0.0481
		95% KM (t) UCL	0.246
Assuming Gamma Distribution		95% KM (z) UCL	0.246
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	0.249
Minimum	0.09	95% KM (bootstrap t) UCL	1.015
Maximum	0.64	95% KM (BCA) UCL	0.259
Mean	0.187	95% KM (Percentile Bootstrap) UCL	0.246
Median	0.183	95% KM (Chebyshev) UCL	0.376
SD	0.0618	97.5% KM (Chebyshev) UCL	0.467
k star	13.21	99% KM (Chebyshev) UCL	0.645
Theta star	0.0141		
Nu star	2430	**Potential UCLs to Use**	
AppChi2	2317	95% KM (t) UCL	0.246
95% Gamma Approximate UCL	0.196	95% KM (% Bootstrap) UCL	0.246
95% Adjusted Gamma UCL	0.196		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Ely Brook

CADMIUM

General Statistics

Number of Valid Data	92	Number of Detected Data	74
Number of Distinct Detected Data	54	Number of Non-Detect Data	18
		Percent Non-Detects	19.57%

Raw Statistics

Minimum Detected	0.02
Maximum Detected	55.8
Mean of Detected	4.076
SD of Detected	7.059
Minimum Non-Detect	0.02
Maximum Non-Detect	0.5

Log-transformed Statistics

Minimum Detected	-3.912
Maximum Detected	4.022
Mean of Detected	0.528
SD of Detected	1.51
Minimum Non-Detect	-3.912
Maximum Non-Detect	-0.693

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	30
Number treated as Detected	62
Single DL Non-Detect Percentage	32.61%

UCL Statistics

Normal Distribution Test with Detected Values Only

Lilliefors Test Statistic	0.283
5% Lilliefors Critical Value	0.103

Data not Normal at 5% Significance Level

Lognormal Distribution Test with Detected Values Only

Lilliefors Test Statistic	0.124
5% Lilliefors Critical Value	0.103

Data not Lognormal at 5% Significance Level

Assuming Normal Distribution

DL/2 Substitution Method	
Mean	3.316
SD	6.51
95% DL/2 (t) UCL	4.444
Maximum Likelihood Estimate(MLE) Method	
Mean	1.33
SD	8.386
95% MLE (t) UCL	2.783
95% MLE (Tiku) UCL	2.869

Assuming Lognormal Distribution

DL/2 Substitution Method	
Mean	0.0288
SD	1.773
95% H-Stat (DL/2) UCL	8.839
Log ROS Method	
Mean in Log Scale	-0.0118
SD in Log Scale	1.785
Mean in Original Scale	3.307
SD in Original Scale	6.514
95% t UCL	4.436
95% Percentile Bootstrap UCL	4.567
95% BCA Bootstrap UCL	5.045

Gamma Distribution Test with Detected Values Only

k star (bias corrected)	0.672
Theta Star	6.069
nu star	99.39
A-D Test Statistic	1.457
5% A-D Critical Value	0.799
K-S Test Statistic	0.799
5% K-S Critical Value	0.108

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data	
Minimum	1E-12
Maximum	55.8
Mean	3.318
Median	1.35
SD	6.511
k star	0.156
Theta star	21.3
Nu star	28.66
AppChi2	17.44
95% Gamma Approximate UCL	5.452
95% Adjusted Gamma UCL	5.496

Note: DL/2 is not a recommended method.

Data Distribution Test with Detected Values Only

Data do not follow a Discernable Distribution (0.05)

Nonparametric Statistics

Kaplan-Meier (KM) Method	
Mean	3.301
SD	6.482
SE of Mean	0.68
95% KM (t) UCL	4.432
95% KM (z) UCL	4.42
95% KM (jackknife) UCL	4.43
95% KM (bootstrap t) UCL	5.288
95% KM (BCA) UCL	4.592
95% KM (Percentile Bootstrap) UCL	4.485
95% KM (Chebyshev) UCL	6.267
97.5% KM (Chebyshev) UCL	7.55
99% KM (Chebyshev) UCL	10.07

Potential UCLs to Use

97.5% KM (Chebyshev) UCL 7.55

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Ely Brook

CHROMIUM

General Statistics

Number of Valid Data	92	Number of Detected Data	49
Number of Distinct Detected Data	39	Number of Non-Detect Data	43
		Percent Non-Detects	46.74%

Raw Statistics

Minimum Detected	0.1
Maximum Detected	125
Mean of Detected	6.587
SD of Detected	17.74
Minimum Non-Detect	0.16
Maximum Non-Detect	10

Log-transformed Statistics

Minimum Detected	-2.303
Maximum Detected	4.828
Mean of Detected	0.956
SD of Detected	1.301
Minimum Non-Detect	-1.833
Maximum Non-Detect	2.303

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	84
Number treated as Detected	8
Single DL Non-Detect Percentage	91.30%

UCL Statistics

Normal Distribution Test with Detected Values Only

Shapiro Wilk Test Statistic	0.308
5% Shapiro Wilk Critical Value	0.947

Data not Normal at 5% Significance Level

Lognormal Distribution Test with Detected Values Only

Shapiro Wilk Test Statistic	0.973
5% Shapiro Wilk Critical Value	0.947

Data appear Lognormal at 5% Significance Level

Assuming Normal Distribution

DL/2 Substitution Method	
Mean	5.294
SD	13.03
95% DL/2 (t) UCL	7.551
Maximum Likelihood Estimate(MLE) Method	N/A

MLE yields a negative mean

Assuming Lognormal Distribution

DL/2 Substitution Method	
Mean	0.93
SD	1.287
95% H-Stat (DL/2) UCL	8.177
Log ROS Method	
Mean in Log Scale	0.423
SD in Log Scale	1.377
Mean in Original Scale	4.272
SD in Original Scale	13.19
95% t UCL	6.556
95% Percentile Bootstrap UCL	6.91
95% BCA Bootstrap UCL	9.656

Gamma Distribution Test with Detected Values Only

k star (bias corrected)	0.63
Theta Star	10.46
nu star	61.7
A-D Test Statistic	1.983
5% A-D Critical Value	0.801
K-S Test Statistic	0.801
5% K-S Critical Value	0.132

Data not Gamma Distributed at 5% Significance Level

Assuming Gamma Distribution

Gamma ROS Statistics using Extrapolated Data	
Minimum	1E-12
Maximum	125
Mean	6.406
Median	2.6
SD	14
k star	0.124
Theta star	51.47
Nu star	22.9
AppChi2	13.01
95% Gamma Approximate UCL	11.27
95% Adjusted Gamma UCL	11.37

Note: DL/2 is not a recommended method.

Data Distribution Test with Detected Values Only

Data appear Lognormal at 5% Significance Level

Nonparametric Statistics

Kaplan-Meier (KM) Method	
Mean	4.289
SD	13.11
SE of Mean	1.39
95% KM (t) UCL	6.598
95% KM (z) UCL	6.575
95% KM (jackknife) UCL	6.587
95% KM (bootstrap t) UCL	11.26
95% KM (BCA) UCL	7.044
95% KM (Percentile Bootstrap) UCL	6.93
95% KM (Chebyshev) UCL	10.35
97.5% KM (Chebyshev) UCL	12.97
99% KM (Chebyshev) UCL	18.12

Potential UCLs to Use

95% KM (Chebyshev) UCL 10.35

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Ely Brook

COBALT

General Statistics

Number of Valid Data	92	Number of Detected Data	84
Number of Distinct Detected Data	79	Number of Non-Detect Data	8
		Percent Non-Detects	8.70%

Raw Statistics		**Log-transformed Statistics**	
Minimum Detected	0.16	Minimum Detected	-1.833
Maximum Detected	1174	Maximum Detected	7.068
Mean of Detected	92.33	Mean of Detected	3.174
SD of Detected	153.4	SD of Detected	2.194
Minimum Non-Detect	50	Minimum Non-Detect	3.912
Maximum Non-Detect	50	Maximum Non-Detect	3.912

UCL Statistics

Normal Distribution Test with Detected Values Only		**Lognormal Distribution Test with Detected Values Only**	
Lilliefors Test Statistic	0.274	Lilliefors Test Statistic	0.17
5% Lilliefors Critical Value	0.0967	5% Lilliefors Critical Value	0.0967
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	86.48	Mean	3.178
SD	147.7	SD	2.095
95% DL/2 (t) UCL	112.1	95% H-Stat (DL/2) UCL	466.5
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE yields a negative mean		Mean in Log Scale	3.057
		SD in Log Scale	2.154
		Mean in Original Scale	85.19
		SD in Original Scale	148.3
		95% t UCL	110.9
		95% Percentile Bootstrap UCL	112.5
		95% BCA Bootstrap UCL	125.6

Gamma Distribution Test with Detected Values Only		**Data Distribution Test with Detected Values Only**
k star (bias corrected)	0.465	Data Follow Appr. Gamma Distribution at 5% Significance Level
Theta Star	198.6	
nu star	78.12	
A-D Test Statistic	0.854	**Nonparametric Statistics**
5% A-D Critical Value	0.825	Kaplan-Meier (KM) Method
K-S Test Statistic	0.825	Mean 85.85
5% K-S Critical Value	0.103	SD 147.3
Data follow Appr. Gamma Distribution at 5% Significance Level		SE of Mean 15.45
		95% KM (t) UCL 111.5
Assuming Gamma Distribution		95% KM (z) UCL 111.3
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL 111.5
Minimum	1E-12	95% KM (bootstrap t) UCL 123.7
Maximum	1174	95% KM (BCA) UCL 115
Mean	86.74	95% KM (Percentile Bootstrap) UCL 114.1
Median	34.65	95% KM (Chebyshev) UCL 153.2
SD	148	97.5% KM (Chebyshev) UCL 182.4
k star	0.268	99% KM (Chebyshev) UCL 239.6
Theta star	323.3	
Nu star	49.36	**Potential UCLs to Use**
AppChi2	34.23	95% KM (Chebyshev) UCL 153.2
95% Gamma Approximate UCL	125.1	
95% Adjusted Gamma UCL	125.8	

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Ely Brook

COPPER

General Statistics

Number of Valid Observations	92	Number of Distinct Observations	86

Raw Statistics

Minimum	1.3
Maximum	33178
Mean	3230
Median	1298
SD	5031
Coefficient of Variation	1.558
Skewness	3.074

Log-transformed Statistics

Minimum of Log Data	0.262
Maximum of Log Data	10.41
Mean of log Data	6.363
SD of log Data	2.603

Relevant UCL Statistics

Normal Distribution Test

Lilliefors Test Statistic	0.282
Lilliefors Critical Value	0.0924

Data not Normal at 5% Significance Level

Lognormal Distribution Test

Lilliefors Test Statistic	0.193
Lilliefors Critical Value	0.0924

Data not Lognormal at 5% Significance Level

Assuming Normal Distribution

95% Student's-t UCL	4101

95% UCLs (Adjusted for Skewness)

95% Adjusted-CLT UCL (Chen-1995)	4272
95% Modified-t UCL (Johnson-1978)	4129

Assuming Lognormal Distribution

95% H-UCL	53796
95% Chebyshev (MVUE) UCL	45406
97.5% Chebyshev (MVUE) UCL	58644
99% Chebyshev (MVUE) UCL	84647

Gamma Distribution Test

k star (bias corrected)	0.38
Theta Star	8494
MLE of Mean	3230
MLE of Standard Deviation	5238
nu star	69.96
Approximate Chi Square Value (.05)	51.71
Adjusted Level of Significance	0.0474
Adjusted Chi Square Value	51.46
Anderson-Darling Test Statistic	1.073
Anderson-Darling 5% Critical Value	0.847
Kolmogorov-Smirnov Test Statistic	0.0956
Kolmogorov-Smirnov 5% Critical Value	0.1

Data follow Appr. Gamma Distribution at 5% Significance Level

Assuming Gamma Distribution

95% Approximate Gamma UCL	4370
95% Adjusted Gamma UCL	4391

Data Distribution

Data Follow Appr. Gamma Distribution at 5% Significance Level

Nonparametric Statistics

95% CLT UCL	4092
95% Jackknife UCL	4101
95% Standard Bootstrap UCL	4086
95% Bootstrap-t UCL	4306
95% Hall's Bootstrap UCL	4508
95% Percentile Bootstrap UCL	4142
95% BCA Bootstrap UCL	4286
95% Chebyshev(Mean, Sd) UCL	5516
97.5% Chebyshev(Mean, Sd) UCL	6505
99% Chebyshev(Mean, Sd) UCL	8448

Potential UCL to Use

Use 95% Adjusted Gamma UCL 4391

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Ely Brook

IRON

General Statistics

Number of Valid Data	92	Number of Detected Data	83
Number of Distinct Detected Data	79	Number of Non-Detect Data	9
		Percent Non-Detects	9.78%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	19.3	Minimum Detected	2.96
Maximum Detected	28300	Maximum Detected	10.25
Mean of Detected	3083	Mean of Detected	6.496
SD of Detected	5690	SD of Detected	2.006
Minimum Non-Detect	50	Minimum Non-Detect	3.912
Maximum Non-Detect	100	Maximum Non-Detect	4.605

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	29
Number treated as Detected	63
Single DL Non-Detect Percentage	31.52%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Lilliefors Test Statistic	0.295	Lilliefors Test Statistic	0.105
5% Lilliefors Critical Value	0.0973	5% Lilliefors Critical Value	0.0973
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	2786	Mean	6.236
SD	5477	SD	2.065
95% DL/2 (t) UCL	3734	95% H-Stat (DL/2) UCL	9128
Maximum Likelihood Estimate(MLE) Method		Log ROS Method	
Mean	1142	Mean in Log Scale	6.239
SD	7053	SD in Log Scale	2.075
95% MLE (t) UCL	2364	Mean in Original Scale	2787
95% MLE (Tiku) UCL	2427	SD in Original Scale	5476
		95% t UCL	3736
		95% Percentile Bootstrap UCL	3763
		95% BCA Bootstrap UCL	3999

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	0.417	Data do not follow a Discernable Distribution (0.05)	
Theta Star	7398		
nu star	69.17		
A-D Test Statistic	1.751	**Nonparametric Statistics**	
5% A-D Critical Value	0.837	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.837	Mean	2786
5% K-S Critical Value	0.105	SD	5447
Data not Gamma Distributed at 5% Significance Level		SE of Mean	571.3
		95% KM (t) UCL	3735
Assuming Gamma Distribution		95% KM (z) UCL	3725
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	3734
Minimum	1E-12	95% KM (bootstrap t) UCL	4014
Maximum	28300	95% KM (BCA) UCL	3798
Mean	2781	95% KM (Percentile Bootstrap) UCL	3751
Median	505.3	95% KM (Chebyshev) UCL	5276
SD	5479	97.5% KM (Chebyshev) UCL	6354
k star	0.162	99% KM (Chebyshev) UCL	8470
Theta star	17208		
Nu star	29.74	**Potential UCLs to Use**	
AppChi2	18.29	97.5% KM (Chebyshev) UCL	6354
95% Gamma Approximate UCL	4522		
95% Adjusted Gamma UCL	4558		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Ely Brook

Lithium

General Statistics

Number of Valid Data	10	Number of Detected Data	8
Number of Distinct Detected Data	8	Number of Non-Detect Data	2
		Percent Non-Detects	20.00%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.1	Minimum Detected	-2.303
Maximum Detected	15.1	Maximum Detected	2.715
Mean of Detected	4.35	Mean of Detected	0.63
SD of Detected	5.001	SD of Detected	1.68
Minimum Non-Detect	0.1	Minimum Non-Detect	-2.303
Maximum Non-Detect	0.1	Maximum Non-Detect	-2.303

Warning: There are only 8 Detected Values in this data
Note: It should be noted that even though bootstrap may be performed on this data set
the resulting calculations may not be reliable enough to draw conclusions

It is recommended to have 10-15 or more distinct observations for accurate and meaningful results.

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.83	Shapiro Wilk Test Statistic	0.946
5% Shapiro Wilk Critical Value	0.818	5% Shapiro Wilk Critical Value	0.818
Data appear Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	3.49	Mean	-0.0949
SD	4.769	SD	2.129
95% DL/2 (t) UCL	6.254	95% H-Stat (DL/2) UCL	504.1
Maximum Likelihood Estimate(MLE) Method		Log ROS Method	
Mean	2.82	Mean in Log Scale	-0.174
SD	5.328	SD in Log Scale	2.264
95% MLE (t) UCL	5.908	Mean in Original Scale	3.488
95% MLE (Tiku) UCL	5.898	SD in Original Scale	4.771
		95% t UCL	6.253
		95% Percentile Bootstrap UCL	6.03
		95% BCA Bootstrap UCL	6.992

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	0.532	Data appear Normal at 5% Significance Level	
Theta Star	8.184		
nu star	8.505		
A-D Test Statistic	0.137	**Nonparametric Statistics**	
5% A-D Critical Value	0.747	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.747	Mean	3.5
5% K-S Critical Value	0.305	SD	4.516
Data appear Gamma Distributed at 5% Significance Level		SE of Mean	1.527
		95% KM (t) UCL	6.299
Assuming Gamma Distribution		95% KM (z) UCL	6.011
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	6.239
Minimum	1E-12	95% KM (bootstrap t) UCL	9.007
Maximum	15.1	95% KM (BCA) UCL	6.36
Mean	3.48	95% KM (Percentile Bootstrap) UCL	6.07
Median	1.75	95% KM (Chebyshev) UCL	10.16
SD	4.777	97.5% KM (Chebyshev) UCL	13.04
k star	0.155	99% KM (Chebyshev) UCL	18.69
Theta star	22.5		
Nu star	3.093	**Potential UCLs to Use**	
AppChi2	0.401	95% KM (t) UCL	6.299
95% Gamma Approximate UCL	26.87	95% KM (Percentile Bootstrap) UCL	6.07
95% Adjusted Gamma UCL	39.1		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Ely Brook

MANGANESE

General Statistics

Number of Valid Observations	92	Number of Distinct Observations	85

Raw Statistics		Log-transformed Statistics	
Minimum	2.4	Minimum of Log Data	0.875
Maximum	5230	Maximum of Log Data	8.562
Mean	613.7	Mean of log Data	5.423
Median	271.5	SD of log Data	1.755
SD	795.3		
Coefficient of Variation	1.296		
Skewness	2.648		

Relevant UCL Statistics

Normal Distribution Test		Lognormal Distribution Test	
Lilliefors Test Statistic	0.229	Lilliefors Test Statistic	0.123
Lilliefors Critical Value	0.0924	Lilliefors Critical Value	0.0924
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		Assuming Lognormal Distribution	
95% Student's-t UCL	751.5	95% H-UCL	1867
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	2212
95% Adjusted-CLT UCL (Chen-1995)	774.5	97.5% Chebyshev (MVUE) UCL	2730
95% Modified-t UCL (Johnson-1978)	755.3	99% Chebyshev (MVUE) UCL	3747

Gamma Distribution Test		Data Distribution	
k star (bias corrected)	0.605	Data Follow Appr. Gamma Distribution at 5% Significance Level	
Theta Star	1015		
MLE of Mean	613.7		
MLE of Standard Deviation	789.2		
nu star	111.3		
Approximate Chi Square Value (.05)	87.91	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0474	95% CLT UCL	750
Adjusted Chi Square Value	87.58	95% Jackknife UCL	751.5
		95% Standard Bootstrap UCL	750.1
Anderson-Darling Test Statistic	0.886	95% Bootstrap-t UCL	779.6
Anderson-Darling 5% Critical Value	0.807	95% Hall's Bootstrap UCL	803.4
Kolmogorov-Smirnov Test Statistic	0.0899	95% Percentile Bootstrap UCL	750.4
Kolmogorov-Smirnov 5% Critical Value	0.0978	95% BCA Bootstrap UCL	771.5
Data follow Appr. Gamma Distribution at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	975.1
		97.5% Chebyshev(Mean, Sd) UCL	1131
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	1439
95% Approximate Gamma UCL	776.6		
95% Adjusted Gamma UCL	779.6		

Potential UCL to Use		Use 95% Approximate Gamma UCL	776.6

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Ely Brook

MERCURY

General Statistics

Number of Valid Data	76	Number of Detected Data	1
Number of Distinct Detected Data	1	Number of Non-Detect Data	75
		Percent Non-Detects	98.68%

Warning: Only one distinct data value was detected! ProUCL (or any other software) should not be used on such a data set!
It is suggested to use alternative site specific values determined by the Project Team to estimate environmental parameters (e.g., EPC, BTV).

The data set for variable MERCURY was not processed!

Appendix D
Surface Water - Ely Brook

NICKEL

General Statistics

Number of Valid Data	76	Number of Detected Data	69
Number of Distinct Detected Data	63	Number of Non-Detect Data	7
		Percent Non-Detects	9.21%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.4	Minimum Detected	-0.916
Maximum Detected	308	Maximum Detected	5.73
Mean of Detected	27.22	Mean of Detected	2.248
SD of Detected	45.98	SD of Detected	1.628
Minimum Non-Detect	0.4	Minimum Non-Detect	-0.916
Maximum Non-Detect	40	Maximum Non-Detect	3.689

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	63
Number treated as Detected	13
Single DL Non-Detect Percentage	82.89%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Lilliefors Test Statistic	0.296	Lilliefors Test Statistic	0.141
5% Lilliefors Critical Value	0.107	5% Lilliefors Critical Value	0.107
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	
Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	26.29	Mean	2.257
SD	43.93	SD	1.627
95% DL/2 (t) UCL	34.69	95% H-Stat (DL/2) UCL	62.5
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE yields a negative mean		Mean in Log Scale	2.15
		SD in Log Scale	1.653
		Mean in Original Scale	25.38
		SD in Original Scale	44.22
		95% t UCL	33.82
		95% Percentile Bootstrap UCL	34.17
		95% BCA Bootstrap UCL	37.14

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	0.571	Data do not follow a Discernable Distribution (0.05)	
Theta Star	47.64		
nu star	78.84		
A-D Test Statistic	1.427	**Nonparametric Statistics**	
5% A-D Critical Value	0.808	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.808	Mean	25.52
5% K-S Critical Value	0.113	SD	43.91
Data not Gamma Distributed at 5% Significance Level		SE of Mean	5.083
		95% KM (t) UCL	33.99
Assuming Gamma Distribution		95% KM (z) UCL	33.88
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	33.98
Minimum	1E-12	95% KM (bootstrap t) UCL	38.04
Maximum	308	95% KM (BCA) UCL	34.56
Mean	26.41	95% KM (Percentile Bootstrap) UCL	34.43
Median	11.85	95% KM (Chebyshev) UCL	47.68
SD	44.28	97.5% KM (Chebyshev) UCL	57.27
k star	0.368	99% KM (Chebyshev) UCL	76.1
Theta star	71.86		
Nu star	55.86	**Potential UCLs to Use**	
AppChi2	39.68	97.5% KM (Chebyshev) UCL	57.27
95% Gamma Approximate UCL	37.18		
95% Adjusted Gamma UCL	37.42		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Ely Brook

VANADIUM

General Statistics

Number of Valid Data	92	Number of Detected Data	62
Number of Distinct Detected Data	47	Number of Non-Detect Data	30
		Percent Non-Detects	32.61%

Raw Statistics		**Log-transformed Statistics**	
Minimum Detected	0.06	Minimum Detected	-2.813
Maximum Detected	11.3	Maximum Detected	2.425
Mean of Detected	1.033	Mean of Detected	-0.607
SD of Detected	1.789	SD of Detected	1.007
Minimum Non-Detect	0.2	Minimum Non-Detect	-1.609
Maximum Non-Detect	5	Maximum Non-Detect	1.609

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	89
Number treated as Detected	3
Single DL Non-Detect Percentage	96.74%

UCL Statistics

Normal Distribution Test with Detected Values Only		**Lognormal Distribution Test with Detected Values Only**	
Lilliefors Test Statistic	0.306	Lilliefors Test Statistic	0.141
5% Lilliefors Critical Value	0.113	5% Lilliefors Critical Value	0.113
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	

Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	1.159	Mean	-0.521
SD	1.618	SD	1.15
95% DL/2 (t) UCL	1.439	95% H-Stat (DL/2) UCL	1.537
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE yields a negative mean		Mean in Log Scale	-0.839
		SD in Log Scale	1.02
		Mean in Original Scale	0.825
		SD in Original Scale	1.512
		95% t UCL	1.087
		95% Percentile Bootstrap UCL	1.105
		95% BCA Bootstrap UCL	1.171

Gamma Distribution Test with Detected Values Only		**Data Distribution Test with Detected Values Only**
k star (bias corrected)	0.879	Data do not follow a Discernable Distribution (0.05)
Theta Star	1.174	
nu star	109	
A-D Test Statistic	4.175	**Nonparametric Statistics**
5% A-D Critical Value	0.784	Kaplan-Meier (KM) Method
K-S Test Statistic	0.784	Mean 0.833
5% K-S Critical Value	0.117	SD 1.519
Data not Gamma Distributed at 5% Significance Level		SE of Mean 0.163
		95% KM (t) UCL 1.104
Assuming Gamma Distribution		95% KM (z) UCL 1.102
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL 1.104
Minimum	1E-12	95% KM (bootstrap t) UCL 1.253
Maximum	11.3	95% KM (BCA) UCL 1.126
Mean	1.003	95% KM (Percentile Bootstrap) UCL 1.127
Median	0.483	95% KM (Chebyshev) UCL 1.545
SD	1.587	97.5% KM (Chebyshev) UCL 1.853
k star	0.2	99% KM (Chebyshev) UCL 2.459
Theta star	5.02	
Nu star	36.75	**Potential UCLs to Use**
AppChi2	23.87	95% KM (Chebyshev) UCL 1.545
95% Gamma Approximate UCL	1.543	
95% Adjusted Gamma UCL	1.554	

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Ely Brook

ZINC

General Statistics

Number of Valid Data	92	Number of Detected Data	88
Number of Distinct Detected Data	85	Number of Non-Detect Data	4
		Percent Non-Detects	4.35%

Raw Statistics		**Log-transformed Statistics**	
Minimum Detected	0.8	Minimum Detected	-0.223
Maximum Detected	8192	Maximum Detected	9.011
Mean of Detected	478.3	Mean of Detected	4.902
SD of Detected	960.5	SD of Detected	1.932
Minimum Non-Detect	60	Minimum Non-Detect	4.094
Maximum Non-Detect	60	Maximum Non-Detect	4.094

UCL Statistics

Normal Distribution Test with Detected Values Only		**Lognormal Distribution Test with Detected Values Only**	
Lilliefors Test Statistic	0.31	Lilliefors Test Statistic	0.169
5% Lilliefors Critical Value	0.0944	5% Lilliefors Critical Value	0.0944
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	
Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	458.8	Mean	4.837
SD	943.6	SD	1.914
95% DL/2 (t) UCL	622.3	95% H-Stat (DL/2) UCL	1524
Maximum Likelihood Estimate(MLE) Method		Log ROS Method	
Mean	153.1	Mean in Log Scale	4.811
SD	1229	SD in Log Scale	1.942
95% MLE (t) UCL	366	Mean in Original Scale	458.4
95% MLE (Tiku) UCL	380.5	SD in Original Scale	943.9
		95% t UCL	621.9
		95% Percentile Bootstrap UCL	634.2
		95% BCA Bootstrap UCL	755.6

Gamma Distribution Test with Detected Values Only		**Data Distribution Test with Detected Values Only**	
k star (bias corrected)	0.491	Data do not follow a Discernable Distribution (0.05)	
Theta Star	973.4		
nu star	86.48		
A-D Test Statistic	1.062	**Nonparametric Statistics**	
5% A-D Critical Value	0.818	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.818	Mean	458.3
5% K-S Critical Value	0.101	SD	938.7
Data not Gamma Distributed at 5% Significance Level		SE of Mean	98.43
		95% KM (t) UCL	621.9
Assuming Gamma Distribution		95% KM (z) UCL	620.2
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	621.8
Minimum	1E-12	95% KM (bootstrap t) UCL	764.4
Maximum	8192	95% KM (BCA) UCL	658.2
Mean	457.5	95% KM (Percentile Bootstrap) UCL	630.1
Median	184.5	95% KM (Chebyshev) UCL	887.4
SD	944.3	97.5% KM (Chebyshev) UCL	1073
k star	0.265	99% KM (Chebyshev) UCL	1438
Theta star	1726		
Nu star	48.77	**Potential UCLs to Use**	
AppChi2	33.74	97.5% KM (Chebyshev) UCL	1073
95% Gamma Approximate UCL	661.3		
95% Adjusted Gamma UCL	665.2		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Schoolhouse Brook

CHROMIUM

General Statistics

Number of Valid Data	22	Number of Detected Data	12
Number of Distinct Detected Data	12	Number of Non-Detect Data	10
		Percent Non-Detects	45.45%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.44	Minimum Detected	-0.821
Maximum Detected	4.5	Maximum Detected	1.504
Mean of Detected	2.654	Mean of Detected	0.78
SD of Detected	1.416	SD of Detected	0.738
Minimum Non-Detect	0.39	Minimum Non-Detect	-0.942
Maximum Non-Detect	10	Maximum Non-Detect	2.303

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

		Number treated as Non-Detect	22
		Number treated as Detected	0
		Single DL Non-Detect Percentage	100.00%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.934	Shapiro Wilk Test Statistic	0.88
5% Shapiro Wilk Critical Value	0.859	5% Shapiro Wilk Critical Value	0.859
Data appear Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	
Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	2.479	Mean	0.486
SD	1.86	SD	1.058
95% DL/2 (t) UCL	3.162	95% H-Stat (DL/2) UCL	5.269
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	0.303
		SD in Log Scale	0.898
		Mean in Original Scale	1.903
		SD in Original Scale	1.448
		95% t UCL	2.434
		95% Percentile Bootstrap UCL	2.416
		95% BCA Bootstrap UCL	2.448

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	2.083	Data appear Normal at 5% Significance Level	
Theta Star	1.274		
nu star	49.99		
A-D Test Statistic	0.441	**Nonparametric Statistics**	
5% A-D Critical Value	0.74	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.74	Mean	1.95
5% K-S Critical Value	0.248	SD	1.492
Data appear Gamma Distributed at 5% Significance Level		SE of Mean	0.369
		95% KM (t) UCL	2.585
Assuming Gamma Distribution		95% KM (z) UCL	2.557
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	2.557
Minimum	0.44	95% KM (bootstrap t) UCL	2.636
Maximum	4.893	95% KM (BCA) UCL	2.762
Mean	2.584	95% KM (Percentile Bootstrap) UCL	2.672
Median	2.561	95% KM (Chebyshev) UCL	3.559
SD	1.342	97.5% KM (Chebyshev) UCL	4.255
k star	2.665	99% KM (Chebyshev) UCL	5.623
Theta star	0.97		
Nu star	117.3	**Potential UCLs to Use**	
AppChi2	93.27	95% KM (t) UCL	2.585
95% Gamma Approximate UCL	3.25	95% KM (Percentile Bootstrap) UCL	2.672
95% Adjusted Gamma UCL	3.306		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Schoolhouse Brook

COBALT

General Statistics

Number of Valid Data	22	Number of Detected Data	18
Number of Distinct Detected Data	17	Number of Non-Detect Data	4
		Percent Non-Detects	18.18%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	1.4	Minimum Detected	0.336
Maximum Detected	25	Maximum Detected	3.219
Mean of Detected	4.216	Mean of Detected	1.151
SD of Detected	5.292	SD of Detected	0.639
Minimum Non-Detect	2	Minimum Non-Detect	0.693
Maximum Non-Detect	50	Maximum Non-Detect	3.912

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

		Number treated as Non-Detect	22
		Number treated as Detected	0
		Single DL Non-Detect Percentage	100.00%

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.434	Shapiro Wilk Test Statistic	0.82
5% Shapiro Wilk Critical Value	0.897	5% Shapiro Wilk Critical Value	0.897
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	
Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	5.847	Mean	1.26
SD	7.862	SD	0.898
95% DL/2 (t) UCL	8.731	95% H-Stat (DL/2) UCL	8.509
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	1.094
		SD in Log Scale	0.609
		Mean in Original Scale	3.899
		SD in Original Scale	4.827
		95% t UCL	5.669
		95% Percentile Bootstrap UCL	5.888
		95% BCA Bootstrap UCL	6.984

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	1.608	Data do not follow a Discernable Distribution (0.05)	
Theta Star	2.622		
nu star	57.88		
A-D Test Statistic	1.821	**Nonparametric Statistics**	
5% A-D Critical Value	0.753	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.753	Mean	3.992
5% K-S Critical Value	0.207	SD	4.93
Data not Gamma Distributed at 5% Significance Level		SE of Mean	1.135
		95% KM (t) UCL	5.945
Assuming Gamma Distribution		95% KM (z) UCL	5.858
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	5.944
Minimum	0.275	95% KM (bootstrap t) UCL	10.99
Maximum	25	95% KM (BCA) UCL	6.517
Mean	4.062	95% KM (Percentile Bootstrap) UCL	6.15
Median	3.27	95% KM (Chebyshev) UCL	8.939
SD	4.87	97.5% KM (Chebyshev) UCL	11.08
k star	1.49	99% KM (Chebyshev) UCL	15.28
Theta star	2.726		
Nu star	65.56	**Potential UCLs to Use**	
AppChi2	47.93	95% KM (Chebyshev) UCL	8.939
95% Gamma Approximate UCL	5.556		
95% Adjusted Gamma UCL	5.689		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Schoolhouse Brook

COPPER

General Statistics

Number of Valid Data	22	Number of Detected Data	20
Number of Distinct Detected Data	19	Number of Non-Detect Data	2
		Percent Non-Detects	9.09%

Raw Statistics		**Log-transformed Statistics**	
Minimum Detected	55	Minimum Detected	4.007
Maximum Detected	1100	Maximum Detected	7.003
Mean of Detected	141.6	Mean of Detected	4.586
SD of Detected	227.6	SD of Detected	0.655
Minimum Non-Detect	2	Minimum Non-Detect	0.693
Maximum Non-Detect	2	Maximum Non-Detect	0.693

UCL Statistics

Normal Distribution Test with Detected Values Only		**Lognormal Distribution Test with Detected Values Only**	
Shapiro Wilk Test Statistic	0.349	Shapiro Wilk Test Statistic	0.706
5% Shapiro Wilk Critical Value	0.905	5% Shapiro Wilk Critical Value	0.905
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	
Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	128.8	Mean	4.169
SD	220.4	SD	1.487
95% DL/2 (t) UCL	209.7	95% H-Stat (DL/2) UCL	573.1
Maximum Likelihood Estimate(MLE) Method		Log ROS Method	
Mean	115.4	Mean in Log Scale	4.466
SD	229.9	SD in Log Scale	0.736
95% MLE (t) UCL	199.8	Mean in Original Scale	131.1
95% MLE (Tiku) UCL	194.2	SD in Original Scale	219.1
		95% t UCL	211.5
		95% Percentile Bootstrap UCL	222.2
		95% BCA Bootstrap UCL	266.9

Gamma Distribution Test with Detected Values Only		**Data Distribution Test with Detected Values Only**	
k star (bias corrected)	1.315	Data do not follow a Discernable Distribution (0.05)	
Theta Star	107.6		
nu star	52.62		
A-D Test Statistic	3.003	**Nonparametric Statistics**	
5% A-D Critical Value	0.758	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.758	Mean	133.7
5% K-S Critical Value	0.197	SD	213
Data not Gamma Distributed at 5% Significance Level		SE of Mean	46.59
		95% KM (t) UCL	213.9
Assuming Gamma Distribution		95% KM (z) UCL	210.3
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	213.7
Minimum	1E-12	95% KM (bootstrap t) UCL	577.4
Maximum	1100	95% KM (BCA) UCL	231.1
Mean	128.7	95% KM (Percentile Bootstrap) UCL	225.3
Median	82.25	95% KM (Chebyshev) UCL	336.8
SD	220.5	97.5% KM (Chebyshev) UCL	424.6
k star	0.225	99% KM (Chebyshev) UCL	597.2
Theta star	571.6		
Nu star	9.907	**Potential UCLs to Use**	
AppChi2	3.884	95% KM (Chebyshev) UCL	336.8
95% Gamma Approximate UCL	328.3		
95% Adjusted Gamma UCL	353.6		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Schoolhouse Brook

MANGANESE

General Statistics

Number of Valid Observations	22	Number of Distinct Observations	21

Raw Statistics		**Log-transformed Statistics**	
Minimum	8	Minimum of Log Data	2.079
Maximum	260	Maximum of Log Data	5.561
Mean	42.9	Mean of log Data	3.462
Median	31.5	SD of log Data	0.693
SD	51.07		
Coefficient of Variation	1.19		
Skewness	3.986		

Relevant UCL Statistics

Normal Distribution Test		**Lognormal Distribution Test**	
Shapiro Wilk Test Statistic	0.496	Shapiro Wilk Test Statistic	0.923
Shapiro Wilk Critical Value	0.911	Shapiro Wilk Critical Value	0.911
Data not Normal at 5% Significance Level		Data appear Lognormal at 5% Significance Level	
Assuming Normal Distribution		**Assuming Lognormal Distribution**	
95% Student's-t UCL	61.64	95% H-UCL	56.46
95% UCLs (Adjusted for Skewness)		95% Chebyshev (MVUE) UCL	67.52
95% Adjusted-CLT UCL (Chen-1995)	70.7	97.5% Chebyshev (MVUE) UCL	79.44
95% Modified-t UCL (Johnson-1978)	63.18	99% Chebyshev (MVUE) UCL	102.8
Gamma Distribution Test		**Data Distribution**	
k star (bias corrected)	1.611	Data appear Lognormal at 5% Significance Level	
Theta Star	26.63		
MLE of Mean	42.9		
MLE of Standard Deviation	33.8		
nu star	70.88		
Approximate Chi Square Value (.05)	52.5	**Nonparametric Statistics**	
Adjusted Level of Significance	0.0386	95% CLT UCL	60.81
Adjusted Chi Square Value	51.32	95% Jackknife UCL	61.64
		95% Standard Bootstrap UCL	60.02
Anderson-Darling Test Statistic	1.341	95% Bootstrap-t UCL	97.76
Anderson-Darling 5% Critical Value	0.757	95% Hall's Bootstrap UCL	134
Kolmogorov-Smirnov Test Statistic	0.219	95% Percentile Bootstrap UCL	62.1
Kolmogorov-Smirnov 5% Critical Value	0.188	95% BCA Bootstrap UCL	74.87
Data not Gamma Distributed at 5% Significance Level		95% Chebyshev(Mean, Sd) UCL	90.36
		97.5% Chebyshev(Mean, Sd) UCL	110.9
Assuming Gamma Distribution		99% Chebyshev(Mean, Sd) UCL	151.2
95% Approximate Gamma UCL	57.93		
95% Adjusted Gamma UCL	59.26		

Potential UCL to Use Use 95% H-UCL 56.46

ProUCL computes and outputs H-statistic based UCLs for historical reasons only.
H-statistic often results in unstable (both high and low) values of UCL95 as shown in examples in the Technical Guide.
It is therefore recommended to avoid the use of H-statistic based 95% UCLs.
Use of nonparametric methods are preferred to compute UCL95 for skewed data sets which do not follow a gamma distribution.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002)
and Singh and Singh (2003). For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Schoolhouse Brook

MERCURY

General Statistics

Number of Valid Data	9	Number of Detected Data	3
Number of Distinct Detected Data	3	Number of Non-Detect Data	6
		Percent Non-Detects	66.67%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.13	Minimum Detected	-2.04
Maximum Detected	0.17	Maximum Detected	-1.772
Mean of Detected	0.152	Mean of Detected	-1.892
SD of Detected	0.0202	SD of Detected	0.136
Minimum Non-Detect	0.1	Minimum Non-Detect	-2.303
Maximum Non-Detect	0.2	Maximum Non-Detect	-1.609

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	9
Number treated as Detected	0
Single DL Non-Detect Percentage	100.00%

Warning: There are only 3 Distinct Detected Values in this data set
The number of detected data may not be adequate enough to perform GOF tests, bootstrap, and ROS methods.
Those methods will return a 'N/A' value on your output display!

It is necessary to have 4 or more Distinct Values for bootstrap methods.
However, results obtained using 4 to 9 distinct values may not be reliable.
It is recommended to have 10 to 15 or more observations for accurate and meaningful results and estimates.

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.98	Shapiro Wilk Test Statistic	0.969
5% Shapiro Wilk Critical Value	0.767	5% Shapiro Wilk Critical Value	0.767
Data appear Normal at 5% Significance Level		**Data appear Lognormal at 5% Significance Level**	
Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	0.095	Mean	-2.474
SD	0.0482	SD	0.524
95% DL/2 (t) UCL	0.125	95% H-Stat (DL/2) UCL	0.147
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	-2.171
		SD in Log Scale	0.265
		Mean in Original Scale	0.118
		SD in Original Scale	0.0313
		95% t UCL	0.137
		95% Percentile Bootstrap UCL	0.133
		95% BCA Bootstrap UCL	0.137

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	N/A	**Data appear Normal at 5% Significance Level**	
Theta Star	N/A		
nu star	N/A		
A-D Test Statistic	N/A	**Nonparametric Statistics**	
5% A-D Critical Value	N/A	Kaplan-Meier (KM) Method	
K-S Test Statistic	N/A	Mean	0.139
5% K-S Critical Value	N/A	SD	0.0152
Data not Gamma Distributed at 5% Significance Level		SE of Mean	0.00705
		95% KM (t) UCL	0.152
Assuming Gamma Distribution		95% KM (z) UCL	0.151
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	0.157
Minimum	N/A	95% KM (bootstrap t) UCL	0.146
Maximum	N/A	95% KM (BCA) UCL	0.17
Mean	N/A	95% KM (Percentile Bootstrap) UCL	0.17
Median	N/A	95% KM (Chebyshev) UCL	0.17
SD	N/A	97.5% KM (Chebyshev) UCL	0.183
k star	N/A	99% KM (Chebyshev) UCL	0.209
Theta star	N/A		
Nu star	N/A	**Potential UCLs to Use**	
AppChi2	N/A	95% KM (t) UCL	0.152
95% Gamma Approximate UCL	N/A	95% KM (Percentile Bootstrap) UCL	0.17
95% Adjusted Gamma UCL	N/A		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

Appendix D
Surface Water - Schoolhouse Brook

VANADIUM

General Statistics

Number of Valid Data	22	Number of Detected Data	8
Number of Distinct Detected Data	7	Number of Non-Detect Data	14
		Percent Non-Detects	63.64%

Raw Statistics		Log-transformed Statistics	
Minimum Detected	0.3	Minimum Detected	-1.204
Maximum Detected	0.86	Maximum Detected	-0.151
Mean of Detected	0.536	Mean of Detected	-0.732
SD of Detected	0.269	SD of Detected	0.489
Minimum Non-Detect	0.2	Minimum Non-Detect	-1.609
Maximum Non-Detect	50	Maximum Non-Detect	3.912

Note: Data have multiple DLs - Use of KM Method is recommended
For all methods (except KM, DL/2, and ROS Methods),
Observations < Largest ND are treated as NDs

Number treated as Non-Detect	22
Number treated as Detected	0
Single DL Non-Detect Percentage	100.00%

Warning: There are only 8 Detected Values in this data
Note: It should be noted that even though bootstrap may be performed on this data set
the resulting calculations may not be reliable enough to draw conclusions

It is recommended to have 10-15 or more distinct observations for accurate and meaningful results.

UCL Statistics

Normal Distribution Test with Detected Values Only		Lognormal Distribution Test with Detected Values Only	
Shapiro Wilk Test Statistic	0.728	Shapiro Wilk Test Statistic	0.764
5% Shapiro Wilk Critical Value	0.818	5% Shapiro Wilk Critical Value	0.818
Data not Normal at 5% Significance Level		Data not Lognormal at 5% Significance Level	
Assuming Normal Distribution		**Assuming Lognormal Distribution**	
DL/2 Substitution Method		DL/2 Substitution Method	
Mean	1.424	Mean	-1.272
SD	5.271	SD	1.249
95% DL/2 (t) UCL	3.358	95% H-Stat (DL/2) UCL	1.363
Maximum Likelihood Estimate(MLE) Method	N/A	Log ROS Method	
MLE method failed to converge properly		Mean in Log Scale	-1.444
		SD in Log Scale	0.724
		Mean in Original Scale	0.305
		SD in Original Scale	0.245
		95% t UCL	0.395
		95% Percentile Bootstrap UCL	0.391
		95% BCA Bootstrap UCL	0.407

Gamma Distribution Test with Detected Values Only		Data Distribution Test with Detected Values Only	
k star (bias corrected)	3.092	Data Follow Appr. Gamma Distribution at 5% Significance Level	
Theta Star	0.173		
nu star	49.46		
A-D Test Statistic	1.02	**Nonparametric Statistics**	
5% A-D Critical Value	0.719	Kaplan-Meier (KM) Method	
K-S Test Statistic	0.719	Mean	0.394
5% K-S Critical Value	0.295	SD	0.192
Data follow Appr. Gamma Distribution at 5% Significance Level		SE of Mean	0.045
		95% KM (t) UCL	0.471
Assuming Gamma Distribution		95% KM (z) UCL	0.468
Gamma ROS Statistics using Extrapolated Data		95% KM (jackknife) UCL	0.462
Minimum	0.149	95% KM (bootstrap t) UCL	0.49
Maximum	1.035	95% KM (BCA) UCL	0.496
Mean	0.596	95% KM (Percentile Bootstrap) UCL	0.478
Median	0.606	95% KM (Chebyshev) UCL	0.59
SD	0.255	97.5% KM (Chebyshev) UCL	0.675
k star	4.214	99% KM (Chebyshev) UCL	0.841
Theta star	0.141		
Nu star	185.4	**Potential UCLs to Use**	
AppChi2	154.9	95% KM (t) UCL	0.471
95% Gamma Approximate UCL	0.713		
95% Adjusted Gamma UCL	0.723		

Note: DL/2 is not a recommended method.

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL.
These recommendations are based upon the results of the simulation studies summarized in Singh, Maichle, and Lee (2006).
For additional insight, the user may want to consult a statistician.

APPENDIX E

APPENDIX E

TABLE E-1
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Recreational Visitor
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Ely Copper Mine Surface Soil	Ingestion	Aluminum	10820	mg/kg	6.8E-04	mg/kg-day	NA	---	NA	5.9E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0059
				Antimony	1.3	mg/kg	8.0E-08	mg/kg-day	NA	---	NA	7.0E-07	mg/kg-day	4.0E-04	mg/kg-day	0.0017
				Arsenic	2.7	mg/kg	1.7E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.6E-07	1.5E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0050
				Cadmium	0.99	mg/kg	6.2E-08	mg/kg-day	NA	---	NA	5.4E-07	mg/kg-day	1.0E-03	mg/kg-day	0.00054
				Chromium	44	mg/kg	2.7E-06	mg/kg-day	NA	---	NA	2.4E-05	mg/kg-day	1.5E+00	mg/kg-day	0.000016
				Cobalt	25	mg/kg	1.5E-06	mg/kg-day	NA	---	NA	1.3E-05	mg/kg-day	3.0E-04	mg/kg-day	0.045
				Copper	1086	mg/kg	6.8E-05	mg/kg-day	NA	---	NA	6.0E-04	mg/kg-day	4.0E-02	mg/kg-day	0.015
				Iron	66914	mg/kg	4.2E-03	mg/kg-day	NA	---	NA	3.7E-02	mg/kg-day	7.0E-01	mg/kg-day	0.052
				Manganese	449	mg/kg	2.8E-05	mg/kg-day	NA	---	NA	2.5E-04	mg/kg-day	7.0E-02	mg/kg-day	0.0035
				Mercury	0.15	mg/kg	9.3E-09	mg/kg-day	NA	---	NA	8.1E-08	mg/kg-day	3.0E-04	mg/kg-day	0.00027
				Molybdenum	6.9	mg/kg	4.3E-07	mg/kg-day	NA	---	NA	3.8E-06	mg/kg-day	5.0E-03	mg/kg-day	0.00075
				Selenium	21	mg/kg	1.3E-06	mg/kg-day	NA	---	NA	1.2E-05	mg/kg-day	5.0E-03	mg/kg-day	0.0023
				Vanadium	56	mg/kg	3.5E-06	mg/kg-day	NA	---	NA	3.1E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0015
			Ingestion Total								2.6E-07					0.13
			Dermal	Aluminum	10820	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	1.3	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	6.0E-05	mg/kg-day	NA
				Arsenic	2.7	mg/kg	6.0E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	9.1E-08	5.3E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0018
				Cadmium	0.99	mg/kg	7.3E-10	mg/kg-day	NA	---	NA	6.4E-09	mg/kg-day	2.5E-05	mg/kg-day	0.00026
				Chromium	44	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	25	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	1086	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	66914	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	449	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Mercury	0.15	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.1E-05	mg/kg-day	NA
				Molybdenum	6.9	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Selenium	21	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	56	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								9.1E-08					0.0020
			Inhalation	Aluminum	10820	mg/kg	1.5E-02	µg/m^3	NA	---	NA	1.3E-04	mg/m^3	5.0E-03	mg/m^3	0.026
				Antimony	1.3	mg/kg	1.7E-06	µg/m^3	NA	---	NA	1.5E-08	mg/m^3	NA	---	NA
				Arsenic	2.7	mg/kg	3.7E-06	µg/m^3	4.3E-03	(µg/m3)^-1	1.6E-08	3.3E-08	mg/m^3	1.5E-05	mg/m^3	0.0022
				Cadmium	1.0	mg/kg	1.3E-06	µg/m^3	1.8E-03	(µg/m3)^-1	2.4E-09	1.2E-08	mg/m^3	1.0E-05	mg/m^3	0.0012
				Chromium	44	mg/kg	6.0E-05	µg/m^3	NA	---	NA	5.2E-07	mg/m^3	NA	---	NA
				Cobalt	25	mg/kg	3.4E-05	µg/m^3	9.0E-03	(µg/m3)^-1	3.0E-07	2.9E-07	mg/m^3	6.0E-06	mg/m^3	0.049
				Copper	1086	mg/kg	1.5E-03	µg/m^3	NA	---	NA	1.3E-05	mg/m^3	NA	---	NA
				Iron	66914	mg/kg	9.2E-02	µg/m^3	NA	---	NA	8.0E-04	mg/m^3	NA	---	NA
				Manganese	449	mg/kg	6.1E-04	µg/m^3	NA	---	NA	5.4E-06	mg/m^3	5.0E-05	mg/m^3	0.11
				Mercury	0.15	mg/kg	2.0E-07	µg/m^3	NA	---	NA	1.8E-09	mg/m^3	3.0E-04	mg/m^3	0.0000059
				Molybdenum	6.9	mg/kg	9.4E-06	µg/m^3	NA	---	NA	8.2E-08	mg/m^3	NA	---	NA
				Selenium	21	mg/kg	2.9E-05	µg/m^3	NA	---	NA	2.5E-07	mg/m^3	2.0E-02	mg/m^3	0.000013
				Vanadium	56	mg/kg	7.7E-05	µg/m^3	NA	---	NA	6.7E-07	mg/m^3	NA	---	NA
			Inhalation Total								3.2E-07					0.19
Soil Total											6.7E-07					0.32
											Total of Receptor Risks Across All Media					
											6.7E-07					0.32
												Total of Receptor Hazards Across All Media				

APPENDIX E

TABLE E-2
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Recreational Visitor
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Ely Copper Mine Surface Soil	Ingestion	Aluminum	10820	mg/kg	1.5E-03	mg/kg-day	NA	---	NA	4.4E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0044
				Antimony	1.3	mg/kg	1.8E-07	mg/kg-day	NA	---	NA	5.2E-07	mg/kg-day	4.0E-04	mg/kg-day	0.0013
				Arsenic	2.7	mg/kg	3.8E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	5.7E-07	1.1E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0037
				Cadmium	0.99	mg/kg	1.4E-07	mg/kg-day	NA	---	NA	4.0E-07	mg/kg-day	1.0E-03	mg/kg-day	0.00040
				Chromium	44	mg/kg	6.1E-06	mg/kg-day	NA	---	NA	1.8E-05	mg/kg-day	1.5E+00	mg/kg-day	0.000012
				Cobalt	25	mg/kg	3.4E-06	mg/kg-day	NA	---	NA	1.0E-05	mg/kg-day	3.0E-04	mg/kg-day	0.033
				Copper	1086	mg/kg	1.5E-04	mg/kg-day	NA	---	NA	4.4E-04	mg/kg-day	4.0E-02	mg/kg-day	0.011
				Iron	66914	mg/kg	9.3E-03	mg/kg-day	NA	---	NA	2.7E-02	mg/kg-day	7.0E-01	mg/kg-day	0.039
				Manganese	449	mg/kg	6.3E-05	mg/kg-day	NA	---	NA	1.8E-04	mg/kg-day	7.0E-02	mg/kg-day	0.0026
				Mercury	0.15	mg/kg	2.1E-08	mg/kg-day	NA	---	NA	6.0E-08	mg/kg-day	3.0E-04	mg/kg-day	0.00020
				Molybdenum	6.9	mg/kg	9.6E-07	mg/kg-day	NA	---	NA	2.8E-06	mg/kg-day	5.0E-03	mg/kg-day	0.00056
				Selenium	21	mg/kg	2.9E-06	mg/kg-day	NA	---	NA	8.6E-06	mg/kg-day	5.0E-03	mg/kg-day	0.0017
				Vanadium	56	mg/kg	7.9E-06	mg/kg-day	NA	---	NA	2.3E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0011
			Ingestion Total								5.7E-07					0.099
			Dermal	Aluminum	10820	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	1.3	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	6.0E-05	mg/kg-day	NA
				Arsenic	2.7	mg/kg	1.6E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.4E-07	4.6E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0015
				Cadmium	0.99	mg/kg	1.9E-09	mg/kg-day	NA	---	NA	5.5E-09	mg/kg-day	2.5E-05	mg/kg-day	0.00022
				Chromium	44	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	25	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	1086	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	66914	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	449	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Mercury	0.15	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.1E-05	mg/kg-day	NA
				Molybdenum	6.9	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Selenium	21	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	56	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								2.4E-07					0.0018
			Inhalation	Aluminum	10820	mg/kg	4.4E-02	µg/m^3	NA	---	NA	1.3E-04	mg/m^3	5.0E-03	mg/m^3	0.026
				Antimony	1.3	mg/kg	5.2E-06	µg/m^3	NA	---	NA	1.5E-08	mg/m^3	NA	---	NA
				Arsenic	2.7	mg/kg	1.1E-05	µg/m^3	4.3E-03	(µg/m3)^-1	4.8E-08	3.3E-08	mg/m^3	1.5E-05	mg/m^3	0.0022
				Cadmium	1.0	mg/kg	4.0E-06	µg/m^3	1.8E-03	(µg/m3)^-1	7.3E-09	1.2E-08	mg/m^3	1.0E-05	mg/m^3	0.0012
				Chromium	44	mg/kg	1.8E-04	µg/m^3	NA	---	NA	5.2E-07	mg/m^3	NA	---	NA
				Cobalt	25	mg/kg	1.0E-04	µg/m^3	9.0E-03	(µg/m3)^-1	9.1E-07	2.9E-07	mg/m^3	6.0E-06	mg/m^3	0.049
				Copper	1086	mg/kg	4.5E-03	µg/m^3	NA	---	NA	1.3E-05	mg/m^3	NA	---	NA
				Iron	66914	mg/kg	2.7E-01	µg/m^3	NA	---	NA	8.0E-04	mg/m^3	NA	---	NA
				Manganese	449	mg/kg	1.8E-03	µg/m^3	NA	---	NA	5.4E-06	mg/m^3	5.0E-05	mg/m^3	0.11
				Mercury	0.15	mg/kg	6.1E-07	µg/m^3	NA	---	NA	1.8E-09	mg/m^3	3.0E-04	mg/m^3	0.0000059
				Molybdenum	6.9	mg/kg	2.8E-05	µg/m^3	NA	---	NA	8.2E-08	mg/m^3	NA	---	NA
				Selenium	21	mg/kg	8.6E-05	µg/m^3	NA	---	NA	2.5E-07	mg/m^3	2.0E-02	mg/m^3	0.000013
				Vanadium	56	mg/kg	2.3E-04	µg/m^3	NA	---	NA	6.7E-07	mg/m^3	NA	---	NA
			Inhalation Total								9.6E-07					0.19
Soil Total											1.8E-06					0.29
										Total of Receptor Risks Across All Media	1.8E-06			Total of Receptor Hazards Across All Media		0.29

APPENDIX E

TABLE E-3
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Recreational Visitor
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Ely Copper Mine Surface Soil	Ingestion	Aluminum	10820	mg/kg	1.1E-04	mg/kg-day	NA	---	NA	1.0E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0010
				Antimony	1.3	mg/kg	1.3E-08	mg/kg-day	NA	---	NA	1.2E-07	mg/kg-day	4.0E-04	mg/kg-day	0.00029
				Arsenic	2.7	mg/kg	2.9E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	4.3E-08	2.5E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00084
				Cadmium	0.99	mg/kg	1.0E-08	mg/kg-day	NA	---	NA	9.1E-08	mg/kg-day	1.0E-03	mg/kg-day	0.000091
				Chromium	44	mg/kg	4.6E-07	mg/kg-day	NA	---	NA	4.0E-06	mg/kg-day	1.5E+00	mg/kg-day	0.0000027
				Cobalt	25	mg/kg	2.6E-07	mg/kg-day	NA	---	NA	2.3E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0076
				Copper	1086	mg/kg	1.1E-05	mg/kg-day	NA	---	NA	1.0E-04	mg/kg-day	4.0E-02	mg/kg-day	0.0025
				Iron	66914	mg/kg	7.1E-04	mg/kg-day	NA	---	NA	6.2E-03	mg/kg-day	7.0E-01	mg/kg-day	0.0088
				Manganese	449	mg/kg	4.7E-06	mg/kg-day	NA	---	NA	4.1E-05	mg/kg-day	7.0E-02	mg/kg-day	0.00059
				Mercury	0.15	mg/kg	1.6E-09	mg/kg-day	NA	---	NA	1.4E-08	mg/kg-day	3.0E-04	mg/kg-day	0.000045
				Molybdenum	6.9	mg/kg	7.2E-08	mg/kg-day	NA	---	NA	6.3E-07	mg/kg-day	5.0E-03	mg/kg-day	0.00013
				Selenium	21	mg/kg	2.2E-07	mg/kg-day	NA	---	NA	1.9E-06	mg/kg-day	5.0E-03	mg/kg-day	0.00039
				Vanadium	56	mg/kg	5.9E-07	mg/kg-day	NA	---	NA	5.2E-06	mg/kg-day	2.0E-02	mg/kg-day	0.00026
			Ingestion Total								4.3E-08					0.023
			Dermal	Aluminum	10820	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	1.3	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	6.0E-05	mg/kg-day	NA
				Arsenic	2.7	mg/kg	6.2E-09	mg/kg-day	1.5E+00	(mg/kg-day)^-1	9.3E-09	5.4E-08	mg/kg-day	3.0E-04	mg/kg-day	0.00018
				Cadmium	0.99	mg/kg	7.5E-11	mg/kg-day	NA	---	NA	6.6E-10	mg/kg-day	2.5E-05	mg/kg-day	0.000026
				Chromium	44	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	25	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	1086	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	66914	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	449	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Mercury	0.15	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.1E-05	mg/kg-day	NA
				Molybdenum	6.9	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Selenium	21	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	56	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								9.3E-09					0.00021
			Inhalation	Aluminum	10820	mg/kg	2.6E-03	µg/m^3	NA	---	NA	2.3E-05	mg/m^3	5.0E-03	mg/m^3	0.0046
				Antimony	1.3	mg/kg	3.1E-07	µg/m^3	NA	---	NA	2.7E-09	mg/m^3	NA	---	NA
				Arsenic	2.7	mg/kg	6.6E-07	µg/m^3	4.3E-03	(µg/m3)^-1	2.8E-09	5.8E-09	mg/m^3	1.5E-05	mg/m^3	0.00039
				Cadmium	1.0	mg/kg	2.4E-07	µg/m^3	1.8E-03	(µg/m3)^-1	4.3E-10	2.1E-09	mg/m^3	1.0E-05	mg/m^3	0.00021
				Chromium	44	mg/kg	1.1E-05	µg/m^3	NA	---	NA	9.3E-08	mg/m^3	NA	---	NA
				Cobalt	25	mg/kg	6.0E-06	µg/m^3	9.0E-03	(µg/m3)^-1	5.4E-08	5.2E-08	mg/m^3	6.0E-06	mg/m^3	0.0087
				Copper	1086	mg/kg	2.6E-04	µg/m^3	NA	---	NA	2.3E-06	mg/m^3	NA	---	NA
				Iron	66914	mg/kg	1.6E-02	µg/m^3	NA	---	NA	1.4E-04	mg/m^3	NA	---	NA
				Manganese	449	mg/kg	1.1E-04	µg/m^3	NA	---	NA	9.5E-07	mg/m^3	5.0E-05	mg/m^3	0.019
				Mercury	0.15	mg/kg	3.6E-08	µg/m^3	NA	---	NA	3.1E-10	mg/m^3	3.0E-04	mg/m^3	0.0000010
				Molybdenum	6.9	mg/kg	1.7E-06	µg/m^3	NA	---	NA	1.5E-08	mg/m^3	NA	---	NA
				Selenium	21	mg/kg	5.1E-06	µg/m^3	NA	---	NA	4.5E-08	mg/m^3	2.0E-02	mg/m^3	0.0000022
				Vanadium	56	mg/kg	1.4E-05	µg/m^3	NA	---	NA	1.2E-07	mg/m^3	NA	---	NA
			Inhalation Total								5.7E-08					0.033
Soil Total											1.1E-07					0.056
											Total of Receptor Risks Across All Media					
											1.1E-07					0.056
												Total of Receptor Hazards Across All Media				

APPENDIX E

TABLE E-4
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Recreational Visitor
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Ely Copper Mine Surface Soil	Ingestion	Aluminum	10820	mg/kg	2.5E-04	mg/kg-day	NA	---	NA	7.4E-04	mg/kg-day	1.0E+00	mg/kg-day	0.00074
				Antimony	1.3	mg/kg	3.0E-08	mg/kg-day	NA	---	NA	8.7E-08	mg/kg-day	4.0E-04	mg/kg-day	0.00022
				Arsenic	2.7	mg/kg	6.4E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	9.6E-08	1.9E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00062
				Cadmium	0.99	mg/kg	2.3E-08	mg/kg-day	NA	---	NA	6.8E-08	mg/kg-day	1.0E-03	mg/kg-day	0.000068
				Chromium	44	mg/kg	1.0E-06	mg/kg-day	NA	---	NA	3.0E-06	mg/kg-day	1.5E+00	mg/kg-day	0.0000020
				Cobalt	25	mg/kg	5.8E-07	mg/kg-day	NA	---	NA	1.7E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0056
				Copper	1086	mg/kg	2.6E-05	mg/kg-day	NA	---	NA	7.4E-05	mg/kg-day	4.0E-02	mg/kg-day	0.0019
				Iron	66914	mg/kg	1.6E-03	mg/kg-day	NA	---	NA	4.6E-03	mg/kg-day	7.0E-01	mg/kg-day	0.0065
				Manganese	449	mg/kg	1.1E-05	mg/kg-day	NA	---	NA	3.1E-05	mg/kg-day	7.0E-02	mg/kg-day	0.00044
				Mercury	0.15	mg/kg	3.5E-09	mg/kg-day	NA	---	NA	1.0E-08	mg/kg-day	3.0E-04	mg/kg-day	0.000034
				Molybdenum	6.9	mg/kg	1.6E-07	mg/kg-day	NA	---	NA	4.7E-07	mg/kg-day	5.0E-03	mg/kg-day	0.000094
				Selenium	21	mg/kg	4.9E-07	mg/kg-day	NA	---	NA	1.4E-06	mg/kg-day	5.0E-03	mg/kg-day	0.00029
				Vanadium	56	mg/kg	1.3E-06	mg/kg-day	NA	---	NA	3.9E-06	mg/kg-day	2.0E-02	mg/kg-day	0.00019
			Ingestion Total								9.6E-08					0.017
			Dermal	Aluminum	10820	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	1.3	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	6.0E-05	mg/kg-day	NA
				Arsenic	2.7	mg/kg	1.7E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.6E-08	5.0E-08	mg/kg-day	3.0E-04	mg/kg-day	0.00017
				Cadmium	0.99	mg/kg	2.1E-10	mg/kg-day	NA	---	NA	6.1E-10	mg/kg-day	2.5E-05	mg/kg-day	0.000024
				Chromium	44	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	25	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	1086	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	66914	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	449	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Mercury	0.15	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.1E-05	mg/kg-day	NA
				Molybdenum	6.9	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Selenium	21	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	56	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								2.6E-08					0.00019
			Inhalation	Aluminum	10820	mg/kg	7.5E-03	µg/m^3	NA	---	NA	2.2E-05	mg/m^3	5.0E-03	mg/m^3	0.0044
				Antimony	1.3	mg/kg	8.8E-07	µg/m^3	NA	---	NA	2.6E-09	mg/m^3	NA	---	NA
				Arsenic	2.7	mg/kg	1.9E-06	µg/m^3	4.3E-03	(µg/m3)^-1	8.1E-09	5.5E-09	mg/m^3	1.5E-05	mg/m^3	0.00037
				Cadmium	1.0	mg/kg	6.8E-07	µg/m^3	1.8E-03	(µg/m3)^-1	1.2E-09	2.0E-09	mg/m^3	1.0E-05	mg/m^3	0.00020
				Chromium	44	mg/kg	3.0E-05	µg/m^3	NA	---	NA	8.8E-08	mg/m^3	NA	---	NA
				Cobalt	25	mg/kg	1.7E-05	µg/m^3	9.0E-03	(µg/m3)^-1	1.5E-07	4.9E-08	mg/m^3	6.0E-06	mg/m^3	0.0082
				Copper	1086	mg/kg	7.5E-04	µg/m^3	NA	---	NA	2.2E-06	mg/m^3	NA	---	NA
				Iron	66914	mg/kg	4.6E-02	µg/m^3	NA	---	NA	1.3E-04	mg/m^3	NA	---	NA
				Manganese	449	mg/kg	3.1E-04	µg/m^3	NA	---	NA	9.0E-07	mg/m^3	5.0E-05	mg/m^3	0.018
				Mercury	0.15	mg/kg	1.0E-07	µg/m^3	NA	---	NA	3.0E-10	mg/m^3	3.0E-04	mg/m^3	0.0000010
				Molybdenum	6.9	mg/kg	4.7E-06	µg/m^3	NA	---	NA	1.4E-08	mg/m^3	NA	---	NA
				Selenium	21	mg/kg	1.5E-05	µg/m^3	NA	---	NA	4.2E-08	mg/m^3	2.0E-02	mg/m^3	0.0000021
				Vanadium	56	mg/kg	3.9E-05	µg/m^3	NA	---	NA	1.1E-07	mg/m^3	NA	---	NA
			Inhalation Total								1.6E-07					0.031
Soil Total											2.8E-07					0.048
										Total of Receptor Risks Across All Media	2.8E-07			Total of Receptor Hazards Across All Media		0.048

APPENDIX E

TABLE E-5
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Construction Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Total Soil	Ely Copper Mine Soil	Ingestion	Aluminum	11814	mg/kg	1.3E-04	mg/kg-day	NA	---	NA	9.2E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0092
				Antimony	1.1	mg/kg	1.2E-08	mg/kg-day	NA	---	NA	8.2E-07	mg/kg-day	4.0E-04	mg/kg-day	0.0020
				Arsenic	2.3	mg/kg	2.6E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	3.9E-08	1.8E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0060
				Cadmium	1.9	mg/kg	2.1E-08	mg/kg-day	NA	---	NA	1.4E-06	mg/kg-day	1.0E-03	mg/kg-day	0.0014
				Chromium	48	mg/kg	5.3E-07	mg/kg-day	NA	---	NA	3.7E-05	mg/kg-day	1.5E+00	mg/kg-day	0.000025
				Cobalt	50	mg/kg	5.6E-07	mg/kg-day	NA	---	NA	3.9E-05	mg/kg-day	3.0E-04	mg/kg-day	0.13
				Copper	1887	mg/kg	2.1E-05	mg/kg-day	NA	---	NA	1.5E-03	mg/kg-day	4.0E-02	mg/kg-day	0.037
				Iron	67413	mg/kg	7.5E-04	mg/kg-day	NA	---	NA	5.2E-02	mg/kg-day	7.0E-01	mg/kg-day	0.075
				Manganese	411	mg/kg	4.5E-06	mg/kg-day	NA	---	NA	3.2E-04	mg/kg-day	7.0E-02	mg/kg-day	0.0045
				Mercury	0.13	mg/kg	1.4E-09	mg/kg-day	NA	---	NA	9.7E-08	mg/kg-day	3.0E-04	mg/kg-day	0.00032
				Molybdenum	6.4	mg/kg	7.1E-08	mg/kg-day	NA	---	NA	5.0E-06	mg/kg-day	5.0E-03	mg/kg-day	0.00099
				Nickel	27	mg/kg	3.0E-07	mg/kg-day	NA	---	NA	2.1E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0011
				Selenium	22	mg/kg	2.4E-07	mg/kg-day	NA	---	NA	1.7E-05	mg/kg-day	5.0E-03	mg/kg-day	0.0034
				Vanadium	57	mg/kg	6.3E-07	mg/kg-day	NA	---	NA	4.4E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0022
				Zinc	262	mg/kg	2.9E-06	mg/kg-day	NA	---	NA	2.0E-04	mg/kg-day	3.0E-01	mg/kg-day	0.00068
			Ingestion Total								3.9E-08					0.27
			Dermal	Aluminum	11814	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	1.1	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	6.0E-05	mg/kg-day	NA
				Arsenic	2.3	mg/kg	1.9E-09	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.8E-09	1.3E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00043
				Cadmium	1.9	mg/kg	4.9E-11	mg/kg-day	NA	---	NA	3.5E-09	mg/kg-day	2.5E-05	mg/kg-day	0.00014
				Chromium	48	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	50	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	1887	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	67413	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	411	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Mercury	0.13	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.1E-05	mg/kg-day	NA
				Molybdenum	6.4	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Nickel	27	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	8.0E-04	mg/kg-day	NA
				Selenium	22	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	57	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
				Zinc	262	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-01	mg/kg-day	NA
			Dermal Total								2.8E-09					0.00057
			Inhalation	Aluminum	11814	mg/kg	1.7E-02	µg/m^3	NA	---	NA	1.2E-03	mg/m^3	5.0E-03	mg/m^3	0.24
				Antimony	1.1	mg/kg	1.5E-06	µg/m^3	NA	---	NA	1.1E-07	mg/m^3	NA	---	NA
				Arsenic	2.3	mg/kg	3.4E-06	µg/m^3	4.3E-03	(µg/m3)^-1	1.5E-08	2.4E-07	mg/m^3	1.5E-05	mg/m^3	0.016
				Cadmium	1.9	mg/kg	2.7E-06	µg/m^3	1.8E-03	(µg/m3)^-1	4.9E-09	1.9E-07	mg/m^3	1.0E-05	mg/m^3	0.019
				Chromium	48	mg/kg	7.1E-05	µg/m^3	NA	---	NA	5.0E-06	mg/m^3	NA	---	NA
				Cobalt	50	mg/kg	7.4E-05	µg/m^3	9.0E-03	(µg/m3)^-1	6.6E-07	5.2E-06	mg/m^3	6.0E-06	mg/m^3	0.86
				Copper	1887	mg/kg	2.8E-03	µg/m^3	NA	---	NA	1.9E-04	mg/m^3	NA	---	NA
				Iron	67413	mg/kg	9.9E-02	µg/m^3	NA	---	NA	6.9E-03	mg/m^3	NA	---	NA
				Manganese	411	mg/kg	6.0E-04	µg/m^3	NA	---	NA	4.2E-05	mg/m^3	5.0E-05	mg/m^3	0.85
				Mercury	0.13	mg/kg	1.8E-07	µg/m^3	NA	---	NA	1.3E-08	mg/m^3	3.0E-04	mg/m^3	0.000043
				Molybdenum	6.4	mg/kg	9.4E-06	µg/m^3	NA	---	NA	6.6E-07	mg/m^3	NA	---	NA
				Nickel	27	mg/kg	4.0E-05	µg/m^3	2.6E-04	(µg/m3)^-1	1.0E-08	2.8E-06	mg/m^3	9.0E-05	mg/m^3	0.031
				Selenium	22	mg/kg	3.2E-05	µg/m^3	NA	---	NA	2.2E-06	mg/m^3	2.0E-02	mg/m^3	0.00011
				Vanadium	57	mg/kg	8.4E-05	µg/m^3	NA	---	NA	5.9E-06	mg/m^3	NA	---	NA
				Zinc	262	mg/kg	3.8E-04	µg/m^3	NA	---	NA	2.7E-05	mg/m^3	NA	---	NA
			Inhalation Total								6.9E-07					2.0
Soil Total											7.4E-07					2.3
											Total of Receptor Risks Across All Media					7.4E-07
																Total of Receptor Hazards Across All Media
																2.3

APPENDIX E

TABLE E-6
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Construction Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Total Soil	Ely Copper Mine Soil	Ingestion	Aluminum	11814	mg/kg	2.0E-05	mg/kg-day	NA	---	NA	1.4E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0014
				Antimony	1.1	mg/kg	1.8E-09	mg/kg-day	NA	---	NA	1.2E-07	mg/kg-day	4.0E-04	mg/kg-day	0.00031
				Arsenic	2.3	mg/kg	3.9E-09	mg/kg-day	1.5E+00	(mg/kg-day)^-1	5.8E-09	2.7E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00091
				Cadmium	1.9	mg/kg	3.1E-09	mg/kg-day	NA	---	NA	2.2E-07	mg/kg-day	1.0E-03	mg/kg-day	0.00022
				Chromium	48	mg/kg	8.1E-08	mg/kg-day	NA	---	NA	5.7E-06	mg/kg-day	1.5E+00	mg/kg-day	0.0000038
				Cobalt	50	mg/kg	8.4E-08	mg/kg-day	NA	---	NA	5.9E-06	mg/kg-day	3.0E-04	mg/kg-day	0.020
				Copper	1887	mg/kg	3.2E-06	mg/kg-day	NA	---	NA	2.2E-04	mg/kg-day	4.0E-02	mg/kg-day	0.0055
				Iron	67413	mg/kg	1.1E-04	mg/kg-day	NA	---	NA	7.9E-03	mg/kg-day	7.0E-01	mg/kg-day	0.011
				Manganese	411	mg/kg	6.9E-07	mg/kg-day	NA	---	NA	4.8E-05	mg/kg-day	7.0E-02	mg/kg-day	0.00069
				Mercury	0.13	mg/kg	2.1E-10	mg/kg-day	NA	---	NA	1.5E-08	mg/kg-day	3.0E-04	mg/kg-day	0.000049
				Molybdenum	6.4	mg/kg	1.1E-08	mg/kg-day	NA	---	NA	7.5E-07	mg/kg-day	5.0E-03	mg/kg-day	0.00015
				Nickel	27	mg/kg	4.6E-08	mg/kg-day	NA	---	NA	3.2E-06	mg/kg-day	2.0E-02	mg/kg-day	0.00016
				Selenium	22	mg/kg	3.6E-08	mg/kg-day	NA	---	NA	2.6E-06	mg/kg-day	5.0E-03	mg/kg-day	0.00051
				Vanadium	57	mg/kg	9.5E-08	mg/kg-day	NA	---	NA	6.7E-06	mg/kg-day	2.0E-02	mg/kg-day	0.00033
				Zinc	262	mg/kg	4.4E-07	mg/kg-day	NA	---	NA	3.1E-05	mg/kg-day	3.0E-01	mg/kg-day	0.00010
			Ingestion Total								5.8E-09					0.041
			Dermal	Aluminum	11814	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	1.1	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	6.0E-05	mg/kg-day	NA
				Arsenic	2.3	mg/kg	5.4E-10	mg/kg-day	1.5E+00	(mg/kg-day)^-1	8.1E-10	3.8E-08	mg/kg-day	3.0E-04	mg/kg-day	0.00013
				Cadmium	1.9	mg/kg	1.4E-11	mg/kg-day	NA	---	NA	1.0E-09	mg/kg-day	2.5E-05	mg/kg-day	0.000040
				Chromium	48	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	50	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	1887	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	67413	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	411	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Mercury	0.13	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.1E-05	mg/kg-day	NA
				Molybdenum	6.4	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Nickel	27	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	8.0E-04	mg/kg-day	NA
				Selenium	22	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	57	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
				Zinc	262	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-01	mg/kg-day	NA
			Dermal Total								8.1E-10					0.00017
			Inhalation	Aluminum	11814	mg/kg	3.4E-03	µg/m^3	NA	---	NA	2.4E-04	mg/m^3	5.0E-03	mg/m^3	0.047
				Antimony	1.1	mg/kg	3.0E-07	µg/m^3	NA	---	NA	2.1E-08	mg/m^3	NA	---	NA
				Arsenic	2.3	mg/kg	6.7E-07	µg/m^3	4.3E-03	(µg/m3)^-1	2.9E-09	4.7E-08	mg/m^3	1.5E-05	mg/m^3	0.0031
				Cadmium	1.9	mg/kg	5.3E-07	µg/m^3	1.8E-03	(µg/m3)^-1	9.6E-10	3.7E-08	mg/m^3	1.0E-05	mg/m^3	0.0037
				Chromium	48	mg/kg	1.4E-05	µg/m^3	NA	---	NA	9.6E-07	mg/m^3	NA	---	NA
				Cobalt	50	mg/kg	1.4E-05	µg/m^3	9.0E-03	(µg/m3)^-1	1.3E-07	1.0E-06	mg/m^3	6.0E-06	mg/m^3	0.17
				Copper	1887	mg/kg	5.4E-04	µg/m^3	NA	---	NA	3.8E-05	mg/m^3	NA	---	NA
				Iron	67413	mg/kg	1.9E-02	µg/m^3	NA	---	NA	1.4E-03	mg/m^3	NA	---	NA
				Manganese	411	mg/kg	1.2E-04	µg/m^3	NA	---	NA	8.2E-06	mg/m^3	5.0E-05	mg/m^3	0.16
				Mercury	0.13	mg/kg	3.6E-08	µg/m^3	NA	---	NA	2.5E-09	mg/m^3	3.0E-04	mg/m^3	0.0000084
				Molybdenum	6.4	mg/kg	1.8E-06	µg/m^3	NA	---	NA	1.3E-07	mg/m^3	NA	---	NA
				Nickel	27	mg/kg	7.8E-06	µg/m^3	2.6E-04	(µg/m3)^-1	2.0E-09	5.5E-07	mg/m^3	9.0E-05	mg/m^3	0.0061
				Selenium	22	mg/kg	6.2E-06	µg/m^3	NA	---	NA	4.4E-07	mg/m^3	2.0E-02	mg/m^3	0.000022
				Vanadium	57	mg/kg	1.6E-05	µg/m^3	NA	---	NA	1.1E-06	mg/m^3	NA	---	NA
				Zinc	262	mg/kg	7.5E-05	µg/m^3	NA	---	NA	5.2E-06	mg/m^3	NA	---	NA
			Inhalation Total								1.4E-07					0.39
Soil Total											1.4E-07					0.43
											Total of Receptor Risks Across All Media					Total of Receptor Hazards Across All Media
											1.4E-07					0.43

APPENDIX E

TABLE E-7
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - CURRENT CHILD RESIDENT SOIL EXPOSURE
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Ely Copper Mine Surface Soil	Ingestion	Aluminum	10820	mg/kg	1.2E-02	mg/kg-day	NA	---	NA	1.4E-01	mg/kg-day	1.0E+00	mg/kg-day	0.14
				Antimony	1.3	mg/kg	1.4E-06	mg/kg-day	NA	---	NA	1.6E-05	mg/kg-day	4.0E-04	mg/kg-day	0.041
				Arsenic	2.7	mg/kg	3.0E-06	mg/kg-day	1.5E+00	(mg/kg-day)^-1	4.5E-06	3.5E-05	mg/kg-day	3.0E-04	mg/kg-day	0.12
				Cadmium	0.99	mg/kg	1.1E-06	mg/kg-day	NA	---	NA	1.3E-05	mg/kg-day	1.0E-03	mg/kg-day	0.013
				Chromium	44	mg/kg	4.8E-05	mg/kg-day	NA	---	NA	5.6E-04	mg/kg-day	1.5E+00	mg/kg-day	0.00037
				Cobalt	25	mg/kg	2.7E-05	mg/kg-day	NA	---	NA	3.1E-04	mg/kg-day	3.0E-04	mg/kg-day	1.0
				Copper	1086	mg/kg	1.2E-03	mg/kg-day	NA	---	NA	1.4E-02	mg/kg-day	4.0E-02	mg/kg-day	0.35
				Iron	66914	mg/kg	7.3E-02	mg/kg-day	NA	---	NA	8.6E-01	mg/kg-day	7.0E-01	mg/kg-day	1.2
				Manganese	449	mg/kg	4.9E-04	mg/kg-day	NA	---	NA	5.7E-03	mg/kg-day	7.0E-02	mg/kg-day	0.082
				Mercury	0.15	mg/kg	1.6E-07	mg/kg-day	NA	---	NA	1.9E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0063
				Molybdenum	6.9	mg/kg	7.5E-06	mg/kg-day	NA	---	NA	8.8E-05	mg/kg-day	5.0E-03	mg/kg-day	0.018
				Selenium	21	mg/kg	2.3E-05	mg/kg-day	NA	---	NA	2.7E-04	mg/kg-day	5.0E-03	mg/kg-day	0.054
				Vanadium	56	mg/kg	6.2E-05	mg/kg-day	NA	---	NA	7.2E-04	mg/kg-day	2.0E-02	mg/kg-day	0.036
			Ingestion Total								4.5E-06					3.1
			Dermal	Aluminum	10820	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	1.3	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	6.0E-05	mg/kg-day	NA
				Arsenic	2.7	mg/kg	2.5E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	3.8E-07	2.9E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0098
				Cadmium	0.99	mg/kg	3.0E-09	mg/kg-day	NA	---	NA	3.5E-08	mg/kg-day	2.5E-05	mg/kg-day	0.0014
				Chromium	44	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	25	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	1086	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	66914	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	449	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Mercury	0.15	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.1E-05	mg/kg-day	NA
				Molybdenum	6.9	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Selenium	21	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	56	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								3.8E-07					0.011
			Inhalation	Aluminum	10820	mg/kg	1.4E-04	µg/m^3	NA	---	NA	1.7E-06	mg/m^3	5.0E-03	mg/m^3	0.00033
				Antimony	1.3	mg/kg	1.7E-08	µg/m^3	NA	---	NA	2.0E-10	mg/m^3	NA	---	NA
				Arsenic	2.7	mg/kg	3.6E-08	µg/m^3	4.3E-03	(µg/m3)^-1	1.5E-10	4.2E-10	mg/m^3	1.5E-05	mg/m^3	0.000028
				Cadmium	1.0	mg/kg	1.3E-08	µg/m^3	1.8E-03	(µg/m3)^-1	2.3E-11	1.5E-10	mg/m^3	1.0E-05	mg/m^3	0.000015
				Chromium	44	mg/kg	5.7E-07	µg/m^3	NA	---	NA	6.7E-09	mg/m^3	NA	---	NA
				Cobalt	25	mg/kg	3.2E-07	µg/m^3	9.0E-03	(µg/m3)^-1	2.9E-09	3.8E-09	mg/m^3	6.0E-06	mg/m^3	0.00063
				Copper	1086	mg/kg	1.4E-05	µg/m^3	NA	---	NA	1.7E-07	mg/m^3	NA	---	NA
				Iron	66914	mg/kg	8.8E-04	µg/m^3	NA	---	NA	1.0E-05	mg/m^3	NA	---	NA
				Manganese	449	mg/kg	5.9E-06	µg/m^3	NA	---	NA	6.9E-08	mg/m^3	5.0E-05	mg/m^3	0.0014
				Mercury	0.15	mg/kg	1.9E-09	µg/m^3	NA	---	NA	2.3E-11	mg/m^3	3.0E-04	mg/m^3	0.000000076
				Molybdenum	6.9	mg/kg	9.0E-08	µg/m^3	NA	---	NA	1.1E-09	mg/m^3	NA	---	NA
				Selenium	21	mg/kg	2.8E-07	µg/m^3	NA	---	NA	3.2E-09	mg/m^3	2.0E-02	mg/m^3	0.00000016
				Vanadium	56	mg/kg	7.4E-07	µg/m^3	NA	---	NA	8.6E-09	mg/m^3	NA	---	NA
			Inhalation Total								3.1E-09					0.0024
Soil Total											4.9E-06					3.1
											Total of Receptor Risks Across All Media					
											4.9E-06					3.1
																Total of Receptor Hazards Across All Media

APPENDIX E

TABLE E-8
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - CURRENT ADULT RESIDENT SOIL EXPOSURE
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Ely Copper Mine Surface Soil	Ingestion	Aluminum	10820	mg/kg	5.1E-03	mg/kg-day	NA	---	NA	1.5E-02	mg/kg-day	1.0E+00	mg/kg-day	0.015
				Antimony	1.3	mg/kg	6.0E-07	mg/kg-day	NA	---	NA	1.7E-06	mg/kg-day	4.0E-04	mg/kg-day	0.0044
				Arsenic	2.7	mg/kg	1.3E-06	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.9E-06	3.7E-06	mg/kg-day	3.0E-04	mg/kg-day	0.012
				Cadmium	0.99	mg/kg	4.6E-07	mg/kg-day	NA	---	NA	1.4E-06	mg/kg-day	1.0E-03	mg/kg-day	0.0014
				Chromium	44	mg/kg	2.1E-05	mg/kg-day	NA	---	NA	6.0E-05	mg/kg-day	1.5E+00	mg/kg-day	0.000040
				Cobalt	25	mg/kg	1.2E-05	mg/kg-day	NA	---	NA	3.4E-05	mg/kg-day	3.0E-04	mg/kg-day	0.11
				Copper	1086	mg/kg	5.1E-04	mg/kg-day	NA	---	NA	1.5E-03	mg/kg-day	4.0E-02	mg/kg-day	0.037
				Iron	66914	mg/kg	3.1E-02	mg/kg-day	NA	---	NA	9.2E-02	mg/kg-day	7.0E-01	mg/kg-day	0.13
				Manganese	449	mg/kg	2.1E-04	mg/kg-day	NA	---	NA	6.1E-04	mg/kg-day	7.0E-02	mg/kg-day	0.0088
				Mercury	0.15	mg/kg	7.0E-08	mg/kg-day	NA	---	NA	2.0E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00068
				Molybdenum	6.9	mg/kg	3.2E-06	mg/kg-day	NA	---	NA	9.4E-06	mg/kg-day	5.0E-03	mg/kg-day	0.0019
				Selenium	21	mg/kg	9.9E-06	mg/kg-day	NA	---	NA	2.9E-05	mg/kg-day	5.0E-03	mg/kg-day	0.0058
				Vanadium	56	mg/kg	2.6E-05	mg/kg-day	NA	---	NA	7.7E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0039
			Ingestion Total								1.9E-06					0.33
			Dermal	Aluminum	10820	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	1.3	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	6.0E-05	mg/kg-day	NA
				Arsenic	2.7	mg/kg	1.5E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.3E-07	4.5E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0015
				Cadmium	0.99	mg/kg	1.8E-09	mg/kg-day	NA	---	NA	5.4E-09	mg/kg-day	2.5E-05	mg/kg-day	0.00022
				Chromium	44	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	25	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	1086	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	66914	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	449	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Mercury	0.15	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.1E-05	mg/kg-day	NA
				Molybdenum	6.9	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Selenium	21	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	56	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								2.3E-07					0.0017
			Inhalation	Aluminum	10820	mg/kg	5.7E-04	µg/m^3	NA	---	NA	1.7E-06	mg/m^3	5.0E-03	mg/m^3	0.00033
				Antimony	1.3	mg/kg	6.7E-08	µg/m^3	NA	---	NA	2.0E-10	mg/m^3	NA	---	NA
				Arsenic	2.7	mg/kg	1.4E-07	µg/m^3	4.3E-03	(µg/m3)^-1	6.2E-10	4.2E-10	mg/m^3	1.5E-05	mg/m^3	0.000028
				Cadmium	1.0	mg/kg	5.2E-08	µg/m^3	1.8E-03	(µg/m3)^-1	9.3E-11	1.5E-10	mg/m^3	1.0E-05	mg/m^3	0.000015
				Chromium	44	mg/kg	2.3E-06	µg/m^3	NA	---	NA	6.7E-09	mg/m^3	NA	---	NA
				Cobalt	25	mg/kg	1.3E-06	µg/m^3	9.0E-03	(µg/m3)^-1	1.2E-08	3.8E-09	mg/m^3	6.0E-06	mg/m^3	0.00063
				Copper	1086	mg/kg	5.7E-05	µg/m^3	NA	---	NA	1.7E-07	mg/m^3	NA	---	NA
				Iron	66914	mg/kg	3.5E-03	µg/m^3	NA	---	NA	1.0E-05	mg/m^3	NA	---	NA
				Manganese	449	mg/kg	2.4E-05	µg/m^3	NA	---	NA	6.9E-08	mg/m^3	5.0E-05	mg/m^3	0.0014
				Mercury	0.15	mg/kg	7.8E-09	µg/m^3	NA	---	NA	2.3E-11	mg/m^3	3.0E-04	mg/m^3	0.00000008
				Molybdenum	6.9	mg/kg	3.6E-07	µg/m^3	NA	---	NA	1.1E-09	mg/m^3	NA	---	NA
				Selenium	21	mg/kg	1.1E-06	µg/m^3	NA	---	NA	3.2E-09	mg/m^3	2.0E-02	mg/m^3	0.00000016
				Vanadium	56	mg/kg	3.0E-06	µg/m^3	NA	---	NA	8.6E-09	mg/m^3	NA	---	NA
			Inhalation Total								1.2E-08					0.0024
Soil Total											2.2E-06					0.34
											Total of Receptor Risks Across All Media					
											2.2E-06					0.34
																Total of Receptor Hazards Across All Media

APPENDIX E

TABLE E-9
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - CURRENT CHILD RESIDENT SOIL EXPOSURE
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Ely Copper Mine Surface Soil	Ingestion	Aluminum	10820	mg/kg	5.9E-03	mg/kg-day	NA	---	NA	6.9E-02	mg/kg-day	1.0E+00	mg/kg-day	0.069
				Antimony	1.3	mg/kg	7.0E-07	mg/kg-day	NA	---	NA	8.2E-06	mg/kg-day	4.0E-04	mg/kg-day	0.020
				Arsenic	2.7	mg/kg	1.5E-06	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.2E-06	1.7E-05	mg/kg-day	3.0E-04	mg/kg-day	0.058
				Cadmium	0.99	mg/kg	5.4E-07	mg/kg-day	NA	---	NA	6.3E-06	mg/kg-day	1.0E-03	mg/kg-day	0.0063
				Chromium	44	mg/kg	2.4E-05	mg/kg-day	NA	---	NA	2.8E-04	mg/kg-day	1.5E+00	mg/kg-day	0.00019
				Cobalt	25	mg/kg	1.3E-05	mg/kg-day	NA	---	NA	1.6E-04	mg/kg-day	3.0E-04	mg/kg-day	0.52
				Copper	1086	mg/kg	6.0E-04	mg/kg-day	NA	---	NA	6.9E-03	mg/kg-day	4.0E-02	mg/kg-day	0.17
				Iron	66914	mg/kg	3.7E-02	mg/kg-day	NA	---	NA	4.3E-01	mg/kg-day	7.0E-01	mg/kg-day	0.61
				Manganese	449	mg/kg	2.5E-04	mg/kg-day	NA	---	NA	2.9E-03	mg/kg-day	7.0E-02	mg/kg-day	0.041
				Mercury	0.15	mg/kg	8.1E-08	mg/kg-day	NA	---	NA	9.5E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0032
				Molybdenum	6.9	mg/kg	3.8E-06	mg/kg-day	NA	---	NA	4.4E-05	mg/kg-day	5.0E-03	mg/kg-day	0.0088
				Selenium	21	mg/kg	1.2E-05	mg/kg-day	NA	---	NA	1.3E-04	mg/kg-day	5.0E-03	mg/kg-day	0.027
				Vanadium	56	mg/kg	3.1E-05	mg/kg-day	NA	---	NA	3.6E-04	mg/kg-day	2.0E-02	mg/kg-day	0.018
			Ingestion Total								2.2E-06					1.6
			Dermal	Aluminum	10820	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	1.3	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	6.0E-05	mg/kg-day	NA
				Arsenic	2.7	mg/kg	5.0E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	7.5E-08	5.9E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0020
				Cadmium	0.99	mg/kg	6.1E-10	mg/kg-day	NA	---	NA	7.1E-09	mg/kg-day	2.5E-05	mg/kg-day	0.00028
				Chromium	44	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	25	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	1086	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	66914	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	449	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Mercury	0.15	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.1E-05	mg/kg-day	NA
				Molybdenum	6.9	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Selenium	21	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	56	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								7.5E-08					0.0022
			Inhalation	Aluminum	10820	mg/kg	9.5E-05	µg/m^3	NA	---	NA	1.1E-06	mg/m^3	5.0E-03	mg/m^3	0.00022
				Antimony	1.3	mg/kg	1.1E-08	µg/m^3	NA	---	NA	1.3E-10	mg/m^3	NA	---	NA
				Arsenic	2.7	mg/kg	2.4E-08	µg/m^3	4.3E-03	(µg/m3)^-1	1.0E-10	2.8E-10	mg/m^3	1.5E-05	mg/m^3	0.000019
				Cadmium	1.0	mg/kg	8.7E-09	µg/m^3	1.8E-03	(µg/m3)^-1	1.6E-11	1.0E-10	mg/m^3	1.0E-05	mg/m^3	0.000010
				Chromium	44	mg/kg	3.8E-07	µg/m^3	NA	---	NA	4.5E-09	mg/m^3	NA	---	NA
				Cobalt	25	mg/kg	2.2E-07	µg/m^3	9.0E-03	(µg/m3)^-1	1.9E-09	2.5E-09	mg/m^3	6.0E-06	mg/m^3	0.00042
				Copper	1086	mg/kg	9.5E-06	µg/m^3	NA	---	NA	1.1E-07	mg/m^3	NA	---	NA
				Iron	66914	mg/kg	5.9E-04	µg/m^3	NA	---	NA	6.8E-06	mg/m^3	NA	---	NA
				Manganese	449	mg/kg	3.9E-06	µg/m^3	NA	---	NA	4.6E-08	mg/m^3	5.0E-05	mg/m^3	0.00092
				Mercury	0.15	mg/kg	1.3E-09	µg/m^3	NA	---	NA	1.5E-11	mg/m^3	3.0E-04	mg/m^3	0.00000005
				Molybdenum	6.9	mg/kg	6.0E-08	µg/m^3	NA	---	NA	7.0E-10	mg/m^3	NA	---	NA
				Selenium	21	mg/kg	1.8E-07	µg/m^3	NA	---	NA	2.2E-09	mg/m^3	2.0E-02	mg/m^3	0.00000011
				Vanadium	56	mg/kg	4.9E-07	µg/m^3	NA	---	NA	5.8E-09	mg/m^3	NA	---	NA
			Inhalation Total								2.1E-09					0.0016
Soil Total											2.3E-06					1.6
											Total of Receptor Risks Across All Media					
											2.3E-06					1.6
												Total of Receptor Hazards Across All Media				

APPENDIX E

TABLE E-10
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - CURRENT ADULT RESIDENT SOIL EXPOSURE
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Surface Soil	Ely Copper Mine Surface Soil	Ingestion	Aluminum	10820	mg/kg	9.5E-04	mg/kg-day	NA	---	NA	7.4E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0074
				Antimony	1.3	mg/kg	1.1E-07	mg/kg-day	NA	---	NA	8.7E-07	mg/kg-day	4.0E-04	mg/kg-day	0.0022
				Arsenic	2.7	mg/kg	2.4E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	3.6E-07	1.9E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0062
				Cadmium	0.99	mg/kg	8.7E-08	mg/kg-day	NA	---	NA	6.8E-07	mg/kg-day	1.0E-03	mg/kg-day	0.00068
				Chromium	44	mg/kg	3.8E-06	mg/kg-day	NA	---	NA	3.0E-05	mg/kg-day	1.5E+00	mg/kg-day	0.000020
				Cobalt	25	mg/kg	2.2E-06	mg/kg-day	NA	---	NA	1.7E-05	mg/kg-day	3.0E-04	mg/kg-day	0.056
				Copper	1086	mg/kg	9.6E-05	mg/kg-day	NA	---	NA	7.4E-04	mg/kg-day	4.0E-02	mg/kg-day	0.019
				Iron	66914	mg/kg	5.9E-03	mg/kg-day	NA	---	NA	4.6E-02	mg/kg-day	7.0E-01	mg/kg-day	0.065
				Manganese	449	mg/kg	4.0E-05	mg/kg-day	NA	---	NA	3.1E-04	mg/kg-day	7.0E-02	mg/kg-day	0.0044
				Mercury	0.15	mg/kg	1.3E-08	mg/kg-day	NA	---	NA	1.0E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00034
				Molybdenum	6.9	mg/kg	6.0E-07	mg/kg-day	NA	---	NA	4.7E-06	mg/kg-day	5.0E-03	mg/kg-day	0.00094
				Selenium	21	mg/kg	1.9E-06	mg/kg-day	NA	---	NA	1.4E-05	mg/kg-day	5.0E-03	mg/kg-day	0.0029
				Vanadium	56	mg/kg	5.0E-06	mg/kg-day	NA	---	NA	3.9E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0019
			Ingestion Total								3.6E-07					0.17
			Dermal	Aluminum	10820	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	1.3	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	6.0E-05	mg/kg-day	NA
				Arsenic	2.7	mg/kg	8.2E-09	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.2E-08	6.4E-08	mg/kg-day	3.0E-04	mg/kg-day	0.00021
				Cadmium	0.99	mg/kg	9.9E-11	mg/kg-day	NA	---	NA	7.7E-10	mg/kg-day	2.5E-05	mg/kg-day	0.000031
				Chromium	44	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	25	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	1086	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	66914	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	449	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Mercury	0.15	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.1E-05	mg/kg-day	NA
				Molybdenum	6.9	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Selenium	21	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	56	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								1.2E-08					0.00024
			Inhalation	Aluminum	10820	mg/kg	1.4E-04	µg/m^3	NA	---	NA	1.1E-06	mg/m^3	5.0E-03	mg/m^3	0.00022
				Antimony	1.3	mg/kg	1.7E-08	µg/m^3	NA	---	NA	1.3E-10	mg/m^3	NA	---	NA
				Arsenic	2.7	mg/kg	3.6E-08	µg/m^3	4.3E-03	(µg/m3)^-1	1.5E-10	2.8E-10	mg/m^3	1.5E-05	mg/m^3	0.000019
				Cadmium	1.0	mg/kg	1.3E-08	µg/m^3	1.8E-03	(µg/m3)^-1	2.3E-11	1.0E-10	mg/m^3	1.0E-05	mg/m^3	0.000010
				Chromium	44	mg/kg	5.7E-07	µg/m^3	NA	---	NA	4.5E-09	mg/m^3	NA	---	NA
				Cobalt	25	mg/kg	3.2E-07	µg/m^3	9.0E-03	(µg/m3)^-1	2.9E-09	2.5E-09	mg/m^3	6.0E-06	mg/m^3	0.00042
				Copper	1086	mg/kg	1.4E-05	µg/m^3	NA	---	NA	1.1E-07	mg/m^3	NA	---	NA
				Iron	66914	mg/kg	8.8E-04	µg/m^3	NA	---	NA	6.8E-06	mg/m^3	NA	---	NA
				Manganese	449	mg/kg	5.9E-06	µg/m^3	NA	---	NA	4.6E-08	mg/m^3	5.0E-05	mg/m^3	0.00092
				Mercury	0.15	mg/kg	1.9E-09	µg/m^3	NA	---	NA	1.5E-11	mg/m^3	3.0E-04	mg/m^3	0.00000005
				Molybdenum	6.9	mg/kg	9.0E-08	µg/m^3	NA	---	NA	7.0E-10	mg/m^3	NA	---	NA
				Selenium	21	mg/kg	2.8E-07	µg/m^3	NA	---	NA	2.2E-09	mg/m^3	2.0E-02	mg/m^3	0.00000011
				Vanadium	56	mg/kg	7.4E-07	µg/m^3	NA	---	NA	5.8E-09	mg/m^3	NA	---	NA
			Inhalation Total								3.1E-09					0.0016
Soil Total											3.8E-07					0.17
											Total of Receptor Risks Across All Media	3.8E-07		Total of Receptor Hazards Across All Media		0.17

APPENDIX E

TABLE E-11
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - FUTURE CHILD RESIDENT SOIL EXPOSURE
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Total Soil	Ely Copper Mine Total Soil	Ingestion	Aluminum	11814	mg/kg	1.3E-02	mg/kg-day	NA	---	NA	1.5E-01	mg/kg-day	1.0E+00	mg/kg-day	0.15
				Antimony	1.1	mg/kg	1.2E-06	mg/kg-day	NA	---	NA	1.3E-05	mg/kg-day	4.0E-04	mg/kg-day	0.034
				Arsenic	2.3	mg/kg	2.5E-06	mg/kg-day	1.5E+00	(mg/kg-day)^-1	3.8E-06	3.0E-05	mg/kg-day	3.0E-04	mg/kg-day	0.099
				Cadmium	1.86	mg/kg	2.0E-06	mg/kg-day	NA	---	NA	2.4E-05	mg/kg-day	1.0E-03	mg/kg-day	0.024
				Chromium	48	mg/kg	5.3E-05	mg/kg-day	NA	---	NA	6.2E-04	mg/kg-day	1.5E+00	mg/kg-day	0.00041
				Cobalt	50	mg/kg	5.5E-05	mg/kg-day	NA	---	NA	6.4E-04	mg/kg-day	3.0E-04	mg/kg-day	2.1
				Copper	1887	mg/kg	2.1E-03	mg/kg-day	NA	---	NA	2.4E-02	mg/kg-day	4.0E-02	mg/kg-day	0.60
				Iron	67413	mg/kg	7.4E-02	mg/kg-day	NA	---	NA	8.6E-01	mg/kg-day	7.0E-01	mg/kg-day	1.2
				Manganese	411	mg/kg	4.5E-04	mg/kg-day	NA	---	NA	5.3E-03	mg/kg-day	7.0E-02	mg/kg-day	0.075
				Mercury	0.13	mg/kg	1.4E-07	mg/kg-day	NA	---	NA	1.6E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0053
				Molybdenum	6.4	mg/kg	7.0E-06	mg/kg-day	NA	---	NA	8.2E-05	mg/kg-day	5.0E-03	mg/kg-day	0.016
				Nickel	27.3	mg/kg	3.0E-05	mg/kg-day	NA	---	NA	3.5E-04	mg/kg-day	2.0E-02	mg/kg-day	0.017
				Selenium	22	mg/kg	2.4E-05	mg/kg-day	NA	---	NA	2.8E-04	mg/kg-day	5.0E-03	mg/kg-day	0.056
				Vanadium	57	mg/kg	6.2E-05	mg/kg-day	NA	---	NA	7.3E-04	mg/kg-day	2.0E-02	mg/kg-day	0.036
				Zinc	262	mg/kg	2.9E-04	mg/kg-day	NA	---	NA	3.3E-03	mg/kg-day	3.0E-01	mg/kg-day	0.011
			Ingestion Total								3.8E-06					4.5
			Dermal	Aluminum	11814	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	1.1	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	6.0E-05	mg/kg-day	NA
				Arsenic	2.3	mg/kg	2.1E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	3.2E-07	2.5E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0083
				Cadmium	1.86	mg/kg	5.7E-09	mg/kg-day	NA	---	NA	6.7E-08	mg/kg-day	2.5E-05	mg/kg-day	0.0027
				Chromium	48	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	50	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	1887	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	67413	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	411	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Mercury	0.13	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.1E-05	mg/kg-day	NA
				Molybdenum	6.4	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Nickel	27.3	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	8.0E-04	mg/kg-day	NA
				Selenium	22	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	57	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
				Zinc	262	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-01	mg/kg-day	NA
			Dermal Total								3.2E-07					0.0110
			Inhalation	Aluminum	11814	mg/kg	1.6E-04	µg/m^3	NA	---	NA	1.8E-06	mg/m^3	5.0E-03	mg/m^3	0.00036
				Antimony	1.1	mg/kg	1.4E-08	µg/m^3	NA	---	NA	1.6E-10	mg/m^3	NA	---	NA
				Arsenic	2.3	mg/kg	3.1E-08	µg/m^3	4.3E-03	(µg/m3)^-1	1.3E-10	3.6E-10	mg/m^3	1.5E-05	mg/m^3	0.000024
				Cadmium	1.9	mg/kg	2.4E-08	µg/m^3	1.8E-03	(µg/m3)^-1	4.4E-11	2.9E-10	mg/m^3	1.0E-05	mg/m^3	0.000029
				Chromium	48	mg/kg	6.3E-07	µg/m^3	NA	---	NA	7.4E-09	mg/m^3	NA	---	NA
				Cobalt	50	mg/kg	6.6E-07	µg/m^3	9.0E-03	(µg/m3)^-1	5.9E-09	7.7E-09	mg/m^3	6.0E-06	mg/m^3	0.0013
				Copper	1887	mg/kg	2.5E-05	µg/m^3	NA	---	NA	2.9E-07	mg/m^3	NA	---	NA
				Iron	67413	mg/kg	8.9E-04	µg/m^3	NA	---	NA	1.0E-05	mg/m^3	NA	---	NA
				Manganese	411	mg/kg	5.4E-06	µg/m^3	NA	---	NA	6.3E-08	mg/m^3	5.0E-05	mg/m^3	0.0013
				Mercury	0.13	mg/kg	1.6E-09	µg/m^3	NA	---	NA	1.9E-11	mg/m^3	3.0E-04	mg/m^3	0.00000006
				Molybdenum	6.4	mg/kg	8.4E-08	µg/m^3	NA	---	NA	9.8E-10	mg/m^3	NA	---	NA
				Nickel	27.3	mg/kg	3.6E-07	µg/m^3	2.6E-04	(µg/m3)^-1	9.3E-11	4.2E-09	mg/m^3	9.0E-05	mg/m^3	0.000047
				Selenium	22	mg/kg	2.9E-07	µg/m^3	NA	---	NA	3.3E-09	mg/m^3	2.0E-02	mg/m^3	0.00000017
				Vanadium	57	mg/kg	7.5E-07	µg/m^3	NA	---	NA	8.7E-09	mg/m^3	NA	---	NA
				Zinc	262	mg/kg	3.4E-06	µg/m^3	NA	---	NA	4.0E-08	mg/m^3	NA	---	NA
			Inhalation Total								6.2E-09					0.0030
Soil Total											4.1E-06					4.5
											Total of Receptor Risks Across All Media					Total of Receptor Hazards Across All Media
											4.1E-06					4.5

APPENDIX E

TABLE E-12
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - FUTURE ADULT RESIDENT SOIL EXPOSURE
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Total Soil	Ely Copper Mine Total Soil	Ingestion	Aluminum	11814	mg/kg	5.5E-03	mg/kg-day	NA	---	NA	1.6E-02	mg/kg-day	1.0E+00	mg/kg-day	0.016
				Antimony	1.1	mg/kg	5.0E-07	mg/kg-day	NA	---	NA	1.4E-06	mg/kg-day	4.0E-04	mg/kg-day	0.0036
				Arsenic	2.3	mg/kg	1.1E-06	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.6E-06	3.2E-06	mg/kg-day	3.0E-04	mg/kg-day	0.011
				Cadmium	1.86	mg/kg	8.7E-07	mg/kg-day	NA	---	NA	2.5E-06	mg/kg-day	1.0E-03	mg/kg-day	0.0025
				Chromium	48	mg/kg	2.3E-05	mg/kg-day	NA	---	NA	6.6E-05	mg/kg-day	1.5E+00	mg/kg-day	0.000044
				Cobalt	50	mg/kg	2.4E-05	mg/kg-day	NA	---	NA	6.9E-05	mg/kg-day	3.0E-04	mg/kg-day	0.23
				Copper	1887	mg/kg	8.9E-04	mg/kg-day	NA	---	NA	2.6E-03	mg/kg-day	4.0E-02	mg/kg-day	0.065
				Iron	67413	mg/kg	3.2E-02	mg/kg-day	NA	---	NA	9.2E-02	mg/kg-day	7.0E-01	mg/kg-day	0.13
				Manganese	411	mg/kg	1.9E-04	mg/kg-day	NA	---	NA	5.6E-04	mg/kg-day	7.0E-02	mg/kg-day	0.0080
				Mercury	0.13	mg/kg	5.9E-08	mg/kg-day	NA	---	NA	1.7E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00057
				Molybdenum	6.4	mg/kg	3.0E-06	mg/kg-day	NA	---	NA	8.8E-06	mg/kg-day	5.0E-03	mg/kg-day	0.0018
				Nickel	27.3	mg/kg	1.3E-05	mg/kg-day	NA	---	NA	3.7E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0019
				Selenium	22	mg/kg	1.0E-05	mg/kg-day	NA	---	NA	3.0E-05	mg/kg-day	5.0E-03	mg/kg-day	0.0060
				Vanadium	57	mg/kg	2.7E-05	mg/kg-day	NA	---	NA	7.8E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0039
				Zinc	262	mg/kg	1.2E-04	mg/kg-day	NA	---	NA	3.6E-04	mg/kg-day	3.0E-01	mg/kg-day	0.0012
			Ingestion Total								1.6E-06					0.48
			Dermal	Aluminum	11814	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	1.1	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	6.0E-05	mg/kg-day	NA
				Arsenic	2.3	mg/kg	1.3E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.0E-07	3.8E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0013
				Cadmium	1.86	mg/kg	3.5E-09	mg/kg-day	NA	---	NA	1.0E-08	mg/kg-day	2.5E-05	mg/kg-day	0.00041
				Chromium	48	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	50	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	1887	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	67413	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	411	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Mercury	0.13	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.1E-05	mg/kg-day	NA
				Molybdenum	6.4	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Nickel	27.3	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	8.0E-04	mg/kg-day	NA
				Selenium	22	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	57	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
				Zinc	262	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-01	mg/kg-day	NA
			Dermal Total								2.0E-07					0.0017
			Inhalation	Aluminum	11814	mg/kg	6.2E-04	µg/m^3	NA	---	NA	1.8E-06	mg/m^3	5.0E-03	mg/m^3	0.00036
				Antimony	1.1	mg/kg	5.5E-08	µg/m^3	NA	---	NA	1.6E-10	mg/m^3	NA	---	NA
				Arsenic	2.3	mg/kg	1.2E-07	µg/m^3	4.3E-03	(µg/m3)^-1	5.3E-10	3.6E-10	mg/m^3	1.5E-05	mg/m^3	0.000024
				Cadmium	1.9	mg/kg	9.8E-08	µg/m^3	1.8E-03	(µg/m3)^-1	1.8E-10	2.9E-10	mg/m^3	1.0E-05	mg/m^3	0.000029
				Chromium	48	mg/kg	2.5E-06	µg/m^3	NA	---	NA	7.4E-09	mg/m^3	NA	---	NA
				Cobalt	50	mg/kg	2.6E-06	µg/m^3	9.0E-03	(µg/m3)^-1	2.4E-08	7.7E-09	mg/m^3	6.0E-06	mg/m^3	0.0013
				Copper	1887	mg/kg	9.9E-05	µg/m^3	NA	---	NA	2.9E-07	mg/m^3	NA	---	NA
				Iron	67413	mg/kg	3.5E-03	µg/m^3	NA	---	NA	1.0E-05	mg/m^3	NA	---	NA
				Manganese	411	mg/kg	2.2E-05	µg/m^3	NA	---	NA	6.3E-08	mg/m^3	5.0E-05	mg/m^3	0.0013
				Mercury	0.13	mg/kg	6.6E-09	µg/m^3	NA	---	NA	1.9E-11	mg/m^3	3.0E-04	mg/m^3	0.00000006
				Molybdenum	6.4	mg/kg	3.4E-07	µg/m^3	NA	---	NA	9.8E-10	mg/m^3	NA	---	NA
				Nickel	27.3	mg/kg	1.4E-06	µg/m^3	2.6E-04	(µg/m3)^-1	3.7E-10	4.2E-09	mg/m^3	9.0E-05	mg/m^3	0.000047
				Selenium	22	mg/kg	1.1E-06	µg/m^3	NA	---	NA	3.3E-09	mg/m^3	2.0E-02	mg/m^3	0.00000017
				Vanadium	57	mg/kg	3.0E-06	µg/m^3	NA	---	NA	8.7E-09	mg/m^3	NA	---	NA
				Zinc	262	mg/kg	1.4E-05	µg/m^3	NA	---	NA	4.0E-08	mg/m^3	NA	---	NA
			Inhalation Total								2.5E-08					0.0030
Soil Total											1.9E-06					0.49
											Total of Receptor Risks Across All Media 1.9E-06					Total of Receptor Hazards Across All Media 0.49

APPENDIX E

TABLE E-13
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - FUTURE CHILD RESIDENT SOIL EXPOSURE
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Total Soil	Ely Copper Mine Total Soil	Ingestion	Aluminum	11814	mg/kg	6.5E-03	mg/kg-day	NA	---	NA	7.6E-02	mg/kg-day	1.0E+00	mg/kg-day	0.076
				Antimony	1.1	mg/kg	5.8E-07	mg/kg-day	NA	---	NA	6.7E-06	mg/kg-day	4.0E-04	mg/kg-day	0.017
				Arsenic	2.3	mg/kg	1.3E-06	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.9E-06	1.5E-05	mg/kg-day	3.0E-04	mg/kg-day	0.050
				Cadmium	1.86	mg/kg	1.0E-06	mg/kg-day	NA	---	NA	1.2E-05	mg/kg-day	1.0E-03	mg/kg-day	0.012
				Chromium	48	mg/kg	2.6E-05	mg/kg-day	NA	---	NA	3.1E-04	mg/kg-day	1.5E+00	mg/kg-day	0.00021
				Cobalt	50	mg/kg	2.8E-05	mg/kg-day	NA	---	NA	3.2E-04	mg/kg-day	3.0E-04	mg/kg-day	1.1
				Copper	1887	mg/kg	1.0E-03	mg/kg-day	NA	---	NA	1.2E-02	mg/kg-day	4.0E-02	mg/kg-day	0.30
				Iron	67413	mg/kg	3.7E-02	mg/kg-day	NA	---	NA	4.3E-01	mg/kg-day	7.0E-01	mg/kg-day	0.62
				Manganese	411	mg/kg	2.3E-04	mg/kg-day	NA	---	NA	2.6E-03	mg/kg-day	7.0E-02	mg/kg-day	0.038
				Mercury	0.13	mg/kg	6.8E-08	mg/kg-day	NA	---	NA	8.0E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0027
				Molybdenum	6.4	mg/kg	3.5E-06	mg/kg-day	NA	---	NA	4.1E-05	mg/kg-day	5.0E-03	mg/kg-day	0.0082
				Nickel	27.3	mg/kg	1.5E-05	mg/kg-day	NA	---	NA	1.7E-04	mg/kg-day	2.0E-02	mg/kg-day	0.0087
				Selenium	22	mg/kg	1.2E-05	mg/kg-day	NA	---	NA	1.4E-04	mg/kg-day	5.0E-03	mg/kg-day	0.028
				Vanadium	57	mg/kg	3.1E-05	mg/kg-day	NA	---	NA	3.6E-04	mg/kg-day	2.0E-02	mg/kg-day	0.018
				Zinc	262	mg/kg	1.4E-04	mg/kg-day	NA	---	NA	1.7E-03	mg/kg-day	3.0E-01	mg/kg-day	0.0056
			Ingestion Total								1.9E-06					2.3
			Dermal	Aluminum	11814	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	1.1	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	6.0E-05	mg/kg-day	NA
				Arsenic	2.3	mg/kg	4.3E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	6.4E-08	5.0E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0017
				Cadmium	1.86	mg/kg	1.1E-09	mg/kg-day	NA	---	NA	1.3E-08	mg/kg-day	2.5E-05	mg/kg-day	0.00053
				Chromium	48	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	50	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	1887	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	67413	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	411	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Mercury	0.13	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.1E-05	mg/kg-day	NA
				Molybdenum	6.4	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Nickel	27.3	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	8.0E-04	mg/kg-day	NA
				Selenium	22	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	57	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
				Zinc	262	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-01	mg/kg-day	NA
			Dermal Total								6.4E-08					0.0022
			Inhalation	Aluminum	11814	mg/kg	1.0E-04	µg/m^3	NA	---	NA	1.2E-06	mg/m^3	5.0E-03	mg/m^3	0.00024
				Antimony	1.1	mg/kg	9.2E-09	µg/m^3	NA	---	NA	1.1E-10	mg/m^3	NA	---	NA
				Arsenic	2.3	mg/kg	2.0E-08	µg/m^3	4.3E-03	(µg/m3)^-1	8.8E-11	2.4E-10	mg/m^3	1.5E-05	mg/m^3	0.000016
				Cadmium	1.9	mg/kg	1.6E-08	µg/m^3	1.8E-03	(µg/m3)^-1	2.9E-11	1.9E-10	mg/m^3	1.0E-05	mg/m^3	0.000019
				Chromium	48	mg/kg	4.2E-07	µg/m^3	NA	---	NA	4.9E-09	mg/m^3	NA	---	NA
				Cobalt	50	mg/kg	4.4E-07	µg/m^3	9.0E-03	(µg/m3)^-1	4.0E-09	5.1E-09	mg/m^3	6.0E-06	mg/m^3	0.00086
				Copper	1887	mg/kg	1.7E-05	µg/m^3	NA	---	NA	1.9E-07	mg/m^3	NA	---	NA
				Iron	67413	mg/kg	5.9E-04	µg/m^3	NA	---	NA	6.9E-06	mg/m^3	NA	---	NA
				Manganese	411	mg/kg	3.6E-06	µg/m^3	NA	---	NA	4.2E-08	mg/m^3	5.0E-05	mg/m^3	0.00084
				Mercury	0.13	mg/kg	1.1E-09	µg/m^3	NA	---	NA	1.3E-11	mg/m^3	3.0E-04	mg/m^3	0.00000004
				Molybdenum	6.4	mg/kg	5.6E-08	µg/m^3	NA	---	NA	6.5E-10	mg/m^3	NA	---	NA
				Nickel	27.3	mg/kg	2.4E-07	µg/m^3	2.6E-04	(µg/m3)^-1	6.2E-11	2.8E-09	mg/m^3	9.0E-05	mg/m^3	0.000031
				Selenium	22	mg/kg	1.9E-07	µg/m^3	NA	---	NA	2.2E-09	mg/m^3	2.0E-02	mg/m^3	0.00000011
				Vanadium	57	mg/kg	5.0E-07	µg/m^3	NA	---	NA	5.8E-09	mg/m^3	NA	---	NA
				Zinc	262	mg/kg	2.3E-06	µg/m^3	NA	---	NA	2.7E-08	mg/m^3	NA	---	NA
			Inhalation Total								4.1E-09					0.0020
Soil Total											2.0E-06					2.3
											Total of Receptor Risks Across All Media					
											2.0E-06					2.3
																Total of Receptor Hazards Across All Media

APPENDIX E

TABLE E-14
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - FUTURE ADULT RESIDENT SOIL EXPOSURE
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Soil	Total Soil	Ely Copper Mine Total Soil	Ingestion	Aluminum	11814	mg/kg	1.0E-03	mg/kg-day	NA	---	NA	8.1E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0081
				Antimony	1.1	mg/kg	9.3E-08	mg/kg-day	NA	---	NA	7.2E-07	mg/kg-day	4.0E-04	mg/kg-day	0.0018
				Arsenic	2.3	mg/kg	2.0E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	3.1E-07	1.6E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0053
				Cadmium	1.86	mg/kg	1.6E-07	mg/kg-day	NA	---	NA	1.3E-06	mg/kg-day	1.0E-03	mg/kg-day	0.0013
				Chromium	48	mg/kg	4.2E-06	mg/kg-day	NA	---	NA	3.3E-05	mg/kg-day	1.5E+00	mg/kg-day	0.000022
				Cobalt	50	mg/kg	4.4E-06	mg/kg-day	NA	---	NA	3.4E-05	mg/kg-day	3.0E-04	mg/kg-day	0.11
				Copper	1887	mg/kg	1.7E-04	mg/kg-day	NA	---	NA	1.3E-03	mg/kg-day	4.0E-02	mg/kg-day	0.032
				Iron	67413	mg/kg	5.9E-03	mg/kg-day	NA	---	NA	4.6E-02	mg/kg-day	7.0E-01	mg/kg-day	0.066
				Manganese	411	mg/kg	3.6E-05	mg/kg-day	NA	---	NA	2.8E-04	mg/kg-day	7.0E-02	mg/kg-day	0.0040
				Mercury	0.13	mg/kg	1.1E-08	mg/kg-day	NA	---	NA	8.6E-08	mg/kg-day	3.0E-04	mg/kg-day	0.00029
				Molybdenum	6.4	mg/kg	5.6E-07	mg/kg-day	NA	---	NA	4.4E-06	mg/kg-day	5.0E-03	mg/kg-day	0.00088
				Nickel	27.3	mg/kg	2.4E-06	mg/kg-day	NA	---	NA	1.9E-05	mg/kg-day	2.0E-02	mg/kg-day	0.00094
				Selenium	22	mg/kg	1.9E-06	mg/kg-day	NA	---	NA	1.5E-05	mg/kg-day	5.0E-03	mg/kg-day	0.0030
				Vanadium	57	mg/kg	5.0E-06	mg/kg-day	NA	---	NA	3.9E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0019
				Zinc	262	mg/kg	2.3E-05	mg/kg-day	NA	---	NA	1.8E-04	mg/kg-day	3.0E-01	mg/kg-day	0.00060
			Ingestion Total								3.1E-07					0.24
			Dermal	Aluminum	11814	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	1.1	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	6.0E-05	mg/kg-day	NA
				Arsenic	2.3	mg/kg	7.0E-09	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.0E-08	5.4E-08	mg/kg-day	3.0E-04	mg/kg-day	0.00018
				Cadmium	1.86	mg/kg	1.9E-10	mg/kg-day	NA	---	NA	1.5E-09	mg/kg-day	2.5E-05	mg/kg-day	0.000058
				Chromium	48	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	50	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	1887	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	67413	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	411	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Mercury	0.13	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.1E-05	mg/kg-day	NA
				Molybdenum	6.4	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Nickel	27.3	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	8.0E-04	mg/kg-day	NA
				Selenium	22	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	57	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
				Zinc	262	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-01	mg/kg-day	NA
			Dermal Total								1.0E-08					0.00024
			Inhalation	Aluminum	11814	mg/kg	1.6E-04	µg/m^3	NA	---	NA	1.2E-06	mg/m^3	5.0E-03	mg/m^3	0.00024
				Antimony	1.1	mg/kg	1.4E-08	µg/m^3	NA	---	NA	1.1E-10	mg/m^3	NA	---	NA
				Arsenic	2.3	mg/kg	3.1E-08	µg/m^3	4.3E-03	(µg/m3)^-1	1.3E-10	2.4E-10	mg/m^3	1.5E-05	mg/m^3	0.000016
				Cadmium	1.9	mg/kg	2.4E-08	µg/m^3	1.8E-03	(µg/m3)^-1	4.4E-11	1.9E-10	mg/m^3	1.0E-05	mg/m^3	0.000019
				Chromium	48	mg/kg	6.3E-07	µg/m^3	NA	---	NA	4.9E-09	mg/m^3	NA	---	NA
				Cobalt	50	mg/kg	6.6E-07	µg/m^3	9.0E-03	(µg/m3)^-1	5.9E-09	5.1E-09	mg/m^3	6.0E-06	mg/m^3	0.00086
				Copper	1887	mg/kg	2.5E-05	µg/m^3	NA	---	NA	1.9E-07	mg/m^3	NA	---	NA
				Iron	67413	mg/kg	8.9E-04	µg/m^3	NA	---	NA	6.9E-06	mg/m^3	NA	---	NA
				Manganese	411	mg/kg	5.4E-06	µg/m^3	NA	---	NA	4.2E-08	mg/m^3	5.0E-05	mg/m^3	0.00084
				Mercury	0.13	mg/kg	1.6E-09	µg/m^3	NA	---	NA	1.3E-11	mg/m^3	3.0E-04	mg/m^3	0.00000004
				Molybdenum	6.4	mg/kg	8.4E-08	µg/m^3	NA	---	NA	6.5E-10	mg/m^3	NA	---	NA
				Nickel	27.3	mg/kg	3.6E-07	µg/m^3	2.6E-04	(µg/m3)^-1	9.3E-11	2.8E-09	mg/m^3	9.0E-05	mg/m^3	0.000031
				Selenium	22	mg/kg	2.9E-07	µg/m^3	NA	---	NA	2.2E-09	mg/m^3	2.0E-02	mg/m^3	0.00000011
				Vanadium	57	mg/kg	7.5E-07	µg/m^3	NA	---	NA	5.8E-09	mg/m^3	NA	---	NA
				Zinc	262	mg/kg	3.4E-06	µg/m^3	NA	---	NA	2.7E-08	mg/m^3	NA	---	NA
			Inhalation Total								6.2E-09					0.0020
Soil Total											3.2E-07					0.24
											Total of Receptor Risks Across All Media					Total of Receptor Hazards Across All Media
											3.2E-07					0.24

APPENDIX E

TABLE E-15
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADOLESCENT SWIMMER/WADER ELY BROOK
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ely Brook	Sediment	Ely Brook Sediment	Ingestion	Aluminum	34526	mg/kg	4.6E-04	mg/kg-day	NA	---	NA	4.0E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0040
				Arsenic	3.7	mg/kg	4.9E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	7.3E-08	4.2E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0014
				Chromium	54	mg/kg	7.1E-07	mg/kg-day	NA	---	NA	6.3E-06	mg/kg-day	1.5E+00	mg/kg-day	0.0000042
				Cobalt	45	mg/kg	6.0E-07	mg/kg-day	NA	---	NA	5.2E-06	mg/kg-day	3.0E-04	mg/kg-day	0.017
				Copper	3118	mg/kg	4.1E-05	mg/kg-day	NA	---	NA	3.6E-04	mg/kg-day	4.0E-02	mg/kg-day	0.0090
				Iron	74219	mg/kg	9.8E-04	mg/kg-day	NA	---	NA	8.6E-03	mg/kg-day	7.0E-01	mg/kg-day	0.012
				Lithium	34	mg/kg	4.5E-07	mg/kg-day	NA	---	NA	4.0E-06	mg/kg-day	2.0E-03	mg/kg-day	0.0020
				Manganese	973	mg/kg	1.3E-05	mg/kg-day	NA	---	NA	1.1E-04	mg/kg-day	7.0E-02	mg/kg-day	0.0016
				Selenium	35	mg/kg	4.6E-07	mg/kg-day	NA	---	NA	4.1E-06	mg/kg-day	5.0E-03	mg/kg-day	0.00081
				Vanadium	75	mg/kg	9.9E-07	mg/kg-day	NA	---	NA	8.7E-06	mg/kg-day	2.0E-02	mg/kg-day	0.00043
			Ingestion Total								7.3E-08					0.049
			Dermal	Aluminum	34526	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	3.7	mg/kg	2.7E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	4.1E-08	2.4E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00080
				Chromium	54	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	45	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	3118	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	74219	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	34	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	973	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Selenium	35	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	75	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								4.1E-08					0.00080
Sediment Total											1.1E-07					0.050
Ely Brook	Surface Water	Ely Brook Surface Water	Ingestion	Aluminum	12885	µg/L	1.7E-04	mg/kg-day	NA	---	NA	1.5E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0015
				Arsenic	0.246	µg/L	3.3E-09	mg/kg-day	1.5E+00	(mg/kg-day)^-1	4.9E-09	2.9E-08	mg/kg-day	3.0E-04	mg/kg-day	0.00010
				Cadmium	7.55	µg/L	1.0E-07	mg/kg-day	NA	---	NA	8.8E-07	mg/kg-day	1.0E-03	mg/kg-day	0.00088
				Chromium	10.35	µg/L	1.4E-07	mg/kg-day	NA	---	NA	1.2E-06	mg/kg-day	1.5E+00	mg/kg-day	0.00000080
				Cobalt	153.2	µg/L	2.0E-06	mg/kg-day	NA	---	NA	1.8E-05	mg/kg-day	3.0E-04	mg/kg-day	0.059
				Copper	4391	µg/L	5.8E-05	mg/kg-day	NA	---	NA	5.1E-04	mg/kg-day	4.0E-02	mg/kg-day	0.013
				Iron	6354	µg/L	8.4E-05	mg/kg-day	NA	---	NA	7.4E-04	mg/kg-day	7.0E-01	mg/kg-day	0.0011
				Lithium	6.299	µg/L	8.3E-08	mg/kg-day	NA	---	NA	7.3E-07	mg/kg-day	2.0E-03	mg/kg-day	0.00037
				Manganese	776.6	µg/L	1.0E-05	mg/kg-day	NA	---	NA	9.0E-05	mg/kg-day	2.4E-02	mg/kg-day	0.0038
				Mercury	0.099	µg/L	1.3E-09	mg/kg-day	NA	---	NA	1.1E-08	mg/kg-day	3.0E-04	mg/kg-day	0.000038
				Nickel	57.27	µg/L	7.6E-07	mg/kg-day	NA	---	NA	6.6E-06	mg/kg-day	2.0E-02	mg/kg-day	0.00033
				Vanadium	1.545	µg/L	2.0E-08	mg/kg-day	NA	---	NA	1.8E-07	mg/kg-day	2.0E-02	mg/kg-day	0.0000090
				Zinc	1073	µg/L	1.4E-05	mg/kg-day	NA	---	NA	1.2E-04	mg/kg-day	3.0E-01	mg/kg-day	0.00041
			Ingestion Total								4.9E-09					0.080
			Dermal	Aluminum	12885	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	0.246	µg/L	9.7E-10	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.5E-09	8.5E-09	mg/kg-day	3.0E-04	mg/kg-day	0.000028
				Cadmium	7.55	µg/L	3.0E-08	mg/kg-day	NA	---	NA	2.6E-07	mg/kg-day	2.5E-05	mg/kg-day	0.010
				Chromium	10.35	µg/L	8.2E-08	mg/kg-day	NA	---	NA	7.2E-07	mg/kg-day	2.0E-02	mg/kg-day	0.000037
				Cobalt	153.2	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	4391	µg/L	1.7E-05	mg/kg-day	NA	---	NA	1.5E-04	mg/kg-day	4.0E-02	mg/kg-day	0.0038
				Iron	6354	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	6.299	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	776.6	µg/L	3.1E-06	mg/kg-day	NA	---	NA	2.7E-05	mg/kg-day	9.6E-04	mg/kg-day	0.028
				Mercury	0.099	µg/L	3.9E-10	mg/kg-day	NA	---	NA	3.4E-09	mg/kg-day	2.1E-05	mg/kg-day	0.00016
				Nickel	57.27	µg/L	4.5E-08	mg/kg-day	NA	---	NA	4.0E-07	mg/kg-day	8.0E-04	mg/kg-day	0.00049
				Vanadium	1.545	µg/L	6.1E-09	mg/kg-day	NA	---	NA	5.3E-08	mg/kg-day	5.2E-04	mg/kg-day	0.00010
				Zinc	1073	µg/L	2.5E-06	mg/kg-day	NA	---	NA	2.2E-05	mg/kg-day	3.0E-01	mg/kg-day	0.000074
			Dermal Total								1.5E-09					0.043
Surface Water Total											6.3E-09					0.12
										Total of Receptor Risks Across All Media	1.2E-07				Total of Receptor Hazards Across All Media	0.17

APPENDIX E

TABLE E-16
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADULT SWIMMER/WADER ELY BROOK
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ely Brook	Sediment	Ely Brook Sediment	Ingestion	Aluminum	34526	mg/kg	1.0E-03	mg/kg-day	NA	---	NA	3.0E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0030
				Arsenic	3.7	mg/kg	1.1E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.6E-07	3.2E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0011
				Chromium	54	mg/kg	1.6E-06	mg/kg-day	NA	---	NA	4.6E-06	mg/kg-day	1.5E+00	mg/kg-day	0.0000031
				Cobalt	45	mg/kg	1.3E-06	mg/kg-day	NA	---	NA	3.9E-06	mg/kg-day	3.0E-04	mg/kg-day	0.013
				Copper	3118	mg/kg	9.2E-05	mg/kg-day	NA	---	NA	2.7E-04	mg/kg-day	4.0E-02	mg/kg-day	0.0067
				Iron	74219	mg/kg	2.2E-03	mg/kg-day	NA	---	NA	6.4E-03	mg/kg-day	7.0E-01	mg/kg-day	0.0091
				Lithium	34	mg/kg	1.0E-06	mg/kg-day	NA	---	NA	2.9E-06	mg/kg-day	2.0E-03	mg/kg-day	0.0015
				Manganese	973	mg/kg	2.9E-05	mg/kg-day	NA	---	NA	8.4E-05	mg/kg-day	7.0E-02	mg/kg-day	0.0012
				Selenium	35	mg/kg	1.0E-06	mg/kg-day	NA	---	NA	3.0E-06	mg/kg-day	5.0E-03	mg/kg-day	0.00060
				Vanadium	75	mg/kg	2.2E-06	mg/kg-day	NA	---	NA	6.4E-06	mg/kg-day	2.0E-02	mg/kg-day	0.00032
			Ingestion Total								1.6E-07					0.036
			Dermal	Aluminum	34526	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	3.7	mg/kg	7.2E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.1E-07	2.1E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00070
				Chromium	54	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	45	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	3118	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	74219	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	34	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	973	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Selenium	35	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	75	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								1.1E-07					0.00070
Sediment Total											2.7E-07					0.037
Ely Brook	Surface Water	Ely Brook Surface Water	Ingestion	Aluminum	12885	µg/L	3.8E-04	mg/kg-day	NA	---	NA	1.1E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0011
				Arsenic	0.246	µg/L	7.3E-09	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.1E-08	2.1E-08	mg/kg-day	3.0E-04	mg/kg-day	0.000071
				Cadmium	7.55	µg/L	2.2E-07	mg/kg-day	NA	---	NA	6.5E-07	mg/kg-day	1.0E-03	mg/kg-day	0.00065
				Chromium	10.35	µg/L	3.1E-07	mg/kg-day	NA	---	NA	8.9E-07	mg/kg-day	1.5E+00	mg/kg-day	0.00000059
				Cobalt	153.2	µg/L	4.5E-06	mg/kg-day	NA	---	NA	1.3E-05	mg/kg-day	3.0E-04	mg/kg-day	0.044
				Copper	4391	µg/L	1.3E-04	mg/kg-day	NA	---	NA	3.8E-04	mg/kg-day	4.0E-02	mg/kg-day	0.0095
				Iron	6354	µg/L	1.9E-04	mg/kg-day	NA	---	NA	5.5E-04	mg/kg-day	7.0E-01	mg/kg-day	0.00078
				Lithium	6.299	µg/L	1.9E-07	mg/kg-day	NA	---	NA	5.4E-07	mg/kg-day	2.0E-03	mg/kg-day	0.00027
				Manganese	776.6	µg/L	2.3E-05	mg/kg-day	NA	---	NA	6.7E-05	mg/kg-day	2.4E-02	mg/kg-day	0.0028
				Mercury	0.099	µg/L	2.9E-09	mg/kg-day	NA	---	NA	8.5E-09	mg/kg-day	3.0E-04	mg/kg-day	0.000028
				Nickel	57.27	µg/L	1.7E-06	mg/kg-day	NA	---	NA	4.9E-06	mg/kg-day	2.0E-02	mg/kg-day	0.00025
				Vanadium	1.545	µg/L	4.6E-08	mg/kg-day	NA	---	NA	1.3E-07	mg/kg-day	2.0E-02	mg/kg-day	0.0000067
				Zinc	1073	µg/L	3.2E-05	mg/kg-day	NA	---	NA	9.2E-05	mg/kg-day	3.0E-01	mg/kg-day	0.00031
			Ingestion Total								1.1E-08					0.060
			Dermal	Aluminum	12885	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	0.246	µg/L	2.6E-09	mg/kg-day	1.5E+00	(mg/kg-day)^-1	3.9E-09	7.6E-09	mg/kg-day	3.0E-04	mg/kg-day	0.000025
				Cadmium	7.55	µg/L	8.0E-08	mg/kg-day	NA	---	NA	2.3E-07	mg/kg-day	2.5E-05	mg/kg-day	0.0094
				Chromium	10.35	µg/L	2.2E-07	mg/kg-day	NA	---	NA	6.4E-07	mg/kg-day	2.0E-02	mg/kg-day	0.000033
				Cobalt	153.2	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	4391	µg/L	4.7E-05	mg/kg-day	NA	---	NA	1.4E-04	mg/kg-day	4.0E-02	mg/kg-day	0.0034
				Iron	6354	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	6.299	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	776.6	µg/L	8.3E-06	mg/kg-day	NA	---	NA	2.4E-05	mg/kg-day	9.6E-04	mg/kg-day	0.025
				Mercury	0.099	µg/L	1.1E-09	mg/kg-day	NA	---	NA	3.1E-09	mg/kg-day	2.1E-05	mg/kg-day	0.00015
				Nickel	57.27	µg/L	1.2E-07	mg/kg-day	NA	---	NA	3.6E-07	mg/kg-day	8.0E-04	mg/kg-day	0.00044
				Vanadium	1.545	µg/L	1.6E-08	mg/kg-day	NA	---	NA	4.8E-08	mg/kg-day	5.2E-04	mg/kg-day	0.000092
				Zinc	1073	µg/L	6.8E-06	mg/kg-day	NA	---	NA	2.0E-05	mg/kg-day	3.0E-01	mg/kg-day	0.000067
			Dermal Total								3.9E-09					0.039
Surface Water Total											1.5E-08					0.10
										Total of Receptor Risks Across All Media	2.8E-07				Total of Receptor Hazards Across All Media	0.14

APPENDIX E

TABLE E-17
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADOLESCENT SWIMMER/WADER ELY BROOK
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ely Brook	Sediment	Ely Brook Sediment	Ingestion	Aluminum	34526	mg/kg	1.1E-04	mg/kg-day	NA	---	NA	1.0E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0010
				Arsenic	3.7	mg/kg	1.2E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.8E-08	1.1E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00035
				Chromium	54	mg/kg	1.8E-07	mg/kg-day	NA	---	NA	1.6E-06	mg/kg-day	1.5E+00	mg/kg-day	0.0000010
				Cobalt	45	mg/kg	1.5E-07	mg/kg-day	NA	---	NA	1.3E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0044
				Copper	3118	mg/kg	1.0E-05	mg/kg-day	NA	---	NA	9.0E-05	mg/kg-day	4.0E-02	mg/kg-day	0.0023
				Iron	74219	mg/kg	2.5E-04	mg/kg-day	NA	---	NA	2.2E-03	mg/kg-day	7.0E-01	mg/kg-day	0.0031
				Lithium	34	mg/kg	1.1E-07	mg/kg-day	NA	---	NA	9.9E-07	mg/kg-day	2.0E-03	mg/kg-day	0.00049
				Manganese	973	mg/kg	3.2E-06	mg/kg-day	NA	---	NA	2.8E-05	mg/kg-day	7.0E-02	mg/kg-day	0.00040
				Selenium	35	mg/kg	1.2E-07	mg/kg-day	NA	---	NA	1.0E-06	mg/kg-day	5.0E-03	mg/kg-day	0.00020
				Vanadium	75	mg/kg	2.5E-07	mg/kg-day	NA	---	NA	2.2E-06	mg/kg-day	2.0E-02	mg/kg-day	0.00011
			Ingestion Total								1.8E-08					0.012
			Dermal	Aluminum	34526	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	3.7	mg/kg	1.8E-09	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.8E-09	1.6E-08	mg/kg-day	3.0E-04	mg/kg-day	0.000053
				Chromium	54	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	45	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	3118	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	74219	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	34	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	973	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Selenium	35	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	75	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								2.8E-09					0.000053
Sediment Total											2.1E-08					0.012
Ely Brook	Surface Water	Ely Brook Surface Water	Ingestion	Aluminum	12885	µg/L	4.3E-05	mg/kg-day	NA	---	NA	3.7E-04	mg/kg-day	1.0E+00	mg/kg-day	0.00037
				Arsenic	0.246	µg/L	8.1E-10	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.2E-09	7.1E-09	mg/kg-day	3.0E-04	mg/kg-day	0.000024
				Cadmium	7.55	µg/L	2.5E-08	mg/kg-day	NA	---	NA	2.2E-07	mg/kg-day	1.0E-03	mg/kg-day	0.00022
				Chromium	10.35	µg/L	3.4E-08	mg/kg-day	NA	---	NA	3.0E-07	mg/kg-day	1.5E+00	mg/kg-day	0.00000020
				Cobalt	153.2	µg/L	5.1E-07	mg/kg-day	NA	---	NA	4.4E-06	mg/kg-day	3.0E-04	mg/kg-day	0.015
				Copper	4391	µg/L	1.5E-05	mg/kg-day	NA	---	NA	1.3E-04	mg/kg-day	4.0E-02	mg/kg-day	0.003
				Iron	6354	µg/L	2.1E-05	mg/kg-day	NA	---	NA	1.8E-04	mg/kg-day	7.0E-01	mg/kg-day	0.00026
				Lithium	6.299	µg/L	2.1E-08	mg/kg-day	NA	---	NA	1.8E-07	mg/kg-day	2.0E-03	mg/kg-day	0.000091
				Manganese	776.6	µg/L	2.6E-06	mg/kg-day	NA	---	NA	2.3E-05	mg/kg-day	2.4E-02	mg/kg-day	0.00094
				Mercury	0.099	µg/L	3.3E-10	mg/kg-day	NA	---	NA	2.9E-09	mg/kg-day	3.0E-04	mg/kg-day	0.000010
				Nickel	57.27	µg/L	1.9E-07	mg/kg-day	NA	---	NA	1.7E-06	mg/kg-day	2.0E-02	mg/kg-day	0.000083
				Vanadium	1.545	µg/L	5.1E-09	mg/kg-day	NA	---	NA	4.5E-08	mg/kg-day	2.0E-02	mg/kg-day	0.0000022
				Zinc	1073	µg/L	3.6E-06	mg/kg-day	NA	---	NA	3.1E-05	mg/kg-day	3.0E-01	mg/kg-day	0.00010
			Ingestion Total								1.2E-09					0.020
			Dermal	Aluminum	12885	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	0.246	µg/L	2.4E-10	mg/kg-day	1.5E+00	(mg/kg-day)^-1	3.6E-10	2.1E-09	mg/kg-day	3.0E-04	mg/kg-day	0.0000071
				Cadmium	7.55	µg/L	7.5E-09	mg/kg-day	NA	---	NA	6.5E-08	mg/kg-day	2.5E-05	mg/kg-day	0.0026
				Chromium	10.35	µg/L	2.0E-08	mg/kg-day	NA	---	NA	1.8E-07	mg/kg-day	2.0E-02	mg/kg-day	0.0000092
				Cobalt	153.2	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	4391	µg/L	4.3E-06	mg/kg-day	NA	---	NA	3.8E-05	mg/kg-day	4.0E-02	mg/kg-day	0.00095
				Iron	6354	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	6.299	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	776.6	µg/L	7.7E-07	mg/kg-day	NA	---	NA	6.7E-06	mg/kg-day	9.6E-04	mg/kg-day	0.0070
				Mercury	0.099	µg/L	9.8E-11	mg/kg-day	NA	---	NA	8.5E-10	mg/kg-day	2.1E-05	mg/kg-day	0.000041
				Nickel	57.27	µg/L	1.1E-08	mg/kg-day	NA	---	NA	9.9E-08	mg/kg-day	8.0E-04	mg/kg-day	0.00012
				Vanadium	1.545	µg/L	1.5E-09	mg/kg-day	NA	---	NA	1.3E-08	mg/kg-day	5.2E-04	mg/kg-day	0.000026
				Zinc	1073	µg/L	6.4E-07	mg/kg-day	NA	---	NA	5.6E-06	mg/kg-day	3.0E-01	mg/kg-day	0.000019
			Dermal Total								3.6E-10					0.011
Surface Water Total											1.6E-09					0.031
										Total of Receptor Risks Across All Media	2.3E-08				Total of Receptor Hazards Across All Media	0.043

APPENDIX E

TABLE E-18
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADULT SWIMMER/WADER ELY BROOK
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ely Brook	Sediment	Ely Brook Sediment	Ingestion	Aluminum	34526	mg/kg	2.5E-04	mg/kg-day	NA	---	NA	7.4E-04	mg/kg-day	1.0E+00	mg/kg-day	0.00074
				Arsenic	3.7	mg/kg	2.7E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	4.1E-08	7.9E-08	mg/kg-day	3.0E-04	mg/kg-day	0.00026
				Chromium	54	mg/kg	4.0E-07	mg/kg-day	NA	---	NA	1.2E-06	mg/kg-day	1.5E+00	mg/kg-day	0.00000077
				Cobalt	45	mg/kg	3.3E-07	mg/kg-day	NA	---	NA	9.7E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0032
				Copper	3118	mg/kg	2.3E-05	mg/kg-day	NA	---	NA	6.7E-05	mg/kg-day	4.0E-02	mg/kg-day	0.0017
				Iron	74219	mg/kg	5.5E-04	mg/kg-day	NA	---	NA	1.6E-03	mg/kg-day	7.0E-01	mg/kg-day	0.0023
				Lithium	34	mg/kg	2.5E-07	mg/kg-day	NA	---	NA	7.3E-07	mg/kg-day	2.0E-03	mg/kg-day	0.00037
				Manganese	973	mg/kg	7.2E-06	mg/kg-day	NA	---	NA	2.1E-05	mg/kg-day	7.0E-02	mg/kg-day	0.00030
				Selenium	35	mg/kg	2.6E-07	mg/kg-day	NA	---	NA	7.5E-07	mg/kg-day	5.0E-03	mg/kg-day	0.00015
				Vanadium	75	mg/kg	5.5E-07	mg/kg-day	NA	---	NA	1.6E-06	mg/kg-day	2.0E-02	mg/kg-day	0.000080
			Ingestion Total								4.1E-08					0.0091
			Dermal	Aluminum	34526	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	3.7	mg/kg	5.1E-09	mg/kg-day	1.5E+00	(mg/kg-day)^-1	7.7E-09	1.5E-08	mg/kg-day	3.0E-04	mg/kg-day	0.000050
				Chromium	54	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	45	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	3118	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	74219	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	34	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	973	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Selenium	35	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Vanadium	75	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								7.7E-09					0.000050
Sediment Total											4.8E-08					0.0091
Ely Brook	Surface Water	Ely Brook Surface Water	Ingestion	Aluminum	12885	µg/L	9.5E-05	mg/kg-day	NA	---	NA	2.8E-04	mg/kg-day	1.0E+00	mg/kg-day	0.00028
				Arsenic	0.246	µg/L	1.8E-09	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.7E-09	5.3E-09	mg/kg-day	3.0E-04	mg/kg-day	0.000018
				Cadmium	7.55	µg/L	5.6E-08	mg/kg-day	NA	---	NA	1.6E-07	mg/kg-day	1.0E-03	mg/kg-day	0.00016
				Chromium	10.35	µg/L	7.6E-08	mg/kg-day	NA	---	NA	2.2E-07	mg/kg-day	1.5E+00	mg/kg-day	0.00000015
				Cobalt	153.2	µg/L	1.1E-06	mg/kg-day	NA	---	NA	3.3E-06	mg/kg-day	3.0E-04	mg/kg-day	0.011
				Copper	4391	µg/L	3.2E-05	mg/kg-day	NA	---	NA	9.5E-05	mg/kg-day	4.0E-02	mg/kg-day	0.0024
				Iron	6354	µg/L	4.7E-05	mg/kg-day	NA	---	NA	1.4E-04	mg/kg-day	7.0E-01	mg/kg-day	0.00020
				Lithium	6.299	µg/L	4.6E-08	mg/kg-day	NA	---	NA	1.4E-07	mg/kg-day	2.0E-03	mg/kg-day	0.000068
				Manganese	776.6	µg/L	5.7E-06	mg/kg-day	NA	---	NA	1.7E-05	mg/kg-day	2.4E-02	mg/kg-day	0.00070
				Mercury	0.099	µg/L	7.3E-10	mg/kg-day	NA	---	NA	2.1E-09	mg/kg-day	3.0E-04	mg/kg-day	0.0000071
				Nickel	57.27	µg/L	4.2E-07	mg/kg-day	NA	---	NA	1.2E-06	mg/kg-day	2.0E-02	mg/kg-day	0.000062
				Vanadium	1.545	µg/L	1.1E-08	mg/kg-day	NA	---	NA	3.3E-08	mg/kg-day	2.0E-02	mg/kg-day	0.0000017
				Zinc	1073	µg/L	7.9E-06	mg/kg-day	NA	---	NA	2.3E-05	mg/kg-day	3.0E-01	mg/kg-day	0.000077
			Ingestion Total								2.7E-09					0.015
			Dermal	Aluminum	12885	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	0.246	µg/L	6.5E-10	mg/kg-day	1.5E+00	(mg/kg-day)^-1	9.8E-10	1.9E-09	mg/kg-day	3.0E-04	mg/kg-day	0.0000064
				Cadmium	7.55	µg/L	2.0E-08	mg/kg-day	NA	---	NA	5.9E-08	mg/kg-day	2.5E-05	mg/kg-day	0.0023
				Chromium	10.35	µg/L	5.5E-08	mg/kg-day	NA	---	NA	1.6E-07	mg/kg-day	2.0E-02	mg/kg-day	0.0000082
				Cobalt	153.2	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	4391	µg/L	1.2E-05	mg/kg-day	NA	---	NA	3.4E-05	mg/kg-day	4.0E-02	mg/kg-day	0.00085
				Iron	6354	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	6.299	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	776.6	µg/L	2.1E-06	mg/kg-day	NA	---	NA	6.0E-06	mg/kg-day	9.6E-04	mg/kg-day	0.0063
				Mercury	0.099	µg/L	2.6E-10	mg/kg-day	NA	---	NA	7.7E-10	mg/kg-day	2.1E-05	mg/kg-day	0.000037
				Nickel	57.27	µg/L	3.0E-08	mg/kg-day	NA	---	NA	8.9E-08	mg/kg-day	8.0E-04	mg/kg-day	0.00011
				Vanadium	1.545	µg/L	4.1E-09	mg/kg-day	NA	---	NA	1.2E-08	mg/kg-day	5.2E-04	mg/kg-day	0.000023
				Zinc	1073	µg/L	1.7E-06	mg/kg-day	NA	---	NA	5.0E-06	mg/kg-day	3.0E-01	mg/kg-day	0.000017
			Dermal Total								9.8E-10					0.010
Surface Water Total											3.7E-09					0.025
										Total of Receptor Risks Across All Media	5.2E-08				Total of Receptor Hazards Across All Media	0.034

APPENDIX E

TABLE E-19
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADOLESCENT SWIMMER/WADER SCHOOLHOUSE BROOK
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Schoolhouse Brook	Sediment	Schoolhouse Brook Sediment	Ingestion	Aluminum	42733	mg/kg	5.7E-04	mg/kg-day	NA	---	NA	5.0E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0050
				Arsenic	1.4	mg/kg	1.9E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.8E-08	1.6E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00055
				Chromium	29	mg/kg	3.9E-07	mg/kg-day	NA	---	NA	3.4E-06	mg/kg-day	1.5E+00	mg/kg-day	0.0000023
				Cobalt	11	mg/kg	1.4E-07	mg/kg-day	NA	---	NA	1.2E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0041
				Copper	853	mg/kg	1.1E-05	mg/kg-day	NA	---	NA	9.9E-05	mg/kg-day	4.0E-02	mg/kg-day	0.0025
				Iron	32270	mg/kg	4.3E-04	mg/kg-day	NA	---	NA	3.7E-03	mg/kg-day	7.0E-01	mg/kg-day	0.0053
				Lithium	24	mg/kg	3.2E-07	mg/kg-day	NA	---	NA	2.8E-06	mg/kg-day	2.0E-03	mg/kg-day	0.0014
				Manganese	739	mg/kg	9.8E-06	mg/kg-day	NA	---	NA	8.6E-05	mg/kg-day	7.0E-02	mg/kg-day	0.0012
				Vanadium	45	mg/kg	5.9E-07	mg/kg-day	NA	---	NA	5.2E-06	mg/kg-day	2.0E-02	mg/kg-day	0.00026
			Ingestion Total								2.8E-08					0.020
			Dermal	Aluminum	42733	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	1.4	mg/kg	1.1E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.6E-08	9.3E-08	mg/kg-day	3.0E-04	mg/kg-day	0.00031
				Chromium	29	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	11	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	853	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	32270	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	24	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	739	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Vanadium	45	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								1.6E-08					0.00031
Sediment Total											4.4E-08					0.021
Schoolhouse Brook	Surface Water	Schoolhouse Brook Surface Water	Ingestion	Chromium	2.6	µg/L	3.4E-08	mg/kg-day	NA	---	NA	3.0E-07	mg/kg-day	1.5E+00	mg/kg-day	0.00000020
				Cobalt	8.9	µg/L	1.2E-07	mg/kg-day	NA	---	NA	1.0E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0035
				Copper	336.8	µg/L	4.5E-06	mg/kg-day	NA	---	NA	3.9E-05	mg/kg-day	4.0E-02	mg/kg-day	0.0010
				Manganese	56	µg/L	7.5E-07	mg/kg-day	NA	---	NA	6.5E-06	mg/kg-day	2.4E-02	mg/kg-day	0.00027
				Mercury	0.17	µg/L	2.3E-09	mg/kg-day	NA	---	NA	2.0E-08	mg/kg-day	3.0E-04	mg/kg-day	0.000066
				Vanadium	0.47	µg/L	6.2E-09	mg/kg-day	NA	---	NA	5.5E-08	mg/kg-day	2.0E-02	mg/kg-day	0.0000027
			Ingestion Total								0.0E+00					0.0048
			Dermal	Chromium	2.6	µg/L	2.0E-08	mg/kg-day	NA	---	NA	1.8E-07	mg/kg-day	2.0E-02	mg/kg-day	0.0000092
				Cobalt	8.9	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	336.8	µg/L	1.3E-06	mg/kg-day	NA	---	NA	1.2E-05	mg/kg-day	4.0E-02	mg/kg-day	0.00029
				Manganese	56	µg/L	2.2E-07	mg/kg-day	NA	---	NA	2.0E-06	mg/kg-day	9.6E-04	mg/kg-day	0.0020
				Mercury	0.17	µg/L	6.7E-10	mg/kg-day	NA	---	NA	5.9E-09	mg/kg-day	2.1E-05	mg/kg-day	0.00028
				Vanadium	0.47	µg/L	1.9E-09	mg/kg-day	NA	---	NA	1.6E-08	mg/kg-day	5.2E-04	mg/kg-day	0.000031
			Dermal Total								0.0E+00					0.0026
Surface Water Total											0.0E+00					0.0074
											Total of Receptor Risks Across All Media					4.4E-08	Total of Receptor Hazards Across All Media				0.028

APPENDIX E

TABLE E-20
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADULT SWIMMER/WADER SCHOOLHOUSE BROOK
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Schoolhouse Brook	Sediment	Schoolhouse Brook Sediment	Ingestion	Aluminum	42733	mg/kg	1.3E-03	mg/kg-day	NA	---	NA	3.7E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0037
				Arsenic	1.4	mg/kg	4.2E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	6.3E-08	1.2E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00041
				Chromium	29	mg/kg	8.6E-07	mg/kg-day	NA	---	NA	2.5E-06	mg/kg-day	1.5E+00	mg/kg-day	0.0000017
				Cobalt	11	mg/kg	3.2E-07	mg/kg-day	NA	---	NA	9.2E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0031
				Copper	853	mg/kg	2.5E-05	mg/kg-day	NA	---	NA	7.3E-05	mg/kg-day	4.0E-02	mg/kg-day	0.0018
				Iron	32270	mg/kg	9.5E-04	mg/kg-day	NA	---	NA	2.8E-03	mg/kg-day	7.0E-01	mg/kg-day	0.0040
				Lithium	24	mg/kg	7.1E-07	mg/kg-day	NA	---	NA	2.1E-06	mg/kg-day	2.0E-03	mg/kg-day	0.0010
				Manganese	739	mg/kg	2.2E-05	mg/kg-day	NA	---	NA	6.4E-05	mg/kg-day	7.0E-02	mg/kg-day	0.00091
				Vanadium	45	mg/kg	1.3E-06	mg/kg-day	NA	---	NA	3.8E-06	mg/kg-day	2.0E-02	mg/kg-day	0.00019
			Ingestion Total								6.3E-08					0.015
			Dermal	Aluminum	42733	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	1.4	mg/kg	2.8E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	4.1E-08	8.1E-08	mg/kg-day	3.0E-04	mg/kg-day	0.00027
				Chromium	29	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	11	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	853	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	32270	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	24	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	739	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Vanadium	45	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								4.1E-08					0.00027
Sediment Total											1.0E-07					0.015
Schoolhouse Brook	Surface Water	Schoolhouse Brook Surface Water	Ingestion	Chromium	2.6	µg/L	7.6E-08	mg/kg-day	NA	---	NA	2.2E-07	mg/kg-day	1.5E+00	mg/kg-day	0.00000015
				Cobalt	8.9	µg/L	2.6E-07	mg/kg-day	NA	---	NA	7.7E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0026
				Copper	336.8	µg/L	9.9E-06	mg/kg-day	NA	---	NA	2.9E-05	mg/kg-day	4.0E-02	mg/kg-day	0.00073
				Manganese	56	µg/L	1.7E-06	mg/kg-day	NA	---	NA	4.9E-06	mg/kg-day	2.4E-02	mg/kg-day	0.00020
				Mercury	0.17	µg/L	5.0E-09	mg/kg-day	NA	---	NA	1.5E-08	mg/kg-day	3.0E-04	mg/kg-day	0.000049
				Vanadium	0.47	µg/L	1.4E-08	mg/kg-day	NA	---	NA	4.1E-08	mg/kg-day	2.0E-02	mg/kg-day	0.0000020
			Ingestion Total								0.0E+00					0.0035
			Dermal	Chromium	2.6	µg/L	5.5E-08	mg/kg-day	NA	---	NA	1.6E-07	mg/kg-day	2.0E-02	mg/kg-day	0.0000082
				Cobalt	8.9	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	336.8	µg/L	3.6E-06	mg/kg-day	NA	---	NA	1.0E-05	mg/kg-day	4.0E-02	mg/kg-day	0.00026
				Manganese	56	µg/L	6.0E-07	mg/kg-day	NA	---	NA	1.8E-06	mg/kg-day	9.6E-04	mg/kg-day	0.0018
				Mercury	0.17	µg/L	1.8E-09	mg/kg-day	NA	---	NA	5.3E-09	mg/kg-day	2.1E-05	mg/kg-day	0.00025
				Vanadium	0.47	µg/L	5.0E-09	mg/kg-day	NA	---	NA	1.5E-08	mg/kg-day	5.2E-04	mg/kg-day	0.000028
			Dermal Total								0.0E+00					0.0024
Surface Water Total											0.0E+00					0.0059
										Total of Receptor Risks Across All Media	1.0E-07			Total of Receptor Hazards Across All Media		0.021

APPENDIX E

TABLE E-21
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADOLESCENT SWIMMER/WADER SCHOOLHOUSE BROOK
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Schoolhouse Brook	Sediment	Schoolhouse Brook Sediment	Ingestion	Aluminum	42733	mg/kg	1.4E-04	mg/kg-day	NA	---	NA	1.2E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0012
				Arsenic	1.4	mg/kg	4.7E-09	mg/kg-day	1.5E+00	(mg/kg-day)^-1	7.0E-09	4.1E-08	mg/kg-day	3.0E-04	mg/kg-day	0.00014
				Chromium	29	mg/kg	9.7E-08	mg/kg-day	NA	---	NA	8.5E-07	mg/kg-day	1.5E+00	mg/kg-day	0.00000056
				Cobalt	11	mg/kg	3.5E-08	mg/kg-day	NA	---	NA	3.1E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0010
				Copper	853	mg/kg	2.8E-06	mg/kg-day	NA	---	NA	2.5E-05	mg/kg-day	4.0E-02	mg/kg-day	0.00062
				Iron	32270	mg/kg	1.1E-04	mg/kg-day	NA	---	NA	9.4E-04	mg/kg-day	7.0E-01	mg/kg-day	0.0013
				Lithium	24	mg/kg	7.9E-08	mg/kg-day	NA	---	NA	7.0E-07	mg/kg-day	2.0E-03	mg/kg-day	0.00035
				Manganese	739	mg/kg	2.4E-06	mg/kg-day	NA	---	NA	2.1E-05	mg/kg-day	7.0E-02	mg/kg-day	0.00031
				Vanadium	45	mg/kg	1.5E-07	mg/kg-day	NA	---	NA	1.3E-06	mg/kg-day	2.0E-02	mg/kg-day	0.000065
			Ingestion Total								7.0E-09					0.0051
			Dermal	Aluminum	42733	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	1.4	mg/kg	7.1E-10	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.1E-09	6.2E-09	mg/kg-day	3.0E-04	mg/kg-day	0.000021
				Chromium	29	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	11	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	853	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	32270	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	24	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	739	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Vanadium	45	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								1.1E-09					0.000021
Sediment Total											8.1E-09					0.0051
Schoolhouse Brook	Surface Water	Schoolhouse Brook Surface Water	Ingestion	Chromium	2.6	µg/L	8.6E-09	mg/kg-day	NA	---	NA	7.5E-08	mg/kg-day	1.5E+00	mg/kg-day	0.000000050
				Cobalt	8.9	µg/L	3.0E-08	mg/kg-day	NA	---	NA	2.6E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00086
				Copper	336.8	µg/L	1.1E-06	mg/kg-day	NA	---	NA	9.8E-06	mg/kg-day	4.0E-02	mg/kg-day	0.00024
				Manganese	56	µg/L	1.9E-07	mg/kg-day	NA	---	NA	1.6E-06	mg/kg-day	2.4E-02	mg/kg-day	0.000068
				Mercury	0.17	µg/L	5.6E-10	mg/kg-day	NA	---	NA	4.9E-09	mg/kg-day	3.0E-04	mg/kg-day	0.000016
				Vanadium	0.47	µg/L	1.6E-09	mg/kg-day	NA	---	NA	1.4E-08	mg/kg-day	2.0E-02	mg/kg-day	0.00000068
			Ingestion Total								0.0E+00					0.0012
			Dermal	Chromium	2.6	µg/L	5.1E-09	mg/kg-day	NA	---	NA	4.5E-08	mg/kg-day	2.0E-02	mg/kg-day	0.0000023
				Cobalt	8.9	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	336.8	µg/L	3.3E-07	mg/kg-day	NA	---	NA	2.9E-06	mg/kg-day	4.0E-02	mg/kg-day	0.000073
				Manganese	56	µg/L	5.6E-08	mg/kg-day	NA	---	NA	4.9E-07	mg/kg-day	9.6E-04	mg/kg-day	0.00051
				Mercury	0.17	µg/L	1.7E-10	mg/kg-day	NA	---	NA	1.5E-09	mg/kg-day	2.1E-05	mg/kg-day	0.000070
				Vanadium	0.47	µg/L	4.6E-10	mg/kg-day	NA	---	NA	4.1E-09	mg/kg-day	5.2E-04	mg/kg-day	0.0000078
			Dermal Total								0.0E+00					0.00066
Surface Water Total											0.0E+00					0.0019
											Total of Receptor Risks Across All Media					Total of Receptor Hazards Across All Media
											8.1E-09					0.0070

APPENDIX E

TABLE E-22
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADULT SWIMMER/WADER SCHOOLHOUSE BROOK
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Schoolhouse Brook	Sediment	Schoolhouse Brook Sediment	Ingestion	Aluminum	42733	mg/kg	3.2E-04	mg/kg-day	NA	---	NA	9.2E-04	mg/kg-day	1.0E+00	mg/kg-day	0.00092
				Arsenic	1.4	mg/kg	1.0E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.6E-08	3.0E-08	mg/kg-day	3.0E-04	mg/kg-day	0.00010
				Chromium	29	mg/kg	2.2E-07	mg/kg-day	NA	---	NA	6.3E-07	mg/kg-day	1.5E+00	mg/kg-day	0.00000042
				Cobalt	11	mg/kg	7.9E-08	mg/kg-day	NA	---	NA	2.3E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00077
				Copper	853	mg/kg	6.3E-06	mg/kg-day	NA	---	NA	1.8E-05	mg/kg-day	4.0E-02	mg/kg-day	0.00046
				Iron	32270	mg/kg	2.4E-04	mg/kg-day	NA	---	NA	6.9E-04	mg/kg-day	7.0E-01	mg/kg-day	0.0010
				Lithium	24	mg/kg	1.8E-07	mg/kg-day	NA	---	NA	5.2E-07	mg/kg-day	2.0E-03	mg/kg-day	0.00026
				Manganese	739	mg/kg	5.5E-06	mg/kg-day	NA	---	NA	1.6E-05	mg/kg-day	7.0E-02	mg/kg-day	0.00023
				Vanadium	45	mg/kg	3.3E-07	mg/kg-day	NA	---	NA	9.6E-07	mg/kg-day	2.0E-02	mg/kg-day	0.000048
			Ingestion Total								1.6E-08					0.0038
			Dermal	Aluminum	42733	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	1.4	mg/kg	2.0E-09	mg/kg-day	1.5E+00	(mg/kg-day)^-1	3.0E-09	5.8E-09	mg/kg-day	3.0E-04	mg/kg-day	0.000019
				Chromium	29	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	11	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	853	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	4.0E-02	mg/kg-day	NA
				Iron	32270	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	24	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	739	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Vanadium	45	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								3.0E-09					0.000019
Sediment Total											1.9E-08					0.0038
Schoolhouse Brook	Surface Water	Schoolhouse Brook Surface Water	Ingestion	Chromium	2.6	µg/L	1.9E-08	mg/kg-day	NA	---	NA	5.6E-08	mg/kg-day	1.5E+00	mg/kg-day	0.000000037
				Cobalt	8.9	µg/L	6.6E-08	mg/kg-day	NA	---	NA	1.9E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00064
				Copper	336.8	µg/L	2.5E-06	mg/kg-day	NA	---	NA	7.3E-06	mg/kg-day	4.0E-02	mg/kg-day	0.00018
				Manganese	56	µg/L	4.2E-07	mg/kg-day	NA	---	NA	1.2E-06	mg/kg-day	2.4E-02	mg/kg-day	0.000051
				Mercury	0.17	µg/L	1.3E-09	mg/kg-day	NA	---	NA	3.7E-09	mg/kg-day	3.0E-04	mg/kg-day	0.000012
				Vanadium	0.47	µg/L	3.5E-09	mg/kg-day	NA	---	NA	1.0E-08	mg/kg-day	2.0E-02	mg/kg-day	0.00000051
			Ingestion Total								0.0E+00					0.00089
			Dermal	Chromium	2.6	µg/L	1.4E-08	mg/kg-day	NA	---	NA	4.0E-08	mg/kg-day	2.0E-02	mg/kg-day	0.0000021
				Cobalt	8.9	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	336.8	µg/L	8.9E-07	mg/kg-day	NA	---	NA	2.6E-06	mg/kg-day	4.0E-02	mg/kg-day	0.000065
				Manganese	56	µg/L	1.5E-07	mg/kg-day	NA	---	NA	4.4E-07	mg/kg-day	9.6E-04	mg/kg-day	0.00046
				Mercury	0.17	µg/L	4.5E-10	mg/kg-day	NA	---	NA	1.3E-09	mg/kg-day	2.1E-05	mg/kg-day	0.000063
				Vanadium	0.47	µg/L	1.3E-09	mg/kg-day	NA	---	NA	3.7E-09	mg/kg-day	5.2E-04	mg/kg-day	0.0000070
			Dermal Total								0.0E+00					0.00059
Surface Water Total											0.0E+00					0.0015
											Total of Receptor Risks Across All Media					Total of Receptor Hazards Across All Media
											1.9E-08					0.0053

APPENDIX E

TABLE E-23
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADOLESCENT SWIMMER/WADER OMPOMPANOOSUC RIVER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ompompanoosuc River	Sediment	Ompompanoosuc River Sediment	Ingestion	Aluminum	35400	mg/kg	4.7E-04	mg/kg-day	NA	---	NA	4.1E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0041
				Arsenic	5.0	mg/kg	6.6E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	9.9E-08	5.8E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0019
				Chromium	31	mg/kg	4.1E-07	mg/kg-day	NA	---	NA	3.6E-06	mg/kg-day	1.5E+00	mg/kg-day	0.0000024
				Cobalt	13	mg/kg	1.7E-07	mg/kg-day	NA	---	NA	1.5E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0050
				Iron	22800	mg/kg	3.0E-04	mg/kg-day	NA	---	NA	2.6E-03	mg/kg-day	7.0E-01	mg/kg-day	0.0038
				Lithium	24	mg/kg	3.2E-07	mg/kg-day	NA	---	NA	2.8E-06	mg/kg-day	2.0E-03	mg/kg-day	0.0014
				Manganese	1120	mg/kg	1.5E-05	mg/kg-day	NA	---	NA	1.3E-04	mg/kg-day	7.0E-02	mg/kg-day	0.0019
				Vanadium	49	mg/kg	6.5E-07	mg/kg-day	NA	---	NA	5.7E-06	mg/kg-day	2.0E-02	mg/kg-day	0.00028
			Ingestion Total								9.9E-08					0.018
			Dermal	Aluminum	35400	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	5.0	mg/kg	3.8E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	5.6E-08	3.3E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0011
				Chromium	31	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	13	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Iron	22800	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	24	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	1120	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Vanadium	49	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								5.6E-08					0.0011
Sediment Total											1.6E-07					0.019
Ompompanoosuc River	Surface Water	Ompompanoosuc River Surface Water	Ingestion	Mercury	0.2	µg/L	2.6E-09	mg/kg-day	NA	---	NA	2.3E-08	mg/kg-day	3.0E-04	mg/kg-day	0.000077
			Ingestion Total								0.0E+00					0.000077
			Dermal	Mercury	0.2	µg/L	7.9E-10	mg/kg-day	NA	---	NA	6.9E-09	mg/kg-day	2.1E-05	mg/kg-day	0.00033
			Dermal Total								0.0E+00					0.00033
Surface Water Total											0.0E+00					0.00041
											Total of Receptor Risks Across All Media					Total of Receptor Hazards Across All Media
											1.6E-07					0.020

APPENDIX E

TABLE E-24
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADULT SWIMMER/WADER OMPOMPANOOSUC RIVER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ompompanoosuc River	Sediment	Ompompanoosuc River Sediment	Ingestion	Aluminum	35400	mg/kg	1.0E-03	mg/kg-day	NA	---	NA	3.0E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0030
				Arsenic	5.0	mg/kg	1.5E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.2E-07	4.3E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0014
				Chromium	31	mg/kg	9.2E-07	mg/kg-day	NA	---	NA	2.7E-06	mg/kg-day	1.5E+00	mg/kg-day	0.0000018
				Cobalt	13	mg/kg	3.8E-07	mg/kg-day	NA	---	NA	1.1E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0037
				Iron	22800	mg/kg	6.7E-04	mg/kg-day	NA	---	NA	2.0E-03	mg/kg-day	7.0E-01	mg/kg-day	0.0028
				Lithium	24	mg/kg	7.1E-07	mg/kg-day	NA	---	NA	2.1E-06	mg/kg-day	2.0E-03	mg/kg-day	0.0010
				Manganese	1120	mg/kg	3.3E-05	mg/kg-day	NA	---	NA	9.6E-05	mg/kg-day	7.0E-02	mg/kg-day	0.0014
				Vanadium	49	mg/kg	1.4E-06	mg/kg-day	NA	---	NA	4.2E-06	mg/kg-day	2.0E-02	mg/kg-day	0.00021
			Ingestion Total								2.2E-07					0.014
			Dermal	Aluminum	35400	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	5.0	mg/kg	9.8E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.5E-07	2.9E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0010
				Chromium	31	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	13	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Iron	22800	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	24	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	1120	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Vanadium	49	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								1.5E-07					0.0010
Sediment Total											3.7E-07					0.015
Ompompanoosuc River	Surface Water	Ompompanoosuc River Surface Water	Ingestion	Mercury	0.2	µg/L	5.9E-09	mg/kg-day	NA	---	NA	1.7E-08	mg/kg-day	3.0E-04	mg/kg-day	0.000057
			Ingestion Total								0.0E+00					0.000057
			Dermal	Mercury	0.2	µg/L	2.1E-09	mg/kg-day	NA	---	NA	6.2E-09	mg/kg-day	2.1E-05	mg/kg-day	0.00030
			Dermal Total								0.0E+00					0.00030
Surface Water Total											0.0E+00					0.00035
											Total of Receptor Risks Across All Media					3.7E-07
																Total of Receptor Hazards Across All Media
																0.015

APPENDIX E

TABLE E-25
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADOLESCENT SWIMMER/WADER OMPOMPANOOSUC RIVER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ompompanoosuc River	Sediment	Ompompanoosuc River Sediment	Ingestion	Aluminum	35400	mg/kg	1.2E-04	mg/kg-day	NA	---	NA	1.0E-03	mg/kg-day	1.0E+00	mg/kg-day	0.0010
				Arsenic	5.0	mg/kg	1.7E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.5E-08	1.4E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00048
				Chromium	31	mg/kg	1.0E-07	mg/kg-day	NA	---	NA	9.0E-07	mg/kg-day	1.5E+00	mg/kg-day	0.00000060
				Cobalt	13	mg/kg	4.3E-08	mg/kg-day	NA	---	NA	3.8E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0013
				Iron	22800	mg/kg	7.6E-05	mg/kg-day	NA	---	NA	6.6E-04	mg/kg-day	7.0E-01	mg/kg-day	0.00094
				Lithium	24	mg/kg	7.9E-08	mg/kg-day	NA	---	NA	7.0E-07	mg/kg-day	2.0E-03	mg/kg-day	0.00035
				Manganese	1120	mg/kg	3.7E-06	mg/kg-day	NA	---	NA	3.2E-05	mg/kg-day	7.0E-02	mg/kg-day	0.00046
				Vanadium	49	mg/kg	1.6E-07	mg/kg-day	NA	---	NA	1.4E-06	mg/kg-day	2.0E-02	mg/kg-day	0.000071
			Ingestion Total								2.5E-08					0.0046
			Dermal	Aluminum	35400	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	5.0	mg/kg	2.5E-09	mg/kg-day	1.5E+00	(mg/kg-day)^-1	3.8E-09	2.2E-08	mg/kg-day	3.0E-04	mg/kg-day	0.000073
				Chromium	31	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	13	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Iron	22800	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	24	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	1120	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Vanadium	49	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								3.8E-09					0.000073
Sediment Total											2.9E-08					0.0047
Ompompanoosuc River	Surface Water	Ompompanoosuc River Surface Water	Ingestion	Mercury	0.2	µg/L	6.6E-10	mg/kg-day	NA	---	NA	5.8E-09	mg/kg-day	3.0E-04	mg/kg-day	0.000019
			Ingestion Total								0.0E+00					0.000019
			Dermal	Mercury	0.2	µg/L	2.0E-10	mg/kg-day	NA	---	NA	1.7E-09	mg/kg-day	2.1E-05	mg/kg-day	0.000082
			Dermal Total								0.0E+00					0.000082
Surface Water Total											0.0E+00					0.00010
											Total of Receptor Risks Across All Media					
											2.9E-08					0.0048
												Total of Receptor Hazards Across All Media				

APPENDIX E

TABLE E-26
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADULT SWIMMER/WADER OMPOMPANOOSUC RIVER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Ompompanoosuc River	Sediment	Ompompanoosuc River Sediment	Ingestion	Aluminum	35400	mg/kg	2.6E-04	mg/kg-day	NA	---	NA	7.6E-04	mg/kg-day	1.0E+00	mg/kg-day	0.00076
				Arsenic	5.0	mg/kg	3.7E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	5.5E-08	1.1E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00036
				Chromium	31	mg/kg	2.3E-07	mg/kg-day	NA	---	NA	6.7E-07	mg/kg-day	1.5E+00	mg/kg-day	0.00000044
				Cobalt	13	mg/kg	9.6E-08	mg/kg-day	NA	---	NA	2.8E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00093
				Iron	22800	mg/kg	1.7E-04	mg/kg-day	NA	---	NA	4.9E-04	mg/kg-day	7.0E-01	mg/kg-day	0.00070
				Lithium	24	mg/kg	1.8E-07	mg/kg-day	NA	---	NA	5.2E-07	mg/kg-day	2.0E-03	mg/kg-day	0.00026
				Manganese	1120	mg/kg	8.3E-06	mg/kg-day	NA	---	NA	2.4E-05	mg/kg-day	7.0E-02	mg/kg-day	0.00034
				Vanadium	49	mg/kg	3.6E-07	mg/kg-day	NA	---	NA	1.1E-06	mg/kg-day	2.0E-02	mg/kg-day	0.000053
			Ingestion Total								5.5E-08					0.0034
			Dermal	Aluminum	35400	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	5.0	mg/kg	7.0E-09	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.0E-08	2.0E-08	mg/kg-day	3.0E-04	mg/kg-day	0.000068
				Chromium	31	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Cobalt	13	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Iron	22800	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lithium	24	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	2.0E-03	mg/kg-day	NA
				Manganese	1120	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-02	mg/kg-day	NA
				Vanadium	49	mg/kg	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.2E-04	mg/kg-day	NA
			Dermal Total								1.0E-08					0.000068
Sediment Total											6.6E-08					0.0035
Ompompanoosuc River	Surface Water	Ompompanoosuc River Surface Water	Ingestion	Mercury	0.2	µg/L	1.5E-09	mg/kg-day	NA	---	NA	4.3E-09	mg/kg-day	3.0E-04	mg/kg-day	0.000014
			Ingestion Total								0.0E+00					0.000014
			Dermal	Mercury	0.2	µg/L	5.3E-10	mg/kg-day	NA	---	NA	1.5E-09	mg/kg-day	2.1E-05	mg/kg-day	0.000074
			Dermal Total								0.0E+00					0.000074
Surface Water Total											0.0E+00					0.000088
											Total of Receptor Risks Across All Media					6.6E-08
																Total of Receptor Hazards Across All Media	0.0036

APPENDIX E

TABLE E-27
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - CHILD OVERBURDEN GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Overburden Groundwater	Ingestion	Aluminum	150000	µg/L	8.2E-01	mg/kg-day	NA	---	NA	9.6E+00	mg/kg-day	1.0E+00	mg/kg-day	9.6
				Arsenic	7.6	µg/L	4.2E-05	mg/kg-day	1.5E+00	(mg/kg-day)^-1	6.2E-05	4.9E-04	mg/kg-day	3.0E-04	mg/kg-day	1.6
				Barium	1202	µg/L	6.6E-03	mg/kg-day	NA	---	NA	7.7E-02	mg/kg-day	2.0E-01	mg/kg-day	0.38
				Cadmium	37	µg/L	2.0E-04	mg/kg-day	NA	---	NA	2.3E-03	mg/kg-day	5.0E-04	mg/kg-day	4.7
				Chromium	365	µg/L	2.0E-03	mg/kg-day	NA	---	NA	2.3E-02	mg/kg-day	1.5E+00	mg/kg-day	0.016
				Cobalt	668	µg/L	3.7E-03	mg/kg-day	NA	---	NA	4.3E-02	mg/kg-day	3.0E-04	mg/kg-day	142
				Copper	49581	µg/L	2.7E-01	mg/kg-day	NA	---	NA	3.2E+00	mg/kg-day	4.0E-02	mg/kg-day	79
				Iron	200000	µg/L	1.1E+00	mg/kg-day	NA	---	NA	1.3E+01	mg/kg-day	7.0E-01	mg/kg-day	18
				Lead	52	µg/L	2.9E-04	mg/kg-day	NA	---	NA	3.3E-03	mg/kg-day	NA	---	NA
				Manganese	4026	µg/L	2.2E-02	mg/kg-day	NA	---	NA	2.6E-01	mg/kg-day	2.4E-02	mg/kg-day	11
				Molybdenum	52	µg/L	2.9E-04	mg/kg-day	NA	---	NA	3.3E-03	mg/kg-day	5.0E-03	mg/kg-day	0.67
				Nickel	402	µg/L	2.2E-03	mg/kg-day	NA	---	NA	2.6E-02	mg/kg-day	2.0E-02	mg/kg-day	1.3
				Vanadium	261	µg/L	1.4E-03	mg/kg-day	NA	---	NA	1.7E-02	mg/kg-day	2.0E-02	mg/kg-day	0.83
				Zinc	3867	µg/L	2.1E-02	mg/kg-day	NA	---	NA	2.5E-01	mg/kg-day	3.0E-01	mg/kg-day	0.82
			Ingestion Total								6.2E-05					270
			Dermal	Aluminum	150000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	7.6	µg/L	2.7E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	4.1E-07	3.2E-06	mg/kg-day	3.0E-04	mg/kg-day	0.011
				Barium	1202	µg/L	4.3E-05	mg/kg-day	NA	---	NA	5.1E-04	mg/kg-day	1.4E-02	mg/kg-day	0.036
				Cadmium	37	µg/L	1.3E-06	mg/kg-day	NA	---	NA	1.5E-05	mg/kg-day	2.5E-05	mg/kg-day	0.62
				Chromium	365	µg/L	2.6E-05	mg/kg-day	NA	---	NA	3.1E-04	mg/kg-day	2.0E-02	mg/kg-day	0.016
				Cobalt	668	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	49581	µg/L	1.8E-03	mg/kg-day	NA	---	NA	2.1E-02	mg/kg-day	4.0E-02	mg/kg-day	0.52
				Iron	200000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lead	52	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	NA	---	NA
				Manganese	4026	µg/L	1.5E-04	mg/kg-day	NA	---	NA	1.7E-03	mg/kg-day	9.6E-04	mg/kg-day	1.77
				Molybdenum	52	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Nickel	402	µg/L	2.9E-06	mg/kg-day	NA	---	NA	3.4E-05	mg/kg-day	8.0E-04	mg/kg-day	0.042
				Vanadium	261	µg/L	9.4E-06	mg/kg-day	NA	---	NA	1.1E-04	mg/kg-day	5.2E-04	mg/kg-day	0.21
				Zinc	3867	µg/L	8.4E-05	mg/kg-day	NA	---	NA	9.8E-04	mg/kg-day	3.0E-01	mg/kg-day	0.0033
			Dermal Total								4.1E-07					3.2
Overburden Groundwater Total											6.3E-05					274
											Total of Receptor Risks Across All Media					6.3E-05
																Total of Receptor Hazards Across All Media
																274

APPENDIX E

TABLE E-28
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADULT OVERBURDEN GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Overburden Groundwater	Ingestion	Aluminum	150000	µg/L	1.4E+00	mg/kg-day	NA	---	NA	4.1E+00	mg/kg-day	1.0E+00	mg/kg-day	4.1
				Arsenic	7.6	µg/L	7.1E-05	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.1E-04	2.1E-04	mg/kg-day	3.0E-04	mg/kg-day	0.69
				Barium	1202	µg/L	1.1E-02	mg/kg-day	NA	---	NA	3.3E-02	mg/kg-day	2.0E-01	mg/kg-day	0.16
				Cadmium	37	µg/L	3.4E-04	mg/kg-day	NA	---	NA	1.0E-03	mg/kg-day	5.0E-04	mg/kg-day	2.0
				Chromium	365	µg/L	3.4E-03	mg/kg-day	NA	---	NA	1.0E-02	mg/kg-day	1.5E+00	mg/kg-day	0.0067
				Cobalt	668	µg/L	6.3E-03	mg/kg-day	NA	---	NA	1.8E-02	mg/kg-day	3.0E-04	mg/kg-day	61
				Copper	49581	µg/L	4.7E-01	mg/kg-day	NA	---	NA	1.4E+00	mg/kg-day	4.0E-02	mg/kg-day	34
				Iron	200000	µg/L	1.9E+00	mg/kg-day	NA	---	NA	5.5E+00	mg/kg-day	7.0E-01	mg/kg-day	7.8
				Lead	52	µg/L	4.9E-04	mg/kg-day	NA	---	NA	1.4E-03	mg/kg-day	NA	---	NA
				Manganese	4026	µg/L	3.8E-02	mg/kg-day	NA	---	NA	1.1E-01	mg/kg-day	2.4E-02	mg/kg-day	4.6
				Molybdenum	52	µg/L	4.9E-04	mg/kg-day	NA	---	NA	1.4E-03	mg/kg-day	5.0E-03	mg/kg-day	0.29
				Nickel	402	µg/L	3.8E-03	mg/kg-day	NA	---	NA	1.1E-02	mg/kg-day	2.0E-02	mg/kg-day	0.55
				Vanadium	261	µg/L	2.4E-03	mg/kg-day	NA	---	NA	7.1E-03	mg/kg-day	2.0E-02	mg/kg-day	0.36
				Zinc	3867	µg/L	3.6E-02	mg/kg-day	NA	---	NA	1.1E-01	mg/kg-day	3.0E-01	mg/kg-day	0.35
			Ingestion Total								1.1E-04					116
			Dermal	Aluminum	150000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	7.6	µg/L	3.7E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	5.6E-07	1.1E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0036
				Barium	1202	µg/L	5.9E-05	mg/kg-day	NA	---	NA	1.7E-04	mg/kg-day	1.4E-02	mg/kg-day	0.012
				Cadmium	37	µg/L	1.8E-06	mg/kg-day	NA	---	NA	5.2E-06	mg/kg-day	2.5E-05	mg/kg-day	0.21
				Chromium	365	µg/L	3.6E-05	mg/kg-day	NA	---	NA	1.0E-04	mg/kg-day	2.0E-02	mg/kg-day	0.0054
				Cobalt	668	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	49581	µg/L	2.4E-03	mg/kg-day	NA	---	NA	7.1E-03	mg/kg-day	4.0E-02	mg/kg-day	0.18
				Iron	200000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lead	52	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	NA	---	NA
				Manganese	4026	µg/L	2.0E-04	mg/kg-day	NA	---	NA	5.8E-04	mg/kg-day	9.6E-04	mg/kg-day	0.60
				Molybdenum	52	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Nickel	402	µg/L	3.9E-06	mg/kg-day	NA	---	NA	1.2E-05	mg/kg-day	8.0E-04	mg/kg-day	0.014
				Vanadium	261	µg/L	1.3E-05	mg/kg-day	NA	---	NA	3.7E-05	mg/kg-day	5.2E-04	mg/kg-day	0.072
				Zinc	3867	µg/L	1.1E-04	mg/kg-day	NA	---	NA	3.3E-04	mg/kg-day	3.0E-01	mg/kg-day	0.0011
			Dermal Total								5.6E-07					1.1
Overburden Groundwater Total											1.1E-04					117
											Total of Receptor Risks Across All Media					1.1E-04
																Total of Receptor Hazards Across All Media
																117

APPENDIX E

TABLE E-29
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - CHILD OVERBURDEN GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Overburden Groundwater	Ingestion	Aluminum	150000	µg/L	4.1E-01	mg/kg-day	NA	---	NA	4.8E+00	mg/kg-day	1.0E+00	mg/kg-day	4.8
				Arsenic	7.6	µg/L	2.1E-05	mg/kg-day	1.5E+00	(mg/kg-day)^-1	3.1E-05	2.4E-04	mg/kg-day	3.0E-04	mg/kg-day	0.81
				Barium	1202	µg/L	3.3E-03	mg/kg-day	NA	---	NA	3.8E-02	mg/kg-day	2.0E-01	mg/kg-day	0.19
				Cadmium	37	µg/L	1.0E-04	mg/kg-day	NA	---	NA	1.2E-03	mg/kg-day	5.0E-04	mg/kg-day	2.3
				Chromium	365	µg/L	1.0E-03	mg/kg-day	NA	---	NA	1.2E-02	mg/kg-day	1.5E+00	mg/kg-day	0.0078
				Cobalt	668	µg/L	1.8E-03	mg/kg-day	NA	---	NA	2.1E-02	mg/kg-day	3.0E-04	mg/kg-day	71
				Copper	49581	µg/L	1.4E-01	mg/kg-day	NA	---	NA	1.6E+00	mg/kg-day	4.0E-02	mg/kg-day	40
				Iron	200000	µg/L	5.5E-01	mg/kg-day	NA	---	NA	6.4E+00	mg/kg-day	7.0E-01	mg/kg-day	9.1
				Lead	52	µg/L	1.4E-04	mg/kg-day	NA	---	NA	1.7E-03	mg/kg-day	NA	---	NA
				Manganese	4026	µg/L	1.1E-02	mg/kg-day	NA	---	NA	1.3E-01	mg/kg-day	2.4E-02	mg/kg-day	5.4
				Molybdenum	52	µg/L	1.4E-04	mg/kg-day	NA	---	NA	1.7E-03	mg/kg-day	5.0E-03	mg/kg-day	0.33
				Nickel	402	µg/L	1.1E-03	mg/kg-day	NA	---	NA	1.3E-02	mg/kg-day	2.0E-02	mg/kg-day	0.64
				Vanadium	261	µg/L	7.1E-04	mg/kg-day	NA	---	NA	8.3E-03	mg/kg-day	2.0E-02	mg/kg-day	0.42
				Zinc	3867	µg/L	1.1E-02	mg/kg-day	NA	---	NA	1.2E-01	mg/kg-day	3.0E-01	mg/kg-day	0.41
			Ingestion Total								3.1E-05					135
			Dermal	Aluminum	150000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	7.6	µg/L	1.4E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.1E-07	1.6E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0053
				Barium	1202	µg/L	2.2E-05	mg/kg-day	NA	---	NA	2.5E-04	mg/kg-day	1.4E-02	mg/kg-day	0.018
				Cadmium	37	µg/L	6.6E-07	mg/kg-day	NA	---	NA	7.7E-06	mg/kg-day	2.5E-05	mg/kg-day	0.31
				Chromium	365	µg/L	1.3E-05	mg/kg-day	NA	---	NA	1.5E-04	mg/kg-day	2.0E-02	mg/kg-day	0.0079
				Cobalt	668	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	49581	µg/L	9.0E-04	mg/kg-day	NA	---	NA	1.0E-02	mg/kg-day	4.0E-02	mg/kg-day	0.26
				Iron	200000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lead	52	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	NA	---	NA
				Manganese	4026	µg/L	7.3E-05	mg/kg-day	NA	---	NA	8.5E-04	mg/kg-day	9.6E-04	mg/kg-day	0.88
				Molybdenum	52	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Nickel	402	µg/L	1.5E-06	mg/kg-day	NA	---	NA	1.7E-05	mg/kg-day	8.0E-04	mg/kg-day	0.021
				Vanadium	261	µg/L	4.7E-06	mg/kg-day	NA	---	NA	5.5E-05	mg/kg-day	5.2E-04	mg/kg-day	0.11
				Zinc	3867	µg/L	4.2E-05	mg/kg-day	NA	---	NA	4.9E-04	mg/kg-day	3.0E-01	mg/kg-day	0.0016
			Dermal Total								2.1E-07					1.6
Overburden Groundwater Total											3.1E-05					137
										Total of Receptor Risks Across All Media	3.1E-05			Total of Receptor Hazards Across All Media		137

APPENDIX E

TABLE E-30
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADULT OVERBURDEN GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Overburden Groundwater	Ingestion	Aluminum	150000	µg/L	2.6E-01	mg/kg-day	NA	---	NA	2.1E+00	mg/kg-day	1.0E+00	mg/kg-day	2.1
				Arsenic	7.6	µg/L	1.3E-05	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.0E-05	1.0E-04	mg/kg-day	3.0E-04	mg/kg-day	0.35
				Barium	1202	µg/L	2.1E-03	mg/kg-day	NA	---	NA	1.6E-02	mg/kg-day	2.0E-01	mg/kg-day	0.082
				Cadmium	37	µg/L	6.4E-05	mg/kg-day	NA	---	NA	5.0E-04	mg/kg-day	5.0E-04	mg/kg-day	1.0
				Chromium	365	µg/L	6.4E-04	mg/kg-day	NA	---	NA	5.0E-03	mg/kg-day	1.5E+00	mg/kg-day	0.0033
				Cobalt	668	µg/L	1.2E-03	mg/kg-day	NA	---	NA	9.2E-03	mg/kg-day	3.0E-04	mg/kg-day	31
				Copper	49581	µg/L	8.7E-02	mg/kg-day	NA	---	NA	6.8E-01	mg/kg-day	4.0E-02	mg/kg-day	17
				Iron	200000	µg/L	3.5E-01	mg/kg-day	NA	---	NA	2.7E+00	mg/kg-day	7.0E-01	mg/kg-day	3.9
				Lead	52	µg/L	9.2E-05	mg/kg-day	NA	---	NA	7.2E-04	mg/kg-day	NA	---	NA
				Manganese	4026	µg/L	7.1E-03	mg/kg-day	NA	---	NA	5.5E-02	mg/kg-day	2.4E-02	mg/kg-day	2.3
				Molybdenum	52	µg/L	9.2E-05	mg/kg-day	NA	---	NA	7.2E-04	mg/kg-day	5.0E-03	mg/kg-day	0.14
				Nickel	402	µg/L	7.1E-04	mg/kg-day	NA	---	NA	5.5E-03	mg/kg-day	2.0E-02	mg/kg-day	0.28
				Vanadium	261	µg/L	4.6E-04	mg/kg-day	NA	---	NA	3.6E-03	mg/kg-day	2.0E-02	mg/kg-day	0.18
				Zinc	3867	µg/L	6.8E-03	mg/kg-day	NA	---	NA	5.3E-02	mg/kg-day	3.0E-01	mg/kg-day	0.18
			Ingestion Total								2.0E-05					58
			Dermal	Aluminum	150000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	7.6	µg/L	7.0E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.0E-07	5.4E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0018
				Barium	1202	µg/L	1.1E-05	mg/kg-day	NA	---	NA	8.6E-05	mg/kg-day	1.4E-02	mg/kg-day	0.0061
				Cadmium	37	µg/L	3.4E-07	mg/kg-day	NA	---	NA	2.6E-06	mg/kg-day	2.5E-05	mg/kg-day	0.10
				Chromium	365	µg/L	6.7E-06	mg/kg-day	NA	---	NA	5.2E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0027
				Cobalt	668	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	49581	µg/L	4.6E-04	mg/kg-day	NA	---	NA	3.5E-03	mg/kg-day	4.0E-02	mg/kg-day	0.089
				Iron	200000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lead	52	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	NA	---	NA
				Manganese	4026	µg/L	3.7E-05	mg/kg-day	NA	---	NA	2.9E-04	mg/kg-day	9.6E-04	mg/kg-day	0.30
				Molybdenum	52	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Nickel	402	µg/L	7.4E-07	mg/kg-day	NA	---	NA	5.8E-06	mg/kg-day	8.0E-04	mg/kg-day	0.0072
				Vanadium	261	µg/L	2.4E-06	mg/kg-day	NA	---	NA	1.9E-05	mg/kg-day	5.2E-04	mg/kg-day	0.036
				Zinc	3867	µg/L	2.1E-05	mg/kg-day	NA	---	NA	1.7E-04	mg/kg-day	3.0E-01	mg/kg-day	0.00055
			Dermal Total								1.0E-07					0.55
Overburden Groundwater Total											2.0E-05					59
											Total of Receptor Risks Across All Media					
											2.0E-05					59
												Total of Receptor Hazards Across All Media				

APPENDIX E

TABLE E-31
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - CHILD SHALLOW BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Shallow Bedrock Groundwater	Ingestion	Aluminum	360000	µg/L	2.0E+00	mg/kg-day	NA	---	NA	2.3E+01	mg/kg-day	1.0E+00	mg/kg-day	23
				Antimony	4.3	µg/L	2.4E-05	mg/kg-day	NA	---	NA	2.8E-04	mg/kg-day	4.0E-04	mg/kg-day	0.69
				Arsenic	4.9	µg/L	2.7E-05	mg/kg-day	1.5E+00	(mg/kg-day)^-1	4.0E-05	3.1E-04	mg/kg-day	3.0E-04	mg/kg-day	1.0
				Beryllium	7.3	µg/L	4.0E-05	mg/kg-day	NA	---	NA	4.7E-04	mg/kg-day	2.0E-03	mg/kg-day	0.23
				Cadmium	13	µg/L	7.1E-05	mg/kg-day	NA	---	NA	8.3E-04	mg/kg-day	5.0E-04	mg/kg-day	1.7
				Chromium	172	µg/L	9.4E-04	mg/kg-day	NA	---	NA	1.1E-02	mg/kg-day	1.5E+00	mg/kg-day	0.0073
				Cobalt	267	µg/L	1.5E-03	mg/kg-day	NA	---	NA	1.7E-02	mg/kg-day	3.0E-04	mg/kg-day	57
				Copper	10600	µg/L	5.8E-02	mg/kg-day	NA	---	NA	6.8E-01	mg/kg-day	4.0E-02	mg/kg-day	17
				Iron	310000	µg/L	1.7E+00	mg/kg-day	NA	---	NA	2.0E+01	mg/kg-day	7.0E-01	mg/kg-day	28
				Lead	27	µg/L	1.5E-04	mg/kg-day	NA	---	NA	1.7E-03	mg/kg-day	NA	---	NA
				Manganese	3000	µg/L	1.6E-02	mg/kg-day	NA	---	NA	1.9E-01	mg/kg-day	2.4E-02	mg/kg-day	8.0
				Molybdenum	93	µg/L	5.1E-04	mg/kg-day	NA	---	NA	5.9E-03	mg/kg-day	5.0E-03	mg/kg-day	1.2
				Nickel	125	µg/L	6.8E-04	mg/kg-day	NA	---	NA	8.0E-03	mg/kg-day	2.0E-02	mg/kg-day	0.40
				Vanadium	37	µg/L	2.0E-04	mg/kg-day	NA	---	NA	2.3E-03	mg/kg-day	2.0E-02	mg/kg-day	0.12
				Zinc	1902	µg/L	1.0E-02	mg/kg-day	NA	---	NA	1.2E-01	mg/kg-day	3.0E-01	mg/kg-day	0.41
			Ingestion Total								4.0E-05					139
			Dermal	Aluminum	360000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	4.3	µg/L	1.6E-07	mg/kg-day	NA	---	NA	1.8E-06	mg/kg-day	6.0E-05	mg/kg-day	0.030
				Arsenic	4.9	µg/L	1.8E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.6E-07	2.1E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0069
				Beryllium	7.3	µg/L	2.6E-07	mg/kg-day	NA	---	NA	3.1E-06	mg/kg-day	1.4E-05	mg/kg-day	0.22
				Cadmium	13	µg/L	4.7E-07	mg/kg-day	NA	---	NA	5.5E-06	mg/kg-day	2.5E-05	mg/kg-day	0.22
				Chromium	172	µg/L	1.2E-05	mg/kg-day	NA	---	NA	1.5E-04	mg/kg-day	2.0E-02	mg/kg-day	0.0074
				Cobalt	267	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	10600	µg/L	3.8E-04	mg/kg-day	NA	---	NA	4.5E-03	mg/kg-day	4.0E-02	mg/kg-day	0.11
				Iron	310000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lead	27	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	NA	---	NA
				Manganese	3000	µg/L	1.1E-04	mg/kg-day	NA	---	NA	1.3E-03	mg/kg-day	9.6E-04	mg/kg-day	1.3
				Molybdenum	93	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Nickel	125	µg/L	9.0E-07	mg/kg-day	NA	---	NA	1.1E-05	mg/kg-day	8.0E-04	mg/kg-day	0.013
				Vanadium	37	µg/L	1.3E-06	mg/kg-day	NA	---	NA	1.5E-05	mg/kg-day	5.2E-04	mg/kg-day	0.030
				Zinc	1902	µg/L	4.1E-05	mg/kg-day	NA	---	NA	4.8E-04	mg/kg-day	3.0E-01	mg/kg-day	0.0016
			Dermal Total								2.6E-07					2.0
Shallow Bedrock Groundwater Total											4.0E-05					141
											Total of Receptor Risks Across All Media	4.0E-05		Total of Receptor Hazards Across All Media		141

APPENDIX E

TABLE E-32
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADULT SHALLOW BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Shallow Bedrock Groundwater	Ingestion	Aluminum	360000	µg/L	3.4E+00	mg/kg-day	NA	---	NA	9.9E+00	mg/kg-day	1.0E+00	mg/kg-day	9.9
				Antimony	4.3	µg/L	4.1E-05	mg/kg-day	NA	---	NA	1.2E-04	mg/kg-day	4.0E-04	mg/kg-day	0.30
				Arsenic	4.9	µg/L	4.6E-05	mg/kg-day	1.5E+00	(mg/kg-day)^-1	6.9E-05	1.3E-04	mg/kg-day	3.0E-04	mg/kg-day	0.45
				Beryllium	7.3	µg/L	6.9E-05	mg/kg-day	NA	---	NA	2.0E-04	mg/kg-day	2.0E-03	mg/kg-day	0.10
				Cadmium	13	µg/L	1.2E-04	mg/kg-day	NA	---	NA	3.6E-04	mg/kg-day	5.0E-04	mg/kg-day	0.71
				Chromium	172	µg/L	1.6E-03	mg/kg-day	NA	---	NA	4.7E-03	mg/kg-day	1.5E+00	mg/kg-day	0.0031
				Cobalt	267	µg/L	2.5E-03	mg/kg-day	NA	---	NA	7.3E-03	mg/kg-day	3.0E-04	mg/kg-day	24
				Copper	10600	µg/L	1.0E-01	mg/kg-day	NA	---	NA	2.9E-01	mg/kg-day	4.0E-02	mg/kg-day	7.3
				Iron	310000	µg/L	2.9E+00	mg/kg-day	NA	---	NA	8.5E+00	mg/kg-day	7.0E-01	mg/kg-day	12
				Lead	27	µg/L	2.6E-04	mg/kg-day	NA	---	NA	7.5E-04	mg/kg-day	NA	---	NA
				Manganese	3000	µg/L	2.8E-02	mg/kg-day	NA	---	NA	8.2E-02	mg/kg-day	2.4E-02	mg/kg-day	3.4
				Molybdenum	93	µg/L	8.7E-04	mg/kg-day	NA	---	NA	2.5E-03	mg/kg-day	5.0E-03	mg/kg-day	0.51
				Nickel	125	µg/L	1.2E-03	mg/kg-day	NA	---	NA	3.4E-03	mg/kg-day	2.0E-02	mg/kg-day	0.17
				Vanadium	37	µg/L	3.4E-04	mg/kg-day	NA	---	NA	1.0E-03	mg/kg-day	2.0E-02	mg/kg-day	0.050
				Zinc	1902	µg/L	1.8E-02	mg/kg-day	NA	---	NA	5.2E-02	mg/kg-day	3.0E-01	mg/kg-day	0.17
			Ingestion Total								6.9E-05					60
			Dermal	Aluminum	360000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	4.3	µg/L	2.1E-07	mg/kg-day	NA	---	NA	6.2E-07	mg/kg-day	6.0E-05	mg/kg-day	0.010
				Arsenic	4.9	µg/L	2.4E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	3.6E-07	7.0E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0023
				Beryllium	7.3	µg/L	3.6E-07	mg/kg-day	NA	---	NA	1.0E-06	mg/kg-day	1.4E-05	mg/kg-day	0.075
				Cadmium	13	µg/L	6.4E-07	mg/kg-day	NA	---	NA	1.9E-06	mg/kg-day	2.5E-05	mg/kg-day	0.074
				Chromium	172	µg/L	1.7E-05	mg/kg-day	NA	---	NA	4.9E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0025
				Cobalt	267	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	10600	µg/L	5.2E-04	mg/kg-day	NA	---	NA	1.5E-03	mg/kg-day	4.0E-02	mg/kg-day	0.038
				Iron	310000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lead	27	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	NA	---	NA
				Manganese	3000	µg/L	1.5E-04	mg/kg-day	NA	---	NA	4.3E-04	mg/kg-day	9.6E-04	mg/kg-day	0.45
				Molybdenum	93	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Nickel	125	µg/L	1.2E-06	mg/kg-day	NA	---	NA	3.6E-06	mg/kg-day	8.0E-04	mg/kg-day	0.0045
				Vanadium	37	µg/L	1.8E-06	mg/kg-day	NA	---	NA	5.2E-06	mg/kg-day	5.2E-04	mg/kg-day	0.010
				Zinc	1902	µg/L	5.6E-05	mg/kg-day	NA	---	NA	1.6E-04	mg/kg-day	3.0E-01	mg/kg-day	0.00054
			Dermal Total								3.6E-07					0.66
Shallow Bedrock Groundwater Total											6.9E-05					60
										Total of Receptor Risks Across All Media	6.9E-05			Total of Receptor Hazards Across All Media		60

APPENDIX E

TABLE E-33
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - CHILD SHALLOW BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Shallow Bedrock Groundwater	Ingestion	Aluminum	360000	µg/L	9.9E-01	mg/kg-day	NA	---	NA	1.2E+01	mg/kg-day	1.0E+00	mg/kg-day	12
				Antimony	4.3	µg/L	1.2E-05	mg/kg-day	NA	---	NA	1.4E-04	mg/kg-day	4.0E-04	mg/kg-day	0.35
				Arsenic	4.9	µg/L	1.3E-05	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.0E-05	1.6E-04	mg/kg-day	3.0E-04	mg/kg-day	0.52
				Beryllium	7.3	µg/L	2.0E-05	mg/kg-day	NA	---	NA	2.3E-04	mg/kg-day	2.0E-03	mg/kg-day	0.12
				Cadmium	13	µg/L	3.6E-05	mg/kg-day	NA	---	NA	4.2E-04	mg/kg-day	5.0E-04	mg/kg-day	0.83
				Chromium	172	µg/L	4.7E-04	mg/kg-day	NA	---	NA	5.5E-03	mg/kg-day	1.5E+00	mg/kg-day	0.0037
				Cobalt	267	µg/L	7.3E-04	mg/kg-day	NA	---	NA	8.5E-03	mg/kg-day	3.0E-04	mg/kg-day	28
				Copper	10600	µg/L	2.9E-02	mg/kg-day	NA	---	NA	3.4E-01	mg/kg-day	4.0E-02	mg/kg-day	8.5
				Iron	310000	µg/L	8.5E-01	mg/kg-day	NA	---	NA	9.9E+00	mg/kg-day	7.0E-01	mg/kg-day	14
				Lead	27	µg/L	7.5E-05	mg/kg-day	NA	---	NA	8.7E-04	mg/kg-day	NA	---	NA
				Manganese	3000	µg/L	8.2E-03	mg/kg-day	NA	---	NA	9.6E-02	mg/kg-day	2.4E-02	mg/kg-day	4.0
				Molybdenum	93	µg/L	2.5E-04	mg/kg-day	NA	---	NA	3.0E-03	mg/kg-day	5.0E-03	mg/kg-day	0.59
				Nickel	125	µg/L	3.4E-04	mg/kg-day	NA	---	NA	4.0E-03	mg/kg-day	2.0E-02	mg/kg-day	0.20
				Vanadium	37	µg/L	1.0E-04	mg/kg-day	NA	---	NA	1.2E-03	mg/kg-day	2.0E-02	mg/kg-day	0.058
				Zinc	1902	µg/L	5.2E-03	mg/kg-day	NA	---	NA	6.1E-02	mg/kg-day	3.0E-01	mg/kg-day	0.20
			Ingestion Total								2.0E-05					69
			Dermal	Aluminum	360000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	4.3	µg/L	7.8E-08	mg/kg-day	NA	---	NA	9.1E-07	mg/kg-day	6.0E-05	mg/kg-day	0.015
				Arsenic	4.9	µg/L	8.8E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.3E-07	1.0E-06	mg/kg-day	3.0E-04	mg/kg-day	0.0034
				Beryllium	7.3	µg/L	1.3E-07	mg/kg-day	NA	---	NA	1.5E-06	mg/kg-day	1.4E-05	mg/kg-day	0.11
				Cadmium	13	µg/L	2.4E-07	mg/kg-day	NA	---	NA	2.7E-06	mg/kg-day	2.5E-05	mg/kg-day	0.11
				Chromium	172	µg/L	6.2E-06	mg/kg-day	NA	---	NA	7.3E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0037
				Cobalt	267	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	10600	µg/L	1.9E-04	mg/kg-day	NA	---	NA	2.2E-03	mg/kg-day	4.0E-02	mg/kg-day	0.056
				Iron	310000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lead	27	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	NA	---	NA
				Manganese	3000	µg/L	5.4E-05	mg/kg-day	NA	---	NA	6.3E-04	mg/kg-day	9.6E-04	mg/kg-day	0.66
				Molybdenum	93	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Nickel	125	µg/L	4.5E-07	mg/kg-day	NA	---	NA	5.3E-06	mg/kg-day	8.0E-04	mg/kg-day	0.0066
				Vanadium	37	µg/L	6.6E-07	mg/kg-day	NA	---	NA	7.7E-06	mg/kg-day	5.2E-04	mg/kg-day	0.015
				Zinc	1902	µg/L	2.1E-05	mg/kg-day	NA	---	NA	2.4E-04	mg/kg-day	3.0E-01	mg/kg-day	0.00080
			Dermal Total								1.3E-07					0.98
Shallow Bedrock Groundwater Total											2.0E-05					70
											Total of Receptor Risks Across All Media	2.0E-05		Total of Receptor Hazards Across All Media		70

APPENDIX E

TABLE E-34
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADULT SHALLOW BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Shallow Bedrock Groundwater	Ingestion	Aluminum	360000	µg/L	6.3E-01	mg/kg-day	NA	---	NA	4.9E+00	mg/kg-day	1.0E+00	mg/kg-day	4.9
				Antimony	4.3	µg/L	7.6E-06	mg/kg-day	NA	---	NA	5.9E-05	mg/kg-day	4.0E-04	mg/kg-day	0.15
				Arsenic	4.9	µg/L	8.6E-06	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.3E-05	6.7E-05	mg/kg-day	3.0E-04	mg/kg-day	0.22
				Beryllium	7.3	µg/L	1.3E-05	mg/kg-day	NA	---	NA	1.0E-04	mg/kg-day	2.0E-03	mg/kg-day	0.050
				Cadmium	13	µg/L	2.3E-05	mg/kg-day	NA	---	NA	1.8E-04	mg/kg-day	5.0E-04	mg/kg-day	0.36
				Chromium	172	µg/L	3.0E-04	mg/kg-day	NA	---	NA	2.4E-03	mg/kg-day	1.5E+00	mg/kg-day	0.0016
				Cobalt	267	µg/L	4.7E-04	mg/kg-day	NA	---	NA	3.7E-03	mg/kg-day	3.0E-04	mg/kg-day	12
				Copper	10600	µg/L	1.9E-02	mg/kg-day	NA	---	NA	1.5E-01	mg/kg-day	4.0E-02	mg/kg-day	3.6
				Iron	310000	µg/L	5.5E-01	mg/kg-day	NA	---	NA	4.2E+00	mg/kg-day	7.0E-01	mg/kg-day	6.1
				Lead	27	µg/L	4.8E-05	mg/kg-day	NA	---	NA	3.7E-04	mg/kg-day	NA	---	NA
				Manganese	3000	µg/L	5.3E-03	mg/kg-day	NA	---	NA	4.1E-02	mg/kg-day	2.4E-02	mg/kg-day	1.7
				Molybdenum	93	µg/L	1.6E-04	mg/kg-day	NA	---	NA	1.3E-03	mg/kg-day	5.0E-03	mg/kg-day	0.25
				Nickel	125	µg/L	2.2E-04	mg/kg-day	NA	---	NA	1.7E-03	mg/kg-day	2.0E-02	mg/kg-day	0.086
				Vanadium	37	µg/L	6.4E-05	mg/kg-day	NA	---	NA	5.0E-04	mg/kg-day	2.0E-02	mg/kg-day	0.025
				Zinc	1902	µg/L	3.3E-03	mg/kg-day	NA	---	NA	2.6E-02	mg/kg-day	3.0E-01	mg/kg-day	0.087
			Ingestion Total								1.3E-05					30
			Dermal	Aluminum	360000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Antimony	4.3	µg/L	4.0E-08	mg/kg-day	NA	---	NA	3.1E-07	mg/kg-day	6.0E-05	mg/kg-day	0.0051
				Arsenic	4.9	µg/L	4.5E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	6.7E-08	3.5E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0012
				Beryllium	7.3	µg/L	6.7E-08	mg/kg-day	NA	---	NA	5.2E-07	mg/kg-day	1.4E-05	mg/kg-day	0.037
				Cadmium	13	µg/L	1.2E-07	mg/kg-day	NA	---	NA	9.3E-07	mg/kg-day	2.5E-05	mg/kg-day	0.037
				Chromium	172	µg/L	3.2E-06	mg/kg-day	NA	---	NA	2.5E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0013
				Cobalt	267	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	10600	µg/L	9.7E-05	mg/kg-day	NA	---	NA	7.6E-04	mg/kg-day	4.0E-02	mg/kg-day	0.019
				Iron	310000	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Lead	27	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	NA	---	NA
				Manganese	3000	µg/L	2.8E-05	mg/kg-day	NA	---	NA	2.1E-04	mg/kg-day	9.6E-04	mg/kg-day	0.22
				Molybdenum	93	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	5.0E-03	mg/kg-day	NA
				Nickel	125	µg/L	2.3E-07	mg/kg-day	NA	---	NA	1.8E-06	mg/kg-day	8.0E-04	mg/kg-day	0.0022
				Vanadium	37	µg/L	3.4E-07	mg/kg-day	NA	---	NA	2.6E-06	mg/kg-day	5.2E-04	mg/kg-day	0.0050
				Zinc	1902	µg/L	1.0E-05	mg/kg-day	NA	---	NA	8.2E-05	mg/kg-day	3.0E-01	mg/kg-day	0.00027
			Dermal Total								6.7E-08					0.33
Shallow Bedrock Groundwater Total											1.3E-05					30
										Total of Receptor Risks Across All Media	1.3E-05			Total of Receptor Hazards Across All Media		30

APPENDIX E

TABLE E-35
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - CHILD DEEP BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Deep Bedrock Groundwater	Ingestion	Bis(2-Ethylhexyl)Phthalate	7.5	µg/L	4.1E-05	mg/kg-day	1.4E-02	(mg/kg-day)^-1	5.8E-07	4.8E-04	mg/kg-day	2.0E-02	mg/kg-day	0.024
				Aluminum	6810	µg/L	3.7E-02	mg/kg-day	NA	---	NA	4.4E-01	mg/kg-day	1.0E+00	mg/kg-day	0.44
				Arsenic	2.6	µg/L	1.4E-05	mg/kg-day	1.5E+00	(mg/kg-day)^-1	2.1E-05	1.7E-04	mg/kg-day	3.0E-04	mg/kg-day	0.55
				Cadmium	6.6	µg/L	3.6E-05	mg/kg-day	NA	---	NA	4.2E-04	mg/kg-day	5.0E-04	mg/kg-day	0.84
				Chromium	132	µg/L	7.2E-04	mg/kg-day	NA	---	NA	8.4E-03	mg/kg-day	1.5E+00	mg/kg-day	0.0056
				Cobalt	153	µg/L	8.4E-04	mg/kg-day	NA	---	NA	9.8E-03	mg/kg-day	3.0E-04	mg/kg-day	33
				Copper	8910	µg/L	4.9E-02	mg/kg-day	NA	---	NA	5.7E-01	mg/kg-day	4.0E-02	mg/kg-day	14
				Iron	16948	µg/L	9.3E-02	mg/kg-day	NA	---	NA	1.1E+00	mg/kg-day	7.0E-01	mg/kg-day	1.5
				Manganese	1433	µg/L	7.9E-03	mg/kg-day	NA	---	NA	9.2E-02	mg/kg-day	2.4E-02	mg/kg-day	3.8
				Nickel	87	µg/L	4.8E-04	mg/kg-day	NA	---	NA	5.6E-03	mg/kg-day	2.4E-02	mg/kg-day	0.23
				Vanadium	5.0	µg/L	2.7E-05	mg/kg-day	NA	---	NA	3.2E-04	mg/kg-day	2.0E-02	mg/kg-day	0.016
			Ingestion Total								2.2E-05					54
			Dermal	Bis(2-Ethylhexyl)Phthalate	7.5	µg/L	NA	mg/kg-day	1.4E-02	(mg/kg-day)^-1	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Aluminum	6810	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	2.6	µg/L	9.4E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.4E-07	1.1E-06	mg/kg-day	3.0E-04	mg/kg-day	0.004
				Cadmium	6.6	µg/L	2.4E-07	mg/kg-day	NA	---	NA	2.8E-06	mg/kg-day	2.5E-05	mg/kg-day	0.11
				Chromium	132	µg/L	9.5E-06	mg/kg-day	NA	---	NA	1.1E-04	mg/kg-day	2.0E-02	mg/kg-day	0.0057
				Cobalt	153	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	8910	µg/L	3.2E-04	mg/kg-day	NA	---	NA	3.8E-03	mg/kg-day	4.0E-02	mg/kg-day	0.094
				Iron	16948	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	1433	µg/L	5.2E-05	mg/kg-day	NA	---	NA	6.0E-04	mg/kg-day	9.6E-04	mg/kg-day	0.63
				Nickel	87	µg/L	6.3E-07	mg/kg-day	NA	---	NA	7.4E-06	mg/kg-day	8.0E-04	mg/kg-day	0.0092
				Vanadium	5.0	µg/L	1.8E-07	mg/kg-day	NA	---	NA	2.1E-06	mg/kg-day	5.2E-04	mg/kg-day	0.004
			Dermal Total								1.4E-07					0.86
Deep Bedrock Groundwater Total											2.2E-05					55
										Total of Receptor Risks Across All Media	2.2E-05			Total of Receptor Hazards Across All Media		55

APPENDIX E

TABLE E-36
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADULT DEEP BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Deep Bedrock Groundwater	Ingestion	Bis(2-Ethylhexyl)Phthalate	7.5	µg/L	7.0E-05	mg/kg-day	1.4E-02	(mg/kg-day)^-1	9.9E-07	2.1E-04	mg/kg-day	2.0E-02	mg/kg-day	0.010
				Aluminum	6810	µg/L	6.4E-02	mg/kg-day	NA	---	NA	1.9E-01	mg/kg-day	1.0E+00	mg/kg-day	0.19
				Arsenic	2.6	µg/L	2.4E-05	mg/kg-day	1.5E+00	(mg/kg-day)^-1	3.7E-05	7.1E-05	mg/kg-day	3.0E-04	mg/kg-day	0.24
				Cadmium	6.6	µg/L	6.2E-05	mg/kg-day	NA	---	NA	1.8E-04	mg/kg-day	5.0E-04	mg/kg-day	0.36
				Chromium	132	µg/L	1.2E-03	mg/kg-day	NA	---	NA	3.6E-03	mg/kg-day	1.5E+00	mg/kg-day	0.0024
				Cobalt	153	µg/L	1.4E-03	mg/kg-day	NA	---	NA	4.2E-03	mg/kg-day	3.0E-04	mg/kg-day	14
				Copper	8910	µg/L	8.4E-02	mg/kg-day	NA	---	NA	2.4E-01	mg/kg-day	4.0E-02	mg/kg-day	6.1
				Iron	16948	µg/L	1.6E-01	mg/kg-day	NA	---	NA	4.6E-01	mg/kg-day	7.0E-01	mg/kg-day	0.66
				Manganese	1433	µg/L	1.3E-02	mg/kg-day	NA	---	NA	3.9E-02	mg/kg-day	2.4E-02	mg/kg-day	1.6
				Nickel	87	µg/L	8.2E-04	mg/kg-day	NA	---	NA	2.4E-03	mg/kg-day	2.4E-02	mg/kg-day	0.10
				Vanadium	5.0	µg/L	4.7E-05	mg/kg-day	NA	---	NA	1.4E-04	mg/kg-day	2.0E-02	mg/kg-day	0.007
			Ingestion Total								3.8E-05					23
			Dermal	Bis(2-Ethylhexyl)Phthalate	7.5	µg/L	NA	mg/kg-day	1.4E-02	(mg/kg-day)^-1	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Aluminum	6810	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	2.6	µg/L	1.3E-07	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.9E-07	3.7E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0012
				Cadmium	6.6	µg/L	3.2E-07	mg/kg-day	NA	---	NA	9.4E-07	mg/kg-day	2.5E-05	mg/kg-day	0.038
				Chromium	132	µg/L	1.3E-05	mg/kg-day	NA	---	NA	3.8E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0019
				Cobalt	153	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	8910	µg/L	4.4E-04	mg/kg-day	NA	---	NA	1.3E-03	mg/kg-day	4.0E-02	mg/kg-day	0.032
				Iron	16948	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	1433	µg/L	7.0E-05	mg/kg-day	NA	---	NA	2.0E-04	mg/kg-day	9.6E-04	mg/kg-day	0.21
				Nickel	87	µg/L	8.6E-07	mg/kg-day	NA	---	NA	2.5E-06	mg/kg-day	8.0E-04	mg/kg-day	0.0031
				Vanadium	5.0	µg/L	2.5E-07	mg/kg-day	NA	---	NA	7.2E-07	mg/kg-day	5.2E-04	mg/kg-day	0.0014
			Dermal Total								1.9E-07					0.29
Deep Bedrock Groundwater Total											3.8E-05					24
										Total of Receptor Risks Across All Media	3.8E-05			Total of Receptor Hazards Across All Media		24

APPENDIX E

TABLE E-37
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - CHILD DEEP BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Deep Bedrock Groundwater	Ingestion	Bis(2-Ethylhexyl)Phthalate	7.5	µg/L	2.1E-05	mg/kg-day	1.4E-02	(mg/kg-day)^-1	2.9E-07	2.4E-04	mg/kg-day	2.0E-02	mg/kg-day	0.012
				Aluminum	6810	µg/L	1.9E-02	mg/kg-day	NA	---	NA	2.2E-01	mg/kg-day	1.0E+00	mg/kg-day	0.22
				Arsenic	2.6	µg/L	7.1E-06	mg/kg-day	1.5E+00	(mg/kg-day)^-1	1.1E-05	8.3E-05	mg/kg-day	3.0E-04	mg/kg-day	0.28
				Cadmium	6.6	µg/L	1.8E-05	mg/kg-day	NA	---	NA	2.1E-04	mg/kg-day	5.0E-04	mg/kg-day	0.42
				Chromium	132	µg/L	3.6E-04	mg/kg-day	NA	---	NA	4.2E-03	mg/kg-day	1.5E+00	mg/kg-day	0.0028
				Cobalt	153	µg/L	4.2E-04	mg/kg-day	NA	---	NA	4.9E-03	mg/kg-day	3.0E-04	mg/kg-day	16
				Copper	8910	µg/L	2.4E-02	mg/kg-day	NA	---	NA	2.8E-01	mg/kg-day	4.0E-02	mg/kg-day	7.1
				Iron	16948	µg/L	4.6E-02	mg/kg-day	NA	---	NA	5.4E-01	mg/kg-day	7.0E-01	mg/kg-day	0.77
				Manganese	1433	µg/L	3.9E-03	mg/kg-day	NA	---	NA	4.6E-02	mg/kg-day	2.4E-02	mg/kg-day	1.9
				Nickel	87	µg/L	2.4E-04	mg/kg-day	NA	---	NA	2.8E-03	mg/kg-day	2.4E-02	mg/kg-day	0.12
				Vanadium	5.0	µg/L	1.4E-05	mg/kg-day	NA	---	NA	1.6E-04	mg/kg-day	2.0E-02	mg/kg-day	0.0080
			Ingestion Total								1.1E-05					27
			Dermal	Bis(2-Ethylhexyl)Phthalate	7.5	µg/L	NA	mg/kg-day	1.4E-02	(mg/kg-day)^-1	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Aluminum	6810	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	2.6	µg/L	4.7E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	7.1E-08	5.5E-07	mg/kg-day	3.0E-04	mg/kg-day	0.0018
				Cadmium	6.6	µg/L	1.2E-07	mg/kg-day	NA	---	NA	1.4E-06	mg/kg-day	2.5E-05	mg/kg-day	0.056
				Chromium	132	µg/L	4.8E-06	mg/kg-day	NA	---	NA	5.6E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0029
				Cobalt	153	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	8910	µg/L	1.6E-04	mg/kg-day	NA	---	NA	1.9E-03	mg/kg-day	4.0E-02	mg/kg-day	0.047
				Iron	16948	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	1433	µg/L	2.6E-05	mg/kg-day	NA	---	NA	3.0E-04	mg/kg-day	9.6E-04	mg/kg-day	0.31
				Nickel	87	µg/L	3.2E-07	mg/kg-day	NA	---	NA	3.7E-06	mg/kg-day	8.0E-04	mg/kg-day	0.0046
				Vanadium	5.0	µg/L	9.0E-08	mg/kg-day	NA	---	NA	1.1E-06	mg/kg-day	5.2E-04	mg/kg-day	0.0020
			Dermal Total								7.1E-08					0.43
Deep Bedrock Groundwater Total											1.1E-05					28
											Total of Receptor Risks Across All Media					
											1.1E-05	Total of Receptor Hazards Across All Media				28

APPENDIX E

TABLE E-38
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADULT DEEP BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Deep Bedrock Groundwater	Ingestion	Bis(2-Ethylhexyl)Phthalate	7.5	µg/L	1.3E-05	mg/kg-day	1.4E-02	(mg/kg-day)^-1	1.8E-07	1.0E-04	mg/kg-day	2.0E-02	mg/kg-day	0.0051
				Aluminum	6810	µg/L	1.2E-02	mg/kg-day	NA	---	NA	9.3E-02	mg/kg-day	1.0E+00	mg/kg-day	0.093
				Arsenic	2.6	µg/L	4.6E-06	mg/kg-day	1.5E+00	(mg/kg-day)^-1	6.9E-06	3.6E-05	mg/kg-day	3.0E-04	mg/kg-day	0.12
				Cadmium	6.6	µg/L	1.2E-05	mg/kg-day	NA	---	NA	9.0E-05	mg/kg-day	5.0E-04	mg/kg-day	0.18
				Chromium	132	µg/L	2.3E-04	mg/kg-day	NA	---	NA	1.8E-03	mg/kg-day	1.5E+00	mg/kg-day	0.0012
				Cobalt	153	µg/L	2.7E-04	mg/kg-day	NA	---	NA	2.1E-03	mg/kg-day	3.0E-04	mg/kg-day	7.0
				Copper	8910	µg/L	1.6E-02	mg/kg-day	NA	---	NA	1.2E-01	mg/kg-day	4.0E-02	mg/kg-day	3.1
				Iron	16948	µg/L	3.0E-02	mg/kg-day	NA	---	NA	2.3E-01	mg/kg-day	7.0E-01	mg/kg-day	0.33
				Manganese	1433	µg/L	2.5E-03	mg/kg-day	NA	---	NA	2.0E-02	mg/kg-day	2.4E-02	mg/kg-day	0.82
				Nickel	87	µg/L	1.5E-04	mg/kg-day	NA	---	NA	1.2E-03	mg/kg-day	2.4E-02	mg/kg-day	0.050
				Vanadium	5.0	µg/L	8.8E-06	mg/kg-day	NA	---	NA	6.8E-05	mg/kg-day	2.0E-02	mg/kg-day	0.003
			Ingestion Total								7.1E-06					12
			Dermal	Bis(2-Ethylhexyl)Phthalate	7.5	µg/L	NA	mg/kg-day	1.4E-02	(mg/kg-day)^-1	NA	NA	mg/kg-day	2.0E-02	mg/kg-day	NA
				Aluminum	6810	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	1.0E+00	mg/kg-day	NA
				Arsenic	2.6	µg/L	2.4E-08	mg/kg-day	1.5E+00	(mg/kg-day)^-1	3.6E-08	1.9E-07	mg/kg-day	3.0E-04	mg/kg-day	0.00062
				Cadmium	6.6	µg/L	6.1E-08	mg/kg-day	NA	---	NA	4.7E-07	mg/kg-day	2.5E-05	mg/kg-day	0.019
				Chromium	132	µg/L	2.4E-06	mg/kg-day	NA	---	NA	1.9E-05	mg/kg-day	2.0E-02	mg/kg-day	0.00097
				Cobalt	153	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	3.0E-04	mg/kg-day	NA
				Copper	8910	µg/L	8.2E-05	mg/kg-day	NA	---	NA	6.4E-04	mg/kg-day	4.0E-02	mg/kg-day	0.016
				Iron	16948	µg/L	NA	mg/kg-day	NA	---	NA	NA	mg/kg-day	7.0E-01	mg/kg-day	NA
				Manganese	1433	µg/L	1.3E-05	mg/kg-day	NA	---	NA	1.0E-04	mg/kg-day	9.6E-04	mg/kg-day	0.11
				Nickel	87	µg/L	1.6E-07	mg/kg-day	NA	---	NA	1.2E-06	mg/kg-day	8.0E-04	mg/kg-day	0.0016
				Vanadium	5.0	µg/L	4.6E-08	mg/kg-day	NA	---	NA	3.6E-07	mg/kg-day	5.2E-04	mg/kg-day	0.0007
			Dermal Total								3.6E-08					0.15
Deep Bedrock Groundwater Total											7.1E-06					12
										Total of Receptor Risks Across All Media	7.1E-06			Total of Receptor Hazards Across All Media		12

APPENDIX E

TABLE E-39
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - CHILD RESIDENTIAL GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Residential Groundwater	Ingestion	Copper	192	µg/L	1.1E-03	mg/kg-day	NA	---	NA	1.2E-02	mg/kg-day	4.0E-02	mg/kg-day	0.31
				Vanadium	1.7	µg/L	9.3E-06	mg/kg-day	NA	---	NA	1.1E-04	mg/kg-day	2.0E-02	mg/kg-day	0.0054
			Ingestion Total								---					0.31
			Dermal	Copper	192	µg/L	6.9E-06	mg/kg-day	NA	---	NA	8.1E-05	mg/kg-day	4.0E-02	mg/kg-day	0.0020
				Vanadium	1.7	µg/L	6.1E-08	mg/kg-day	NA	---	NA	7.2E-07	mg/kg-day	5.2E-04	mg/kg-day	0.0014
			Dermal Total								---					0.0034
Residential Groundwater Total											---					0.32
											Total of Receptor Risks Across All Media					---					Total of Receptor Hazards Across All Media	0.32

APPENDIX E

TABLE E-40
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADULT RESIDENTIAL GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Residential Groundwater	Ingestion	Copper	192	µg/L	1.8E-03	mg/kg-day	NA	---	NA	5.3E-03	mg/kg-day	4.0E-02	mg/kg-day	0.13
				Vanadium	1.7	µg/L	1.6E-05	mg/kg-day	NA	---	NA	4.7E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0023
			Ingestion Total								---					0.13
			Dermal	Copper	192	µg/L	9.4E-06	mg/kg-day	NA	---	NA	2.7E-05	mg/kg-day	4.0E-02	mg/kg-day	0.00069
				Vanadium	1.7	µg/L	8.3E-08	mg/kg-day	NA	---	NA	2.4E-07	mg/kg-day	5.2E-04	mg/kg-day	0.00047
			Dermal Total								---					0.0012
Residential Groundwater Total											---					0.13
											Total of Receptor Risks Across All Media	---		Total of Receptor Hazards Across All Media		0.13

APPENDIX E

TABLE E-41
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - CHILD RESIDENTIAL GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Residential Groundwater	Ingestion	Copper	192	µg/L	5.3E-04	mg/kg-day	NA	---	NA	6.1E-03	mg/kg-day	4.0E-02	mg/kg-day	0.15
				Vanadium	1.7	µg/L	4.7E-06	mg/kg-day	NA	---	NA	5.4E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0027
			Ingestion Total								---					0.16
			Dermal	Copper	192	µg/L	3.5E-06	mg/kg-day	NA	---	NA	4.1E-05	mg/kg-day	4.0E-02	mg/kg-day	0.0010
				Vanadium	1.7	µg/L	3.1E-08	mg/kg-day	NA	---	NA	3.6E-07	mg/kg-day	5.2E-04	mg/kg-day	0.00069
			Dermal Total								---					0.0017
Residential Groundwater Total											---					0.16
											Total of Receptor Risks Across All Media	---		Total of Receptor Hazards Across All Media		0.16

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TABLE E-42
RAGS PART D TABLE 7 - CALCULATION OF COPC CANCER RISKS AND NON-CANCER HAZARDS - ADULT RESIDENTIAL GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Exposure Route	Contaminant of Potential Concern	EPC		Cancer Risk Calculations					Non-Cancer Hazard Calculations				
					Value	Units	Intake/Exposure Concentration		CSF/Unit Risk		Cancer Risk	Intake/Exposure Concentration		RfD/RfC		Hazard Quotient
							Value	Units	Value	Units		Value	Units	Value	Units	
Groundwater	Groundwater	Residential Groundwater	Ingestion	Copper	192	µg/L	3.4E-04	mg/kg-day	NA	---	NA	2.6E-03	mg/kg-day	4.0E-02	mg/kg-day	0.066
				Vanadium	1.7	µg/L	3.0E-06	mg/kg-day	NA	---	NA	2.3E-05	mg/kg-day	2.0E-02	mg/kg-day	0.0012
			Ingestion Total								---					0.067
			Dermal	Copper	192	µg/L	1.8E-06	mg/kg-day	NA	---	NA	1.4E-05	mg/kg-day	4.0E-02	mg/kg-day	0.00034
				Vanadium	1.7	µg/L	1.6E-08	mg/kg-day	NA	---	NA	1.2E-07	mg/kg-day	5.2E-04	mg/kg-day	0.00023
			Dermal Total								---					0.00058
Residential Groundwater Total											---					0.067
											Total of Receptor Risks Across All Media					---					Total of Receptor Hazards Across All Media	0.067

APPENDIX E

TABLE E-43
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADOLESCENT RECREATIONAL VISITOR
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Recreational Visitor
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Ely Copper Mine Surface Soil	Aluminum	---	---	---	---	Nervous system	0.0059	0.026	---	0.032
			Antimony	---	---	---	---	Blood	0.0017	---	---	0.0017
			Arsenic	2.6E-07	1.6E-08	9.1E-08	3.6E-07	Skin	0.0050	0.0022	0.0018	0.0089
			Cadmium	---	2.4E-09	---	2.4E-09	Kidney	0.00054	0.0012	0.00026	0.0020
			Chromium	---	---	---	---	None observed	0.000016	---	---	0.000016
			Cobalt	---	3.0E-07	---	3.0E-07	Thyroid	0.045	0.049	---	0.094
			Copper	---	---	---	---	Gastrointestinal	0.015	---	---	0.015
			Iron	---	---	---	---	Gastrointestinal	0.052	---	---	0.052
			Manganese	---	---	---	---	Nervous system	0.0035	0.11	---	0.11
			Mercury	---	---	---	---	Nervous system	0.00027	0.0000059	---	0.00028
			Molybdenum	---	---	---	---	Increased uric acid	0.00075	---	---	0.00075
			Selenium	---	---	---	---	Selenosis	0.0023	0.000013	---	0.0023
			Vanadium	---	---	---	---	None reported	0.0015	---	---	0.0015
		Exposure Point Total		2.6E-07	3.2E-07	9.1E-08	6.7E-07		0.13	0.19	0.0020	0.32
	Surface Soil						6.7E-07					0.32
Soil Total							6.7E-07					0.32

Total Risk Across All Media	6.7E-07
Total Hazard Across All Media	0.32
Total Nervous System HI Across All Media	0.14
Total Skin HI Across All Media	0.0089
Total Thyroid HI Across All Media	0.094
Total Kidney HI Across All Media	0.0020
Total Gastrointestinal HI Across All Media	0.067
Total Blood HI Across All Media	0.0017
Total Urinary Tract HI Across All Media	0.00075
Total Selenosis HI Across All Media	0.0023

APPENDIX E

TABLE E-44
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT RECREATIONAL VISITOR
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Recreational Visitor
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Ely Copper Mine Surface Soil	Aluminum	---	---	---	---	Nervous system	0.0044	0.026	---	0.030
			Antimony	---	---	---	---	Blood	0.0013	---	---	0.0013
			Arsenic	5.7E-07	4.8E-08	2.4E-07	8.5E-07	Skin	0.0037	0.0022	0.0015	0.0074
			Cadmium	---	7.3E-09	---	7.3E-09	Kidney	0.00040	0.0012	0.00022	0.0018
			Chromium	---	---	---	---	None observed	0.000012	---	---	0.000012
			Cobalt	---	9.1E-07	---	9.1E-07	Thyroid	0.033	0.049	---	0.082
			Copper	---	---	---	---	Gastrointestinal	0.011	---	---	0.011
			Iron	---	---	---	---	Gastrointestinal	0.039	---	---	0.039
			Manganese	---	---	---	---	Nervous system	0.0026	0.11	---	0.11
			Mercury	---	---	---	---	Nervous system	0.00020	0.0000059	---	0.00021
			Molybdenum	---	---	---	---	Increased uric acid	0.00056	---	---	0.00056
			Selenium	---	---	---	---	Selenosis	0.0017	0.000013	---	0.0017
			Vanadium	---	---	---	---	None reported	0.0011	---	---	0.0011
		Exposure Point Total		5.7E-07	9.6E-07	2.4E-07	1.8E-06		0.099	0.19	0.0018	0.29
	Surface Soil						1.8E-06					0.29
Soil Total							1.8E-06					0.29

Total Risk Across All Media	1.8E-06
Total Hazard Across All Media	0.29
Total Nervous System HI Across All Media	0.14
Total Skin HI Across All Media	0.0074
Total Thyroid HI Across All Media	0.082
Total Kidney HI Across All Media	0.0018
Total Gastrointestinal HI Across All Media	0.050
Total Blood HI Across All Media	0.0013
Total Urinary Tract HI Across All Media	0.00056
Total Selenosis HI Across All Media	0.0017

APPENDIX E

TABLE E-45
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADOLESCENT RECREATIONAL VISITOR
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Recreational Visitor
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Ely Copper Mine Surface Soil	Aluminum	---	---	---	---	Nervous system	0.0010	0.0046	---	0.0056
			Antimony	---	---	---	---	Blood	0.00029	---	---	0.00029
			Arsenic	4.3E-08	2.8E-09	9.3E-09	5.5E-08	Skin	0.00084	0.00039	0.00018	0.0014
			Cadmium	---	4.3E-10	---	4.3E-10	Kidney	0.000091	0.00021	0.000026	0.00033
			Chromium	---	---	---	---	None observed	0.0000027	---	---	0.0000027
			Cobalt	---	5.4E-08	---	5.4E-08	Thyroid	0.0076	0.0087	---	0.016
			Copper	---	---	---	---	Gastrointestinal	0.0025	---	---	0.0025
			Iron	---	---	---	---	Gastrointestinal	0.0088	---	---	0.0088
			Manganese	---	---	---	---	Nervous system	0.00059	0.019	---	0.020
			Mercury	---	---	---	---	Nervous system	0.000045	0.0000010	---	0.000047
			Molybdenum	---	---	---	---	Increased uric acid	0.00013	---	---	0.00013
			Selenium	---	---	---	---	Selenosis	0.00039	0.0000022	---	0.00039
			Vanadium	---	---	---	---	None reported	0.00026	---	---	0.00026
		Exposure Point Total		4.3E-08	5.7E-08	9.3E-09	1.1E-07		0.023	0.033	0.00021	0.056
	Surface Soil						1.1E-07					0.056
Soil Total							1.1E-07					0.056

Total Risk Across All Media	1.1E-07
Total Hazard Across All Media	0.056
Total Nervous System HI Across All Media	0.025
Total Skin HI Across All Media	0.0014
Total Thyroid HI Across All Media	0.016
Total Kidney HI Across All Media	0.00033
Total Gastrointestinal HI Across All Media	0.011
Total Blood HI Across All Media	0.00029
Total Urinary Tract HI Across All Media	0.00013
Total Selenosis HI Across All Media	0.00039

APPENDIX E

TABLE E-46
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT RECREATIONAL VISITOR
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Recreational Visitor
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Ely Copper Mine Surface Soil	Aluminum	---	---	---	---	Nervous system	0.00074	0.0044	---	0.0051
			Antimony	---	---	---	---	Blood	0.00022	---	---	0.00022
			Arsenic	9.6E-08	8.1E-09	2.6E-08	1.3E-07	Skin	0.00062	0.00037	0.00017	0.0012
			Cadmium	---	1.2E-09	---	1.2E-09	Kidney	0.000068	0.00020	0.000024	0.00029
			Chromium	---	---	---	---	None observed	0.0000020	---	---	0.0000020
			Cobalt	---	1.5E-07	---	1.5E-07	Thyroid	0.0056	0.0082	---	0.014
			Copper	---	---	---	---	Gastrointestinal	0.0019	---	---	0.0019
			Iron	---	---	---	---	Gastrointestinal	0.0065	---	---	0.0065
			Manganese	---	---	---	---	Nervous system	0.00044	0.018	---	0.019
			Mercury	---	---	---	---	Nervous system	0.000034	0.00000099	---	0.000035
			Molybdenum	---	---	---	---	Increased uric acid	0.000094	---	---	0.000094
			Selenium	---	---	---	---	Selenosis	0.00029	0.0000021	---	0.00029
			Vanadium	---	---	---	---	None reported	0.00019	---	---	0.00019
		Exposure Point Total		9.6E-08	1.6E-07	2.6E-08	2.8E-07		0.017	0.031	0.00019	0.048
	Surface Soil						2.8E-07					0.048
Soil Total							2.8E-07					0.048

Total Risk Across All Media	2.8E-07
Total Hazard Across All Media	0.048
Total Nervous System HI Across All Media	0.024
Total Skin HI Across All Media	0.0012
Total Thyroid HI Across All Media	0.014
Total Kidney HI Across All Media	0.00029
Total Gastrointestinal HI Across All Media	0.0084
Total Blood HI Across All Media	0.00022
Total Urinary Tract HI Across All Media	0.000094
Total Selenosis HI Across All Media	0.00029

APPENDIX E

TABLE E-47
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - WORKER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Construction Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Total Soil	Ely Copper Mine Soil	Aluminum	---	---	---	---	Nervous system	0.0092	0.24	---	0.25
			Antimony	---	---	---	---	Blood	0.0020	---	---	0.0020
			Arsenic	3.9E-08	1.5E-08	2.8E-09	5.6E-08	Skin	0.0060	0.016	0.00043	0.022
			Cadmium	---	4.9E-09	---	4.9E-09	Kidney	0.0014	0.019	0.00014	0.021
			Chromium	---	---	---	---	None observed	0.000025	---	---	0.000025
			Cobalt	---	6.6E-07	---	6.6E-07	Thyroid	0.13	0.86	---	0.99
			Copper	---	---	---	---	Gastrointestinal	0.037	---	---	0.037
			Iron	---	---	---	---	Gastrointestinal	0.075	---	---	0.075
			Manganese	---	---	---	---	Nervous system	0.0045	0.85	---	0.85
			Mercury	---	---	---	---	Nervous system	0.00032	0.000043	---	0.00037
			Molybdenum	---	---	---	---	Increased uric acid	0.00099	---	---	0.00099
			Nickel	---	1.0E-08	---	1.0E-08	Body weight	0.0011	0.031	---	0.032
			Selenium	---	---	---	---	Selenosis	0.0034	0.00011	---	0.0035
			Vanadium	---	---	---	---	None reported	0.0022	---	---	0.0022
			Zinc	---	---	---	---	Blood	0.00068	---	---	0.00068
		Exposure Point Total		3.9E-08	6.9E-07	2.8E-09	7.4E-07		0.27	2.0	0.00057	2.3
	Surface Soil						7.4E-07					2.3
Soil Total							7.4E-07					2.3

Total Risk Across All Media	7.4E-07
Total Hazard Across All Media	2.3
Total Nervous System HI Across All Media	1.1
Total Skin HI Across All Media	0.022
Total Thyroid HI Across All Media	0.99
Total Kidney HI Across All Media	0.021
Total Gastrointestinal HI Across All Media	0.11
Total Blood HI Across All Media	0.0027
Total Urinary Tract HI Across All Media	0.00099
Total Body Weight HI Across All Media	0.032
Total Selenosis HI Across All Media	0.0035

APPENDIX E

TABLE E-48
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - WORKER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Construction Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Total Soil	Ely Copper Mine Soil	Aluminum	---	---	---	---	Nervous system	0.0014	0.047	---	0.049
			Antimony	---	---	---	---	Blood	0.00031	---	---	0.00031
			Arsenic	5.8E-09	2.9E-09	8.1E-10	9.5E-09	Skin	0.00091	0.0031	0.00013	0.0041
			Cadmium	---	9.6E-10	---	9.6E-10	Kidney	0.00022	0.0037	0.000040	0.0040
			Chromium	---	---	---	---	None observed	0.0000038	---	---	0.0000038
			Cobalt	---	1.3E-07	---	1.3E-07	Thyroid	0.020	0.17	---	0.19
			Copper	---	---	---	---	Gastrointestinal	0.0055	---	---	0.0055
			Iron	---	---	---	---	Gastrointestinal	0.011	---	---	0.011
			Manganese	---	---	---	---	Nervous system	0.00069	0.16	---	0.17
			Mercury	---	---	---	---	Nervous system	0.000049	0.0000084	---	0.000057
			Molybdenum	---	---	---	---	Increased uric acid	0.00015	---	---	0.00015
			Nickel	---	2.0E-09	---	2.0E-09	Body weight	0.00016	0.0061	---	0.0062
			Selenium	---	---	---	---	Selenosis	0.00051	0.000022	---	0.00053
			Vanadium	---	---	---	---	None reported	0.00033	---	---	0.00033
			Zinc	---	---	---	---	Blood	0.00010	---	---	0.00010
		Exposure Point Total		5.8E-09	1.4E-07	8.1E-10	1.4E-07		0.041	0.39	0.00017	0.43
	Surface Soil						1.4E-07					0.43
Soil Total							1.4E-07					0.43

Total Risk Across All Media	1.4E-07

Total Hazard Across All Media	0.43
Total Nervous System HI Across All Media	0.21
Total Skin HI Across All Media	0.0041
Total Thyroid HI Across All Media	0.19
Total Kidney HI Across All Media	0.0040
Total Gastrointestinal HI Across All Media	0.017
Total Blood HI Across All Media	0.00041
Total Urinary Tract HI Across All Media	0.00015
Total Body Weight HI Across All Media	0.0062
Total Selenosis HI Across All Media	0.00053

APPENDIX E

TABLE E-49
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CURRENT CHILD RESIDENT
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Ely Copper Mine Surface Soil	Aluminum	---	---	---	---	Nervous system	0.14	0.00033	---	0.14
			Antimony	---	---	---	---	Blood	0.041	---	---	0.041
			Arsenic	4.5E-06	1.5E-10	3.8E-07	4.9E-06	Skin	0.12	0.000028	0.0098	0.13
			Cadmium	---	2.3E-11	---	2.3E-11	Kidney	0.013	0.000015	0.0014	0.014
			Chromium	---	---	---	---	None observed	0.00037	---	---	0.00037
			Cobalt	---	2.9E-09	---	2.9E-09	Thyroid	1.0	0.00063	---	1.0
			Copper	---	---	---	---	Gastrointestinal	0.35	---	---	0.35
			Iron	---	---	---	---	Gastrointestinal	1.2	---	---	1.2
			Manganese	---	---	---	---	Nervous system	0.082	0.0014	---	0.083
			Mercury	---	---	---	---	Nervous system	0.0063	0.000000076	---	0.0063
			Molybdenum	---	---	---	---	Increased uric acid	0.018	---	---	0.018
			Selenium	---	---	---	---	Selenosis	0.054	0.00000016	---	0.054
			Vanadium	---	---	---	---	None reported	0.036	---	---	0.036
		Exposure Point Total		4.5E-06	3.1E-09	3.8E-07	4.9E-06		3.1	0.0024	0.011	3.1
	Surface Soil						4.9E-06					3.1
Soil Total							4.9E-06					3.1

Total Risk Across All Media	4.9E-06
Total Hazard Across All Media	3.1
Total Nervous System HI Across All Media	0.23
Total Skin HI Across All Media	0.13
Total Thyroid HI Across All Media	1.0
Total Kidney HI Across All Media	0.014
Total Gastrointestinal HI Across All Media	1.6
Total Blood HI Across All Media	0.041
Total Urinary Tract HI Across All Media	0.018
Total Selenosis HI Across All Media	0.054

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TABLE E-50
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CURRENT ADULT RESIDENT
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Ely Copper Mine Surface Soil	Aluminum	---	---	---	---	Nervous system	0.015	0.00033	---	0.015
			Antimony	---	---	---	---	Blood	0.0044	---	---	0.0044
			Arsenic	1.9E-06	6.2E-10	2.3E-07	2.2E-06	Skin	0.012	0.000028	0.0015	0.014
			Cadmium	---	9.3E-11	---	9.3E-11	Kidney	0.0014	0.000015	0.00022	0.0016
			Chromium	---	---	---	---	None observed	0.000040	---	---	0.000040
			Cobalt	---	1.2E-08	---	1.2E-08	Thyroid	0.11	0.00063	---	0.11
			Copper	---	---	---	---	Gastrointestinal	0.037	---	---	0.037
			Iron	---	---	---	---	Gastrointestinal	0.13	---	---	0.13
			Manganese	---	---	---	---	Nervous system	0.0088	0.0014	---	0.010
			Mercury	---	---	---	---	Nervous system	0.00068	0.000000076	---	0.00068
			Molybdenum	---	---	---	---	Increased uric acid	0.0019	---	---	0.0019
			Selenium	---	---	---	---	Selenosis	0.0058	0.00000016	---	0.0058
			Vanadium	---	---	---	---	None reported	0.0039	---	---	0.0039
		Exposure Point Total		1.9E-06	1.2E-08	2.3E-07	2.2E-06		0.33	0.0024	0.0017	0.34
	Surface Soil						2.2E-06					0.34
Soil Total							2.2E-06					0.34

Total Risk Across All Media	2.2E-06
Total Hazard Across All Media	0.34
Total Nervous System HI Across All Media	0.026
Total Skin HI Across All Media	0.014
Total Thyroid HI Across All Media	0.11
Total Kidney HI Across All Media	0.0016
Total Gastrointestinal HI Across All Media	0.17
Total Blood HI Across All Media	0.0044
Total Urinary Tract HI Across All Media	0.0019
Total Selenosis HI Across All Media	0.0058

APPENDIX E

TABLE E-51
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CURRENT CHILD RESIDENT
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Ely Copper Mine Surface Soil	Aluminum	---	---	---	---	Nervous system	0.069	0.00022	---	0.069
			Antimony	---	---	---	---	Blood	0.020	---	---	0.020
			Arsenic	2.2E-06	1.0E-10	7.5E-08	2.3E-06	Skin	0.058	0.000019	0.0020	0.060
			Cadmium	---	1.6E-11	---	1.6E-11	Kidney	0.0063	0.000010	0.00028	0.0066
			Chromium	---	---	---	---	None observed	0.00019	---	---	0.00019
			Cobalt	---	1.9E-09	---	1.9E-09	Thyroid	0.52	0.00042	---	0.52
			Copper	---	---	---	---	Gastrointestinal	0.17	---	---	0.17
			Iron	---	---	---	---	Gastrointestinal	0.61	---	---	0.61
			Manganese	---	---	---	---	Nervous system	0.041	0.00092	---	0.042
			Mercury	---	---	---	---	Nervous system	0.0032	0.000000050	---	0.0032
			Molybdenum	---	---	---	---	Increased uric acid	0.0088	---	---	0.0088
			Selenium	---	---	---	---	Selenosis	0.027	0.00000011	---	0.027
			Vanadium	---	---	---	---	None reported	0.018	---	---	0.018
		Exposure Point Total		2.2E-06	2.1E-09	7.5E-08	2.3E-06		1.6	0.0016	0.0022	1.6
	Surface Soil						2.3E-06					1.6
Soil Total							2.3E-06					1.6

Total Risk Across All Media	2.3E-06

Total Hazard Across All Media	1.6
Total Nervous System HI Across All Media	0.11
Total Skin HI Across All Media	0.060
Total Thyroid HI Across All Media	0.52
Total Kidney HI Across All Media	0.0066
Total Gastrointestinal HI Across All Media	0.78
Total Blood HI Across All Media	0.020
Total Urinary Tract HI Across All Media	0.0088
Total Selenosis HI Across All Media	0.027

APPENDIX E

TABLE E-52
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CURRENT ADULT RESIDENT
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Ely Copper Mine Surface Soil	Aluminum	---	---	---	---	Nervous system	0.0074	0.00022	---	0.0076
			Antimony	---	---	---	---	Blood	0.0022	---	---	0.0022
			Arsenic	3.6E-07	1.5E-10	1.2E-08	3.7E-07	Skin	0.0062	0.000019	0.00021	0.0065
			Cadmium	---	2.3E-11	---	2.3E-11	Kidney	0.00068	0.000010	0.000031	0.00072
			Chromium	---	---	---	---	None observed	0.000020	---	---	0.000020
			Cobalt	---	2.9E-09	---	2.9E-09	Thyroid	0.056	0.00042	---	0.057
			Copper	---	---	---	---	Gastrointestinal	0.019	---	---	0.019
			Iron	---	---	---	---	Gastrointestinal	0.065	---	---	0.065
			Manganese	---	---	---	---	Nervous system	0.0044	0.00092	---	0.0053
			Mercury	---	---	---	---	Nervous system	0.00034	0.000000050	---	0.00034
			Molybdenum	---	---	---	---	Increased uric acid	0.00094	---	---	0.00094
			Selenium	---	---	---	---	Selenosis	0.0029	0.00000011	---	0.0029
			Vanadium	---	---	---	---	None reported	0.0019	---	---	0.0019
		Exposure Point Total		3.6E-07	3.1E-09	1.2E-08	3.8E-07		0.17	0.0016	0.00024	0.17
	Surface Soil						3.8E-07					0.17
Soil Total							3.8E-07					0.17

Total Risk Across All Media	3.8E-07
Total Hazard Across All Media	0.17
Total Nervous System HI Across All Media	0.013
Total Skin HI Across All Media	0.0065
Total Thyroid HI Across All Media	0.057
Total Kidney HI Across All Media	0.00072
Total Gastrointestinal HI Across All Media	0.084
Total Blood HI Across All Media	0.0022
Total Urinary Tract HI Across All Media	0.00094
Total Selenosis HI Across All Media	0.0029

APPENDIX E

TABLE E-53
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - FUTURE CHILD RESIDENT
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Total Soil	Ely Copper Mine Soil	Aluminum	---	---	---	---	Nervous system	0.15	0.00036	---	0.15
			Antimony	---	---	---	---	Blood	0.034	---	---	0.034
			Arsenic	3.8E-06	1.3E-10	3.2E-07	4.1E-06	Skin	0.099	0.000024	0.0083	0.11
			Cadmium	---	4.4E-11	---	4.4E-11	Kidney	0.024	0.000029	0.0027	0.026
			Chromium	---	---	---	---	None observed	0.00041	---	---	0.00041
			Cobalt	---	5.9E-09	---	5.9E-09	Thyroid	2.1	0.00128	---	2.1
			Copper	---	---	---	---	Gastrointestinal	0.60	---	---	0.60
			Iron	---	---	---	---	Gastrointestinal	1.2	---	---	1.2
			Manganese	---	---	---	---	Nervous system	0.075	0.0013	---	0.076
			Mercury	---	---	---	---	Nervous system	0.0053	0.000000064	---	0.0053
			Molybdenum	---	---	---	---	Increased uric acid	0.016	---	---	0.016
			Nickel	---	9.3E-11	---	9.3E-11	Body weight	0.017	0.000047	---	0.018
			Selenium	---	---	---	---	Selenosis	0.056	0.00000017	---	0.056
			Vanadium	---	---	---	---	None reported	0.036	---	---	0.036
			Zinc	---	---	---	---	Blood	0.011	---	---	0.011
		Exposure Point Total		3.8E-06	6.2E-09	3.2E-07	4.1E-06		4.5	0.0030	0.011	4.5
	Surface Soil						4.1E-06					4.5
Soil Total							4.1E-06					4.5

Total Risk Across All Media	4.1E-06
Total Hazard Across All Media	4.5
Total Nervous System HI Across All Media	0.23
Total Skin HI Across All Media	0.11
Total Thyroid HI Across All Media	2.1
Total Kidney HI Across All Media	0.026
Total Gastrointestinal HI Across All Media	1.8
Total Blood HI Across All Media	0.034
Total Urinary Tract HI Across All Media	0.016
Total Body Weight HI Across All Media	0.018
Total Selenosis HI Across All Media	0.056

APPENDIX E

TABLE E-54
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - FUTURE ADULT RESIDENT
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Total Soil	Ely Copper Mine Soil	Aluminum	---	---	---	---	Nervous system	0.016	0.00036	---	0.017
			Antimony	---	---	---	---	Blood	0.0036	---	---	0.0036
			Arsenic	1.6E-06	5.3E-10	2.0E-07	1.8E-06	Skin	0.011	0.000024	0.0013	0.012
			Cadmium	---	1.8E-10	---	1.8E-10	Kidney	0.0025	0.000029	0.00041	0.0030
			Chromium	---	---	---	---	None observed	0.000044	---	---	0.000044
			Cobalt	---	2.4E-08	---	2.4E-08	Thyroid	0.23	0.0013	---	0.23
			Copper	---	---	---	---	Gastrointestinal	0.065	---	---	0.065
			Iron	---	---	---	---	Gastrointestinal	0.13	---	---	0.13
			Manganese	---	---	---	---	Nervous system	0.0080	0.0013	---	0.0093
			Mercury	---	---	---	---	Nervous system	0.00057	0.000000064	---	0.00057
			Molybdenum	---	---	---	---	Increased uric acid	0.0018	---	---	0.0018
			Nickel	---	3.7E-10	---	3.7E-10	Body weight	0.0019	0.000047	---	0.0019
			Selenium	---	---	---	---	Selenosis	0.0060	0.00000017	---	0.0060
			Vanadium	---	---	---	---	None reported	0.0039	---	---	0.0039
			Zinc	---	---	---	---	Blood	0.0012	---	---	0.0012
		Exposure Point Total		1.6E-06	2.5E-08	2.0E-07	1.9E-06		0.48	0.0030	0.0017	0.49
	Surface Soil						1.9E-06					0.49
Soil Total							1.9E-06					0.49

Total Risk Across All Media	1.9E-06
Total Hazard Across All Media	0.49
Total Nervous System HI Across All Media	0.026
Total Skin HI Across All Media	0.012
Total Thyroid HI Across All Media	0.23
Total Kidney HI Across All Media	0.0030
Total Gastrointestinal HI Across All Media	0.20
Total Blood HI Across All Media	0.0036
Total Urinary Tract HI Across All Media	0.0018
Total Body Weight HI Across All Media	0.0019
Total Selenosis HI Across All Media	0.0060

APPENDIX E

TABLE E-55
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - FUTURE CHILD RESIDENT
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Total Soil	Ely Copper Mine Soil	Aluminum	---	---	---	---	Nervous system	0.076	0.00024	---	0.076
			Antimony	---	---	---	---	Blood	0.017	---	---	0.017
			Arsenic	1.9E-06	8.8E-11	6.4E-08	2.0E-06	Skin	0.050	0.000016	0.0017	0.051
			Cadmium	---	2.9E-11	---	2.9E-11	Kidney	0.012	0.000019	0.00053	0.012
			Chromium	---	---	---	---	None observed	0.00021	---	---	0.00021
			Cobalt	---	4.0E-09	---	4.0E-09	Thyroid	1.1	0.00086	---	1.1
			Copper	---	---	---	---	Gastrointestinal	0.30	---	---	0.30
			Iron	---	---	---	---	Gastrointestinal	0.62	---	---	0.62
			Manganese	---	---	---	---	Nervous system	0.038	0.00084	---	0.038
			Mercury	---	---	---	---	Nervous system	0.0027	0.000000043	---	0.0027
			Molybdenum	---	---	---	---	Increased uric acid	0.0082	---	---	0.0082
			Nickel	---	6.2E-11	---	6.2E-11	Body weight	0.0087	0.000031	---	0.0088
			Selenium	---	---	---	---	Selenosis	0.028	0.000000111	---	0.028
			Vanadium	---	---	---	---	None reported	0.018	---	---	0.018
			Zinc	---	---	---	---	Blood	0.0056	---	---	0.0056
		Exposure Point Total		1.9E-06	4.1E-09	6.4E-08	2.0E-06		2.3	0.0020	0.0022	2.3
	Surface Soil						2.0E-06					2.3
Soil Total							2.0E-06					2.3

Total Risk Across All Media	2.0E-06

Total Hazard Across All Media	2.3
Total Nervous System HI Across All Media	0.12
Total Skin HI Across All Media	0.051
Total Thyroid HI Across All Media	1.1
Total Kidney HI Across All Media	0.012
Total Gastrointestinal HI Across All Media	0.92
Total Blood HI Across All Media	0.017
Total Urinary Tract HI Across All Media	0.0082
Total Body Weight HI Across All Media	0.0088
Total Selenosis HI Across All Media	0.028

APPENDIX E

TABLE E-56
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - FUTURE ADULT RESIDENT
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Total Soil	Ely Copper Mine Soil	Aluminum	---	---	---	---	Nervous system	0.0081	0.00024	---	0.0083
			Antimony	---	---	---	---	Blood	0.00180	---	---	0.00180
			Arsenic	3.1E-07	1.3E-10	1.0E-08	3.2E-07	Skin	0.0053	0.000016	0.00018	0.0055
			Cadmium	---	4.4E-11	---	4.4E-11	Kidney	0.0013	0.000019	0.000058	0.0014
			Chromium	---	---	---	---	None observed	0.000022	---	---	0.000022
			Cobalt	---	5.9E-09	---	5.9E-09	Thyroid	0.11	0.00086	---	0.12
			Copper	---	---	---	---	Gastrointestinal	0.032	---	---	0.032
			Iron	---	---	---	---	Gastrointestinal	0.066	---	---	0.066
			Manganese	---	---	---	---	Nervous system	0.0040	0.00084	---	0.0049
			Mercury	---	---	---	---	Nervous system	0.00029	0.000000043	---	0.00029
			Molybdenum	---	---	---	---	Increased uric acid	0.00088	---	---	0.00088
			Nickel	---	9.3E-11	---	9.3E-11	Body weight	0.00094	0.000031	---	0.00097
			Selenium	---	---	---	---	Selenosis	0.0030	0.00000011	---	0.0030
			Vanadium	---	---	---	---	None reported	0.0019	---	---	0.0019
			Zinc	---	---	---	---	Blood	0.00060	---	---	0.00060
		Exposure Point Total		3.1E-07	6.2E-09	1.0E-08	3.2E-07		0.24	0.0020	0.00024	0.24
	Surface Soil						3.2E-07					0.24
Soil Total							3.2E-07					0.24

Total Risk Across All Media	3.2E-07

Total Hazard Across All Media	0.24
Total Nervous System HI Across All Media	0.013
Total Skin HI Across All Media	0.0055
Total Thyroid HI Across All Media	0.12
Total Kidney HI Across All Media	0.0014
Total Gastrointestinal HI Across All Media	0.098
Total Blood HI Across All Media	0.0018
Total Urinary Tract HI Across All Media	0.00088
Total Body Weight HI Across All Media	0.00097
Total Selenosis HI Across All Media	0.0030

APPENDIX E

TABLE E-57
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADOLESCENT SWIMMER/WADER ELY BROOK
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Ely Brook Sediment	Sediment	Ely Brook Sediment	Aluminum	---	---	---	Nervous system	0.0040	---	0.0040
			Arsenic	7.3E-08	4.1E-08	1.1E-07	Skin	0.0014	0.00080	0.0022
			Chromium	---	---	---	None observed	0.0000042	---	0.0000042
			Cobalt	---	---	---	Thyroid	0.017	---	0.017
			Copper	---	---	---	Gastrointestinal	0.0090	---	0.0090
			Iron	---	---	---	Gastrointestinal	0.012	---	0.012
			Lithium	---	---	---	No information	0.0020	---	0.0020
			Manganese	---	---	---	Nervous system	0.0016	---	0.0016
			Selenium	---	---	---	Selenosis	0.00081	---	0.00081
			Vanadium	---	---	---	None reported	0.00043	---	0.00043
		Exposure Point Total		7.3E-08	4.1E-08	1.1E-07		0.049	0.00080	0.050
	Surface Soil					1.1E-07				0.050
Sediment Total						1.1E-07				0.050
Ely Brook Surface Water	Surface Water	Ely Brook Surface Water	Aluminum	---	---	---	Nervous system	0.0015	---	0.0015
			Arsenic	4.9E-09	1.5E-09	6.3E-09	Skin	0.000095	0.000028	0.00012
			Cadmium	---	---	---	Kidney	0.00088	0.010	0.011
			Chromium	---	---	---	None observed	0.00000080	0.000037	0.000037
			Cobalt	---	---	---	Thyroid	0.059	---	0.059
			Copper	---	---	---	Gastrointestinal	0.013	0.0038	0.017
			Iron	---	---	---	Gastrointestinal	0.0011	---	0.0011
			Lithium	---	---	---	No information	0.00037	---	0.00037
			Manganese	---	---	---	Nervous system	0.0038	0.02794	0.0317
			Mercury	---	---	---	Nervous system	0.000038	0.00016	0.00020
			Nickel	---	---	---	Body weight	0.00033	0.00049	0.00083
			Vanadium	---	---	---	None reported	0.0000090	0.00010	0.00011
			Zinc	---	---	---	Blood	0.00041	0.000074	0.00049
		Exposure Point Total		4.9E-09	1.5E-09	6.3E-09		0.080	0.043	0.12
	Surface Soil					6.3E-09				0.12
Surface Water Total						6.3E-09				0.12

Total Risk Across All Media	1.2E-07
Total Hazard Across All Media	0.17
Total Nervous System HI Across All Media	0.0390
Total Skin HI Across All Media	0.0023
Total Thyroid HI Across All Media	0.077
Total Kidney HI Across All Media	0.011
Total Gastrointestinal HI Across All Media	0.039
Total Blood HI Across All Media	0.00049
Total Body Weight HI Across All Media	0.00083
Total Selenosis HI Across All Media	0.00081

APPENDIX E

TABLE E-58
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT SWIMMER/WADER ELY BROOK
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Ely Brook Sediment	Sediment	Ely Brook Sediment	Aluminum	---	---	---	Nervous system	0.0030	---	0.0030
			Arsenic	1.6E-07	1.1E-07	2.7E-07	Skin	0.0011	0.00070	0.0017
			Chromium	---	---	---	None observed	0.0000031	---	0.0000031
			Cobalt	---	---	---	Thyroid	0.013	---	0.013
			Copper	---	---	---	Gastrointestinal	0.0067	---	0.0067
			Iron	---	---	---	Gastrointestinal	0.0091	---	0.0091
			Lithium	---	---	---	No information	0.0015	---	0.0015
			Manganese	---	---	---	Nervous system	0.0012	---	0.0012
			Selenium	---	---	---	Selenosis	0.00060	---	0.00060
			Vanadium	---	---	---	None reported	0.00032	---	0.00032
		Exposure Point Total		1.6E-07	1.1E-07	2.7E-07		0.036	0.00070	0.037
	Surface Soil					2.7E-07				0.037
Sediment Total						2.7E-07				0.037
Ely Brook Surface Water	Surface Water	Ely Brook Surface Water	Aluminum	---	---	---	Nervous system	0.0011	---	0.0011
			Arsenic	1.1E-08	3.9E-09	1.5E-08	Skin	0.000071	0.000025	0.000096
			Cadmium	---	---	---	Kidney	0.00065	0.0094	0.010
			Chromium	---	---	---	None observed	0.00000059	0.000033	0.000033
			Cobalt	---	---	---	Thyroid	0.044	---	0.044
			Copper	---	---	---	Gastrointestinal	0.0095	0.0034	0.013
			Iron	---	---	---	Gastrointestinal	0.00078	---	0.00078
			Lithium	---	---	---	No information	0.00027	---	0.00027
			Manganese	---	---	---	Nervous system	0.0028	0.02508	0.0279
			Mercury	---	---	---	Nervous system	0.000028	0.00015	0.00017
			Nickel	---	---	---	Body weight	0.00025	0.00044	0.00069
			Vanadium	---	---	---	None reported	0.0000067	0.000092	0.000099
			Zinc	---	---	---	Blood	0.00031	0.000067	0.00037
		Exposure Point Total		1.1E-08	3.9E-09	1.5E-08		0.060	0.039	0.098
	Surface Soil					1.5E-08				0.098
Surface Water Total						1.5E-08				0.098

Total Risk Across All Media	2.8E-07
Total Hazard Across All Media	0.14
Total Nervous System HI Across All Media	0.0333
Total Skin HI Across All Media	0.0018
Total Thyroid HI Across All Media	0.057
Total Kidney HI Across All Media	0.010
Total Gastrointestinal HI Across All Media	0.029
Total Blood HI Across All Media	0.00037
Total Body Weight HI Across All Media	0.00069
Total Selenosis HI Across All Media	0.00060

APPENDIX E

TABLE E-59
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADOLESCENT SWIMMER/WADER ELY BROOK
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Ely Brook Sediment	Sediment	Ely Brook Sediment	Aluminum	---	---	---	Nervous system	0.0010	---	0.0010
			Arsenic	1.8E-08	2.8E-09	2.1E-08	Skin	0.00035	0.000053	0.00041
			Chromium	---	---	---	None observed	0.0000010	---	0.0000010
			Cobalt	---	---	---	Thyroid	0.0044	---	0.0044
			Copper	---	---	---	Gastrointestinal	0.0023	---	0.0023
			Iron	---	---	---	Gastrointestinal	0.0031	---	0.0031
			Lithium	---	---	---	No information	0.00049	---	0.00049
			Manganese	---	---	---	Nervous system	0.00040	---	0.00040
			Selenium	---	---	---	Selenosis	0.00020	---	0.00020
			Vanadium	---	---	---	None reported	0.00011	---	0.00011
		Exposure Point Total		1.8E-08	2.8E-09	2.1E-08		0.012	0.000053	0.012
	Surface Soil					2.1E-08				0.012
Sediment Total						2.1E-08				0.012
Ely Brook Surface Water	Surface Water	Ely Brook Surface Water	Aluminum	---	---	---	Nervous system	0.00037	---	0.00037
			Arsenic	1.2E-09	3.6E-10	1.6E-09	Skin	0.000024	0.0000071	0.000031
			Cadmium	---	---	---	Kidney	0.00022	0.0026	0.0028
			Chromium	---	---	---	None observed	0.00000020	0.0000092	0.0000094
			Cobalt	---	---	---	Thyroid	0.015	---	0.015
			Copper	---	---	---	Gastrointestinal	0.0032	0.00095	0.0041
			Iron	---	---	---	Gastrointestinal	0.00026	---	0.00026
			Lithium	---	---	---	No information	0.000091	---	0.000091
			Manganese	---	---	---	Nervous system	0.00094	0.006986	0.00792
			Mercury	---	---	---	Nervous system	0.0000096	0.000041	0.000050
			Nickel	---	---	---	Body weight	0.000083	0.00012	0.00021
			Vanadium	---	---	---	None reported	0.0000022	0.000026	0.000028
			Zinc	---	---	---	Blood	0.00010	0.000019	0.00012
		Exposure Point Total		1.2E-09	3.6E-10	1.6E-09		0.020	0.0108	0.031
	Surface Soil					1.6E-09				0.031
Surface Water Total						1.6E-09				0.031

Total Risk Across All Media	2.3E-08
Total Hazard Across All Media	0.043
Total Nervous System HI Across All Media	0.0098
Total Skin HI Across All Media	0.00044
Total Thyroid HI Across All Media	0.019
Total Kidney HI Across All Media	0.0028
Total Gastrointestinal HI Across All Media	0.0097
Total Blood HI Across All Media	0.00012
Total Body Weight HI Across All Media	0.00021
Total Selenosis HI Across All Media	0.00020

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TABLE E-60
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT SWIMMER/WADER ELY BROOK
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Ely Brook Sediment	Sediment	Ely Brook Sediment	Aluminum	---	---	---	Nervous system	0.00074	---	0.00074
			Arsenic	4.1E-08	7.7E-09	4.8E-08	Skin	0.00026	0.000050	0.00031
			Chromium	---	---	---	None observed	0.00000077	---	0.00000077
			Cobalt	---	---	---	Thyroid	0.0032	---	0.0032
			Copper	---	---	---	Gastrointestinal	0.0017	---	0.0017
			Iron	---	---	---	Gastrointestinal	0.0023	---	0.0023
			Lithium	---	---	---	No information	0.00037	---	0.00037
			Manganese	---	---	---	Nervous system	0.00030	---	0.00030
			Selenium	---	---	---	Selenosis	0.00015	---	0.00015
			Vanadium	---	---	---	None reported	0.000080	---	0.000080
		Exposure Point Total		4.1E-08	7.7E-09	4.8E-08		0.0091	0.000050	0.0091
	Surface Soil					4.8E-08				0.0091
Sediment Total						4.8E-08				0.0091
Ely Brook Surface Water	Surface Water	Ely Brook Surface Water	Aluminum	---	---	---	Nervous system	0.00028	---	0.00028
			Arsenic	2.7E-09	9.8E-10	3.7E-09	Skin	0.000018	0.0000064	0.000024
			Cadmium	---	---	---	Kidney	0.00016	0.0023	0.0025
			Chromium	---	---	---	None observed	0.00000015	0.0000082	0.0000084
			Cobalt	---	---	---	Thyroid	0.011	---	0.011
			Copper	---	---	---	Gastrointestinal	0.0024	0.00085	0.0032
			Iron	---	---	---	Gastrointestinal	0.00020	---	0.00020
			Lithium	---	---	---	No information	0.000068	---	0.000068
			Manganese	---	---	---	Nervous system	0.00070	0.006269	0.00697
			Mercury	---	---	---	Nervous system	0.0000071	0.000037	0.000044
			Nickel	---	---	---	Body weight	0.000062	0.00011	0.00017
			Vanadium	---	---	---	None reported	0.0000017	0.000023	0.000025
			Zinc	---	---	---	Blood	0.000077	0.000017	0.000094
		Exposure Point Total		2.7E-09	9.8E-10	3.7E-09		0.015	0.0097	0.025
	Surface Soil					3.7E-09				0.025
Surface Water Total						3.7E-09				0.025

Total Risk Across All Media	5.2E-08
Total Hazard Across All Media	0.034
Total Nervous System HI Across All Media	0.0083
Total Skin HI Across All Media	0.00034
Total Thyroid HI Across All Media	0.014
Total Kidney HI Across All Media	0.0025
Total Gastrointestinal HI Across All Media	0.0074
Total Blood HI Across All Media	0.000094
Total Body Weight HI Across All Media	0.00017
Total Selenosis HI Across All Media	0.00015

APPENDIX E

TABLE E-61
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADOLESCENT SWIMMER/WADER SCHOOLHOUSE BROOK
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Schoolhouse Brook Sediment	Sediment	Schoolhouse Brook Sediment	Aluminum	---	---	---	Nervous system	0.0050	---	0.0050
			Arsenic	2.8E-08	1.6E-08	4.4E-08	Skin	0.00055	0.00031	0.00086
			Chromium	---	---	---	None observed	0.0000023	---	0.0000023
			Cobalt	---	---	---	Thyroid	0.0041	---	0.0041
			Copper	---	---	---	Gastrointestinal	0.0025	---	0.0025
			Iron	---	---	---	Gastrointestinal	0.0053	---	0.0053
			Lithium	---	---	---	No information	0.0014	---	0.0014
			Manganese	---	---	---	Nervous system	0.0012	---	0.0012
			Vanadium	---	---	---	None reported	0.00026	---	0.00026
		Exposure Point Total		2.8E-08	1.6E-08	4.4E-08		0.020	0.00031	0.021
	Surface Soil					4.4E-08				0.021
Sediment Total						4.4E-08				0.021
Schoolhouse Brook Surface Water	Surface Water	Schoolhouse Brook Surface Water	Chromium	---	---	---	None observed	0.0000002	0.0000092	0.0000094
			Cobalt	---	---	---	Thyroid	0.0035	---	0.0035
			Copper	---	---	---	Gastrointestinal	0.0010	0.00029	0.0013
			Manganese	---	---	---	Nervous system	0.000273	0.002031	0.00230
			Mercury	---	---	---	Nervous system	0.000066	0.00028	0.00035
			Vanadium	---	---	---	None reported	0.0000027	0.000031	0.000034
		Exposure Point Total		0.0E+00	0.0E+00	0.0E+00		0.0048	0.0026	0.0074
	Surface Soil					0.0E+00				0.0074
Surface Water Total						0.0E+00				0.0074

Total Risk Across All Media	4.4E-08
Total Hazard Across All Media	0.028
Total Nervous System HI Across All Media	0.0088
Total Skin HI Across All Media	0.00086
Total Thyroid HI Across All Media	0.0076
Total Gastrointestinal HI Across All Media	0.0091

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TABLE E-62
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT SWIMMER/WADER SCHOOLHOUSE BROOK
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Schoolhouse Brook Sediment	Sediment	Schoolhouse Brook Sediment	Aluminum	---	---	---	Nervous system	0.0037	---	0.0037
			Arsenic	6.3E-08	4.1E-08	1.0E-07	Skin	0.00041	0.00027	0.00067
			Chromium	---	---	---	None observed	0.0000017	---	0.0000017
			Cobalt	---	---	---	Thyroid	0.0031	---	0.0031
			Copper	---	---	---	Gastrointestinal	0.0018	---	0.0018
			Iron	---	---	---	Gastrointestinal	0.0040	---	0.0040
			Lithium	---	---	---	No information	0.0010	---	0.0010
			Manganese	---	---	---	Nervous system	0.00091	---	0.00091
			Vanadium	---	---	---	None reported	0.00019	---	0.00019
		Exposure Point Total		6.3E-08	4.1E-08	1.0E-07		0.015	0.00027	0.015
	Surface Soil					1.0E-07				0.015
Sediment Total						1.0E-07				0.015
Schoolhouse Brook Surface Water	Surface Water	Schoolhouse Brook Surface Water	Chromium	---	---	---	None observed	0.00000015	0.0000082	0.0000084
			Cobalt	---	---	---	Thyroid	0.0026	---	0.0026
			Copper	---	---	---	Gastrointestinal	0.00073	0.00026	0.0010
			Manganese	---	---	---	Nervous system	0.000203	0.001823	0.002026
			Mercury	---	---	---	Nervous system	0.000049	0.00025	0.00030
			Vanadium	---	---	---	None reported	0.0000020	0.000028	0.000030
		Exposure Point Total		0.0E+00	0.0E+00	0.0E+00		0.0035	0.0024	0.0059
	Surface Soil					0.0E+00				0.0059
Surface Water Total						0.0E+00				0.0059

Total Risk Across All Media	1.0E-07
Total Hazard Across All Media	0.021
Total Nervous System HI Across All Media	0.0069
Total Skin HI Across All Media	0.00067
Total Thyroid HI Across All Media	0.0056
Total Gastrointestinal HI Across All Media	0.0068

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TABLE E-63
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADOLESCENT SWIMMER/WADER SCHOOLHOUSE BROOK
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Schoolhouse Brook Sediment	Sediment	Schoolhouse Brook Sediment	Aluminum	---	---	---	Nervous system	0.0012	---	0.0012
			Arsenic	7.0E-09	1.1E-09	8.1E-09	Skin	0.00014	0.000021	0.00016
			Chromium	---	---	---	None observed	0.00000056	---	0.00000056
			Cobalt	---	---	---	Thyroid	0.0010	---	0.0010
			Copper	---	---	---	Gastrointestinal	0.00062	---	0.00062
			Iron	---	---	---	Gastrointestinal	0.0013	---	0.0013
			Lithium	---	---	---	No information	0.00035	---	0.00035
			Manganese	---	---	---	Nervous system	0.00031	---	0.00031
			Vanadium	---	---	---	None reported	0.000065	---	0.000065
		Exposure Point Total		7.0E-09	1.1E-09	8.1E-09		0.0051	0.000021	0.0051
	Surface Soil					8.1E-09				0.0051
Sediment Total						8.1E-09				0.0051
Schoolhouse Brook Surface Water	Surface Water	Schoolhouse Brook Surface Water	Chromium	---	---	---	None observed	0.000000050	0.0000023	0.0000023
			Cobalt	---	---	---	Thyroid	0.00086	---	0.00086
			Copper	---	---	---	Gastrointestinal	0.00024	0.000073	0.00032
			Manganese	---	---	---	Nervous system	0.000068	0.0005079	0.000576
			Mercury	---	---	---	Nervous system	0.000016	0.000070	0.000086
			Vanadium	---	---	---	None reported	0.00000068	0.0000078	0.0000085
		Exposure Point Total		0.0E+00	0.0E+00	0.0E+00		0.0012	0.00066	0.0019
	Surface Soil					0.0E+00				0.0019
Surface Water Total						0.0E+00				0.0019

Total Risk Across All Media	8.1E-09
Total Hazard Across All Media	0.0070
Total Nervous System HI Across All Media	0.0022
Total Skin HI Across All Media	0.00016
Total Thyroid HI Across All Media	0.0019
Total Gastrointestinal HI Across All Media	0.0023

APPENDIX E

TABLE E-64
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT SWIMMER/WADER SCHOOLHOUSE BROOK
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Schoolhouse Brook Sediment	Sediment	Schoolhouse Brook Sediment	Aluminum	---	---	---	Nervous system	0.00092	---	0.00092
			Arsenic	1.6E-08	3.0E-09	1.9E-08	Skin	0.00010	0.000019	0.00012
			Chromium	---	---	---	None observed	0.00000042	---	0.00000042
			Cobalt	---	---	---	Thyroid	0.00077	---	0.00077
			Copper	---	---	---	Gastrointestinal	0.00046	---	0.00046
			Iron	---	---	---	Gastrointestinal	0.00099	---	0.00099
			Lithium	---	---	---	No information	0.00026	---	0.00026
			Manganese	---	---	---	Nervous system	0.00023	---	0.00023
			Vanadium	---	---	---	None reported	0.000048	---	0.000048
		Exposure Point Total		1.6E-08	3.0E-09	1.9E-08		0.0038	0.000019	0.0038
	Surface Soil					1.9E-08				0.0038
Sediment Total						1.9E-08				0.0038
Schoolhouse Brook Surface Water	Surface Water	Schoolhouse Brook Surface Water	Chromium	---	---	---	None observed	0.000000037	0.0000021	0.0000021
			Cobalt	---	---	---	Thyroid	0.00064	---	0.00064
			Copper	---	---	---	Gastrointestinal	0.00018	0.000065	0.00025
			Manganese	---	---	---	Nervous system	0.000051	0.0004558	0.000506
			Mercury	---	---	---	Nervous system	0.000012	0.000063	0.000075
			Vanadium	---	---	---	None reported	0.00000051	0.0000070	0.0000075
		Exposure Point Total		0.0E+00	0.0E+00	0.0E+00		0.00089	0.00059	0.0015
	Surface Soil					0.0E+00				0.0015
Surface Water Total						0.0E+00				0.0015

Total Risk Across All Media	1.9E-08
Total Hazard Across All Media	0.0053
Total Nervous System HI Across All Media	0.0017
Total Skin HI Across All Media	0.00012
Total Thyroid HI Across All Media	0.0014
Total Gastrointestinal HI Across All Media	0.0017

APPENDIX E

TABLE E-65
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADOLESCENT SWIMMER/WADER OMPOMPANOOSUC RIVER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Ompompanoosuc River Sediment	Sediment	Ompompanoosuc River Sediment	Aluminum	---	---	---	Nervous system	0.0041	---	0.0041
			Arsenic	9.9E-08	5.6E-08	1.6E-07	Skin	0.0019	0.0011	0.0030
			Chromium	---	---	---	None observed	0.0000024	---	0.0000024
			Cobalt	---	---	---	Thyroid	0.0050	---	0.0050
			Iron	---	---	---	Gastrointestinal	0.0038	---	0.0038
			Lithium	---	---	---	No information	0.0014	---	0.0014
			Manganese	---	---	---	Nervous system	0.0019	---	0.0019
			Vanadium	---	---	---	None reported	0.00028	---	0.00028
		Exposure Point Total		9.9E-08	5.6E-08	1.6E-07		0.018	0.0011	0.019
	Surface Soil					1.6E-07				0.019
Sediment Total						1.6E-07				0.019
Ompompanoosuc River Surface Water	Surface Water	Ompompanoosuc River Surface Water	Mercury	---	---	---	Nervous system	0.000077	0.00033	0.00041
		Exposure Point Total		0.0E+00	0.0E+00	0.0E+00		0.000077	0.00033	0.00041
	Surface Soil					0.0E+00				0.00041
Surface Water Total						0.0E+00				0.00041

Total Risk Across All Media	1.6E-07
Total Hazard Across All Media	0.020
Total Nervous System HI Across All Media	0.0064
Total Skin HI Across All Media	0.0030
Total Thyroid HI Across All Media	0.0050
Total Gastrointestinal HI Across All Media	0.0038

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TABLE E-66
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT SWIMMER/WADER OMPOMPANOOSUC RIVER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Ompompanoosuc River Sediment	Sediment	Ompompanoosuc River Sediment	Aluminum	---	---	---	Nervous system	0.0030	---	0.0030
			Arsenic	2.2E-07	1.5E-07	3.7E-07	Skin	0.0014	0.00095	0.0024
			Chromium	---	---	---	None observed	0.0000018	---	0.0000018
			Cobalt	---	---	---	Thyroid	0.0037	---	0.0037
			Iron	---	---	---	Gastrointestinal	0.0028	---	0.0028
			Lithium	---	---	---	No information	0.0010	---	0.0010
			Manganese	---	---	---	Nervous system	0.0014	---	0.0014
			Vanadium	---	---	---	None reported	0.00021	---	0.00021
		Exposure Point Total		2.2E-07	1.5E-07	3.7E-07		0.014	0.00095	0.015
	Surface Soil					3.7E-07				0.015
Sediment Total						3.7E-07				0.015
Ompompanoosuc River Surface Water	Surface Water	Ompompanoosuc River Surface Water	Mercury	---	---	---	Nervous system	0.000057	0.00030	0.00035
		Exposure Point Total		0.0E+00	0.0E+00	0.0E+00		0.000057	0.00030	0.00035
	Surface Soil					0.0E+00				0.00035
Surface Water Total						0.0E+00				0.00035

Total Risk Across All Media	3.7E-07
Total Hazard Across All Media	0.015
Total Nervous System HI Across All Media	0.0048
Total Skin HI Across All Media	0.0024
Total Thyroid HI Across All Media	0.0037
Total Gastrointestinal HI Across All Media	0.0028

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TABLE E-67
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADOLESCENT SWIMMER/WADER OMPOMPANOOSUC RIVER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Ompompanoosuc River Sediment	Sediment	Ompompanoosuc River Sediment	Aluminum	---	---	---	Nervous system	0.0010	---	0.0010
			Arsenic	2.5E-08	3.8E-09	2.9E-08	Skin	0.00048	0.000073	0.00056
			Chromium	---	---	---	None observed	0.00000060	---	0.00000060
			Cobalt	---	---	---	Thyroid	0.0013	---	0.0013
			Iron	---	---	---	Gastrointestinal	0.00094	---	0.00094
			Lithium	---	---	---	No information	0.00035	---	0.00035
			Manganese	---	---	---	Nervous system	0.00046	---	0.00046
			Vanadium	---	---	---	None reported	0.000071	---	0.000071
		Exposure Point Total		2.5E-08	3.8E-09	2.9E-08		0.0046	0.000073	0.0047
	Surface Soil					2.9E-08				0.0047
Sediment Total						2.9E-08				0.0047
Ompompanoosuc River Surface Water	Surface Water	Ompompanoosuc River Surface Water	Mercury	---	---	---	Nervous system	0.000019	0.000082	0.00010
		Exposure Point Total		0.0E+00	0.0E+00	0.0E+00		0.000019	0.000082	0.00010
	Surface Soil					0.0E+00				0.00010
Surface Water Total						0.0E+00				0.00010

Total Risk Across All Media	2.9E-08
Total Hazard Across All Media	0.0048
Total Nervous System HI Across All Media	0.0016
Total Skin HI Across All Media	0.00056
Total Thyroid HI Across All Media	0.0013
Total Gastrointestinal HI Across All Media	0.00094

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TABLE E-68
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT SWIMMER/WADER OMPOMPANOOSUC RIVER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current/Future
Receptor Population: Swimmer/Wader
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Ompompanoosuc River Sediment	Sediment	Ompompanoosuc River Sediment	Aluminum	---	---	---	Nervous system	0.00076	---	0.00076
			Arsenic	5.5E-08	1.0E-08	6.6E-08	Skin	0.00036	0.000068	0.00043
			Chromium	---	---	---	None observed	0.00000044	---	0.00000044
			Cobalt	---	---	---	Thyroid	0.00093	---	0.00093
			Iron	---	---	---	Gastrointestinal	0.00070	---	0.00070
			Lithium	---	---	---	No information	0.00026	---	0.00026
			Manganese	---	---	---	Nervous system	0.00034	---	0.00034
			Vanadium	---	---	---	None reported	0.000053	---	0.000053
		Exposure Point Total		5.5E-08	1.0E-08	6.6E-08		0.0034	0.000068	0.0035
	Surface Soil					6.6E-08				0.0035
Sediment Total						6.6E-08				0.0035
Ompompanoosuc River Surface Water	Surface Water	Ompompanoosuc River Surface Water	Mercury	---	---	---	Nervous system	0.000014	0.000074	0.000088
		Exposure Point Total		0.0E+00	0.0E+00	0.0E+00		0.000014	0.000074	0.000088
	Surface Soil					0.0E+00				0.000088
Surface Water Total						0.0E+00				0.000088

Total Risk Across All Media	6.6E-08
Total Hazard Across All Media	0.0036
Total Nervous System HI Across All Media	0.0012
Total Skin HI Across All Media	0.00043
Total Thyroid HI Across All Media	0.00093
Total Gastrointestinal HI Across All Media	0.00070

APPENDIX E

TABLE E-69
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CHILD OVERBURDEN GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Overburden Groundwater	Aluminum	---	---	---	Nervous system	9.6	---	9.6
			Arsenic	6.2E-05	4.1E-07	6.3E-05	Skin	1.6	0.011	1.6
			Barium	---	---	---	Kidney	0.38	0.036	0.42
			Cadmium	---	---	---	Kidney	4.7	0.62	5.3
			Chromium	---	---	---	None observed	0.016	0.016	0.031
			Cobalt	---	---	---	Thyroid	142	---	142
			Copper	---	---	---	Gastrointestinal	79	0.52	80
			Iron	---	---	---	Gastrointestinal	18	---	18
			Manganese	---	---	---	Nervous system	11	1.8	12
			Molybdenum	---	---	---	Increased uric acid	0.67	---	0.67
			Nickel	---	---	---	Body weight	1.3	0.042	1.3
			Vanadium	---	---	---	None reported	0.83	0.21	1.0
			Zinc	---	---	---	Blood	0.82	0.0033	0.83
		Exposure Point Total		6.2E-05	4.1E-07	6.3E-05		270	3.2	274
	Surface Soil					6.3E-05				274
Overburden Groundwater Total						6.3E-05				274

Total Risk Across All Media	6.3E-05
Total Hazard Across All Media	274
Total Nervous System HI Across All Media	22
Total Skin HI Across All Media	1.6
Total Thyroid HI Across All Media	142
Total Kidney HI Across All Media	6.4
Total Gastrointestinal HI Across All Media	98
Total Body Weight HI Across All Media	1.3
Total Blood HI Across All Media	0.83

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TABLE E-70
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT OVERBURDEN GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Overburden Groundwater	Aluminum	---	---	---	Nervous system	4.1	---	4.1
			Arsenic	1.1E-04	5.6E-07	1.1E-04	Skin	0.69	0.0036	0.70
			Barium	---	---	---	Kidney	0.16	0.012	0.18
			Cadmium	---	---	---	Kidney	2.0	0.209	2.2
			Chromium	---	---	---	None observed	0.0067	0.0054	0.012
			Cobalt	---	---	---	Thyroid	61	---	61
			Copper	---	---	---	Gastrointestinal	34	0.18	34
			Iron	---	---	---	Gastrointestinal	7.8	---	7.8
			Manganese	---	---	---	Nervous system	4.6	0.60	5.2
			Molybdenum	---	---	---	Increased uric acid	0.29	---	0.29
			Nickel	---	---	---	Body weight	0.55	0.014	0.57
			Vanadium	---	---	---	None reported	0.36	0.072	0.43
			Zinc	---	---	---	Blood	0.35	0.0011	0.35
		Exposure Point Total		1.1E-04	5.6E-07	1.1E-04		116	1.1	117
	Surface Soil					1.1E-04				117
Overburden Groundwater Total						1.1E-04				117

Total Risk Across All Media	1.1E-04
Total Hazard Across All Media	117
Total Nervous System HI Across All Media	9.3
Total Skin HI Across All Media	0.70
Total Thyroid HI Across All Media	61
Total Kidney HI Across All Media	2.7
Total Gastrointestinal HI Across All Media	42
Total Body Weight HI Across All Media	0.57
Total Blood HI Across All Media	0.35

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TABLE E-71
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CHILD OVERBURDEN GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Overburden Groundwater	Aluminum	---	---	---	Nervous system	4.8	---	4.8
			Arsenic	3.1E-05	2.1E-07	3.1E-05	Skin	0.81	0.0053	0.82
			Barium	---	---	---	Kidney	0.19	0.018	0.21
			Cadmium	---	---	---	Kidney	2.3	0.31	2.6
			Chromium	---	---	---	None observed	0.0078	0.0079	0.016
			Cobalt	---	---	---	Thyroid	71	---	71
			Copper	---	---	---	Gastrointestinal	40	0.26	40
			Iron	---	---	---	Gastrointestinal	9.1	---	9.1
			Manganese	---	---	---	Nervous system	5.4	0.88	6.2
			Molybdenum	---	---	---	Increased uric acid	0.33	---	0.33
			Nickel	---	---	---	Body weight	0.64	0.021	0.66
			Vanadium	---	---	---	None reported	0.42	0.11	0.52
			Zinc	---	---	---	Blood	0.41	0.0016	0.41
		Exposure Point Total		3.1E-05	2.1E-07	3.1E-05		135	1.6	137
	Surface Soil					3.1E-05				137
Overburden Groundwater Total						3.1E-05				137

Total Risk Across All Media	3.1E-05
Total Hazard Across All Media	137
Total Nervous System HI Across All Media	11
Total Skin HI Across All Media	0.82
Total Thyroid HI Across All Media	71
Total Kidney HI Across All Media	3.2
Total Gastrointestinal HI Across All Media	49
Total Body Weight HI Across All Media	0.66
Total Blood HI Across All Media	0.41

APPENDIX E

TABLE E-72
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT OVERBURDEN GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Overburden Groundwater	Aluminum	---	---	---	Nervous system	2.1	---	2.1
			Arsenic	2.0E-05	1.0E-07	2.0E-05	Skin	0.35	0.0018	0.35
			Barium	---	---	---	Kidney	0.082	0.0061	0.088
			Cadmium	---	---	---	Kidney	1.0	0.10	1.1
			Chromium	---	---	---	None observed	0.0033	0.0027	0.0060
			Cobalt	---	---	---	Thyroid	31	---	31
			Copper	---	---	---	Gastrointestinal	17	0.089	17
			Iron	---	---	---	Gastrointestinal	3.9	---	3.9
			Manganese	---	---	---	Nervous system	2.3	0.30	2.6
			Molybdenum	---	---	---	Increased uric acid	0.14	---	0.14
			Nickel	---	---	---	Body weight	0.28	0.0072	0.28
			Vanadium	---	---	---	None reported	0.18	0.036	0.21
			Zinc	---	---	---	Blood	0.18	0.00055	0.18
		Exposure Point Total		2.0E-05	1.0E-07	2.0E-05		58	0.55	59
	Surface Soil					2.0E-05				59
Overburden Groundwater Total						2.0E-05				59

Total Risk Across All Media	2.0E-05
Total Hazard Across All Media	59
Total Nervous System HI Across All Media	4.7
Total Skin HI Across All Media	0.35
Total Thyroid HI Across All Media	31
Total Kidney HI Across All Media	1.3
Total Gastrointestinal HI Across All Media	21
Total Body Weight HI Across All Media	0.28
Total Blood HI Across All Media	0.18

APPENDIX E

TABLE E-73
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CHILD SHALLOW BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Shallow Bedrock Groundwater	Aluminum	---	---	---	Nervous system	23	---	23
			Antimony	---	---	---	Blood	0.69	0.030	0.72
			Arsenic	4.0E-05	2.6E-07	4.0E-05	Skin	1.0	0.0069	1.0
			Beryllium	---	---	---	Gastrointestinal	0.23	0.22	0.45
			Cadmium	---	---	---	Kidney	1.7	0.22	1.9
			Chromium	---	---	---	None observed	0.0073	0.0074	0.015
			Cobalt	---	---	---	Thyroid	57	---	57
			Copper	---	---	---	Gastrointestinal	17	0.11	17
			Iron	---	---	---	Gastrointestinal	28	---	28
			Manganese	---	---	---	Nervous system	8.0	1.3	9.3
			Molybdenum	---	---	---	Increased uric acid	1.2	---	1.2
			Nickel	---	---	---	Body weight	0.40	0.013	0.41
			Vanadium	---	---	---	None reported	0.12	0.030	0.15
			Zinc	---	---	---	Blood	0.41	0.0016	0.41
		Exposure Point Total		4.0E-05	2.6E-07	4.0E-05		139	2.0	141
	Surface Soil					4.0E-05				141
Shallow Bedrock Groundwater Total						4.0E-05				141

Total Risk Across All Media	4.0E-05
Total Hazard Across All Media	141
Total Nervous System HI Across All Media	32
Total Skin HI Across All Media	1.0
Total Thyroid HI Across All Media	57
Total Kidney HI Across All Media	3.1
Total Gastrointestinal HI Across All Media	46
Total Body Weight HI Across All Media	0.41
Total Blood HI Across All Media	1.1

APPENDIX E

TABLE E-74
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT SHALLOW BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Shallow Bedrock Groundwater	Aluminum	---	---	---	Nervous system	9.9	---	9.9
			Antimony	---	---	---	Blood	0.30	0.010	0.31
			Arsenic	6.9E-05	3.6E-07	6.9E-05	Skin	0.45	0.0023	0.45
			Beryllium	---	---	---	Gastrointestinal	0.10	0.075	0.17
			Cadmium	---	---	---	Kidney	0.71	0.074	0.79
			Chromium	---	---	---	None observed	0.0031	0.0025	0.0057
			Cobalt	---	---	---	Thyroid	24	---	24
			Copper	---	---	---	Gastrointestinal	7.3	0.038	7.3
			Iron	---	---	---	Gastrointestinal	12	---	12.1
			Manganese	---	---	---	Nervous system	3.4	0.45	3.9
			Molybdenum	---	---	---	Increased uric acid	0.51	---	0.51
			Nickel	---	---	---	Body weight	0.17	0.0045	0.18
			Vanadium	---	---	---	None reported	0.050	0.010	0.060
			Zinc	---	---	---	Blood	0.17	0.00054	0.17
		Exposure Point Total		6.9E-05	3.6E-07	6.9E-05		60	0.66	60
	Surface Soil					6.9E-05				60
Shallow Bedrock Groundwater Total						6.9E-05				60

Total Risk Across All Media	6.9E-05
Total Hazard Across All Media	60
Total Nervous System HI Across All Media	14
Total Skin HI Across All Media	0.45
Total Thyroid HI Across All Media	24
Total Kidney HI Across All Media	1.3
Total Gastrointestinal HI Across All Media	20
Total Body Weight HI Across All Media	0.18
Total Blood HI Across All Media	0.48

APPENDIX E

TABLE E-75
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CHILD SHALLOW BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Shallow Bedrock Groundwater	Aluminum	---	---	---	Nervous system	12	---	12
			Antimony	---	---	---	Blood	0.35	0.015	0.36
			Arsenic	2.0E-05	1.3E-07	2.0E-05	Skin	0.52	0.0034	0.52
			Beryllium	---	---	---	Gastrointestinal	0.12	0.11	0.23
			Cadmium	---	---	---	Kidney	0.83	0.11	0.94
			Chromium	---	---	---	None observed	0.0037	0.0037	0.0074
			Cobalt	---	---	---	Thyroid	28	---	28
			Copper	---	---	---	Gastrointestinal	8.5	0.056	8.5
			Iron	---	---	---	Gastrointestinal	14	---	14
			Manganese	---	---	---	Nervous system	4.0	0.66	4.7
			Molybdenum	---	---	---	Increased uric acid	0.59	---	0.59
			Nickel	---	---	---	Body weight	0.20	0.0066	0.21
			Vanadium	---	---	---	None reported	0.058	0.015	0.073
			Zinc	---	---	---	Blood	0.20	0.00080	0.20
		Exposure Point Total		2.0E-05	1.3E-07	2.0E-05		69	0.98	70
	Surface Soil					2.0E-05				70
Shallow Bedrock Groundwater Total						2.0E-05				70

Total Risk Across All Media	2.0E-05
Total Hazard Across All Media	70
Total Nervous System HI Across All Media	16
Total Skin HI Across All Media	0.52
Total Thyroid HI Across All Media	28
Total Kidney HI Across All Media	1.5
Total Gastrointestinal HI Across All Media	22.9
Total Body Weight HI Across All Media	0.21
Total Blood HI Across All Media	0.56

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TABLE E-76
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT SHALLOW BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Shallow Bedrock Groundwater	Aluminum	---	---	---	Nervous system	4.9	---	4.9
			Antimony	---	---	---	Blood	0.15	0.0051	0.15
			Arsenic	1.3E-05	6.7E-08	1.3E-05	Skin	0.22	0.0012	0.22
			Beryllium	---	---	---	Gastrointestinal	0.050	0.037	0.087
			Cadmium	---	---	---	Kidney	0.36	0.037	0.39
			Chromium	---	---	---	None observed	0.0016	0.0013	0.0028
			Cobalt	---	---	---	Thyroid	12	---	12
			Copper	---	---	---	Gastrointestinal	3.6	0.019	3.6
			Iron	---	---	---	Gastrointestinal	6.1	---	6.1
			Manganese	---	---	---	Nervous system	1.7	0.22	1.9
			Molybdenum	---	---	---	Increased uric acid	0.25	---	0.25
			Nickel	---	---	---	Body weight	0.086	0.0022	0.088
			Vanadium	---	---	---	None reported	0.025	0.0050	0.030
			Zinc	---	---	---	Blood	0.087	0.00027	0.087
		Exposure Point Total		1.3E-05	6.7E-08	1.3E-05		30	0.33	30
	Surface Soil					1.3E-05				30
Shallow Bedrock Groundwater Total						1.3E-05				30

Total Risk Across All Media	1.3E-05
Total Hazard Across All Media	30
Total Nervous System HI Across All Media	6.9
Total Skin HI Across All Media	0.22
Total Thyroid HI Across All Media	12
Total Kidney HI Across All Media	0.65
Total Gastrointestinal HI Across All Media	10
Total Body Weight HI Across All Media	0.088
Total Blood HI Across All Media	0.24

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TABLE E-77
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CHILD DEEP BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Deep Bedrock Groundwater	Bis(2-Ethylhexyl)Phthalate	5.8E-07	---	5.8E-07	Liver	0.024	---	0.024
			Aluminum	---	---	---	Nervous system	0.44	---	0.44
			Arsenic	2.1E-05	1.4E-07	2.2E-05	Skin	0.6	0.004	0.56
			Cadmium	---	---	---	Kidney	0.84	0.11	0.96
			Chromium	---	---	---	None observed	0.0056	0.0057	0.011
			Cobalt	---	---	---	Thyroid	33	---	33
			Copper	---	---	---	Gastrointestinal	14	0.094	14
			Iron	---	---	---	Gastrointestinal	1.5	---	1.5
			Manganese	---	---	---	Nervous system	3.8	0.63	4.4
			Nickel	---	---	---	Body weight	0.23	0.0092	0.24
			Vanadium	---	---	---	None reported	0.02	0.004	0.020
		Exposure Point Total		2.2E-05	1.4E-07	2.2E-05		54	0.86	55
	Surface Soil					2.2E-05				55
Deep Bedrock Groundwater Total						2.2E-05				55

Total Risk Across All Media	2.2E-05
Total Hazard Across All Media	55
Total Nervous System HI Across All Media	4.9
Total Skin HI Across All Media	0.56
Total Thyroid HI Across All Media	33
Total Kidney HI Across All Media	0.96
Total Gastrointestinal HI Across All Media	16
Total Body Weight HI Across All Media	0.24
Total Liver HI Across All Media	0.024

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TABLE E-78
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT DEEP BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Deep Bedrock Groundwater	Bis(2-Ethylhexyl)Phthalate	9.9E-07	---	9.9E-07	Liver	0.010	---	0.010
			Aluminum	---	---	---	Nervous system	0.19	---	0.19
			Arsenic	3.7E-05	1.9E-07	3.7E-05	Skin	0.24	0.0012	0.24
			Cadmium	---	---	---	Kidney	0.36	0.038	0.40
			Chromium	---	---	---	None observed	0.0024	0.0019	0.0043
			Cobalt	---	---	---	Thyroid	14	---	14
			Copper	---	---	---	Gastrointestinal	6.1	0.032	6.1
			Iron	---	---	---	Gastrointestinal	0.66	---	0.66
			Manganese	---	---	---	Nervous system	1.6	0.21	1.8
			Nickel	---	---	---	Body weight	0.10	0.0031	0.10
			Vanadium	---	---	---	None reported	0.0068	0.0014	0.0082
		Exposure Point Total		3.8E-05	1.9E-07	3.8E-05		23	0.29	24
	Surface Soil					3.8E-05				24
Deep Bedrock Groundwater Total						3.8E-05				24

Total Risk Across All Media	3.8E-05
Total Hazard Across All Media	24
Total Nervous System HI Across All Media	2.0
Total Skin HI Across All Media	0.24
Total Thyroid HI Across All Media	14
Total Kidney HI Across All Media	0.40
Total Gastrointestinal HI Across All Media	6.8
Total Body Weight HI Across All Media	0.10
Total Liver HI Across All Media	0.010

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TABLE E-79
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CHILD DEEP BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Deep Bedrock Groundwater	Bis(2-Ethylhexyl)Phthalate	2.9E-07	---	2.9E-07	Liver	0.012	---	0.012
			Aluminum	---	---	---	Nervous system	0.22	---	0.22
			Arsenic	1.1E-05	7.1E-08	1.1E-05	Skin	0.28	0.0018	0.28
			Cadmium	---	---	---	Kidney	0.42	0.056	0.48
			Chromium	---	---	---	None observed	0.0028	0.0029	0.0057
			Cobalt	---	---	---	Thyroid	16	---	16
			Copper	---	---	---	Gastrointestinal	7.1	0.047	7.2
			Iron	---	---	---	Gastrointestinal	0.77	---	0.77
			Manganese	---	---	---	Nervous system	1.9	0.31	2.2
			Nickel	---	---	---	Body weight	0.12	0.0046	0.12
			Vanadium	---	---	---	None reported	0.0080	0.0020	0.010
		Exposure Point Total		1.1E-05	7.1E-08	1.1E-05		27	0.43	28
	Surface Soil					1.1E-05				28
Deep Bedrock Groundwater Total						1.1E-05				28

Total Risk Across All Media	1.1E-05
Total Hazard Across All Media	28
Total Nervous System HI Across All Media	2.4
Total Skin HI Across All Media	0.28
Total Thyroid HI Across All Media	16
Total Kidney HI Across All Media	0.48
Total Gastrointestinal HI Across All Media	7.9
Total Body Weight HI Across All Media	0.12
Total Liver HI Across All Media	0.012

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TABLE E-80
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT DEEP BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Deep Bedrock Groundwater	Bis(2-Ethylhexyl)Phthalate	1.8E-07	---	1.8E-07	Liver	0.0051	---	0.0051
			Aluminum	---	---	---	Nervous system	0.093	---	0.093
			Arsenic	6.9E-06	3.6E-08	6.9E-06	Skin	0.12	0.00062	0.12
			Cadmium	---	---	---	Kidney	0.18	0.019	0.20
			Chromium	---	---	---	None observed	0.0012	0.00097	0.0022
			Cobalt	---	---	---	Thyroid	7.0	---	7.0
			Copper	---	---	---	Gastrointestinal	3.1	0.016	3.1
			Iron	---	---	---	Gastrointestinal	0.33	---	0.33
			Manganese	---	---	---	Nervous system	0.82	0.11	0.92
			Nickel	---	---	---	Body weight	0.050	0.0016	0.051
			Vanadium	---	---	---	None reported	0.0034	0.00069	0.0041
		Exposure Point Total		7.1E-06	3.6E-08	7.1E-06		12	0.15	12
	Surface Soil					7.1E-06				12
Deep Bedrock Groundwater Total						7.1E-06				12

Total Risk Across All Media	7.1E-06
Total Hazard Across All Media	12
Total Nervous System HI Across All Media	1.0
Total Skin HI Across All Media	0.12
Total Thyroid HI Across All Media	7.0
Total Kidney HI Across All Media	0.20
Total Gastrointestinal HI Across All Media	3.4
Total Body Weight HI Across All Media	0.051
Total Liver HI Across All Media	0.0051

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TABLE E-81
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CHILD RESIDENTIAL GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Residential Groundwater	Copper	---	---	---	Gastrointestinal	0.31	0.0020	0.31
			Vanadium	---	---	---	None reported	0.0054	0.0014	0.0068
		Exposure Point Total		---	---	---		0.31	0.0034	0.32
	Surface Soil					---				0.32
Residential Groundwater Total						---				0.32

Total Risk Across All Media: ---

Total Hazard Across All Media: 0.32

Total Gastrointestinal HI Across All Media: 0.31

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TABLE E-73
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CHILD SHALLOW BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Shallow Bedrock Groundwater	Aluminum	---	---	---	Nervous system	23	---	23
			Antimony	---	---	---	Blood	0.69	0.030	0.72
			Arsenic	4.0E-05	2.6E-07	4.0E-05	Skin	1.0	0.0069	1.0
			Beryllium	---	---	---	Gastrointestinal	0.23	0.22	0.45
			Cadmium	---	---	---	Kidney	1.7	0.22	1.9
			Chromium	---	---	---	None observed	0.0073	0.0074	0.015
			Cobalt	---	---	---	Thyroid	57	---	57
			Copper	---	---	---	Gastrointestinal	17	0.11	17
			Iron	---	---	---	Gastrointestinal	28	---	28
			Manganese	---	---	---	Nervous system	8.0	1.3	9.3
			Molybdenum	---	---	---	Increased uric acid	1.2	---	1.2
			Nickel	---	---	---	Body weight	0.40	0.013	0.41
			Vanadium	---	---	---	None reported	0.12	0.030	0.15
			Zinc	---	---	---	Blood	0.41	0.0016	0.41
		Exposure Point Total		4.0E-05	2.6E-07	4.0E-05		139	2.0	141
	Surface Soil					4.0E-05				141
Shallow Bedrock Groundwater Total						4.0E-05				141

Total Risk Across All Media	4.0E-05
Total Hazard Across All Media	141
Total Nervous System HI Across All Media	32
Total Skin HI Across All Media	1.0
Total Thyroid HI Across All Media	57
Total Kidney HI Across All Media	3.1
Total Gastrointestinal HI Across All Media	46
Total Body Weight HI Across All Media	0.41
Total Blood HI Across All Media	1.1

APPENDIX E

TABLE E-74
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT SHALLOW BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Shallow Bedrock Groundwater	Aluminum	---	---	---	Nervous system	9.9	---	9.9
			Antimony	---	---	---	Blood	0.30	0.010	0.31
			Arsenic	6.9E-05	3.6E-07	6.9E-05	Skin	0.45	0.0023	0.45
			Beryllium	---	---	---	Gastrointestinal	0.10	0.075	0.17
			Cadmium	---	---	---	Kidney	0.71	0.074	0.79
			Chromium	---	---	---	None observed	0.0031	0.0025	0.0057
			Cobalt	---	---	---	Thyroid	24	---	24
			Copper	---	---	---	Gastrointestinal	7.3	0.038	7.3
			Iron	---	---	---	Gastrointestinal	12	---	12.1
			Manganese	---	---	---	Nervous system	3.4	0.45	3.9
			Molybdenum	---	---	---	Increased uric acid	0.51	---	0.51
			Nickel	---	---	---	Body weight	0.17	0.0045	0.18
			Vanadium	---	---	---	None reported	0.050	0.010	0.060
			Zinc	---	---	---	Blood	0.17	0.00054	0.17
		Exposure Point Total		6.9E-05	3.6E-07	6.9E-05		60	0.66	60
	Surface Soil					6.9E-05				60
Shallow Bedrock Groundwater Total						6.9E-05				60

Total Risk Across All Media	6.9E-05
Total Hazard Across All Media	60
Total Nervous System HI Across All Media	14
Total Skin HI Across All Media	0.45
Total Thyroid HI Across All Media	24
Total Kidney HI Across All Media	1.3
Total Gastrointestinal HI Across All Media	20
Total Body Weight HI Across All Media	0.18
Total Blood HI Across All Media	0.48

APPENDIX E

TABLE E-75
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CHILD SHALLOW BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Shallow Bedrock Groundwater	Aluminum	---	---	---	Nervous system	12	---	12
			Antimony	---	---	---	Blood	0.35	0.015	0.36
			Arsenic	2.0E-05	1.3E-07	2.0E-05	Skin	0.52	0.0034	0.52
			Beryllium	---	---	---	Gastrointestinal	0.12	0.11	0.23
			Cadmium	---	---	---	Kidney	0.83	0.11	0.94
			Chromium	---	---	---	None observed	0.0037	0.0037	0.0074
			Cobalt	---	---	---	Thyroid	28	---	28
			Copper	---	---	---	Gastrointestinal	8.5	0.056	8.5
			Iron	---	---	---	Gastrointestinal	14	---	14
			Manganese	---	---	---	Nervous system	4.0	0.66	4.7
			Molybdenum	---	---	---	Increased uric acid	0.59	---	0.59
			Nickel	---	---	---	Body weight	0.20	0.0066	0.21
			Vanadium	---	---	---	None reported	0.058	0.015	0.073
			Zinc	---	---	---	Blood	0.20	0.00080	0.20
		Exposure Point Total		2.0E-05	1.3E-07	2.0E-05		69	0.98	70
	Surface Soil					2.0E-05				70
Shallow Bedrock Groundwater Total						2.0E-05				70

Total Risk Across All Media	2.0E-05
Total Hazard Across All Media	70
Total Nervous System HI Across All Media	16
Total Skin HI Across All Media	0.52
Total Thyroid HI Across All Media	28
Total Kidney HI Across All Media	1.5
Total Gastrointestinal HI Across All Media	22.9
Total Body Weight HI Across All Media	0.21
Total Blood HI Across All Media	0.56

APPENDIX E

TABLE E-76
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT SHALLOW BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Shallow Bedrock Groundwater	Aluminum	---	---	---	Nervous system	4.9	---	4.9
			Antimony	---	---	---	Blood	0.15	0.0051	0.15
			Arsenic	1.3E-05	6.7E-08	1.3E-05	Skin	0.22	0.0012	0.22
			Beryllium	---	---	---	Gastrointestinal	0.050	0.037	0.087
			Cadmium	---	---	---	Kidney	0.36	0.037	0.39
			Chromium	---	---	---	None observed	0.0016	0.0013	0.0028
			Cobalt	---	---	---	Thyroid	12	---	12
			Copper	---	---	---	Gastrointestinal	3.6	0.019	3.6
			Iron	---	---	---	Gastrointestinal	6.1	---	6.1
			Manganese	---	---	---	Nervous system	1.7	0.22	1.9
			Molybdenum	---	---	---	Increased uric acid	0.25	---	0.25
			Nickel	---	---	---	Body weight	0.086	0.0022	0.088
			Vanadium	---	---	---	None reported	0.025	0.0050	0.030
			Zinc	---	---	---	Blood	0.087	0.00027	0.087
		Exposure Point Total		1.3E-05	6.7E-08	1.3E-05		30	0.33	30
	Surface Soil					1.3E-05				30
Shallow Bedrock Groundwater Total						1.3E-05				30

Total Risk Across All Media	1.3E-05
Total Hazard Across All Media	30
Total Nervous System HI Across All Media	6.9
Total Skin HI Across All Media	0.22
Total Thyroid HI Across All Media	12
Total Kidney HI Across All Media	0.65
Total Gastrointestinal HI Across All Media	10
Total Body Weight HI Across All Media	0.088
Total Blood HI Across All Media	0.24

APPENDIX E

TABLE E-77
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CHILD DEEP BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Deep Bedrock Groundwater	Bis(2-Ethylhexyl)Phthalate	5.8E-07	---	5.8E-07	Liver	0.024	---	0.024
			Aluminum	---	---	---	Nervous system	0.44	---	0.44
			Arsenic	2.1E-05	1.4E-07	2.2E-05	Skin	0.6	0.004	0.56
			Cadmium	---	---	---	Kidney	0.84	0.11	0.96
			Chromium	---	---	---	None observed	0.0056	0.0057	0.011
			Cobalt	---	---	---	Thyroid	33	---	33
			Copper	---	---	---	Gastrointestinal	14	0.094	14
			Iron	---	---	---	Gastrointestinal	1.5	---	1.5
			Manganese	---	---	---	Nervous system	3.8	0.63	4.4
			Nickel	---	---	---	Body weight	0.23	0.0092	0.24
			Vanadium	---	---	---	None reported	0.02	0.004	0.020
		Exposure Point Total		2.2E-05	1.4E-07	2.2E-05		54	0.86	55
	Surface Soil					2.2E-05				55
Deep Bedrock Groundwater Total						2.2E-05				55

Total Risk Across All Media	2.2E-05
Total Hazard Across All Media	55
Total Nervous System HI Across All Media	4.9
Total Skin HI Across All Media	0.56
Total Thyroid HI Across All Media	33
Total Kidney HI Across All Media	0.96
Total Gastrointestinal HI Across All Media	16
Total Body Weight HI Across All Media	0.24
Total Liver HI Across All Media	0.024

APPENDIX E

TABLE E-78
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT DEEP BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Deep Bedrock Groundwater	Bis(2-Ethylhexyl)Phthalate	9.9E-07	---	9.9E-07	Liver	0.010	---	0.010
			Aluminum	---	---	---	Nervous system	0.19	---	0.19
			Arsenic	3.7E-05	1.9E-07	3.7E-05	Skin	0.24	0.0012	0.24
			Cadmium	---	---	---	Kidney	0.36	0.038	0.40
			Chromium	---	---	---	None observed	0.0024	0.0019	0.0043
			Cobalt	---	---	---	Thyroid	14	---	14
			Copper	---	---	---	Gastrointestinal	6.1	0.032	6.1
			Iron	---	---	---	Gastrointestinal	0.66	---	0.66
			Manganese	---	---	---	Nervous system	1.6	0.21	1.8
			Nickel	---	---	---	Body weight	0.10	0.0031	0.10
			Vanadium	---	---	---	None reported	0.0068	0.0014	0.0082
		Exposure Point Total		3.8E-05	1.9E-07	3.8E-05		23	0.29	24
	Surface Soil					3.8E-05				24
Deep Bedrock Groundwater Total						3.8E-05				24

Total Risk Across All Media	3.8E-05
Total Hazard Across All Media	24
Total Nervous System HI Across All Media	2.0
Total Skin HI Across All Media	0.24
Total Thyroid HI Across All Media	14
Total Kidney HI Across All Media	0.40
Total Gastrointestinal HI Across All Media	6.8
Total Body Weight HI Across All Media	0.10
Total Liver HI Across All Media	0.010

APPENDIX E

TABLE E-79
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CHILD DEEP BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Deep Bedrock Groundwater	Bis(2-Ethylhexyl)Phthalate	2.9E-07	---	2.9E-07	Liver	0.012	---	0.012
			Aluminum	---	---	---	Nervous system	0.22	---	0.22
			Arsenic	1.1E-05	7.1E-08	1.1E-05	Skin	0.28	0.0018	0.28
			Cadmium	---	---	---	Kidney	0.42	0.056	0.48
			Chromium	---	---	---	None observed	0.0028	0.0029	0.0057
			Cobalt	---	---	---	Thyroid	16	---	16
			Copper	---	---	---	Gastrointestinal	7.1	0.047	7.2
			Iron	---	---	---	Gastrointestinal	0.77	---	0.77
			Manganese	---	---	---	Nervous system	1.9	0.31	2.2
			Nickel	---	---	---	Body weight	0.12	0.0046	0.12
			Vanadium	---	---	---	None reported	0.0080	0.0020	0.010
		Exposure Point Total		1.1E-05	7.1E-08	1.1E-05		27	0.43	28
	Surface Soil					1.1E-05				28
Deep Bedrock Groundwater Total						1.1E-05				28

Total Risk Across All Media	1.1E-05
Total Hazard Across All Media	28
Total Nervous System HI Across All Media	2.4
Total Skin HI Across All Media	0.28
Total Thyroid HI Across All Media	16
Total Kidney HI Across All Media	0.48
Total Gastrointestinal HI Across All Media	7.9
Total Body Weight HI Across All Media	0.12
Total Liver HI Across All Media	0.012

APPENDIX E

TABLE E-80
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT DEEP BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Deep Bedrock Groundwater	Bis(2-Ethylhexyl)Phthalate	1.8E-07	---	1.8E-07	Liver	0.0051	---	0.0051
			Aluminum	---	---	---	Nervous system	0.093	---	0.093
			Arsenic	6.9E-06	3.6E-08	6.9E-06	Skin	0.12	0.00062	0.12
			Cadmium	---	---	---	Kidney	0.18	0.019	0.20
			Chromium	---	---	---	None observed	0.0012	0.00097	0.0022
			Cobalt	---	---	---	Thyroid	7.0	---	7.0
			Copper	---	---	---	Gastrointestinal	3.1	0.016	3.1
			Iron	---	---	---	Gastrointestinal	0.33	---	0.33
			Manganese	---	---	---	Nervous system	0.82	0.11	0.92
			Nickel	---	---	---	Body weight	0.050	0.0016	0.051
			Vanadium	---	---	---	None reported	0.0034	0.00069	0.0041
		Exposure Point Total		7.1E-06	3.6E-08	7.1E-06		12	0.15	12
	Surface Soil					7.1E-06				12
Deep Bedrock Groundwater Total						7.1E-06				12

Total Risk Across All Media	7.1E-06
Total Hazard Across All Media	12
Total Nervous System HI Across All Media	1.0
Total Skin HI Across All Media	0.12
Total Thyroid HI Across All Media	7.0
Total Kidney HI Across All Media	0.20
Total Gastrointestinal HI Across All Media	3.4
Total Body Weight HI Across All Media	0.051
Total Liver HI Across All Media	0.0051

APPENDIX E

TABLE E-81
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CHILD RESIDENTIAL GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Residential Groundwater	Copper	---	---	---	Gastrointestinal	0.31	0.0020	0.31
			Vanadium	---	---	---	None reported	0.0054	0.0014	0.0068
		Exposure Point Total		---	---	---		0.31	0.0034	0.32
	Surface Soil					---				0.32
Residential Groundwater Total						---				0.32

Total Risk Across All Media: ---

Total Hazard Across All Media: 0.32

Total Gastrointestinal HI Across All Media: 0.31

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TABLE E-82
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT RESIDENTIAL GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Residential Groundwater	Copper	---	---	---	Gastrointestinal	0.13	0.00069	0.13
			Vanadium	---	---	---	None reported	0.0023	0.00047	0.0028
		Exposure Point Total		---	---	---		0.13	0.0012	0.13
	Surface Soil					---				0.13
Residential Groundwater Total						---				0.13

Total Risk Across All Media: ---

Total Hazard Across All Media: 0.13

Total Gastrointestinal HI Across All Media: 0.13

APPENDIX E

TABLE E-83
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - CHILD RESIDENTIAL GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Residential Groundwater	Copper	---	---	---	Gastrointestinal	0.15	0.0010	0.15
			Vanadium	---	---	---	None reported	0.0027	0.00069	0.0034
		Exposure Point Total		---	---	---		0.16	0.0017	0.16
	Surface Soil					---				0.16
Residential Groundwater Total						---				0.16

Total Risk Across All Media: ---

Total Hazard Across All Media: 0.16

Total Gastrointestinal HI Across All Media: 0.15

APPENDIX E

TABLE E-84
RAGS PART D TABLE 9 - SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs - ADULT RESIDENTIAL GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Contaminant of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Residential Groundwater	Copper	---	---	---	Gastrointestinal	0.066	0.00034	0.066
			Vanadium	---	---	---	None reported	0.0012	0.00023	0.0014
		Exposure Point Total		---	---	---		0.067	0.00058	0.067
	Surface Soil					---				0.067
Residential Groundwater Total						---				0.067

Total Risk Across All Media: ---

Total Hazard Across All Media: 0.067

Total Gastrointestinal HI Across All Media: 0.066

APPENDIX E

TABLE E-85
RAGS PART D TABLE 10 - RISK SUMMARY - CURRENT CHILD RESIDENT
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Ely Copper Mine	Aluminum	---	---	---	---	---	---	---	---	---
			Antimony	---	---	---	---	---	---	---	---	---
			Arsenic	4.5E-06	---	---	4.5E-06	---	---	---	---	---
			Cadmium	---	---	---	---	---	---	---	---	---
			Chromium	---	---	---	---	---	---	---	---	---
		Surface Soil	Cobalt	---	---	---	---	Thyroid	1.0	---	---	1.0
			Copper	---	---	---	---	---	---	---	---	---
			Iron	---	---	---	---	Gastrointestinal	1.2	---	---	1.2
			Manganese	---	---	---	---	---	---	---	---	---
			Mercury	---	---	---	---	---	---	---	---	---
			Molybdenum	---	---	---	---	---	---	---	---	---
			Selenium	---	---	---	---	---	---	---	---	---
			Chemical Total	4.5E-06	---	---	4.5E-06		2.3	---	---	2.3
		Ely Copper Mine Surface Soil Total					4.5E-06					2.3
	Surface Soil Total						4.5E-06					2.3
Soil Total							4.5E-06					2.3

Total Risk Across All Media: 4.5E-06

Total Hazard Across All Media: 2.3

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TABLE E-86
RAGS PART D TABLE 10 - RISK SUMMARY - CURRENT ADULT RESIDENT
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Ely Copper Mine	Aluminum	---	---	---	---	---	---	---	---	---
			Antimony	---	---	---	---	---	---	---	---	---
			Arsenic	1.9E-06	---	---	1.9E-06	---	---	---	---	---
			Cadmium	---	---	---	---	---	---	---	---	---
			Chromium	---	---	---	---	---	---	---	---	---
			Cobalt	---	---	---	---	---	---	---	---	---
			Copper	---	---	---	---	---	---	---	---	---
			Iron	---	---	---	---	---	---	---	---	---
			Manganese	---	---	---	---	---	---	---	---	---
			Mercury	---	---	---	---	---	---	---	---	---
			Molybdenum	---	---	---	---	---	---	---	---	---
			Selenium	---	---	---	---	---	---	---	---	---
		Surface Soil	Chemical Total	1.9E-06	---	---	1.9E-06		---	---	---	---
		Ely Copper Mine Surface Soil Total					1.9E-06					---
	Surface Soil Total						1.9E-06					---
Soil Total							1.9E-06					---

Total Risk Across All Media: 1.9E-06

Total Hazard Across All Media: ---

APPENDIX E

TABLE E-87
RAGS PART D TABLE 10 - RISK SUMMARY - CURRENT CHILD RESIDENT
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Current
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Surface Soil	Ely Copper Mine	Aluminum	---	---	---	---	---	---	---	---	---
			Antimony	---	---	---	---	---	---	---	---	---
			Arsenic	2.2E-06	---	---	2.2E-06	---	---	---	---	---
			Cadmium	---	---	---	---	---	---	---	---	---
			Chromium	---	---	---	---	---	---	---	---	---
			Cobalt	---	---	---	---	---	---	---	---	---
			Copper	---	---	---	---	---	---	---	---	---
			Iron	---	---	---	---	---	---	---	---	---
			Manganese	---	---	---	---	---	---	---	---	---
			Mercury	---	---	---	---	---	---	---	---	---
			Molybdenum	---	---	---	---	---	---	---	---	---
			Selenium	---	---	---	---	---	---	---	---	---
		Surface Soil	Chemical Total	2.2E-06	---	---	2.2E-06		---	---	---	---
		Ely Copper Mine Surface Soil Total					2.2E-06					---
	Surface Soil Total						2.2E-06					---
Soil Total							2.2E-06					---

Total Risk Across All Media: 2.2E-06

Total Hazard Across All Media: ---

APPENDIX E

TABLE E-88
RAGS PART D TABLE 10 - RISK SUMMARY - FUTURE CHILD RESIDENT
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Total Soil	Ely Copper Mine	Aluminum	---	---	---	---	---	---	---	---	---
			Antimony	---	---	---	---	---	---	---	---	---
			Arsenic	3.8E-06	---	---	3.8E-06	---	---	---	---	---
			Cadmium	---	---	---	---	---	---	---	---	---
			Chromium	---	---	---	---	---	---	---	---	---
		Total Soil	Cobalt	---	---	---	---	Thyroid	2.1	---	---	2.1
			Copper	---	---	---	---	---	---	---	---	---
			Iron	---	---	---	---	Gastrointestinal	1.2	---	---	1.2
			Manganese	---	---	---	---	---	---	---	---	---
			Mercury	---	---	---	---	---	---	---	---	---
			Molybdenum	---	---	---	---	---	---	---	---	---
			Nickel	---	---	---	---	---	---	---	---	---
			Selenium	---	---	---	---	---	---	---	---	---
			Zinc	---	---	---	---	---	---	---	---	---
			Chemical Total	3.8E-06	---	---	3.8E-06		3.4	---	---	3.4
		Ely Copper Mine Total Soil Total					3.8E-06					3.4
	Total Soil Total						3.8E-06					3.4
Soil Total							3.8E-06					3.4

Total Risk Across All Media: 3.8E-06

Total Hazard Across All Media: 3.4

APPENDIX E

TABLE E-89
RAGS PART D TABLE 10 - RISK SUMMARY - FUTURE ADULT RESIDENT
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Total Soil	Ely Copper Mine	Aluminum	---	---	---	---	---	---	---	---	---
			Antimony	---	---	---	---	---	---	---	---	---
			Arsenic	1.6E-06	---	---	1.6E-06	---	---	---	---	---
			Cadmium	---	---	---	---	---	---	---	---	---
			Chromium	---	---	---	---	---	---	---	---	---
			Cobalt	---	---	---	---	---	---	---	---	---
			Copper	---	---	---	---	---	---	---	---	---
			Iron	---	---	---	---	---	---	---	---	---
			Manganese	---	---	---	---	---	---	---	---	---
			Mercury	---	---	---	---	---	---	---	---	---
			Molybdenum	---	---	---	---	---	---	---	---	---
			Nickel	---	---	---	---	---	---	---	---	---
			Selenium	---	---	---	---	---	---	---	---	---
		Total Soil	Zinc	---	---	---	---	---	---	---	---	---
		Total Soil	Chemical Total	1.6E-06	---	---	1.6E-06		---	---	---	---
		Ely Copper Mine Total Soil Total					1.6E-06					---
	Total Soil Total						1.6E-06					---
Soil Total							1.6E-06					---

Total Risk Across All Media: 1.6E-06

Total Hazard Across All Media: ---

APPENDIX E

TABLE E-90
RAGS PART D TABLE 10 - RISK SUMMARY - FUTURE CHILD RESIDENT
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk				Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Inhalation	Dermal	Exposure Routes Total
Soil	Total Soil	Ely Copper Mine	Aluminum	---	---	---	---	---	---	---	---	---
			Antimony	---	---	---	---	---	---	---	---	---
			Arsenic	1.9E-06	---	---	1.9E-06	---	---	---	---	---
			Cadmium	---	---	---	---	---	---	---	---	---
			Chromium	---	---	---	---	---	---	---	---	---
		Total Soil	Cobalt	---	---	---	---	Thyroid	1.1	---	---	1.1
			Copper	---	---	---	---	---	---	---	---	---
			Iron	---	---	---	---	---	---	---	---	---
			Manganese	---	---	---	---	---	---	---	---	---
			Mercury	---	---	---	---	---	---	---	---	---
			Molybdenum	---	---	---	---	---	---	---	---	---
			Nickel	---	---	---	---	---	---	---	---	---
			Selenium	---	---	---	---	---	---	---	---	---
			Zinc	---	---	---	---	---	---	---	---	---
			Chemical Total	1.9E-06	---	---	1.9E-06		1.1	---	---	1.1
		Ely Copper Mine Total Soil Total					1.9E-06					1.1
	Total Soil Total						1.9E-06					1.1
Soil Total							1.9E-06					1.1

Total Risk Across All Media: 1.9E-06

Total Hazard Across All Media: 1.1

APPENDIX E

TABLE E-91
RAGS PART D TABLE 10 - RISK SUMMARY - CHILD OVERBURDEN GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Overburden	Aluminum	---	---	---	Nervous system	9.6	---	9.6
			Arsenic	6.2E-05	---	6.2E-05	Skin	1.6	---	1.6
			Barium	---	---	---	---	---	---	---
			Cadmium	---	---	---	Kidney	4.7	---	4.7
			Chromium	---	---	---	---	---	---	---
			Cobalt	---	---	---	Thyroid	142	---	142
			Copper	---	---	---	Gastrointestinal	79	---	79
			Iron	---	---	---	Gastrointestinal	18	---	18
			Manganese	---	---	---	Nervous system	11	1.8	12
			Molybdenum	---	---	---	---	---	---	---
			Nickel	---	---	---	Body weight	1.3	---	1.3
			Chemical Total	6.2E-05	---	6.2E-05		268	1.77	270
		Overburden Groundwater Total				6.2E-05				270
	Groundwater Total					6.2E-05				270
Groundwater Total						6.2E-05				270

Total Risk Across All Media: 6.2E-05

Total Hazard Across All Media: 270

APPENDIX E

TABLE E-92
RAGS PART D TABLE 10 - RISK SUMMARY - ADULT OVERBURDEN GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Overburden	Aluminum	---	---	---	Nervous system	4.1	---	4.1
			Arsenic	1.1E-04	---	1.1E-04	---	---	---	---
			Barium	---	---	---	---	---	---	---
			Cadmium	---	---	---	Kidney	2.0	---	2.0
			Chromium	---	---	---	---	---	---	---
			Cobalt	---	---	---	Thyroid	61	---	61
			Copper	---	---	---	Gastrointestinal	34	---	34
			Iron	---	---	---	Gastrointestinal	7.8	---	7.8
			Manganese	---	---	---	Nervous system	4.6	---	4.6
			Molybdenum	---	---	---	---	---	---	---
			Nickel	---	---	---	---	---	---	---
			Chemical Total	1.1E-04	---	1.1E-04		113	---	113
		Overburden Groundwater Total				1.1E-04				113
	Groundwater Total					1.1E-04				113
Groundwater Total						1.1E-04				113

Total Risk Across All Media: 1.1E-04

Total Hazard Across All Media: 113

APPENDIX E

TABLE E-93
RAGS PART D TABLE 10 - RISK SUMMARY - CHILD OVERBURDEN GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Overburden	Aluminum	---	---	---	Nervous system	4.8	---	4.8
			Arsenic	3.1E-05	---	3.1E-05	---	---	---	---
			Barium	---	---	---	---	---	---	---
			Cadmium	---	---	---	Kidney	2.3	---	2.3
			Chromium	---	---	---	---	---	---	---
			Cobalt	---	---	---	Thyroid	71	---	71
			Copper	---	---	---	Gastrointestinal	40	---	40
			Iron	---	---	---	Gastrointestinal	9.1	---	9.1
			Manganese	---	---	---	Nervous system	5.4	---	5.4
			Molybdenum	---	---	---	---	---	---	---
			Nickel	---	---	---	---	---	---	---
			Chemical Total	3.1E-05	---	3.1E-05		132	---	132
		Overburden Groundwater Total				3.1E-05				132
	Groundwater Total					3.1E-05				132
Groundwater Total						3.1E-05				132

Total Risk Across All Media: 3.1E-05

Total Hazard Across All Media: 132

APPENDIX E

TABLE E-94
RAGS PART D TABLE 10 - RISK SUMMARY - ADULT OVERBURDEN GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Overburden	Aluminum	---	---	---	Nervous system	2.1	---	2.1
			Arsenic	2.0E-05	---	2.0E-05	---	---	---	---
			Barium	---	---	---	---	---	---	---
			Cadmium	---	---	---	---	---	---	---
			Chromium	---	---	---	---	---	---	---
			Cobalt	---	---	---	Thyroid	31	---	31
			Copper	---	---	---	Gastrointestinal	17	---	17
			Iron	---	---	---	Gastrointestinal	3.9	---	3.9
			Manganese	---	---	---	Nervous system	2.3	---	2.3
			Molybdenum	---	---	---	---	---	---	---
			Nickel	---	---	---	---	---	---	---
			Chemical Total	2.0E-05	---	2.0E-05		56	---	56
		Overburden Groundwater Total				2.0E-05				56
	Groundwater Total					2.0E-05				56
Groundwater Total						2.0E-05				56

Total Risk Across All Media: 2.0E-05

Total Hazard Across All Media: 56

APPENDIX E

TABLE E-95
RAGS PART D TABLE 10 - RISK SUMMARY - CHILD SHALLOW BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Shallow Bedrock	Aluminum	---	---	---	Nervous system	23	---	23
			Antimony	---	---	---	---	---	---	---
			Arsenic	4.0E-05	---	4.0E-05	Skin	1.0	---	1.0
			Beryllium	---	---	---	---	---	---	---
			Cadmium	---	---	---	Kidney	1.7	---	1.7
			Chromium	---	---	---	---	---	---	---
			Cobalt	---	---	---	Thyroid	57	---	57
			Copper	---	---	---	Gastrointestinal	17	---	17
			Iron	---	---	---	Gastrointestinal	28	---	28
			Manganese	---	---	---	Nervous system	8.0	1.3	9.3
			Molybdenum	---	---	---	Increased uric acid	1.2	---	1.2
			Nickel	---	---	---	---	---	---	---
			Zinc	---	---	---	---	---	---	---
			Chemical Total	4.0E-05	---	4.0E-05		137	1.3	138
		Shallow Bedrock Groundwater Total				4.0E-05				138
	Groundwater Total					4.0E-05				138
Groundwater Total						4.0E-05				138

Total Risk Across All Media: 4.0E-05

Total Hazard Across All Media: 138

APPENDIX E

TABLE E-96
RAGS PART D TABLE 10 - RISK SUMMARY - ADULT SHALLOW BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Shallow Bedrock	Aluminum	---	---	---	Nervous system	9.9	---	9.9
			Antimony	---	---	---	---	---	---	---
			Arsenic	6.9E-05	---	6.9E-05	---	---	---	---
			Beryllium	---	---	---	---	---	---	---
			Cadmium	---	---	---	---	---	---	---
			Chromium	---	---	---	---	---	---	---
			Cobalt	---	---	---	Thyroid	24	---	24
			Copper	---	---	---	Gastrointestinal	7.3	---	7.3
			Iron	---	---	---	Gastrointestinal	12	---	12
			Manganese	---	---	---	Nervous system	3.4	---	3.4
			Molybdenum	---	---	---	---	---	---	---
			Nickel	---	---	---	---	---	---	---
			Zinc	---	---	---	---	---	---	---
			Chemical Total	6.9E-05	---	6.9E-05		57	---	57
		Shallow Bedrock Groundwater Total				6.9E-05				57
	Groundwater Total					6.9E-05				57
Groundwater Total						6.9E-05				57

Total Risk Across All Media: 6.9E-05

Total Hazard Across All Media: 57

APPENDIX E

TABLE E-97
RAGS PART D TABLE 10 - RISK SUMMARY - CHILD SHALLOW BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Shallow Bedrock	Aluminum	---	---	---	Nervous system	12	---	12
			Antimony	---	---	---	---	---	---	---
			Arsenic	2.0E-05	---	2.0E-05	---	---	---	---
			Beryllium	---	---	---	---	---	---	---
			Cadmium	---	---	---	---	---	---	---
			Chromium	---	---	---	---	---	---	---
			Cobalt	---	---	---	Thyroid	28	---	28
			Copper	---	---	---	Gastrointestinal	8.5	---	8.5
			Iron	---	---	---	Gastrointestinal	14	---	14
			Manganese	---	---	---	Nervous system	4.0	---	4.0
			Molybdenum	---	---	---	---	---	---	---
			Nickel	---	---	---	---	---	---	---
			Zinc	---	---	---	---	---	---	---
			Chemical Total	2.0E-05	---	2.0E-05		67	---	67
		Shallow Bedrock Groundwater Total				2.0E-05				67
	Groundwater Total					2.0E-05				67
Groundwater Total						2.0E-05				67

Total Risk Across All Media: 2.0E-05

Total Hazard Across All Media: 67

APPENDIX E

TABLE E-98
RAGS PART D TABLE 10 - RISK SUMMARY - ADULT SHALLOW BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Shallow Bedrock	Aluminum	---	---	---	Nervous system	4.9	---	4.9
			Antimony	---	---	---	---	---	---	---
			Arsenic	1.3E-05	---	1.3E-05	---	---	---	---
			Beryllium	---	---	---	---	---	---	---
			Cadmium	---	---	---	---	---	---	---
			Chromium	---	---	---	---	---	---	---
			Cobalt	---	---	---	Thyroid	12	---	12
			Copper	---	---	---	Gastrointestinal	3.6	---	3.6
			Iron	---	---	---	Gastrointestinal	6.1	---	6.1
			Manganese	---	---	---	Nervous system	1.7	---	1.7
			Molybdenum	---	---	---	---	---	---	---
			Nickel	---	---	---	---	---	---	---
			Zinc	---	---	---	---	---	---	---
			Chemical Total	1.3E-05	---	1.3E-05		29	---	29
		Shallow Bedrock Groundwater Total				1.3E-05				29
	Groundwater Total					1.3E-05				29
Groundwater Total						1.3E-05				29

Total Risk Across All Media: 1.3E-05

Total Hazard Across All Media: 29

APPENDIX E

TABLE E-99
RAGS PART D TABLE 10 - RISK SUMMARY - CHILD DEEP BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Deep Bedrock	Bis(2-Ethylhexyl)Phthalate	---	---	---	---	---	---	---
			Aluminum	---	---	---	---	---	---	---
			Arsenic	2.1E-05	---	2.1E-05	---	---	---	---
			Cadmium	---	---	---	---	---	---	---
			Chromium	---	---	---	---	---	---	---
			Cobalt	---	---	---	Thyroid	33	---	33
			Copper	---	---	---	Gastrointestinal	14	---	14
			Iron	---	---	---	Gastrointestinal	1.5	---	1.5
			Manganese	---	---	---	Nervous system	3.8	---	3.8
			Nickel	---	---	---	---	---	---	---
			Chemical Total	2.1E-05	---	2.1E-05		52	---	52
		Deep Bedrock Groundwater Total				2.1E-05				52
	Groundwater Total					2.1E-05				52
Groundwater Total						2.1E-05				52

Total Risk Across All Media: 2.1E-05

Total Hazard Across All Media: 52

APPENDIX E

TABLE E-100
RAGS PART D TABLE 10 - RISK SUMMARY - ADULT DEEP BEDROCK GROUNDWATER
REASONABLE MAXIMUM EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Deep Bedrock	Bis(2-Ethylhexyl)Phthalate	---	---	---	---	---	---	---
			Aluminum	---	---	---	---	---	---	---
			Arsenic	3.7E-05	---	3.7E-05	---	---	---	---
			Cadmium	---	---	---	---	---	---	---
			Chromium	---	---	---	---	---	---	---
		Groundwater	Cobalt	---	---	---	Thyroid	14	---	14
			Copper	---	---	---	Gastrointestinal	6.1	---	6.1
			Iron	---	---	---	---	---	---	---
			Manganese	---	---	---	Nervous system	1.6	---	1.6
			Nickel	---	---	---	---	---	---	---
			Chemical Total	3.7E-05	---	3.7E-05		22	---	22
		Deep Bedrock Groundwater Total				3.7E-05				22
	Groundwater Total					3.7E-05				22
Groundwater Total						3.7E-05				22

Total Risk Across All Media: 3.7E-05

Total Hazard Across All Media: 22

APPENDIX E

TABLE E-101
RAGS PART D TABLE 10 - RISK SUMMARY - CHILD DEEP BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Deep Bedrock	Bis(2-Ethylhexyl)Phthalate	---	---	---	---	---	---	---
			Aluminum	---	---	---	---	---	---	---
			Arsenic	1.1E-05	---	1.1E-05	---	---	---	---
			Cadmium	---	---	---	---	---	---	---
			Chromium	---	---	---	---	---	---	---
		Groundwater	Cobalt	---	---	---	Thyroid	16	---	16
			Copper	---	---	---	Gastrointestinal	7.1	---	7.1
			Iron	---	---	---	---	---	---	---
			Manganese	---	---	---	Nervous system	1.9	---	1.9
			Nickel	---	---	---	---	---	---	---
			Chemical Total	1.1E-05	---	1.1E-05		25	---	25
		Deep Bedrock Groundwater Total				1.1E-05				25
	Groundwater Total					1.1E-05				25
Groundwater Total						1.1E-05				25

Total Risk Across All Media: 1.1E-05

Total Hazard Across All Media: 25

APPENDIX E

TABLE E-102
RAGS PART D TABLE 10 - RISK SUMMARY - ADULT DEEP BEDROCK GROUNDWATER
CENTRAL TENDENCY EXPOSURE
ELY COPPER MINE SUPERFUND SITE

Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical of Potential Concern	Carcinogenic Risk			Non-Carcinogenic Hazard Quotient			
				Ingestion	Dermal	Exposure Routes Total	Primary Target Organ(s)	Ingestion	Dermal	Exposure Routes Total
Groundwater	Groundwater	Deep Bedrock	Bis(2-Ethylhexyl)Phthalate	---	---	---	---	---	---	---
			Aluminum	---	---	---	---	---	---	---
			Arsenic	6.9E-06	---	6.9E-06	---	---	---	---
			Cadmium	---	---	---	---	---	---	---
			Chromium	---	---	---	---	---	---	---
		Groundwater	Cobalt	---	---	---	Thyroid	7.0	---	7.0
			Copper	---	---	---	Gastrointestinal	3.1	---	3.1
			Iron	---	---	---	---	---	---	---
			Manganese	---	---	---	---	---	---	---
			Nickel	---	---	---	---	---	---	---
			Chemical Total	6.9E-06	---	6.9E-06		10	---	10
		Deep Bedrock Groundwater Total				6.9E-06				10
	Groundwater Total					6.9E-06				10
Groundwater Total						6.9E-06				10

Total Risk Across All Media: 6.9E-06

Total Hazard Across All Media: 10

APPENDIX F

APPENDIX F

Table F-1
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM INCIDENTAL INGESTION OF SOIL
CURRENT/FUTURE ADOLESCENT RECREATIONAL VISITOR

PURPOSE: To estimate intake, cancer and non-cancer risks from incidental ingestion of COPCs in surface soil by the current/future adolescent recreational visitor for the Reasonable Maximum Exposure (RME).

EQUATION:

$$\text{Intake} = \frac{EPC \times IRS \times FI \times EF \times ED \times CF}{BW \times AT}$$

Where:

Intake	=	estimated exposure intake (mg/kg-day)
EPC	=	exposure point concentration in soil (mg/kg)
IRS	=	ingestion rate of soil (mg/day)
FI	=	fraction ingested from contaminated source (unitless)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
CF	=	conversion factor (kg/mg)
BW	=	body weight (kg)
AT	=	averaging time (days)
CSFo	=	oral carcinogenic slope factor ((mg/kg-day)$^{-1}$)
RfDo	=	oral noncarcinogenic reference dose (mg/kg-day)

RISKS:

ILCR (Carcinogens) = Intake (mg/kg-day) x CSFo ((mg/kg-day)$^{-1}$)
HQ (Noncarcinogens) = Intake (mg/kg-day) / RfDo (mg/kg-day)

ASSUMPTIONS:

EPC	=	2.7	mg/kg	Arsenic, Current/Future Surface Soil
IRS	=	100	mg/day	
FI	=	1	unitless	
EF	=	104	days/year	
ED	=	8	years	
CF	=	1.0E-06	kg/mg	
BW	=	52	kg	
ATc	=	25550	days	
ATnc	=	2920	days	
CSFo	=	1.50E+00	(mg/kg-day)$^{-1}$	
RfDo	=	3.00E-04	mg/kg-day	

APPENDIX F

Table F-1
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM INCIDENTAL INGESTION OF SOIL
CURRENT/FUTURE ADOLESCENT RECREATIONAL VISITOR

EXAMPLE CANCER CALCULATION

$$Intake_c = \frac{2.7 \text{ mg/kg} \times 100 \text{ mg/day} \times 1 \times 104 \text{ days/year} \times 8 \text{ years} \times 0.000001 \text{ kg/mg}}{52 \text{ kg} \times 25550 \text{ days}}$$

$Intake_c$ = 1.7E-07 mg/kg-day

ILCR = 1.7E-07 mg/kg-day x 1.5 (mg/kg-day)-1 = Incremental Lifetime Cancer Risk

ILCR = 2.6E-07

EXAMPLE NON-CANCER CALCULATION

$$Intake_{nc} = \frac{2.7 \text{ mg/kg} \times 100 \text{ mg/day} \times 1 \times 104 \text{ days/year} \times 8 \text{ years} \times 0.000001 \text{ kg/mg}}{52 \text{ kg} \times 2920 \text{ days}}$$

$Intake_{nc}$ = 1.5E-06 mg/kg-day

HQ = 1.5E-06 mg/kg-day / 0.0003 mg/kg-day = Hazard Quotient

HQ = 0.0050

Table F-2
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM DERMAL CONTACT WITH SOIL
CURRENT/FUTURE ADOLESCENT RECREATIONAL VISITOR

PURPOSE: To estimate intake, cancer and non-cancer risks from dermal contact with COPCs in surface soil by the current/future adolescent recreational visitor for the Reasonable Maximum Exposure (RME).

EQUATION:

$$\text{Intake} = \frac{\text{EPC} \times \text{SA} \times \text{AF} \times \text{ABS} \times \text{EF} \times \text{ED} \times \text{CF}}{\text{BW} \times \text{AT}}$$

Where:

Intake	=	estimated exposure intake (mg/kg-day)
EPC	=	exposure point concentration in soil (mg/kg)
SA	=	exposed skin surface area (cm^2/day)
AF	=	soil-to-skin adherence factor (mg/cm^2)
ABS	=	dermal absorption factor (unitless)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
CF	=	conversion factor (kg/mg)
BW	=	body weight (kg)
AT	=	averaging time (days)
CSFd	=	dermal carcinogenic slope factor ($(mg/kg\text{-}day)^{-1}$)
RfDd	=	dermal noncarcinogenic reference dose (mg/kg-day)

RISKS:

ILCR (Carcinogens) = Intake (mg/kg-day) x CSFd ($(mg/kg\text{-}day)^{-1}$)
HQ (Noncarcinogens) = Intake (mg/kg-day) / RfDd (mg/kg-day)

ASSUMPTIONS:

EPC	=	2.7	mg/kg	Arsenic, Current/Future Surface Soil
SA	=	5900	cm^2/day	
AF	=	0.2	mg/cm^2	
ABS	=	0.03	unitless	Arsenic value
EF	=	104	days/year	
ED	=	8	years	
CF	=	1.0E-06	kg/mg	
BW	=	52	kg	
ATc	=	25550	days	
ATnc	=	2920	days	
CSFd	=	1.50E+00	$(mg/kg\text{-}day)^{-1}$	
RfDd	=	3.00E-04	mg/kg-day	

APPENDIX F

Table F-2
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM DERMAL CONTACT WITH SOIL
CURRENT/FUTURE ADOLESCENT RECREATIONAL VISITOR

EXAMPLE CANCER CALCULATION

$$Intake_c = \frac{2.7 \text{ mg/kg} \times 5900 \text{ cm2/day} \times 0.2 \text{ mg/cm2} \times 0.03 \times 104 \text{ days/year} \times 8 \text{ years} \times 0.000001 \text{ kg/mg}}{52 \text{ kg} \times 25550 \text{ days}}$$

$Intake_c$ = 6.0E-08 mg/kg-day

ILCR = 6.0E-08 mg/kg-day x 1.5 (mg/kg-day)-1 = Incremental Lifetime Cancer Risk

ILCR = 9.1E-08

EXAMPLE NON-CANCER CALCULATION

$$Intake_{nc} = \frac{2.7 \text{ mg/kg} \times 5900 \text{ cm2/day} \times 0.2 \text{ mg/cm2} \times 0.03 \times 104 \text{ days/year} \times 8 \text{ years} \times 0.000001 \text{ kg/mg}}{52 \text{ kg} \times 2920 \text{ days}}$$

$Intake_{nc}$ = 5.3E-07 mg/kg-day

HQ = 5.3E-07 mg/kg-day / 0.0003 mg/kg-day = Hazard Quotient

HQ = 0.0018

APPENDIX F

Table F-3
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM INHALATION OF SOIL
CURRENT/FUTURE ADOLESCENT RECREATIONAL VISITOR

PURPOSE: To estimate intake, cancer and non-cancer risks from inhalation of COPCs from fugative dust and volatile emissions from surface soil by the current/future adolescent recreational visitor for the Reasonable Maximum Exposure (RME).

EQUATION:

$$EPCair = \frac{CA \times ET \times EF \times ED \times CF2}{AT}$$

Where:

EC = exposure concentration (µg/m^3)
CA = chemical concentration in air (µg/m^3)

$$CA = \frac{EPC}{PEF} \times CF3$$

EPC = exposure point concentration in soil (mg/kg)
PEF = particulate emission factor from soil (m^3/kg)
CF2 = conversion factor 2 (days/hour)
CF3 = conversion factor 3 (µg/mg)
ET = exposure time (hours/day)
EF = exposure frequency (days/year)
ED = exposure duration (years)
AT = averaging time (days)
URFi = inhalation unit risk factor ((µg/m^3)$^{-1}$)
RfC = inhalation noncarcinogenic reference concentration (mg/m^3)

RISKS:

ILCR (Carcinogens) = EC (µg/m^3) x URFi ((µg/m^3)$^{-1}$)
HQ (Noncarcinogens) = EC (µg/m^3) / RfC (mg/m^3) / 1000 (µg/mg)

ASSUMPTIONS:

CA	=	1.36E-03	µg/m^3	or	$\frac{2.725 \text{ mg/kg}}{2000000 \text{ m3/kg}}$ x 1000 µg/mg
EPC	=	2.7	mg/kg	Arsenic, Current/Future Surface Soil	
PEF	=	2.00E+06	m^3/kg		
CF2	=	0.042	days/hour		
CF3	=	1000	µg/mg		
ET	=	2	hours/day		
EF	=	104	days/year		
ED	=	8	years		
ATc	=	25550	days		
ATnc	=	2920	days		
URFi	=	4.30E-03	(µg/m^3)$^{-1}$		
RfC	=	1.50E-05	mg/m^3		

Table F-3
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM INHALATION OF SOIL
CURRENT/FUTURE ADOLESCENT RECREATIONAL VISITOR

EXAMPLE CARCINOGENIC CALCULATION

$$EC = \frac{1.36E\text{-}03\ \mu g/m3 \times 2\ hours/day \times 104\ days/year \times 8\ years \times 0.042\ days/hour}{25550\ days}$$

EC = 3.7E-06 $\mu g/m^3$

ILCR = 3.7E-06 µg/m3 x 0.0043 (µg/m3)-1 = Incremental Lifetime Cancer Risk

ILCR = 1.6E-08

EXAMPLE NON-CANCER CALCULATION

$$EC = \frac{1.36E\text{-}03\ \mu g/m3 \times 2\ hours/day \times 104\ days/year \times 8\ years \times 0.042\ days/hour}{2920\ days}$$

EC = 3.3E-05 $\mu g/m^3$

HQ = 3.3E-05 µg/m3 / 1000 µg/mg / 0.000015 mg/m3 = Hazard Quotient

HQ = 0.0022

Table F-4
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM INGESTION OF OVERBURDEN GROUNDWATER
CURRENT/FUTURE CHILD RESIDENT

PURPOSE: To estimate intake, cancer and non-cancer risks from ingestion of COPCs from overburden groundwater by the current/future child resident for the Reasonable Maximum Exposure (RME).

EQUATION:

$$\text{Intake} = \frac{\text{EPC} \times \text{IRW} \times \text{FI} \times \text{EF} \times \text{ED} \times \text{CF}}{\text{BW} \times \text{AT}}$$

Where:

Intake	=	estimated exposure intake (mg/kg-day)
EPC	=	exposure point concentration in water (µg/L)
IRW	=	water ingestion rate (L/day)
FI	=	fraction ingested from contaminated source (unitless)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
CF	=	conversion factor (mg/µg)
BW	=	body weight (kg)
AT	=	averaging time (days)
CSFo	=	oral carcinogenic slope factor ((mg/kg-day)$^{-1}$)
RfDo	=	oral noncarcinogenic reference dose (mg/kg-day)

RISKS:

ILCR (Carcinogens) = Intake (mg/kg-day) x CSFo ((mg/kg-day)$^{-1}$)
HQ (Noncarcinogens) = Intake (mg/kg-day) / RfDo (mg/kg-day)

ASSUMPTIONS:

EPC	=	5.8	µg/L	Arsenic, Current/Future Overburden Groundwater
IRW	=	1	L/day	
FI	=	1	unitless	
EF	=	350	days/year	
ED	=	6	years	
CF	=	1.0E-03	mg/µg	
BW	=	15	kg	
ATc	=	25550	days	
ATnc	=	2190	days	
CSFo	=	1.50E+00	(mg/kg-day)$^{-1}$	
RfDo	=	3.00E-04	mg/kg-day	

APPENDIX F

Table F-4
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM INGESTION OF OVERBURDEN GROUNDWATER
CURRENT/FUTURE CHILD RESIDENT

EXAMPLE CANCER CALCULATION

$$Intake_c = \frac{5.8\ \mu g/L \times 1\ L/day \times 1 \times 350\ days/year \times 6\ years \times 0.001\ mg/\mu g}{15\ kg \times 25550\ days}$$

$Intake_c$ = 3.2E-05 mg/kg-day

ILCR = 3.2E-05 mg/kg-day x 1.5 (mg/kg-day)-1 = Incremental Lifetime Cancer Risk

ILCR = 4.8E-05

EXAMPLE NON-CANCER CALCULATION

$$Intake_{nc} = \frac{5.8\ \mu g/L \times 1\ L/day \times 1 \times 350\ days/year \times 6\ years \times 0.001\ mg/\mu g}{15\ kg \times 2190\ days}$$

$Intake_{nc}$ = 3.7E-04 mg/kg-day

HQ = 3.7E-04 mg/kg-day / 0.0003 mg/kg-day = Hazard Quotient

HQ = 1.2

APPENDIX F

Table F-5
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM DERMAL CONTACT WITH OVERBURDEN GROUNDWATER
CURRENT/FUTURE CHILD RESIDENT

PURPOSE: To estimate intake, cancer and non-cancer risks from dermal contact with COPCs from overburden groundwater by the current/future child resident for the Reasonable Maximum Exposure (RME).

EQUATION:

$$Intake = \frac{EPC \times Kp \times t_{event} \times SA \times EV \times EF \times ED \times CF}{BW \times AT}$$

Where:

Intake	=	estimated exposure intake (mg/kg-day)
EPC	=	exposure point concentration in water (µg/L)
Kp	=	dermal permeability coefficient (cm/hour)
t_{event}	=	event duration (hour/event)
SA	=	exposed skin surface area (cm^2/day)
EV	=	event frequency (event/day)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
CF	=	conversion factor (mg-L/µg-cm^3)
BW	=	body weight (kg)
AT	=	averaging time (days)
CSFd	=	dermal carcinogenic slope factor ((mg/kg-day)$^{-1}$)
RfDd	=	dermal noncarcinogenic reference dose (mg/kg-day)

RISKS:

ILCR (Carcinogens) = Intake (mg/kg-day) x CSFd ((mg/kg-day)$^{-1}$)
HQ (Noncarcinogens) = Intake (mg/kg-day) / RfDd (mg/kg-day)

ASSUMPTIONS:

EPC	=	5.8	µg/L	Arsenic, Current/Future Overburden Groundwater
Kp	=	0.001	cm/hour	Arsenic value
t_{event}	=	1	hour/event	
SA	=	6600	cm^2/day	
EV	=	1	event/day	
EF	=	350	days/year	
ED	=	6	years	
CF	=	1.0E-06	mg-L/µg-cm^3	
BW	=	15	kg	
ATc	=	25550	days	
ATnc	=	2190	days	
CSFd	=	1.50E+00	(mg/kg-day)$^{-1}$	
RfDd	=	3.00E-04	mg/kg-day	

Table F-5
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM DERMAL CONTACT WITH OVERBURDEN GROUNDWATER
CURRENT/FUTURE CHILD RESIDENT

EXAMPLE CANCER CALCULATION

$$Intake_c = \frac{5.8\ \mu g/L \times 0.001\ cm/hour \times 1\ hour/event \times 6600\ cm2/day \times 1\ event/day \times 350\ days/year \times 6\ years \times 0.000001 mg\text{-}L/\mu g\text{-}cm3}{15\ kg \times 25550\ days}$$

$Intake_c$ = 2.1E-07 mg/kg-day

ILCR = 2.1E-07 mg/kg-day x 1.5 (mg/kg-day)-1 = Incremental Lifetime Cancer Risk

ILCR = 3.2E-07

EXAMPLE NON-CANCER CALCULATION

$$Intake_{nc} = \frac{5.8\ \mu g/L \times 0.001\ cm/hour \times 1\ hour/event \times 6600\ cm2/day \times 1\ event/day \times 350\ days/year \times 6\ years \times 0.000001 mg\text{-}L/\mu g\text{-}cm3}{15\ kg \times 2190\ days}$$

$Intake_{nc}$ = 2.5E-06 mg/kg-day

HQ = 2.5E-06 mg/kg-day / 0.0003 mg/kg-day = Hazard Quotient

HQ = 0.0082

Table F-6
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM INCIDENTAL INGESTION OF ELY BROOK SEDIMENT
CURRENT/FUTURE ADULT SWIMMER/WADER

PURPOSE: To estimate intake, cancer and non-cancer risks from incidental ingestion of COPCs in Ely Brook sediment by the current/future adult swimmer/wader for the Reasonable Maximum Exposure (RME).

EQUATION:

$$\text{Intake} = \frac{EPC \times IRSED \times FI \times EF \times ED \times CF}{BW \times AT}$$

Where:

Intake	=	estimated exposure intake (mg/kg-day)
EPC	=	exposure point concentration in sediment (mg/kg)
IRSED	=	incidental sediment ingestion rate (mg/day)
FI	=	fraction ingested from contaminated source (unitless)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
CF	=	conversion factor (kg/mg)
BW	=	body weight (kg)
AT	=	averaging time (days)
CSFo	=	oral carcinogenic slope factor ((mg/kg-day)$^{-1}$)
RfDo	=	oral noncarcinogenic reference dose (mg/kg-day)

RISKS:

ILCR (Carcinogens) = Intake (mg/kg-day) x CSFo ((mg/kg-day)$^{-1}$)
HQ (Noncarcinogens) = Intake (mg/kg-day) / RfDo (mg/kg-day)

ASSUMPTIONS:

EPC	=	3.7	mg/kg	Arsenic, Current/Future Ely Brook Sediment
IRSED	=	100	mg/day	
FI	=	1	unitless	
EF	=	22	days/year	
ED	=	24	years	
CF	=	1.0E-06	kg/mg	
BW	=	70	kg	
ATc	=	25550	days	
ATnc	=	8760	days	
CSFo	=	1.50E+00	(mg/kg-day)$^{-1}$	
RfDo	=	3.00E-04	mg/kg-day	

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Table F-6
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont
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SUBJECT:
CALCULATION OF INTAKE/RISK FROM INCIDENTAL INGESTION OF ELY BROOK SEDIMENT
CURRENT/FUTURE ADULT SWIMMER/WADER

EXAMPLE CANCER CALCULATION

$$Intake_c = \frac{3.7\ mg/kg \times 100\ mg/day \times 1 \times 22\ days/year \times 24\ years \times 0.000001\ kg/mg}{70\ kg \times 25550\ days}$$

$Intake_c$ = 1.1E-07 mg/kg-day

ILCR = 1.1E-07 mg/kg-day x 1.5 (mg/kg-day)-1 = Incremental Lifetime Cancer Risk

ILCR = 1.6E-07

EXAMPLE NON-CANCER CALCULATION

$$Intake_{nc} = \frac{3.7\ mg/kg \times 100\ mg/day \times 1 \times 22\ days/year \times 24\ years \times 0.000001\ kg/mg}{70\ kg \times 8760\ days}$$

$Intake_{nc}$ = 3.2E-07 mg/kg-day

HQ = 3.2E-07 mg/kg-day / 0.0003 mg/kg-day = Hazard Quotient

HQ = 0.0011

APPENDIX F

Table F-7
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM DERMAL CONTACT WITH ELY BROOK SEDIMENT
CURRENT/FUTURE ADULT SWIMMER/WADER

PURPOSE: To estimate intake, cancer and non-cancer risks from dermal contact with COPCs in Ely Brook sediment by the current/future adult swimmer/wader for the Reasonable Maximum Exposure (RME).

EQUATION:

$$\text{Intake} = \frac{\text{EPC} \times \text{SA} \times \text{AF} \times \text{ABS} \times \text{EF} \times \text{ED} \times \text{CF}}{\text{BW} \times \text{AT}}$$

Where:

Intake	=	estimated exposure intake (mg/kg-day)
EPC	=	exposure point concentration in sediment (mg/kg)
SA	=	exposed skin surface area (cm^2/day)
AF	=	sediment-to-skin adherence factor (mg/cm^2)
ABS	=	dermal absorption factor (unitless)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
CF	=	conversion factor (kg/mg)
BW	=	body weight (kg)
AT	=	averaging time (days)
CSFd	=	dermal carcinogenic slope factor ($(mg/kg\text{-}day)^{-1}$)
RfDd	=	dermal noncarcinogenic reference dose (mg/kg-day)

RISKS:

ILCR (Carcinogens) = Intake (mg/kg-day) x CSFd ($(mg/kg\text{-}day)^{-1}$)
HQ (Noncarcinogens) = Intake (mg/kg-day) / RfDd (mg/kg-day)

ASSUMPTIONS:

EPC	=	3.7	mg/kg	Arsenic, Current/Future Ely Brook Sediment
SA	=	6900	cm^2/day	
AF	=	0.32	mg/cm^2	
ABS	=	0.03	unitless	Arsenic value
EF	=	22	days/year	
ED	=	24	years	
CF	=	1.0E-06	kg/mg	
BW	=	70	kg	
ATc	=	25550	days	
ATnc	=	8760	days	
CSFd	=	1.50E+00	$(mg/kg\text{-}day)^{-1}$	
RfDd	=	3.00E-04	mg/kg-day	

Table F-7
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM DERMAL CONTACT WITH ELY BROOK SEDIMENT
CURRENT/FUTURE ADULT SWIMMER/WADER

EXAMPLE CANCER CALCULATION

$$Intake_c = \frac{3.7\ mg/kg \times 6900\ cm2/day \times 0.32\ mg/cm2 \times 0.03 \times 22\ days/year \times 24\ years \times 0.000001\ kg/mg}{70\ kg \times 25550\ days}$$

$Intake_c$ = 7.2E-08 mg/kg-day

ILCR = 7.2E-08 mg/kg-day x 1.5 (mg/kg-day)-1 = Incremental Lifetime Cancer Risk

ILCR = 1.1E-07

EXAMPLE NON-CANCER CALCULATION

$$Intake_{nc} = \frac{3.7\ mg/kg \times 6900\ cm2/day \times 0.32\ mg/cm2 \times 0.03 \times 22\ days/year \times 24\ years \times 0.000001\ kg/mg}{70\ kg \times 8760\ days}$$

$Intake_{nc}$ = 2.1E-07 mg/kg-day

HQ = 2.1E-07 mg/kg-day / 0.0003 mg/kg-day = Hazard Quotient

HQ = 0.00070

Table F-8
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM INCIDENTAL INGESTION OF ELY BROOK SURFACE WATER
CURRENT/FUTURE ADULT SWIMMER/WADER

PURPOSE: To estimate intake, cancer and non-cancer risks from ingestion of COPCs from Ely Brook surface water by the current/future adult swimmer/wader for the Reasonable Maximum Exposure (RME).

EQUATION:

$$\text{Intake} = \frac{\text{EPC} \times \text{IRW} \times \text{FI} \times \text{ET} \times \text{EF} \times \text{ED} \times \text{CF}}{\text{BW} \times \text{AT}}$$

Where:

Intake	=	estimated exposure intake (mg/kg-day)
EPC	=	exposure point concentration in water (µg/L)
IRW	=	incidental water ingestion rate (L/hr)
FI	=	fraction ingested from contaminated source (unitless)
ET	=	exposure time (hours/day)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
CF	=	conversion factor (mg/µg)
BW	=	body weight (kg)
AT	=	averaging time (days)
CSFo	=	oral carcinogenic slope factor ((mg/kg-day)$^{-1}$)
RfDo	=	oral noncarcinogenic reference dose (mg/kg-day)

RISKS:

ILCR (Carcinogens) = Intake (mg/kg-day) x CSFo ((mg/kg-day)$^{-1}$)
HQ (Noncarcinogens) = Intake (mg/kg-day) / RfDo (mg/kg-day)

ASSUMPTIONS:

EPC	=	0.25	µg/L	Arsenic, Current/Future Ely Brook Surface Water
IRW	=	0.05	L/hr	
FI	=	1	unitless	
ET	=	2	hours/day	
EF	=	22	days/year	
ED	=	24	years	
CF	=	1.0E-03	mg/µg	
BW	=	70	kg	
ATc	=	25550	days	
ATnc	=	8760	days	
CSFo	=	1.50E+00	(mg/kg-day)$^{-1}$	
RfDo	=	3.00E-04	mg/kg-day	

APPENDIX F

Table F-8
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM INCIDENTAL INGESTION OF ELY BROOK SURFACE WATER
CURRENT/FUTURE ADULT SWIMMER/WADER

EXAMPLE CANCER CALCULATION

$$Intake_c = \frac{0.25\ \mu g/L \times 0.05\ L/hr \times 1 \times 2\ hours/day \times 22\ days/year \times 24\ years \times 0.001\ mg/\mu g}{70\ kg \times 25550\ days}$$

$Intake_c$ = 7.3E-09 mg/kg-day

ILCR = 7.3E-09 mg/kg-day x 1.5 (mg/kg-day)-1 = Incremental Lifetime Cancer Risk

ILCR = 1.1E-08

EXAMPLE NON-CANCER CALCULATION

$$Intake_{nc} = \frac{0.25\ \mu g/L \times 0.05\ L/hr \times 1 \times 2\ hours/day \times 22\ days/year \times 24\ years \times 0.001\ mg/\mu g}{70\ kg \times 8760\ days}$$

$Intake_{nc}$ = 2.1E-08 mg/kg-day

HQ = 2.1E-08 mg/kg-day / 0.0003 mg/kg-day = Hazard Quotient

HQ = 0.000071

Table F-9
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM DERMAL CONTACT WITH ELY BROOK SURFACE WATER
CURRENT/FUTURE ADULT SWIMMER/WADER

PURPOSE: To estimate intake, cancer and non-cancer risks from dermal contact with COPCs from Ely Brook surface water by the current/future adult swimmer/wader for the Reasonable Maximum Exposure (RME).

EQUATION:

$$Intake = \frac{EPC \times Kp \times t_{event} \times SA \times EV \times EF \times ED \times CF}{BW \times AT}$$

Where:

Intake	=	estimated exposure intake (mg/kg-day)
EPC	=	exposure point concentration in water (µg/L)
Kp	=	dermal permeability coefficient (cm/hour)
t_{event}	=	event duration (hour/event)
SA	=	exposed skin surface area (cm^2/day)
EV	=	event frequency (event/day)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
CF	=	conversion factor (mg-L/µg-cm^3)
BW	=	body weight (kg)
AT	=	averaging time (days)
CSFd	=	dermal carcinogenic slope factor ((mg/kg-day)$^{-1}$)
RfDd	=	dermal noncarcinogenic reference dose (mg/kg-day)

RISKS:

ILCR (Carcinogens) = Intake (mg/kg-day) x CSFd ((mg/kg-day)$^{-1}$)
HQ (Noncarcinogens) = Intake (mg/kg-day) / RfDd (mg/kg-day)

ASSUMPTIONS:

EPC	=	0.25	µg/L	Arsenic, Current/Future Ely Brook Surface Water
Kp	=	0.001	cm/hour	Arsenic value
t_{event}	=	2	hour/event	
SA	=	18000	cm^2/day	
EV	=	1	event/day	
EF	=	22	days/year	
ED	=	24	years	
CF	=	1.0E-06	mg-L/µg-cm^3	
BW	=	70	kg	
ATc	=	25550	days	
ATnc	=	8760	days	
CSFd	=	1.50E+00	(mg/kg-day)$^{-1}$	
RfDd	=	3.00E-04	mg/kg-day	

APPENDIX F

Table F-9
Sample Calculations
Ely Copper Mine Superfund Site
Vershire, Vermont

SUBJECT:
CALCULATION OF INTAKE/RISK FROM DERMAL CONTACT WITH ELY BROOK SURFACE WATER
CURRENT/FUTURE ADULT SWIMMER/WADER

EXAMPLE CANCER CALCULATION

$$Intake_c = \frac{0.25\ \mu g/L \times 0.001\ cm/hour \times 2\ hour/event \times 18000\ cm2/day \times 1\ event/day \times 22\ days/year \times 24\ years \times 0.000001 mg\text{-}L/\mu g\text{-}cm3}{70\ kg \times 25550\ days}$$

$Intake_c$ = 2.6E-09 mg/kg-day

ILCR = 2.6E-09 mg/kg-day x 1.5 (mg/kg-day)-1 = Incremental Lifetime Cancer Risk

ILCR = 3.9E-09

EXAMPLE NON-CANCER CALCULATION

$$Intake_{nc} = \frac{0.25\ \mu g/L \times 0.001\ cm/hour \times 2\ hour/event \times 18000\ cm2/day \times 1\ event/day \times 22\ days/year \times 24\ years \times 0.000001 mg\text{-}L/\mu g\text{-}cm3}{70\ kg \times 8760\ days}$$

$Intake_{nc}$ = 7.6E-09 mg/kg-day

HQ = 7.6E-09 mg/kg-day / 0.0003 mg/kg-day = Hazard Quotient

HQ = 0.000025