INTERTOX

RICHARD C. PLEUS, PHD, EXPERT REPORT:

CRITIQUE OF THE *FINAL BASELINE HUMAN HEALTH RISK ASSESSMENT FOR THE ANACONDA SMELTER NPL SITE, ANACONDA, MONTANA* (CDM, 1996) AND REASSESSMENT OF SOIL SCREENING LEVELS FOR THE OPPORTUNITY COMMUNITY

Regarding

Montana Second Judicial District Court, Silver Bow County
Christian et al. v. BP Amoco Corp., et al.,
Cause No. DV-08-173 BN

Signature _____

April 12, 2013

INTERTOX, INC.
600 Stewart St.
Suite 1101
Seattle, WA 98101

206.443.2115 phone
206.443.2117 facsimile

TABLE OF CONTENTS

EXECUTIVE SUMMARY iv
1.0 INTRODUCTION 1
2.0 SCIENTIFIC REVIEW OF THE BASELINE HUMAN HEALTH RISK ASSESSMENT FOR THE ANACONDA SMELTER NPL SITE 2
2.1 Soil Screening Level Calculations 2
2.2 Review of Key Exposure Parameters and Risk Assumptions 5
2.2.1 Soil Ingestion Rate (IR) 5
2.2.2 Fraction Ingested that is Soil (FS) and Dust (FD) 6
2.2.3 Relative Concentration of Arsenic in Dust ($CR_{soil\text{-}dust}$) 7
2.2.4 Oral Bioavailability Factor for Soil and Dust (BAF_{soil} and BAF_{dust}) 8
2.2.5 Exposure Duration (ED_a and ED_c) 23
2.2.6 Toxicity Criteria 23
2.3 Exposure Pathways Not Considered 23
3.0 RECALCULATION OF SITE-RELATED RISKS AND SOIL SCREENING LEVELS 25
3.1 Quantification of Exposure 25
3.1.1 Estimation of Exposure Point Concentrations 26
3.1.2 Additional Complete Exposure Pathways 27
3.1.3 Revised Exposure Assumptions for Soil and Dust Ingestion Pathways 39
3.2 Recalculation of Site-Related Risks 40
3.3 Recalculation of the Soil Screening Level 42
4.0 COMPARISON TO CLEANUP LEVELS AT OTHER SITES 43
5.0 IMPACT OF ALTERNATIVE EXPOSURE ASSUMPTIONS 53
5.1 Relative Oral Bioavailability (RBA) Factors 53
5.2 Relative Concentration of Arsenic in Dust 54
5.3 Soil + Dust Ingestion Rates 54
5.4 Produce Consumption Rates 55
5.5 Meat Consumption Rates 56
6.0 SUMMARY AND CONCLUSIONS 56
7.0 REFERENCES 58

ED_001802_00024760-00002

LIST OF APPENDICES

APPENDIX A. Curriculum Vitae of Richard C. Pleus

LIST OF TABLES

TABLE 1. ACTION LEVELS FOR ARSENIC IN SURFACE SOIL AND WASTE IN THE ARWW&S OU (U.S. EPA, 1998A) 1
TABLE 2. RISK-BASED SCREENING LEVELS FOR ARSENIC IN SOIL CALCULATED FOR THE RESIDENTIAL EXPOSURE SCENARIO IN THE ANACONDA SMELTER NPL SITE BASELINE HHRA (CDM, 1996) ... 3
TABLE 3. EXPOSURE PARAMETERS USED TO CALCULATE ARSENIC SOIL SCREENING LEVELS FOR THE RESIDENTIAL SCENARIO IN THE ANACONDA SMELTER BASELINE HHRA (CDM, 1996) 4
TABLE 4. SUMMARY OF RELATIVE ORAL BIOAVAILABILITY ESTIMATES FOR ARSENIC IN SOIL IMPACTED BY MINING OR SMELTING ACTIVITIES 18
TABLE 5. SUMMARY OF SOIL AND GROUNDWATER SAMPLING DATA FOR ARSENIC COLLECTED IN THE OPPORTUNITY COMMUNITY IN JUNE 2012 26
TABLE 6. EXPOSURE POINT CONCENTRATIONS (EPCS) USED IN THE REVISED HHRA FOR THE OPPORTUNITY, MONTANA COMMUNITY 27
TABLE 7. EXPOSURE PARAMETERS USED IN THE RISK CALCULATIONS FOR THE RESIDENTIAL SCENARIO AT THE OPPORTUNITY, MT SITE 29
TABLE 8. SUMMARY OF PATHWAY-SPECIFIC LIFETIME EXCESS CANCER RISKS FOR OPPORTUNITY COMMUNITY RESIDENTS, RME SCENARIO 41
TABLE 9. SOIL CLEANUP LEVELS FOR ARSENIC IN SOIL FOR RESIDENTIAL/ UNRESTRICTED USE FOR U.S. STATES[A] 43
TABLE 10. ACTION LEVELS FOR ARSENIC IN SOIL AT SELECTED U.S. SITES 45

ED_001802_00024760-00003

EXECUTIVE SUMMARY

I have been asked by attorneys (Beck & Amsden, PLLC, of Bozeman, MT) representing a group of citizens in Opportunity, MT to assess the U.S. EPA risk assessment used to derive soil action levels for arsenic, with particular focus on the residential clean-up action level of 250 parts per million (ppm, equivalent to milligrams/kilogram or mg/kg) soil. Arsenic and other chemicals were deposited on the land from the operation of a local smelter. The particular point in question is whether the clean-up level for arsenic in residential soil was developed in a manner consistent with U.S. EPA risk assessment practices.

Montana's Anaconda Regional Water, Waste, and Soils (ARWW&S) Operable Unit (OU) covers approximately 300 square miles in the southern Deer Lodge Valley and its surrounding foothills, and includes the Opportunity Ponds, North Opportunity, South Opportunity, Old Works/ Stucky Ridge, and Smelter Hill subareas. The North Opportunity and South Opportunity subareas in particular include a mixture of rural/residential, agricultural, and open space/ recreational uses. The U.S. EPA Superfund Record of Decision (ROD) for the Anaconda Company Smelter in Anaconda, MT set action levels for arsenic in soils at the ARWW&S OU that vary according to land-use designation. The action levels range from 250 ppm (residential) to 2,500 ppm (open space).

The ROD action levels were selected in part according to the risk-based screening levels presented in the *Final Baseline Human Health Risk Assessment, Anaconda Smelter NPL Site, Anaconda, MT,* (Baseline HHRA) prepared by CDM (1996) for U.S. EPA, which calculated human health risk-based screening levels for various receptors, including residents. As such, I conducted a review of the Baseline HHRA. I note in this report a number of problems with the Baseline HHRA, including omissions of exposure pathways and incorrect calculation of the bioavailability factor. Other concerns include the experimental design of the monkey study used to determine the bioavailability factor.

A site-specific risk assessment for arsenic was performed that included relevant and defensible exposure pathways and corrected mistakes in the Baseline HHRA where applicable. The risk assessment was conducted in a manner consistent with standard U.S. EPA guidance, and incorporates site-specific exposure information, including the concentration of arsenic in dust relative to soil and information from community residents that indicates they consume, or have consumed, homegrown produce, and in some cases have raised livestock for consumption.

This risk assessment yields an estimated arsenic cancer risk of 2.5×10^{-4}, which is 4.5-fold higher than the risk of 5.51×10^{-5} estimated for the Opportunity resident Reasonable Maximum Exposure (RME) scenario in the Baseline HHRA. If the ROD action level of 250 ppm is used with the equations and assumptions applied in this assessment, the estimated lifetime excess cancer risk for an Opportunity resident would be 3.6×10^{-4}, which is 4.3-fold higher than the risk of 8.4×10^{-5} that the Baseline HHRA and the ROD assumes is associated with this action level. These risks are greater than U.S. EPA's general acceptable risk threshold of 1×10^{-4}. These risk estimates are solely arsenic based; other chemical exposures, not included here, would increase the risk estimate.

The soil screening level calculated in the current report, based on an acceptable lifetime excess cancer risk level of 1×10^{-5}, is approximately 8 ppm, compared to the ROD action level of 250 ppm. This screening level is appropriate and consistent with other arsenic soil action levels established nationwide. The U.S. EPA ROD action level of 250 ppm is one of the highest levels for arsenic in the U.S. EPA RODs nationwide.

ED_001802_00024760-00004

Regarding our risk calculations, where we could not obtain better scientific data, we used the same parameters as the Baseline HHRA conducted in 1996. For example, while I believe for several reasons that the site-specific monkey study underestimates bioavailability, we used values from that study in this assessment. If the bioavailability was in fact higherthan indicated by the monkey study, the risk estimated here would be greater than 2.5×10^{-4}; as such, my assessment is conservative and if an alternative estimate of relative bioavailability were used, the estimated risks would be calculated to be higher.

My review of the U.S. EPA Superfund ROD for the Anaconda Company Smelter in Anaconda, MT (U.S. EPA, 1998a), the *Final Baseline Human Health Risk Assessment, Anaconda Smelter NPL Site, Anaconda, MT* (CDM, 1996) has found that their arsenic risk estimate and residential action level of 250 ppm is not appropriate. According to the standard practice of toxicological risk assessment, the documents' calculations contain a number of errors and omissions. I further note that, when using a standard risk assessment approach, the 250 ppm figure presents a greater estimate of cancer risk than the documents indicate. Using current data and practices, I have found a scientifically reliable soil screening level to be approximately 8 ppm. This is assessed on a more probable than not basis.

ED_001802_00024760-00005

1.0 INTRODUCTION

I have been asked by attorneys (Beck & Amsden, PLLC, of Bozeman, MT) representing a group of citizens in Opportunity, MT to assess the U.S. EPA risk assessment that was the basis for soil action levels for arsenic at the Anaconda Regional Water, Waste, and Soils (ARWW&S) Operable Unit (OU) of the Anaconda Company Smelter National Priority List (NPL) site, with particular focus on the residential soil action level of 250 parts per million (ppm, equivalent to milligrams arsenic /kilogram soil or mg arsenic/kg soil). Arsenic and other chemicals were deposited on the land from the operation of the smelter. I have been asked whether the action level for residential soil was developed in a manner that reflects valid methodologies for predicting exposure and risk, and whether the action levels are appropriately health protective according to U.S. EPA risk assessment practices. I reviewed in more detail the assumptions and results of the Baseline HHRA for the Anaconda Smelter NPL Site and the soil screening levels. I was assisted by scientists in my office, who I directed to conduct specific tasks, including research and calculations. In conducting the evaluation, we relied upon current risk assessment practices, knowledge of arsenic behavior, fate, and toxicity, site-specific information, and comparison to other sites. I base the opinion stated on my education, training, and experience, research I have conducted on similar smelters, and research I have performed specific to this project.

Montana's Anaconda Regional Water, Waste, and Soils (ARWW&S) Operable Unit (OU) covers approximately 300 square miles in the southern Deer Lodge Valley and its surrounding foothills, and includes the Opportunity Ponds, North Opportunity, South Opportunity, Old Works/ Stucky Ridge, and Smelter Hill subareas. The North Opportunity and South Opportunity subareas in particular include a mixture of rural/residential, agricultural, and open space/ recreational uses. The U.S. EPA Superfund Record of Decision (ROD) for the Anaconda Company Smelter in Anaconda, MT (U.S. EPA, 1998a) set action levels for arsenic in soils at the ARWW&S OU according to land-use designation. Table 1 summarizes the action levels for arsenic in surface soil and waste reported in the ROD; the action levels range from 250 to 2,500 ppm. The lowest level action levels are for residential soil and the highest are for steep slopes and open spaces.

Table 1. Action Levels for Arsenic in Surface Soil and Waste in the ARWW&S OU (U.S. EPA, 1998a)

Land Use Designation	Media	Concentration (ppm)	Risk
Residential	Soil and Waste	250	8×10^{-5}
Commercial/Industrial	Soil and Waste	500	4×10^{-5}
Recreational	Soil and Waste	1,000	4×10^{-5}
Agricultural	Soil only	1,000	1×10^{-4}
Steep Slope/ Open Space	Soil only	2,500	1×10^{-5}

Per the ROD, these action levels were selected in part based on the risk-based screening levels presented in the *Final Baseline Human Health Risk Assessment, Anaconda Smelter NPL Site, Anaconda, MT*, prepared by CDM (1996) for the U.S. EPA, which calculated human health risk-based screening levels for residents, commercial/ industrial workers, agricultural workers, and dirt

ED_001802_00024760-00006

bike riders exposed to soils within the Community Soils OU located within the ARWW&S OU geographic area. As described in the ROD (p. 28):

> Because the Community Soils OU is located within the ARWW&S OU geographic area and shares one of the primary sources of contamination (i.e., soils contaminated by deposition of historical aerial emissions from the smelter), the risk-based screening levels presented in the Anaconda Smelter Site HHRA [Human Health Risk Assessment] are applicable to soils of the ARWW&S OU contaminated by historical smelter emissions.

The ROD describes the source of contamination to the local soils as follows (p. 28):

> The two primary sources of contamination within the ARWW&S OU are soils impacted by historic air emissions from the Old Works and Anaconda Smelter stacks, and tailings and other wastes remaining from the smelting processes. Historical smelting activities resulted in widespread, aerial deposition of fugitive dusts and contaminants released from stacks, resulting in contamination of soils in the ARWW&S OU. Materials released from the smelter stacks were small particulates not captured by emission controls in place…

The ROD further states (p. 29):

> …The primary release mechanism for tailings and slag is wind erosion, although release to ground water via infiltration/percolation and to soils and surface water via runoff also occurs. Contamination in air emissions is transported via dry or wet deposition from the air into three secondary sources: soil, surface water, and sediment. Transport of contaminants also occurs among secondary sources.

Regarding the level of cancer risk that the Agency deemed acceptable, the ROD (p. 31-32) reports that "EPA has developed action levels for surface soil and wastes for the targeted cancer risk range of 1E-04 to 1E-06." Regarding specific action levels, the criteria they used to select the levels are not defined, described as "based on technical and risk management considerations."

The results, briefly found that the Baseline HHRA contains errors or considerations not accounted for and/or assumptions that have not been updated according to scientific and site-related knowledge. Based on these findings, and our knowledge of the site, I evaluated potential human health risks to residents of the Opportunity, MT community associated with arsenic concentrations in soil, dust, and other media, and recalculated residential soil screening levels. We compared these screening levels to action levels established by U.S. EPA at other sites, and found that the action levels set in the ROD are high relative to these sites.

2.0 SCIENTIFIC REVIEW OF THE BASELINE HUMAN HEALTH RISK ASSESSMENT FOR THE ANACONDA SMELTER NPL SITE

In Sections 2.1, 2.2, and 2.3, we describe and comment on the assumptions used to calculate these screening levels.

2.1 Soil Screening Level Calculations

The action level established for arsenic in residential soils within the ARWW&S OU is based on the risk-based soil screening levels calculated in the Baseline HHRA (CDM, 1996). U.S. EPA currently describes arsenic as a Group A human carcinogen (IRIS, 2013); it was classified the same in 1996. The expression of risk for cancer causing agents is a risk level of "N" in a million ("N" $\times 10^{-6}$),

which implies a likelihood that up to "N" people, out of one million equally exposed, would develop cancer if exposed to the given concentration at the assumed exposure level over 70 years (an assumed lifetime). This risk estimate is an upper bound excess cancer risk that is in addition to any cancer risk borne by the person if they were not exposed to this agent. A risk of 1×10^{-5} is equal to 1 in 100,000 and 1×10^{-4} is equal to 1 in 10,000.

The Baseline HHRA calculates a range of "risk-based screening levels" for arsenic in soil for the reasonable maximum exposure (RME) scenario (which represents "an exposure well above the average but still within the range of those possible;" CDM, 1996) and central tendency exposure (CTE) scenario (which "uses exposure assumptions that predict an average or best estimate exposure to an individual;" CDM, 1996), assuming residential exposure. Per U.S. EPA's *Superfund Standard Default Exposure Factors for the Central Tendency and Reasonable Maximum Exposure* (U.S. EPA, 1993a), which is cited by CDM (1996) as the source of default exposure parameter assumptions used in the Baseline HHRA, the RME "can be equated to about the 90th percentile of the population distribution," and is defined as "the highest exposure that is reasonably expected to occur at a site and in practice is estimated by combining upper bound (90-95th percentile) values for some but not all exposure parameters."

Arsenic soil screening levels calculated in the Baseline HHRA using different acceptable lifetime excess cancer risk assumptions for the RME and CTE residential scenarios are shown in Table 2.

Table 2. Risk-Based Screening Levels for Arsenic in Soil Calculated for the Residential Exposure Scenario in the Anaconda Smelter NPL Site Baseline HHRA (CDM, 1996)

Excess Carcinogenic Risk Risk in 70 Years	Reasonable Maximum Exposure (RME) Scenario (ppm)	Central Tendency Exposure (CTE) Scenario (ppm)
1×10^{-6}	2.97	18.5
1×10^{-5}	29.7	185.2
1×10^{-4}	297	1,852

The action level of 250 ppm for the residential land use designation cited in the ROD equates to a lifetime excess cancer risk of approximately 8.4×10^{-5} for the RME scenario (i.e., 8.4 in 100,000), or nearly 1 in 10,000, based on these assumptions.

The screening levels were derived for the residential scenario based on a resident exposed to arsenic by only two exposure pathways, soil ingestion and ingestion of indoor dust, as follows (CDM, 1996):

$$SL_{soil}\ (mg/kg, or\ ppm) = \frac{TR \times AT}{CF \times EF \times SF_o \times \left(\frac{IRc \times EDc}{BWc} + \frac{IRa \times EDa}{BWa}\right) \times \left[(FS \times BAF_{soil}) + (CR_{soil-dust} \times FD \times BAF_{dust})\right]}$$

Where:

SL_{soil}	=	Screening level for soil (mg/kg soil, or ppm)
TR	=	Target cancer risk (unitless)
AT	=	Averaging time (d)
CF	=	Conversion factor (0.001 kg/mg)
EF	=	Exposure frequency (d/yr)
SF_o	=	Oral cancer slope factor $(mg/kg\text{-}d)^{-1}$

IR_c	=	Soil ingestion rate by a child (mg/d)
ED_c	=	Exposure duration for a child (yr)
BW_c	=	Body weight for a child (kg)
IR_a	=	Soil ingestion rate by an adult (mg/d)
ED_a	=	Exposure duration for an adult (yr)
BW_a	=	Body weight for an adult (kg)
FS	=	Fraction ingested that is soil (unitless)
BAF_{soil}	=	Oral bioavailability of arsenic in soil (unitless)
$CR_{soil\text{-}dust}$	=	Relative concentration of arsenic in dust (unitless)
FD	=	Fraction ingested that is dust (unitless)
BAF_{dust}	=	Oral bioavailability of arsenic in dust (unitless)

Numerical values for the parameters used to calculate the soil screening levels are shown in Table 3.

Table 3. Exposure Parameters Used to Calculate Arsenic Soil Screening Levels for the Residential Scenario in the Anaconda Smelter Baseline HHRA (CDM, 1996)

Exposure Parameter	Value for the RME Scenario	Value for the CTE Scenario
AT	25,550 d	25,550 d
EF	350 d/yr	350 d/yr
SF_o	1.5 $(mg/kg\text{-}d)^{-1}$	1.5 $(mg/kg\text{-}d)^{-1}$
IR_c	200 mg/d	100 mg/d
ED_c	6 yr	2 yr
BW_c	15 kg	15 kg
IR_a	100 mg/d	50 mg/d
ED_a	24 yr	7 yr
BW_a	70 kg	70 kg
FS	45%	45%
BAF_{soil}	18.3%	18.3%
$CR_{soil\text{-}dust}$	43%	43%
FD	55%	55%
BAF_{dust}	25.8%	25.8%

Per U.S. EPA (1993a), for the RME scenario, exposure assumptions corresponding to the 90 to 95th percentile of values for a given assumption were targeted as default factors for intake/contact rate, exposure frequency, and exposure duration. For body weight and exposure concentration, average values were targeted.

In addition, the Baseline HHRA calculates "total arsenic cancer risk" for a number of subareas of the site, including Opportunity, based on assumed exposure to arsenic through ingestion of soil and dust and the above exposure assumptions. For Opportunity, the estimated cancer risk is 5.5×10^{-5} for the residential RME scenario and 7×10^{-6} for the residential CTE scenario. These estimates were based on assumed 95th percentile upper confidence limit (UCL) of the mean soil and dust concentrations in Opportunity of 145.1 ppm and 100.8 mg/kg dust, respectively.

2.2 Review of Key Exposure Parameters and Risk Assumptions

2.2.1 *Soil Ingestion Rate (IR)*

The soil ingestion rate parameter describes how much soil (or dust) a person (adult or child) consumes on a daily basis. Soil ingestion typically occurs incidentally, as a result, for example, of "hand-to-mouth" behaviors. CDM (1996) assumed the following soil ingestion rates (to reflect the combined rate of consumption of soil and dust):

- RME adult: 100 mg soil/d
- RME child: 200 mg soil/d
- CTE adult: 50 mg soil/d
- CTE child: 100 mg soil/d

These are U.S. EPA default assumptions for the RME and CTE scenarios (US. EPA, 1993a), and are widely cited and used by the U.S. EPA (the Agency) and state agencies in Baseline HHRAs for these scenarios. In general, these are consensus values based on a compendium of data from many studies that focus largely on children. While U.S. EPA (1993a) states that the adult RME value of 100 mg soil/d is based on a study by Sedman (1989), the Sedman (1989) paper indicates that it lacks scientific support for the value for consumption of soil and dust. The author states:

> No information could be identified that provides a sound technical basis for estimating the quantity of soil ingested by older children and adults. While other investigators have assumed that adults ingest a significant quantity of soil and employ an assumed rate of soil ingestion for adults, no basis for these assumptions could be identified…Investigators who have advanced exposure scenarios that include soil ingestion for adults assume that adults ingest less soil than children.

Sedman develops other soil ingestion rates for a range of increasing ages via extrapolation. These values are based on measured and estimated soil ingestion rates for toddlers (they report that for children from 1 to 3 years old, daily soil ingestion is 590 mg soil/d) combined with data about increases in blood lead levels at increasing ages and assumptions about changes in mouthing behaviors. For children ages 2 to 9, the cited soil ingestion rates range from 590 mg soil/d to 300 mg soil/d, and for adults (age 18-70) the cited rate is 100 mg soil/d.

The child RME value of 200 mg soil/d reportedly reflects the consensus opinion of a U.S. EPA workgroup and is "believed to correspond to a conservative estimate of an average ingestion rate for this age group over a chronic period of exposure" (U.S. EPA, 1993a). However, per U.S. EPA "the available data did not support identification of the 90 or 95 percentile value. It was the consensus among workgroup participants that over the 6 year period of concern for this receptor category, the value of 200 mg soil/day was reasonable to assume."

ED_001802_00024760-00010

While CDM (1996) describes a week-long study to measure soil and dust ingestion in 64 people living in Anaconda that was performed by Dr. Edward Calabrese, the results of this study were only used as scientific support for the default values. CDM states (p. 3-37):

> Using a single 'best tracer' methodology, the soil and dust ingestion rate median was 51 mg/d, the mean was 117 mg/d, and the 90th percentile was 277 mg/d. The 'four best tracers' study resulted in an ingestion rate median of 39 mg/d, a mean of 83 mg/d and a 90th percentile of 273 mg/d. The findings in the Anaconda soil and dust ingestion study support the Superfund Program's usual approach of assuming ingestion of 100 mg soil and dust per day as a CTE assumption and 200 mg soil and dust per day as a RME assumption for soil and dust ingestion rates (IRs) of children age 0-6 years. Though default assumptions are used for soil and dust IRs for children, these assumptions are clearly consistent with the available site-specific data.

Overall, these estimated soil ingestion rates are assumed to represent average incidental soil ingestion rates by children. It is further assumed that the potential for incidental ingestion of soil is greater in young children than older children and adults because of the increased hand-to-mouth behaviors at younger ages.

These assumptions do not consider pica behaviors (i.e., the recurrent intentional ingestion of unusually high amounts of soil, typically by children, on the order of 1,000-5,000 mg soil/day or more; U.S. EPA, 2008), which would dramatically increase the dose for a child that engages in these behaviors.

A very large number of additional studies have been conducted to attempt to measure soil and dust ingestion by children, most using "tracer" methods. These have been reviewed by U.S. EPA (2008) and other regulatory agencies (e.g., CA OEHHA, 2002). Because of the uncertainties in the results of soil ingestion studies, these agencies continue to recommend use of the default values for the soil ingestion parameter. As such, use of the default soil ingestion assumptions of 100 mg soil/d for adults and 200 mg soil/d for children in the Baseline HHRA appear reasonable.

2.2.2 *Fraction Ingested that is Soil (FS) and Dust (FD)*

CDM (1996) assumed that 45% of the total soil and dust consumed in a day is soil and 55% is dust. Regarding these assumptions, CDM (1996) states (p. 3-38),

> It was assumed for both adults and children that of the total soil and dust ingested, 55% derives from indoor dust and 45% from soil. An assumption for fractionating dose between soil and dust is necessary since (1) indoor dust and soil arsenic and lead concentrations are not the same at exposure points, (2) different bioavailability estimates are used for dust and soil for arsenic, and (3) many studies have found a significant contribution of indoor dust to exposure.

While U.S. EPA is not specific about its rationale for selecting these values, these are typical U.S. EPA default values applied for the soil/dust ingestion pathways: in their *Exposure Factors Handbook*, U.S. EPA (2011b) recommends assuming that the relative proportions of soil and dust ingested by children are 45% soil and 55% dust, and this assumption is also used in U.S. EPA's Integrated Exposure and Uptake Biokinetic (IEUBK) model for lead in children (U.S. EPA, 1994a).

In the absence of other data, use of 45%/55% soil/dust for the relative fraction ingested assumption appears reasonable for evaluating exposure to arsenic in soil and dust.

2.2.3 *Relative Concentration of Arsenic in Dust ($CR_{soil-dust}$)*

CDM (1996) assumed that the concentration of arsenic in dust was 43% of the concentration of arsenic in soil. CDM (1996) bases this estimate on measurements of arsenic concentrations in soil and dust in Anaconda and Opportunity reported by Bornschein (1992 and 1994). As described by CDM (1996), soil samples were collected from several locations within each yard, and in general, three interior dust samples were collected from each home, with samples "intended to represent areas frequented by children, and included a floor area directly inside the main entry to the home, a floor area in the most frequently occupied room (usually living room or kitchen) and a floor area in the child's bedroom" (p. 2-11). Regarding the relative concentrations in soil and dust, CDM states that (p. 3-24) "…analysis of paired soil and interior dust measurements for arsenic suggest a transfer coefficient of 0.43 for movement from soil to dust (Bornschein 1994)."

The basis for this original assumption was not verified. However, a more recent study of arsenic concentrations in soil and dust was conducted in the Community Soils OU, including Opportunity, in 2006 and 2007 (Pioneer Technical Services, 2009). Samples of exterior soil and interior dust (from the main living space and the attic) were collected from 10 homes in Opportunity, as well as 17 homes in Anaconda West and 15 homes in Anaconda East. The results showed the following:

- In Opportunity (sample size (n) = 10), the average relative concentration of arsenic in main indoor living area dust vs outdoor soil was 170% (range 60% to 490%), with median and 75th percentile values of 110% and 200%, respectively. In other words, on average, the concentration of arsenic in indoor living area dust was nearly twice that in outdoor soil, for samples collected at the same residence.

  When concentrations in attic dust were compared to those in dust from the main indoor living area of the same home, attic concentrations were higher in six of 10 cases (the average relative concentration of arsenic in dust from the attic vs. the main indoor living area was 240%, range 10% to 730%, with median and 75th percentile values of 120% and 350%, respectively). In other words, on average, the concentration of arsenic in attic dust was more than twice that in dust from the indoor living area, for samples collected at the same residence.
- Combining all data collected outside of Opportunity in Anaconda West and Anaconda East (n = 32), the average relative concentration of arsenic in main indoor living area dust vs outdoor soil was 110% (range 30% to 310%), with median and 75th percentile values of 90% and 140%, respectively. Thus, the tendency for higher concentrations in indoor dust vs. outdoor soil was consistent in Anaconda as well as Opportunity.

  Attic dust concentrations tended to be higher than main indoor living area dust concentrations at these locations as well: attic concentrations were higher in all but three of the cases, with concentrations sometimes dramatically higher in the attics (the average relative concentration of arsenic in dust from the attic vs. the main indoor living area was 1,160%, range 7% to 2,970%;. Median and 75th percentile relative values were 870% and 1,880%, respectively).
- Combining all data for Opportunity, Anaconda West, and Anaconda East (n = 42) to increase statistical robustness, the average relative concentration of arsenic in main indoor living area dust vs outdoor soil was 130% (range 30% to 490%), with median and 75th percentile values of 100% and 150%, respectively.

  The average relative concentration of arsenic in dust from the attic vs. the main indoor living area was 940% (range 7% to 2970%), with median and 75th percentile values of 720% and 1,610%, respectively.

These data indicate that the ratio of concentrations in indoor dust in the main area versus concentrations in outdoor soil in Opportunity and Anaconda homes are comparable, and that the relative concentration in indoor dust to outdoor soil is much higher than that assumed in the CDM (1996) Baseline HHRA. In addition, at most of the sampled locations, the attic dust concentration was much higher than the concentration in dust in the main living area.

Whereas the CDM (1996) assessment assumed that the indoor dust concentration was 43% of the outdoor soil concentration, more recent data, including data specific to the Opportunity community, indicate that the concentration of arsenic in indoor dust should be assumed to be higher than in outdoor soil.

2.2.4 *Oral Bioavailability Factor for Soil and Dust (BAF_{soil} and BAF_{dust})*

CDM (1996) assumed that 18.3% of arsenic in ingested soil and 25.8% of arsenic in ingested dust is bioavailable. Oral bioavailability factors are critical for evaluation of soil ingestion pathways, as the assumed value directly affects estimated dose and thereby risk. The bioavailability estimates assumed in CDM (1996) are based on a site-specific study (cited as Battelle 1994[1]) in which cynomolgus monkeys were administered either soluble arsenic or arsenic in soil or dust collected in the Anaconda community. Arsenic was then measured in urine, feces, and blood.

Appendix C of CDM (1996) (*Review of the Battelle Columbus Report: Determination of the Bioavailability of Soluble Arsenic and Arsenic in Soil and Dust Impacted by Smelter Activities following Oral Administration in Cynomolgus Monkeys, Memorandum from Christopher Weis to Charlie Coleman and Susan Griffin, November 10, 1994*) and Freeman et al. (1995) describe the monkey study and its results. As described, arsenic was administered to monkeys as a single dose in one of the following ways:

- Intravenous (IV) dosing of a soluble arsenic solution (as sodium arsenate)
- Oral gavage dosing of a soluble arsenic solution (as sodium arsenate)
- Oral ingestion dosing, in capsules, of a test soil containing arsenic
- Oral ingestion dosing, in capsules, of a test dust containing arsenic

The test soil was a composite of samples collected in the Anaconda community from the surface (0-2") horizon of six play areas or bare area soils. The test house dust was collected with a vacuum from carpets in living areas and children's bedrooms. Samples were dried at 80°C, then sieved to a particle size of <250 μm. The samples were analyzed for arsenic concentration then blended to yield a final concentration of 410 ppm.

The study group consisted of only three animals that were cycled through each of the four treatment groups at different times during the study. Urine, fecal and cage rinse samples were reportedly collected prior to dosing and, after dosing, once every 24 hours for 7 days. Excretion of arsenic to urine and feces was reportedly essentially complete after 72hours.

Since we did not have access to all of the study details (e.g., animal handling, dose administration, and other variables that can significantly influence experimental outcomes), we cannot comment on the validity of all of the experimental methods. However, some available data cause concern and

[1] We were unable to locate the original study (cited as: Battelle. 1994. Determination of the Bioavailability of Soluble Arsenic and Arsenic in Soil and Dust Impacted by the Smelter Activities Following Oral Administration in cynomolgus Monkeys. Amended Final Report. March). However, Appendix C of CDM (1996) reviews the study, and Freeman et al. (1995) presents the study methodology and results (although the calculated bioavailability levels presented in Freeman et al. (1995) differ somewhat from those applied in CDM (1996)).

ED_001802_00024760-00013

suggest significant uncertainty about arsenic dosing and recovery methods. For example, the rate of recovery of urinary and fecal arsenic from the IV dosing groups was consistently lower than from the other groups; per Freeman et al. (1995), the total percentage recovery of arsenic from the IV group was 79.7%, compared to 94.4%, 101%, and 95.4% for the gavage sodium arsenate, oral soil, and oral dust groups, respectively. The reasons for this difference were not determined but, per the Appendix C review:

> It is likely that the IV dose remained bound to tissue components, cellular blood components or plasma proteins making it more inaccessible to glomerular filtration or biliary excretion that the oral doses.

As applied in the Baseline HHRA, absolute percent bioavailability of arsenic via each of the routes was estimated by determining the "area under the curve" (AUC) based on the urinary arsenic measurements. In addition, for application to the Baseline HHRA, the urinary recovery results were "normalized" to compensate for the poor recovery from the IV group by dividing the absolute bioavailability reported for the ingestion route by the absolute bioavailability for the IV route for the same animal. CDM (1996) reported the following mean adjusted absolute bioavailability estimates from urine concentrations based on these calculations:

- Gavage: 90.9%
- Soil: 18.3%
- Dust: 25.8%

These bioavailability estimates were applied in the cleanup level calculations[2].

However, as described below, we note that the bioavailability estimates were calculated erroneously, which resulted in lower levels. In addition, some of the study design variables created in an experimental exposure scenario that differed vastly from what might occur in a residential exposure situation. Furthermore, the bioavailability factors used in this study are low when compared to other values in the literature, in some cases by two- to three-fold. Together, these factors raise concern about the scientific validity of this study and suggest that these bioavailability estimates underestimate site-related risks.

2.2.4.1 CDM (1996) Incorrectly Applies Absolute Rather than Relative Bioavailability Factors

Per U.S. EPA (2007a), the term "bioavailability" as applied to the evaluation of chemical exposure refers to, "The fraction of an ingested dose that crosses the gastrointestinal epithelium and becomes available for distribution to internal target tissues and organs."

However, when applying a bioavailability factor in risk assessment calculations, a distinction must be made between "absolute" and "relative" bioavailability. Absolute bioavailability describes that total fraction of the dose that is absorbed and available, whereas relative bioavailability compares the bioavailability of the compound in the exposure medium of interest (e.g., soil) to that in the medium upon which the toxicity criterion is based.

[2] Freeman et al. (1995) also attempt to correct the results of urinary recovery for the oral routes to compensate for the poor recovery from the IV group. However, in their calculations, they apply the "correction factor" twice in a manner that it cancels itself out and the net result is no correction factor is applied. Consequently, the adjusted relative bioavailability estimates reported in Freeman et al. (1995) (19.2% for dust and 13.8% for soil) differ from those reported and applied in CDM (1996).

ED_001802_00024760-00014

With regard to arsenic, the application of relative bioavailability estimates is described by Roberts et al. (2007) in a review of oral bioavailability factors for arsenic:

> The default assumption used in risk assessments is that the extent of gastrointestinal absorption of arsenic from soil is equivalent to its absorption under the conditions in which the toxicity value was derived (NRC, 2003), which in the case of arsenic is from water. Absorption from water is the relevant comparison for arsenic because the cancer slope factor used to estimate excess cancer risks was developed from studies of individuals exposed to arsenic in drinking water.

Thus, for arsenic, the relative oral bioavailability (RBA) based on urinary excretion data should be calculated as follows (U.S. EPA, 2010):

Since the oral bioavailability estimates applied in CDM (1996) reflect the percent of the total delivered dose in soil that is excreted, and are not normalized relative to the excreted dose following ingestion of arsenic in water, they are absolute, not relative, bioavailability factors.

Applying the Freeman et al. (1995) data on urinary excretion of arsenic following ingestion of soil or dust and following gavage administration of sodium arsenate in water to this equation yields relative bioavailability estimates of 20.3 ± 3.4% for soil and 29.1 ± 0.7% for dust. Thus, the RBA values for soil and dust are 2.0 and 3.3 percentage points higher, respectively, than the bioavailability factors assumed in the Baseline HHRA. Use of the RBA values results in higher assumed doses and, therefore, higher assumed risks at a given soil or dust concentration.

In another study of the oral bioavailability of arsenic in primates, Roberts et al. (2007) reported that the average percentage of arsenic recovered in urine after oral (gavage) administration of sodium arsenate in water (40.6% ± 10.1%) was substantially lower than after IV administration of sodium arsenate (80.5% ± 10.2%), "indicating incomplete oral absorption of arsenic from the oral dose in water." Thus, even highly soluble sodium arsenate in water may not be completely absorbed in a given experimental system, and it is essential to determine the relative absorption of arsenic following oral administration in soil (or dust) compared to oral administration in water within the system of interest, rather than simply assume that absorption of arsenic in water would be complete (which is essentially what is assumed if the urinary excretion fraction in soil is not determined relative to water).

The importance of making the "relative" adjustment when using urinary excretion as a surrogate for absorbed arsenic is further illustrated by the potential fate of some of the absorbed arsenic. A fraction of administered arsenic that is absorbed may be eliminated in bile in the feces, or it may enter tissue compartments and thus not be reflected in the recovered urine. U.S. EPA (2010) notes,

> If 100% of all absorbed arsenic were excreted in the urine, the UEF [urinary excretion fraction] would be equal to the oral absorption fraction or ABA. However, some absorbed arsenic is excreted in the feces via the bile and some absorbed arsenic enters tissue compartments (e.g., skin, hair) from which it is cleared very slowly or not at all. Thus, the urinary excretion fraction should not be equated with the absolute absorption fraction.

However,

> The RBA [relative bioavailability] of two orally administered materials (e.g., a test soil and sodium arsenate) can be calculated from the ratio of the urinary excretion fraction of the two

materials. This calculation is independent of the extent of tissue binding or biliary excretion, because the fraction of absorbed arsenic that is excreted in urine (Ku), which does depend on tissue binding and biliary excretion, cancels in the calculation.

Thus the relative efficiency of recovery of arsenic in the experimental environment (i.e., in urine) is implicitly accounted for by applying relative, as opposed to absolute, bioavailability factors in a risk assessment.

2.2.4.2 Methodological Factors in the Monkey Study, Are More Likely (Than Not) to Have Under-Predicted Absorption

As noted above, it is critical to review the experimental design of a study. The original bioavailability study report was not available; however, the documentation we reviewed indicated that large (bolus) doses of the test material were given to the monkeys during a single dosing period. This experimental protocol is more likely than not to contribute to less efficient absorption compared to if smaller doses are delivered over a period of time, which is more representative of exposure to soil and discussed further below.

In the monkey study, the soil or dust dose was delivered to each monkey during a single dosing period in four capsules, with a 1- to 4-minute interval between each capsule (Freeman et al., 1995). Overall, approximately 3 grams of soil or 3.8 grams of dust were administered to each animal (CDM, 1996). Assuming an average animal body weight of 2.5 kg, this rate of intake (1200 mg soil/kg BW for soil[3]) is approximately 840-fold higher[4] than the amount of soil U.S. EPA typically assumes is consumed by an adult under the RME scenario (i.e., 100 mg/70 kg soil-d, or 1.4 mg/kg BW-d) and approximately 90-fold higher[5] than the amount U.S. EPA typically assumes is consumed by a child under the RME scenario (200 mg/15 kg BW-d, or 13.3 mg/kg BW-d). The review in Appendix C of CDM (1996) notes that "It is plausible that such high doses may have a negative influence on the estimates of arsenic absorption made by the authors;" yet, they do not attempt to characterize its impact on the bioavailability measurement, stating "however, further work is necessary to determine the relationship between arsenic dose and percent absorption."

Other authors have observed that bolus doses can reduce absorption of metals. For example, data on absorption of lead from soils (Mushak, 1998) suggest that bolus administration of a large mass of metal and/or metal-containing soil matrix may be associated with a lesser degree of absorption from the gastrointestinal tract than might result from administration of the same mass in smaller, divided doses. Mushak (1998) references a study by Kierski (1992; a PhD thesis not located) in which soil was administered to rabbits to estimate lead absorption. When soil mass was reduced, relative bioavailability of lead from soil (relative to ingested soluble lead acetate in solution) increased significantly compared to larger soil doses. A number of mechanisms are thought to contribute, including the greater availability of smaller dosing amounts to stomach acids and to chelating and binding effects that may enhance absorption, and the potential that large doses can overwhelm typical biochemical and physiologic mechanisms. Thus, the low bioavailablity factors in the Freeman et al. (1995) study, compared to other studies, could be explained by the poor experimental design (as discussed in Section 2.2.4.4, many of the relative bioavailability estimates determined in other studies use an experimental model in which smaller soil doses are administered over a longer period of time).

[3] Freeman et al. (1995) estimates that the dose was 1500 mg/kg BW of soil or house dust.
[4] Appendix C of CDM (1996) states that the dose is 190-fold higher than the typical adult soil ingestion rate.
[5] Appendix C of CDM (1996) states that the dose is 20-fold higher than the typical child soil ingestion rate.

Steady-state models more closely approximate likely human exposures

In the Freeman et al. (1995) monkey study, arsenic was administered in single doses and arsenic excretion was determined based on collection of urine over 72 hours. U.S. EPA (2012a) identifies several disadvantages of single-dose studies versus steady-state models (in which animals are dosed over several days), including:

- Steady state models more closely mimic the status of the human receptor who receives continuous daily exposure to soil.
- At steady state, urinary excretion of arsenic will be relatively constant over time, and as a result, urinary arsenic excretion rate and urinary excretion fraction can be estimated by averaging multiple estimates obtained from several urine samples collected over time. By contrast, in a single dose study, urinary excretion fraction must be estimated as the cumulative urinary arsenic excretion. This requires absolute accuracy in sampling urine at each interval of the post-dosing observation period.
- Random errors in urine sampling (e.g., reflecting completeness of collection) would be expected to have a larger impact on estimates of the cumulative arsenic excretion in single-dose studies than on average steady-state arsenic excretion in repeat dose studies.

Further, because of the very small number of animals in the monkey study (a total of three animals per group), any biases or errors in the experimental design would have a significant impact on average urinary measurements and result in low bioavailability estimates.

2.2.4.3 The Study of Arsenic in Urine from Anaconda Children Cannot Be Reliably Used to Compare Predicted and Measured Concentrations

CDM (1996) defends the oral bioavailability assumptions applied in the Baseline HHRA in part based on a study in which urine samples were collected from Anaconda children and measured arsenic levels were compared to levels predicted based on the exposure equation and assumptions described above, including the site-specific bioavailability estimates. The urine study is discussed in Appendix D of the Baseline HHRA; the authors suggest that based on their comparison of measured vs. predicted "speciated" (i.e., inorganic) urinary arsenic, the exposure equations and assumptions used in the Baseline HHRA over-predict arsenic uptake from soil (speciated arsenic is assumed to come from soil, dust, and water, where inorganic species predominate, whereas organic arsenic is largely derived from food sources such as fish). However, numerous uncertainties in the methodology limit the ability to draw conclusions based on this urinary arsenic study.

Urine samples were reportedly collected from 373 children in Anaconda. Per Bornschein (1992), urine samples represented the "first catch" of the day. Bornschein (1992) indicates that urine samples were analyzed for total arsenic (inorganic + organic) and/or speciated (inorganic) arsenic, with speciated arsenic representing the sum of As^{+3}, As^{+5}, dimethyl arsenic acid (DMAA) and monomethylarsonic acid (MMAA) (i.e., it does not include organic arsenic such as arsenobentaine, which is a component of total arsenic and is considered to largely come from food, such as seafood). For each child, measured urinary arsenic was compared to levels that were predicted as a function of the estimated absorbed dose of arsenic from various sources and assumed urinary production rates. For speciated arsenic, excretion in g-arsenic/day was predicted as the sum of predicted excretion from ingestion of arsenic in soil, dust, and water, which was in turn was estimated from measured concentrations of arsenic in soil and dust and, where available, water (if residences used community water, a default concentration in water of 0.5 parts per billion (ppb; equivalent to micrograms per liter or μg/L) was assumed) and assumed intake rates. The same assumptions were used to predict

intake as were used in the HHRA: the bioavailability of arsenic in soil, dust, soil, and water was assumed to be 18.3%, 25.8%, and 100%, respectively, and the soil/dust ingestion rate was assumed to be 200 mg/d for the RME estimate and 200 mg/d for the CTE estimate. This "absorbed" daily dose was assumed to be equivalent to the daily urinary excretion rate. Urinary concentrations were estimated by dividing the estimated daily excretion rate (in g/d) by the assumed daily urine volume (in L/d) per child.

Regarding the environmental media concentration measurements:

- For soil, the 95% upper confidence limit (UCL) concentration of surface soil samples collected in each yard (Bornschein 1992) was used. Bornschein collected composite soil samples to a depth of 2 cm "from several different types of surface conditions within the yard of each home or building, including the perimeter of the home or building, garden areas, play areas, and bare areas of the yard."
- For dust, composite indoor dust samples were collected, using a small vacuum pump, from three areas within each home or apartment. These areas were intended to represent areas frequented by children, and included floor areas directly inside the main entry to the home, in the most frequently occupied room (usually living room or kitchen), and in the child's bedroom.
- For water, the authors used a value measured from the primary water faucet, normally the kitchen sink, from a subset of 36 homes that obtained drinking water from local groundwater. Samples consisted of 100 milliliter (mL) taken immediately upon opening the tap. For subjects without a location-specific water sample, the concentration of arsenic in drinking water was assumed to be 0.5 ppb (these samples were assumed to not be contaminated, i.e., not from local groundwater).

The authors compared measured to predicted speciated arsenic in urine (n = 366). Total arsenic excretion (i.e., reflecting inorganic and organic arsenic) was also measured in urine and predicted assuming uptake from food (based on analysis of arsenic in food for a subset of 30 study participants) in addition to uptake from soil, dust, and water. However, CDM (1996) did not consider the total arsenic predictions to be very reliable,

> Because of the large standard deviation in the arsenic concentration in food and because of the lack of information on the bioavailability of arsenic in food, there is a large degree of uncertainty associated with predicted total urinary arsenic excretion.

Bornschein states further, "the uncertainty associated with predicted total arsenic excretion is greater than the uncertainty associated with the predicted speciated arsenic excretion."

Based on review of information provided by CDM (1996) and Bornschein (1992), the following sources of uncertainty in these results are evident:

- *It is unclear how measured concentrations of arsenic in urine reflect longer term average concentrations (and exposures).* The protocol discussed in Bornschein (1992) suggests that repeated urine samples would be collected from subgroups of Anaconda children throughout the year, to capture seasonal variations in concentrations (e.g., exposure to arsenic in airborne dust might be expected to be higher during drier months, and direct contact and ingestion of arsenic in homegrown produce might be expected to be higher during warmer periods). However, CDM (1996) does not indicate when the urine measurements presented in Appendix D were collected. Consequently, it is not known whether measurements reflect "high," "low," or "average" exposure periods.
- *"First catch" samples may not reflect within- and between day variations in urine dilution and*

arsenic intake. "First catch" samples reflect measurement of urinary arsenic at a single point in time on the collection day. For chemicals with a short biological half-life (e.g., arsenic), within-individual concentrations in spot urine samples can vary highly between samples, due to within- and between-day variations in urine volume and intake of exogenous compounds (Barr et al., 2005). Factors shown to influence urinary concentrations of these types of chemicals include fasting time, time of day, nature of the last meal, sample dilution, collection method, preservation method, sample interferences, and analytical method (Rasmussen et al., 1999).

- *Urinary excretion volumes were estimated, not measured.* To calculate the predicted concentration of arsenic in urine, the daily excretion rate (in g/d) was multiplied by an assumed urine excretion volume. The assumed excretion volume was based on the child's age, as follows: for ages 8 to 36 months, 240 mL/d; for ages 36 to 60 months, 355 mL/d; and for ages 60 to 76 months, 432 mL/d. It is unknown how, on an individual basis, estimated and actual excretion volumes varied. Use of a default value could under- or over-estimate urinary concentrations for a given child.
- *It was assumed without verification that all arsenic in water, soil, and dust was "speciated" arsenic (i.e., As^{+3}, As^{+5}, dimethyl arsenic acid (DMAA) and/or monomethylarsonic acid (MMAA)).* Based on the study description and identified protocols for the analysis of arsenic in water, soil, or dust (i.e., Appendix D of the Baseline HHRA and Bornschein 1992), it appears that no special separation procedures were used to speciate arsenic in soil, dust, or water. While, Bornschein (1994) suggests that a small subset of all soil and dust samples were analyzed for speciated arsenic, no details or results are provided. Some of the arsenic measured in soil, dust, or water may have been organic arsenic, which would yield a larger estimated urinary concentration than the measured concentration in urine, which is based only on speciated arsenic.
- *Some absorbed arsenic may accumulate in other tissues and not be excreted in urine; thus, the urinary concentration does not equal the absorbed dose.* As discussed previously, U.S. EPA (2010) notes, "If 100% of all absorbed arsenic were excreted in the urine, the UEF [urinary excretion fraction] would be equal to the oral absorption fraction or ABA. However, some absorbed arsenic is excreted in the feces via the bile and some absorbed arsenic enters tissue compartments (e.g., skin, hair) from which it is cleared very slowly or not at all. Thus, the urinary excretion fraction should not be equated with the absolute absorption fraction." The urinary excretion concentration could underestimate the actual absorbed dose.
- *The equation used to predict urinary arsenic concentrations does not calculate the absolute absorbed dose, nor does it reflect the same absorbed fraction as the measured concentration in urine.* The measured and predicted urinary concentrations of arsenic reflect two different measures. The bioavailability factor in the risk assessment equation predicts the absorption of arsenic from soil, dust, or other media *relative* to the absorption of the arsenic in the study upon which the toxicity criterion is based—it does not predict the total absorbed dose. In contrast, the measured urinary concentration reflects the fraction of the total absorbed dose that is excreted in urine. Since it is probably that experimental variables in the monkey study impacted the reported RBA values (as discussed in Section 2.2.4.2, above), it does not appear that urinary concentrations can be accurately predicted in children using these values.

Thus, because of one or more deficiencies or gaps in scientific information , it does not appear that it is valid to compare the measured and predicted urinary arsenic concentrations using these methods, or that these comparisons reveal anything about the accuracy of the equations and assumptions used in the Baseline HHRA for estimating doses of arsenic for these scenarios relative to the toxicity criterion dose.

2.2.4.4 Estimates for Other Sites Show Potentially Higher Uptake

Numerous studies have been conducted to measure or estimate the oral bioavailabilty of arsenic in soil at other sites. In U.S. EPA's (2012a) summary of a total of 103 RBA estimates of arsenic in soil using 88 unique test materials, RBA estimates for arsenic in soil following ingestion ranged from 8 to 61%, including soils from Montana. While the Agency recommends that site-specific data be used where available, in the absence of such data U.S. EPA (2012b) recommended a default value for RBA of arsenic in soil of 60%, based on an upper percentile of the RBAs presented in U.S. EPA (2012a).

Comparison to estimates at other sites provides some insight into the range of possible values and the factors that can impact bioavailability estimates. For example, site- and study-specific factors can influence bioavailability estimates, as follows:

- *The mineral phase of arsenic in soil can impact its bioavailability.* Roberts et al. (2007) suggests that the iron sulfate mineral phase fraction of arsenic (FeAs sulfate) is the best single linear predictor of arsenic RBA, with these two variables being inversely related (i.e., soils with lower iron sulfate fractions have higher bioavailability). For example, Roberts et al. (2007) reports that "Colorado smelter soil" had an iron sulfate fraction of 76.7% and a corresponding RBA of 5 ± 4% in a cynomolgus monkey study, while a "New York pesticide facility" had an iron sulfate fraction of 0.5% and a RBA in monkeys of 20 ± 10%. However, Freeman et al. (1995) reports the arsenic iron sulfate fraction for the tested Anaconda materials to be 5% for soil and 1% for dust, suggesting that a higher RBA might be expected. Further, while other authors report an inverse association between RBA and the iron oxide fraction (Yang et al., 2005), this relationship isn't necessarily consistent either: for example, Casteel et al. (2003b) reported an iron oxide fraction in two soils from a Butte, Montana Superfund site of 20% and 39%, with reported RBAs (based on studies in immature swine) of 17% and 22% (i.e., the soil with a lower iron oxide fraction had a lower RBA). Thus, clear relationships between mineralogy and RBA estimates are not apparent and it is not evident that soil mineralogy is sufficient to predict RBA given that many other site- and study-specific variables can also affect bioavailability.
- *Arsenic bioavailability may differ in different animal models.* Arsenic bioavailability in soil has been evaluated in rodents, monkeys, and juvenile swine. In attempting to directly compare the results of rodent, monkey, and swine models for the same soils, U.S. EPA (2012a) identifies only one study that evaluated RBA in all three species. This study investigated the RBA of arsenic in soils from four different sources (with arsenic concentrations of 290-388 ppm). For these soils, RBA estimates in swine were highest (range 31-52%), and estimates in monkey and mice were similar (range 25-38% and 21-35%, respectively). However, U.S. EPA indicates that because the sample size was small (n=4 soil samples), there is substantial overlap in the uncertainty bounds on the experimental estimates and statistically meaningful comparisons of between-species estimates cannot be made.

  In another study, U.S. EPA compares RBA estimates of the same soil materials in swine and mice (U.S. EPA, 2012a). Out of 11 materials, similar RBA estimates were reported for five materials and dissimilar estimates were reported for six. Of the dissimilar estimates, the RBA predicted in mice was less than that predicted in swine in every case. The absolute differences in the RBA estimates (swine – mouse) ranged from ≤1% to 28% (average 12%).

  When comparing rodents and swine, U.S. EPA (1996a) indicates a preference for the immature swine model to predict bioavailability in humans:

  > Immature swine were preferred as the test animal for this study because of characteristics

> comparable to young children (the age group at greatest risk of ingesting soil or other material containing contaminants). These included similar body size, weight, bone-to-body weight ratio and gastrointestinal anatomy and physiology. In addition, unlike other species such as rats or rabbits, the rate of growth and maturation is slower (a smaller portion of the prepubertal period will occur during the experiment), the cecum (a diverticulum of the large intestine where prolonged exposure to digestive enzymes and fluids occurs) is small, and coprophagia (reingestion of feces) is not required to maintain normal nutritional status. Like humans, swine are monogastric omnivores (stomach and intestinal fluid and bacterial composition are different than herbivores or carnivores), are adaptable to a periodic feeding schedule and have a gall bladder which excretes bile into the small intestine when food is present (some contaminants, such as lead are excreted in bile). Unlike the rat, metabolism and excretion of arsenic in swine is similar to humans. The results of pharmacokinetic studies of lead in immature swine and humans are similar (Weis et al., 1994).

Roberts et al. (2007) derived RBA estimates in monkeys using soil from two sites that had previously been evaluated using a swine model, although the evaluations were not conducted on the same soil samples. In both cases, the monkey RBA estimate was less[6]. Like the Freeman et al. (1995) study, the Roberts et al. (2007) monkey study administered single, very large doses of soil to each study animal (at a soil mass that "did not exceed 1,000 mg/kg[ppm]", which would be equal to 4,000 to 5,000 mg/d in a single dose for a 4-5 kg monkey). For the reasons discussed in Section 2.2.4.2, administration of these very large bolus soil doses probably resulted in a lower estimate of bioavailability in the monkeys relative to what might occur if smaller doses were administered over time.

U.S. EPA (2012a) summarized results for all three species and concluded the results were generally comparable, but suggests no direct species comparisons are possible at this time. Per U.S. EPA,

> Although estimates of RBA of arsenic in soil materials in animal models have not been quantitatively compared to estimates made in humans for the same material, this report shows that RBA estimates obtained from swine, monkey, and mouse for the same test materials are sufficiently similar to suggest that large differences in RBA across mammalian species are unlikely. This increases confidence in extrapolating RBA estimates obtained from these assays to humans.

- *Other study protocol variables can impact measurements.* As discussed in Section 2.2.4.2, a number of experimental parameters within a given study can influence the amount of arsenic that is absorbed, and these variables may differ substantially between studies and from human exposures. Some of these parameters include dose size and the frequency and duration of dosing.

Overall, differences in these variables limit the ability to compare bioavailability estimates across species and across sites. However, in general, U.S. EPA considers the *in vivo* juvenile swine model to be a good model for predicting bioavailabilty in humans, stating, "available physiological data indicate that young swine are a good model for the human gastrointestinal system" (U.S. EPA, 2010).

[6] The comparative RBA estimates were as follows: (1) Vasquez Boulevard and I-70 Superfund site (CO; a smelting site) residential soil composite: Monkey = 17 ± 8% (Roberts et al., 2007); Swine: 31% (mean, range 18-45%) (Casteel et al., 2001); (2) Western U.S. mining/smelting site soil sample: Monkey = 13 ± 7% (Roberts et al., 2007); Swine = 14.7% (mean, range <LOD-30.1%) (Rodriquez et al., 1999).

Table 4 summarizes *in vivo* RBA estimates for arsenic in soil collected from or near mining or smelting sites, measured in studies using juvenile swine, mice, or monkeys. Estimates range from a high of 98 ± 86% in a study of mining-impacted residential soils from the Aspen/Smuggler NPL site, conducted in juvenile swine (Casteel et al., 1997)[7], to a low of 5 ± 4% in a study of Colorado smelter soil, conducted in monkeys (Roberts et al., 2007). The mean RBA of the 28 values presented in Table 4 is 35%. Values in monkeys (range 5-19%, mean 14.2%, n = 6) tend to be lower than values in swine (range 10-98%, mean 40.7%, n = 19) and mice (range 31-45%, mean 29.7%, n = 4). These data, and the observations discussed in Section 2.2.4.2 regarding methodological concerns with the design of the monkeys studies, suggest that the estimates of RBA of arsenic in soil and dust at the Anaconda Smelter site (Freeman et al., 1995) are likely low.

[7]Per Casteel et al. (1997), recovery of arsenic in urine, feces, and tissues was very low, ranging from 25-35% of the amount given. The authors report "These estimates are based on very low doses of arsenic and are considered to be highly uncertain."

Table 4. Summary of Relative Oral Bioavailability Estimates for Arsenic in Soil Impacted by Mining or Smelting Activities

Site Location and Contamination Source	Arsenic Soil Concentration (ppm)	Model System	Dosing Protocol	Relative Bioavailability Estimate[a]	Reference
Aspen/Smuggler NPL Site (CO) residential soil [mining]	17	Juvenile swine (n = 4 per group)	Soil in 3 dose levels in dough ball for 2x/d for 15 d; urine collected on day 7 and 14	98 ± 86%[b,c]	Casteel et al., 1997
Composite of soil collected in residential area surrounding ASARCO smelter site, Tacoma, WA [smelting]	1,600 ± 31	Juvenile swine (n = 3 per group)	Soil in 4 dose levels, single dose (25-150 mg soil/kg BW/d); urine collected over 12 hour periods up to 6 days after dosing	78% (mean; 95% lower and upper limits = 56-111%)	U.S. EPA, 1996a
Aspen/Smuggler NPL Site (CO) berm soil [mining]	67	Juvenile swine (n = 4 per group)	Soil in 3 dose levels in dough ball for 2x/d for 15 d; urine collected on day 7 and 14	62 ± 55%[b,c]	Casteel et al., 1997
Iron King Mine—Humboldt Smelter Superfund Site, Yavapai County, AZ, from Chaparral Gulch near residential area [mining]	200 (mean)	Juvenile swine (n = 4 per group)	Soil in 3 dose levels in dough ball for 2x/d for 14 d (200-600 mg soil/kg BW/d); urine collected over 48-hr periods, beginning on days 5, 9, and 12	60% (mean; range 57-70%)	Cited by U.S. EPA, 2012a as Casteel and SRC, 2010a
New Jersey Zinc NPL Site (PA) soil composite [smelting]	134 and 110	Juvenile swine (n = 4 per group)	Soil in 3 dose levels in dough ball for 2x/d for 15 d; urine collected on day 7 and 14	52% and 39% respectively[b]	Casteel et al., 1997
Soil stockpile removed from residential properties and stockpiled; Former ASARCO smelter site near Tacoma, WA [smelting]	182 (mean)	Juvenile swine (n = 4 per group)	Soil in 3 dose levels in dough ball for 2x/d for 14 d (220-660 mg soil/kg BW/d); urine collected over 48-hr periods beginning on days 6, 9, and 12	49% (mean; range 46-52%)	Casteel et al., 2012
Along Silver Bow Creek near Butte, MT [mine tailings deposits] [d]	601	Mouse (n = 4 per group)	Soil-amended diet for 9 d; cumulative urine collected daily for 10 d	42.9-45.0%	Bradham et al., 2011

Site Location and Contamination Source	Arsenic Soil Concentration (ppm)	Model System	Dosing Protocol	Relative Bioavailability Estimate[a]	Reference
Along Silver Bow Creek near Butte, MT [mine tailings deposits] [d]	626	Juvenile swine (n = 4 per group)	Soil in 2 dose levels in dough ball for 2x/d for 14 d (93-183 mg soil/kg BW/d); urine collected over 48-hr periods beginning on days 6, 9, and 12	44% (mean; range 41-50%)	Casteel et al., 2009
Along Silver Bow Creek near Butte, MT [mine tailings deposits] [e]	1,513	Mouse (n = 4 per group)	Soil-amended diet for 9 d; cumulative urine collected daily for 10 d	42.9%	Bradham et al., 2011
Flood plain of Silver Bow Creek, five miles west of Butte, MT [mine tailings deposits] [e]	1,540 (mean)	Juvenile swine (n = 4 per group)	Soil in 3 dose levels in dough ball for 2x/d for 14 d (27-79 mg soil/kg BW/d); urine collected over 48-hr periods beginning on days 6, 9, and 12	42%	Cited by U.S. EPA, 2012a as Casteel and SRC, 2010c
Bingham Creek (Kennecott South) NPL Site (UT) channel soil [mining]	149	Juvenile swine (n = 4 per group)	Soil in 3 dose levels in dough ball for 2x/d for 15 d; urine collected on day 7 and 14	37 ± 19%[b]	Casteel et al., 1997
Murray Smelter NPL Site (UT) soil [smelting]	310	Juvenile swine (n = 4 per group)	Soil in 3 dose levels in dough ball for 2x/d for 15 d; urine collected on day 7 and 14	34 ± 3%[b]	Casteel et al., 1997
Residential sites affected by mining or smelting activity [mining/smelting]	280-990 (n=8)	Mouse (n = 4 per group)	Soil-amended diet for 9 d; cumulative urine collected daily for 10 d	31.0% (mean; range 11.1-52.8%)	Bradham et al., 2011
Vasquez Boulevard and I-70 Superfund site (CO) residential soil composite [smelting]	312-983 (n = 5)	Juvenile swine (n = 4 per group)	Soil in 3 dose levels in dough ball for 2x/d for 12 d; urine collected over 48-hr periods, beginning on days 6, 8, and 10	31% (mean; range 18-45%)	Casteel et al., 2001
California mine tailings [mining]	"at least 100"	Cynomolgus monkey (4-5 kg BW; n = 5 per group)	Fasted ; single administration of 1 soil dose in slurry in water via gavage (soil mass did not exceed 1000 mg/kg BW); urine collected for 4 days	19 ± 2%	Roberts et al., 2007

ED_001802_00024760-00024

Site Location and Contamination Source	Arsenic Soil Concentration (ppm)	Model System	Dosing Protocol	Relative Bioavailability Estimate[a]	Reference
Silver Bow Creek/ Butte Area NPL Site (MT) soil composite collected from a residential property located adjacent to a railroad grade in Butte [mining/ smelting]	367	Juvenile swine (n = 4 per group)	Soil in 3 dose levels in dough ball for 2x/d for 12 d; urine collected over 48-hr periods, beginning on days 6, 8, and 10	17-24% (mean)[f]	Casteel et al., 2003a,b; U.S. EPA, 2010
Silver Bow Creek/ Butte Area NPL Site (MT) soil composite from waste rock dumps in Butte Priority Soils Operable Unit [mining/ smelting]	234	Juvenile swine (n = 4 per group)	Soil in 3 dose levels in dough ball for 2x/d for 12 d; urine collected over 48-hr periods, beginning on days 6, 8, and 10	17-22% (mean)[g]	Casteel et al., 2003a,b; U.S. EPA, 2010
Colorado smelter composite soil [smelting]	"at least 100"	Cynomolgus monkey (4-5 kg BW; n = 5 per group)	Fasted ; single administration of 1 soil dose in slurry in water via gavage (soil mass did not exceed 1000 mg/kg BW); urine collected for 4 days	18 ± 6%	Roberts et al., 2007
Vasquez Boulevard and I-70 Superfund site (CO) residential soil composite [smelting]	"at least 100"	Cynomolgus monkey (4-5 kg BW; n = 5 per group)	Fasted ; single administration of 1 soil dose in slurry in water via gavage (soil mass did not exceed 1000 mg/kg BW); urine collected for 4 days	17 ± 8%	Roberts et al., 2007
Western U.S. mining/smelting site[h]	233-17,500 (n= 10)	Juvenile swine (n = 5 per group)	Soil in 3 dose levels in dough ball for 2x/d for 12 d (23-30 mg-soil/dose adjusted based on growth); urine collected over 24-hr periods every 3 d thereafter for 5 collection periods	14.7% (mean; range <LOD-30.1%)	Rodriquez et al., 1999

Site Location and Contamination Source	Arsenic Soil Concentration (ppm)	Model System	Dosing Protocol	Relative Bioavailability Estimate[a]	Reference
Montana smelter soil [mining/smelting]	"at least 100"	Cynomolgus monkey (4-5 kg BW; n = 5 per group)	Fasted ; single administration of 1 soil dose in slurry in water via gavage (soil mass did not exceed 1000 mg/kg BW); urine collected for 4 days	13 ± 5%	Roberts et al., 2007
Western iron slag soil [mining]	"at least 100"	Cynomolgus monkey (4-5 kg BW; n = 5 per group)	Fasted ; single administration of 1 soil dose in slurry in water via gavage (soil mass did not exceed 1000 mg/kg BW); urine collected for 4 days	13 ± 7%	Roberts et al., 2007
Butte NPL Site (MT) soil composite [mining, smelting]	238	Juvenile swine (n = 4 per group)	Soil in 3 dose levels in dough ball for 2x/d for 15 d; urine collected on day 7 and 14	10 ± 5%[b]	Casteel et al., 1997
Colorado smelter soil [smelting]	"at least 100"	Cynomolgus monkey (4-5 kg BW; n = 5 per group)	Fasted ; single administration of 1 soil dose in slurry in water via gavage (soil mass did not exceed 1000 mg/kg BW); urine collected for 4 days	5 ± 4%	Roberts et al., 2007

[a] Bioavailability of arsenic in test substance (e.g., soil) relative to sodium arsenate in drinking water.

[b] Recovery of arsenic in urine, feces, and tissues ranged from 25-35% of amount given (Casteel et al., 1997)

[c] Casteel et al. (1997) reports "These estimates are based on very low doses of arsenic and are considered to be highly uncertain."

[d] National Institute of Standards and Technology (NIST) Standard Reference Material 2710 (NIST, 2003)

[e] National Institute of Standards and Technology (NIST) Standard Reference Material 2710a.

[f] Presented in Casteel et al. (2003a) as 22% (range 17-29%); according to Casteel et al. (2003b) this method was "known to yield low recovery of methyl metabolites." Casteel et al. (2003b) reanalyzed the urine samples using a revised analytical procedure "known to yield good recovery of all urinary metabolites," yielding an estimate of 27% (range 21-35%). U.S. EPA (2010) recalculated estimates of 24% ± 2% and 23%, respectively.

[g] Presented in Casteel et al. (2003a) as 17% (range 14-22%); according to Casteel et al. (2003b) this method was "known to yield low recovery of methyl metabolites." Casteel et al. (2003b) reanalyzed the urine samples using a revised analytical procedure "known to yield good recovery of all urinary metabolites," yielding an estimate of 22% (range 17-28%). U.S. EPA (2010) recalculated estimates of 18% ± 3% and 20%, respectively.

[h] No specific sample location was given. However, it is described by Rodriquez et al. (1999) as "Two matrices were collected for this study from a typical

mining/smelter site in the western U.S. where wastes were deposited between 20 and 50 years ago. These aged and weathered wastes included a calcine material (a waste product which results from the roasting and smelting of arsenopyrite ore for the extraction of arsenic) and an iron slag material (a waste product that results from the smelting of ores for lead which is also high in iron)…Mineralogical composition of one calcine (soil 4) and one slag (soil 9) was determined by microprobe analysis for the various and arsenic-bearing solid phases. Soil 4 contained 38% of total arsenic as an arsenic jarosite analogue and 60% arsenic associated with Fe and Mnoxides. Soil 9 contained 17% of total arsenic as an arsenic jarosite analogue, 53% associated with Fe and Mn oxides, and 30%of total arsenic associated with lead oxide."

2.2.5 *Exposure Duration (ED_a and ED_c)*

CDM (1996) assumed the following exposure durations:

- RME adult: 24 yr
- RME child: 6 yr
- CTE adult: 7 yr
- CTE child: 2 yr

These are U.S. EPA default assumptions for the RME and CTE scenarios (U.S. EPA, 1993a). Both the RME value of 30 years and the CTE value of 9 years are based on data summarized in U.S. EPA's *Exposure Factors Handbook*[8] in which the average length of residence in the same house by people who own their own home was estimated to be 9 years and the 90th percentile was estimated to be 30 years. In the current *Exposure Factors Handbook* (U.S. EPA, 2011b), the recommended values for "residential occupancy period" are a mean of 12 years and an upperbound (95th percentile) of 33 years.

Based on these and site-specific information suggesting a relatively longer residence time for people who live in the Opportunity Community, the RME ED assumptions used in CDM (1996) will not overestimate site-related risks and may underestimate risks for some members of the population.

2.2.6 *Toxicity Criteria*

The oral cancer slope factor (SF_o) for arsenic applied in the CDM (1996) HHRA was 1.5 $(mg/kg\text{-}d)^{-1}$. This value has not changed, and is U.S. EPA's SF_o for inorganic arsenic (U.S. EPA, 2012c). Inorganic arsenic, including arsenic acid, arsenic pentoxide, and sodium arsenate, is classified by U.S. EPA as a known human carcinogen. The slope factor is based on increased lung cancer mortality in multiple human populations exposed to arsenic via inhalation, including in studies of smelter workers (in Tacoma, WA and Anaconda), as well as evidence of increased mortality from multiple internal organ cancers (liver, kidney, lung, bladder, and an increased incidence of skin cancer) in human populations consuming drinking water high in inorganic arsenic.

Because this value has not changed, it does not affect the recalculated site-related risks and soil cleanup levels.

2.3 Exposure Pathways Not Considered

In order to develop an estimate of dose, all relevant exposure pathways need to be evaluated. As described above, the soil cleanup level incorporates only two exposure pathways: soil ingestion and ingestion of interior dust. Based on a site visit to Opportunity in December 2012, the following are plausible exposure pathways that should have been included. They were not included for the reasons noted below, specified in the CDM (1996) document:

- *Dermal contact with soil or dust*: Not included because exposure through this pathway was not expected to contribute significantly. Specifically, CDM (1996) states (p. 3-19), "Only limited data are available on the rate at which metals cross the skin into the blood from soil or dust particles…uptake of metals across skin, especially from soil, is generally believed to be minor." And further, "It is expected that residents…might have dermal contact with contaminated soil. Only limited data are available on the rate at which metals cross the skin into the blood from soil

[8] The estimates in U.S. EPA (1993a) are based on the 1990 version of the *Exposure Factors Handbook*.

or dust particles; therefore, dermal exposure to metals was not included in the quantitative assessment. It is not considered likely that omission of this pathway causes a significant underestimate of risk because uptake of metals across the skin, especially from soil, is generally believed to be minor."

- *Inhalation of soil or dust*: Not included because exposure through this pathway was not expected to contribute significantly. Specifically, CDM (1996) states (p. 3-16), "Monitoring data indicate that levels of arsenic and lead in air are below current regulatory limits (Life Systems 1993). Therefore, inhalation of particulate matter released by wind erosion is not assessed quantitatively for residents or commercial workers."
- *Ingestion of fruits and vegetables*: Not included because exposure through this pathway was not expected to contribute significantly. Specifically, CDM (1996) states (p. 3-7), "Residents may have private gardens in which fruits and vegetables are grown for personal consumption. Anaconda resident survey responses indicate consumption of locally grown fruits and vegetables is minimal (Bornschein, 1993)" (p. 3-7). Further, CDM (1996) states (p. 3-16), "…fruits and vegetables may take up chemicals from the soil into the edible portion of the plant. However, as described in the HHRA for the Mine Flooding OU, Silver Bow Creek/ Butte NPL Site, exposure through this pathway is not expected to contribute significantly to site-related risks, and is, therefore, not further evaluated in this risk assessment."
- *Ingestion of locally raised meats*: Not included because exposure through this pathway was not expected to contribute significantly. Specifically, CDM (1996) states (p. 3-7), "Livestock production in Deer Lodge County is relatively low compared to other Montana counties; Deer Lodge County ranks as 53 out of 56 counties for beef production (personal communication, Montana Agriculture Line 8/3/94). Farms may have cattle, sheep, and hogs; however, there are typically no more than 2 animals per farm. Chickens are raised on most farms in the area. It is estimated that consumption of locally raised beef is low, as the majority of cattle raised are sold out of state (personal communication, Montana Agriculture Line, 8/3/94). Based on this information, it appears that exposure to arsenic and lead through the ingestion of local contaminated livestock is negligible. This assumption is supported by Anaconda resident survey responses (Bornschein, 1993), which also indicated negligible consumption of locally grown livestock. Moreover, analyses in the Streamside Tailings (SST) OU HHRA (CDM Inc. 1994) indicates that, even if local livestock are consumed, exposure to arsenic and lead through this pathway is not expected to be significant due to minimal concentrations sequestered in tissue."

  Further, CDM (1996) states (p. 3-17), "Livestock could, in theory, take up arsenic and/or lead from ingested soil and food and sequester these chemicals in their tissues. However, information presented in the SST OU HHRA indicates that this is unlikely to occur in significant amounts. Additionally, local residents consume little locally grown livestock. Exposure through this pathway is expected to be negligible for arsenic and lead even where local livestock is consumed based on information presented in Section 3.1.2.1. This pathway is not likely to significantly contribute to site-related risks and is, therefore, not further assessed in this HHRA."
- *Ingestion of or dermal contact with surface water*: Not included because exposure through this pathway was not expected to contribute significantly. CDM (1996) states (p. 3-18), "Area residents might visit affected creeks and be exposed to surface water through incidental ingestion during wading and other water play activities. Exposure to arsenic and lead might also occur through aquatic recreation in a small pool near Opportunity Ponds. It seems unlikely that such exposures would be associated with significant risks, based on results of the risk assessment recently completed for the SST OU of the Silver Bow Creek NPL Site (CDM Inc. 1994). Surface

water and sediment are heavily contaminated with arsenic and metals in this OU, yet even conservative estimates of potential exposures were not associated with significant risk. Risk-based screening concentrations are developed in Section 6.0 for this exposure pathway."

Further, CDM (1996) states (p. 3-18), "Only recreational users of surface water within the study area would be potentially exposed through this pathway. Lead is not expected to be significantly absorbed across the skin and several risk assessments performed for sites within the Clark Fork Basin have concluded that dermal exposure is insignificant for this metal. Dissolved arsenic in surface water may, however, be absorbed to some extent, although significant exposures are not expected based on results of the SST OU risk assessment (CDM Inc. 1994). This pathway is addressed using risk-based screening levels developed in Section 6.0."

And CDM (1996) states (p. 3-19), "Potential exposures for recreational visitors should occur during visits to surface water in the study area. Incidental ingestion of sediments might occur in much the same way as incidental ingestion of soils. However, recreational visitors are assumed to spend the majority of their time in the water, where sediments are not expected to adhere to skin. Additionally, visitors are assumed to bathe following swimming. Therefore, this HHRA assumes that contact with sediments would be minimal. This pathway is, therefore, considered insignificant and exposure to recreational users is not quantified in this HHRA."

- *Ingestion of fish*: Not included because exposure through this pathway was not expected to contribute significantly. CDM (1996) states (p. 3-17), "…Current or future residents and recreational users could be exposed through ingestion of these fish. Although this is a plausible exposure pathway, screening level calculations presented in Life Systems (1993) indicate that risks resulting from fish ingestion would be very low. Therefore, exposure from ingestion of contaminated fish is not evaluated further in this HHRA."
- *Ingestion of drinking (ground) water, Dermal contact with groundwater, Ingestion of or dermal contact with sediment.* Not included but reason for exclusion not specified.

Information from surveys of Anaconda residents suggest that many of these exposure pathways (e.g., ingestion of fruits and vegetables) are complete for at least some members of the population and should be considered, or that residents engaged in relevant practices in the past but discontinued them out of concerns about potential soil contamination (e.g., growing fruits and vegetables or raising livestock), current U.S. EPA guidance (e.g., U.S. EPA, 2004a; U.S. EPA, 1996b) also suggests that dermal contact with soil/dust and inhalation of soil particulates should be considered in deriving cleanup levels for soil at a site with residential exposures. Exclusion of these pathways results in an incomplete assessment of potential site-related risks and will underestimate the cancer risk using U.S. EPA methodology.

3.0 RECALCULATION OF SITE-RELATED RISKS AND SOIL SCREENING LEVELS

3.1 Quantification of Exposure

I have been asked whether the action level for residential soil established in the Anaconda Smelter NPL ROD was developed in a manner that reflects valid methodologies for predicting exposure and risk, and whether the action levels are appropriately health protective according to U.S. EPA risk assessment practices. In Section 2.0, I review the approach that was used to establish the action level. Based on my critical review of the U.S. EPA Baseline HHRA and knowledge of the Anaconda site, I reevaluate site-related risks and recalculate a soil cleanup level: in Sections 3.1, 3.2, and 3.3, I discuss the revised exposure assumptions for the soil and dust ingestion pathways, as well as

equations and assumptions for other pathways that were not included in the Baseline HHRA but are evaluated here.

Site-related risks were recalculated based on recently collected soil and groundwater data, revised exposure assumptions for the soil and dust ingestion pathways, and additional exposure pathways that were assumed to be complete. These updates are described below.

3.1.1 *Estimation of Exposure Point Concentrations*

In June 2012, soil and groundwater sample were collected from residential properties in the Opportunity community and analyzed for arsenic. These data were used to calculate exposure point concentrations (EPCs) used to estimate intake of arsenic. Table 5 summarizes these data.

Table 5. Summary of Soil and Groundwater Sampling Data for Arsenic Collected in the Opportunity Community in June 2012

Medium	Depth Interval	Sample Count	**Arsenic Concentration Range** (soil: ppm; groundwater: ppb)	**Arsenic Concentration Average and Standard Deviation** (soil: ppm; groundwater: ppb)
Soil	0-2"	66	5.95-1,420	138.3 ± 203.7
	2-6"	66	7.66-872	140.9 ± 144.4
	6-12"	66	7.38-611	116.0 ± 94.5
	12-24"	18	6.35-181	53.0 ± 48.0
	24-36"	60	1.66-199	19.8 ± 28.7
	36-48"	14	1.86-40.2	9.38 ± 10.3
	48-60"	23	0.994-16.9	5.44 ± 5.25
Groundwater	---	30	0.311-31.4	6.88 ± 7.83

Per U.S. EPA risk assessment guidance (U.S. EPA, 1989; 1992a), the 95 percent upper confidence limit (95% UCL) of the arithmetic mean was used as an estimate of the contaminant's arithmetic average concentration in each river segment for the RME scenario. Use of the 95% UCL provides reasonable confidence that the true average in each exposure area will not be underestimated. Consistent with U.S. EPA guidance, if the calculated 95% UCL exceeded the maximum detected concentration in a given medium, the maximum detected concentration is used (U.S. EPA, 1989; 1992a).

The following assumptions were made for purposes of selecting EPCs for the various potential exposure media:

- **Concentrations in Surface Soil.** Typically, the top 2 cm of soil is assumed to be the depth of soil where direct contact predominantly occurs (U.S. EPA, 1996b). For purposes of this assessment, the EPC for surface soil was calculated based on soil data collected in Opportunity in June 2012 from the 0-2" depth interval.
- **Concentrations in Subsurface Soil, for Estimating Uptake into Produce**. Roots of vegetables

and other garden produce were assumed to uptake arsenic from the shallow subsurface soil. For purposes of this assessment, the EPC for subsurface soil was calculated based on soil data collected in Opportunity in June 2012 from the 2-6" and 6-12" depth intervals.

- **Concentrations in Sediment:** No sampling data for sediments in creeks near Opportunity were identified. Consequently, for the purposes of this assessment, the EPC for sediment was calculated based on soil data collected in Opportunity in June 2012 from the 0-2" depth interval.
- **Concentrations in Ground and Surface Water**: It was assumed that incidental contact with ground or surface water could occur. No sampling data for surface water in creeks near Opportunity were identified. Consequently, for the purposes of this assessment, the EPC for the ground and surface water contact pathways was calculated based on the groundwater data collected in Opportunity in June 2012.

EPCs used in the HHRA are summarized in Table 6.

Table 6. Exposure Point Concentrations (EPCs) Used in the Revised HHRA for the Opportunity, Montana Community

Medium	Sample Count	Maximum Concentration	95% UCL
Surface Soil	66	1,420 ppm	184 ppm
Subsurface Soil	132	872 ppm	155 ppm
Sediment	66	1,420 ppm	184 ppm
Groundwater/ Surface Water	30	31.4 ppb	15.6 ppb

3.1.2 *Additional Complete Exposure Pathways*

The contribution of a number of additional exposure pathways excluded in the Baseline HHRA to total arsenic exposure was evaluated. These pathways, and the rationale for their inclusion in the revised HHRA, are as follows:

- **Dermal contact with soil or dust.** Data collected in the Opportunity Community in June 2012 show arsenic contamination of surface soils. Consequently, this exposure pathway is assumed to be complete. In addition, dermal absorption factors are available to estimate uptake of arsenic across skin: U.S. EPA's *Supplemental Guidance for Dermal Risk Assessment* (U.S. EPA, 2004a) recommends assuming a dermal absorption fraction from soil for arsenic of 0.03.
- **Inhalation of soil particulates.** As discussed, data collected in the Opportunity Community in June 2012 show arsenic contamination of surface soils. U.S. EPA's *Soil Screening Guidance*, which is intended to support calculation of cleanup levels for contaminated soils at sites on the NPL with future residential land use, identifies inhalation of volatiles and fugitive dusts as one of the three most common routes of exposure to environmental contaminants in the residential setting (the other two are direct ingestion and ingestion of potable ground water) (U.S. EPA, 1996b). Consequently, this exposure pathway is assumed to be complete. In addition, the *Soil Screening Guidance* provides exposure equations and default assumptions to support estimation of particulate emissions from soils.

- **Ingestion of fruits and vegetables.** In the Baseline HHRA, this pathway was not evaluated in part because "Anaconda resident survey responses indicate consumption of locally grown fruits and vegetables is minimal" (Section 2.3, above). However, information gathered from Opportunity residents indicates that many of them currently, or have in the past, grow and consume vegetables from backyard gardens, and some also grow and consume fruit from fruit trees, raspberry bushes, and strawberry plants. Consequently, this exposure pathway is assumed to be complete.
- **Ingestion of locally raised meat**. In the Baseline HHRA, this pathway was not evaluated in part because "Livestock production in Deer Lodge County is relatively low compared to other Montana counties…there are typically no more than 2 animals per farm" (Section 2.3, above). However, information gathered from Opportunity residents indicates that a number of them currently, or have in the past, raise and consume chickens and chicken eggs, and several have also raised cattle or pigs. In addition, a number of Opportunity residents report consuming locally caught wild game including venison, elk, and ducks. Consequently, this exposure pathway is assumed to be complete.
- **Ingestion of and dermal contact with ground or surface water and ingestion of or dermal contact with sediment**. Several creeks run adjacent to the Opportunity Community. As a result, it is possible that local residents may visit the creeks and be exposed to surface water through incidental ingestion or dermal contact while wading or splashing in the creek. Consequently, these exposure pathways are assumed to be complete.

In general, for each pathway, the lifetime average daily dose (LADD), in units of milligrams of arsenic per kilogram of body weight per day (mg/kg-d), was calculated based on the assumed concentration in the exposure medium, the contact rate, and the frequency and duration of exposure, as follows:

$$\left(\frac{m}{d}\right) \quad \frac{\left(\frac{m}{\ }\right) \quad \text{ontact rate} \quad \left(\frac{\ }{d}\right) \quad \text{posure fre uency} \quad \left(\frac{d}{yr}\right) \quad \text{posure duration} \quad yr}{\text{ody wei ht} \quad \text{era in time} \quad \left(\frac{yr \quad d}{yr}\right)}$$

Pathway-specific lifetime excess cancer risks were then calculated by multiplying each LADD by the appropriate pathway-specific toxicity criterion (the oral cancer slope factor for ingestion pathways, the dermal slope factor for dermal contact pathways, or the inhalation unit risk value for the particulate inhalation pathway), for example:

$$\text{ifetime e c ess cancer ris} \quad m \quad d \quad m \quad d$$

For arsenic, the following toxicity criteria were used:

- **Oral slope factor (SF_o):** 1.5 $(mg/kg\text{-}d)^{-1}$ (U.S. EPA, 2012c)
- **Dermal slope factor (SF_d):** 1.5 $(mg/kg\text{-}d)^{-1}$ (U.S. EPA, 2012c). In general, it is recommended that dermal slope factors be calculated by adjusting an administered dose slope factor (i.e., the oral slope factor) to an absorbed dose slope factor (i.e., the dermal slope factor) by dividing the administered dose slope factor by the fraction of the contaminant absorbed in the gastrointestinal tract in the critical toxicity study (U.S. EPA, 1992b). However, in recommending a dermal value for arsenic, U.S. EPA assumed that the majority of the ingested dose in the studies upon which the oral slope factor are based (e.g., soluble arsenic in drinking water) is gastrointestinally absorbed. Consequently, U.S. EPA (1992b) does not recommend adjusting the oral slope factor for arsenic, but to also use this value as the dermal slope factor.

- **Inhalation unit risk factor (UR):** 0.0043 $(\mu g/m^3)^{-1}$ (U.S. EPA, 2012c). A UR is the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 $\mu g/m^3$ in air (U.S. EPA, 2012e).

Exposure parameters and assumptions are listed in Table 7.

Table 7. Exposure Parameters Used in the Risk Calculations for the Residential Scenario at the Opportunity, MT Site

Exposure Parameter	Description	Value	Source
$IR_{soil(c\ or\ a)}$	Ingestion rate of soil, child or adults	Child: 200 mg/d Adult: 100 mg/d	U.S. EPA, 1993a
$IR_{sed\ (c\ or\ a)}$	Ingestion rate of soil, child or adults	Child: 100 mg/event Adult: 50 mg/event	U.S. EPA, 1991; U.S. EPA 2011b; Professional judgment
IR_{water}	Incidental ingestion rate of surface or groundwater	0.05 L/d	U.S. EPA, 1989
FI_{soil}	Fraction of soil and dust ingested or contacted that is soil	0.45 (unitless)	CDM, 1996
FI_{dust}	Fraction of soil and dust ingested or contacted that is dust	0.55 (unitless)	CDM, 1996
RBA_{soil}	Relative oral bioavailability factor for soil	20.3%	Freeman et al., 1995
RBA_{dust}	Relative oral bioavailability factor for dust	29.1%	Freeman et al., 1995
RBA_{sed}	Relative oral bioavailability factor, sediment	20.3%	Freeman et al., 1995 (same as soil)
$SA_{soil\ or\ dust\ (c\ or\ a)}$	Skin surface area for dermal contact with soil or dust ,child or adult	Child: 1,720 cm^2/event Adult: 2,825 cm^2/event	U.S. EPA, 1992b; U.S. EPA, 2011b
$SA_{sed(c\ or\ a)}$	Skin surface area for dermal contact with sediment, child or adult	Child: 1,460 cm^2/event Adult: 4,560 cm^2/event	U.S. EPA, 2011b
$SA_{water(c\ or\ a)}$	Skin surface area for incidental dermal contact with surface or groundwater, child or adult	Child: 3,830 cm^2/event Adult: 10,920 cm^2/event	U.S. EPA, 2011b
$AF_{soil(c\ or\ a)}$	Skin adherence factor for soil, child or adult	Child: 0.12 mg/cm^2 Adult: 0.01 mg/cm^2	U.S. EPA, 2004a
$AF_{dust(c\ or\ a)}$	Skin adherence factor for dust, child or adult	Child: 0.01 mg/cm^2 Adult: 0.005 mg/cm^2	U.S. EPA, 2004a; Professional judgment
$AF_{sed(c\ or\ a)}$	Skin adherence factor for sediment, child or adult	Child: 0.18 mg/cm^2 Adult: 0.18 mg/cm^2	U.S. EPA, 2000a
ABS	Dermal absorption fraction of arsenic from soil or sediment	0.032 (unitless)	U.S. EPA, 2004a
EF	Exposure frequency, all but dermal contact with soil, or incidental exposure to surface or groundwater or to sediment	350 events or days/year	U.S. EPA, 1996b; U.S. EPA, 2012d
$EF_{soil(c\ or\ a)}$	Exposure frequency, direct contact with soil, for child or adult	Child: 130 days/year Adult: 48 days/year	Professional judgment
$EF_{water\ or\ sediment(c\ or\ a)}$	Exposure frequency, incidental exposure to surface or groundwater or to sediment, for child or adult	Child: 18 days or events/year Adult: 6 days or events/year	Professional judgment

Exposure Parameter	Description	Value	Source
$ED_{(c\ or\ a)}$	Exposure duration, child or adult	Child: 6 years Adult: 24 years	U.S. EPA, 1996b; U.S. EPA, 2012d
$BW_{(c\ or\ a)}$	Body weight, child or adult	Child: 15 kg Adult: 70 kg	U.S. EPA, 1996b; U.S. EPA, 2012d
AT	Averaging time	25,550 days	U.S. EPA, 1996b; U.S. EPA, 2012d
K_p	Percutaneous absorption factor	0.001 cm/hr	U.S. EPA, 1992b
PEF	Particulate emission factor	1.32×10^9 m^3/kg	U.S. EPA, 1996b
$ET_{(c\ or\ a)}$	Exposure time, incidental dermal contact with surface or groundwater or with sediment, child or adult	Child: 1 hour/event Adult: 0.5 hour /event	Professional judgment
Br_{age}	Arsenic plant/soil bioconcentration factor for above ground exposed produce from root uptake	0.026 (mg/kg dry weight (DW))/ (mg/kg soil)	Cobb et al., 2000; Ramirez-Andreotta et al., 2013
Br_{agp}	Arsenic plant/soil bioconcentration factor for above ground protected produce (e.g., fruits, nuts) from root uptake	0.006 (mg/kg dry weight (DW))/ (mg/kg soil)	Baes et al., 1985
Br_{bg}	Arsenic plant/soil bioconcentration factor for below ground produce from root uptake	0.0036 (mg/kg dry weight (DW))/ (mg/kg soil)	Cobb et al., 2000; Ramirez-Andreotta et al., 2013
$CF_{d\text{-}w(age)}$	Dry-to-wet weight conversion factor for exposed (above ground) produce	0.126 kg DW/kg fresh weight	Baes et al., 1985
$CF_{d\text{-}w(agp)}$	Dry-to-wet weight conversion factor for protected (above ground) produce	0.222 kg DW/kg fresh weight	Baes et al., 1985
$CF_{d\text{-}w(bg)}$	Dry-to-wet weight conversion factor for below ground produce	0.222 kg DW/kg fresh weight	Baes et al., 1985
$IR_{age(c\ or\ a)}$	Ingestion rate of above ground exposed produce, child or adult	Child: 0.00077 kg DW/d Adult: 0.00032 kg DW/d	U.S. EPA, 2011b
$IR_{agp(c\ or\ a)}$	Ingestion rate of above ground protected produce, child or adult	Child: 0.0015 kg DW/d Adult: 0.00061 kg DW/d	U.S. EPA, 2011b
$IR_{bg(c\ or\ a)}$	Ingestion rate of below ground produce, child or adult	Child: 0.00023 kg DW/d Adult: 0.00014 kg DW/d	U.S. EPA, 2011b
$RBA_{veg\text{-}ag,e}$	Relative bioavailability of arsenic in ingested above ground, exposed produce	50%	Juhasz et al., 2008
$RBA_{veg\text{-}ag,p}$	Relative bioavailability of arsenic in ingested above ground, protected produce	75%	Juhasz et al., 2008
$RBA_{veg\text{-}bg}$	Relative bioavailability of arsenic in ingested below ground produce	100%	Juhasz et al., 2008
F_{veg}	Fraction of vegetables consumed that are contaminated	0.25	Professional judgment (U.S. EPA, 2011b indicates home-produced about ¼ of total, for populations that consume home-produced)

Exposure Parameter	Description	Value	Source
$IR_{soil\text{-}cattle}$	Quantity of soil eaten by beef cattle each day	0.002 kg soil/kg BW-d	Mayland et al., 1977
B_{forage}	Arsenic plant/soil bioconcentration factor for forage, from root uptake	0.036(mg/kg dry weight (DW))/ (mg/kg soil)	U.S. EPA, 2005
$IR_{forage\text{-}cattle}$	Quantity of forage ingested by beef cattle per day	0.017 kg forage DW/kg BW-d	NRC, 1987
B_{meat}	Arsenic intake/ muscle bioconcentration factor for cattle	0.097 (mg/kg muscle)/(mg/kg BW-d)	Bruce et al., 2003
$IR_{meat(c\ or\ a)}$	Consumption rate of beef, child or adult	Child: 0.0012 kg FW/kg-BW-d Adult: 0.00059 kg FW/kg-BW-d	U.S. EPA, 2011b
F_{meat}	Fraction of beef from a contaminated source	Resident: 1	Professional judgment (consumption rates based on home-produced meat)
CF	Conversion factor, liters per cm^3 or kilograms per g	0.001 L/cm^3 or 0.001 kg/g	---

3.1.2.1 Contributions from Soil or Dust

Dermal Contact with Soil or Dust

Uptake from incidental dermal contact with soil or dust was calculated using the following equations.

For soil:

$$is_{soil}\left(\frac{s_{soil\ c}\ {}_{soil\ c}\ {}_{soil\ c}\ {}_{soil\ c}}{c}\quad\frac{s_{soil\ a}\ {}_{soil\ a}\ {}_{soil\ a}\ {}_{soil\ a}}{a}\right)$$

Where $Abs_{soil(c)}$ and $Abs_{soil(a)}$ are the absorbed doses in mg/event for the child and the adult, respectively.

For dust:

$$is_{dust}\left(\frac{s_{dust\ c}\ {}_{dust\ c}\ {}_{dust\ c}\ {}_{dust\ c}}{c}\quad\frac{s_{dust\ a}\ {}_{dust\ a}\ {}_{dust\ a}\ {}_{dust\ a}}{a}\right)$$

Where $Abs_{dust(c)}$ and $Abs_{dust(a)}$ are the absorbed doses in mg/event for the child and the adult, respectively.

Several factors can affect the potential for exposure to arsenic from dermal contact with soil. In general, it is assumed that activities associated with incidental soil ingestion are likely to present opportunities for dermal exposure (U.S. EPA, 1992b), although soil ingestion rates are based on annual average values whereas dermal exposure is typically evaluated on a per-event basis. Overall, it is assumed that dermal contact with soil and dust would be greater during warmer months when people spend more time outdoors and/or the surface area of exposed skin is greater. There is also uncertainty about the residence time of soil or dust on skin, which can affect the total absorption

potential, but it is assumed to roughly correspond to the time between washings, or about 8 to 24 hours (U.S. EPA, 1992b).

A range of values for frequency of exposure to outdoor soil have been proposed (U.S. EPA, 1992b), ranging for adults from 43 days/year (an assumed "typical" value for an adult who gardens or works outside 1 to 2 days/week during the warmer months) to 350 days per year (based on the rationale that in warmer climates, people who actively garden or play outdoors could have contact with soil almost every day). For children, a "typical" value of 130 days per year has been proposed. For this assessment, it is assumed that for adults, the exposure frequency (EF) to soil contact is four times/month (or 48 days/year) and for children it is 2-3 times/week (or 130 days/year). It is assumed that adults and children contact dust 350 days/year.

The assumed skin surface area available for soil or dust contact (SA) is based on data presented in U.S. EPA's *Dermal Exposure Assessment* guidance (U.S. EPA, 1992b) and *Exposure Factors Handbook* (U.S. EPA, 2011b), according to the following considerations:

- For children, the value (1,720 cm^2/event) assumes contact by head, hands, lower arms, feet, and lower legs for 25% of events, and head, hands and lower arms for 75% of events, based on the mean surface area by body part averaged for children ages 2 to 6 (U.S. EPA, 2011b).
- For adults, the value (2,825 cm^2/event) assumes contact by head, hands, lower arms, and lower legs for 25% of events, and head and hands for 75% of events (U.S. EPA, 1992b).

The soil- or dust-to-skin adherence factor (AF) describes the mass of soil or dust that adheres to skin per square centimeter of skin surface. Assumed values were drawn from U.S. EPA (2004a), and are based on the following:

- For soil, the adult value is based on the mean value for groundskeepers (0.01 mg/m^2) since it was assumed that adults could come in contact with local soils while doing yard or garden work. The child value is based on the average of mean values for children playing in dry soil and children playing in wet soil (0.12 mg/m^2).
- For dust, the adult value is assumed to be one-half that for soil (0.005 mg/m^2) due to the lack of any other data and the assumption that dust adherence would be less than that of soil. The child value is based on the mean value for children indoors (0.01 mg/m^2).

The dermal absorption factor (ABS) describes the uptake of arsenic across skin when in contact with arsenic in soil. The value (0.032) is U.S. EPA's chemical-specific default for this pathway (U.S. EPA, 2004a).

Inhalation of Soil Particulate

Uptake from inhalation of soil particulate was calculated using the following equation and assumptions:

$$is_{soil} \quad \left(\frac{soil \qquad \overline{m}}{\qquad\qquad\qquad\qquad} \right)$$

The particulate emission factor (PEF) relates the concentration of contaminant in soil to the concentration of dust particles in air. The value assumed is a U.S. EPA default value (1.32×10^9 m^3/kg) and is derived from modeling conducted by U.S. EPA that incorporates assumptions about annual average emission rate based on wind erosion (in g/m^2-s per kg/m^3), fraction of vegetative cover (50%), and annual windspeed (4.69 m/s) (U.S. EPA, 1996b).

The exposure equation results in an estimate of the annual average airborne concentration of arsenic to which an individual is exposed (in $\mu g/m^3$). Lifetime excess cancer risk for this pathway is estimated by multiplying this concentration by the inhalation unit risk factor (UR) for arsenic of 0.0043 per $\mu g/m^3$ (U.S. EPA, 2012c).

A separate equation to estimate uptake from inhalation of dust was not included due to lack of information on re-entrainment of dust into the air. However, it is assumed that if this pathways were included in the risk assessment, the overall site-related risks would increase slightly (as shown in Section 3.2, inhalation is not a major exposure pathway compared to some other exposure pathways).

Ingestion of Locally Grown Produce

Uptake from ingestion of locally grown produce contaminated by deposition of airborne arsenic or uptake of arsenic from soil ($Risk_{veg}$) was calculated using the following equations and assumptions.

Separate plant uptake and produce consumption rates were applied for three different categories of produce: aboveground exposed, aboveground protected, and belowground (root) vegetables or fruit. These categories are used because it is assumed that contaminants are taken up from soil or air at different rates into these groups of produce. Per Baes et al. (1985),

> Exposed produce (snap beans, tomatoes, apples, etc.) intercept atmospherically depositing material on edible surfaces, but surface areas for exposure are relatively small compared to leafy vegetables. Additionally, edible portions are typically concerned with reproductive functions (fruits and seeds). Protected produce (potatoes, peanuts, citrus fruits, etc.) are not directly exposed to atmospherically depositing material because their growth habit is underground, or if aboveground, the edible portions are protected by pods, shells, or nonedible skins or peels. Typically, edible portions are reproductive or storage organs.

First, the concentration of arsenic in above ground exposed (C_{age}), above ground protected (C_{agp}), and below ground (C_{bg}) produce, in mg/kg wet weight, is calculated:

sdeep d w a e

sdeep d w a p

sdeep d w

Then the lifetime average daily dose (LADD) for the child and adult, and the associated cancer risk, are calculated

([( ) ( ) ( )])

([( ) ( ) ( )])

In this assessment, for estimating concentrations of arsenic in produce, uptake from soil only was assumed—deposition of airborne particulate was not evaluated due to the lack of data on airborne arsenic deposition. Nonetheless, in some cases with sufficient air concentrations, atmospheric deposition can contribute significantly to plant arsenic concentrations, with accumulation of arsenic in both the leaves and storage organs of root crops reported as a result of atmospheric deposition near an active metal smelter (De Temmerman et al., 2012).

The assumed homegrown produce consumption rates were as follows:

- *Consumption rate of aboveground exposed produce (IR_{age})*: Values of 1.36 g/ kg BW-d (fresh weight, converted to 0.171 g /kg BW-d dry weight) for adults and 1.45 g/ kg BW-d (fresh weight, converted to 0.183 g /kg BW-d dry weight) for children were assumed. These values represent the mean per capita intake of all exposed vegetables reported for the U.S. population for ages 20-69 (adults) and 2 to 9 (children) (U.S. EPA, 2011b). Assuming a typical adult body weight of 70 kg, the adult rate equates to 95 g/d (or 3.4 ounces/d), which is equivalent to consumption of about 2 cups of lettuce.
- *Consumption rate of aboveground protected produce (IR_{agp})*: Values of 0.55 g/ kg BW-d (fresh weight, converted to 0.122 g /kg BW-d dry weight) for adults and 1.0 g/ kg BW-d (fresh weight, converted to 0.222 g /kg BW-d dry weight) for children were assumed. These values represent the mean per capita intake of all protected vegetables reported for the U.S. population for ages 20-69 (adults) and 2 to 9 (children) (U.S. EPA, 2011b). Assuming a typical adult body weight of 70 kg, the adult value equates to 38.5 g/d (or 1.4 ounces/d), which is equivalent to consumption of about 1/4 cup of fresh peas.
- *Consumption rate of belowground produce (IR_{bg})*: Values of 1.03 g/ kg BW-d (fresh weight, converted to 0.229 g /kg BW-d dry weight) for adults and 1.95 g/ kg BW-d (fresh weight, converted to 0.433 g /kg BW-d dry weight for children were assumed. These values represent the mean per capita intake of all belowground produce reported for the U.S. population for ages 20-69 (adults) and 2 to 9 (children) (U.S. EPA, 2011b). Assuming a typical adult body weight of 70 kg, the adult value equates to 72.1 g/d (or 2.5 ounces/d), which is equivalent to consumption of about one carrot.

To estimate which fraction of all consumed produce is from home gardens, it was assumed that on an annual average basis, 25% of all vegetables consumed was from home gardens (F_{veg}).

A literature search was conducted to identify data on uptake of arsenic from soil into produce. In general, authors have observed that leafy vegetables (e.g., lettuce, spinach) take up more arsenic into their edible parts than non-leafy vegetables (e.g., tomatoes, eggplant, beans, peas) (Ramirez-Andreotta et al., 2013). In addition, uptake can vary not only based on the soil concentration, but also based on the dominant inorganic arsenic species in soil (e.g., plants have been shown to preferentially take up arsenate, the dominant species in oxic environments; Ramirez-Andreotta et al., 2013); and soil characteristics such as pH, organic matter, clay content, water regime, and nutrient balance (e.g., uptake of arsenic from sandy soils is much greater than from clay soils; Ramirez-Andreotta et al., 2013).

Because of the potential impact of soil characteristics on plant uptake of arsenic, plant uptake factors were estimated based on data from similar sites, where available. The following studies evaluated plant uptake of arsenic from soils impacted by mining or smelting operations:

- Cobb et al. (2000) evaluated uptake of heavy metals into vegetables grown in soils mixed with mine wastes collected from areas in the Bingham Creek mining district near Salt Lake City, Utah. Plants were grown in a greenhouse, and soils were prepared with a percentage of mine tailing of 0, 25, 50, 75, or 100%, with average arsenic concentrations of 23.3, 187, 196, 303, and 408ppm, respectively. Bioconcentration factors (BCFs, in units of (mg As/kg plant, DW)/(mg As/kg soil, DW)) were calculated based on concentrations in edible plant parts and in soil, and ranged as follows: Bean = 0.00162-0.00790 (mean 0.00393); Radish = 0.0121-0.0292 (mean 0.0205); Lettuce = 0.00755-0.235 (mean 0.125). Arsenic was not detected in tomatoes (limit of detection = 0.125 mg/kg); assuming arsenic was present in tomatoes at one-half the detection limit resulted

in calculated BCFs for tomatoes ranging from 0.000153-0.00268 (mean 0.000739).

- Ramirez-Andreotta et al. (2013) evaluated plant uptake of arsenic in a controlled greenhouse study (with mean soil concentrations ranging from 27.2 to 533 ppm) and a garden study (with mean soil concentrations ranging from 2.35 to 374 ppm), to characterize the potential uptake of arsenic into homegrown vegetables at residences near the Iron King Mine and Humboldt Smelter Superfund site in southern Arizona. All vegetables in both experiments accumulated arsenic, with a direct correlation between the amount of arsenic in the edible portion of the plant and arsenic in soil for most of the vegetable families including Asteraceae (lettuce; BCF = 0.0478 mg plant DW/mg soil), Brassicaceae (radish, broccoli, kale, cabbage; BCF = 0.0146), Amaranthaceae (beet, Swiss chard, spinach; BCF = 0.00982), and Fabaceae (bean; BCF = 0.00323) families, but not the Solanaceae (tomato, pepper; BCF =0.00391) and Cucurbitaceae (squash, cucumber; 0.00483) families. Per the authors:

  > The results suggest that home gardeners neighboring mining operations or mine tailings with elevated arsenic levels should be made aware that arsenic can accumulate considerably in certain vegetables, and in particular, it is recommended that gardeners limit consumption of vegetables from the Asteraceae and Brassicaceae plant families.

Based on these data, the following BCFs were used in the current assessment:

- *Above ground, exposed produce*: 0.026 mg/kg plant (DW)/mg/kg soil (based on the mean of mean values for lettuce, tomatoes, Brassicaceae, Amaranthaceae, Solanaceae, and Cucurbitaceae families from the above studies). This value is consistent with the value presented in Baes et al. (1985) of 0.04.
- *Above ground, protected produce*: 0.0036 mg/kg plant (DW)/mg/kg soil (based on the mean of mean values for beans from the above studies). This value is consistent with the value in Baes et al. (1985) of 0.006.
- *Below ground, root produce*: 0.006 mg/kg plant (DW)/mg/kg soil (based on the value presented in Baes et al. (1985) for below ground produce). A value derived from smelting or mining contaminated soils was not identified.

Juhasz et al. (2008) and Larios et al. (2012) report that, in general, only inorganic arsenic (i.e., arsenite and arsenate) are detected in the edible portion of vegetables, with organic species rarely found; thus, it is appropriate to assume that the species of arsenic in consumed homegrown produce is the same as that upon which the toxicity criteria for arsenic is based (i.e., sodium arsenate in drinking water). However, as with other matrices, not all of the arsenic in produce may be bioavailable. When Juhasz et al. (2008) assessed arsenic bioavailability after ingestion of arsenic-contaminated vegetables based on blood plasma concentrations in swine, between 50-100% of the total administered arsenic dose was estimated to be absorbed and enter the systemic circulation, with mean values of 52% and 50% for chard and lettuce, respectively, 98% for mung bean, and 77% for radish. Based on these data, in this assessment, 50%, 75%, and 100% of the arsenic estimated to be ingested from above ground exposed, above ground protected, and below ground root produce, respectively, was assumed to be bioavailable.

Ingestion of Locally Raised Meat

Exposure to arsenic from ingestion of locally raised meat was calculated assuming uptake into cattle from ingestion of arsenic in soil and forage, using the following equations and assumptions:

$$meat \quad [\ soil \quad soil\ cattle \quad (\ soil \quad fora\ e \quad fora\ e\ cattle)] \quad meat$$

$$is_{meat} \quad \left(\frac{meat \quad meat \quad [(\ meat\ c \quad c) \quad (\ meat\ a \quad a)]}{} \right)$$

Estimates of uptake of arsenic into muscle of beef cattle were based on a study by Bruce et al. (2003) in which cattle were grazed in paddocks that had known concentrations of arsenic in soil from contamination by mine tailings. The grazing trial continued for approximately 8 months (237 days) and periodic samplings of blood and biopsy of the liver and muscle from the animals were conducted to monitor the accumulation of metals. The authors cite three possible pathways for cattle to ingest arsenic from the tailings: (1) ingesting pasture plants that have accumulated heavy metals in their leaf and stem tissues, (2) directly ingesting contaminated soil that has adhered to pasture plant surfaces, and (3) either deliberate direct ingestion of tailing material or accidental ingestion during grazing. Dose rates for arsenic in ingested plants and arsenic adhered to plants were calculated based on an estimate of dry matter intake per day (2.5% of body weight per day) and the average weight of the animals during the trial (331 kg). The authors also state that direct ingestion of soil (not associated with plant material) may be up to 10% of the daily dry matter intake, equating to as much as 1 kg of soil per day. Per Bruce et al. (2003), "This is consistent with earlier work done by Thornton and Abrahams (1983), who reported arsenic in soil being a more important contamination pathway than via plant material."

Overall, accumulation of arsenic in cattle was greater in organs such as the liver and kidneys. The measured concentration in muscle, relative to a unit intake of 1 mg/kg-BW-d, was 0.097 (mg/kg-muscle)/(mg/kg-BW-d).

Based on this information, the following values for exposure parameters for this pathway were assumed:

- *Ingestion rate of soil by cattle ($IR_{soil\text{-}cattle}$)*: 0.002 kg-soil/kg-BW-d. This value is based on the average soil ingestion rate by grazing cattle of 0.825 kg/day, divided by an assumed average body weight of 350 kg; (Mayland et al., 2005).
- *Ingestion rate of forage by cattle ($IR_{forage\text{-}cattle}$):* 0.017 kg-forage (DW)/kg-BW-d. This value is based on data from NRC (1987) indicating that an 800 pound (364 kg) animal consumes about 8.1- 9.8 kg dry matter/d and a 1,000 pound (454 kg) animal consumes about 8.8- 11.5 kg dry matter/d. These values are in turn predicted by a number of different models that take into account differences in starting body weight, frame size, and net energy of the feed. These values are equivalent to a range of 0.019-0.027 kg dry matter/kg-d and are consistent with U.S. EPA's *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities* which recommends assuming 11.8 kg total daily dry matter intake by beef cattle (cattle weight unspecified), of which 8.8 kg/d (75%) is forage, 2.5 kg/d (21%) is silage, and 0.47 kg/d (4%) is grain (U.S. EPA 2005a). For these calculations, we assume an average forage ingestion rate of 0.017 kg dry matter/kg-d (0.023 kg × 75%).
- *Arsenic plant/soil bioconcentration factor for forage (B_{forage}):* 0.036 mg/kg-plant (DW)/mg/kg soil. This is an arsenic-specific value presented in U.S. EPA (2005), and is consistent with the value for uptake of arsenic from soil into above ground exposed produce assumed above of 0.026

mg/kg plant (DW)/mg/kg soil.

- *Arsenic intake/ muscle bioconcentration factor for cattle (B_{meat}):* 0.097 mg/kg-plant (DW)/mg/kg soil. This is based on the measured concentration in muscle, relative to a unit intake of 1 mg/kg BW-d, reported in Bruce et al. (2003).

The consumption rate of beef was based on the mean per capita intake of home-produced meats reported for U.S. populations that farm or raise animals (U.S. EPA, 2011b). These values were as follows:

- *Consumption rate of aboveground exposed meat* (IR_{meat}): Values of 0.59 g/ kg-BW-d (fresh weight) for adults and 1.2 g/kg-BW-d (fresh weight) for children were assumed. These values represent the mean per capita intake of home-produced meats reported for U.S. populations that farm or raise animals for ages 20-69 (adults) and 2 to 9 (children) (U.S. EPA, 2011b). Assuming a typical adult body weight of 70 kg, the adult value equates to 41 g/d (or 1.5 ounces/d), which is equivalent to consumption of about 10 ounces of homegrown meat per week.

Since this consumption rate is based on data for consumption of home-produced meats, it is assumed that 100% of the home-produced meat consumed is from a contaminated source (F_{meat}).

3.1.2.2 Contributions from Surface or Ground Water and Sediment

Incidental Ingestion of Surface or Ground Water

Uptake from incidental ingestion of surface or ground water was calculated using the following equation and assumptions:

$$is_{water} \left(\frac{water \quad water \quad c \quad water \quad c}{c} \quad \frac{water \quad water \quad a}{a} \right)$$

Where Abs_{water} is the absorbed dose in mg/d.

The incidental ingestion rate of surface or ground water not used as a drinking water source was assumed to be 0.05 L/d, the U.S. EPA default incidental water ingestion rate (U.S. EPA, 1989). This is equivalent to about 1.7 fluid ounces or 3.4 tablespoons per exposure event

The relative bioavailability (RBA) of arsenic in ingested water was assumed to be 100% (i.e., the same as in the studies that are the basis for the toxicity criterion).

It is assumed that for adults, based on professional judgment and the assumption that residents would occasionally but infrequently contact surface water, the exposure frequency (EF) to water contact is six times/year (one day per month for six months). For children, EF was assumed to be 18 times/year (two days per month for six months plus one day per month for six months).

Incidental Dermal Contact with Surface or Ground Water

Uptake from incidental dermal contact with surface or ground water was calculated using the following equation and assumptions:

$$S_{water\ c} \qquad water$$

$$S_{water\ a} \qquad water$$

$$is_{water} \quad \left(\frac{S_{water\ c} \qquad water\ c \qquad c}{c} \quad \frac{S_{water\ a} \qquad water\ a \qquad a}{a} \right)$$

Where $Abs_{water\ (c)}$ and $Abs_{water\ (a)}$ are the absorbed doses in mg/event for the child and the adult, respectively.

The skin surface area available for water contact (SA) is based on data presented in U.S. EPA's *Exposure Factors Handbook* (U.S. EPA, 2011b), and assumes contact during wading or contact from the shore. It is based on the following:

- For adults, the value (4,560 cm^2/event) assumes contact by hands, lower arms, lower legs, and feet for 50% of events, and hands and lower arms for 50% of events, based on mean surface area by body part averaged for males and females.
- For children, the value (1,750 cm^2/event) assumes contact by head, hands, lower arms, feet, and lower legs for 50% of events, and hands and lower arms for 50% of events, based on mean surface area by body part averaged for children ages 2 to 6.

The percutaneous absorption factor (Kp) was assumed to be 0.001 cm/hr and is a U.S. EPA default value based on the assumption that water-soluble compounds have a human skin permeability constant that is unlikely to exceed 0.001 cm/hr (U.S. EPA, 1992b).

It is assumed that for adults, the exposure time (ET) to water contact is one-half hour per event and for children it is one hour per event.

Like the incidental ingestion pathway, it is assumed that for adults, based on professional judgment and the assumption that residents would occasionally but infrequently contact surface water, that the exposure frequency (EF) to water contact is six times/year (one day per month for six months) and for children it is 18 times/year (two days per month for six months and one day per month for six months).

Incidental Ingestion of Sediment

Uptake from incidental ingestion of sediment was calculated using the following equations and assumptions:

$$S_{sed\ in\ c} \qquad sed \qquad sed\ c \qquad sed$$

$$S_{sed\ in\ a} \qquad sed \qquad sed\ a \qquad sed$$

$$\left(\frac{S_{sed\ in\ c} \qquad sed\ c \qquad c}{c} \quad \frac{S_{sed\ in\ a} \qquad sed\ a \qquad a}{a} \right)$$

Where $Abs_{sed\text{-}ing\ (c)}$ and $Abs_{sed\text{-}ing\ (a)}$ are the absorbed doses from ingestion of sediment in mg/day in the child and the adult, respectively.

The ingestion rate of sediment was assumed to one half of the ingestion rate of soil, or 50 mg/d for adults and 100 mg/d for children.

The relative bioavailability (RBA) of arsenic in sediment was assumed to be the same as that assumed for soil (20.3%).

As with the water contact pathways, it is assumed that for adults, based on professional judgment and the assumption that residents would occasionally but infrequently contact surface water, the exposure frequency (EF) to sediment is six times/year (one day per month for six months) and for children it is 18 times/year (two days per month for six months and one day per month for six months).

Incidental Dermal Contact with Sediment

Uptake from incidental dermal contact with sediment was calculated using the following equation and assumptions:

$$S_{sed\ derm\ c} \quad sed \quad sed\ c \quad sed$$

$$S_{sed\ derm\ a} \quad sed \quad sed\ a \quad sed$$

$$iS_{sed} \quad \left(\frac{S_{sed\ derm\ c} \quad c}{c} \quad \frac{S_{sed\ derm\ a} \quad sed\ a \quad a}{a} \right)$$

Where $Abs_{sed\text{-}derm\ (c)}$ and $Abs_{sed\text{-}derm\ (a)}$ are the absorbed doses from dermal contact with sediment in mg/day in the child and the adult, respectively.

The skin surface area available for sediment contact (SA) is based on data presented in U.S. EPA's *Exposure Factors Handbook* (U.S. EPA, 2011b), and assumes contact during swimming or wading, or contact from the shore. It is based on the following:

- For adults, the value (2,960 cm^2/event) assumes contact by hands, lower arms, and feet for 50% of events, and hands and lower arms for 50% of events, based on mean surface area by body part averaged for males and females.
- For children, the value (1,7500 cm^2/event) assumes contact by head, hands, lower arms, feet, and lower legs for 50% of events, and hands and lower arms for 50% of events, based on mean surface area by body part averaged for children ages 2 to 6.

The sediment-to-skin adherence factor (AF) describes the mass of sediment that adheres to skin per square centimeter of skin surface. The assumed value (0.18 mg/cm^2) is from U.S. EPA (2000a) and represents a reasonable upper-bound adherence factor for all exposed skin. This value is higher than that for soil because it is assumed that sediment is wet and so is more adherent.

The same dermal absorption factor (ABS) as used for the soil dermal contact pathway (0.032) was used, and is U.S. EPA's chemical-specific default for dermal contact with arsenic (U.S. EPA, 2004a).

It is assumed that for adults, the exposure frequency (EF) to sediment contact is six times/year and for children it is 18 times/year.

3.1.3 *Revised Exposure Assumptions for Soil and Dust Ingestion Pathways*

Relative bioavailability was recalculated using the data presented in Freeman et al. (1995) and the following equation:

$$\text{of test material} \quad \frac{\text{rinary e cretion fraction of arsenic in test material soil or water}}{\text{rinary e cretion fraction of sodium arsenate in water}}$$

Using the monkey data, which have a number of uncertainties as discussed in Section 2.2.4.2, results in RBA estimates of 20.3 ± 3.4% for soil and 29.1 ± 0.7% for dust. However, as noted in Sections 2.2.4.2 and 2.2.4.4, the bioavailability estimates from the monkey study are likely to underestimate arsenic bioavailability at the site.

In addition, the assumed relative concentration of arsenic in dust (43%) was changed based on the Pioneer Technical Services (2009) study. This study indicates that the average relative indoor dust to outdoor soil concentration ratio, combining all data for Opportunity, Anaconda West, and Anaconda East, was 130%. This value was applied in the current assessment. Particularly in Anaconda homes, concentrations in attic dust tended to be much higher than those in dust in the main living area; however, the enrichment in attic dust did not tend to be quite as high in the Opportunity, and without more data, it was assumed that contact with attic dust would be minimal. Thus, a separate assumption for exposure to attic dust was not included in this assessment. This may underestimate risks in homes where attic dust concentrations are particularly high and may be reentrained into the air, or occupants regularly spend time in the attic.

Other exposure assumptions were not changed from the CDM (1996) Baseline HHRA for the following reasons:

- **Soil ingestion rate.** The RME soil ingestion rates (100 mg/d for adults and 200 mg/d for children) were not changed as no additional site-specific data were identified that warrant an adjustment to this parameter, and these remain the U.S. EPA recommended default assumptions for this scenario (U.S. EPA, 1996b; U.S. EPA, 2012d).
- **Fraction ingested that is soil and dust.** The assumed fraction that is ingested that is soil and dust (45% soil and 55% dust) was not changed, as no additional site-specific data were identified that warrant an adjustment to this parameter, and U.S. EPA's current exposure assessment guidance (U.S. EPA, 2011b) continues to recommend this assumption.
- **Exposure duration.** The RME exposure duration values (24 years as an adult and 6 years as a child) were not changed as no additional site-specific data were identified that warrant an adjustment to this parameter and these remain the U.S. EPA recommended default assumptions for this scenario (U.S. EPA, 1996b; U.S. EPA, 2012d). However, based on site-specific information suggesting a relatively longer residence time for people who live in the Opportunity Community, these assumptions will not overestimate typical site-related risks and may underestimate risks for some members of the population.
- **Exposure frequency.** The standard RME exposure frequency value (350 d/year) was not changed as no additional site-specific data were identified that warrant an adjustment to this parameter and this remains the U.S. EPA recommended default assumptions for this scenario (U.S. EPA, 1996b; U.S. EPA, 2012d).
- **Body weight.** The RME body weight values (70 kg for an adult and 15 kg for a child) were not changed as no additional site-specific data were identified that warrant an adjustment to this parameter and these remain the U.S. EPA recommended default assumptions for this scenario (U.S. EPA, 1996b; U.S. EPA, 2012d).

3.2 Recalculation of Site-Related Risks

Site-related risks recalculated for Opportunity Community residents based on the assumptions described above are summarized in Table 8.

ED_001802_00024760-00045

Table 8. Summary of Pathway-Specific Lifetime Excess Cancer Risks for Opportunity Community Residents, RME Scenario

Pathway	Cancer Risk	Percent of Total Risk
Ingestion of soil	3.9×10^{-5}	15.9%
Ingestion of dust	9.0×10^{-5}	36.2%
Dermal contact with soil	1.7×10^{-6}	0.70%
Dermal contact with dust	1.0×10^{-6}	0.41%
Inhalation of soil particulate	2.5×10^{-7}	0.10%
Ingestion of produce	9.5×10^{-5}	38.4%
Ingestion of meat	1.7×10^{-5}	6.9%
Total soil-related risks	$\mathbf{2.4 \times 10^{-4}}$	**98.6%**
Ingestion of sediment	1.8×10^{-6}	0.73%
Dermal contact with sediment	1.2×10^{-6}	0.47%
Total sediment-related risks	$\mathbf{3.0 \times 10^{-6}}$	**1.2%**
Ingestion of surface or ground water	4.2×10^{-7}	0.17%
Dermal contact with surface or ground water	1.6×10^{-8}	0.01%
Total surface or ground water-related risks	$\mathbf{4.4 \times 10^{-7}}$	**0.18%**
TOTAL	$\mathbf{2.5 \times 10^{-4}}$	

As shown, based on the assumptions described above, four exposure pathways contribute significantly (i.e., >1%) to the total risk estimate: ingestion of soil (15.9%), ingestion of dust (36.2%), ingestion of produce (38.4%), and ingestion of meat (6.9%). All the other pathways contribute <1% to the total risk estimate but estimated risks for four other pathways (dermal contact with soil, dermal contact with dust, ingestion of sediment, and dermal contact with sediment) exceed U.S. EPA's *de minimis* lifetime excess cancer risk level of 1 in 1,000,000 (1×10^{-6}).

3.3 Recalculation of the Soil Screening Level

Overall, non-soil related pathways were estimated to contribute insignificantly to overall risk (i.e., water and sediment-related pathways were estimated to contribute <1% to the total risk). As a result, soil cleanup levels were calculated taking into account only the contribution of the soil-related pathways to total lifetime excess cancer risk from exposure to environmental arsenic. Because there is a linear relationship between soil concentration and the estimated lifetime excess cancer risk for these pathways, a soil cleanup level associated with a target cancer risk can be calculated based on the ratio of the risk estimated for the exposure pathways shown in Table 8 at the assumed soil concentration (i.e., the 95% UCL), as follows:

Rearranging the equation yields:

In regard to target risk, U.S. EPA has stated that a lifetime excess cancer risk of 10^{-6} (1 in 1,000,000) is recommended as the point of departure. For example, Subpart E of the National Contingency Plan (1990) states:

> EPA will set remediation goals for total risk due to carcinogens that represent an excess upperbound lifetime cancer risk to an individual to between 10^{-4} to 10^{-6} lifetime excess cancer risk. A cancer risk of 10^{-6} will serve as the point of departure for these remediation goals.

U.S. EPA's *Risk Assessment Guidance for Superfund* (RAGS) outlines the fundamental methodology for conduct of risk assessments under Superfund. RAGS Part B (U.S. EPA, 1991) provides guidance on methodologies for developing risk-based preliminary remediation goals (PRGs) and states:

> For carcinogenic effects, a concentration is calculated that corresponds to a 10^{-6} incremental risk of an individual developing cancer over a lifetime as a result of exposure to the potential carcinogen from all significant exposure pathways for a given medium (Chapter 2, p. 14).

And, further:

> When the cumulative current or future baseline cancer risk for a medium is within the range of 10^{-6} to 10^{-4}, a decision about whether or not to take action is a site-specific determination (Chapter 2, p. 15).

RAGS Part B further specifies

> …in the absence of ARARS, the 10^{-6} cancer risk 'point of departure' is used as a starting point for analysis of remedial alternatives which reflects EPA's preference for managing risks at the more protective end of the risk range, other things being equal (Chapter 2, p. 18).

U.S. EPA (1996b) (*Soil Screening Guidance*) specifies that for calculation of soil screening levels for residential soil, the "screening level" equation corresponds to a target risk level of 10^{-6}. Overall, based on this guidance, a target risk level that does not exceed 10^{-5} (1 in 100,000) to 10^{-6} (1 in 1,000,000) is recommended.

ED_001802_00024760-00047

Based on the calculated lifetime excess cancer risks for the soil pathways associated with a 95% UCL soil concentration of 184 ppm of 2.4×10^{-4} and a target cancer risk of 1.0×10^{-5}, a soil screening level of approximately 8 ppm is calculated.

oil screenin le el m _____ *ppm*

Thus, based on the assumptions described above, a soil cleanup level of 8 ppm is calculated associated with a lifetime excess cancer risk of 1×10^{-5} (1 in 100,000).

4.0 COMPARISON TO CLEANUP LEVELS AT OTHER SITES

Numerous states have established default soil cleanup levels for arsenic in soil, based on assumed acceptable cancer risk levels and default (non-site specific) exposure assumptions. Cleanup levels in soil for residential/unrestricted use, shown in Table 9, range from 0.039 to 40 ppm. Most of these values are based on an assumed acceptable cancer risk of 1×10^{-5} (1 in 100,000) or 1×10^{-6} (1 in 1,000,000).

Table 9. Soil Cleanup Levels for Arsenic in Soil for Residential/ Unrestricted Use for U.S. States[a]

State	Cleanup Level (ppm)	Basis
WI	0.039	Cancer (10^{-7} risk level), standard risk assessment assumptions and toxicological guidance values
CA	0.07	Cancer (10^{-6} risk level), 4% dermal absorption assumption, California Slope Factors
AL, CO, DE, ID, LA, MD, MS, NC, OK, OR, VA, WV, WY	0.38-0.41	Cancer (10^{-6} risk level), either direct cite to EPA, or state-specific calculation with standard risk assessment assumptions and toxicological guidance values
ME	1.4	Cancer (10^{-5} risk level), California Slope Factors
FL	2.1	Cancer (10^{-6} risk level), 33% oral bioavailability, state-specific exposure assumptions
NM	3.59	Cancer (10^{-5} risk level), standard risk assessment assumptions and toxicological guidance values
IN	3.9	Noncancer soil-plant-human uptake (based on U.S. EPA soil screening guidance)
OH	6.7	Cancer (10^{-5} risk level), 3% dermal absorption assumption
AZ, IA, KS, KY, MA, MN, MO, MT, NH, NJ, NY, PA, RI, WA	7 to 40	State-specific natural background
TX	24	Noncancer (lower than cancer endpoint at 10^{-4} risk; 34 ppm)

[a] Source: Teaf et al. (2010) and research of values set by state agencies.

The U.S. EPA Regional Screening Level (RSL) for arsenic is 0.39 ppm (U.S. EPA, 2012d), based on a lifetime excess cancer risk of 1×10^{-6}. U.S. EPA defines RSLs as follows:

> They are risk-based concentrations derived from standardized equations combining exposure information assumptions with EPA toxicity data. SLs are considered by the Agency to be protective for humans (including sensitive groups) over a lifetime; however, SLs are not always applicable to a particular site and do not address non-human health endpoints, such as

ecological impacts. The SLs contained in the SL table are generic; they are calculated without site-specific information. They may be re-calculated using site-specific data.

The RSL is derived based on the following assumptions:

- Pathways: soil ingestion, inhalation of particulate, dermal contact with soil.
- Oral cancer SF = 1.5 $(mg/kg\ BW\text{-}d)^{-1}$
- 100% oral bioavailability (U.S. EPA default)
- 3% dermal bioavailability (U.S. EPA default for metals)
- Exposure frequency: 350 d/yr
- Exposure duration: 30 years
- Averaging time: 70 year lifetime
- Particulate emission factor (PEF) = 1.4E+09 m^3/kg (EPA default)
- Soil ingestion rate: 200 mg/d for 6 years by a 15 kg child, then 100 mg/d for 24 years by 70 kg adult
- Dermal contact rate: 2800 cm^2/d and absorption fraction of 0.2 mg/cm^2 for 6 years and 5700 cm^2/d and absorption fraction of 0.07 mg/cm^2 for 24 years

Pathway-specific RSLs are 0.43 ppm for soil ingestion, 770 ppm for inhalation of particulate, and 4.5 ppm for dermal contact (U.S. EPA, 2012d).

Table 10 lists site-specific action levels established for arsenic in soil at other U.S. NPL sites. These sites were identified based on a review of arsenic RODs conducted by Davis et al. (2001), a summary prepared by the Agency of Toxic Substances and Disease Registry (ATSDR) on action levels presented in RODs conducted as part of a Health Consultation to evaluate the residential soil arsenic level at the Anaconda site (ATSDR, 2007), and searches of U.S. EPA's ROD database. The table also identifies the type of site (e.g., smelting, mining, wood treating, manufacturing), the basis of the action level where available, the assumed cancer risk level, and the site-specific or local background arsenic soil concentration.

Table 10. Action Levels for Arsenic in Soil at Selected U.S. Sites

Site	Type of Site/ Source of Contamination	Date of ROD	Site-Specific Arsenic Action Level in Soil (ppm)	Risk Level	Local Background Arsenic Soil Concentration (ppm)	Reference
McCormick & Baxter Creosoting Co. (Portland, OR)	Creosote wood treating	1996	8, based on background concentration	2×10^{-6} for an industrial scenario, assuming soil ingestion and dermal contact	8	U.S. EPA, 1996c
Bunker Hill Site (Smelterville, ID)	Inactive mine and mill, lead and zinc smelter, and phosphoric acid fertilizer plant	1992	10 (OU3), risk based concentration and equal to background 100 (OU2), clean replacement soil must contain less than this concentration; ROD for site is based on soil lead concentration of 100	NA	10	U.S. EPA, 1992c; U.S. EPA, 2013
Tacoma Smelter (Commencement Bay, Near Shore/Tide Flats Superfund Site, Ruston, WA)	Lead and copper smelter	1993	20, for child play areas 100, WA DOE level for residential yards 230, U.S. EPA level, propose to use community education to protect below this level	3.0×10^{-5} (based on urban background) 1.5×10^{-5} (set because WA DOE does not have resources to cleanup to 20 ppm) 5×10^{-4}	20	WA DOE, 2011

ED_001802_00024760-00050

Site	Type of Site/ Source of Contamination	Date of ROD	Site-Specific Arsenic Action Level in Soil (ppm)	Risk Level	Local Background Arsenic Soil Concentration (ppm)	Reference
Lava Cap Mine (Nevada County, CA)	Hardrock gold and silver mine; tailings	2004	20 (Mine Area OU), based on background	Between 10^{-4} and 10^{-6} assuming exposure through ingestion (soil, sediment, surface/ground water, dust), dermal contact, and inhalation	20	U.S. EPA, 2004b
Koppers Co. (Oroville, CA)	Wood treating (copper chromated arsenate)	1996	21, for worker exposure (surface soil up to five feet below ground)	1×10^{-5} (industrial worker exposure to surface soil)	7.15	U.S. EPA, 1996d
Wingate Road Municipal Incinerator Dump (Fort Lauderdale, FL)	Municipal incinerator and landfill	1996	23 (for surface soil); (for sediment); scenario not identified	"Fall within EPA's risk range" of 1×10^{-6} to 1×10^{-4}	1.4	U.S. EPA, 1996e
Cleveland Mill (Silver City, NM)	Former ore processing area adjacent to Cleveland Mine; tailings	1993	30 based on background level	NA	30	U.S. EPA, 1999a
RSR Corp. (Dallas, TX)	Lead smelter	1996	32.7, for worker exposure	1×10^{-5} (not based on 1×10^{-6} because that results in cleanup level below background concentrations; industrial exposure through soil ingestion and dermal contact)	NA	U.S. EPA,1996f

ED_001802_00024760-00051

Site	Type of Site/ Source of Contamination	Date of ROD	Site-Specific Arsenic Action Level in Soil (ppm)	Risk Level	Local Background Arsenic Soil Concentration (ppm)	Reference
Rentokil Wood Preserving (Richmond, VA)	Wood treating using chromium zinc arsenate and CCA	1993	33, for worker exposure	1×10^{-6} (worker exposure to soil through ingestion, dermal contact, and inhalation of particulate)	NA	U.S. EPA, 1993b
Joseph Forest Products (Wallowa County, OR)	Wood treating using chromated copper arsenate	1992	36 for surface soils, industrial use; 336 for subsurface soils (deeper than 3 feet), for industrial use (OU1)	1×10^{-5} for industrial PRG for surface soils, approximately equal to 1×10^{-4} for residential scenario; pathways not identified	4-11	U.S. EPA, 1992d
Talache Mine Tailing Site (Atlanta, ID)	Tailings pile; saturation caused "blowout"	Cleanup alternative selected 2000	36, for residential exposure (future proposed use) and based on background	Site-specific cleanup goals	36	U.S. EPA, 2001a
Fremont National Forest/ White King and Lucky Lass Uranium Mines (Lakeview, OR)	Mining	2001	38, recreational use (no current or likely future residential use of site)	1×10^{-6} based on recreational user scenario	6	U.S. EPA, 2001b
Kennecott South Zone (Copperton, UT)	Mining	1999	50-100, residential, daycare, and playgrounds	$<1 \times 10^{-4}$ (50 ppm is generic cleanup level protective for all mining waste sites in the county absent site specific information; bioavailability based on juvenile swine study	16.3 (mean; 23.6 maximum)	U.S. EPA, 1999b

ED_001802_00024760-00052

Site	Type of Site/ Source of Contamination	Date of ROD	Site-Specific Arsenic Action Level in Soil (ppm)	Risk Level	Local Background Arsenic Soil Concentration (ppm)	Reference
National Zinc Corp. (Bartlesville, OK)	Zinc smelting	1995	60, residential	3×10^{-5} (assumed 25% relative oral bioavailabilty based on Anaconda site values)	8	U.S. EPA, 1995a
Midvale Slag (Salt Lake City, UT)	Smelting	1995, 2002	61-73, residential	1×10^{-4} (for soil ingestion only) (80% relative bioavailability factor for soil and dust; U.S. EPA default for Region 8)	NA	U.S. EPA, 1995b; U.S. EPA, 2002a
Sharon Steel Corp. (Midvale, UT)	Smelting and milling	1990	70, for residential exposure	2.6×10^{-5}	<20	U.S. EPA, 1990b
Asarco Globe Plant (Denver, CO)	Gold, silver, lead, and copper smelter	1993	70 for community soils	8×10^{-5}, based on U.S. EPA default (RAGS) methodology; inhalation, soil ingestion, and ingestion of garden vegetables. Oral bioavailability factor for soil = 80% (site used for arsenic trioxide production)	28	Colorado Dept. of Health, 1993
Vasquez Boulevard and I-70 (Denver, CO)	Smelting	2003	70, residential	1×10^{-5} at background level of 15 ppm; preliminary action level was 47 ppm, level at which HQ for noncancer effects exceeds 1; based on soil and dust and garden vegetable ingestion; site-specific relative oral bioavailability estimate of 42% based on juvenile swine study	8-15	U.S. EPA, 2003a

ED_001802_00024760-00053

Site	Type of Site/ Source of Contamination	Date of ROD	Site-Specific Arsenic Action Level in Soil (ppm)	Risk Level	Local Background Arsenic Soil Concentration (ppm)	Reference
Woolfork Chemical Works, Inc. (Fort Valley, GA)	Pesticide production, packaging, and storage	1995, 1998, 2004	71 for indoor dust; 100 for properties proposed for non-industrial development; 113 for a future industrial worker; 317 for paved soils	1×10^{-5} for future industrial worker scenario	5 in southern U.S.	U.S. EPA, 1994b; U.S. EPA, 1995c; U.S. EPA, 1998b
Atlantic Wood Industries (Portsmouth, VA)	Wood treating	2007 (originally 1995)	76, for worker exposure	5×10^{-6} for industrial exposure scenario (soil ingestion, dermal contact with soil, and inhalation of soil particulates by on-site workers)	"below cleanup levels for the site)	U.S. EPA, 1995d; U.S .EPA, 2007b
Upper Tenmile Creek Mining Area (Helena, MT)	Mining	2002	96, residential	Risk-based concentrations for residential scenario, RME: 1×10^{-6} = 0.6 ppm; 1×10^{-5} = 6 ppm; 1×10^{-4} = 60 ppm (default bioavailability); RME: 1×10^{-6} = 12 ppm; 1×10^{-5} = 120 ppm; 1×10^{-4}= 1,200 ppm (using Butte and Anaconda bioavailability estimates) (soil and dust ingestion, inhalation of particulate, surface and groundwater ingestion)	50	U.S. EPA, 2002b
Whitewood Creek (Whitewood, SD)	Gold mining and milling; tailings	1990	100, for residential exposure	1×10^{-4}; assumes residential exposure; oral bioavailability in soil = 50%; soil ingestion pathway only	NA	U.S. EPA, 1990a

Site	Type of Site/ Source of Contamination	Date of ROD	Site-Specific Arsenic Action Level in Soil (ppm)	Risk Level	Local Background Arsenic Soil Concentration (ppm)	Reference
Jacobs Smelter (Stockton, UT)	Smelting/milling (at least nine operations, including silver)	1999	100, residential	$<1 \times 10^{-4}$; Risk-based range for residential scenario = 1.2-117 ppm; "very bioavailable" as lead arsenic oxide	NA	U.S. EPA, 1999c
Blackbird Mine (Lemhi County, ID)	Mining	2003	100, residential scenario (based on background)	Risk-based concentration for residential scenario = 42 ppm	100	U.S. EPA, 2003b
California Gulch (Lake County, CO; includes Leadville)	Mining/ smelting	1998, 2000	120-340 (residential), 330-1,300 (commercial/ industrial) (OU3, OU7)	NA	1.2-24 (CO background)	U.S. EPA, 1998c; U.S. EPA, 2000b
Davenport and Flagstaff Smelters (Sandy, UT)	Smelting	2002	126, residential	1×10^{-4} (based on direct contact, ingestion, or inhalation of soil)	NA	U.S. EPA, 2002c
Clark Fork River (Deer Lodge, MT)	Mining and smelting	2004	150, recreational	1×10^{-4} (based on ingestion and direct contact with soil)	NA	U.S. EPA, 2004c
Kennecott North Zone (Magna, UT)	Smelting	2002	200(site is zoned manufacturing, heavy industrial, mining)	At 261 ppm, 1×10^{-5} (industrial scenario)	NA	U.S. EPA, 2002d

ED_001802_00024760-00055

Site	Type of Site/ Source of Contamination	Date of ROD	Site-Specific Arsenic Action Level in Soil (ppm)	Risk Level	Local Background Arsenic Soil Concentration (ppm)	Reference
Rockwool Industries Inc. (Belton, TX)	Household mineral wool insulation manufacturing	2004	200, for worker exposure	$<5 \times 10^{-4}$; Industrial (worker) exposure; site is zoned for industrial use; based on direct contact)	NA	U.S. EPA, 2004d
Puget Sound Naval Shipyard (Bremerton, WA)	Shipyard	1997	219 (OU1) based on State of Washington Model Toxic Control Act, Method C, Industrial Cleanup Values for Soils, although current MTCA values is 20	Current MTCA value = 20 ppm for 1×10^{-6}; based on soil ingestion only, and protection of groundwater for drinking water use	7.5	U.S. EPA, 1997
US DOE Hanford Area 300 (Benton County, WA)	Nuclear fuels fabrication	1996	219 (OU 1 and 2), based on State of Washington Model Toxic Control Act, Method C, Industrial Cleanup Values for Soils, although current MTCA value is 20	Current MTCA value = 20 ppm at 1×10^{-6}; based on soil ingestion only, and protection of groundwater for drinking water use	2.2	U.S. EPA, 1996g
Silver Bow Creek/ Butte (Butte, MT)	Mining, smelting, industrial and municipal wastes	1990	250 mg/kg, residential	5.3×10^{-5}; based on soil ingestion and dermal contact, inhalation of particulate, and dermal absorption from water; relative oral bioavailability = 18% for soil and 25% for dust	NA	U.S. EPA, 1990c

ED_001802_00024760-00056

Site	Type of Site/ Source of Contamination	Date of ROD	Site-Specific Arsenic Action Level in Soil (ppm)	Risk Level	Local Background Arsenic Soil Concentration (ppm)	Reference
Rhone-Poulenc/ Zoecon (East Palo Alto, CA)	Pesticide manufacturing	1992	300, but will conduct cleanup on Sandoz and Bains properties with arsenic >70	2×10^{-4}; institutional controls prohibit future residential use	9	U.S. EPA, 1992e
Triumph Mine Tailings (Triumph, ID)	Mining tailings piles	1998	300, residential	1×10^{-4} based on soil ingestion and inhalation; "protective whether arsenic bioavailability is assumed to be 16, 28, or 60 percent" (from 1 to 5×10^{-4} depending on bioavailability)	NA	U.S. EPA, 1998d

5.0 IMPACT OF ALTERNATIVE EXPOSURE ASSUMPTIONS

Based on the equations and assumptions presented in the 1996 Baseline HHRA, the action level for arsenic in soil at the Anaconda Smelter NPL site of 250 ppm equates to a lifetime excess cancer risk estimate of 8.4×10^{-5}. If this soil concentration is used with the equations and assumptions presented in this assessment (Section 3.0), the estimated lifetime excess cancer risk would be 3.6×10^{-4}. The latter risk estimate exceeds 1×10^{-4} and is outside of the range that US EPA, in general, deems appropriate. Further, it exceeds nearly all of risk estimates associated with the arsenic action levels identified in RODs for other sites (Table 10) (the only exceptions are the action levels for the Tacoma Smelter site, which is not applied to residential yards; the Rockwool Industries site, which is zoned for industrial use; and the Rhone-Poulenc Zoecon site, which has institutional controls prohibiting future residential use). The risk assessment I developed herein is based on U.S. EPA guidance, incorporating additional likely-to-be complete exposure pathways and correctly calculated variables were appropriate. In addition, my risk assessment incorporates assumptions based on additional defensible sources of information, including the scientific literature and risk assessment guidance as well as a site visit and a survey of members of the Opportunity community. The result of this risk assessment is that the estimated screening level for arsenic soil decreased from 250 ppm to approximately 8 ppm. Following Agency's policy, then this change would require remediation of contaminated soil in Opportunity to an acceptable level.

I selected many exposure parameters to reflect mean or median values (e.g., meat and produce ingestion rates, soil contact rates), as opposed to upper bound estimates, and to reflect realistic estimates of site-related exposures. However, different values could be used for several key parameters in the risk calculations. To assess the potential impact of alternative values for these parameters, I explored the use of alternative values for several exposure parameters that could significantly impact the total estimated risk, focusing on important parameters in the four most significant exposure pathways identified in Section 3.2. These exposure parameters are as follows:

- Relative oral bioavailability factor for soil
- Relative oral bioavailability factor for dust
- Relative concentration of arsenic in dust compared to soil
- Soil and dust ingestion rates
- Produce ingestion rates
- Meat ingestion rates

The alternatives assumptions and effects on the risk calculations and screening levels are discussed below.

5.1 Relative Oral Bioavailability (RBA) Factors

As shown in Table 4, the RBA values applied in the CDM (1996) Baseline HHRA are at the lower end of RBA estimates for arsenic that have been reported in *in vivo* studies conducted using soil from mining or smelting sites: values for other sites where soil was contaminated by mining or smelting activity range from 5% to 98%, with a mean value for the 24 sites of 34.9%. The mean value for the six sites where studies were conducted in monkeys was 14.2%, whereas the mean value for the remaining 18 sites where studies were conducted in swine or mice was 42.4%. As discussed in Section 2.2.4.2 and 2.2.4.4, a number of methodological concerns with the monkey studies suggest

ED_001802_00024760-00058

that use of the monkey model is more likely (than not) to underestimate the oral bioavailability of arsenic in soils.

While U.S. EPA (2012b) recommends using site-specific bioavailability data when available, concerns with the site-specific data suggest reconsideration of the RBA estimates applied in the CDM (1996) Baseline HHRA may be warranted.

If an alternative oral bioavailability factor for soil and dust equal to the mean soil RBA value for all the smelting/ mining sites is used (35%), the resulting lifetime excess cancer risk for the Opportunity Community residential scenario (based on the equations and assumptions described in Section 3.0) increases to 3.0×10^{-4}, and the calculated soil screening level at a target cancer risk of 1×10^{-5} decreases to 6.1 ppm. Note that Freeman et al. (1995) suggests the RBA for arsenic in dust may be greater than that in soil; however, no dust RBA data for any other sites were identified, and so a value based on soil was applied to dust in this recalculation. This may underestimate uptake of arsenic from dust at the site.

5.2 Relative Concentration of Arsenic in Dust

As discussed in Section 2.2.3, data on arsenic concentrations in soil and dust collected in the Community Soils OU in 2006 and 2007 (Pioneer Technical Services, 2009) suggest that concentrations of arsenic in indoor dust were relatively higher than in outdoor soil. In the reassessment of residential risks, a value for the relative concentration of arsenic in dust of 130% is assumed, based on the average relative concentration for the sampled homes in Opportunity, Anaconda West, and Anaconda East. However, in some cases, the relative concentration of arsenic in indoor dust was much higher, and the average relative concentration of arsenic in indoor dust vs outdoor soil is relatively higher in Opportunity (170%) when considered separately from the Anaconda homes (average 110%).

If an alternative value for relative concentration of arsenic in indoor dust of 170% is used, the resulting lifetime excess cancer risk for the Opportunity Community residential scenario (based on the equations and assumptions described in Section 3.0) increases to 2.8×10^{-4}, and the calculated soil screening level at a target cancer risk of 1×10^{-5} decreases to 6.6 ppm.

Available data suggest that the relative concentrations of arsenic in attic dust tend to be much higher than in other areas of the house. However, separate assumptions to estimate exposure to attic dust were not made in this assessment, since it was assumed that contact with attic dust was minimal. However, if a resident regularly spends time in the attic, or if exposures to dust in some other area of the home more closely approximate attic concentrations, this assessment may underestimate total arsenic exposures.

5.3 Soil + Dust Ingestion Rates

In this assessment, we assumed a soil ingestion rate of 100 mg/d for an adult and 200 mg/d for a child; these values were not changed from the CDM (1996) Baseline HHRA as these remain the U.S. EPA recommended default assumptions for this scenario (U.S. EPA, 1996b; U.S. EPA, 2012d). In the 2011 *Exposure Factors Handbook*, U.S. EPA reevaluates soil and dust ingestion rate data for adults and children and suggests average soil + dust ingestion rates of 50 mg/d for adults and 110 mg/d for children (U.S. EPA, 2011).

If these alternative values for soil + dust ingestion are used, the resulting lifetime excess cancer risk for the Opportunity Community residential scenario (based on the equations and assumptions

ED_001802_00024760-00059

described in Section 3.0) decreases to 1.9×10^{-4}, and the calculated soil screening level at a target cancer risk of 1×10^{-5} increases to 9.7 ppm.

5.4 Produce Consumption Rates

Separate produce consumption rates were applied for three different categories of produce: above ground exposed, above ground protected, and below ground (root) vegetables or fruit. These categories are used because it is assumed that contaminants are taken up from soil into these groups of produce at different rates. Per Baes et al. (1985),

> Exposed produce (snap beans, tomatoes, apples, etc.) intercept atmospherically depositing material on edible surfaces, but surface areas for exposure are relatively small compared to leafy vegetables. Additionally, edible portions are typically concerned with reproductive functions (fruits and seeds). Protected produce (potatoes, peanuts, citrus fruits, etc.) are not directly exposed to atmospherically depositing material because their growth habit is underground, or if aboveground, the edible portions are protected by pods, shells, or nonedible skins or peels. Typically, edible portions are reproductive or storage organs.

For estimating concentrations of arsenic in produce, uptake from soil only was assumed (i.e., deposition of airborne particulate onto the plant surface was not evaluated). If deposition of airborne arsenic onto edible produce was significant, this assessment may underestimate total arsenic exposures.

As discussed in Section 3.2, the assumed homegrown produce consumption rates applied in this risk assessment were equivalent to the following for adults:

- *Consumption rate of aboveground exposed produce* = 1.36 g/ kg BW-d (fresh weight, converted to 0.171 g /kg BW-d dry weight). Assuming a typical adult body weight of 70 kg, this equates to 95 g/d (or 3.4 ounces/d), which is equivalent to consumption of about 2 cups of lettuce.
- *Consumption rate of aboveground protected produce* = 0.55 g/ kg BW-d (fresh weight, converted to 0.122 g /kg BW-d dry weight). Assuming a typical adult body weight of 70 kg, this equates to 38.5 g/d (or 1.4 ounces/d), which is equivalent to consumption of about 1/4 cup of fresh peas.
- *Consumption rate of belowground produce* = 1.03 g/ kg BW-d (fresh weight, converted to 0.229 g /kg BW-d dry weight). Assuming a typical adult body weight of 70 kg, this equates to 72.1 g/d (or 2.5 ounces/d), which is equivalent to consumption of about one carrot.

On an annual average basis, 25% of all vegetables consumed was assumed to be from home gardens. Alternatively, some residents may eat more homegrown produce, and some may eat less or no homegrown produce. However, it is clear based on surveys of Opportunity Community residents that at least some residents do consume homegrown produce.

The consumption rates of aboveground exposed produce and aboveground protected produce are based on U.S. mean values for consumption of vegetables only. Mean consumption rates of exposed fruits and protected fruits are also available. Some residents of Opportunity report eating home-grown berries (strawberries, raspberries), which could be classified as above ground exposed fruits. U.S. EPA reports the following for this category, for adults:

- *Consumption rate of mixed berries, by consumers* = 0.23 g/ kg BW-d (fresh weight, converted to 0.029 g /kg BW-d dry weight). This represents the mean intake of mixed berries reported for consumers in the U.S. population, ages 20-69 (U.S. EPA, 2011b). Assuming a typical adult body weight of 70 kg, this equates to 16 g/d (or 0.57 ounces/d), which is equivalent to consumption of

ED_001802_00024760-00060

about 1/8 cup of raspberries per day. On an annual average basis, 50% of all raspberries consumed was assumed to be from home gardens.

Based on these observations and data, several alternative assumptions for this pathway were made as follows:

- If one-third (33%), rather than 25%, of all consumed produce is assumed to be homegrown, the resulting lifetime excess cancer risk for the Opportunity Community residential scenario increases to 2.8×10^{-4}, and the calculated soil screening level at a target cancer risk of 1×10^{-5} decreases to 6.6 ppm.
- If produce consumption is assumed to include consumption of berries per the above assumptions, the resulting lifetime excess cancer risk for the Opportunity Community residential scenario increases slightly to 2.7×10^{-4}, and the calculated soil screening level at a target cancer risk of 1×10^{-5} decreases to 6.8 ppm.
- If it is assumed that no homegrown produce is consumed, the resulting lifetime excess cancer risk for the Opportunity Community residential scenario decreases to 1.5×10^{-4}, and the calculated soil screening level at a target cancer risk of 1×10^{-5} increases to 12.2 ppm.

5.5 Meat Consumption Rates

The homegrown meat consumption rates applied to estimate the lifetime excess cancer risk for the Opportunity Community residential scenario, discussed in Section 3.2, were as follows for adults:

- *Consumption rate of aboveground exposed meat* = 0.59 g/ kg BW-d (fresh weight). Assuming a typical adult body weight of 70 kg, this equates to 41 g/d (or 1.5 ounces/d), which is equivalent to consumption of about 10 ounces of homegrown meat per week.

Alternatively, some residents may eat more homegrown meat, and some may eat less or no homegrown meat. For this pathway, several alternative assumptions were made as follows:

- If a person is assumed to eat twice as much homegrown meat (about 20 ounces per week for an adult), the resulting lifetime excess cancer risk for the Opportunity Community residential scenario increases to 2.6×10^{-4}, and the calculated soil screening level at a target cancer risk of 1×10^{-5} decreases to 7.1 ppm.
- If a person is assumed to eat no homegrown meat, the resulting lifetime excess cancer risk for the Opportunity Community residential scenario decreases slightly to 2.3×10^{-4} and the calculated soil screening level increases slightly to 8.0 ppm.

If a person is assumed to eat no homegrown produce or homegrown meat, the resulting lifetime excess cancer risk for the Opportunity Community residential scenario decreases to 1.4×10^{-4} and the calculated soil screening level increases to 13.1 ppm.

6.0 SUMMARY AND CONCLUSIONS

The risk assessment conducted herein was performed in a manner consistent with standard U.S. EPA risk assessment guidance. It incorporates numerous updated assumptions including site-specific information about the relative concentration of arsenic in soil and dust and information from community residents that indicates they consume, or have consumed, homegrown produce and in some cases raised livestock for consumption.

ED_001802_00024760-00061

The result of this assessment is an estimated lifetime excess cancer risk associated with exposure to arsenic in soil and dust of 2.5×10^{-4}. By comparison, the estimated cancer risks for arsenic exposure presented in the Baseline HHRA for the Opportunity resident scenario were 5.51×10^{-5} for the RME scenario and 7.01×10^{-6} for the CTE scenario—the risks presented in this assessment are 4.5 to 36-fold higher than those presented in the Baseline HHRA. Further, based on the equations and assumptions presented in the 1996 Baseline HHRA, an action level for arsenic in soil at the Anaconda Smelter NPL site of 250 ppm was established. If this soil concentration is used with the equations and assumptions applied in the assessment presented in this report, the estimated lifetime excess cancer risk for an Opportunity resident would be 3.6×10^{-4}, a 4.3-fold increase over the lifetime excess cancer risk of 8.4×10^{-5} assumed to be associated with the 250 ppm action level in the Baseline HHRA and the ROD. These risks are greater than the Agency's general acceptable risk threshold of 1×10^{-4}. Note this risk estimate is solely arsenic-based. Other chemical exposures, not included here, would increase the risk estimate.

The soil screening level calculated in the current report, based on an acceptable lifetime excess cancer risk level of 1×10^{-5} and the assumptions described herein, is approximately 8 ppm. This recalculated screening level is appropriate and consistent with other arsenic soil action levels established nationwide. The original U.S. EPA ROD action level of 250 ppm is one of the highest action levels for arsenic in the U.S. EPA RODs nationwide.

Where we could not obtain better scientific data, we used the same parameters as the Baseline HHRA conducted in 1996. For example, while I believe for several reasons that the site-specific monkey study underestimates bioavailability, we used values from that study in this assessment. If the bioavailability was in fact higher than indicated by the monkey study, the risk estimated here would be greater than 2.5×10^{-4}; as such, my assessment is conservative and if an alternative estimate of relative bioavailability were used, the estimated risks would be calculated to be higher.

My review of the U.S. EPA Superfund ROD for the Anaconda Company Smelter in Anaconda, MT (U.S. EPA, 1998a), the *Final Baseline Human Health Risk Assessment, Anaconda Smelter NPL Site, Anaconda, MT* (CDM, 1996) has found that their arsenic risk estimate and residential action level of 250 ppm is not appropriate. According to the standard practice of toxicological risk assessment, the documents' calculations contain a number of errors and omissions. I further note that, when using a standard risk assessment approach, the 250 ppm figure presents a greater estimate of cancer risk than the documents indicate. Using current data and practices, I have found a scientifically reliable soil screening level to be approximately 8 ppm. This is assessed on a more probable than not basis.

ED_001802_00024760-00062

7.0 REFERENCES

Barr DB, Wilder LC, Caudill SP, Gonzalez AJ, Needham LL, Pirkle JL. 2005. Urinary creatinine concentrations in the U.S. population: implications for urinary biologic monitoring measurements. *Environ. Health Perspect.* 113 (2):192-200.

Bornschein R. 1992. *Anaconda Childhood Arsenic Exposure Study*. University of Cincinnati. Prepared for Deer Lodge County Board of Health. April.

Bornschein R. 1994. *The Anaconda Study: An Assessment of Residential Arsenic Exposures Among Children Living in the Vicinity of a Former Copper Smelter*. University of Cincinnati. September 27.

Bradham KD, Scheckel KG, Nelson CM, Seales PE, Lee GE, Hughes MF, Miller BW, Yeow A, Gilmore T, Serda SM, Harper S, Thomas DJ. 2011. Relative bioavailability and bioaccessibility and speciation of arsenic in contaminated soils. *Environ Health Perspect.* 119(11):1629-34.

Calabrese EJ, Barnes R, Stanek EJ 3rd, Pastides H, Gilbert CE, Veneman P, Wang XR, Lasztity A, Kostecki PT. 1989. How much soil do young children ingest: an epidemiologic study.*Regul Toxicol Pharmacol.* 10(2):123-37.

CA OEHHA. 2002. *Technical Support Document for Exposure Assessment and Stochastic Analysis*. Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. September.

Casteel SW, Brown LD, Dunsmore ME, Weis CP, Henningsen GM, Hoffman E, et al. 1997. *Relative Bioavailability of Arsenic in Mining Wastes.* December. http://www.epa.gov/region8/r8risk/pdf/r8_asrba1997.pdf

Casteel SW, Evans T, Dunsmore ME Weis CP, Lavelle B, et al. 2001. *Relative Bioavailability of Arsenic in Soils from the VBI70 Site.* January. http://www.epa.gov/region8/r8risk/pdf/rba-as_vbi70.pdf

Casteel SW, Evans T, Lemay J, Brattin WJ, Wahlquist AM. 2002. *Relative Bioavailability of Arsenic in Sediments from the Aberjona River.* December. http://www.epa.gov/region8/r8risk/pdf/rba-as_aberjona.pdf

Casteel SW, Evans T, Morris S, Griffin S, Brattin WJ, Wahlquist AM. 2003a. *Relative Bioavailability of Arsenic in Soils from Butte, Montana.* March. http://www.epa.gov/region8/r8risk/pdf/rba-as_butte.pdf

Casteel SW, Evans T, Griffin S, Brattin WJ, Wahlquist AM. 2003b. *Comparison of Relative Bioavailability of Arsenic in Soil Estimated Using Data Derived With Two Alternative Analytical Methods*. May. http://www.epa.gov/region8/r8risk/pdf/rba-as_buttemethod.pdf

Casteel SW, Fent G, Myoungheon L, Brattin WJ, Wahlquist AM. 2009. *Relative Bioavailability of Arsenic in NIST SRM 2710 (Montana Soil).* Prepared for U.S. Environmental Protection Agency, Office of Superfund Remediation Technology Innovation. Prepared by University of Missouri, Columbia and SRC. http://www.epa.gov/superfund/bioavailability/pdfs/NIST1_As%20RBA%20Report%20Final%203-13-09_508.pdf

Casteel SW, Fent G, Myoungheon L, Brattin WJ, Hunter P. 2010. *Relative Bioavailability of Arsenic in Two Soils from the Iron King Mine*. Prepared for U.S. Environmental Protection Agency, Office of Superfund Remediation Technology Innovation. Prepared by University of Missouri, Columbia and SRC. http://www.epa.gov/superfund/bioavailability/pdfs/Iron%20King%20Swine%20RBA%2002-25-10_SRC_508.pdf

Casteel SW, Fent G, Knight LE, Brattin WJ, and Hunter P. 2012. *Relative Bioavailability of Arsenic in an ASARCO and a Hawaii Soil*. Prepared for U.S. Environmental Protection Agency, Office of Superfund Remediation Technology Innovation. Prepared by University of Missouri, Columbia and SRC. March.

CDM. 1996. *Final Baseline Human Health Risk Assessment, Anaconda Smelter NPL Site, Anaconda, MT*. CDM Federal Programs Corporation, Golden, CO. Prepared for U.S. Environmental Protection Agency, Helena, MT. January 24.

Cobb GP, Sands K, Waters M, Wixson BG, Dorward-King E. 2000. Accumulation of heavy metals by vegetables grown in mine wastes. *Environ Toxicol Chem*. 19: 600–607.

Colorado Department of Health. 1993. *Record of Decision, Asarco Globe Plant Site, Denver, Colorado*. Denver, CO. February 18.

Davis A, Sherwin D, Ditmars R, Hoenke KA. 2001. An analysis of soil arsenic records of decision. *Environ Sci Technol*. 35(12):2401-6.

De Temmerman L, Ruttens A, Waegeneers N. 2012. Impact of atmospheric deposition of As, Cd and Pb on their concentration in carrot and celeriac. *Environ Pollut*. 166:187-95.

Freeman GB, Schoof RA, Ruby MV, Davis AO, Dill JA, Liao SC, Lapin CA, Bergstrom PD. 1995. Bioavailability of arsenic in soil and house dust impacted by smelter activities following oral administration in cynomolgus monkeys. *Fundam Appl Toxicol*. 28(2):215-22.

Juhasz AL, Smith E, Weber J, Rees M, Rofe A, Kuchel T, Sansom L, Naidu R. 2008. Application of an in vivo swine model for the determination of arsenic bioavailability in contaminated vegetables. *Chemosphere*. 71(10):1963-9.

Larios R, Fernández-Martínez R, Lehecho I, Rucandio I. 2012. A methodological approach to evaluate arsenic speciation and bioaccumulation in different plant species from two highly polluted mining areas. *Sci Total Environ*. 414:600-7.

Mayland HF, Shewmaker GE, and Bull RC. 1977. Soil ingestion by cattle grazing crested wheatgrass. *J Range Management*. 30(4): 264-265.

Mushak P. 1998. Uses and limits of empirical data in measuring and modeling human lead exposure. *Environ Health Perspect*. 106 Suppl 6:1467-84.

NRC (National Research Council). 1987. *Predicting Feed Intake of Food-Producing Animals. Subcommittee on Beef Cattle Nutrition*. National Academy Press, Washington, D.C.

NIST (National Institute of Standards and Technology). 2003. *Standard Reference Material® 2710: Montana Soil Highly Elevated Trace Element Concentrations*. Gaithersburg, MD. http://www.clu-in.org/conf/tio/xrf_082808/cd/nist-standard-reference-materials/nist_srm_2710.pdf

Pioneer Technical Services. 2009. *Final: Community Soils Interior and Attic Dust Characterization Study Data Summary Report (DSR)*. Anaconda Smelter NPL Site: Community Soils Operable Unit. Butte, MT. January 9.

ED_001802_00024760-00064

Ramirez-Andreotta MD, Brusseau ML, Artiola JF, Maier RM. 2013. A greenhouse and field-based study to determine the accumulation of arsenic in common homegrown vegetables grown in mining-affected soils. *Sci Total Environ.* 443:299-306.

Rasmussen LB, Ovesen L, and Christiansen E. 1999. Day-to-day and within-day variation in urinary iodine excretion. *Eur J Clin Nutr.* 53 (5):401-7.

Roberts SM, Munson JW, Lowney YW, Ruby MV. 2007. Relative oral bioavailability of arsenic from contaminated soils measured in the cynomolgus monkey. *Toxicol Sci.* 95(1):281-8.

Sedman RM. 1989. The development of applied action levels for soil contact: a scenario for the exposure of humans to soil in a residential setting. *Environ Health Perspect.* 79:291-313.

Teaf CM, Covert DJ, Teaf PA, Page E, Starks MJ. 2010. Arsenic Cleanup Criteria for Soils in the US and Abroad: Comparing Guidelines and Understanding Inconsistencies. Chapter 9 of *Proceedings of the Annual International Conference on Soils, Sediments, Water, and Energy*, Vol. 15.

U.S. EPA. 1989. *Risk Assessment Guidance for Superfund (RAGS), Volume I. Human Health Evaluation Manual, Part A.* Interim Final. Office of Solid Waste and Emergency Response, United States Environmental Protection Agency. Washington, D.C. EPA/540/1-89/002. December.

U.S. EPA. 1990a. *EPA Superfund Record of Decision: Whitewood Creek.* EPA ID: SDD980717136. OU 01. Whitewood, SD. March 30, 1990. http://www.epa.gov/superfund/sites/rods/fulltext/r0890028.pdf

U.S. EPA. 1990b. *EPA Superfund Record of Decision: Sharon Steel Corp. (Midvale Tailings).* EPA ID: UTD980951388. OU 02. Midvale, UT. EPA/ROD/R08-90/044. September 24, 1990. http://www.epa.gov/superfund/sites/rods/fulltext/r0890044.pdf

U.S. EPA. 1990c. *EPA Superfund Record of Decision: Silver Bow Creek/Butte Area.* EPA ID: MTD980502777. OU 04. Butte, MT. EPA/ROD/R08-90/030. September 28, 1990. http://www.epa.gov/superfund/sites/rods/fulltext/r0890030.pdf

U.S. EPA. 1991. *Risk Assessment Guidance for Superfund, Volume I—Human Health Evaluation Manual (Part B, Development of Risk-based Preliminary Remediation Goals).* Office of Emergency and Remedial Response, United States Environmental Protection Agency. Washington, D.C. EPA/540/R-92/003. December. http://www.epa.gov/oerrpage/superfund/programs/risk/ragsa/index.htm

U.S. EPA. 1992a. *Supplemental Guidance to RAGS: Calculating the Concentration Term.* Office of Solid Waste and Emergency Response, United States Environmental Protection Agency. Washington, D.C. May.

U.S. EPA. 1992b. *Dermal Exposure Assessment: Principles and Applications.* Office of Health and Environmental Assessment, United States Environmental Protection Agency. January. http://rais.ornl.gov/documents/DERM_EXP.PDF

U.S. EPA. 1992c. *EPA Superfund Record of Decision: Bunker Hill Mining & Metallurgical Complex.* EPA ID: IDD048340921. OU 2. Smelterville, ID. United States Environmental Protection Agency. September 22.

ED_001802_00024760-00065

U.S. EPA. 1992d. *EPA Superfund Record of Decision: Joseph Forest Products.* EPA ID: ORD068782820. OU 01. Joseph, OR. United States Environmental Protection Agency. EPA/ROD/R10-92/043. September 30. http://www.epa.gov/superfund/sites/rods/fulltext/r1092043.pdf

U.S. EPA. 1992e. *EPA Superfund Record of Decision: Rhone-Poulenc, Inc./Zoecon Corp.* EPA ID: CAT000611350. OU 01. East Palo Alto, CA. EPA/ROD/R09-92/080. March 4, 1992. http://www.epa.gov/superfund/sites/rods/fulltext/r0992080.pdf

U.S. EPA. 1993a. *Superfund Standard Default Exposure Factors for the Central Tendency and Reasonable Maximum Exposure.* Preliminary Review Draft. United States Environmental Protection Agency. May.

U.S. EPA. 1993b. *EPA Superfund Record of Decision: Rentokil, Inc. (Virginia Wood Preserving Division).* EPA ID: VAD071040752. OU 01. Richmond, VA. United States Environmental Protection Agency. EPA/ROD/R03-93/168. June 22. http://www.epa.gov/superfund/sites/rods/fulltext/r0393168.pdf

U.S. EPA. 1994a. *Technical Support Document for the Integrated Exposure Uptake Biokinetic Model for Lead in Children (v0.99d).* United States Environmental Protection Agency. NTIS #PB94-963505, EPA 9285.7-22. December. http://www.epa.gov/superfund/lead/products.htm#techsupport

U.S. EPA. 1994b. *EPA Superfund Record of Decision: Woolfolk Chemical Works, Inc.* EPA ID: GAD003269578. OU 01. Fort Valley, GA. EPA/ROD/R04-94/171. March 25, 1994. http://www.epa.gov/superfund/sites/rods/fulltext/r0494171.pdf

U.S. EPA. 1995a. *EPA Superfund Record of Decision: National Zinc Corp.* EPA ID: OKD000829440. OU 01. Bartlesville, OK. United States Environmental Protection Agency. December 13, 1994. http://www.epa.gov/superfund/sites/rods/fulltext/r0695098.pdf

U.S. EPA. 1995b. *EPA Superfund Record of Decision: Midvale Slag.* EPA ID: UTD081834277. OU 01. Midvale, UT. EPA/ROD/R08-95/106. April 28, 1995. http://www.epa.gov/superfund/sites/rods/fulltext/r0895106.pdf

U.S. EPA. 1995c. *EPA Superfund Record of Decision: Woolfolk Chemical Works, Inc.* EPA ID: GAD003269578. OU 02. Fort Valley, GA. EPA/ROD/R04-95/246. September 29, 1995. http://www.epa.gov/superfund/sites/rods/fulltext/r0494171.pdf

U.S. EPA. 1995d. *EPA Superfund Record of Decision: Atlantic Wood Industries, Inc.* EPA ID: VAD990710410. OU 01. Portsmouth, VA. EPA/ROD/R03-95/199. September 29, 1995. http://www.epa.gov/superfund/sites/rods/fulltext/r0395199.pdf

U.S. EPA. 1996a. *Bioavailability of Arsenic and Lead in Environmental Substrates.* United States Environmental Protection Agency, Seattle, WA. February. http://www.epa.gov/region10/pdf/bioavailability_arsenic_lead_in_substrates_1996.pdf

U.S. EPA. 1996b. *oil creenin Guidance: ser's Guide* . United States Environmental Protection Agency, Washington, DC. July. http://www.epa.gov/superfund/health/conmedia/soil/pdfs/ssg496.pdf

ED_001802_00024760-00066

U.S. EPA. 1996c. EPA Superfund Record of Decision: McCormick & Baxter Creosoting Co. (Portland Plant). EPA ID: ORD009020603. OU 01. Portland, OR. United States Environmental Protection Agency. EPA/ROD/R10-96/140. March 29. http://www.epa.gov/superfund/sites/rods/fulltext/r1096140.pdf

U.S. EPA. 1996d. *EPA Superfund Record of Decision: Koppers Co., Inc. (Oroville Plant).* EPA ID: CAD009112087. OU 01. Oroville, CA. United States Environmental Protection Agency. EPA/AMD/R09-96/151. August 29.

U.S. EPA. 1996e. *EPA Superfund Record of Decision: Wingate Road Municipal Incinerator Dump.* OU 01. EPA ID: FLD981021470. Fort Lauderdale, FL. United States Environmental Protection Agency. EPA/ROD/R04-96/274. May 14. http://www.epa.gov/superfund/sites/rods/fulltext/r0496274.pdf

U.S. EPA. 1996f. *EPA Superfund Record of Decision: RSR Corporation.* EPA ID: TXD079348397. OU 04. Dallas, TX. United States Environmental Protection Agency. EPA/ROD/R06-96/099. February 28. http://www.epa.gov/superfund/sites/rods/fulltext/r0696099.pdf

U.S. EPA. 1996g. *EPA Superfund Record of Decision: Hanford 300-Area (USDOE).* EPA ID: WA2890090077. OU 01, 02. Benton County, WA. EPA/ROD/R10-96/143. July 17, 1996. http://www.epa.gov/superfund/sites/rods/fulltext/r1096143.pdf

U.S. EPA. 1997. *EPA Superfund Record of Decision: Puget Sound Naval Shipyard Complex.* EPA ID: WA2170023418. OU 01. Bremerton, WA. EPA/ROD/R10-97/046. January 24, 1997. http://www.epa.gov/superfund/sites/rods/fulltext/r1097046.pdf

U.S. EPA. 1998a. *EPA Superfund Record of Decision: Anaconda Co. Smelter.* EPA ID: MTD093291656. OU 04. Anaconda, MT. United States Environmental Protection Agency, Helena, MT. EPA/ROD/R08-98/096. September 29.

U.S. EPA. 1998b. *EPA Superfund Record of Decision: Woolfolk Chemical Works, Inc.* EPA ID: GAD003269578. OU 03. Fort Valley, GA. EPA/ROD/R04-98/111. August 6, 1998. http://www.epa.gov/superfund/sites/rods/fulltext/r0498111.pdf

U.S. EPA. 1998c. *EPA Superfund Record of Decision: California Gulch.* EPA ID: COD980717938. OU 03. Leadville, CO. United States Environmental Protection Agency. EPA/ROD/R08-98/076. May 6, 1998. http://www.epa.gov/superfund/sites/rods/fulltext/r0898076.pdf

U.S. EPA. 1998d. *EPA Superfund Record of Decision: Triumph Mine Tailings Piles.* EPA ID: IDD984666024. OU 01. Cheyenne, WY. EPA/ROD/R10-98/506. March 19, 1998.

U.S. EPA. 1999a. *EPA Superfund Record of Decision Amendment: Cleveland Mill Site, Silver City, New Mexico.* United States Environmental Protection Agency. EPA 541-R99-057. September 20.

U.S. EPA. 1999b. *EPA Superfund Record of Decision: Kennecott (South Zone).* EPA ID: UTD000826404. OU 01. Copperton, UT. United States Environmental Protection Agency. EPA/ROD/R08-99/034. November 3, 1998. http://www.epa.gov/superfund/sites/rods/fulltext/r0899034.pdf

U.S. EPA. 1999c. *EPA Superfund Record of Decision: Jacobs Smelter. EPA ID: UT0002391472. OU 01. Stockton, UT.* United States Environmental Protection Agency. EPA/ROD/R08-99/047. July 29, 1999. http://www.epa.gov/superfund/sites/rods/fulltext/r0899047.pdf

ED_001802_00024760-00067

U.S. EPA. 2000a. *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health.*Office of Water, United States Environmental Protection Agency. Washington, D.C. EPA-822-B-00-004. October.

U.S. EPA. 2000b. EPA Superfund Record of Decision: California Gulch. EPA ID: COD980717938. OU 07. Leadville, CO. United States Environmental Protection Agency. EPA/ROD/R08-00/512. June 6, 2000. http://www.epa.gov/superfund/sites/rods/fulltext/r0800512.pdf

U.S. EPA. 2001a. *Superfund Fact Sheet: Talache Mine Tailings Site, Atlanta, Idaho.* United States Environmental Protection Agency. May. http://yosemite.epa.gov/R10/CLEANUP.NSF/9f3c21896330b4898825687b007a0f33/8e3cf18004b0a8bb882568640001b3a8/$FILE/0501talache.pdf

U.S. EPA. 2001b. *EPA Superfund Record of Decision: Fremont National Forest/White King and Lucky Lass Uranium Mines (USDA).* EPA ID: OR7122307658. OU 02. Lakeview County, OR. EPA/ROD/R10-01/536. September 28, 2001. http://www.epa.gov/superfund/sites/rods/fulltext/r1001536.pdf

U.S. EPA. 2002a. *EPA Superfund Record of Decision: Midvale Slag.* EPA ID: UTD081834277. OU 02. Midvale, UT. EPA/ROD/R08-03/002. October 29, 2002. http://www.epa.gov/superfund/sites/rods/fulltext/r0803002.pdf

U.S. EPA. 2002b. *EPA Superfund Record of Decision: Upper Tenmile Creek Mining Area.* EPA ID: MTSFN7578012. OU 04. Helena, MT. EPA/ROD/R08-02/068. June 28, 2002. http://www.epa.gov/superfund/sites/rods/fulltext/r0802068.pdf

U.S. EPA. 2002c. *EPA Superfund Record of Decision: Davenport and Flagstaff Smelters.* EPA ID: UTD988075719. OU 01. Sandy, UT. EPA/ROD/R08-02/055. September 20, 2002. http://www.epa.gov/superfund/sites/rods/fulltext/r0802055.pdf

U.S. EPA, 2002d. *Record of Decision Kennecott North Zone Site, Kennecott South Zone Site.* United States Environmental Detection Agency, Denver, CO. September 26. http://www.epa.gov/region8/superfund/ut/kennecottnorth/KennNorthROD.pdf

U.S. EPA. 2003a. *EPA Superfund Record of Decision: Vasquez Boulevard and I-70.* EPA ID: CO0002259588. OU 01. Denver, CO. EPA/ROD/R08-03/014. September 25, 2003. http://www.epa.gov/superfund/sites/rods/fulltext/r0803014.pdf

U.S. EPA. 2003b. *Blackbird Mine Record of Decision. Lemhi County, ID.* EPA Region 10. February. http://yosemite.epa.gov/r10/CLEANUP.NSF/sites/Blackbird/$FILE/Blackbird+ROD+-+Feb+2003.pdf

U.S. EPA. 2004a. *Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment).* United States Environmental Protection Agency. EPA/540/R/99/005. http://www.epa.gov/oswer/riskassessment/ragse/pdf/part_e_final_revision_10-03-07.pdf

U.S. EPA. 2004b. *EPA Superfund Record of Decision: Lava Cap Mine.* EPA ID: CAD983618893. OU 01. Nevada City, CA. United States Environmental Protection Agency. September 28.

U.S. EPA. 2004c. *Clark Fork River Operable Unit of the Milltown Reservoir/Clark Fork River Superfund Site Record Of Decision.* U.S. Environmental Protection Agency, Region 8, Helena, MT. April. http://www.epa.gov/region8/superfund/mt/milltowncfr/cfr/Pt2DecisionSummary.pdf

ED_001802_00024760-00068

U.S. EPA. 2004d. EPA Superfund Record of Decision: Rockwool Industries Inc. EPA ID: TXD066379645. OU 01. Bell County, TX. EPA/ROD/R06-04/672. September 20, 3004.

U.S. EPA. 2005. *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities.* United States Environmental Protection Agency, Office of Solid Waste and Emergency Response. EPA530-R-05-006.

U.S. EPA. 2007a. *Guidance for Evaluating the Oral Bioavailability of Metals in Soils for Use in Human Health Risk Assessment.* United States Environmental Protection Agency. OSWER 9285.7-80. May. http://www.epa.gov/superfund/bioavailability/bio_guidance.pdf

U.S. EPA. 2007b. *Record of Decision, Operable Units 1, 2, and 3. Atlantic Wood Industries, Inc. Superfund Site. Portsmouth, VA.* United States Environmental Protection Agency, Philadelphia, PA. December.

U.S. EPA. 2008. *Child-Specific Exposure Factors Handbook.* United States Environmental Protection Agency, Washington, DC.

U.S. EPA. 2010. *Relative Bioavailability of Arsenic in Soils at 11 Hazardous Waste Sites Using an In Vivo Juvenile Swine Method.* United States Environmental Protection Agency, Washington, DC. OSWER Directive #9300.0-76. June. http://www.epa.gov/superfund/bioavailability/pdfs/as_in_vivo_rba_main.pdf

U.S. EPA. 2011a. *Technology Transfer Network Air Toxics 2005 National-Scale Air Toxics Assessment: Glossary of Key Terms.* United States Environmental Protection Agency. February 22. http://www.epa.gov/nata/gloss1.html

U.S. EPA. 2011b. *Exposure Factors Handbook.* United States Environmental Protection Agency.

U.S. EPA. 2012a. *Compilation and Review of Data on Relative Bioavailability of Arsenic in Soil.* United States Environmental Protection Agency. OSWER 9200.1-113. http://www.epa.gov/superfund/bioavailability/pdfs/Arsenic%20Bioavailability%20SCIENCE%20Report_SRC%2009-20-12.pdf

U.S. EPA. 2012b. *Recommendations for Default Value for Relative Bioavailability of Arsenic in Soil.* United States Environmental Protection Agency. http://www.epa.gov/superfund/bioavailability/pdfs/Arsenic%20Bioavailability%20POLICY%20Memorandum%2012-20-12.pdf

U.S. EPA. 2012c. *Arsenic, inorganic (CASRN 7440-38-2).* Integrated Risk Information System. United States Environmental Protection Agency. http://www.epa.gov/iris/subst/0278.htm

U.S. EPA. 2012d. *ser's Guide to the is ased oncentration Ta le* . United States Environmental Protection Agency, Mid-Atlantic Region. November. http://www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/usersguide.htm

U.S. EPA. 2012e. *Risk Assessment: Glossary.* United States Environmental Protection Agency. July. http://www.epa.gov/risk_assessment/glossary.htm

U.S. EPA. 2013. *Record of Decision System (RODS): Bunker Hill Mining & Metallurgical Complex.* United States Environmental Protection Agency. March. http://cfpub.epa.gov/superrods/index.cfm?fuseaction=data.rodinfo&id=1000195&mRod=10001951992ROD041

ED_001802_00024760-00069

WA DOE. 2011. *Frequently Asked Questions: Tacoma Smelter Plume. Cleanup Levels & Action Levels for Soil Arsenic and Lead.* Washington Department of Ecology. October.

Yang JK, Barnett MO, Zhuang J, Fendorf SE, Jardine PM. 2005. Adsorption, oxidation, and bioaccessibility of As(III) in soils. *Environ Sci Technol.* 39(18):7102-10.

ED_001802_00024760-00070

ATTACHMENT A

CURRICULUM VITAE OF RICHARD C. PLEUS

ED_001802_00024760-00071

Richard C. Pleus, Ph.D.

FIELDS OF EXPERTISE

Toxicology and Pharmacology: Neurological / Endocrinological; Reproductive / Developmental; Respiratory, General; Risk Assessment

EDUCATION

Postdoctoral training, University of Nebraska Medical Center, 1992, Neuropharmacology.

Ph.D., University of Minnesota, 1991, Environmental Toxicology. Dissertation title: *Neurobehavioral assessment in offspring of the influence of maternal hypoxia and hypercapnia induced by injection of methadone in pregnant rats.*

M.S., University of Minnesota, 1983, Environmental Health.

B.S., Michigan State University, 1977, Physiology, Honors Graduate.

CURRENT AND PREVIOUS POSITIONS

Founder and Managing Director, Intertox, Inc., Seattle, WA (1995 - present).

Co-Founder, Intertox Decision Sciences, Inc., Seattle, WA (2009 – present).

Adjunct Associate Professor, University of Nebraska, Center for Environmental Toxicology, Omaha, NE (2002 - present).

Adjunct Associate Professor, University of Nebraska Medical Center, Department of Pharmacology, Omaha, NE (1999 - present).

President, Environmental Toxicology International, Inc., Seattle, WA (1993-1995).

Vice President, Marketing & Communications, Environmental Toxicology International, Inc., Seattle, WA (1993).

Senior Toxicologist, Environmental Toxicology International, Inc., Seattle, WA (1992-1993).

Community Faculty, Minnesota State University, St. Paul, MN (1989-1996; taught courses in physiological psychology and psychopharmacology).

Research Associate, Department of Pharmacology, College of Medicine, University of Nebraska Medical Center, Omaha, NE (1989-1992).

Instructor, General College, University of Minnesota, Minneapolis, MN (1985-1989).

Research Assistant, Department of Pharmacology, Medical School, University of Minnesota, Minneapolis, MN (1985-1989).

Instructor, Lowthian College, Minneapolis, MN (1983-1985).

Instructor, Department of Continuing Education and Extension, University of Minnesota, Minneapolis, MN (1979-1983; taught courses on toxicology of cosmetic products and physiological factors contributing to accident susceptibility).

ED_001802_00024760-00072

SELECT PROJECT EXPERIENCE

AIR

- Assessed human health risk of workers in a facility that was being built to decommission chemical warfare agents. Evaluated human acute exposures to sarin and mustard gases. Route of exposure was inhalation. Compared and contrasted reported health effects from acute exposures to health effects reported to those published in the toxicological literature.13 Non-cancer endpoints, with a focus on the nervous system, were assessed.
- Conducted a toxicological assessment for human exposure to chloroform and hydrogen sulfide. These chemicals were released as gases from the wastewater treatment system of a pulp and paper mill. Exposures to residents were via inhalation. Cancer and non-cancer effects were assessed with non-cancer effects focused on the respiratory and neurological systems.
- Conducted a peer review of a multi-pathway human and ecological chemical risk assessment prepared for a Portland cement facility. Chemicals of concern included metals (including As, Cu, Cr, Cd, Zn, Pb, Hg, Ni, Zn), dioxins, and furans, and polyaromatic hydrocarbons (PAHs). The facility used supplemental fuels to make clinker. The supplemental fuels are identified by US EPA as "hazardous waste". Followed US EPA guidelines for the assessment. Corrected errors in the methodology and assisted in correcting a report to both state and federal officials. Cancer and non-cancer endpoints assessed.
- Conducted a toxicological assessment of emissions for a Portland cement plant that supplements its fuel with hazardous waste. The chemicals evaluated included metals (As, Cr, Cd, Pb, Hg, Ni), dioxins, furans, polyaromatic cyclic hydrocarbons, nitrogen oxides, sulfur oxides, and many other US EPA-identified hazardous air pollutants. Focused the research on which of these chemicals would cause neuroanatomical effects on the developing fetuses of local pregnant women. Exposure was primarily via inhalation. Assessment included a review of laboratory data, review of the toxicological literature, the medical literature, and medical records. Research also focused on genetic determinants of nervous system affects.
- Conducted toxicological assessment related to the human and ecological risks of exposure to hydrogen chloride released as stack emissions from a Portland cement plant. Used US EPA guidelines as a basis for the evaluation. Exposures to populations were via inhalation (humans) and deposition (ecological receptors). The facility used hazardous waste to supplement fossil fuel use in the production of cement. Conducted review of laboratory data, assessed the toxicological literature, and used air dispersion modeling to estimate ambient air concentrations to nearby receptors. Cancer and non-cancer endpoints were evaluated for human exposures.
- Assessed human health risk of workers in a facility that was being built to decommission chemical warfare agents. Evaluated human acute exposures to sarin and mustard gases. Route of exposure was inhalation. Compared and contrasted reported health effects from acute exposures to health effects reported to those published in the toxicological literature.
- Conducted research, reviewed laboratory data, and prepared written responses to US EPA Region VII for six cement plants required to submit human health risk assessments for the operation of cement kilns. Followed US EPA guidelines for the assessment. Each facility used supplemental fuels to make clinker. The supplemental fuels are identified by US EPA as "hazardous waste." Conducted toxicological assessment for metals (including As, Cr, Cu, Cd, Zn, Pb, Hg, Ni), dioxins, and furans, and polyaromatic hydrocarbons, nitrogen oxides, sulfur oxides, and other US EPA-identified hazardous air pollutants. Human health risk assessment was conducted for all chemicals for the routes of exposure that included direct exposure from inhalation and

ED_001802_00024760-00073

multipathway exposures from oral and dermal exposures. Cancer, non-cancer, and ecological endpoints were assessed.

- Conducted human health risk assessment for exposures to metals (including As, Cu, Cr, Cd, Pb, Hg, Ni, Zn), dioxins, and furans from exposures to cement kiln dust. Followed US EPA guidelines for the assessment. Exposure routes were inhalation, oral, and dermal. Cancer and non-cancer endpoints assessed.
- Assessed human health risk due to emissions from a coal-burning cement plant. Triggering events were related to foul odors. Developed ambient air-monitoring plan to determine the chemicals of interest. Followed US EPA and the State of California guidelines for the assessment. Carbonyl sulfide was identified as the chemical likely to have caused the foul odors. Exposures were from inhalation. Toxicological assessment included respiratory and nervous organ systems. Non-cancer endpoints assessed. Risk communication was used to disseminate information to local citizens.
- Conducted a multi-pathway human health risk assessment related to a portland cement facility's stack emissions. Followed US EPA guidelines for the assessment. The supplemental fuels are identified by US EPA as "hazardous waste." Chemicals of concern included metals (including As, Cr, Cu, Cd, Zn, Pb, Hg, Ni), dioxins, furans, and polyaromatic hydrocarbons (PAHs). Cancer and-non-cancer endpoints assessed. Documents submitted to US EPA and relevant state agencies.
- Conducted a toxicological assessment to residents living nearby a lead smelting and refining operation. Chemicals of concern were lead and arsenic. Routes of exposure where multipathway but eventually focused on inhalation and ingestion as major routes. Evaluation consisted of assessing laboratory data, review of the toxicological literature, and included information derived from government agencies. US EPA guidelines were used as a basis for the evaluation. All body systems were evaluated.
- Assessed adverse health consequences to an off-site neighborhood resident from an accidental release of chlorine gas from a manufacturing plant. Exposure was via inhalation. Toxicological assessment was focused on the respiratory system. Cancer and non-cancer endpoints assessed.
- Evaluated human health risks from exposure to stack emissions from a proposed fluidized bed incinerator. Air dispersion monitoring was used to estimate air concentrations at critical receptors at nearby neighborhoods. Followed US EPA guidelines for the assessment. Toxicological assessment was conducted for metals (As, Cr, Cd, Pb, Hg, Ni), dioxins, furans, polyaromatic cyclic hydrocarbons, nitrogen oxides, sulfur oxides, and other US EPA identified hazardous air pollutants. Reviewed potential effects for all body systems for cancer and non-cancer endpoints. Presented results to citizens.
- Assessed human health risk from gases released from a landfill. Triggering events for residents were from foul odors. Laboratory data was obtained and carbon disulfide, formaldehyde, and hydrogen sulfide were identified as the chemicals being released. Route of exposure was inhalation. Cancer and non-cancer endpoints were assessed, with a focus on nervous system for non-cancer effects.
- Conducted a review of a state's proposal for biological monitoring of residents and their pets residing in a town that built and operates a hazardous waste incinerator. Followed US EPA guidelines for the assessment. Assessed the reliability and accuracy of biomonitoring parameters relative to chemicals of potential concern. Exposure route was inhalation. Non-cancer endpoints assessed.

ED_001802_00024760-00074

- Evaluated a risk assessment for carcinogenic polycyclic aromatic hydrocarbons for a coal burning power plant. Focus was eventually narrowed to assessing a system of toxic equivalency factors based on non-validated assumptions. US EPA guidelines were used as a basis of the assessment. Exposures were considered multipathway. Cancer and non-cancer endpoints considered.
- Conducted toxicological assessment of multiple emission sources within an industrial park in the Bahamas to nearby residents. Oil terminals, pharmaceutical plants, power plants, and chemical plants operated in the park. Conducted extensive emissions inventories and source evaluation surveys to gather data to assess individual contributions and cumulative effects of emissions. Toxicological assessment for all body systems; and included cancer and non-cancer endpoints.
- Conducted multiple human health risk assessments for a number of different facilities in a US state. Facilities included, a newly designed bus manufacturing facility and several beet processing plants. These were the first air screening risk assessments conducted by this state and we were useful in assisting in the process. Conducted toxicological review of nearly a 100 chemicals, including metals (As, Cr, Cu, Cd, Pb, Hg, Ni, Zn) emitted from each facility. Assessed oral, dermal and inhalation routes of exposures through multipathway analysis. Cancer and non-cancer endpoints were evaluated.
- Conducted a toxicological assessment to residents living nearby a lead smelting and refining operation. Chemicals of concern were lead and arsenic. Routes of exposure where multipathway but eventually focused on inhalation and ingestion as major routes. Evaluation consisted of assessing laboratory data, review of the toxicological literature, and included information derived from government agencies. US EPA guidelines were used as a basis for the evaluation. All body systems were evaluated.
- Reviewed multipathway human health risk assessment for a medical waste facility for the Chris Hani Baragwanath Hospital, the largest hospital in the world. The hospital is in Soweto, South Africa. Chemicals evaluated included metals, (As, Cr, Cu, Cd, Pb, Hg, Ni, Zn), dioxin, furans and polycyclic aromatic hydrocarbons. Exposure was evaluated for residents living nearby. Cancer and non-cancer endpoints assessed.
- Assessed human health risks related to emissions from a composting facility to nearby community. Followed US EPA guidelines for the assessment. Triggering events were foul odors releases. Exposure was via inhalation. Developed a monitoring program to measure 23 reduced sulfurs, volatile organic compounds, and ammonia. Toxicological assessment consisted of all organ systems, with eventual focus on nervous system. Non-cancer endpoints assessed.
- Conducted human health risk assessment from emissions of a thermoplastic extrusion plant. Assessed laboratory data, air dispersion modeling, and calculated estimates of hazards for acute exposures to residents living near the facility. Used US EPA guidelines as a basis for the evaluation. Chemicals evaluated included acrolein, 1,3-butadiene, 4-vinylcyclohexane, styrene, and triphenylphosphate. Exposure was via inhalation. Cancer and non-cancer endpoints for all body systems were considered.
- Conducted human health risk assessments for emissions from several coal-fired electric generating stations in Texas, Illinois, Massachusetts, Michigan, and Washington. Followed US EPA and pertinent state guidelines for the assessments. The evaluations included metals (As, Cr, Cd, Pb, Hg, Ni), dioxins, and furans, and polyaromatic hydrocarbons, which became newly reportable under the US EPA's Toxic Release Inventory (TRI) program. Toxicological assessment was conducted for other additional metals. Exposure was via inhalation. For some

ED_001802_00024760-00075

facilities, potential risks through oral, inhalation, and dermal pathways were assessed. Evaluated all organ systems for both cancer, non-cancer, and ecological endpoints.

- Evaluated human health risks to residents living near an accidental chemical release and subsequent fire from rail cars filled with chlorine and methyl mercaptan. Route of exposure was via inhalation of parent chemical agents and work included assessing the human health risk from byproducts of pyrolysis. Non-cancer endpoints assessed.
- Conducted toxicological research and reviewed laboratory data to determine the technical feasibility of burning medical waste as an alternative fuel source in cement kilns. Followed US EPA guidelines for the assessment. Toxicological assessment was conducted for Chemicals of concern included metals (including As, Cr, Cu, Cd, Zn, Pb, Hg, Ni), dioxins, and furans, and polyaromatic hydrocarbons, nitrogen oxides, sulfur oxides, and other US EPA identified hazardous air pollutants. Human health risk assessment was conducted for all chemicals for the routes of exposure that included direct exposure from inhalation and multipathway exposures from oral and dermal exposures. Cancer and non-cancer endpoints assessed.
- Conducted toxicological evaluation of sewer gases and their impact to community health. Evaluated over 60 chemicals produced from degradation of plant, animal, and human wastes. All organ systems were evaluated with focus eventual focus on risk from adverse effects to the nervous system. Exposure was via inhalation.

WORKPLACE

- Conducted an occupational exposure assessment for workers at a cement facility in Belgium that supplements fuel with "hazardous waste." Assessed nearly 100 chemicals including polycyclic aromatic hydrocarbons, metals (As, Cr, Cd, Pb, Hg, Ni), and chlorinated hydrocarbons. Worker exposure was inhalation; and dermal. Cancer and non-cancer endpoints were evaluated.
- Assessed health effects of a worker allegedly exposed to fumes from a construction work site. The chemicals of concern included asphalt roofing fumes, solvent-based and latex paints, dusts, and various other construction-related fumes. Exposures were via inhalation and dermal routes. Non-cancer endpoints assessed.
- Assessed risk to human health of workers exposed to chemicals from the re-entrainment of exhaust air in a pharmaceutical research laboratory. Exposure was via inhalation. Odors were detected and were the triggering event at the workplace. Chemicals were identified and estimated concentrations calculated. Toxicological assessment included all body systems, with eventual focus on reproductive and nervous systems. Non-cancer endpoints assessed.
- Conducted a toxicological assessment related to occupational chemical exposures of tricresyl phosphate (TCP) on human health through the operation of a commercial airplane's Auxiliary Power Unit (APU). Assessment included analyzing laboratory data, medical records, and medical literature analyses.
- Conducted toxicological evaluation of a worker to exposure to beryllium (Be) and polonium (Po) dust and residue. Evaluated exposures via inhalation, dermal, and to a lesser degree oral from dust. Cancer and non-cancer endpoints assessed.
- Conducted toxicological assessment for workers exposed to trichloroethylene (TCE). Conducted evaluation of the literature and laboratory data from the facility. TCE was used extensively in the facility as a degreaser and all routes of exposure were assessed. However, assessment eventually focused on risks for the development of neurobehavioral effects to offspring of women exposed to TCE from ingestion of drinking water as that was deemed the most sensitive endpoint. Evaluated the use of medical monitoring for this population.

ED_001802_00024760-00076

- Conducted a toxicological assessment to human health from exposure to perchloroethylene (PCE, or tetrachloroethylene) from contaminated ground water. Evaluated ingestion and dermal contact with water and inhalation of volatiles during showering or bathing. Toxicological assessment included general toxicology and focused on the nervous system.
- Conducted toxicological assessments related to the waste from production of plutonium (Pu) at the Hanford Reservation in eastern Washington, for the US DOE site contractor. We conducted toxicological assessments for chemicals present in underground storage tanks, including metals and organic compounds. This included developing Temporary Emergency Exposure Limits (TEELs) and other toxicity guidelines for worker exposure that were peer reviewed by Argonne National Laboratories, evaluating exposures to workers related to potential accident scenarios, and developing computer visualization tools to assist workers with understanding the significance of detected chemical concentrations.
- Conducted an exposure assessment of airborne nanoparticles to workers. Nano-sized particles are thought be released from sanding or grinding of composite materials containing carbon nanotubules. Exposure routes include inhalation, ingestion, and dermal exposures.

PESTICIDES

- Conducted a toxicological evaluation of pesticides and their combustion by-products to residents living nearby a pesticide warehouse. Chemicals evaluated pesticides (e.g., organophosphates) and herbicides (e.g., glyphosate). Toxicological assessment considered all organ systems, however, assessment eventually focused on the nervous system.
- Conducted a toxicological assessment for Malathion (pesticide) exposure on human health. Assessment included analyzing laboratory data, medical records, and medical literature analyses. Non-cancer endpoints evaluated, with eventual focus on the nervous system.
- Conducted a toxicological assessment of human exposure to a chemical intermediate used in the production of carbamate pesticides. Worker exposures were assessed for inhalation of intermediate compounds. Conducted toxicological assessment of the literature and prepared a toxicology profile for the chemical. The work was submitted to the US EPA. A surrogate reference dose was developed and presented for information purposes. Toxicological assessment of all body systems was conducted for cancer and non-cancer endpoints.
- Conducted a toxicological assessment related to a consumer from the use of indoor pesticides. Pesticides contained boron, which was the chemical evaluated. Exposure was predominately via inhalation. All organ systems were considered for non-cancer endpoints.

WATER

- Assisted in a toxicological assessment of human health risk from ingestion drinking water containing trace amounts of personal care products, endocrine disrupting chemicals (EDCs) and pharmaceuticals in drinking water. Both cancer and non-cancer endpoints were assessed. Non-cancer endpoints included nervous system, endocrine system, reproductive system, and immune system. Ecological endpoints were examined.
- Evaluated potential human health and ecological consequences of exposure to EDCs and pharmaceuticals and personal care products (PPCPs) in reuse water for a large reuse water management agency. Identified contaminants of greatest concern based on likelihood of occurrence and resistance to treatment processes used at various facilities as well as potential for environmental exposure and health effects. Communicated the potential risks to the public and to regulators.

ED_001802_00024760-00077

- Provided scientific assessment of the human health risk for exposure to perchlorate. Followed US EPA guidelines for the human health assessment. Addressed multipathway exposures to perchlorate that also included exposures to sensitive populations, such as pregnant women, and ecological endpoints. Provided scientific comments to US EPA's development of a reference dose, as well as to the State of California regarding the development of a Public Health Goal and proposed Proposition 65 listing. Cancer and non-cancer endpoints assessed.
- Conducted a review of a state's proposal for biological monitoring of residents and their fluoride. This assessment also considered human exposures from metals, found as a contaminants of sodium hexafluorosilicate or hexafluorosilicic acid. Evaluated ingestion of fluoride in sensitive human populations, including the elderly and children. Both cancer and non-cancer endpoints evaluated.
- Conducted toxicological assessment for health risk from drinking water. Chemicals evaluated included perchlorate, TCE, and N-nitrosodidimethylamine (NDMA). Assessment consisted of evaluating laboratory data, modeling data, toxicological literature, medical literature, and medical records. The major route of exposure was ingestion, however, dermal and inhalation routes were also reviewed.
- Conducted toxicological evaluation of human health related to fluoride. Water fluoridation is the practice of adding fluoride compounds to water with the intended purpose of reducing tooth decay in the general population. Conducted a review of the toxicological and medical literature in addition to US and European governmental assessments of fluoridation.
- Conducted toxicological assessment of the risk of oral exposure to lead to children via drinking water from public schools. Used physiologically based pharmacokinetic modeling to develop blood lead estimates for children of different ages. Non-cancer endpoints were focused on neurodevelopmental affects.
- Conducted a toxicological assessment related to oral exposures of ground water contaminated with low levels of perchlorate to residents of a community. Toxicological assessment considered possible doses, exposures via dermal, ingestion, and inhalation (e.g., taking a shower). Evaluated sensitive populations that included the pregnant woman and children.
- Conducted an evaluation of non-standard neuropsychological tests as a means to demonstrate adverse effects to chemical exposure. Chemicals evaluated were perchlorate and a number of petroleum chlorinated solvents (e.g., TCE, PCE) in ground water. Routes of exposure were via oral and inhalation. Non-cancer endpoints evaluated with focus on the nervous system.

PHARMACEUTICALS

- Conducted a toxicological assessment for opiate and opioid narcotic analgesic agents and their possible cause of death in two separate cases. Assessment included analyzing laboratory data, medical records, and medical literature analyses. Route of exposure was oral.
- Provided a toxicological assessment for over 10 projects related to the testing and evaluation of biologic tissue (e.g., urine, hair, serum) samples for concerns of drug exposure, including ethanol, methamphetamine, benzoylecgonine, phencyclidine, nortriptyline, and amphetamine. Tasks included evaluations of test results for indications of adulterants or dilution, assessing methodological techniques, and determining the toxicological impacts and the signs and symptoms that might be associated with the levels of drugs detected. Routes of exposure where inhalation and ingestion. Research also focused on genetic determinants of nervous system affects. Non-cancer endpoints were evaluated.

ED_001802_00024760-00078

PRODUCT SAFETY

- Conducted a scientific assessment for a large multinational company. A toy distributed to the US, Europe and Asia was observed to have fungal growth. Laboratory tests were conducted, biological assessment was conducted, and an approach to possible disposal was developed to address possible disposal options. Assisted in developing forensic analysis to determine the cause of the mold. Evaluated acute and subchronic exposures.
- Conducted a toxicological assessment of human exposure to chemicals found in cell-phone and its packaging. Employees were exposed to unknown chemicals and subsequently reported acute health effects. Developed a testing program to determine chemicals of potential concern. Identified a number of solvents and assessed toxicological effects from exposure via inhalation and dermal contact. Developed a forensic program to evaluate source. Acute non-cancer endpoints evaluated.
- Conducted a toxicological assessment to human health from several volatile organic compounds, silane, and siloxane released from weather treating products. Route of exposure was inhalation and population of concern included children. Non-cancer endpoints were assessed and focused on the nervous and respiratory systems.
- Conducted a toxicological assessment of multiple chemical cleaning solutions and carbon monoxide on reproductive effects. Exposure to pregnant woman occurred while visiting a commercial art store. Exposure was via inhalation. Toxicological assessment focused on reproductive and neurological systems. Research also focused on genetic determinants of nervous system affects.
- Conducted human and ecological risk assessment of ethylene vinyl alcohol, a chemical used to make shipping packaging "peanuts." Reviewed laboratory data, the toxicological literature, and the use and fate of the material as a consumer product. Endpoints of evaluation were human and ecological receptors. US EPA guidelines were used as a basis of the evaluation. For human exposure, cancer and non-cancer endpoints were assessed.
- Conducted a toxicological assessment of a consumer product, an ink-pen barrel which was constructed of pressed recycled rubber-tire. Reviewed laboratory data and conducted a review of the toxicological literature. Used US EPA risk assessment guidelines as a basis for assessment. Chemicals evaluated included metals (As, Cr, Cd, Pb, Hg, Ni) and organic hydrocarbons. Cancer and non-cancer endpoints for all body systems were considered.
- Prepared a human health risk assessment of occupational exposures to cellulose insulation. Conducted a review of the toxicological literature of paper dust, wood dust, and chemicals found in newsprint. Assessed the exposure from inhalation to paper and wood dusts. Evaluated cancer and non-cancer endpoints.
- Conducted a toxicological assessment of human exposure to the combustion of jet oil and hydraulic fluid in commercial aircraft. Chemicals evaluated were a group of organophosphates that included tricresyl phosphate and tributyl phosphate. The triggering events were foul odors. Evaluation focused on neurological system.
- Evaluated potential human health and ecological consequences of exposure to EDCs and pharmaceuticals and personal care products (PPCPs) in reuse water for a large reuse water management agency. Identified contaminants of greatest concern based on likelihood of occurrence and resistance to treatment processes used at various facilities as well as potential for environmental exposure and health effects. Communicated the potential risks to the public and to regulators.

ED_001802_00024760-00079

- Conducted toxicological, human, and ecological risk assessment of a variety of herbicides used by WA State's Department of Transportation for use on state roadways. Assessed human and ecological health risk associated with roadside vegetation management practice. Multipathway exposures were conducted and included the evaluation of sensitive human and ecological populations. Cancer and non-cancer endpoints were assessed.
- Conducted toxicological assessment of a consumer-related use of a product for cleaning outdoor camping equipment. The chemicals evaluated were ethylenediaminetetraacetic acid (EDTA), sodium hydroxide, nonylphenol polyethylene glycol ether, and dipropylene glycol monomethyl ether. The concern was the adverse impact of these chemicals on the non-cancer endpoints of the nervous system, particularly the eye.
- Conducted a toxicological assessment of fog-oil released from a military training facility. Fog-oil is used as a chemical obscurant in training exercises. Fog-oil migrated off-base into residential neighborhoods. Benzene and a number of other volatile organic compounds were evaluated toxicologically via inhalation exposure. Evaluation focused on cancer and non-cancer hematological effects.
- Conducted toxicological assessment on human health risks from of cellulose insulation containing ammonium sulfate-based flame retardant. Exposure was via inhalation. Developed a single-compartment, first-order model to describe the environmental fate and transport of ammonia in a residential setting. Used US EPA guidelines for the evaluation. Cancer and non-cancer endpoints were considered. Also, the production of foul odor was evaluated.
- Conducted toxicological evaluation of boron (B)-containing pesticide. All organ systems were evaluated. Exposures were ingestion, dermal, and via inhalation. Conducted exposure assessment with major university laboratory as a part of the assessment. Eventually focused on the reproductive effects of boron pesticide. All information was forwarded to the State of California for review and assessment of the data. Cancer and non-cancer endpoints were considered.
- Conducted toxicological assessment for consumer-used ink products for Japanese manufacturer. Assessed human health risk using American Society for Testing and Materials (ASTM) standards. Over 50 chemicals were evaluated for various oral, dermal, and inhalation routes of exposure. Toxicological assessment for all body systems, and included cancer and non-cancer endpoints.
- Reviewed and assessed the toxicity of multiple perfluorinated chemicals, including perfluorooctnoate (PFOA) and perfluooctosulfonate (PFOS). Assessed the current state of toxicological knowledge, evaluated guideline levels of state and federal government. Issues of concern included assessment of the appropriateness of extrapolating from other species, the use of safety factors, and co-exposure to other chemicals. Cancer and non-cancer endpoints assessed.

SOIL

- Conducted field research on workers in wood treatment facilities to copper chromium arsenate (CCA), formerly used as a wood preservative. Airborne exposures to hexavalent chromium (Cr VI) and arsenic (As) were of primary focus. Data was submitted to US EPA. New technique for detecting lower quantities was developed. Route of exposure was primarily via inhalation. Cancer and non-cancer endpoints were assessed.
- Conducted a comprehensive risk assessment addressing human health risks related to dioxins and polyaromatic hydrocarbons (PAHs) in soil at a wood treating facility listed by US EPA as a Superfund site. Followed US EPA guidelines for the assessment. Exposure assessment was

ED_001802_00024760-00080

multipathway and included exposures to workers and residents living near the facility. Conducted a probabilistic risk assessment to characterize uncertainty and variability in worker exposures and identify parameters contributing most significantly to uncertainty in risk estimates. Developed site-specific parameter distributions, and characterized current scientific knowledge of the bioavailability of dioxins and PAHs in soil. Work was submitted to US EPA. Cancer and non-cancer endpoints assessed.

BIOLOGICAL RISK ASSESSMENT

- Conducted biological assessment of workers exposed to bioaerosols and particulates from an advanced wastewater treatment plant. Evaluated the chemical and biological constituents of dewatered sludge. Reviewed laboratory data, conducted microbial, viral, and chemical literature review, and conducted site assessments. Route of exposures were inhalation and ingestion. Non-cancer endpoints were assessed. A review of employee protection was also conducted.
- Conducted biological assessment of workers exposed to bioaerosols and to a conditioning agent used to dewater sludge (CLARIFLOC® C-9525 POLYMER) at an advanced wastewater treatment plant. Reviewed laboratory data, conducted microbial, viral, and chemical literature review, and conducted site assessments. Route of exposures were inhalation and ingestion. Non-cancer endpoints were assessed.
- Evaluated the performance of immunoassay biological agent detection instrumentation for instrument developer. Conducted independent tests to determine the minimum level of spore delectability of two specific instruments for viable anthrax spore vaccine and *Bacillus thuringiensis*.
- Conducted an anthrax investigation at a military mail facility. Developed and implemented a sampling and analysis program to determine whether *Bacillus anthracis* (anthrax) organisms could be detected. The biological assessment included a review of the microbiological literature; development of state-of-the-science sampling approach; refining a work plan to address site-specific elements; preparation of work plan; collection of samples; coordination of laboratory analysis; interpretation of results; and preparation of final project documentation and results reporting.
- Provided scientific support for numerous commercial and residential indoor air quality claims concerning alleged adverse health effects due to exposures to microbiological agents. Critically evaluated laboratory data, the method of collection and analysis, medical records, and toxicological literature regarding exposure to fungal toxins in indoor air and their potential for causing long-term adverse health effects. Also included in some assessments was the release of volatile organic compounds from fungi. Assessed validity and reliability of information on the nature and extent of exposure, interpreted receptor health status, evaluated toxicological basis for health complaints, and identified potential sources of confounding causality. Many organisms have been evaluated, however, most commonly evaluated are *Aspergillus, Penicillium* and *Stachybotrys*.
- Developed and conducted an independent sampling program to test certain building materials for the presence of fungal spores and/or hyphal fragments that might indicate current fungal growth or the possibility of fungal growth in the future. Analyses included both direct microscopy for identification of any fungal spores to the genus level and culture of viable samples on appropriate nutrient media.
- Conducted biological assessment of residents exposed to *Salmonella* and *E. coli* emanating as bioaerosols from a cattle feedlot. Reviewed laboratory data, conducted microbial, viral, and

ED_001802_00024760-00081

chemical literature review, and conducted site assessments. Route of exposures were inhalation and ingestion. Non-cancer endpoints were assessed. Prepared a work plan to assess the adverse health effects related to microbial contamination of a potable private well-water supply. Work plan included examination of exposure events, identification of microbial agents of concern (MAOC), and identification of possible health effects associated with direct and indirect contact with MAOCs detected in sewage.

ECOLOGICAL RECEPTORS

- Assisted in the evaluation and identification of sources of contamination for a screening-level ecotoxicologic assessment of select chemicals of potential concern (COPCs). Analysis concluded that sampling locations were associated with several COPCs exceeding their level of concern (LOC) for water. Further evaluation indicated that the relationship between sediment, water, and animal tissue was not always synchronous, COPCs with identified benchmarks should be evaluated, and LOCs not likely to pose a risk should be identified appropriately. Ecological endpoints were assessed. COPCs including organics (chlorpyrifos, hexachlorobenzene, organochlorine pesticides, pentachloroanisole, pentachorobenzene, PCBs, and tetrachlorobenzene) and inorganics (Al, Sb, As, Ba, Be, B, Cd, Cr, Cu, Fe, Pb, Mg, Mn, Hg, Mo, Ni, Se, Sr, Ti, V, and Zn) were analyzed and assessed in water, sediment, fish, and bird eggs.
- Managed a human and ecological risk assessment project for a state Department of Transportation agency. The purpose of the project was to estimate the potential human health and ecological risks associated with agency use of herbicides for roadside vegetation management. The evaluation addressed the general public and workers applying herbicides. Produced a risk assessment report and made herbicide risk management recommendations to the client.
- Conducted toxicological assessment for clean-up of lead and arsenic contaminated soil from smelter operating in the 1900s. Conducted historical toxicological research on articles and records dating back to the 1700s. Assessed what was known and when regarding the toxicology of lead (Pb) and arsenic (As) for all body systems and all exposure pathways for both human and ecological endpoints.
- Conducted a toxicological assessment of human health risks from lead deposited in agricultural soil. Lead was released from the operation of a steel manufacturing plant. Assessment used the US EPA Uptake/Biokinetic Model for Lead to evaluate human exposure. Environmental fate was followed through the food chain from soil to human food sources. A review of the literature was conducted. Route of exposure was primarily ingestion. Non-cancer endpoints were assessed with the focus on nervous system risks.
- Assisted with toxicological assessment of human exposure to an herbicide, acrolein, a pungent chemical used in urban settings to control weeds in public water systems. Odor is likely a human trigger to the presence of this chemical. Routes of exposure included inhalation, ingestion (from water and fish) and dermal exposure (from swimming). Toxicological assessment consisted of a review of the toxicological literature, evaluation of communities, and assessment of accident scenarios. Non-cancer endpoints were assessed.
- Assisted in the evaluation of a proposed Concentrated Animal Feeding Operation (CAFO) facility for potential human health and ecological impacts from possible facility releases for both human and ecological endpoints. The facility was intended for closed loop operation and designed to prepare 20,000 head of cattle for market. Chemicals of concern included hormones, antibiotics, and pesticides. Pathogens were also assessed. This unique CAFO design comprised of the following operating units: beef facility, ethanol producing facility, combined heat and

ED_001802_00024760-00082

power facility, nutrient separator, greenhouse facility, water treatment facility, fluidized bed reactor, and composing facility.

- Conducted human and ecological risk assessment from the effects copper slag leachates. Reviewed laboratory data, conducted toxicological literature review on human and ecological receptors. Evaluated arsenic (As), copper (Cu), cadmium (Cd), and zinc (Zn). US EPA and Washington state guidelines were used as basis for this assessment. For human exposures, cancer and non-cancer endpoints were considered.

POLICY AND LEGISLATION

- Participated in technical discussions with South African governmental and private industry representatives on land, water, and air legislation, and the benefits of science and risk-based environmental legislation.
- Participated in technical discussions with several members of the European Parliament, in Brussels, Belgium, on developing appropriate scientifically based regulations to prevent adverse health effects from burning hazardous waste in cement kilns.

EXPERT PEER REVIEW PANELS

- 2010 Science Advisor for the Nanosafety Consortium for Carbon, Washington, D.C.
- 2004-present Ad Hoc Science Review Board Member of the US Environmental Protection Agency (US EPA) Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) Scientific Advisory Panel.
- 2009 US Environmental Protection Agency (US EPA) Nanomaterial Case Studies Workshop: Developing a Comprehensive Environmental Assessment Research Strategy for Nanoscale Titanium Dioxide, Durham, NC, September 29 and 30.
- 2009 US Environmental Protection Agency (US EPA) - External Peer Review Panel of the Toxicological Review of Nitrobenzene (CAS No. 98-95-3), In Support of Summary Information on the Integrated Risk Information System (IRIS), published January 2009.
- 2009 Expert Peer Panel of Tertiary-Butyl Acetate (TBAC), Toxicology Excellence for Risk Assessment (TERA), Cincinnati, OH. January 7 & 8.
- 2007 US Environmental Protection Agency (US EPA) - Integrated Risk Information System (IRIS) Peer Review of Nitrobenzene, Washington, DC, May 15.
- 2004 Resorcinol Peer Review Meeting: Follow-up Review to 2003, Toxicology Excellence for Risk Assessment, Harrisburg, PA. November 17 & 18.
- 2003 Resorcinol Peer Review Meeting, Toxicology Excellence for Risk Assessment (TBAC), Cincinnati, OH. April 18 & 19.

CONFERENCES AND SYMPOSIUMS

- 2013 Steering Committee member for the upcoming Gordon Conference "Environmental Nanotechnology: Novel Approaches to Meet Global Challenges." Vermont, USA.
- 2012 Invited Expert for the "BRE Cabin Air Quality Workshop." The BRE Group, London, England, February 20-21.
- 2011 Roundtable Participant for the "Washington State Green Chemistry Roundup," the Pacific Northwest Pollution Prevention Resource Center (PPRC), May 25-26.

ED_001802_00024760-00083

- 2010 Invited Speaker for the "Capstone Meeting: Risk Management Methods & Ethical, Legal, and Societal Implications of Nanotechnology", the National Nanotechnology Initiative (NNI), Washington DC, USA, March 30-31.
- 2009 Co-Chair for the In Vitro Plenary of the "Nanomaterials and Human Health & Instrumentation, Metrology, an Analytical Methods" Workshop, the National Nanotechnology Initiative, Washington DC, USA, November. The workshop brings together thoughts and ideas to recommend which direction the federal government's nano EHS research strategy.
- 2009 Planning Committee Chair Member for the "Nanotechnology Health & Safety Forum (NHSF)", Seattle, USA, June. The NHSF explored the multiple perspectives of nanotechnology.
- 2007 Committee Member for the "Naphthalene State-of-the-Science Symposium," University of Nebraska Center for Environmental Toxicology, Monterey, USA. October. The Symposium is a scholarly peer review of critical scientific information underlying a federal health risk assessment.
- 2003 Organizing Committee Member for the "Perchlorate State-of-the-Science Symposium," University of Nebraska Medical Center, Omaha, USA, September. The Symposium is a scholarly peer review of critical scientific information underlying a federal health risk assessment.

SELECT EDUCATIONAL COURSES

- Lecturer 2012. Guidance on Physicochemical Characterization for Manufactured Nano-objects Submitted for Toxicological Testing: ISO TC-229 Project Work. Presented at a Bar-Ilan Institute of Nanotechnology and Advanced Materials Seminar. Tel Aviv, Israel, October 15.
- Lecturer for Department of Pharmacology, University of Nebraska Medical Center. Provided lectures on toxicology for medical, pharmacy, graduate and physician assistant students at the University of Nebraska Medical Center, Department of Pharmacology.
- Presented two courses in human health risk assessment for staff of the Technical Research Council in South Africa. The purpose of the course was to introduce multi-pathway risk assessment as a means for evaluating potential chemical exposures associated with various industrial activities in South Africa.
- Developed and taught over five courses on risk assessment and risk communication for the Air & Waste Management Association. The courses address toxicology, multi-pathway risk assessments for combustion sources, uncertainty analyses, and risk communication.
- Lectured on toxicology of the sensory system and neuroimaging in graduate student courses at the University of Washington.
- Developed and taught a course for public utility workers in the Puget Sound area on the subject of electromagnetic fields and their potential for adverse health effects.
- Developed, managed, and team-taught several courses on toxicology, risk assessment, and risk communication for the managers and staff of chemical plants, utilities, oil companies, railroads, and for government officials. Courses have been presented in California, Pennsylvania, Arizona, Missouri, France, and South Africa.

GRANTS AND AWARDS

- Grant Awarded, WaterRF 4387: Development of a Water Utility Primer on EDCs/PPCPs for Public Outreach (Gretchen Bruce and Richard Pleus, Principal Investigators) 2012.

ED_001802_00024760-00084

- Grant Awarded, Awwarf /WRF 4214: Development of Acceptable Daily Intakes (ADIs) for Pharmaceutical and Personal Care Product Ingredients, Hormonally Active Compounds, and Other Potentially Highly Toxic Compounds of Emerging Interest in Water Using the Minimum Anticipated Biological Effect Level (MABEL) Approach (Richard Pleus and Gretchen Bruce, Principal Investigators) 2008.
- Grant Awarded, Awwarf/WRF 3085/04-003: Toxicological Relevance of Endocrine Disruptors and Pharmaceuticals in Drinking Water (2004-2008); in collaboration with Southern Nevada Water Authority (SNWA) (Shane Snyder, Principal Investigator), 2008.
- Grant Awarded, AwwaRF 3033: Comprehensive Utility Guide for Endocrine Disrupting Chemicals, Pharmaceuticals, and Personal Care Products in Drinking Water (2005-2007); in collaboration with SNWA (Shane Snyder, Principal Investigator), 2007.
- Grant Awarded, WRF-06-018: Tools to Assess and Understand the Relative Risks of Indirect Potable Reuse and Aquifer Storage and Recovery Projects (2006-present) (*DRAFT*); in collaboration with Nellor Environmental Associates, Inc. and Soller Environmental, LLC (Margie Nellor and Jeff Soller, Principal Investigators)
- Grant Awarded, WRF-05-005: Identifying Hormonally Active Compounds, Pharmaceutical Ingredients, and Personal Care Product Ingredients of Most Health Concern from Their Potential Presence in Water Intended for Indirect Potable Reuse (2006-present) (*DRAFT*); in collaboration with SNWA (Shane Snyder, Principal Investigator)
- Elected to Delta Omega Honorary Society in Public Health, 2003.
- Best Paper: Pleus R.C., Goodman G. and Mattie D.R. Development of a Reference Dose for Perchlorate: Current Issues and Status. Presented at the 50th Joint Army-Navy-NASA-Air Force (JANNAF) Propellant Development and Characterization and Safety and Environmental Protection Subcommittees Joint Meeting, Cocoa Beach, FL. May 2000.
- Faculty Mentor of the Year Award, General College Student government, University of Minnesota, Minneapolis, MN. 1989.
- Director of Undergraduate Research Opportunities Program Award, University of Minnesota, for the research proposal, *The effect of fetal hypoxia on fetal brain development.* 1987.
- Director of Undergraduate Research Opportunities Program Award, University of Minnesota, for the research proposal, *Use and operation of autoshaping and fixed ratio paradigm in environmental toxicology research.* 1986.
- Scholl Fellowship, National Sudden Infant Death Foundation, Landover, MD. 1985.
- US Public Health Traineeship Award, United States Public Health Service, Washington, DC. 1979.

PROFESSIONAL MEMBERSHIPS

- American Society for Pharmacology and Experimental Therapeutics
- Association for the Advancement of Science
- Society for Neuroscience
- Society for Risk Analysis
- Society of Toxicology

ED_001802_00024760-00085

DIRECTORSHIPS

- 2010 – present Member, Board of Directors for the Nanotechnology Industries Association (NIA), Brussels, Belgium
- 2004-2007 Member, Board of Directors, Frontier Geosciences, Inc. Seattle, WA.
- 2001-present Member and former Secretary, Board of Directors, Urban Environmental Institute. Seattle, WA.
- 1998-1999 Member, Board of Directors, Northwest Sculling Association. Seattle, WA.
- 1996-1998 Vice President, Seattle Yacht Club Rowing Foundation. Seattle, WA.
- 1989 Member, Board of Directors, Insight, Inc. Stillwater, MN.

ADVISORY POSITIONS

- 2011 U.S. delegate for the U.S.-Russia Bilateral Presidential Commission on Science and Technology, March 1 through 5. Selected for expertise on nano-related EHS issues.
- 2010 – present Chair of the Science Advisory Board, *National Institute of Biomedical Imaging and Bioengineering (NBIB),* Development and Launch of an Interoperable and Curated Nanomaterial Registry, Principal Investigator: Michele L. Ostraat, PhD.
- 2009 – 2011 Project Advisory Committee, WateReuse Foundation / Kennedy/Jenks Consultants, WRF 09-07: Risk Assessment Study of PPCPs in Recycled Water to Support Public Review.
- 2009 Peer Review Panel Member for the National Institute for Occupational Safety and Health (NIOSH) Intramural Proposal "International Coordination of Nanoscale Reference Materials" for the Nanotechnology Research Center (NTRC).
- 2008 - 2011 Project Advisory Committee, WateReuse Foundation, WRF 06-019: Monitoring for Microconstituents in an Advanced Wastewater Treatment (AWT) Facility and Modeling Discharge of Reclaimed Water to Surface Canals for Indirect Potable Use, Florida, USA
- 2008 - 2012 Advisory Board Member, Center for Risk Communication Research, University of Maryland, College Park, MD, USA
- 2007 - 2010 International Advisory Board, USA, International Symposium on Nanotechnology in Environmental Protection and Pollution, Fort Lauderdale, FL, USA
- 2007 - present US Delegate on the International Organization for Standardization (ISO) Technical Committee (TC) 229, Nanotechnologies, leading the U.S. Technical Advisory Group (TAG) Working Group 3 to develop a comprehensive list of physical and chemical characterization parameters of engineered nano-objects for toxicologic assessment.
- 2006 - 2008 Counselor to the Regional Central States Chapter of the Society of Toxicology (CS-SOT).

ED_001802_00024760-00086

- 2006 Stakeholder Advisory Committee Member, to review Development of Indicators and Surrogates for Chemical Contaminant Removal During Wastewater Treatment and Reclamation, WateReuse Foundation Project WRF-03-014. May 16-17, in Phoenix, Arizona.
- 2002 Odors and Toxic Air Emissions Conference Program Committee Member. New Mexico, Rocky Mountain Water Environment Association, Air and Waste Management Association, and the International Water Association.
- 2000-2008 Member, Board of Advisors, Good Company. Eugene, OR.

COMMUNITY SERVICE

- Panelist, Lakeside School Annual Biology Assessment Program. Seattle, WA (2001).
- Member & Co-Director, Mayor's Small Business Task Force. Seattle, WA (1997-2001).
- Member, Sustainable Seattle: a voluntary network and civic forum for sustainability. Seattle, WA (1992-1993).

SELECTED PROFESSIONAL PRESENTATIONS

2012. **Pleus R.C.** Innovative Nano-toxicological Risk Assessment Process for Regulatory Purposes. Presented at the 40th Annual ISEES Conference. Tel Aviv, Israel, October 16-18.

2012. **Pleus R.C.** Brief Examination of the Current Toxicology Research for Carbon Nanotubes (CNTs). Presented to the Nanotubes Empowerment Solutions Consortium. Tel Aviv, Israel, October 15.

2012. **Pleus R.C.** Chemicals, Bleed Air, and Health Effects: What the Science Says. Presented at Aviation Health Conference 2012. London, United Kingdom, October 2-3, 2012.

2010. **Pleus R.C.** Nanomaterials – Understanding and Managing ESOH Risks. Presented at the 8th Annual NanoTechnology for Defense Conference (NT4D). Atlanta, GA, May 3-6, 2010.

2010. **Pleus R.C.** The Importance of Defining Chemical and Physical Parameters for Toxicological Testing of Nanomaterials: Getting Two Scientific Groups to Help Each Other. Presented at the Bureau International des Poids et Mesures (BIPM) Workshop on Metrology at the Nanoscale. Sevres, France, February 18-19.

2009. **Pleus R.C.** Global Standardization: ISO TC 229. Nanotechnology Symposium California Department of Toxic Substances Control (DTSC). Sacramento, CA, November 16.

2009. **Pleus R.C.** Nanomaterials: Steps to Address EHS Concerns That Businesses Should Consider Before Placing Nanomaterials on the Market. Nanotech in the Marketplace Webinar. Nanotechnology Today: A Web Series. June 4.

2009. **Pleus R.C.** Hexavalent Chromium and Mercury in the Cement Industry – Recent Concerns About Human Health Issues. Presented at the 2009 IEEE-IAS/PCA 51st Cement Industry Technical Conference. Palm Springs, CA, June 2.

2009. **Pleus R.C.** Pharmaceuticals & Endocrine Disrupting Chemicals (EDCs) in Water: Development of Health Risk-Based Screening Levels. Presented at the Water Quality Committee Program 2009 ACWA Spring Conference. Sacramento, CA, May 19-22.

2009. **Pleus R.C.** EHS: Policy, Regulation & Product Safety. Presented at the Nano Science and

ED_001802_00024760-00087

Technology Institute (NSTI) 2009 Products and Liability Panel. Houston, TX, May 5.

2009. **Pleus R.C.** Environmental Health & Safety and Nanotechnology: Possible Issues in the Water Industry. Presented at the Washington Innovation Summit 2009. Bellevue, WA, April 9.

2009. **Pleus R.C.** Perchlorate, Pharmaceuticals and Personal Care Products, Endocrine Disrupting Chemicals, and Nanotechnology in Water. Presented for the Association of California Water Agencies (ACWA). Sacramento, CA, February 9.

2008. **Pleus R.C.**, Walker, N., and Canady, R. A Minimal Set of Characterization Parameters. Presented at the Ensuring Appropriate Material Characterization in Nano-Toxicity Studies: A Workshop, Washington, D.C., October 28.

2008. **Pleus R.C.** What We Are Learning About Micro Constituents in Drinking Water: Pharmaceuticals and Endocrine Disruptors. Presented at the 2008 Water Quality and Regulatory Conference, Ontario, CA, October 16.

2008. **Pleus R.C.** Pharmaceuticals, Endocrine Disrupting Chemicals (EDCs), and Personal Care Products (PCPs) in Untreated and Treated Drinking Water: What We Know So Far. Presented at the AWWA / PNWS sponsored seminar, Pharmaceuticals in Water and Wastewater, Hillsboro, OR, September 11.

2008. **Pleus R.C.** Nanotechnology: Risk, Health, and Environmental Perspectives: Toxicology and Nano-objects. Boeing, Seattle, WA, July 25.

2008. **Pleus R.C.** Endocrine Disrupting Compounds (EDCs) and Pharmaceuticals and Personal Care Products (PPCPs). AWWA Webcast, May 7.

2008. **Pleus R.C.** Toxicology of Endocrine Disruptors (EDCs): Excerpts from *Toxicological Relevance of Endocrine Disruptors and Pharmaceuticals in Drinking Water, project #3085.* Presented at the EDC Communication - AwwaRF Research On EDC's and Risk Communication for D.C. Region Stakeholders, Washington, D.C., April 11.

2008. **Pleus R.C.** Toxicological Relevance of Endocrine Disrupting Chemicals (EDCs) and Pharmaceuticals in Water. AwwaRF Project #3085 / WRF 04-003. AwwaRF Webcast, March 6.

2008. **Pleus R.C.** Strategy Used to Build Toxicological Database for Emerging Toxic Chemicals in Litigation Focusing on the Mechanism of Action—Yes, This Is Rocket Science! Presented at the DRI Conference, Phoenix, AZ, February 6, 2008.

2007. Linkov I., Peterson M.K., Corey L.M., and **Pleus R.C.** Assessing Environmental Risk of Nanomaterials: Approaches and Tools. 2007 NSTI Nanotechnology Conference and Trade Show, Santa Clara, CA, May 20-24.

2007. Snyder E.M., Bruce G.M., **Pleus R.C**., and Snyder S.A. Incidence and Toxicological Significance of Selected Endocrine Disrupting Chemicals (EDCs) in Drinking Water. Presented at the World Environmental and Water Resources Congress 2007, Tampa, FL, May 15-19.

2006. Snyder S.A., **Pleus R.C.** Human Health Implications from Nanoparticles in Water. Presented at The2nd International Symposium on Environmental Nanotechnology, South Korea, November 3.

2006. **Pleus R.C**., Snyder S.A. Risk Assessment of Pharmaceuticals and Endocrine Disruptors in Drinking Water. Presented at The Western Coalition of Arid States Conference, Tucson, AZ, November 2.

ED_001802_00024760-00088

2006. **Pleus R.C**., Snyder S.A. Toxicological Relevance of Pharmaceuticals and Endocrine Disruptors in Drinking Water. Presented to the Orange County Utilities; Water Quality Section Conference, Orlando, FL, October 26.

2006. Linkov I., **Pleus R.C.**, Stevens J., and Ferguson E. EPA Peer Review Panel Recommendations on Environmental Risk of Nanomaterials & Multi-Criteria Decision Analysis and Environmental Risk Assessment for Nanomaterials. Presented at the US Army Nanotechnology Development Coordination Meeting, Cambridge, MA, August 15-17.

2006. **Pleus, R.C.** Perchlorate in 2006: Where are we and where are we going? Invited speaker. Presented at the 2006 Superfund Program Managers Symposium, Scottsdale, AZ, August 13-16.

2006. Corey L.M., Peterson M.K., **Pleus R.C**. Nanotechnology Environmental Health and Safety (EHS): Current Knowledge and Future Challenges. Presented at the 9th Annual Force Health Protection Conference, Albuquerque, NM, August 6-11.

2006. Corey L.M., Peterson M.K., **Pleus R.C**. Nanotoxicology: Special Considerations for Assessing Risks from Very Small Particles. Presented at the 9th Annual Force Health Protection Conference, Albuquerque, NM, August 6-11.

2006. Corey L.M., Peterson M.K., **Pleus R.C.** Developing Nanotechnology Health and Safety Standards. Invited speaker. Presented at the 2006 Micro Nano Breakthrough Conference, Vancouver, WA, July 25, 2006.

2006. **Pleus R.C.**, Bruce G.M., Snyder E.M., Snyder S.A., and Corey L.M. Toxicological Relevance of EDCs and Pharmaceuticals. Invited speaker. Presented at the 2006 AWWA Annual Conference in San Antonio, TX. June 11-15.

2006. **Pleus R.C.**, Bruce G.M., Snyder E.M., Snyder S.A., and Corey L.M. Incidence and Toxicological Significance of Selected Pharmaceuticals in Drinking Water. Presented at the Groundwater Resources Association's Emerging Contaminants in Groundwater Symposium, Concord, CA. June 7-8.

2006. **Pleus R.C.,** Bruce G.M., Snyder E.M. Addressing the Significance of Trace Level Findings. Presented at the Association of California Water Agencies Groundwater/ Water Quality Track: Pharmaceuticals in Groundwater: Public Health Issue or Public Relations Nightmare? Monterey, CA. May 10.

2006. Bruce G.M., **Pleus R.C.,** Snyder S.A., and Snyder E.M. Toxicological Relevance of Pharmaceuticals and Endocrine Disrupting Chemicals in Water. Presented at the National Ground Water Association's 5th International Conference on Pharmaceuticals and Endocrine Disrupting Chemicals in Water, Costa Mesa, CA. March 14, 2006.

2005. Snyder E.M., Snyder S.A., **Pleus R.C.**, Bruce G.M., Hemming J.D.C., and Hulsey R.A. Approach for Assessing the Toxicological Relevance of Endocrine Disruptors and Pharmaceuticals in Drinking Water. Submitted to Water Quality Technology Conference and Exhibition, Quebec, Canada. November 6-10.

2005. Corey L.M., Bruce G.M., **Pleus R.C.** Development of Nano-Based Risk Assessments: Challenges for the Present and Future. Mechanisms of Action of Inhaled Fibers, Particles and Nanoparticles in Lung and Cardiovascular Disease, Research Triangle Park, NC. October 25-28.

2005. **Pleus, R.C.** Perchlorate: Where We Are and Where We Are Going? Presented at the Environmental Law Conference at Yosemite, CA. October 22.

ED_001802_00024760-00089

2005. **Pleus, R.C.** Emerging Chemicals of Concern-Effective Toxicological Assessment. Presented for the Society of Toxicology, Central State Chapter, Ames, IA. September 30.

2005. **Pleus, R.C.** Emerging Chemicals—Health Concerns About Endocrine Disruptors & Pharmaceuticals in Drinking Water Supplies. Presented at Mealey's Water Contamination Conference in Los Angeles, CA. September 26-27.

2005. **Pleus R.C.** Methods to Derive Safe Drinking Water Levels for Chemicals in Drinking Water. Invited to present at the 2005 Annual Conference & Exposition of the American Water Works Association: Natural Poisons & Unnatural Products Session, San Francisco, CA. June 14.

2005. Bruce G.M. and **Pleus R.C.** Private Toxicology: Testing and Analysis. Invited to present at the Winning: Hot Topics in Criminal Law—Alternatives to the State Crime Lab, Seattle, WA. May 24.

2005. Bruce G.M., Peterson M.K., and **Pleus R.C.** Comparative Risk Assessment Of Multimedia Environmental Exposure To Perchlorate and Other Agents That Inhibit Iodide Uptake Into The Thyroid. Poster presented at the Society of Toxicology 44th Annual Meeting, New Orleans, LA. March 10.

2005. Peterson M.K., **Pleus R.C.,** and Hays S.M. Assessing the Risks Associated with Children Ingesting Lead in School Drinking Water: PBPK Modeling and Risk Communication. Poster presented at the Society of Toxicology 44th Annual Meeting, New Orleans, LA. March 8.

2005. Dodge D.G., Peterson M.K., and **Pleus R.C.** Addressing Toxicological Challenges to Community Water Fluoridation in Washington State. Poster presented at the Society of Toxicology 44th Annual Meeting, New Orleans, LA. March 7.

2004. **Pleus R.C.** 2004 Update: What Do Human Data Tell Us About How Much Perchlorate Exposure is 'Safe'? Presented at the 2004 Water Quality Conference, Ontario, CA. October 26-28.

2004. **Pleus R.C.** Perchlorate dose-response relationship and the likelihood of effects at environmentally relevant levels. Presented at the URS Seminar—Perchlorate: Rush to Judgment or Serious Health Threat, Seattle, WA. September 28th.

2004. **Pleus R.C.** Research, Discovery, and Contribution: Professional Experience in the Republic of South Africa. Invited speaker. Presented at the Pacific Northwest Association of Toxicologists (PANWAT) Annual Meeting: Toxicology in Third World Settings, Bend, OR. September 19th.

2004. **Pleus R.C.** Product Liability: Emerging Contaminants of Concern. Presented to Bullivant Houser Bailey, Seattle, WA and their satellite offices via video-conference. September 2.

2004. **Pleus R.C.** Perchlorate dose-response relationship and the likelihood of effects at environmentally relevant levels. Presented at the 228th ACS National Meeting, Philadelphia, PA. August 22-26.

2004. **Pleus R.C.** and Bruce G.M. Where Are We Now? An Update on the Perchlorate Action Level Debate. Presented at the 7th Annual Force Health Protection Conference, Albuquerque, NM. August 8-12.

2004. Belzer R.B., **Pleus R.C.,** Bruce G.M., and Peterson M.K. Using Comparative Exposure Analysis to Validate Low-Dose Human Health Risk Assessment: The Case of Perchlorate. Presented at the 7th Annual Force Health Protection Conference, Albuquerque, NM. August 8-

ED_001802_00024760-00090

12.

2004. **Pleus R.C.** and Bruce G.M. Perchlorate dose-response relationship and the likelihood of effects at environmentally relevant levels. Presented at the Groundwater Resources Association of California Conference, Glendale, CA. August 4th.

2004. **Pleus R.C.** Asthma and Fungi: State of the Science. Presented at the ASTM International Boulder Conference on Mold in the Indoor Environment: Assessment, Health and Physical Effects, and Remediation, University of Colorado at Boulder, Boulder, CO. July 25-30.

2004. **Pleus R.C.**, Bruce G.M., Peterson M.K., and Dodge D.G. Comparative Contribution of Perchlorate and Anti-Thyroid Agents in American Diets to Iodide Uptake Inhibition. Paper presented at the 32nd Propellant Development & Characterization Subcommittee (PDCS) and the 21st Safety & Environmental Protection Subcommittee (S&EPS) Joint Meeting, Seattle, WA, July 25-29.

2004. **Pleus R.C.** Perchlorate dose-response relationship: Evidence from human studies. Presented at the 227th American Chemical Society National Meeting, Anaheim, CA. April 1.

2004. **Pleus R.C.** Establishing a Safe Dose for Perchlorate Based on Human Evidence of a No Effect Level. Presented at the Society of Toxicology 43rd Annual Meeting, Baltimore, MD. March 24.

2004. Peterson M.K. and **Pleus R.C.** Comparative Analysis of Neuropsychological Toxicity of Biological, Chemical, and Pharmaceutical Agents. Presented at the Society of Toxicology Annual Meeting, Baltimore, MD. March 22.

2004. **Pleus R.C.** Perchlorate: The Greer Study, the Critical Animal Studies, and the Process of Evaluation by the National Academy of Science. Presented at the 14th Annual West Coast Conference on Soils, Sediments, and Water, San Diego, CA. March 16.

2004. **Pleus R.C.** and Bruce G.M. Adverse Effect Levels for Neurodevelopmental Effects Associated with Maternal Perchlorate Exposure: What do Existing Data Indicate? Presented at the 21st International Neurotoxicology Conference, Honolulu, Hawaii. February 12.

2003. **Pleus R.C.** Considerations related to sampling for bioterrorism agents. Presented at the Society for Risk Analysis Meeting—Bridging Risk Divides: Risk Assessment and Risk Communications Methodologies for Bioterrorism Incident Response Symposia, Baltimore, MD. December 8.

2003. **Pleus R.C.** A Review of the Science Required to Establish an Informed MCL for Perchlorate in Drinking Water. Presented to the Perchlorate Review Scholars Committee Urban Water Research Center, University of California, Irvine, CA. October 21.

2003. **Pleus R.C.** Perchlorate: The Questions You Have and The Answers You Need Presented at Fresh Summit 2003: Produce Marketing Association's 54th International Convention & Exposition, Orlando, FL. October 19.

2003. **Pleus R.C.** What do we know about the neurotoxic effects of chemicals in aircraft cabin air? Presented at the Northwest Occupational Health Conference Pacific Northwest Section of the American Industrial Hygiene Association, Seattle, WA. October 16.

2003. **Pleus R.C.** The Greer Study: Discussion of the Key Points. Presented at the UNMC Perchlorate State-of-the-Science Symposium, Omaha, NE. September 30.

ED_001802_00024760-00091

2003. **Pleus R.C.** Making Sense of the Perchlorate Action Level Debate. Presented at the 6th Annual Force Health Protection Conference, Albuquerque, NM. August 11-14.

2003. **Pleus R.C.** Quantifying the Effects of Perchlorate. Presented at the California Minor Crops Council Technical Committee Meeting, Irvine, CA. June 26.

2003. **Pleus R.C.**, Bruce G.M., and Peterson M.K. Assessing Neurodevelopmental Effects of Environmental Exposures to Anti-Thyroid Agents: How Relevant are High Dose Rat Studies? Presented at the Society of Toxicology 42nd Annual Meeting, Salt Lake City, UT. March 9-13.

2003. Bruce G.M., **Pleus R.C.,** and Peterson M.K. Dose-Response Investigation of Tricresyl Phosphates Potentially Present in Airplane Cabin Air from Jet Engine Oils. Presented at the Society of Toxicology 42nd Annual Meeting, Salt Lake City, UT. March 9-13.

2003. **Pleus R.C.** Invited as Expert Panelist and presented, Making Sense of the Perchlorate Action Level Debate at AFCEE Technology Transfer Workshop, San Antonio, TX. February 24-27.

2003. **Pleus R.C.** Making Sense of the Perchlorate Action Level Debate. Presented at the 22nd Meeting of the RCC – Environmental Group AFCEE, San Francisco, CA. February 11-13.

2003. **Pleus R.C.** Dose-Response Investigation of Tricresyl Phosphates Potentially Present in Airplane Cabin Air from Jet Engine Oils. Presented at the 20th Annual International Aircraft Cabin Safety Symposium. Universal City, CA. February 10-13.

2002. Belzer R.B., Johnson D., Peterson M.K., and **Pleus R.C.** Comparative Risk Assessment for Perchlorate: How does the US EPA's RfD Compare to Other Goitrogens that are Found in the US Diet. Presented at the Society for Risk Analysis Annual Meeting: Symposium on Perchlorate: Policy Implications, New Orleans, LA, December 8-11.

2002. **Pleus R.C.** and Bruce G.M. Assessing Developmental Neurotoxicity for Environmental Chemicals. Presented at the Society for Risk Analysis Annual Meeting Symposium on Perchlorate: Policy Implications, New Orleans, LA, December 8-11.

2002. Peterson M.K., Bruce G.M., and **Pleus R.C.** Implications for the Use of Thyroid Endpoints from Rat Reproductive/Developmental Toxicity Studies in Human Risk Assessment. Presented at the Society for Risk Analysis Annual Meeting Symposium on Perchlorate: Policy Implications, New Orleans, LA, December 8-11.

2002. **Pleus R.C.** Making Sense of the Perchlorate Action Level Debate. Presented at the SERDP Partners in Environmental Technology Technical Symposium & Workshop, Washington, D.C. December 5.

2002. **Pleus R.C.** What Do Human Data Tell Us About How Much Perchlorate Exposure is 'Safe'? Presented at the Perchlorate Conference, Ontario, CA, October 16.

2002. Bruce G.M., Peterson M.K., and **Pleus R.C.** Sequence of Neurodevelopmental Effects of Anti-thyroid Agents in Rat Offspring: What Should We Expect to See? Poster presented at the NIEHS Thyroid Hormone & Brain Development Conference, Research Triangle Park, NC, September 23-25.

2002. Wahlsten D., Colbourne F., and **Pleus R.C.** High throughput rat and mouse brain morphometry for toxicology research. Poster presented at the NIEHS Thyroid Hormone & Brain Development Conference, Research Triangle Park, NC, September 23-25.

ED_001802_00024760-00092

2002. Belzer R.B., Bruce G.M., Peterson M.K., and **Pleus R.C.** Exposure to Anti-thyroid Chemicals in the Environment and Diet. Poster presented at the NIEHS Thyroid Hormone & Brain Development Conference, Research Triangle Park, NC, September 23-25.

2002. **Pleus R.C.** Understanding Mold-Related Health Effects. Presented at the Emerging Environmental Issues Workshop Environmental Issues In Transactions: The New Landscape & Mold: Why a Headline Now? Sidley Austin Brown & Wood, Chicago, IL, June 7.

2002. **Pleus R.C**. with Boss, et al. Decommissioning – Biological Risk course: Risk Assessment and Risk Communication: Strategic Tools. Presented at the American Industrial Hygiene Conference & Exposition, San Diego, CA, June 1-2.

2002. Peterson M.K., Bruce G.M., and **Pleus R.C.** Identification and Risk Assessment of Odorous Chemicals Associated with Combustion Processes. Poster presented at the Air & Waste Management Association's Hazardous Waste Combustors Specialty Conference & Exhibition, St. Louis, MO, April 17-19.

2002. **Pleus R.C.** The Toxicology of Terror and Tragedy. Presented at the Western Washington Emergency Network Conference, Bellevue, WA, April 2-3.

2002. **Pleus R.C.** Understanding Mold-Related Health Effects. Presented at the Mold Mania! A Growing Concern for the Insurance Industry seminar, Pacific Northwest Chapter of the CPCU Society, Seattle, WA, March 13.

2002. Bruce G.M., Johnson D. and **Pleus R.C.** Assessment of the Validity of US EPA's Interpretation of an Effect of Altered Neurobehavior in Offspring Treated with Perchlorate *in utero*: A Critical Review of the Argus (1998) and Bekkedal *et al.* (2000) Studies. Submitted to Eastern Research Group, Inc. for the US EPA/ORD Peer Review Workshop-Perchlorate Environmental Contamination: Toxicological Review and Risk Characterization, March 5-6, Sacramento, CA. February 19.

2002. Bruce G., Peterson M.K., Lincoln D.L., and **Pleus R.C.** Review and assessment of TSH and Thyroid Hormones during Pregnancy in the Rat and Human and Comparison to Hormone Values in the 2001 Effects Study. Submitted to Eastern Research Group, Inc. for the US EPA/ORD Peer Review Workshop-Perchlorate Environmental Contamination: Toxicological Review and Risk Characterization, March 5-6, Sacramento, CA. February 19.

2002. Bruce G. and **Pleus R.C.** Summary of the Expert Review of the Argus, 2001 ("Effects Study") Evaluation of Perchlorate Effects on Brain Morphometry in Neonatal Rats. Submitted to Eastern Research Group, Inc. for the US EPA/ORD Peer Review Workshop-Perchlorate Environmental Contamination: Toxicological Review and Risk Characterization. March 5-6, Sacramento, CA. February 19.

2002. INTERTOX, INC. Summary of the 1999 External Peer Review Panel Workshop, Submitted to Eastern Research Group, Inc. for the US EPA/ORD Peer Review Workshop-Perchlorate Environmental Contamination: Toxicological Review and Risk Characterization. March 5-6, Sacramento, CA. February 19.

2002. Johnson D. and **Pleus R.C.** Assessment of Neuropsychological Studies by Haddow et al. (1999) and Others Cited By US EPA to Support Their Concerns for Developmental Deficits Related to Maternal Thyroid Deficiency. Submitted to Eastern Research Group, Inc. for the US EPA/ORD Peer Review Workshop-Perchlorate Environmental Contamination: Toxicological Review and Risk Characterization. March 5-6, Sacramento, CA. February 19.

ED_001802_00024760-00093

2001. **Pleus R.C.** Mercury Toxicology: Managing for Mercury in the Waste Stream. Presented at The WA State Recycling Association Workshop: Managing for Mercury in the Recycling Stream, Olympia, WA, November 16.

2001. **Pleus R.C.** Understanding Mold-Related Health Effects. Is the Mold Rush the New Litigation Gold Rush? Presented at the Strategies for Taking the "Gold" Out of Mold Claims workshop, Bullivant Houser Bailey, PC, Seattle, WA, November 8.

2001. Peterson M.K., Bruce G.M., Johnson D.L., and **Pleus R.C.** Evaluation of Risks and Health Effects in Humans Exposed to the Herbicide Dinoseb: A Case Study. Poster presented at the 2001 Society for Risk Analysis Annual Meeting, Seattle, WA, December 2-5.

2001. Goodman G. and **Pleus R.C.** Report on Six Expert Reviews of the Levy Report (J. Levy, J.D. Spengler, D. Hlinka, and D. Sullivan, Estimated Public Health Impacts of Criteria Pollutant Air Emissions from the Salem Harbor and Brayton Point Power Plants, May 2000). Prepared for USGen New England, Inc., Boston, MA. August 4.

2001. **Pleus R.C.**, Goodman G. and Mattie D.R. Development of a Reference Dose for Perchlorate: Current Issues and Status. Paper presented at 50th JANNAF (Joint Army-Navy-NASA-Air Force) Propulsion Meeting, Salt Lake City, UT, July 12.

2001. **Pleus R.C.** and Bruce G. Report on Five Expert Reviews of the Primedica 2001 Study Report (Hormone, Thyroid and Neurohistological Effects of Oral (Drinking Water) Exposure to Ammonium Perchlorate in Pregnant and Lactating Rats and in Fetuses and Nursing Pups Exposed to Ammonium Perchlorate During Gestation or via Maternal Milk, March 2001). Prepared for the Perchlorate Study Group. May 16.

2000. Greer M.A., Goodman G., **Pleus R.C.** and Greer S. Does Environmental Perchlorate Exposure Alter Human Thyroid Function? Determination of the Dose-Response for Inhibition of Radioiodine Uptake. Paper presented at the International Thyroid Meeting, Kyoto, Japan, October 22-27.

2000. Greer M.A., Goodman G., **Pleus R.C.** and Greer S.E. Dose-Response for Perchlorate Effects on Thyroid Function in Human Subjects: Assessment of Environmental Risks. Submitted to US EPA. June 30.

2000. **Pleus R.C.** and Fulton K. Risk Assessment and Risk Communication: Strategic Tools. Course presented at the Air & Waste Management Association 93rd Annual Conference, Salt Lake City, UT, June 18-22.

2000. **Pleus R.C.**, Goodman G., and Mattie D.R. Development of a Reference Dose for Perchlorate: Current Issues and Status. Paper presented at the JANNAF Propellant Development & Characterization Subcommittee and 18th Safety & Environmental Protection Subcommittee Joint Meeting, NASA Kennedy Space Center, FL, May 11.

1999. Goodman G. and **Pleus R.C.** Study of Perchlorate Pharmacokinetics and Inhibition of RAIU in Humans. Protocol submitted to and approved by the Institutional Review Board of Oregon Health Sciences University. Study director: Monte Greer, MD. November 19.

1998. Goodman G. and **Pleus R.C.** Recommendations to the US EPA concerning the derivation of a reference dose for perchlorate. Prepared for the American Pacific Corporation, Submitted to the National Center for Environmental Assessment. Research Triangle Park, NC. September.

1998. Rogers D.E.C., Terblanche P., and **Pleus R.C.** Health risk assessment: its introduction to

ED_001802_00024760-00094

South Africa for the regulation of emissions from medical waste incinerators. Presented at the *Papers of 11th World Clean Air and Environmental Congress, Volume 3.* Durban, South Africa.

1998. **Pleus R.C.** Risk Assessment and Risk Communication: Strategic Tools. Course presented at the Air & Waste Management Association Waste Combustion in Boilers and Industrial Furnaces Specialty Conference, Kansas City, MO, April 14.

1998. Johnson D.L. and **Pleus R.C.** Current Issues that Effect the Estimate of Cancer Risks and Noncancer Hazards in Multipathway Risk Assessments for BIF Facilities. Paper presented at the Air & Waste Management Association Waste Combustion in Boilers and Industrial Furnaces Specialty Conference, Kansas City, MO, April 15-16.

1998. **Pleus R.C.** and Johnson D.L. Assessing the Risks and Costs to Environmental Cases: Case Studies of Management Responses to Community Discontent. Paper presented at the Air & Waste Management Association Waste Combustion in Boilers and Industrial Furnaces Specialty Conference, Kansas City, MO, April 15-16.

1998. **Pleus R.C.**, Dunn L. and Rogers D.E.C. Comparison of the Use of Risk Assessment for Human Health and Ecological Assessments in Developed and Developing Countries. Paper presented at the 11th World Clean Air and Environment Congress, Durban, South Africa, September 13-18.

1997. **Pleus R.C.** and Boss M.J. Risk Assessment and Risk Communication: Strategic Tools. Course presented at the Air & Waste Management Association Waste Combustion in Boilers and Industrial Furnaces Specialty Conference, St. Louis, MO, April 7.

1997. Shirai J., **Pleus R.C.** and Perry M. Chemical Characteristics of Cement Kiln Dust and their Effect on Dioxin-Related Health Risks. Paper presented at the Air & Waste Management Association Waste Combustion in Boilers and Industrial Furnaces Specialty Conference, St. Louis, MO, April 8-10.

1997. **Pleus R.C.** Introduction to Risk Assessment and Risk Communication: Training Course I. Course presented at the Council on Scientific and Industrial Research (CSIR), Pretoria, South Africa, March 3.

1997. **Pleus R.C.** International Trend in Health Risk Assessment. Lecture to South African governmental officials, CSIR Boardroom, Building 46, Pretoria, South Africa, March 11.

1997. Perry M. and **Pleus R.C.** What are the Neighbors Smelling? Odor Investigation of a Portland Cement Plant. Paper presented at the Nevada Water Pollution Control Association Annual Conference, Las Vegas, NV, March 7.

1997. Perry M. and **Pleus R.C.** Managing Corporate and Citizen Response to Foul Odors: Case Studies Involving Commercial Disposal and Production Facilities. Paper presented at the Air & Waste Management Association Annual Meeting, Toronto, Canada, June 8-13.

1996. **Pleus R.C.** Risk Assessment and Risk Communication: Strategic Tools. Course presented at the Air & Waste Management Association Waste Combustion in Boilers and Industrial Furnaces Specialty Conference, Kansas City, MO, March 25.

1996. **Pleus R.C.** and Boss M.J. Risk Assessment, Risk Communication, and Risk Management: Strategic Tools. Course presented at the Air & Waste Management Association Clean Air Conference, Orlando, FL, November 19.

1995. **Pleus R.C.** Risk Assessment and Risk Communication: Strategic Tools. Course presented

ED_001802_00024760-00095

at the Air & Waste Management Association Waste Combustion in Boilers and Industrial Furnaces Specialty Conference, Kansas City, MO, March 27.

1995. **Pleus R.C.** and Minter S.L. Odor Investigation of a Portland Cement Plant. Paper presented at the Air and Waste Management Association Odors: Indoor and Environmental Air International Specialty Conference, Bloomington, MN, September 13-15.

1995. Brankovan V., **Pleus R.C.,** and Molholt B. A Reference Dose Concentration (Rfd) for Lithium. Paper presented at the Vth COMTOX Symposium on Toxicology and Clinical Chemistry of Metals, Vancouver, British Columbia, Canada, July 10-13.

1995. **Pleus R.C.** and Kelly K.E. Health Effects Of Hazardous Waste Incineration Facilities…More of the Rest of the Story; An Updated Review of the Scientific Basis of Alleged Adverse Health Effects of Hazardous Waste Incineration. Paper presented at the Incineration Conference, Bellevue, WA, June.

1994. **Pleus R.C.** Q & A's Regarding Recent US EPA Testing of Dioxins and Furans in CKD.

1994. Kelly K.E. and **Pleus R.C.** Health effects of hazardous waste incineration...more of the story. Paper presented at Incineration Conference, Houston, TX, May.

1994. Kelly K.E. and **Pleus R.C.** Health effects of hazardous waste incineration. Paper presented at the Medichem Conference, Melbourne, Australia, October 18-21.

1993. Kelly K.E. and **Pleus R.C.** Identifying the species of metal air toxics of greatest concern to human health and the environment. Paper presented at the Air & Waste Management Association Current Issues in Air Toxics Conference, Sacramento, CA, November 15-16.

1993. **Pleus R.C.** and Kelly K.E. Health effects of burning hazardous waste in hazardous waste incinerators: US and international experience. Paper presented at the International Congress on Health Effects of Hazardous Waste, Health and Human Services, Atlanta, GA, May 3-6.

1993. **Pleus R.C.** and Kelly K.E. Health effects of burning hazardous waste in cement kilns. Paper presented at the International Congress on Health Effects of Hazardous Waste, Health and Human Services, Atlanta, GA, May 3-6.

1993. **Pleus R.C.** and Kelly K.E. The health effects of burning hazardous waste in cement kilns in the US. Paper presented at the Spalování, nebezpecnych odpadu v cementárnách, Bmo, Republic of Czech, January 27.

1992. O'Rourke M.F., **Pleus R.C.**, Iversen L.J. and Bylund D.B. Pharmacologic characterization of alpha-2 adrenergic receptor heterogeneity in rat brain. Abstract. *Soc. Neurosci. Abstr.* 18: 457.

1992. Blaxall H.S., **Pleus R.C.**, Cerutis D.R., Hass N.A. and Bylund D.B. Regulation of the alpha-2C adrenergic and $5HT_{1B}$ serotonergic receptors by dexamethasone in an opossum kidney (OK) cell line. Abstract. *Soc. Neurosci. Abstr.* 18: 1538.

1992. Cerutis D., **Pleus R.C.**, Blaxall H., Hass N. and Bylund D.B. Characterization of an alpha-2 adrenergic receptor expressed in the human pineal gland. Abstract. *FASEB*, April.

1992. **Pleus R.C.**, Shiue C.Y., Shiue G.G., Rysavy J.A., Huang H., Frick M.P. and Bylund D.B. Carbon-11 labeled alpha-2 adrenergic receptor antagonist: Synthesis of [^{11}C]WY 26703 and its biodistribution in rodents. Abstract. IXth International Symposium on Radiopharmaceutical Chemistry, Paris, France, 6-10 April.

1992. Shiue C.Y., Bai L., Shiue G.G., Rysavy J.A., **Pleus R.C.**, Huang H., Frick M.P., Catt J.D.

ED_001802_00024760-00096

and Yevich J.P. Fluorine-18 labeled BMY 14802: Synthesis and anatomical distribution in rodents. Abstract. IXth International Symposium on Radiopharmaceutical Chemistry, Paris, France, 6-10 April.

1992. **Pleus R.C.**, Shiue C.Y., Shiue G.G., Rysavy J.A., Huang H., Frick M.P., and Bylund D.B. 1992. Comparison of [^{11}C]MK-912 and[^{11}C]WY26703 as alpha-2 adrenergic receptor ligands. Abstract. *J. Nucl. Med.* 5: 861.

1991. **Pleus R.C.** and Bylund D.B. 1991. Regulation of the $5HT_{1B}$ receptor in opossum kidney cells by serotonin. Abstract. *Soc. Neurosci. Abstr.* 17: 1175.

1991. Shiue C.Y., Shiue G.G., Bai L.Q., Huang H., Rysavy J.A., **Pleus R.C.**, Sunderland J.J. and Frick M.P. Fluorine-18 and carbon-11 labeled amphetamine analogs: Synthesis, biodistribution in mice and the effect on D-2 receptor binding. Abstract. *J. Nucl. Med.* 32: 994.

1990. **Pleus R.C.** and Bylund, D.B. 1990. Serotonin down-regulates $5HT_{1B}$ receptors in opossum kidney cells. Abstract. *Soc. Neurosci. Abstr.* 16: 495.

1988. **Pleus R.C.** and Sparber S.B. Acute toxicity of methadone, measured as hypoxia and hypercapnia, in pregnant rats. Abstract. *Soc. Neurosci. Abstr.* 14: 34.

1987. **Pleus R.C.** and Sparber S.B. 1987. Transcutaneous monitoring of O_2 and CO_2 in conscious and methadone treated rats: Underestimates of hypoxia due to physiological adaptation in control subjects. Abstract. International Narcotics Research Conference, Adelaide, South Australia, 31 August-4 September.

1986. **Pleus R.C.** Reactive astrogliosis and astrocytosis in infants who died of sudden infant death syndrome. Abstract. 7th European Conference on Brain Research, Val Thorens, France, 9-14 March.

1985. **Pleus R.C.** Chemical composition of cosmetic products. Department of Professional Development and Conferences, University of Minnesota, St. Paul, MN, Lecturer (also 1979, 1980, and 1982).

1983. **Pleus R.C.** Physiological factors contributing to accidental susceptibility. Midwest Center for Occupational Health and Safety, St. Paul Ramsey Hospital, St. Paul, MN, Lecturer (also 1981 and 1982).

1983. **Pleus R.C**. 1983. Physiological factors contributing to accident susceptibility. Course notes. Midwest Center for Occupational Health and Safety, St. Paul, MN.

1981. **Pleus R.C.** Effects of hazardous wastes on ground water in Minnesota. Paper presented at the University YMCA, Minneapolis, MN.

SELECTED PROFESSIONAL PUBLICATIONS

ISO/PDTR 13014:2012. 2012. Project Leader: **Pleus R.C.** Nanotechnologies – Guidance on Physico-Chemical Characterization for Manufactured Nano-Objects Submitted for Toxicological Testing, Geneva.

Bruce, G.M., L.M. Corey, J.H. Mandel, and **R.C. Pleus**. 2012. Urinary Nitrate, Thiocyanate, and Perchlorate and Serum Thyroid Endpoints Based on Nhanes 2001 to 2002.*J Occup Environ Med.* E-pub Ahead of Print.

Snyder S., Lue-Hing C., Cotruvo J., Drewes J.E., Eaton A., **Pleus R.C.**, Schlenk D. 2010.

ED_001802_00024760-00097

Pharmaceuticals in the Water Environment. In association with *the National Association of Clean Water Agencies* and *the Association of Metropolitan Water Agencies.*

Bruce G.M., **Pleus R.C.**, Snyder S.A. 2009. Toxicological Relevance of Pharmaceuticals in Drinking Water. *Environmental Science & Technology* 44(14): 5619-26.

Bruce G.M., **Pleus R.C.**, Peterson M.K., Snyder S.A. 2009. Toxicological Relevance of Endocrine Disrupting Chemicals in Untreated and Treated Drinking Water. In process.

Belzer R.B., Bus J.C., Cavalieri E.L., Lewis S.C., North D.W., **Pleus R.C.** 2008. The Naphthalene State of the Science Symposium: Objectives, Organization, Structure, and Charge. *Regulatory Toxicology and Pharmacology* 51, 2; Suppl. 1: 1-5.

Snyder S.A., Vanderford B.J., Drewes J., Dickenson E., Snyder E.M., Bruce G.M., **Pleus R.C.** 2008. State of Knowledge of Endocrine Disruptors and Pharmaceuticals in Drinking Water, *Awwa Research Foundation*, Denver, CO.

Linkov I., Satterstrom F.K., Steevens J., Ferguson, E., **Pleus R.C.** 2007. Multi-criteria decision analysis and environmental risk assessment for nanomaterials. *Journal of Nanoparticle Research: 543-554.*

Snyder S.A., **Pleus R.C.**, Vanderford B.J., Holady J.C. 2006. Perchlorate and chlorate in dietary supplements and flavor enhancing ingredients. *Analytica Chimica Acta* 567, 1: 26–32.

Snyder E.M., **Pleus R.C.**, Snyder S.A. 2005. Pharmaceuticals and EDCs in the US water industry- an update. *Journal of the American Water Works Association* 97, 11: 32-36.

Chow J.C., Watson J.G., Savage N., Solomon C.J., Cheng Y., McMurry PH, Corey LM, Bruce GM, **Pleus RC**, Biswas P, Wu C. 2005. Critical Review: Nanoparticles and the Environment. *Air & Waste Management Association.* 55: 1411-1417

Wahlsten D., Colbourne F. and **Pleus R.C.** 2003. A robust, efficient and flexible method for staining myelinated axons in blocks of brain tissue. *Journal of Neuroscience Methods* 123: 207-214.

Greer M.A., Goodman G., **Pleus R.C.** and Greer S.E. 2002. Health Effects Assessment for Environmental Perchlorate Contamination: The Dose Response for Inhibition of Thyroidal Radioiodine Uptake in Humans. *Environmental Health Perspectives.* 110: 927-937.

Pleus R.C., Goodman G. and Mattie D.R. 2000. Development of a Reference Dose for Perchlorate: Current Issues and Status. *CIPA Publication:* 698.

Greer M.A., Goodman G., **Pleus R.C.**, and Greer S.E. 2000. Does environmental perchlorate exposure alter human thyroid function? Determination of the dose-response for inhibition of radioiodine uptake. Abstract. Endocrine Journal 47 : 148.

Bylund D.B. and **Pleus R.C.** 2000. Alpha-2 adrenergic receptor binding in human pineal gland. *Pharmacology Reviews and Communications* 11: 1-10.

Shiue C., **Pleus R.C.**, Shiue G., Rysavy J.A., Sunderland J.L., Cornish K.G., Young S.D. and Bylund D.B. 1998. Synthesis and Biological Evaluation of [^{11}C]MK-912 As an Alpha-2 Adrenergic Receptor Radioligand for PET Studies. *Nuclear Medicine and Biology* 25: 127-133.

Pleus R.C., Dunn L. and Rogers D.E.C. 1998. Comparison of the use of risk assessment for human health and ecological assessments in developed and developing countries. In *Papers of 11th World Clean Air and Environmental Congress, Volume 2.* Durban, South Africa, National

ED_001802_00024760-00098

Association for Clean Air: 6D-5.

Pleus R.C. and Kelly K.E. 1998. Heath Effects from Hazardous Waste Incineration Facilities: Five Case Studies. *Advances in Modern Environmental Toxicology* 25: 179-192.

Shirai J., **Pleus R.C.** and Perry M. 1997. Chemical Characteristics of Cement Kiln Dust and their Effect on Dioxin-Related Health Risks. In *Waste Combustion in Boilers and Industrial Furnaces.* Pittsburgh, PA; Air & Waste Management Association: 193-205.

Pleus R.C., Shiue C.Y., Shiue G.G., Rysavy J.A., Huang H., Sunderland J.J. and Bylund D.B. 1993. Synthesis and biodistribution of the α_2-adrenergic receptor antagonist (^{11}C)WY26703: Use as a radioligand for Positron Emission Tomography. *Receptor* 2: 241-252.

Pleus, R.C., Suder D.R., and C.E. Schmidt. 1993. Methodology for assessing the health impact of gaseous emissions from a pulp mill. Paper presented at the 86th Annual Meeting of the Air and Waste Management Association, Denver, Colorado, 13-18 June. Paper 93-TA-36A.05.

Pleus R.C. and Pascoe G.A. 1993. Assessing health risks from inhalation and oral exposure to chloroform in water. Abstract. *FASEB Federation Proceedings.* 7: 3323.

Shiue C.Y., Shiue G.G., Rysavy J.A., **Pleus R.C.**, Huang H., Bai L.Q., Cornish K.C., Sunderland J.J. and M.P. Frick. 1993. Fluorine-18 and carbon-11 labeled amphetamine analogs—Synthesis, distribution, binding characteristics in mice and rats and a PET study in monkey. *Nuclear Medicine and Biology* 20: 973-981.

Shiue C.Y., Bai L.Q., Shiue G.G., Rysavy J.A., **Pleus R.C.**, Hui H., Frick M.P. and Catt J.D. 1993. Synthesis of (±)-[^{18}F]BMY 14802, its enantiomers and their anatomical distributions in rodents. *Nuclear Medicine and Biology* 20: 625-630.

Pleus R.C., Shreve P.E., Toews M.L. and Bylund D.B. 1993. Down-regulation of alpha-2 adrenoceptor subtypes. *European Journal of Pharmacology* 244: 181-185.

Shiue C.Y., Shiue G.G., **Pleus R.C.**, Rysavy J.A., Huang H., Frick M.P. and Menolascino F.J. 1992. A comparison of the utility of N-[^{11}C]methylketanserin and N-[^{11}C]methylaltanserin for mapping serotonin receptors *in vivo*. *Journal of Nuclear Medicine* 33: 1026.

Pleus R.C., Shiue C.Y., Shiue J., Rysavy J., Huang H., Sunderland J., Cornish K., Bai L., Frick M. and Bylund D.B. 1992. [^{11}C]MK-912 and [^{11}C]WY26703 as positron emitting radioligands to label alpha-2 adrenergic receptors *in vivo*: Assessing their use in Rhesus monkey brain. Abstract. *Abstracts – Society for Neuroscience* 18: 590.

Pleus R.C. and Bylund D.B. 1992. Desensitization and down-regulation of the 5-HT_{1B} receptor in the opossum kidney cell line. *Journal of Pharmacology and Experimental Therapeutics* 261: 271-277.

Gary V., Oatman L., **Pleus R.C.** and Gray D. 1984. Formaldehyde in the home: Some disease perspectives. *Minnesota Medicine* 63: 107-110.

CONTRIBUTIONS TO BOOK CHAPTERS

Pleus R.C. 2012. The State of Science – Human Health, Toxicology, and Nanotechnological Risk. In Shatkin J., *Nanotechnology Health and Environmental Risks.* CRC Press.

Locascio L., Reipa V., Zook J., **Pleus R.C.** 2011. Nanomaterial Toxicity: Emerging Standards and Efforts to Support Standards Development. In Murashov V. and Howard J., Eds.

ED_001802_00024760-00099

Nanotechnology Standards. Springer: 179-208. New York, NY.

Belzer R.B., Bruce G.M., Peterson M.K. and **Pleus R.C.** 2004. Using Comparative Exposure Analysis to Validate Low-Dose Human Health Risk Assessment: The Case of Perchlorate. In Linkov, I. and Ramadan, A., Eds. *Comparative Risk Assessment and Environmental Decision Making.* Kluwer: 57-74. Norwell, MA.

Borak J.B. and **Pleus R.C.** 2003. Toxicology (Chapter 39). In McCunney R.J., Eds. *A Practical Approach to Occupational and Environmental Medicine, 3rd Edition.* Lippincott Williams & Wilkins Publishers: 554-570. Philadelphia, PA.

Pleus R.C. 2003. Perchlorate Regulation and Regulatory Activity (Chapter IX). In Schilt A.A. and McBride L.C., *Perchloric Acid and Perchlorate, 2nd Edition.* GFS Chemicals. Powell, OH.

Pleus R.C., Ammann H.M., Miller R.V., and Robles H. 2003. Toxicology (Chapter 4). In Boss M. and Day D., Eds. *Biological Risk Engineering Handbook: Infection Control and Decontamination.* Lewis Publishers: 97-110. Boca Raton, FL.

Ammann H.M., Miller R.V. Robles H., and **Pleus R.C.** 2003. Risk Assessment (Chapter 5). In Boss M. and Day D., Eds. *Biological Risk Engineering Handbook: Infection Control and Decontamination.* Lewis Publishers: 111-134. Boca Raton, FL.

OTHER PROFESSIONAL PUBLICATIONS

Pleus R.C. 2003. Perchlorate: Using Good Science to Derive a Safe Drinking Water Level. *Water Conditioning & Purification Magazine.* August: 36-40.

Pleus R.C. 2000. Riding the Rocket – How a Perchlorate Standard May Affect Those for Chlorine Dioxide and Its DBPs. *Water Conditioning & Purification Magazine.* September: 104-107.

Pleus R.C. 2000. Strategies to Manage POPs and PBTs in the Cement Industry. *Cement Americas.* May/June: 38-40.

Pleus R.C. and Kelly K.E. 1993. Health Effects of Hazardous Waste Incineration Facilities: Five Case Studies. *US Department of Health and Human Services. ATSDR* Hazardous Waste and Public Health: International Congress on the Health Effects of Hazardous Waste:815-825.

Pleus R.C. 1991. Book review of Jonsen, A.: The new medicine and the old ethics. Science Books and Films 27: 71.

Pleus, R.C. 1988. Book review of Thackray, S: Looking at pollution. *Science Books and Films* 24,1: 21.

Pleus, R.C. 1988. Biology textbook review of Wallace R. A., Biology; The world of life, 4th edition. *Science Books and Films* 23, 4: 217-218.

Pleus R.C. 1985. Biology textbook review of Bauer et al. Experience in biology. 2nd edition. *Science Books and Films* 29.

Pleus R.C. 1985. Chemical composition of cosmetic products. Course notes. Minnesota Cosmetology Workshop, Minneapolis, MN.

Curriculum vitae last updated March 1, 2013.

ED_001802_00024760-00100