Evaluation of Human Health Risks from Exposure to Arsenic Associated with CCA-Treated Wood

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Table of Contents

List of Tables

List of Acronyms

ACS	American Cancer Society
Arch	Arch Wood Protection, Inc.
AF	Soil Adherence Factor
AT	Averaging Time
ATSDR	Agency for Toxic Substances and Disease Registry
AWPA	American Wood Preservers Association
AWPI	American Wood Preservers Institute
BTM	Best Tracer Method
BW	Body Weight
C_{DA}	concentration of dislodgeable arsenic on the hands
CAA	Clean Air Act
CADHS	California Department of Health Services
CCA	Chromated Copper Arsenate
CIS	Consumer Information Sheets
COCs	Constituents of Concern
CPSC	United States Consumer Product Safety Commission
$CrAsO_4$	Chromium Arsenate
CSF	Cancer Slope Factor
CSF_{dermal}	Dermal Cancer Slope Factor
CSF_{oral}	Oral Cancer Slope Factor
CTDEP	Connecticut Department of Environmental Protection
CTE	Central Tendency Exposure
CUG	Cleanup Goal
CWPA	Canadian Wood Preservers Association
DA	Dermal Absorption Fraction
DSMA	disodium methanearsenate
ED	Exposure Duration
EF	Exposure Frequency
EPC	Exposure Point Concentration
ET	Exposure Time
EWG	Environmental Working Group
FDEP	Florida Department of Environmental Protection
FS	Fraction of Source
GI	gastrointestinal
Gradient	Gradient Corporation
HHRA	Human Health Risk Assessment
HS&WMR	Hazardous Substance & Waste Management Research, Inc.
HTE	Hand Transfer Efficiency
IEUBK	Integrated Exposure Uptake Biokinetic Model
IRIS	Integrated Risk Information System
LOAEL	Lowest Observed Adverse Effect-Level
MADEP	Massachusetts Department of Environmental Protection
MCL	Maximum Contaminant Level
MDEQ	Montana Department of Environmental Quality
MDL	Method Detection Limit
MEDHS	Maine Department of Human Services
MIDEQ	Michigan Department of Environmental Quality
MRL	Minimal Risk Level

MSMA	monosodium methanarsonate
MTCA Stat	MTCA Stat Site Module-Version 2.1
n	sample size
NCP	National Contingency Plan
ND	non-detect
NEPI	National Environmental Policy Institute
NIST	National Institute of Standards and Technology
NJDEP	New Jersey Department of Environmental Protection
NMED	New Mexico Environment Department
NOAEL	No Observed-Adverse Effect Level
NRC	National Research Council
ODEQ	Oklahoma Department of Environmental Quality
OSHA	Occupational Safety and Health Administration's
Osmose	Osmose, Inc.
OSWER	USEPA Office of Solid Waste and Emergency Response
PADEP	Pennsylvania Department of Environmental Protection
PEL	Permissible Exposure Limit
PM_{10}	Particulate Emissions (less than 10 μm in diameter)
PRA	Preliminary Risk Assessment
RAGS	Risk Assessment Guidance for Superfund
RBA	Relative Bioavailability Absorption
RED	Registration Eligibility Decision
Region 8	USEPA Region 8 Office
RfC	Reference Concentration
RfD	Reference Dose
RfD_{dermal}	Dermal Reference Dose
RfD_{oral}	Oral Reference Dose
RISI	Resource Information Systems, Inc.
RL	Reporting Limit
RME	Reasonable Maximum Exposure
ROD	Record of Decision
RSD	Relative Standard Deviation
SA	Skin Surface Area
SCS	Scientific Certification Systems
SDWA	Safe Drinking Water Act
SEM	Scanning Electron Microscopy
Subchronic RfD_{oral}	Subchronic Oral Reference Dose
SP	Southern Pine
TEQs	Toxicity Equivalents
TNRCC	Texas Natural Resource Conservation Commission
95% UCLM	95% Upper Confidence Limit on the Mean
UR_{inhal}	Inhalation Unit Risk
USDA	United States Department of Agriculture
USEPA	United States Environmental Protection Agency
WA Ecology	Washington State Department of Ecology
WEM	Wind Erosion Model
WVDEP	West Virginia Department of Environmental Protection

List of Units

cm^2	squared centimeter
cm^2/day	squared centimeter per day
km	kilometer
L/day	liter per day
m^3	cubic meter
meters/sec	meters per second
mg/day	milligram per day
mg/kg	milligram per kilogram
mg/kg-day	milligram of chemical per kilogram of human body weight a day
mg/L	milligram per liter
mg/m^3	milligram per cubic meter
mL	milliliter
mm	millimeter
ppb	parts per billion
ppm	parts per million
µg	microgram
$\mu g/cm^2$	microgram per squared centimeter
µg/g	microgram per gram
µg/kg	microgram per kilogram
µg/L	microgram per liter
$\mu g/m^3$	microgram per cubic meter
µm	micrometers
yr	year

Executive Summary

Gradient Corporation (Gradient) prepared a human health risk assessment (HHRA) for Arch Wood Protection, Inc., and Osmose, Inc., to quantify potential health risks from exposure to arsenic associated with chromated copper arsenate (CCA) treated wood. Using conservative assumptions and parameters to evaluate exposures, the results of the HHRA indicate that use of CCA-treated wood in both a residential and playground setting does not pose a significant health risk to children or adults.

The HHRA is conducted in accordance with current United States Environmental Protection Agency (USEPA) risk assessment guidance and recent scientific literature. Central tendency exposure (CTE) or average, and reasonable maximum exposure (RME) parameters are used to quantify exposures for a residential and a playground exposure scenario. The residential scenario includes a male/female child ages 2-6 years, and a male/female child and adult ages 2-31 years. The playground scenario includes a male/female child ages 2-6 and 7-12 years. Both exposure scenarios evaluate incidental ingestion and dermal contact with "dislodgeable arsenic,"[1] which is arsenic on the surface of CCA-treated wood that can be removed from the wood surface by dermal contact with the hands. Both scenarios also evaluate incidental ingestion, dermal contact, and inhalation exposure to arsenic-impacted soil located below a CCA-treated structure at a residence or at a public playground. It is assumed in the HHRA that most of the arsenic in these soils is the result of CCA that has migrated (*via* rainwater run-off) from the treated wood to the soil below.

A number of conservative assumptions are used to evaluate exposure and risk for both the CTE and RME scenarios, including the assumption that the amount of dislodgeable arsenic does not decrease with the age of the treated structure, and that all of the time outdoors at either a residence or a playground is spent exposed to both dislodgeable and soil arsenic, simultaneously. These assumptions are likely to result in an overestimate of exposure, and consequently, risk.

The estimated cancer and non-cancer health risks are summarized below in Tables ES-1 through ES-4. Cancer risks for the residential and playground scenarios are summarized in Tables ES-1 and ES-2, respectively. Non-cancer risks for the residential and playground scenarios are summarized in Tables ES-3 and ES-4, respectively.

[1] It should be noted that arsenic in the dislodgeable form is present as chromium arsenate (Bull, 2000, 2001; and Cooper *et al*., 1993; Kamdem, 2001) and is only a small fraction of the dislodgeable material on treated wood (Cui, 2001; Osmose, 2001).

Tables ES-1 and ES-2
Summary of Cancer Risks

Table ES-1
Residential Scenario

Exposure Medium	Exposure Pathway	Estimated Lifetime Cancer Risk			
		Central Tendency Exposure		Reasonable Maximum Exposure	
		Ages 2-6	Ages 2-31	Ages 2-6	Ages 2-31
Soil Arsenic	Ingestion, dermal, and inhalation	1.7×10^{-7}	3.9×10^{-7}	8.2×10^{-7}	1.4×10^{-6}
Dislodgeable Arsenic from Southern Pine*	Ingestion and dermal	9.6×10^{-7}	2.3×10^{-6}	3.0×10^{-6}	5.2×10^{-6}
Dislodgeable Arsenic from Southern Pine w/ Pressure-Applied Water Repellent**	Ingestion and dermal	2.0×10^{-6}	5.0×10^{-6}	6.3×10^{-6}	1.1×10^{-5}

Table ES-2
Playground Scenario

Exposure Medium	Exposure Pathway	Estimated Lifetime Cancer Risk			
		Central Tendency Exposure		Reasonable Maximum Exposure	
		Ages 2-6	Ages 7-12	Ages 2-6	Ages 7-12
Soil Arsenic	Ingestion, dermal, and inhalation	2.5×10^{-8}	1.4×10^{-8}	1.3×10^{-7}	6.0×10^{-8}
Dislodgeable Arsenic from Southern Pine*	Ingestion and dermal	5.4×10^{-7}	4.8×10^{-7}	1.5×10^{-6}	1.3×10^{-6}
Dislodgeable Arsenic from Southern Pine w/ Pressure-Applied Water Repellent**	Ingestion and dermal	1.2×10^{-6}	1.0×10^{-6}	3.3×10^{-6}	2.7×10^{-6}

** Most commonly used type of treated wood in the U.S. (AWPA, 1998).*

*** Treated wood type resulting in greatest risk. This wood type accounts for only about 6% of the treated lumber sold in the U.S. (RISI, 1990).*

Tables ES-3 and ES-4
Summary of Non-Cancer Risks

Table ES-3
Residential Scenario

Exposure Medium	Exposure Pathway	Hazard Quotient			
		Central Tendency Exposure		Reasonable Maximum Exposure	
		Ages 2-6	Ages 2-31	Ages 2-6	Ages 2-31
Soil Arsenic	Ingestion and dermal	9.3×10^{-5}	1.6×10^{-3}	4.9×10^{-4}	6.2×10^{-3}
Dislodgeable Arsenic from Southern Pine*	Ingestion and dermal	6.0×10^{-4}	1.2×10^{-2}	1.9×10^{-3}	2.7×10^{-2}
Dislodgeable Arsenic from Southern Pine w/ Water Repellent**	Ingestion and dermal	1.3×10^{-3}	2.6×10^{-2}	3.9×10^{-3}	5.8×10^{-2}

Table ES-4
Playground Scenario

Exposure Medium	Exposure Pathway	Hazard Quotient			
		Central Tendency Exposure		Reasonable Maximum Exposure	
		Ages 2-6	Ages 7-12	Ages 2-6	Ages 7-12
Soil Arsenic	Ingestion and dermal	1.5×10^{-5}	6.9×10^{-6}	8.0×10^{-5}	3.0×10^{-5}
Dislodgeable Arsenic from Southern Pine*	Ingestion and dermal	3.4×10^{-4}	2.5×10^{-4}	9.6×10^{-4}	6.6×10^{-4}
Dislodgeable Arsenic from Southern Pine w/ Water Repellent**	Ingestion and dermal	7.2×10^{-4}	5.3×10^{-4}	2.0×10^{-3}	1.4×10^{-3}

** Most commonly used type of treated wood in the U.S. (AWPA, 1998).*
*** Treated wood type resulting in greatest risk. This wood type accounts for only about 6% of the treated lumber sold in the U.S. (RISI, 1990).*

The cancer risk estimates for both exposure scenarios, based on either central tendency or RME parameters, are within the USEPA's acceptable cancer risk range of 1×10^{-6} to 1×10^{-4}; and the non-cancer risk estimates for both exposure scenarios are below the USEPA's acceptable non-cancer risk limit of 1.0. Based on the results of the HHRA, average and high-end exposures to arsenic associated with CCA-treated wood in both a residential and playground setting does not pose a significant health risk to children or adults.

It should be noted that the toxicity of arsenic, and in particular its carcinogenicity, remain an evolving area of scientific discussion. Three major risk assessments for arsenic in drinking water have been published in the last two years. We are unable to incorporate the information in these assessments into this document for several reasons, including the lack of a revised cancer slope factor in these assessments, presentation of a range of risk estimates for arsenic in drinking water rather than single point estimates, and because there continues to be scientific debate regarding the best approach for the evaluation. Still, it is of interest to observe that all of the exposure estimates (and associated risk estimates) in this report for CCA-treated wood are below the exposure and risk estimates associated with the proposed drinking water standard for arsenic and the levels of naturally occurring arsenic in food.

1 Introduction

Gradient Corporation (Gradient) prepared a human health risk assessment (HHRA) for Arch Wood Protection, Inc. (Arch), and Osmose, Inc. (Osmose), to quantify potential health risks from exposure to arsenic associated with chromated copper arsenate (CCA) treated wood. Exposure to arsenic on the surface of CCA-treated wood, and exposure to arsenic in soil near a CCA-treated structure is evaluated. Only arsenic, in the CCA complex of metals, is evaluated in the HHRA since most of the potential health risks associated with CCA-treated wood are from exposure to arsenic.

Gradient submitted a "focused" HHRA to the United States Environmental Protection Agency's (USEPA's) Office of Pesticide Programs on July 6, 2001. The focused HHRA was a streamlined assessment of risks from exposures to arsenic associated with CCA-treated wood in a residential setting. The evaluation included only those exposure routes that contributed significantly to risk (*i.e.*, ingestion and dermal absorption), used reasonable maximum exposure (RME) parameters to estimate exposures, and contained a limited assessment of arsenic toxicity. A number of additional analyses have been performed in this more comprehensive HHRA and are summarized below.

Additional Analyses in the Comprehensive HHRA

- A discussion of background soil arsenic concentrations in the U.S., acceptable regulatory levels of soil arsenic, and arsenic concentrations in fertilizers is included to provide some perspective on the reported concentrations of arsenic in soil near CCA-treated wooden structures.
- Residential and playground exposure scenarios are evaluated.
- A broader set of exposure routes are evaluated, including incidental ingestion and dermal contact with dislodegable arsenic; and incidental ingestion, dermal contact, and inhalation exposure to arsenic in soil.
- Both central tendency and RME parameters are used to quantify exposures.
- A comparison of exposure assumptions and parameters, and risk estimates in this HHRA to other relevant risk assessments is discussed.
- A number of arsenic toxicity issues are considered, including children's relative sensitivity to arsenic, arsenic's purported role in endocrine disruption, and a subchronic Reference Dose.

- More detailed discussion of epidemiological and animal studies involving exposures to CCA-treated wood is provided.

- A sensitivity analysis is performed to identify key sources of uncertainty in the assessment of ingestion exposure to CCA-treated wood.

The HHRA is conducted in accordance with current USEPA risk assessment guidance (USEPA, 1989; 1992a; 1992b; 1999a; 1999b) and recent scientific literature. Cancer and non-cancer health risks are assessed for both a residential and a playground exposure scenario. The residential scenario includes a male/female child ages 2-6 years, and a male/female child and adult ages 2-31 years. The playground scenario includes a male/female child ages 2-6 and 7-12 years. Receptors in both exposure scenarios are evaluated for incidental ingestion and dermal contact with dislodgeable arsenic, which is a chemical compound of arsenic (chromium arsenate) on the surface of CCA-treated wood that can be removed from the wood surface by dermal contact with the hands. (It should be noted that arsenic is a small fraction (*i.e.*, 0.2%) of the dislodgeable material on the surface of treated wood (Cui, 2001; Osmose, 2001)). Residential and playground receptors are also evaluated for incidental ingestion, dermal contact, and inhalation exposure to arsenic in soil (also most likely chromium arsenate). It is assumed in the HHRA that most of the arsenic in these soils is the result of CCA that has migrated (*via* rainwater run-off) from a treated wood structure to underlying and adjacent soils.

2 Use, History, and Chemistry of CCA-Treated Wood

This section is organized as follows: Section 2.1 contains a description of what CCA-treated wood is, what it is used for, and a brief regulatory history. A description of the CCA-treatment process, and the chemistry of the CCA formulation is summarized in Section 2.2. Section 2.3 contains a summary of CCA's wood-preserving properties.

2.1 CCA-Treated Wood: What it is Used for and How its Been Regulated

2.1.1 Uses of CCA Treated Wood

Chromated copper arsenate (CCA) is a chemical mixture consisting of three metal ions: chromium, copper, and arsenic, that has been registered with the USEPA for wood preservative use (USEPA, 2000a). CCA protects wood from dry rot, fungi, termites, and other pests that can threaten the integrity of wood products. Wood treated with CCA is used primarily in outdoor structures, such as decks, walkways, fences, boat docks, sign and utility posts, and retaining walls. Wood treated with CCA lasts at least five times longer than untreated wood used for such purposes, and therefore, untreated wood must be replaced more frequently, which can be expensive and resource intensive.

2.1.2 Regulation of CCA Treated Wood

In October 1978, the USEPA initiated a special review of CCA, and two other major wood preservatives, creosote and pentachlorophenol (USEPA, 2000a). Following conclusion of the special review in January, 1986 (51 FR 1334 January 10, 1986), the Agency required new protective measures for workers who use CCA to treat wood, and instituted a voluntary consumer awareness program that required wood pressure-treaters to provide Consumer Information Sheets (CIS) to all lumber yards and other appropriate retailers. The CIS were designed to give consumers safe handling instructions, *e.g.*, to use protective gloves, coveralls and dust masks when sawing treated wood products.

To reduce exposures and potential health risks to workers, the USEPA enacted a number of safety precautions, including classifying CCA as a "Restricted Use" pesticide. This classification requires that only certified applicators (or persons under their direct supervision) can purchase and use CCA, and that the use must be covered by the applicator's certification. Manufacturers must use a closed system (*i.e.*, a

sealed container) for mixing powdered CCA formulations, workers that treat wood with arsenic compounds are required to wear protective clothing, and a respirator must be used if the level of ambient arsenic is unknown or exceeds the Occupational Safety and Health Administration's (OSHA's) Permissible Exposure Limit (PEL) of 10 $\mu g/m^3$, averaged over an 8-hour work day (USEPA, 2000a). Applicators may not eat, drink, or use tobacco products during the application process, and applicators must wash thoroughly after skin contact with the CCA formulation.

More recently, the USEPA has met with representatives of the wood preservative industry, registrants, major retailers, and public interest groups, and together has developed a number of ways to effectively disseminate consumer information regarding the safe and proper use of CCA-treated wood. As a result of these meetings, a number of consumer information initiatives have been implemented, including revised safety information labels attached to treated wood sold in the U.S., a toll-free safety information phone number, and a website address.

The USEPA is also reviewing CCA as part of the Agency's on-going re-registration program (USEPA, 2000a). All pesticides first registered before November 1984 must undergo re-registration review to ensure that the data supporting their use meet current safety standards. As part of the re-registration process for CCA-treated wood, the USEPA is requiring registrants to submit additional data, including exposure data on wood treatment plant employees, in order to better assess both human and environmental risks. The USEPA will adhere to its public participation process during CCA's re-registration review and will provide ample time for public comment on the Agency's preliminary risk assessment (PRA) regarding the use of CCA as a wood preservative. Once the USEPA has completed its re-registration review process for CCA-treated wood, a re-registration eligibility decision (RED) will be issued and will describe the Agency's findings and its conclusions regarding the continued uses of CCA.

2.2 CCA-Treatment Process

During the chemical treatment process, untreated wood is loaded onto trams that are pushed into a large chemical treatment cylinder (USDA, 1980). The cylinder door is closed and a vacuum is applied to the system in order to remove air from the cylinder and the wood (USDA, 1980). Dilute CCA treating solution (*i.e.*, 0.8-6%) (USDA, 1980) is then pumped into the cylinder while the vacuum pressure is still being applied. The cylinder is then pressurized and the CCA solution impregnates the wood. Treatment time is dependant upon the species of wood, the type of wood (*i.e.*, lumber, plywood, poles, planks, *etc.*), and the desired chemical retention (USDA, 1980). After the treatment cycle is completed, solution not

absorbed by the wood is pumped out of the cylinder and stored for reuse. The major species of wood that is treated is Southern Pine, but there are other western softwoods and eastern hardwoods that are also treated (USDA, 1980).

During and even after the treatment process, a series of chemical reactions termed "fixation" occur. Fixation is the process by which chromium, copper and arsenic oxides in the treatment formulation become complexed in the wood so that they are rendered insoluble. The metal ions generated during the reactions precipitate and combine until eventually copper becomes bonded to the wood and copper chromium arsenate and chromium arsenates are formed (Bull, 2000, 2001; and Bull *et al*., 2000). The chromium arsenate ($CrAsO_4$) that is formed during the fixation process is quite stable and exists as a polymeric solid in the wood (Bull, 2000; Kamdem and Cui, 2001). Arsenic is in the +5 oxidation state, which is generally less toxic and less mobile than arsenic in the +3 oxidation state (ATSDR, 2000; Masscheleyn *et al*., 1991 as cited in Townsend *et al*., 2001).

There are three types of CCA formulations, Types A, B, and C, which vary based on the proportions of the chemical components. Type A is not used frequently, and Type B is confined to use in field and remedial treatments (Lebow, 1996). Type C is the formulation predominantly used by the U.S. wood treatment industry because the treated wood can be effectively used for a number of different purposes and Type C is a stable chemical mixture. The Type C formulation contains chromium (as chromium oxide) at 47.5%, copper (as copper oxide) at 18.5%, and arsenic (as arsenic oxide) at 34% (USDA, 1980). The American Wood Preserver's Association formulation standards for Type C specify that chromium oxide be used in concentrations ranging 44.5 to 50.5%, copper oxide ranging 17 to 21%, and arsenic oxide ranging 30 to 38% (Lebow, 1996).

2.3 CCA's Wood-Preserving Properties

CCA significantly prolongs the life of outdoor wood products. Treated wood can last up to five times longer than untreated wood (USEPA, 2000a). This is because the chemical treatment protects the wood from fungi, insects, and other causes of rot and decay by rendering the wood an unattractive food source to fungi and insects. The three components of CCA: copper, chromium, and arsenic - are integral in the efficacy of the preservative. Chromium's role during these reactions results in insolubilization of the three metals so that they do not migrate from the wood (Lebow, 1996). Copper and arsenic are responsible for CCA's anti-decay properties. This is because copper is a good fungicide, and arsenic is

effective against insects and some copper-resistant fungi (Lebow, 1996). The enhanced durability of CCA treated wood is evidence of the stability of the CCA chemicals in the wood.

3 Exposure Assessment

An exposure assessment is used to identify constituents of concern (COCs), and to estimate the magnitude of human exposure to COCs. Arsenic is the only COC evaluated in the HHRA. Based on a review of the current published literature, the most likely chemical species of arsenic associated with CCA-treated wood, in the dislodgeable form and in soil, is chromium arsenate (see Section 3.1 below). However, the term "arsenic" is used in the HHRA because the toxicity data used to quantify health risks is based on inorganic arsenic.

Arsenic is the only metal in CCA-treated wood that is evaluated in the HHRA because most of the potential subchronic and chronic health risks associated with treated wood are from exposures to arsenic. Based on the toxicity of the other two metals in CCA-treated wood, *i.e.*, copper and trivalent chromium, both of which are non-carcinogens according to the USEPA, their inclusion in the HHRA would not significantly affect estimated non-cancer risks (USEPA, 2001a). It should be noted that confirmation of the chemical species of chromium in CCA is based on the CCA treatment process itself. Hexavalent chromium used in the CCA formulation is reduced to trivalent chromium when the complex of metals is "fixed" to the wood (Bull, 2000, 2001). Indeed, the reduction of hexavalent chromium to trivalent chromium is essential for CCA to properly bind to the wood. Therefore, the species of chromium in dislodgeable material is most likely trivalent chromium. Furthermore, to the extent that minute quantities of non-reduced hexavalent chromium in dislodgeable material on the surface of treated wood or in soil exists, hexavalent chromium has been shown to be non-carcinogenic *via* the oral route of exposure in a report prepared by the Office of Environmental Health Hazard Assessment of the California Environmental Protection Agency (CAEPA, 2001). Therefore, potential incidental ingestion of hexavalent chromium is unlikely to pose a cancer risk. And potential exposures to hexavalent chromium *via* dermal contact and inhalation are not likely to pose a significant risk because metals in general are not absorbed efficiently across the skin (USEPA, 1999a), and based on an evaluation of the ambient concentration of wind-blown soil particulate in both the residential and playground exposure scenarios (see Section 3.3.1.5), the magnitude of inhalation exposure is negligible.

In addition to the species of arsenic associated with CCA-treated wood, this section contains a description of the exposure scenarios, potential receptors, exposure media, and exposure pathways evaluated in the HHRA. Also described in this section is the calculation of exposure point concentrations

(EPCs) and chemical intake for each complete exposure pathway, including the equations and exposure parameters used.

3.1 Form of Arsenic in CCA-Treated Wood

The form of arsenic in CCA treated wood has been a subject of research for decades, yet the precise nature of the fixing process and the resultant metal complexes has remained largely unclear until recently. The difficulty arose because of the complexity of the wood matrix, the limited analytical tools available, and the focus on operational characteristics of the wood. As a result, there had been much speculation on the nature of the metal complexes (Dahlgren and Hartford, 1972 as cited in Bull, 2001; Pizzi, 1982; Cooper *et al*., 1993), but no unequivocal understanding of the nature of arsenic in treated wood. Despite decades of research, it was recently stated in a journal article that "This [article] constitutes the first substantive analysis of CCA's fixation products …" (Bull *et al*., 2000). Based on this recent analysis, it can be concluded that the predominant species of arsenic in wood is chromium arsenate (Bull, 2000; 2001; and Cooper *et al*., 1993). This conclusion is further supported by unpublished results from a scanning electron microscopy (SEM) study performed by Dr. Pascal Kamdem at Michigan State University, in which arsenic on the surface of CCA-treated wood appears to be in the form of amorphous arsenic oxide (Kamdem, 2001). As such, arsenic is in its +5 oxidation state, which is a less soluble and less mobile form than arsenite (As^{+3}) (ATSDR, 2000; Masscheleyn *et al*., 1991 as cited in Townsend, *et al*., 2001). In fact, arsenite (As^{+3}) was looked for and not found in properly treated wood (Nygren and Nilsson, 1993).

Based on the information presented in the preceding paragraph, the predominant chemical species of dislodgeable arsenic is most likely chromium arsenate. Because it is assumed in the HHRA that most of the arsenic in soils located below and adjacent to CCA-treated structures is the result of dislodgeable material that has migrated (*via* rainwater run-off) from the treated wood to the soil, the predominant chemical species of arsenic in soil may also be chromium arsenate.

3.2 Complete Exposure Pathways

For exposure and potential risks to occur, a complete exposure pathway must exist. A complete pathway requires the following elements (USEPA, 1989):

- A source and mechanism for release of constituents,
- A transport or retention medium,
- A point of potential human contact (exposure point) with the affected medium, and
- An exposure route at the exposure point.

If any one of these elements is missing, the pathway is not considered complete. For example, if human activity patterns and/or the location of potentially exposed individuals relative to the location of an affected exposure medium prevent human contact, then that exposure pathway is not complete.

The complete exposure pathways evaluated in the HHRA are based on the source, potential release mechanisms, likely exposure media, potential receptors, and possible intake mechanisms of arsenic associated with CCA-treated wood. Table 3-1 (below) contains a summary of the complete exposure pathways identified in the HHRA. Based on these complete exposure pathways, the exposure routes evaluated in the HHRA include incidental ingestion and dermal contact with dislodgeable arsenic, and incidental ingestion, dermal contact, and inhalation of arsenic in soil.

Table 3-1
Complete Exposure Pathways

Contaminant Source	Release Mechanism	Exposure Route	Type of Exposure Evaluated
CCA-Treated Wood	Dermal Contact	Ingestion Dermal Absorption	Subchronic and Chronic
CCA-Impacted Soil	Dermal Contact	Ingestion Dermal Absorption	Subchronic and Chronic
	Wind Erosion	Inhalation	

3.2.1 Exposure Scenarios and Potential Receptors

Two separate exposure scenarios are evaluated in the HHRA: residential and playground. Both exposure scenarios evaluate incidental ingestion and dermal contact with dislodgeable arsenic, and incidental ingestion, dermal contact, and inhalation of soil arsenic. It is assumed in the residential scenario that a CCA-treated deck and/or play structure is the source of dislodgeable arsenic, and most of the arsenic in soil (arsenic is naturally occurring in soil). The assessment of exposures to dislodgeable and soil arsenic is applicable to either structure.

Subchronic and chronic exposures are evaluated in the residential scenario. A male/female child, ages 2-6 years, is used to quantify subchronic exposure for a period of 5 years. A child is used in the subchronic exposure scenario because of the potential for increased exposure as a result of increased hand-to-mouth behavior in children under 6 years of age, and increased dose because of a child's low bodyweight. The combination of increased exposure and dose can result in potentially greater non-cancer risks for a child compared to an adult. A male/female child and adult, ages 2-31 years, is used to quantify chronic exposure for a period of 30 years based on a combination of exposures for the 2-6 and 7-31 year-old receptors.

Only subchronic exposures are evaluated in the playground scenario, which includes two receptors: a male/female child, ages 2-6 and 7-12 years (6 year exposure period). Both receptors are assumed to be exposed to dislodgeable and soil arsenic associated with a CCA-treated play structure at a public playground.

Both central tendency exposure (CTE) and reasonable maximum exposure (RME) parameters are used to quantify exposures *via* the complete exposure routes evaluated in the HHRA. CTE parameters are used so that health risks associated with typical or average exposures to arsenic from CCA-treated wood can be calculated. RME parameters are used so that health risks associated with high-end exposures can be calculated.

3.2.2 Potential Exposure Media

Two exposure media are evaluated in the HHRA, dislodgeable arsenic residue and soil.

3.3 Calculation of Exposure Point Concentrations

In a risk assessment, an exposure point concentration (EPC) represents the average concentration of a chemical that an individual could be exposed to. Calculation of the EPCs used for soil and dislodgeable arsenic are described below.

3.3.1 Soil Arsenic

This refers to arsenic that has been mobilized from CCA-treated structures to nearby soils, usually *via* rainwater. Gradient reviewed five different studies that evaluated the concentration of metals in soil (*i.e.*, arsenic, chromium and copper) that appear to have been mobilized from CCA-treated decks to underlying soils. All but one of the studies (*i.e.*, SCS, 2000) measured the concentration of all three metals; however, since the focus of the HHRA is exposure to arsenic, study results concerning only this metal will be described.

Each of the studies and the results are described below in Section 3.3.1.1. A summary of each of the studies and the results is also available in Table 1.

3.3.1.1 Summary of Studies of Soil Arsenic From Treated Decks

Stilwell and Gorny, 1997

Stilwell and Gorny (1997) collected 85 soil samples from below a total of 7 decks (construction history unknown) located in the state of Connecticut. The age of the decks ranged from 4 months to 15 years. The number of samples collected below each deck ranged from 9 to 16; 4 or 5 control samples were also collected for each deck at a distance of at least 5 meters. Soil samples were collected in a grid-like fashion from the top 5 cm of soil after brushing away any loose debris that may have been present on top of a sample location.

The reported concentration of arsenic ranged from 3 to 350 mg/kg, the mean was 76 mg/kg. The mean of the control samples was 4 mg/kg. There was significant variability in the reported arsenic concentrations from below each deck (mean relative standard deviation or RSD = 51%; RSD for the arsenic controls was 25%). Differences in the amount of treated wood above where soil samples were collected, and differences in rainwater drainage patterns, were given as possible reasons for the variability

in the reported arsenic concentrations. The authors reported a statistically significant higher average arsenic concentration below each deck, compared to the average concentration of the controls for each deck. The authors also reported that the amount of arsenic in soil below a deck appeared to increase with the age of the deck.

Lebow *et al*., 2000a

Lebow *et al* (2000a) conducted a study for the United States Department of Agriculture to assess the migration of metals from a preservative-treated wooden boardwalk constructed in Mt Hood National Forest in the state of Oregon. (Although this study did not evaluate soil arsenic concentrations associated with treated decks, the structure type and study methodology are similar to the other studies summarized in this section). Four different preservative systems were evaluated in the study; however, only the reported results for soil arsenic from the CCA-treated portion of the boardwalk are discussed here.

CCA-boardwalk construction was initiated in May 1996, at which point 16 shallow (*i.e.*, 0-6") pre-construction samples were collected, and completed in June 1996. A total of 110 shallow post construction samples were collected at 4 different locations below and adjacent to the boardwalk at four post-construction time intervals ranging from 0.5 to 11 months. Four background samples were also collected at each post-construction time interval.

Soil arsenic concentrations from below and adjacent to the structure ranged from 1 to 36 mg/kg with a mean of 3.8 mg/kg. The mean background concentration was 1.6 mg/kg. Soil arsenic levels did not exceed background until 5.5 months post-construction and then generally increased with weathering of the structure. Soil arsenic concentrations were highest directly below the drip lines and decreased with increasing distance from the structure. The author concluded that elevated concentrations of arsenic can be detected in soil beneath a CCA-treated structure and that generally low levels of arsenic were detected in the soils despite high rainfall at the test site.

Scientific Certification Systems (SCS), 2000

SCS (2000) conducted a study for Osmose that evaluated the amount of arsenic in soil that could have migrated from commercially treated CCA decks. SCS selected ten commercially constructed pre-fabricated residential decks made of treated wood that were installed by a known contractor in the state of Virginia. The decks were divided into two groups of 5 based on age, "medium aged" decks were

5-10 years old, and "older" decks were 10-15 years old. Soil samples were collected in January 2000 in a grid-like fashion from the top 5 cm after brushing away any loose debris that may have been present on top of a sample location. Soil samples were deliberately collected below drip lines and in areas of high foot traffic (*e.g.*, deck entrances), because it was hypothesized that the potential for migration and deck wear in theses areas would be greatest; thereby, resulting in higher arsenic concentrations in the soil underlying these areas. A total of 84 soil samples (6 to 9 samples/deck) were collected from below the decks. Control samples were collected at least 5 meters away for 7 of the 10 decks.

The reported concentration of arsenic in soil below all of the decks ranged from non-detect (ND) (laboratory method detection limit or MDL = 5 mg/kg) to 85 mg/kg, with mean of 21.2 mg/kg, and a median of 18.3 mg/kg. Based on the results of the control samples, SCS concluded that the background concentration of arsenic in the soils sampled was below MDLs (*i.e.*, <5 mg/kg). SCS concluded that arsenic in CCA-treated wood does appear to migrate to nearby (*e.g.*, underlying and/or adjacent) soils, that weather conditions can accelerate this migration, and that foot traffic does not seem to be a significant factor in the migration of arsenic from CCA-treated wood.

Osmose, 2000

In February of 1993, Osmose (2000) placed untreated and CCA-treated "deck modules" (number unknown) on its Research Division test plot located Gainesville, Florida. Prior to their installation, three replicate baseline soil samples were collected from the top 4 inches of soil after brushing away any loose debris that may have been present on top of a sample location. The decks were not constructed in-place, and construction debris was not generated during installation. Three replicate soil samples were collected below the decks at a location adjacent to where the baseline samples were collected, at 1,2,3,4,5, and 7 years post installation. The post installation samples were collected in the same manner as the baseline sample, except that the samples were collected to a depth of only 2 inches instead of 4 inches.

For the CCA-treated deck, soil arsenic was 3.6 mg/kg at the time of installation; 5.7 mg/kg at 1 year; ND (reporting limit (RL) = 0.01 mg/kg) at 2 years; 8.3 mg/kg at 3 years; 1.8 mg/kg at 4 years; and ND (RL = 0.05 mg/kg) at 5 years and 7 years (RL = 0.03 mg/kg). For the untreated deck, the initial soil arsenic concentration was higher than the treated deck (11.8 mg/kg *vs*. 3.6 mg/kg); however, the reported results for years 1 to 7 were lower (maximum reported value was 3.2 mg/kg at 1 year). The reported soil arsenic levels in this study do not follow a distinct temporal pattern for either the treated or untreated decks.

Townsend *et al*., 2001

Townsend *et al.* (2001) collected 8 to 9 soil samples from the top one-inch of soil in a grid-like fashion from beneath 9 CCA-treated decks ranging from 2 to 19 years in age in different cities in the state of Florida. The samples were collected in November and December 1999 and in June and July 2000. An equal number of control (*i.e.*, background) samples were also collected at least 50 feet from each deck. A total of 65 samples and 65 controls were collected in the study. Information regarding construction history and presence of construction debris was not provided.

Arsenic concentrations in the deck samples ranged from 1.2 mg/kg up to 217 mg/kg with an average of 28.5 mg/kg. The reported averages for the control samples ranged from 1.3 mg/kg to 1.5 mg/kg. Based on the study results, the authors concluded that CCA-treated decks are capable of impacting adjacent soils.

3.3.1.2 EPC for Soil Arsenic From Treated Decks

Based on Gradient's review of the preceding studies, it is apparent that construction debris (*e.g.,* sawdust and wood chips) can be a significant source of arsenic for soils underlying and/or adjacent to CCA-treated structures. This observation may explain some of the difference between the reported results in the three preceding studies where construction debris was known to be absent (*i.e.*, SCS, 2000, Osmose, 2000, and Lebow *et al*., 2000a), and the two studies where information regarding construction history and debris were not available (*i.e.*, Stilwell and Gorny, 1997 and Townsend *et al*., 2001). It is possible that construction debris contributed to the reported soil arsenic concentrations in the Stilwell and Gorny (1997) and Townsend *et al*. (2001) studies, which had higher reported average and maximum concentrations compared to the other three studies. A number of studies (*e.g.*, Lebow *et al*., 2000b; Townsend *et al*., 2001) have evaluated the migration of metals from CCA-treated sawdust *vs.* CCA-treated wooden blocks and observed significantly greater leaching from the sawdust compared to the blocks. These studies indicate that construction debris can be a significant source of arsenic in soil *via* leaching. Furthermore, it is possible that digestion of construction debris in the form of sawdust in the soil samples collected in the Stilwell and Gorny (1997) and Townsend *et al*. (2001) studies contributed to the elevated arsenic concentrations in these studies.

Gradient chose the results from the SCS (2000) study to quantify exposure to soil arsenic in the residential exposure scenario because the construction history of the test decks was known, and construction debris did not appear to be contributing to soil arsenic levels. Although some of the details regarding the Osmose (2000) study are lacking, the results are consistent with the SCS study because they show that when construction debris is known to be absent, soil arsenic concentrations are lower. In addition, the SCS study included 10 different decks of various age, and collected a sufficient number of soil samples to be representative of soil arsenic levels and to enable statistical analysis of the results. Lebow *et al*. (2000a) appears to have been a well-conducted study; construction debris was controlled for, a large number of test samples were collected, and background samples were collected. However, the wood species used in the study was Western Hemlock, which is not a widely used species for CCA treatment, the single structure used in the study was not a deck, and given the relatively short study period (*i.e.*, less than 1 yr), the concentrations of soil arsenic (assuming they were adjusted for pre-construction levels) seemed prematurely elevated compared to the results of the other studies.

Gradient performed a statistical analysis of the reported soil arsenic concentrations in the SCS study. According to USEPA guidance, most soil quality data are lognormally distributed, and the W-test (Gilbert, 1987) is a statistical method that can be used to determine if a dataset is consistent with a normal or lognormal distribution (USEPA, 1992b). The Shapiro-Wilk W test was used to assess whether each of the SCS soil datasets (*i.e.*, data for each deck is considered a set) was better fit by a normal or lognormal distribution. The statistical software program MTCA Stat Site Module-Version 2.1 (MTCA Stat), was used to evaluate the SCS soil data (MTCA Stat contains a number of statistical tests including the W test). Based on a statistical evaluation of the data, some of the datasets are consistent with a normal distribution and others are better fit by a logarithmic transformation of the data (USEPA, 1992b).

Summary statistics including the mean, median and the 95% upper confidence limit on the mean (95% UCLM) were calculated for each dataset, and for grouped data based on the age of the decks (*i.e.*, 5-10 years and 10+ years) (see Table 2 for a summary of the statistical analysis of the SCS soil data). Because there is little difference between the mean soil arsenic concentrations between the two deck age groups (5-10 years = 21.7 mg/kg; 10+ years = 20.5 mg/kg), the overall data set (*i.e.*, all 10 decks in the study, n=84) was used to calculate an EPC of 28.7 mg/kg. The EPC was used to evaluate subchronic and chronic exposures to arsenic in soil and is the 95% UCLM of the overall data set. Based on the results of D'Agostino's test (this test is used instead of the W test by MTCA Stat for a sample size of 50 or more), the overall dataset was determined to be better fit by a logarithmic transformation of the data; therefore, the 95% UCLM is based on a lognormal distribution.

3.3.1.3 Summary of Studies of Soil Arsenic at Playgrounds

Gradient reviewed three soil arsenic studies conducted at playgrounds in the U.S. and in Canada. The purpose of the review was to identify a study or studies that are representative of playground soil arsenic concentrations in the U.S. that could be used to develop a soil arsenic EPC for the playground exposure scenario evaluated in the HHRA. One of the Canadian studies, Doyle (1992), was conducted at a research test site and not a playground. However, the Doyle study was included here because it is cited in the literature as a playground soil arsenic study. Each of the studies are described below and summarized in Table 3.

Riedel *et al*., 1990

Riedel *et al*. (1990) conducted a study at 10 exiting playgrounds with structures ranging in age from 2 to 10 or more years, in eastern Ontario, Canada. A total of 40 sand and soil samples (*i.e.*, 4 samples/playground) from hot-spot or worst-case areas (*i.e.*, under and adjacent to structure) were collected. One control or background sample was also collected at each playground at a distance of 10 meters from a treated structure. Information regarding construction history and the presence of construction debris at the playgrounds was not provided

The reported mean concentration of arsenic for each of the playgrounds ranged from 0.2 to 7 mg/kg with an overall mean of 3 mg/kg. The reported concentration of arsenic in the background samples ranged from 0.1 to 0.4 mg/kg with a mean of 0.2 mg/kg. The authors concluded that outdoor structures constructed of CCA-treated wood release measurable amounts of arsenic and that the results do not indicate a clear correlation between the age of the structure and the amount of arsenic in soil or sand.

Doyle, 1992

Doyle (1992) conducted a study at a research site in Ottawa, Canada using a test structure made of type C CCA-treated wood built specifically for the study. The test structure was comprised of various horizontal, vertical and diagonal surfaces typically found in outdoor play structures. Four coating materials including stain, varnish, paint, and a water repellent sealer were applied to different areas of the test structure. Background levels of arsenic were based on soil and sand samples (number unknown) randomly collected at the test area prior to construction. A total of 40 post-construction sand and soil

samples were collected during September to November 1991 from hot-spot or worst-case areas (*i.e.*, under and adjacent to structure).

Overall sample range was 0.8 to 80 mg/kg with a mean of 19.4 mg/kg. Reported background concentrations in soil and sand were 1 and 0.2 mg/kg, respectively. The concentration of arsenic in both soil and sand samples was highest at the bases of vertical and diagonal support posts. The authors concluded that run-off from the posts tended to concentrate the leached residue at their bases. In general, higher concentrations were found in samples collected near areas of grater wood surface area, and in samples collected near uncoated surfaces. The authors concluded that there were significant accumulations of metals in soil and sand at the bases of the test structure and that application of various coatings on the treated wood surfaces reduced the amount of accumulated metals in these solid media.

Malcolm Pirnie, 2001

Malcolm Pirnie (2001) conducted a study for the American Chemistry Council to evaluate soil quality at public playgrounds in the U.S. that have CCA-treated play structures. The study was conducted at four existing playgrounds in Newport News, Virginia; Virginia Beach, Virginia; Berkeley, California; and Oakland, California. Information regarding construction history and the presence of construction debris was not provided. The age was known for three of the four playground structures and ranged from 5 to 23 years. Sixteen site-wide and twelve hot-spot or worst-case (*e.g.*, downgradient from treated poles) samples were collected at a depth of 0-2 inches at each playground. Four background samples were also collected at a depth of 0-2 inches at each playground from areas believed to have not been impacted by CCA-treated wood. Analyzed sample matrices included soil, sand, and wood chips.

The overall range of arsenic concentrations for the site-wide samples was 0.2 to 64 mg/kg with a mean of 3.7 mg/kg. The overall background concentrations of arsenic ranged from 0.2 to 7 mg/kg with a mean of 1.5 mg/kg. In general, the highest reported concentrations were found in samples collected within one foot of a treated support pole. Except for the Virginia Beach location, the reported average site-wide concentrations of arsenic were comparable to reported average background levels.

Of the three studies described above, only one was conducted in the U.S., the other two were conducted in Canada. It is possible that the Canadian studies may not be representative of soil arsenic levels at U.S. playgrounds. This is because Canada typically uses "refractory" wood species such as hemlocks and firs that are more difficult to impregnate with the CCA formulation. Refractory species are

resistant to the CCA formulation because of their internal cell structure, and therefore, require higher concentrations of the metals in CCA, and are treated for longer time periods to achieve the proper retention of the CCA constituents. However, even with the more concentrated treatment formulation and modified procedure, the CCA is not able to penetrate the wood as deeply as non-refractory species resulting in a greater amount of leaching of the CCA constituents (Cooper, 1990). Support for the increased leaching potential of refractory wood species is provided in a study by Zahora *et al*. (1993) who measured concentrations of copper, chromium and arsenic in run-off samples from CCA-treated piles of Southern Pine (SP), a non-refractory species, and Hemlock, a refractory species. The reported concentrations of arsenic were 3- to 5-fold higher, on average, in the run-off from the treated Hemlock pile compared to the run-off from the treated SP pile, over a period of 1 to 4 days. Also, McNamara (1982) found higher leaching losses in Hem-Fir (a refractory species) compared to Southern Pine blocks subjected to the USEPA's Leachate Toxicity Extraction Procedure (McNamara, 1982 as cited in Cooper, 1990).

In addition to the possible difference in wood species between the Canadian and U.S. studies, both of the Canadian studies collected only hot-spot or worse-case samples from near treated poles and directly under the structure. And lastly, the Doyle (1992) study was not conducted at a playground and did not evaluate leaching from a CCA-treated play structure.

The Malcolm Pirnie (2001) study was selected because it included 4 playgrounds located in geographically distinct areas of the U.S., three different types of ground cover (*i.e.*, soil, sand, and woodchips) were analyzed, site-wide and hot-spot samples were collected, there is a limited degree of variability in the site-wide datasets for 3 of the 4 playgrounds, and enough site-wide samples were collected to enable statistical analysis of the results.

3.3.1.4 EPC for Soil Arsenic at Playgrounds

Gradient conducted an independent analysis of the data from the Malcolm Pirnie (2001) study and calculated average and 95% UCLM concentrations of soil arsenic for each playground and for the entire dataset. Table 3 contains summary statistics for the overall dataset. There was good agreement between the calculated average concentrations of arsenic across all of the playgrounds in the study, except for the Kids Cove playground in Virginia Beach, Virginia. The variability in the Kids Cove results is likely due to the wood chip ground cover at this facility. The wood chip samples were passed through a 2.4 mm sieve prior to laboratory analysis, and were analyzed as original samples (*i.e.*, whole wood chips).

Variability was observed in both the sieved and non-sieved sample sets. The range in the results for the sieved samples is 0.5 to 63.5 mg/kg with a mean of 9.2 mg/kg and a 95% UCLM of 26.6 mg/kg. The range in the results for the non-sieved samples is 0.4 to 51.5 mg/kg with a mean of 6.7 mg/kg and a 95% UCLM of 20.5 mg/kg.

The wood chip ground cover at the Kids Cove facility is the most likely reason for the greater degree of variability in the sieved and non-sieved datasets. However, since wood chips are not an uncommon ground cover at playgrounds, the Kids Cove results are included in the calculation of the EPC for arsenic in soil at playgrounds. The sieved data from the Kids Cove facility are used because they are slightly higher than the non-sieved data, and because this sample matrix more closely approximates soil, and therefore, is consistent with other exposure parameters (*e.g.*, soil ingestion rate, adherence factor, and bioavailability) used in the HHRA to quantify exposures to arsenic in soil. An EPC of 4.1 mg/kg is used to quantify exposures to soil arsenic for the playground scenario. This value is the 95% UCLM (mean 3.7 mg/kg) of the reported site-wide arsenic concentrations for all 4 playgrounds.

3.3.1.5 EPC for Soil Arsenic Particulate

It is assumed that residents could be exposed to wind-blown soil from beneath a CCA-treated deck while outdoors in the yard, and that children at a playground could be exposed to wind-blown soil from beneath and/or adjacent to a play structure while outdoors at playground. A USEPA-recommended wind erosion model (WEM) is used to calculate a wind-blown emission rate for arsenic-impacted respirable soil particulate (*i.e.*, smaller than 10 μm in diameter or PM_{10}).

The WEM is recommended for use in the USEPA's "*Air/Superfund National Technical Guidance Study Series, Volume II - Estimation of Baseline Air Emissions at Superfund Sites*" (USEPA, 1990a), and is described in more detail in the USEPA's "*Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination Sites*" (USEPA, 1985). The calculation technique is based on the empirical relationship that the wind-blown flux of particles is proportional to the wind speed cubed, and is sometimes referred to as the Gillette model.

The WEM is applicable to bare surfaces of finely divided material such as sandy agricultural soil. Such surfaces are considered to have an "unlimited reservoir" of erodable particles. These surfaces have low threshold wind speeds for wind erosion, and particulate emission rates are assumed to be independent of time at a given wind speed. The following assumptions are inherent to the WEM:

- Uniform contamination of a symmetrical land area,
- Continuous and uniform emissions from the entire area,
- The chemical mass fraction in respirable particulate emissions (PM_{10}) is equivalent to the chemical mass fraction in soil (*i.e.*, PM_{10} particles are not "enriched" in chemical mass),
- There is an endless supply of contaminated material at the surface, resulting in the same emission rate over the time period of interest, and
- Solubility, degradation, and evaporation are not taken into account in evaluating particulate matter.

Default assumptions and parameters from the USEPA's "*Soil Screening Guidance: Technical Background Document*" were used in the WEM to estimate a PM_{10} emission rate at a residence and at a playground (USEPA, 1996a). The Soil Screening Guidance Document was considered an appropriate reference for model assumptions and parameters because it provides a methodology to calculate risk-based soil screening levels for a generic residence that the USEPA believes are protective for residential use of a property (USEPA, 1996a). The key WEM input parameters used are listed below:

- 0.5-acre source area (this is the assumed area of a residential lot)
- 50% of the source area is vegetated
- Mean annual wind speed is 4.7 meters/sec (10.6 miles/hour)
- Aggregate soil particle size is 0.5 mm or 500 μm

The estimated emission rate for impacted soil particulate is 1.12×10^{-6} g/m^2-sec. Using the EPC calculated for exposure to soil arsenic below decks (see Section 3.3.1.2), and additional USEPA default parameters, the PM_{10} concentration for soil arsenic is 3.53×10^{-4} μg/m^3 for the residential exposure scenario. This PM_{10} concentration is used to quantify inhalation exposure at a residence. The same soil particulate emission rate used for the residential scenario is also used for the playground scenario because USEPA default parameters for the WEM are not available for a generic playground, and these parameters are considered reasonable for playground exposures. The EPC calculated for playground soils is used in combination with the soil emission rate to calculate a PM_{10} EPC of 5.05×10^{-5} μg/m^3 to quantify inhalation exposure at a playground. Refer to Appendix A for a detailed description of the WEM, model input parameters, and a copy of the model output.

3.3.2 Dislodgeable Arsenic

As mentioned previously, dislodgeable arsenic refers to arsenic that can be removed from the surface of CCA-treated wood by dermal contact, usually with the hands. Gradient reviewed eleven different studies that evaluated the amount of arsenic removed using either the hands of human volunteers or wipes. Removal of arsenic from the surface of CCA-treated wood with hands is referred to as "arsenic hand loading". Three of the eleven arsenic loading studies used human volunteers and the remaining studies used only a wipe sampling methodology. Gradient considers the hand loading methodology, compared to a wipe technique, to provide a more accurate estimate of the amount of dislodgeable arsenic that is typically removed *via* dermal contact during normal human exposure to CCA-treated wood.

Wipe studies tend to overestimate the amount of surface material that can be transferred to the hands, and therefore, are a less reliable (especially in the absence of any adjustment) measure of exposure. Several studies provide evidence of this overestimate, for example SCS (1998) observed that 2- to 6-fold more arsenic was removed using wipe sampling compared to hands. Similar results were observed in pesticide transfer studies where the amount of pesticide residue transferred from indoor surfaces using either a wipe or "drag-sled" sampling method, and a hand sampling method was compared. The mean percentage of pesticide transferred using either a drag-sled or wipe sampling technique was approximately 3- to 10-fold greater, respectively, than when a hand sampling technique was used (Lu and Fenske, 1999; Camann *et al*., 1995; Fenske *et al*., 1990; and Vacarro, 1990 all as reported in USEPA, 1999c).

Because Gradient is using the results of hand loading studies to quantify exposures to dislodgeable arsenic, only these studies are described below in Section 3.3.2.1. A summary of each of the hand and wipe studies, and their results, is available in Table 4.

3.3.2.1 Summary of Hand Loading Studies

California Department of Health Services (CADHS), 1987

The CADHS (1987) conducted an exposure assessment to measure the amount of dislodgeable arsenic complex that is removed upon dermal contact with CCA-treated wood. Little information is available regarding this study; however, it appears that five human volunteers (age unknown) rubbed both

wetted hands on CCA-treated playground wood (number and type of structure not known) for approximately 3 minutes. Dislodgeable arsenic was removed from the volunteer's hands by washing the hands twice in deionized water and collecting the wash water for laboratory analysis (personal communication with Bob Schlag, 1997 as cited in MEDHS, 1998).

The amount of arsenic reported on the hands (assuming both hands) ranged from an average of 236 μg (average of both hands for all volunteers) to a maximum of 1,260 μg. The results in the CADHS study were not used in the HHRA because of a lack of information regarding how the volunteer's made contact with the wood, the number of samples collected, the type of CCA-treated structure tested, and a lack of controls.

Maine Department of Human Services (MEDHS), 1998

The MEDHS (1998) conducted a hand loading study in October and November of 1997 and in March and April of 1998 using a single 3-year old CCA-treated residential deck located in the central portion of the state of Maine. An adult male volunteer gently rubbed (using a single hand, wet and dry) a 150 cm x 9 cm (1,350 cm^2) and 1,400 cm x 9 cm (12,600 cm^2) section of railing for different time periods (range: 3 seconds to 1 minute). Arsenic was removed from the hand for laboratory analysis using a wipe from a lead dust sampling kit. The study also measured the amount of arsenic on the hands of a 20-month old child allowed to play freely on the same CCA-treated deck for a period of less than 10 minutes.

The reported arsenic hand loads for a number of different experiments using the adult volunteer ranged from 6-88 μg/hand using a dry hand, and 37-110 μg/hand using a wet hand. These reported ranges were comparable, especially since it was reported in the study that there appeared to be arsenic contamination in the field blanks for an experiment using wet hands and that the reported hand arsenic concentrations could be overestimated by 5-10 μg. Assuming the adult hand surface area available for contact with a treated wood surface is 267 cm^2, which is approximately 1/3 of the total surface area of both hands as reported in USEPA (1997a), the reported hand arsenic concentrations using a dry hand range from 0.02 to 0.33 μg/cm^2, and 0.12 to 0.39 μg/cm^2 using a wet hand (assuming a 5 μg correction).

The arsenic hand loading for the child ranged from 22-55 μg for both hands. Assuming the hand surface area available for contact with a treated wood surface is 104 cm^2, which is approximately 1/3 of

the total surface area of both hands for a 2 year old child, as reported in USEPA (1997a), the reported hand arsenic concentrations range from 0.21 to 0.53 $\mu g/cm^2$.

Based on the results of one experiment, the authors concluded that a wet hand removes approximately 6 to 15 times more arsenic than a dry hand from the same section of wood; however, overall study results indicate that the average difference between arsenic loading using dry hands *vs.* wet hands is only about 5-fold. The authors also concluded that repeated rubbing (4 times) of the same section of wood did not appear to "deplete" arsenic from the wood surface, there was a slight increase in the amount of hand arsenic with longer rubbing periods, and there was little difference between the amount of arsenic loaded onto the child's hands compared to the adult volunteer.

Scientific Certification Systems (SCS), 1998

SCS (1998) conducted a hand loading (and wipe) study using eight different samples of CCA type C commercially treated wood and one control sample of untreated wood. Eight of the nine wood samples were Southern Pine, a treated Hemlock/Fir wood sample was also tested. Except for the untreated control, all of the wood samples were treated using CCA from Osmose. In addition, one of the wood samples was treated with CCA and a proprietary water repellent manufactured and marketed by Osmose, this sample is referred to as treated Southern Pine with pressure-applied water repellent. Further treatment of some of the wood samples was performed by SCS prior to testing, and included staining, sealing, and cleaning with two different types of brightening agents. Brightening agents are used to remove the gray coloring of wood exposed to outdoor conditions. Two of the wood samples were also aged 5 years. These commercially treated wood samples were aged by Osmose at their weather exposure test area in Griffin, GA. Table 3-2 below contains a summary of the different wood samples evaluated in the study.

Table 3-2

Study Wood Samples
Untreated Southern Pine (control)
Treated Southern Pine
Treated Southern Pine, stained
Treated Southern Pine, sealed
Treated Southern Pine with pressure-applied water repellent
Treated Southern Pine, brightener
Treated Southern Pine, aged 5 years
Treated Southern Pine, aged 5 years, brightener
Treated Hemlock/Fir

Hand sizes (left and right) for five adult male volunteers (ages 18-40 years) with informed consent were measured by tracing the outline of each hand. Each volunteer rubbed each wood sample 10 times with each hand. Each hand was subsequently rinsed 3 times with reagent grade water and the rinsate was collected and analyzed for arsenic, chromium and copper.

The reported arsenic concentrations in the rinsate samples were converted to arsenic concentrations per hand ($\mu g/cm^2$) by dividing the amount of arsenic in each hand rinsate sample (normalized to 100 mL) by the measured hand size for each volunteer. Gradient performed statistical analysis of each of the hand arsenic data sets (*i.e.*, the reported results for each wood sample (n=10) is considered a data set) to evaluate whether the data were better fit by a normal or lognormal distribution using methodology in accordance with current USEPA risk assessment guidance (see Section 3.3.1.2 for details regarding statistical analysis) (USEPA, 1992b). Summary statistics were calculated for each data set based on the distribution fit, and included the mean, 95% UCLM, and the maximum reported hand arsenic concentration. A summary of the results and the statistical analysis is available in Table 5. The 95% UCLM of the hand arsenic concentrations ranged from 0.005 $\mu g/cm^2$ for the untreated control, up to 0.130 $\mu g/cm^2$ for treated Southern Pine with pressure-applied water repellent.

3.3.2.2 EPCs for Dislodgeable Arsenic

Based on a review of the preceding hand loading studies, Gradient chose the hand arsenic concentrations from the SCS (1998) evaluation to estimate exposures to dislodgeable arsenic. The SCS study was chosen because it tested a number of commonly applied post manufacturer treatments (*e.g.*, stain, sealer, *etc.*), it included both fresh and aged wood samples, it included a negative (untreated) control, it used a conservative yet realistic exposure method, and it included enough samples to enable a

statistical analysis of the hand arsenic concentrations for each wood sample. It should be noted that the adult volunteers in the SCS study grasped each of the wood samples with one hand, and rubbed the wood surface with the other hand using firm downward pressure and a spread palm. This process was repeated using the other hand. This sampling protocol may have overestimated hand loading for typical residential use of treated wood. Furthermore, for the two aged wood samples, not all of the wood surface sampled was exposed to the sun, and therefore, all of the wood sampled may not have been fully aged. Because aging has been shown to reduce the amount of dislodgeable arsenic, it is possible that hand loading from these aged wood samples may overestimate the release of arsenic from aged wood.

Gradient used the 95% UCLM as a conservative estimate of the mean of the reported hand arsenic concentrations for each wood sample tested in the study, based on either a normal or lognormal distribution of the data (the distribution with the best fit was used). The 95% UCLM is used as the EPC to calculate exposures to dislodgeable arsenic for each wood sample, including the control. Table 5 contains a summary of the results and the statistical analysis of the SCS hand loading study (Table 6 contains a summary of the results and the statistical analysis of the wipe sampling portion of the SCS study).

The reported hand loadings for the adult in the MEDHS (1998) study for both the wet and dry hand experiments were on average, approximately 3-fold greater than the findings in the SCS study. The reported hand loadings for the child in the MEDHS study were approximately 4-fold higher than in SCS study. The authors in the MEDHS study concluded that there was little difference between the amount of arsenic loaded onto the child's hands compared to the adult volunteer. This conclusion is probably based on the variability observed in the hand arsenic levels reported for repeated samples in a given experiment, and the difference in the way the adult volunteer and child subject were exposed to the treated wood in the two experiments. The results in the MEDHS study were not used in the HHRA because of the limited nature of the study, including the use of only one wood type, the lack of controls, and the use of only one adult volunteer and child subject.

3.4 Quantification of Exposure

In this section of the HHRA, the basis for calculating human intake levels from exposures to arsenic associated with CCA-treated wood is presented, and each input parameter is described. Exposure estimates represent the daily dose of a chemical taken into the body, averaged over the appropriate exposure period, and expressed in the units of milligram (mg) of chemical per kilogram (kg) of human

body weight a day (mg/kg-day). The primary source for the exposure equations used in the HHRA is the USEPA's "*Risk Assessment Guidance for Superfund* (RAGS)" (USEPA, 1989). The ingestion exposure equation for dislodgeable arsenic is modified slightly to calculate the daily intake of arsenic for this exposure pathway. The generalized equation for calculating chemical intakes is shown below:

$$I = \frac{EPC \times CR \times EF \times ED}{BW \times AT}$$

where:

I	=	Intake, the amount of chemical at the exchange boundary (mg/kg body weight-day),
EPC	=	Exposure Point Concentration, the chemical concentration contacted over the exposure period at the exposure point (*e.g.*, mg/kg in soil),
CR	=	Contact Rate, the amount of contaminated medium contacted per unit time or event (*e.g.*, soil ingestion rate (mg/day) or air inhalation rate (m^3/hr)),
EF	=	Exposure Frequency, describes how often exposure occurs (days/year),
ED	=	Exposure Duration, describes how long exposure occurs (yr),
BW	=	Body Weight, the average body weight over the exposure period (kg), and
AT	=	Averaging Time, the period over which exposure is averaged (days).

Exposure parameters (*e.g.*, exposure frequency, exposure duration, body weight) describe a receptor's exposure for a given exposure scenario. The parameters used in the HHRA as input values in the exposure equations are consistent with current USEPA risk assessment guidance and/or recent peer-reviewed literature. Where appropriate, exposure parameters are based on best professional judgment. Both central tendency exposure (CTE) (*i.e.*, 50^{th} percentile value or mean) and reasonable maximum exposure (RME) (*i.e.*, 90^{th} or 95^{th} percentile value or conservative estimate) parameters are used to assess exposure for the receptors evaluated in the HHRA. CTE parameters provide a typical or average estimate of exposure, and RME parameters provide a high-end estimate of exposure. The key exposure parameters for the receptors evaluated in the HHRA are summarized in Table 7 and are discussed in detail below.

3.4.1 Incidental Ingestion of Soil Arsenic

The intake of arsenic from ingestion of soil for both the residential and playground exposure scenarios is calculated as:

$$Intake\left(\frac{mg}{kg \cdot day}\right) = \frac{EPC\left(\frac{mg}{kg}\right) \times B \times FS \times IR_{soil}\left(\frac{mg}{day}\right) \times EF\left(\frac{day-equiv.}{yr}\right) \times ED(yrs) \times 10^{-6}\,\frac{kg}{mg}}{BW(kg) \times AT(days)}$$

where:

EPC	=	EPC for arsenic in soil (mg/kg),
B	=	Relative Bioavailability of arsenic in soil (unitless),
FS	=	Fraction of Source (unitless),
IR	=	Ingestion Rate (mg/day),
EF	=	Exposure Frequency (day-equivalents/year),
ED	=	Exposure Duration (years),
BW	=	Body Weight (kg), and
AT	=	Averaging Time (days).

The basis for each parameter used to quantify exposure for the soil ingestion pathway is described below.

EPC for Soil Arsenic. As discussed in Section 3.3.1.2, a 95% UCLM of 28.7 mg/kg is used as the EPC for exposure to soil arsenic at a residence. This EPC is based on the reported soil arsenic concentrations from the SCS (2000) arsenic leaching study of soil underneath CCA-treated decks.

As discussed in Section 3.3.1.4, a 95% UCLM of 4.1 mg/kg is used as the EPC for exposure to soil arsenic at a playground. This EPC is based on the reported site-wide soil arsenic concentrations from all 4 playgrounds evaluated in the Malcolm Pirnie (2001) playground study.

Relative Bioavailability (B). The basis for the bioavailability of soil arsenic is briefly discussed here. Refer to Appendix B for a more detailed discussion of the studies and issues related to the bioavailability of soil arsenic.

A critical factor determining the magnitude of potential exposures and risks associated with a chemical is its bioavailability, *i.e.*, the amount of the chemical that is actually absorbed into the body. A chemical's bioavailability is influenced by such factors as the species of the chemical, the matrix in which it is present, the amount of time that a chemical is in a matrix, and the route by which exposure occurs.

When chemicals are ingested, bioavailability is determined by the amount of a chemical that is dissolved in gastrointestinal fluids and absorbed across the gastrointestinal tract into the bloodstream. An ingested chemical that is adsorbed to soil or some other solid medium like wood dust may be absorbed less completely than the same ingested dose of the chemical when dissolved in water (NEPI, 2000).

Another important factor to consider is the relative bioavailability of the chemical under the exposure conditions of interest when compared to the bioavailability of the chemical under the exposure conditions present in the study that forms the basis for the quantitative toxicity factor for the chemical (USEPA, 1989). Frequently, quantitative toxicity factors are calculated based on studies where the chemical was administered in food or water. By contrast, risk assessments for chemicals in the environment often require assessments of the exposures and risks associated with chemicals in soil or other solid media. Where the bioavailability of the chemical observed in the toxicity study is likely to differ from that under the exposure conditions of interest, a relative bioavailability absorption (RBA) factor is derived. The RBA factor for a specific chemical reflects the absorption fraction from soil relative to the absorption fraction from the exposure medium used in the relevant toxicity study (*e.g.,* food or water).

It is widely recognized that the bioavailability of many metals and organic chemicals in soil tends to be considerably lower than bioavailability from food or water (see, for example, Ruby *et al.*, 1999 and Alexander, 2000). Bioavailability from soil can be affected by a number of factors, including the form of the chemical, its solubility, the size distribution of the ingested soil particles, the type of soil, the degree of encapsulation of the chemical within an insoluble matrix, and the nutritional status of the exposed individual.

USEPA guidance recognizes the need to make adjustments for the reduced bioavailability of compounds in soil. Specifically, in Appendix A of the USEPA's "*Risk Assessment Guidance for Superfund*" (USEPA, 1989, pg. A-3), the Agency notes:

> "If the medium of exposure in the site exposure assessment differs from the medium of exposure assumed by the toxicity value (*e.g.*, RfD values usually are based on or have been adjusted to reflect exposure *via* drinking water, while the site medium of concern may be soil), an absorption adjustment may, on occasion, be appropriate. For example, a substance might be more completely absorbed following exposure to contaminated drinking water than following exposure to contaminated food or soil (*e.g.*, if the substance does not desorb from soil in the gastrointestinal tract)."

USEPA guidance also recommends the use of RBA factors "to adjust a food or soil ingestion exposure estimate to match an RfD or slope factor based on the assumption of drinking water ingestion " (USEPA, 1989, pg. A-3).

Appropriate RBA factors for assessing exposure to soil arsenic associated with CCA-treated wood were developed based on review of the extensive information in the scientific literature regarding the generally reduced bioavailability of arsenic in soil. In addition, information regarding the bioavailability and toxicity of arsenic in CCA-treated wood, and in soil from a CCA hazardous waste site, was also reviewed. The chemistry of CCA wood treatment was also considered in assessing the biological availability of arsenic from CCA-treated wood. In selecting RBA factors for use in the HHRA, those studies that used materials derived from CCA-treated wood were judged to be most relevant for these analyses.

Overall, based on rabbit, monkey, dog, and swine studies published in the peer-reviewed literature, relative bioavailability estimates for arsenic in soil range from near zero to approximately 50%. The corresponding oral bioavailability for soluble forms of arsenic (*i.e.,* the type of arsenic present in the epidemiological and animal studies upon which the standard toxicity factors are based) reported in published *in vivo* studies is as high as 95%. Results from two studies of soil from CCA wood treatment sites revealed a similarly reduced relative bioavailability of arsenic. The value used in this risk assessment for CCA in soil (16.3%) is based on the most relevant of these studies, in which primates were fed soil collected at a CCA treatment site (Roberts *et al*., 2001).

The chemistry of arsenic in soil also suggests a reduced bioavailability. Arsenic is generally tightly bound to soils (Cooper, 1990; USDA, 1980). Arsenates, including chromium arsenate, are the form of arsenic in treated wood and released from treated wood as dislodgeable arsenic and soil arsenic. Arsenates strongly bind to ferric hydroxides, which are abundant in soils. Arsenates can also interact with ferric iron, aluminum, or calcium (all abundant in soil) to form insoluble complexes (Cooper, 1990). Unless there are unusual circumstances, such as reducing conditions, which are rare in surface soils, arsenic will remain in the arsenate form. As such, arsenic is in its +5 oxidation state, which is a less soluble and less mobile form than arsenite (As^{+3}) (ATSDR, 2000; Masscheleyn *et al*., 1991 as cited in Townsend, *et al*., 2001). In fact, arsenite (As^{+3}) was looked for and not found in properly treated wood (Nygren and Nilsson, 1993). These observations, regarding the chemical species of arsenic in soil near treated wood structures, its strong binding to soil, and its reduced solubility and mobility, all support the notion of a reduced bioavailability of arsenic in soil.

Fraction of Source (FS). This parameter refers to the fraction of impacted soil that a receptor could potentially be exposed to. The same FS value is used as the CTE and RME estimate; however, different FS values are used for the residential and playground scenarios.

For the residential scenario, it is assumed that CCA-impacted soil is located beneath a residential deck and/or immediately adjacent to a backyard play structure. This assumption is based on data showing that the area of soil affected by treated wood structures is relatively limited (Malcolm Pirnie, 2001; Stilwell and Graetz, 2001), and that arsenic is not very mobile in soil (Cooper, 1990; USDA, 1980). In the Malcolm Pirnie (2001), and Stilwell and Graetz (2001) studies, arsenic concentrations in soil approached background levels within 2-3 feet of a CCA-treated structure. Based on these observations, mobilized arsenic from a treated structure will be confined primarily to the areas beneath a treated deck or immediately adjacent to a treated play structure. Therefore, it is unlikely that a child playing on and around the structure will be exposed only to soil impacted by arsenic from the structure. Instead, a resident playing or undertaking other outdoor activities in the vicinity of a CCA-treated wood structure is likely to encounter soil from a much broader area.

Based on best professional judgment, a FS of 50% is used for both the child resident (ages 2-6) and the child and adult resident (ages 7-31). This parameter reflects the conservative assumption that approximately half of the soil that a resident receptor is exposed to while outside at a residence has been impacted with arsenic from a treated deck and/or play structure. This assumption is conservative for several reasons. First, the soil samples used to calculate the EPC for soil arsenic in the residential scenario were collected directly below a treated deck. Because of the limited mobility of arsenic in soil, such samples are only representative of soil located directly beneath a deck. As discussed above, with increasing distance from the structure, the likely influence of CCA-related arsenic and consequent soil concentrations would be expected to decrease. As a result, residential receptors are likely to receive only a portion of their total soil exposure from areas affected by arsenic from treated wood. Second, the surface of play areas and other residential yard areas frequently is covered by various materials that reduce the potential for direct contact with soil, *e.g.*, wood chips, sand, gravel, or grass. Such coverings would decrease the fraction of total soil intake that would be derived from impacted soil. In addition, to the extent that such surfaces are periodically replaced (*e.g.*, wood chips), such practices would tend to dilute arsenic concentrations in directly contacted materials over time. Finally, using the same FS for the child and adult residents is conservative because older residents are less likely to be exposed to impacted

soil. Thus, the FS value used to quantify exposures to arsenic impacted soil is likely to overestimate actual intake of impacted soil.

For the playground scenario, a value of 100% is used for the FS parameter because the EPC for soil arsenic at a playground is based on a site-wide average, and therefore, is representative of the soil that a child may be exposed to while at a playground. As discussed above, however, to the extent that ground coverings are present at playgrounds, they would tend to reduce the potential for direct contact with impacted soil and could dilute arsenic concentrations in the near vicinity of wood structures over time.

Soil Ingestion Rate (IR_{soil}). Separate soil ingestion rates were used for the child and adult CTE and RME estimates.

CTE Soil Ingestion Rate for Children, Ages 2-6

A value of 36 mg/day is used as the CTE estimate of soil ingestion for children ages 2-6 and is based on the mean soil ingestion rate for the 50th percentile child in a soil ingestion study published by Stanek and Calabrese (1995a).

The Stanek and Calabrese (1995a) study is actually a re-analysis of a previous soil ingestion study of 64 children (ages 1-4) in the town of Amherst, Massachusetts (Calabrese *et al*., 1989). The Amherst study is one of the most comprehensive and detailed studies of children's incidental soil ingestion to date (Calabrese *et al*., 1989). In this study, incidental soil ingestion rates were estimated using a mass balance approach. To evaluate soil and dust intake, soil and dust samples were analyzed for 8 tracer elements. Daily food and fecal samples were collected from each child over an eight day period and were analyzed for the same elements. These results were used to assess the amount of each element excreted by a child and to account for other potential element sources. These data have been extensively analyzed to evaluate the best tracers for assessing soil intake, the best methods for estimating daily soil ingestion rates, and appropriate approaches for extrapolating the results from a short-term study to estimate ingestion rates over longer time periods.

In the re-analysis, the Amherst data were used to develop distributions of potential daily soil ingestion rates, including estimates for various percentiles of the study population. Using this approach, the authors estimated a mean soil ingestion rate for the 50th percentile child of 45 mg/day (Stanek and Calabrese, 1995a). This re-analysis differs from earlier interpretations of the Amherst study (including

evaluations conducted by the study researchers) and reflects a more robust approach that takes into account a greater degree of the information reflected in the study data.

The Amherst study focused on the age range considered to have the highest incidental soil ingestion rates (*i.e.*, ages 1 to 4 years old). By contrast, the age range of interest in the HHRA is 2 to 6 years and includes additional years beyond the period of peak soil ingestion. As a result, the Amherst results were extrapolated to develop an adjusted soil ingestion rate estimate that more accurately reflects the expanded age range. Age-specific differences in incidental soil ingestion rates are widely recognized and reflected in standard risk assessment approaches (*e.g.*, USEPA, 1997a; 1994). To estimate the reduced soil ingestion rates expected in children greater than 4 years old, the estimate based on the Amherst study was adjusted using the relative ratios of age-specific soil ingestion rates presented in the USEPA's Integrated Exposure Uptake Biokinetic (IEUBK) model for assessing children's exposures to lead (USEPA, 1994). The USEPA's soil ingestion rates used in the IEUBK model are based on an analysis of the Amherst study data, and data from a soil ingestion study in children by Davis *et al*. (1990). While subsequent evaluations of these data have raised questions regarding the absolute soil ingestion rates used by the USEPA in the IEUBK model, the relative values derived for specific age ranges still reflects current USEPA guidance (USEPA, 1999d). Gradient used the ratio of the age-specific upper-end soil ingestion estimates in the IEUBK model, and the median soil ingestion rate of 45 mg/day from the re-analysis of the Amherst study, to calculate age-specific soil ingestion rates for children ages 2-6, according to the calculations in Table 3-3, below.

Table 3-3
Calculation of Soil Ingestion Rates (mg/day)
for Children Ages 2-6 Years

Age	IEUBK Default[a]	Ratio of Ingestion Estimates [b]	Adjusted Amherst Study Results[c]
2-3 years	135	1.00	45
3-4 years	135	1.00	45
4-5 years	100	0.74	33
5-6 years	90	0.67	30
6-7 years	85	0.63	28
Average	--	--	**36**

Notes:
a. Source: USEPA, 1994. Table 2-7, p. 2-40.
b. Ratio is based on dividing the IEUBK default soil ingestion estimate by the maximum soil ingestion estimate of 135 mg/day.
c. These values were calculated by multiplying the ingestion ratio by the median soil ingestion estimate of 45 mg/day from Stanek and Calabrese (1995a).

The average soil ingestion estimate based on the calculations in Table 3-3 is 36 mg/day. This value is used to quantify incidental ingestion exposure to soil arsenic, and also dislodgeable arsenic (see Section 3.4.4).

Stanek and Calabrese (1995b) also re-analyzed a combined data set (n=168) based on the Amherst study mentioned above, and another soil ingestion study in children by Davis *et al*. (1990) that involved 104 children (ages 2-7) in the state of Washington. Based on their re-analysis of the combined dataset, the authors estimated a mean soil ingestion rate for the 50th percentile child of 37 mg/day. The median soil ingestion estimate from this study was not used in the HHRA as the CTE estimate because of significant differences between the Amherst and Davis *et al*. (1990) study methodologies and the associated uncertainty in the estimated soil ingestion rates. In contrast with the Amherst study, the Davis *et al*. study did not collect and analyze food and fecal samples on a daily basis, the study used only three soil tracers, and the age range of the children in the studies differed.

Another soil ingestion dataset is available in Stanek and Calabrese (2000), which involved 64 children (ages 1-4) living on a Superfund site in Anaconda, Montana. The reported mean soil ingestion rate for the 50th percentile child from this study is 17 mg/day based on a 7 day study duration. The estimated soil ingestion rate from the Anaconda study is over two-fold less than the authors' estimates based on the re-analysis of the Amherst data (45 mg/day; Stanek and Calabrese, 1995a) and the combined re-analysis of the Amherst and Davis *et al*. (1990) studies (37 mg/day; Stanek and Calabrese, 1995b). This two-fold difference in soil ingestion may be due to differences between the study populations, climates of the study areas, soil-to-dust transfer in the study areas, or knowledge among the Anaconda population that they are living on a Superfund site. The median soil ingestion estimate from the Anaconda study was not used in the HHRA because the estimated value was significantly lower than the authors' other estimated values, and because it is more conservative to use the higher soil ingestion rate based on the Amherst study. Additional information regarding sources of uncertainty and variability in soil ingestion rate estimates is provided in the Uncertainty Assessment in Section 5.5.

RME Soil Ingestion Rate for Children, Ages 2-6

A soil ingestion rate of 100 mg/day is used as the RME estimate for a child ages 2-6 years for both the residential and playground exposure scenarios. This value is based on the USEPA's recommended soil ingestion rate for children under 6 years of age (USEPA, 1997a). Based on recent soil

ingestion studies in the published literature, a soil ingestion rate of 100 mg/day is consistent with an RME estimate (*i.e.*, 95th percentile). For example, the 95th percentile soil ingestion rate from Stanek and Calabrese (2000) ranges from 106 to 133 mg/day, depending on the time period used; and the 95th percentile soil ingestion rate from Stanek *et al*. (2001a) is 91 mg/day.

Adult and Child Over Age 6

A mean soil ingestion rate of 10 mg/day is used as the CTE estimate for the child and adult resident ages 7-31 years, and for the older playground child, ages 7-12. This value is based on a study of 10 adults and was conducted as part of a larger study to evaluate soil ingestion in children (Stanek *et al*., 1997).

There are very few studies in the recently published literature regarding soil ingestion in adults. Most of the published literature only provides estimates of soil ingestion in adults based on different activities (Hawley, 1985 as cited in Sheppard, 1995) or as a percentage of children's ingestion rates (Calabrese and Stanek, 1994). Stanek *et al*. (1997) provides a soil ingestion estimate based on a carefully controlled study, used the best 4 tracer elements identified in the study, and provides estimates based on 280 subject-days, which according to the authors, is the largest amount of data available on soil ingestion rates in adults.

A soil ingestion rate of 50 mg/day is used as the RME estimate for the child and adult resident ages 7-31 years, and for the older playground child, ages 7-12. The USEPA considers 50 mg/day a reasonable central estimate; however the value is considered "highly uncertain" and the Agency does not consider the data sufficient to recommend an upper-percentile value (USEPA, 1997a). Therefore, because a reasonable CTE estimate for older children and adults is available in the recently published literature that is 5-fold less than the USEPA's recommended average value, 50 mg/day is used as the RME estimate.

Exposure Frequency (EF). Different EF values are used for the CTE and RME estimates in both the residential and playground exposure scenarios.

Based on a review of activity studies in the recent peer-reviewed literature, the results of a study by Tsang and Klepeis (1996) were used to calculate EF values for the residential and playground scenarios. Over a 2-year period (*i.e.*, September 1992 to October 1994) Tsang and Klepeis (1996)

conducted a telephone survey of U.S. citizens and obtained information about the amount of time spent in the last 24 hours for a number of different activities, such as the number of minutes spent at a restaurant, at home in the kitchen or traveling in a car. Each respondent maintained a time-log of activities for the previous 24 hours and a total of 3,003 respondents were surveyed in the study.

The Tsang and Klepeis study was used in the HHRA because the results are based on a large number of respondents from all 48 contiguous states, the study was conducted recently, respondents recorded their activities in a time-log for accuracy, information for a range of age groups and activities was obtained, and the results were reported as a distribution of time estimates for different activities. The results of the Tsang and Klepeis study were also used in the USEPA's *Exposure Factors Handbook Volume III – Activity Factors* (1997b) to calculate a distribution of time periods for a variety of different activities.

Residential Exposure Scenario

Children, Ages 2-6

The CTE EF for the child resident is based on the 50th percentile number of minutes that a young child spends at home in the yard or at other areas outside the home (USEPA, 1997b). The 50th percentile estimate is 1.8 hours/day and is based on the two most relevant age ranges (*i.e.*, 1-4 and 5-11 years old) in the USEPA's *Exposure Factors Handbook* (USEPA, 1997b). This value is applied as day-equivalents per year in the HHRA and is based on the number of daylight hours (*i.e.*, approximately 12 hours/day) that a child is likely to be outside and exposed to either soil or dislodgeable arsenic. The day-equivalents approach is based on the following assumptions:

- Exposure to both soil and dislodgeable arsenic occurs only when outside,
- Based on soil ingestion studies by Calabrese *et al*. (1989), soil accounts for about 50% of the daily ingestion of soil, the other 50% comes from ingesting indoor house dust, and
- CCA-impacted soil and dislodgeable arsenic does not significantly contribute to the concentration of arsenic in indoor house dust.

For these reasons, the EF parameter is presented as day-equivalents, averaged over a year.

The calculated EF for the child resident is 55 day-equivalents/year and was calculated as follows:

1.8 hours/day x 7 days = 12.6 hours/week; assuming 12 hours of daylight per day, gives

12.6 hours/week x 1/12 hours/day = 1.1 day-equivalents/week;

using a standard default residential exposure frequency of 350 days/year (50 weeks/year) (USEPA, 1991a), we have

50 weeks/year x 1.1 day-equivalents/week = 55 day-equivalents/year

The RME EF for the child resident is based on the 90th percentile number of minutes that a young child spends at home in the yard or at other areas outside the home (USEPA, 1997b). The 90th percentile estimate is 5.1 hours/day and is based on the two most relevant age ranges (*i.e.*, 1-4 and 5-11 years old) in the USEPA's *Exposure Factors Handbook* (USEPA, 1997b). As described above, this value is applied as day-equivalents per year. The calculated EF for the child resident is 150 day-equivalents/year and was calculated as follows:

5.1 hours/day x 7 days = 35.7 hours/week; assuming 12 hours of daylight per day, gives

35.7 hours/week x 1/12 hours/day = 3 day-equivalents/week;

using a standard default residential exposure frequency of 350 days/year (50 weeks/year) (USEPA, 1991a), we have

50 weeks/year x 3 day-equivalents/week = 150 day-equivalents/year

Child and Adult, Ages 7-31

The CTE EF for the child and adult resident is based on the 50th percentile number of minutes that a child and an adult spend at home in the yard or at other areas outside the home (USEPA, 1997b). The 50th percentile estimate is 1.6 hours/day and is based on the three most relevant age ranges (*i.e.*, 5-11, 12-17, and 18-64 years old) in the USEPA's *Exposure Factors Handbook* (USEPA, 1997b). The calculated EF for the child and adult resident is 45 day-equivalents/year and was calculated as follows:

1.6 hours/day x 7 days = 11.2 hours/week; assuming 12 hours of daylight per day, gives

11.2 hours/week x 1/12 hours/day = 0.9 day-equivalents/week;

using a standard default residential exposure frequency of 350 days/year (50 weeks/year) (USEPA, 1991a), we have

50 weeks/year x 0.9 day-equivalents/week = 45 day-equivalents/year

The RME EF for the child and adult resident is based on the 90th percentile number of minutes that a child and an adult spend at home in the yard or at other areas outside the home (USEPA, 1997b). The 90th percentile estimate is 4.9 hours/day and is based on the three most relevant age ranges (*i.e.*, 5-11, 12-17, and 18-64 years old) in the USEPA's *Exposure Factors Handbook* (USEPA, 1997b). The calculated EF for the child and adult resident is 73 day-equivalents/year and was calculated as follows:

4.9 hours/day x 7 days = 34.3 hours/week; assuming 12 hours of daylight per day, gives

34.3 hours/week x 1/12 hours/day = 2.9 day-equivalents/week;

using a standard default residential exposure frequency of 350 days/year (50 weeks/year) (USEPA, 1991a), we have

50 weeks/year x 2.9 day-equivalents/week = 145 day-equivalents/year

An EF of 73 day-equivalents/year (*i.e.*, 145 day-equivalents/year ÷ 2 = 73 day-equivalents/year) was used for this parameter because unlike the child resident, not all of the time that an older child and adult resident spend outdoors in the yard is assumed to be exposed to either soil or dislodgeable arsenic. Exposure to either soil or dislodgeable arsenic is assumed to occur when a resident is on or around a CCA-treated deck or play structure. It is assumed that the older child and adult will be engaged in other activities in the yard (*e.g.*, non-team sports, home maintenance and repair projects) that will reduce the amount of time spent on or around a CCA-treated deck or play structure.

Another reason for reducing the 90th percentile estimate from the Tsang and Klepeis study is the methodology the investigators used to obtain time estimates for the different activities. The Tsang and Klepeis study relied on the amount of time respondents reported spending on different activities during the last 24 hours. Data collected in this way tends to be skewed towards higher values at the upper percentiles (*i.e.*, 90th and 95th). This result occurs because respondents are asked about the amount of time spent the day before on a specific activity, a range of time estimates will be given, some respondents will indicate a great deal of time spent on an activity and others will indicate little or no time. When these results are compiled, the extreme upper percentiles will contain artificially increased time estimates. Evidence of this effect can be seen in the results of a study conducted by Garlock *et al*. (1999) where 450 adult respondents (*i.e.*, 18 and over) were surveyed by phone about the amount of time spent on various activities during the previous year. The methodology used in the Garlock study forced the respondents to

average the amount of time spent on different activities over the course of the previous year. This study approach tends to remove outliers in the upper percentiles for a specific activity. The Garlock study did not report the 90th percentile estimates of the time use patterns for specific activities; however, the 95th percentile estimate from this study of the average amount of time spent on outdoor activities such as gardening and other yard work is approximately 16.8 hours/week or 70 day-equivalents/year. This result is approximately half of the 90th percentile value form the Tsang and Klepeis study and is consistent with the EF of 73 day-equivalents/year used as the RME estimate for the older child and adult resident.

Playground Exposure Scenario

Children, Ages 2-6

The CTE EF for the playground child is based on the 50th percentile number of minutes that a child spends outdoors on school grounds or a playground (USEPA, 1997b). The 50th percentile estimate is 1 hour/day and is based on the two most relevant age ranges (*i.e.*, 1-4 and 5-11 years old) in the USEPA's *Exposure Factors Handbook* (USEPA, 1997b). The calculated EF for the playground child is 31 day-equivalents/year and was calculated as follows:

1 hour/day x 7 days = 7 hours/week; assuming 12 hours of daylight per day, gives

7 hours/week x 1/12 hours/day = 0.6 day-equivalents/week;

using an exposure frequency of 365 days/year (52 weeks/year), we have

52 weeks/year x 0.6 day-equivalents/week = 31 day-equivalents/year

The RME EF for the playground child is based on the 90th percentile number of minutes that a child spends outdoors on school grounds or a playground (USEPA, 1997b). The 90th percentile estimate is 2.9 hours/day and is based on the two most relevant age ranges (*i.e.*, 1-4 and 5-11 years old) in the USEPA's *Exposure Factors Handbook* (USEPA, 1997b). The calculated EF for the playground child is 88 day-equivalents/year and was calculated as follows:

2.9 hours/day x 7 days = 20.3 hours/week; assuming 12 hours of daylight per day, gives

20.3 hours/week x 1/12 hours/day = 1.7 day-equivalents/week;

using an exposure frequency of 365 days/year (52 weeks/year), we have

52 weeks/year x 1.7 day-equivalents/week = 88 day-equivalents/year

Children, Ages 7-12

The CTE EF for the older playground child is based on the 50th percentile number of minutes that a child spends outdoors on school grounds or a playground (USEPA, 1997b). The 50th percentile estimate is 1 hour/day and is based on the most relevant age range (*i.e.*, 5-11 years old) in the USEPA's *Exposure Factors Handbook* (USEPA, 1997b). The calculated EF for the older playground child is 31 day-equivalents/year and was calculated as follows:

1 hour/day x 7 days = 7 hours/week; assuming 12 hours of daylight per day, gives

7 hours/week x 1/12 hours/day = 0.6 day-equivalents/week;

using an exposure frequency of 365 days/year (52 weeks/year), we have

52 weeks/year x 0.6 day-equivalents/week = 31 day-equivalents/year

The RME EF for the older playground child is based on the 90th percentile number of minutes that a child spends outdoors on school grounds or a playground (USEPA, 1997b). The 90th percentile estimate is 2.8 hours/day and is based on the most relevant age range (*i.e.*, 5-11 years old) in the USEPA's *Exposure Factors Handbook* (USEPA, 1997b). The calculated EF for the older playground child is 83 day-equivalents/year and was calculated as follows:

2.8 hours/day x 7 days = 19.6 hours/week; assuming 12 hours of daylight per day, gives

19.6 hours/week x 1/12 hours/day = 1.6 day-equivalents/week;

using an exposure frequency of 365 days/year (52 weeks/year), we have

52 weeks/year x 1.6 day-equivalents/week = 83 day-equivalents/year

It should be noted that the way the EF values are applied in the HHRA to quantify exposures for both the residential and playground scenarios will overestimate exposure and risk because almost all of the time outdoors at a residence or at a playground is assumed to be spent on or near a CCA-treated structure. In actuality, individuals are likely to spend some portion of their outdoor time in activities and locations that do not include exposure to treated wood structures. As a result, their exposure frequency is likely to be less than that assumed in the HHRA. Moreover, the approach used in the HHRA inherently assumes that exposures to both dislodgeable and soil arsenic are occurring simultaneously. In fact, an

individual is likely to be exposed to only one of these exposure media at a time, and time spent exposed to one of these media should be subtracted from time spent exposed to the other. As a result, the approach taken in the HHRA is likely to overestimate overall exposures and risks.

Exposure Duration (ED). The ED values used for the three receptors evaluated in the HHRA are based on their assumed age ranges. The ED for the child, ages 2-6, in both the residential and playground exposure scenarios is 5 years. The ED for the child and adult resident, ages 7-31, is 25 years. The exposure duration for the older playground child, ages 7-12 years, is 6 years.

Body Weight (BW). In accordance with current USEPA risk assessment guidance, the use of a mean bodyweight is appropriate when either CTE or RME estimates are used to quantify exposure (USEPA, 1989). A bodyweight of 17.8 kg is used for the child receptor, ages 2-6, and is based on the mean bodyweight of boys and girls ages 2-6 (USEPA, 1997a). A bodyweight of 58.6 kg is used for the child and adult receptor, ages 7-31, and is based on the mean bodyweight of males and females ages 7-31 (USEPA, 1997a). A bodyweight of 34.5 kg is used for the older child receptor, ages 7-12, and is based on the mean bodyweight of male and female children ages 7-12 (USEPA, 1997a).

Averaging Time (AT). The AT values used to estimate non-cancer health risks for the three receptors evaluated in the HHRA are based on their assumed age ranges. The AT used to estimate non-cancer risks for the child receptor, ages 2-6 years, is 5 years. The AT used to estimate chronic non-cancer risks for the child and adult resident is 30 years, based on an age range of 2-31 years. Chronic, non-cancer risks for the child and adult resident are based on a time-weighted adjustment of the dose for the child resident, ages 2-6 years, and the child and adult resident, ages 7-31 years. The AT used to estimate non-cancer risks for the older child receptor, ages 7-12 years, is 6 years.

For cancer risk, exposures are averaged over a lifetime. The current USEPA-recommended average life expectancy for women and men in the U.S. is 75 years (USEPA, 1997a). However, the USEPA used a lifetime expectancy of 70 years to calculate cancer potency values (*i.e.,* cancer slope factors and unit risks) in its on-line chemical toxicity database, *i.e.*, the Integrated Risk Information System or IRIS (USEPA, 1997a). Therefore, to be consistent with the derivation of cancer potency values recommended by the USEPA and used in the HHRA, Gradient used a 70-year life expectancy (which equates to an AT of 25,550 days) to estimate potential cancer risks for all three receptors.

3.4.2 Dermal Contact with Arsenic in Soil

For dermal exposure to arsenic in soil, intake is calculated as follows (USEPA, 1999a):

$$Intake\left(\frac{mg}{kg \cdot day}\right) = \frac{EPC\left(\frac{mg}{kg}\right) \times DA \times AF\left(\frac{mg}{cm^2}\right) \times SA\left(\frac{cm^2}{day}\right) \times EF\left(\frac{day-equiv}{yr}\right) \times ED(yrs) \times 10^{-6}\ \frac{kg}{mg}}{BW(kg) \times AT(days)}$$

where:

EPC = EPC of arsenic in soil (mg/kg),
DA = Dermal Absorption fraction (unitless),
AF = Soil-skin Adherence Factor (mg/cm^2),
SA = Skin surface Area exposed (cm^2/day),
EF = Exposure Frequency (day-equiv/year),
ED = Exposure Duration (years),
BW = Body Weight (kg), and
AT = Averaging Time (days).

There are three parameters in this equation that differ from those discussed in the assessment of ingestion exposure to arsenic in soil (Section 3.4.1), and include the dermal absorption fraction (DA), the soil adherence factor (AF), and the skin surface area (SA). The same EPC for soil arsenic used to assess ingestion exposure was used to assess dermal exposure to arsenic in soil.

Note that since absorbed doses are used for the dermal pathway, the toxicity criteria must be adjusted so they apply to absorbed doses. This adjustment is discussed in more detail in the Toxicity Assessment in Section 4.

Dermal Absorption Fraction (DA). The DA used to quantify exposure to soil arsenic is based on the USEPA's recommended dermal absorption fraction of 3% for arsenic in soil (USEPA, 1999a). The same issues that contribute to a reduced oral bioavailability of arsenic in soil hold-true for the dermal bioavailability of arsenic in soil. Therefore, a bioavailability of 16.3% is also used for dermal absorption of arsenic in soil, resulting in a DA of 0.5% (*i.e.*, 0.163 x 0.03 = 0.0049 or 0.5%).

Soil to Skin Adherence Factor (AF). The AF describes the amount of soil that adheres to the skin per unit surface area (USEPA, 1999a). Adherence factors vary depending on the properties of the soil, the part of the body exposed, and the type of activity. The AF values used to quantify dermal exposure to soil arsenic are based on USEPA-recommended AF values of 0.2 mg/cm^2 and 0.07 mg/cm^2 for child (ages

1-6) and adult (18 years and older) residents, respectively (USEPA, 1999a). These AF values are consistent with an RME estimate of exposure for face, hands, forearms, lower legs and feet (USEPA, 1999a).

The USEPA-recommended AF values are weighted based on a hand AF of 1.1 mg/cm^2, and the contribution of the surface area of both hands to the total surface area of the body parts used to derive the recommended value, which includes the face, hands, forearms, lower legs and feet. This was done to be consistent with the AF (1.1 mg/cm^2) used to quantify ingestion exposure to dislodgeable arsenic (see Section 3.4.4). The weighted AF values used for the child ages 2-6, the older child ages 7-12, and the child and adult ages 7-31 are 0.34, 0.33, and 0.27 mg/cm^2, respectively.

Skin Surface Area Exposed (SA). This parameter represents the amount of skin that is assumed to be available for exposure to arsenic in soil. The estimation of exposure based on both CTE and RME estimates assumes that the child and adult receptors are wearing shoes, shorts and a T-shirt, and therefore, the surface areas assumed to be available for dermal exposure include the forearms, hands, and lower legs.

Consistent with USEPA risk assessment guidance, median skin surface areas were calculated for use with both CTE and RME parameters (USEPA, 1989). Median skin surface areas of 3,317, 5,198, and 5,800 cm^2 are used for the child ages 2-6, the older child ages 7-12, and the child and adult ages 7-31 (USEPA, 1997a).

3.4.3 Inhalation Exposure to Soil Arsenic Particulate

For inhalation of soil arsenic particulate, exposure is calculated as:

$$EPC_{air} = \frac{ET\left(\frac{hr}{day}\right) \times EF\left(\frac{day-equiv}{yr}\right) \times ED(yrs) \times C_{air}\left(\frac{mg}{m^3}\right) \times 10^3 \frac{\mu g}{mg}}{24\frac{hours}{day} \times AT(days)}$$

where:

EPC_{air} = Exposure Point Concentration of soil arsenic in air ($\mu g/m^3$),
C_{air} = Modeled Concentration of soil arsenic in air (mg/m^3),
ET = Exposure Time (hours/day),
EF = Exposure Frequency (day-equivalents/yr),
ED = Exposure Duration (yrs), and
AT = Averaging Time (days).

There are two parameters in this equation that are different from those discussed in the quantification of ingestion exposure to soil arsenic (Section 3.4.1), and they include the Concentration of soil arsenic particulate in air (C_{air}) and Exposure Time (ET). Therefore, only these parameters unique to the inhalation exposure equation are discussed below.

Concentration in air (C_{air}). The concentration of soil arsenic particulate in air is based on the PM_{10} emission factor (described in Section 3.3.1.5) for wind-blown soil particulate, and the concentration of soil arsenic in each of the exposure scenarios evaluated in the HHRA. For the residential exposure scenario, the concentration of arsenic in soil beneath a CCA-treated deck (*i.e.*, 28.7 mg/kg) is used to calculate the concentration of soil arsenic particulate in air. For the playground exposure scenario, the concentration of arsenic in soil from the Malcolm Pirnie playground study (*i.e.*, 4.1 mg/kg) is used to calculate the concentration of soil arsenic particulate in air

Exposure Time (ET). The ET used in this equation is 24 hours/day for the residential exposure scenario and 12 hours/day for the playground scenario. It is conservatively assumed that a resident could be exposed to respirable soil particulate during the entire period of time while at home in the yard or indoors. However, it is assumed that a child receptor at a playground would not be exposed to soil particulate for more than 12 hours/day.

3.4.4 Incidental Ingestion of Dislodgeable Arsenic

For hand-to-mouth ingestion of dislodgeable arsenic, intake is calculated as:

$$Intake\left(\frac{mg}{kg\cdot day}\right)=\frac{EPC\left(\frac{\mu g}{cm^2}\right)\times HTE\left(\frac{handtransfer}{day}\right)\times SA\left(\frac{cm^2}{handtransfer}\right)\times EF\left(\frac{day-equiv}{yr}\right)\times ED(yrs)\times B\times CF\left(10^{-3}\,\frac{mg}{\mu g}\right)}{BW(kg)\times AT(days)}$$

where:

EPC = EPC for ingestion of dislodgeable arsenic on the hands ($\mu g/cm^2$),
HTE = Daily Hand Transfer Efficiency factor (hand transfer/day),
SA = Skin Surface Area exposed (cm^2/hand transfer),
EF = Exposure Frequency (day-equivalents/year),
ED = Exposure Duration (years),
B = Bioavailability of dislodgeable arsenic complex (unitless),
CF = Unit Conversion Factor (mg/μg),
BW = Body Weight (kg), and
AT = Averaging Time (days).

Only the parameters that differ from those used in the quantification of ingestion exposure to soil arsenic (Section 3.4.1) are described here, and include the exposure point concentration (EPC), the daily hand transfer efficiency (HTE) factor, and the exposed skin surface area of the hands (SA).

EPC for Dislodgeable Arsenic. The 95% UCLM of the reported hand arsenic concentrations ($\mu g/cm^2$) for each wood sample tested in the SCS hand loading study (see Section 3.3.2.2) are used as the EPCs to evaluate exposure to dislodgeable arsenic *via* incidental ingestion. The same set of EPCs are used with both CTE and RME parameters to quantify exposure.

Daily Hand Transfer Efficiency (HTE). As described in Section 3.3.2.1, several studies were reviewed that evaluated the amount of dislodgeable arsenic transferred to the hands when dermal contact is made with CCA-treated wood. Data regarding the amount of arsenic that might be dislodged from the surface of treated wood is available primarily in units of mass of arsenic present per unit of skin surface area (*i.e.*, $\mu g/cm^2$). Therefore, the amount of arsenic on the hands that might be ingested *via* hand-to-mouth contact was calculated in a two-step process. First, the amount of arsenic on the hands was calculated for several different samples of CCA-treated wood based on the results of the SCS (1998) hand-loading study, which was described previously in Sections 3.3.2.1 and 3.3.2.2. Second, a hand transfer efficiency (HTE) factor was calculated to estimate the proportion of dislodgeable arsenic on the hands that might be subsequently ingested. The basis for the HTE factor that was used to quantify ingestion exposure to dislodgeable arsenic is briefly described below. The data and calculations used to derive the HTE factor are discussed in more detail in Appendix C.

To develop the HTE factor, data regarding children's incidental ingestion of soil, adherence of soil to the hands, and the skin surface area of the hands were reviewed. The HTE factor was calculated based on data regarding lead loading onto hands that were collected as part of a community study of

children's exposures in the vicinity of a smelter (Roels *et al*., 1980). In this study, researchers assessed the mass of lead adhering to children's hands by rinsing the front surface of the children's hands and analyzing the rinsate for lead. Average lead concentrations in soil samples were then used to estimate the average amount of soil adhering to the hands.

These values were then divided by median estimates of the skin surface area of the hands assumed to be available for dermal contact with a treated wood surface to generate a soil adherence factor (AF) of 1.1 mg/cm^2 for both boys and girls. Skin surface area data specific to the average age of the children included in the study (*i.e.*, 11 year old children) were used in these calculations. As described in the next section, the available skin surface area of both hands was assumed to comprise one-third of the total surface area of the hands. Using skin surface area data specific to the age range of interest for young children in the HHRA (*i.e*., 2 to 6 years old), the AF was used to estimate the average mass of soil present on young children's hands.

This loading estimate was then combined with an estimated soil ingestion rate to derive the HTE factor, *i.e.*, to determine what proportion of the mass of soil adhering to the hands would need to be ingested to yield the estimated soil ingestion rate. As described in Section 3.4.1, a soil ingestion rate of 36 mg/day for children, ages 2-6 years, was calculated based on a soil ingestion study conducted in Amherst, Massachusetts (Calabrese *et al*., 1989; Stanek and Calabrese, 1995a). This soil ingestion rate is the average estimate for the 50th percentile child. When divided by the average soil loading estimate for young children's hands (146 mg on both hands) based on the Roels *et al*. (1980) study, a daily HTE factor of approximately 0.25 hand transfers/day is calculated. This value indicates that, on average, incidental ingestion of approximately one-fourth of the soil adhering to the front surface of children's hands yields the typical estimated soil ingestion rate. In the HHRA, the HTE factor is used to estimate the percentage of the total amount of dislodgeable arsenic on the surface of both hands that is incidentally transferred to the mouth during hand-to-mouth contact.

An HTE factor of 0.13 is used for the older child (ages 7 and 12) and for the child and adult receptor (ages 7-31). This value is approximately one-half of the HTE derived for the young child receptor (ages 2-6). The HTE factor for the older child and adult receptors was reduced relative to that for the young child to reflect the reduced hand-to-mouth behavior in children greater than 6 years old. This assumption is further supported by the fact that the USEPA-recommended mean soil ingestion rate for adults is exactly one-half of the recommended value for children less than 6 years of age (USEPA, 1997a).

The same applicable HTE factors for young children, and for older children and adults are used with both CTE and RME parameters to quantify exposure to dislodgeable arsenic *via* incidental ingestion.

The Roels *et al*. (1980) study was selected as the basis for the HTE because it provides direct empirical measurements of soil adherence to the palmar surface of children's hands following a variety of activities. By contrast, many of the more recent studies of soil adherence have focused on adherence associated with specific activities or have evaluated soil adherence over more extensive skin surface areas. Palmar skin surfaces are the primary skin area likely to have contact with treated wood surfaces and soil adherence to palmar surfaces is generally greater than adherence to other skin areas. As a result, Roels *et al*. was selected as the best study for estimating the HTE factor because the data provided in this study are most relevant to the evaluations of dislodgeable arsenic adherence that are of interest in the HHRA.

Skin Surface Area (SA). It is assumed that dislodged arsenic on the surface of both hands could be ingested *via* hand-to-mouth contact. The skin surface area of both hands assumed to be available for contact with a treated wood surface is 1/3 of the total surface area of both hands. The total surface area of both hands is based on male and female hand size data in the USEPA's *Exposure Factors Handbook* (1997a). The 2/3 reduction in total hand surface area is based on three separate sources. The USEPA Office of Pesticides convened an expert panel to evaluate the key issues in the assessment of residential and non-occupational exposure to pesticides (USEPA, 1999c). One of the comments from the expert panel indicated that a value of approximately 1/3 of the total median surface area of both hands represents the hand surface area available for contact with a surface. Rodes *et al*. (2001) conducted hand-press trials to quantify the transfer of particles from indoor surfaces to human skin. One of the findings in this study was that only approximately 1/3 of the hand surface typically came in contact with a smooth test surface (Rodes *et al*., 2001). And finally, measured hand sizes based on tracing each hand of five different adult male volunteers in the SCS (1998) hand loading study were compared to the total surface area of one hand for adult males in USEPA (1997a). Based on this comparison, the measured hand surface areas from the SCS study were on average approximately 1/3 of the total hand surface area calculated for an adult based on data in USEPA (1997a).

The hand surface areas used to quantify ingestion exposure to dislodgeable arsenic on a treated wood surface are 132 cm^2 for the child receptor ages 2-6; 188 cm^2 for the older child receptor ages 7-12; and 267 cm^2 for the child and adult receptor ages 7-31. The same hand surface areas for children and

adults are used with both CTE and RME estimates to quantify exposure to dislodgeable arsenic *via* incidental ingestion.

Relative Bioavailability (B). The basis for the bioavailability of dislodgeable arsenic is briefly discussed here. Refer to Appendix B for a more detailed discussion of the studies and issues related to the bioavailability of dislodgeable arsenic.

An appropriate RBA factor for assessing exposure to arsenic associated with CCA-treated wood was developed based on review of the extensive information in the scientific literature regarding the generally reduced bioavailability of arsenic in soil. To assess the potential exposures and risks associated with arsenic in CCA-treated wood, wood dust and other dislodgeable materials on the surface of treated wood are of interest. For arsenic present in material directly contacted and dislodged from CCA-treated wood, the relative bioavailability estimate used in this risk assessment (47%) is based on two studies of dogs fed sawdust from CCA-treated wood (Peoples, 1976; Peoples and Parker 1979). Other factors supporting a reduced bioavailability of dislodgeable arsenic from CCA-treated wood include the chemistry of the wood treatment process, which is designed to fix arsenic and the other metals within the wood matrix; studies indicating that only a small proportion of dislodgeable arsenic is soluble; and toxicology and epidemiology studies indicating few adverse effects that are attributable to arsenic exposure from CCA-treated wood.

In addition to the animal studies used to derive an estimate of oral bioavailability, other factors support an assumption of reduced bioavailability for dislodgeable arsenic. First, the chemical process which occurs during wood treatment is designed to bind the CCA in the wood so that the fixative will persist and prevent deterioration of the wood over a long period of time (Bull, 2001). Second, a study of the composition of dislodgeable materials suggests that a substantial proportion of the arsenic observed on the surface of CCA-treated wood is insoluble. Specifically, an analysis of dislodgeable surface materials collected from samples of CCA-treated wood found that on average arsenic comprised at most 0.19% of the surface material and that approximately 94-100% of the surface arsenic was insoluble in water (Cui, 2001; Osmose, 2001). This finding supports the assumption that the bioavailability of arsenic present in dislodged materials is less than would be expected based on consideration of the total measured arsenic concentration.

Additional evidence of the reduced bioavailability of dislodgeable arsenic comes from the results of leaching studies. Data from leaching studies indicate that arsenic is not released from treated wood to

any appreciable extent under normal outdoor conditions (*e.g.*, when exposed to rainwater) and is primarily mobilized from the wood through physical transport of dislodgeable particles (*e.g.*, wood particles and/or insoluble arsenate complex). Based on a review of the leaching studies, two key observations support Gradient's estimate of the bioavailability of dislodgeable arsenic: 1) the duration of the leaching studies in acidic solutions ranged from 4 to 40 days, this duration is significantly longer than the period of time that food (or ingested dislodgeable arsenic) is in the human stomach-approximately 4 hours (Vander *et al*., 1994); and 2) the reported amount of leached arsenic in these studies ranged from 17 to 44%. These observations support Gradient's estimation of 47% bioavailability of dislodgeable arsenic.

3.4.5 Dermal Contact with Dislodgeable Arsenic

For dermal exposure to dislodgeable arsenic on the surface of treated wood, intake is calculated as follows:

$$Intake\left(\frac{mg}{kg\cdot day}\right)=\frac{EPC\left(\frac{\mu g}{cm^2}\right)\times DA\times SA\left(\frac{cm^2}{day}\right)\times EF\left(\frac{day-equiv}{yr}\right)\times ED(yrs)\times CF\left(10^{-3}\frac{mg}{\mu g}\right)}{BW(kg)\times AT(days)}$$

where:

EPC = EPC for dislodgeable arsenic on the hands (μg/cm^2),
DA = Dermal Absorption fraction (unitless),
SA = Skin surface Area exposed (cm^2/day),
EF = Exposure Frequency (day-equivalents/year),
ED = Exposure Duration (years),
CF = Conversion Factor (mg/μg),
BW = Body Weight (kg), and
AT = Averaging Time (days).

There are three parameters in this equation that differ from those discussed in the assessment of ingestion exposure to arsenic in soil (Section 3.4.1), and include the exposure point concentration (EPC), dermal absorption fraction (DA), and the skin surface area (SA).

Note that since absorbed doses are used for the dermal pathway, the toxicity criteria must be adjusted so they apply to absorbed doses. This adjustment is discussed in more detail in the Toxicity Assessment in Section 4.

EPC for Dislodgeable Arsenic. The 95% UCLM of the reported hand arsenic concentrations ($\mu g/cm^2$) for each wood sample tested in the SCS (1998) hand loading study (see Section 3.3.2.2) are used as the EPCs to evaluate exposure to dislodgeable arsenic *via* dermal contact. The same set of EPCs are used with both CTE and RME parameters to quantify exposure.

Dermal Absorption Fraction (DA). The DA represents the amount of a chemical that is in contact with the skin and that is absorbed through the skin and into the bloodstream. The DA used to quantify exposure to dislodegable arsenic is based on the USEPA's recommended dermal absorption fraction for arsenic in soil of 3% (USEPA, 1999a). However, this dermal absorption fraction was adjusted to 1.4% (*i.e.*, 0.03 x 0.47 = 0.014 or 1.4%) based on the bioavailability (47%) of dislodgeable arsenic.

Skin Surface Area Exposed (SA). This parameter represents the amount of skin that is assumed to be available for exposure to dislodgeable arsenic on a treated wood surface. It is assumed that both child and adult receptors are wearing shoes, shorts and a T-shirt; however, the only skin surface considered to be exposed to a treated wood surface on a consistent basis are the hands. Therefore, as described in the previous section (Section 3.4.4), 1/3 of the total surface area of both hands is used as the skin surface available for contact with a treated wood surface.

The hand surface areas used to quantify dermal exposure to dislodgeable arsenic on a treated wood surface are 132 cm^2 for the child receptor ages 2-6; 188 cm^2 for the older child receptor ages 7-12; and 267 cm^2 for the child and adult receptor ages 7-31. Except for the child resident (ages 2-6), the same hand surface areas are used as both CTE and RME parameters.

The RME parameter for the child resident, ages 2-6, includes exposure to both hands and feet. It is assumed that the child resident may not be wearing shoes while playing on a treated structure. This assumption did not seem reasonable for the older child and adult resident receptor or for the child receptors at a playground, and therefore, is not used for the RME estimates of exposure for these receptors. Consistent with the assumption regarding the skin surface of the hands available for contact with a treated wood surface, 1/3 of the total skin surface area of both feet and hands is used for the RME estimate of exposure for the child resident. The foot and hand surface area used for the child resident is 300 cm^2.

4 Toxicity Assessment

4.1 Overview of Dose-Response Data

Gradient assessed potential cancer and non-cancer risks from exposure to arsenic associated with the use of CCA-treated wood using dose-response relationships for carcinogenicity (*i.e.*, oral Cancer Slope Factor and inhalation Unit Risk) and systemic toxicity (*i.e.*, oral Reference Dose).

The primary source for the arsenic toxicity criteria used in the HHRA was the USEPA's Integrated Risk Information System (IRIS) (USEPA, 2001a). Toxicity criteria in IRIS undergo a peer review process and represent the generally accepted approach in the Agency. A subchronic oral Reference Dose (subchronic RfD_{oral}) for arsenic is also used in the HHRA to quantify non-cancer health risks for the child receptors ages 2-6 and 7-12. The subchronic RfD was developed by USEPA, Region 8 and is considered appropriate to assess acute and subchronic exposures to inorganic arsenic in drinking water, food, and soil for periods up to 7 years (USEPA, Region 8, 2001).

In addition to describing the basis and application of the arsenic toxicity criteria used in the HHRA to quantify risks, this chapter also addresses other toxicity issues associated with exposure to arsenic, including children's relative sensitivity to arsenic, arsenic's purported role in endocrine disruption, and a review of occupational and animal studies to evaluate potential health effects from exposures to arsenic in CCA-treated wood.

4.2 Cancer Dose-Response Data

4.2.1 Oral Cancer Slope Factor (CSF_{oral})

The CSF is an upper-bound estimate of carcinogenic potency used to calculate risk from exposure to carcinogens by relating estimates of lifetime average chemical intake to the incremental risk of an individual developing cancer over their lifetime (USEPA, 1992c). The CSFs recommended by the USEPA are conservative upper-bound estimates, which means that the USEPA is reasonably confident that the "true" cancer risk does not exceed the estimated risk based on the CSF, and may be as low as zero. A USEPA-recommended CSF_{oral} value is available for arsenic.

4.2.2 Dermal Cancer Slope Factor (CSF_{dermal})

There are no USEPA-derived toxicity values specifically for cancer studies involving dermal exposures. In the absence of dermal-specific CSFs, oral CSFs are used, assuming that once a chemical is absorbed into the blood stream, the carcinogenic effect is similar regardless of whether the route of exposure was oral or dermal. However, since a CSF_{oral} is based on the amount of a chemical *administered* per unit time and body weight (chemical intake), it needs to be adjusted to be applicable to *absorbed* doses (dermal exposures are expressed as absorbed intake levels) (USEPA, 1989; 1999a). If oral absorption is very high (almost 100%), then the absorbed dose is virtually the same as the administered dose, and no adjustment of the CSF_{oral} is necessary. If oral absorption is very low (*e.g.*, 5%), the absorbed dose is much smaller than the administered dose, and an adjustment of the toxicity criteria is necessary. For any given chemical, the USEPA recommends adjusting the CSF_{oral} to evaluate dermal risks only when the oral absorption for a chemical is less than 50%, to "obviate the need to make comparatively small adjustments in the toxicity value that would otherwise impart on the process a level of accuracy that is not supported by the scientific literature" (USEPA, 1999a).

To assess cancer risk from dermal exposure, this adjustment is made by dividing the CSF_{oral} (for applied doses) by the oral absorption fraction (*i.e.*, $CSF_{oral} / Abs_{oral} = CSF_{dermal}$).

4.2.3 Inhalation Unit Risk (UR_{inhal})

A UR_{inhal} is used to assess the risk of developing cancer from inhalation exposure to arsenic. The UR_{inhal} is based on the incremental risk of developing cancer from exposure to 1 µg of a chemical per cubic meter (m^3) of air.

4.3 Non-Cancer Dose-Response Data

4.3.1 Oral Reference Dose (RfD_{oral})

An RfD_{oral} is an estimate of daily exposure to a substance that a sensitive population can experience over a lifetime with a negligible risk of adverse systemic health effects. The USEPA derives RfDs by first identifying the highest dose level that does not cause observable adverse health effects (*i.e.*, the No Observed-Adverse Effect Level, or NOAEL; USEPA, 1993). If a NOAEL was not identified, a Lowest Observed Adverse Effect-Level, or LOAEL, may be used. This dose level is then

divided by uncertainty factors to calculate an RfD. An uncertainty factor of 100 is often used, to account for interspecies differences (if animal studies were used) and sensitive human subpopulations (*e.g.,* children and the elderly; USEPA, 1993). Additional uncertainty factors may be used, depending on the quality of the toxicity study and/or confidence in the data.

4.3.2 Dermal Reference Dose (RfD_{dermal})

There are no USEPA-derived toxicity values based specifically on toxicity studies involving dermal exposures. In the absence of dermal-specific RfDs, oral toxicity factors are used, assuming that once a chemical is absorbed into the blood stream, the health effects are similar regardless of whether the route of exposure was oral or dermal. However, since oral toxicity criteria are based on the amount of a chemical *administered* per unit time and body weight (chemical intake), they need to be adjusted to be applicable to *absorbed* dose (dermal exposures are expressed as absorbed intake levels) (USEPA, 1989; 1999a).

Since most RfDs are based on studies where a chemical is administered in food or water, this adjustment is made using the oral absorption fraction for the chemical. For any given chemical, the USEPA recommends adjusting the RfD_{oral} to evaluate dermal risk only when the oral absorption for that chemical is less than 50%, to "obviate the need to make comparatively small adjustments in the toxicity value that would otherwise impart on the process a level of accuracy that is not supported by the scientific literature" (USEPA, 1999a).

To assess non-cancer health effects from dermal exposure, this adjustment is made by multiplying the RfD_{oral} (for administered dose) by the oral absorption fraction (*i.e.*, $RfD_{oral} \times Abs_{oral} = RfD_{dermal}$).

4.4 Arsenic Toxicity Criteria

In IRIS, the USEPA currently has the following toxicity criteria available for arsenic: CSF_{oral}, UR_{inhal}, and RfD_{oral}. Each of these criterion are used to quantify risk in the HHRA (USEPA, 2001b). As previously noted, a subchronic RfD_{oral} from USEPA, Region 8 is also used in the HHRA (USEPA, Region 8, 2001). The derivation of the CSF_{oral}, UR_{inhal}, chronic RfD_{oral}, and subchronic RfD_{oral}, and the scientific uncertainties concerning these toxicity criteria, are discussed below.

4.4.1 Arsenic CSF_{oral}

The USEPA concluded that arsenic is a "human carcinogen", a weight-of-evidence classification for carcinogenicity of "A" (USEPA, 2001b). This classification is based on sufficient evidence of carcinogenicity in human populations. Lung cancer has been associated with inhalation of arsenic, and skin, bladder, and possibly other internal cancers have been associated with ingestion of arsenic in drinking water.

In IRIS, the USEPA recommends a CSF_{oral} value for arsenic of 1.5 $(mg/kg\text{-}day)^{-1}$. This toxicity criterion is based on the incidence of skin cancer from a study of a large population (over 40,000 people) in Taiwan with chronic exposure to arsenic in drinking water and food (Tseng, 1977; Tseng *et al*., 1968). The CSF_{oral} was calculated using a multistage model, assuming a drinking water ingestion rate of 3.5 L/day for Taiwanese males and 2 L/day for Taiwanese females, an average Taiwanese body weight of 55 kg, and an average U.S. body weight of 70 kg.

There is currently considerable debate among the scientific community regarding the arsenic CSF_{oral}. A number of researchers believe that the current value of 1.5 $(mg/kg/day)^{-1}$ may overestimate cancer risks for U.S. populations (Chappell *et al*., 1997; Slayton and Beck, 2001). The key uncertainties regarding arsenic cancer toxicity are discussed in the Uncertainty Assessment in Section 5.5.

It should be noted that the toxicity of arsenic, and in particular its carcinogenicity, remain an evolving area of scientific discussion. Of particular relevance are three recent risk assessments for arsenic in drinking water: *Arsenic in Drinking Water* (NRC, 1999); the Final Rule for the arsenic MCL (USEPA, 2001c); and *Arsenic in Drinking Water: 2001 Update* (NRC, 2001). In contrast to the evaluation in IRIS that focused on skin cancer, these more recent arsenic risk assessments focus on internal cancer, especially bladder cancer. While future updates to the IRIS file for arsenic will undoubtedly consider these risks assessment, it is not possible at present to incorporate the information from these reports directly into the HHRA for several reasons. Perhaps the most important reason is that the USEPA has not yet used the information in these analyses to derive a revised CSF_{oral} for arsenic in IRIS. In addition, a single CSF_{oral} cannot be derived from the three risk assessments for several reasons. These risk assessments differ among themselves with respect to key assumptions, resulting in different risk values. For example, the USEPA, 2001 report used an internal comparison group, whereas the NRC, 2001 report used an external comparison group for its risk calculations. Even within a single document, different risk calculations are presented. For example, the NRC, 2001 report presents a range of risk calculations based

on different tumor sites, different underlying studies, and different dose-response models. Finally, there continues to be scientific debate regarding the best approach for the evaluation. Still, it is of interest to observe that all of the exposure estimates (and associated risk estimates) for CCA-treated wood in this HHRA are below the exposure and risk estimates associated with the proposed drinking water standard for arsenic, and from naturally occurring arsenic in food.

4.4.2 Arsenic UR_{inhal}

The current UR_{inhal} used for arsenic is from IRIS and is 4.3×10^{-3} $(\mu g/m^3)^{-1}$ (USEPA, 2001a). This value is based on relative risk estimates from occupational studies of lung cancer in adult male workers (Brown and Chu, 1983a,b,c; Lee-Feldstein, 1983; Higgins, 1982; and Enterline and Marsh, 1982).

4.4.3 Arsenic RfD_{oral}

The USEPA recommends an RfD_{oral} for arsenic of 3×10^{-4} mg/kg-day to quantify non-cancer risks from chronic exposure to arsenic (USEPA, 2001b). The arsenic RfD_{oral} is based on increased incidence of hyperpigmentation, keratosis, and possible vascular complications in the same Taiwanese population used as the basis for the CSF_{oral} (Tseng, 1977; Tseng *et al*., 1968). The USEPA characterized a NOAEL of 0.0008 mg/kg-day for skin lesions in the Tseng study, based on the reported drinking water concentration in the NOAEL group (0.009 mg/L), an assumed drinking water ingestion rate of 4.5 L/day, daily arsenic intake from sweet potatoes and rice of 0.002 mg/day, and an average Taiwanese body weight of 55 kg, *i.e.*, [(0.009 mg/L × 4.5 L/day) + 0.002 mg/day]/55 kg (Abernathy *et al*., 1989). An uncertainty factor of 3 (based on a lack of reproductive toxicity data and uncertainty regarding toxicity in sensitive individuals) was applied to the NOAEL to derive an RfD of 3×10^{-4} mg/kg-day (0.0008/3). Overall, the USEPA has "medium" confidence in the study, "medium" confidence in the database (due to poor characterization of the exposure levels in the Tseng and other supporting studies), and "medium" confidence in the RfD_{oral} for arsenic.

It is noted in the arsenic IRIS file that a clear consensus does not exist among Agency scientists regarding arsenic systemic toxicity (USEPA, 2001b). Solid scientific arguments can be made for values within a factor of 2 or 3 of the current recommended RfD_{oral} value (*i.e.*, 0.1 to 0.2 µg/kg-day).

4.4.4 Arsenic Subchronic RfD_{oral}

The USEPA has not provided a toxicity criterion for assessing subchronic exposures to arsenic in the IRIS database. However, USEPA's Region 8 Office (Region 8) has derived an RfD_{oral} for arsenic of 0.015 mg/kg-day (1.5×10^{-2} mg/kg-day) that addresses both acute and subchronic exposures (USEPA, Region 8, 2001). According to Region 8, the subchronic RfD_{oral} is appropriate to quantify non-cancer health risks from acute exposures lasting one to fourteen days, and subchronic exposures lasting 15 days to 7 years, to inorganic arsenic (USEPA, Region 8, 2001). Based on Region 8's criteria for use, the subchronic RfD_{oral} is used to quantify non-cancer health risks for the child receptors ages 2-6 and 7-12.

Region 8 reviewed 18 different studies where arsenic exposure was primarily *via* drinking water. Based on this review as a whole, and a study by Mazumder *et al*. (1998) in particular, Region 8 determined that the NOAEL for arsenic was 0.015 mg/kg-day. At this exposure level, signs of arsenic-related skin effects (hyperkeratosis, hyperpigmentation) were absent in children exposed to arsenic in drinking water (USEPA, Region 8, 2001). Because the NOAEL is based on a review of a large number of studies in human populations, including some involving sensitive subgroups, Region 8 determined that additional safety factors were not required in order to derive an RfD_{oral} from the NOAEL. An alternative derivation, based on applying safety factors to the LOAEL reported in the studies, yielded a similar value of 0.02 mg/kg-day. Because many of the epidemiological studies included a large number of children, the acute/subchronic RfD_{oral} would address particular concerns related to children's health. Region 8 also noted that the NOAEL (and hence the RfD_{oral}) could be as high as 0.03 to 0.04 mg/kg-day based on an evaluation of studies by Tseng (1977) and Tseng *et al*. (1968). Thus, use of an RfD_{oral} of 0.015 mg/kg-day may actually result in overestimate of non-cancer risk.

The Agency for Toxic Substances and Disease Registry (ATSDR) has also developed an acute Minimal Risk Level (MRL) for arsenic of 0.005 mg/kg-day (ATSDR, 2000). The acute MRL value addresses exposures of 14 days or less. The MRL is based on the Mizuta *et al*. (1956) study, which evaluated a Japanese population that became ill after consuming arsenic in soy sauce. The dose received by the individuals consuming the soy sauce was estimated to be 0.05 mg/kg-day. The reported health effects included gastrointestinal symptoms, skin lesions and neuropathy. Using a LOAEL of 0.05 mg/kg-day, the estimated MRL was obtained by dividing the LOAEL by an uncertainty factor of 10 (*i.e.*, 0.05/10 = 0.005 mg/kg-day). The resulting MRL of 0.005 mg/kg-day is one-third the value of the Region 8 acute/subchronic RfD_{oral}. However, because the Region 8 value was derived from a comprehensive review of the literature rather than from a single study, and because it is based on a

NOAEL rather than a LOAEL, it appears to be a more scientifically rigorous estimate and more appropriate for use in human health risk assessment.

4.4.5 Arsenic CSF_{derm}, RfD_{derm} and Subchronic RfD_{derm}

In general, for dermal exposure (expressed as absorbed dose), the CSF_{oral} and RfD_{oral} are adjusted to be applicable to absorbed doses (USEPA, 1989; 1992a). This adjustment is made assuming that once a chemical is absorbed into the blood stream the health effects are similar regardless of whether the route of exposure was oral or dermal. However, since oral absorption for arsenic is about 95% in water (USEPA, 1999a), and the USEPA recommends adjusting toxicity for the dermal route only when oral absorption is less than 50%, no adjustment was made to the CSF_{oral}, RfD_{oral} or subchronic RfD_{oral} to assess health risks from dermal exposure to both soil and dislodgeable arsenic.

4.5 Other Arsenic Toxicity Issues

As mentioned at the beginning of this chapter, a number of other issues regarding the health effects from exposure to arsenic are discussed here. The topics include children's relative sensitivity to arsenic, arsenic's purported role in endocrine disruption, and a review of occupational and animal studies to assess potential health effects from exposures to arsenic.

4.5.1 Children's Relative Sensitivity to Arsenic

The period of early childhood represents a stage of development characterized by increased sensitivity to chemical exposure (USEPA, 2000b). Children may exhibit particular behaviors (*e.g.*, hand to mouth activity) that may result in an increased dose of a chemical relative to their body weight when compared to adults. Furthermore, a child may have a different capacity to detoxify or excrete chemicals compared to an adult. Lastly, because various tissues are undergoing rapid growth during childhood, the cells of those tissues may also be particularly vulnerable to chemical damage. It is therefore important to examine whether data have indicated any special sensitivity of children to the chemical in question when evaluating children's exposure to environmental agents.

The evidence for metabolic differences in arsenic metabolism between children and adults is limited. Concha *et al*. (1998) studied a population of children in Northern Argentina exposed to high levels of arsenic in drinking water. The mean concentration of arsenic in the drinking water was 87 μg/L.

Children in one village (S.A. Cobres) had a higher percentage of inorganic arsenic in their urine than adult women, but this difference was not observed in another village (Taco Pozo) that had similar levels of arsenic in drinking water and measured in the urine (mean urinary arsenic concentration in S.A. Cobres - 323 μg/L, mean value in Taco Pozo - 400 μg/L). These data do not appear to have been evaluated statistically. One possible explanation for the difference in arsenic excretion profiles between the two populations may be ethnic background; the population in S.A. Cobres was largely indigenous while the population in Taco Pozo was predominantly of mixed European-indigenous background. A study by Kalman *et al*. (1990), examining a U.S. population, did not find age-related differences in the percentages of inorganic and organic arsenic excreted in the urine. The population studied was larger than in the Concha *et al*. study (378 children *versus* 57) and reflected a lower range of urinary arsenic levels (*i.e.*, 7.3 to 65.2 μg/L). Thus, potential differences in arsenic metabolism between adults and children, if they exist, may be influenced by ethnicity and may be limited to high dose arsenic exposures.

In addition to studies comparing differences in arsenic metabolism between adults and children, a few studies have described health effects in populations of children exposed to arsenic. Zaldivar and Guillier (1977) studied a population of children in northern Chile exposed to high levels of arsenic in drinking water. The authors indicated that the children and infants in this region showed a "much greater severity of symptoms" than adults. Approximately 340 children were found to have symptoms of arsenic toxicity, particularly dermal effects (*i.e.*, hyperkeratosis, leuko-melanoderma). A dietary survey conducted in the region was used to estimate a high daily arsenic intake of 6.3×10^{-2} mg/kg-day for children in the 0 to 10 year age range. The authors also note that five children who died in this population, apparently of arsenic toxicity, had doses approximately twice this level. By comparison, the maximum estimated dose for a child resident (ages 2-6) exposed to CCA-treated wood is approximately 6×10^{-5} mg/kg-day, which is more than 1,000 times lower than the estimated dose reported to cause health effects in the Chilean population.

Other studies have focused on specific types of health effects in children. Bencko and Symon (1977) and Bencko *et al*. (1977) studied the hearing of children living near a coal-burning power plant that burned coal containing high levels of arsenic. The mean urinary arsenic concentrations of children living in communities near the power plant ranged from 78 to 253 μg/L; the mean urinary arsenic concentration in a control community was 109 μg/L (Bencko and Symon, 1977). The authors found that urinary arsenic was associated with statistically significant decreases in hearing thresholds across a range of frequencies (Bencko *et al*., 1977). One important limitation of this study is that the possible role of

other pollutants produced by the coal burning power plant was not addressed. The individuals conducting the hearing tests and physical examinations also do not appear to have been blinded to the exposure status of the children, another potential source of bias. A study conducted by Milham (1977) evaluated hearing in a population of U.S. children attending school located less than 100 yards from a copper smelter in Tacoma, WA. Operation of the smelter had resulted in significant arsenic emissions to the local environment. No hearing deficits were found in the children compared to a control group attending a school located in another part of the city. A subset of six children with urinary arsenic levels above 200 μg/L had hearing within normal limits. As in the Bencko studies, it is unclear if individuals conducting the hearing tests were aware of the exposure status of the children.

Morse *et al*. (1979) evaluated arsenic exposures in children living near a copper smelter in southwest Arizona. Exposures appeared to be chiefly due to arsenic in airborne dust. Urinary arsenic concentrations in the children from the most exposed community (Ajo, AZ) were higher (59 μg/L) than those in the less exposed community of Gila Bend, AZ (17.8 μg/L). Changes in skin color were reported in children living in the most heavily contaminated community (Ajo) but these were not borne out by physical examinations. In fact, physical examinations did not reveal any dermatologic or neurologic abnormalities in the children. A statistically significant increase in the incidence of mottled teeth was observed in Ajo children compared to those in Gila Bend. This may have been due to elevated fluoride, which is increased in some soils containing elevated arsenic (Wyatt *et al*., 1998).

Finally, Calderon *et al*. (2001) studied the effects of arsenic and lead exposure on neuropsychological development on a group of Mexican children living near a copper and zinc smelter. Urinary arsenic levels in children living near the smelter were higher than those in a control population (mean 62.9 μg/g creatinine *vs.* 40 μg/g). There were no statistically significant differences between children living near the smelter and controls in terms of the raw IQ scores. In fact, children living near the smelter appeared to do better in IQ tests, although the parents of this group had more education and scored higher in socioeconomic status than the control population. Within the exposed group, urinary arsenic was associated with decreased verbal IQ score and several specific sub-scores. Blood lead and nutritional status were also found to be significantly associated with IQ score. Although the authors attempt to address issues of confounding by other variables, the lack of a well-matched control group limits the usefulness of the study results.

Desesso *et al*. (1998) conducted an extensive review of studies on the developmental toxicity of arsenic in both laboratory animals and humans to evaluate the potential for increased sensitivity during the pre-natal period. They concluded that studies which have indicated adverse developmental effects from arsenic exposure involved doses far above those relevant to human exposures and attainable only *via* irrelevant exposure routes (*e.g.*, intraperitoneal injection). Studies using oral dosing have not demonstrated adverse developmental effects. They concluded that, "under realistic human exposure scenarios, inorganic arsenic is unlikely to pose a threat to pregnant humans and their offspring."

As stated previously, the available scientific evidence concerning the potential for increased sensitivity of children (compared to adults) to the toxicological effects of arsenic is quite limited. Children do not appear to absorb arsenic more readily than adults *via* the GI tract (ATSDR, 2000). Although one study has suggested that children are less effective at metabolizing arsenic than adults, this has not been corroborated by other studies. If there is a metabolic difference due to age, it may only be important at high levels of exposure, and likely would not be of concern in the case of children exposed to the low levels of arsenic associated with CCA-treated wood. Some studies have indicated adverse effects on central nervous system development (Bencko and Symon, 1977; Bencko *et al*., 1977; Calderon *et al*., 2001). However, the potential contribution of other chemicals in the exposures evaluated in these studies was not ruled-out.

Evidence that children are not more sensitive to the toxic effects of arsenic compared to adults can be found in the studies that Region 8 used to derive the subchronic RfD_{oral} for arsenic (Section 4.4.4). These studies, which involved a large number of children, evaluated the same health endpoints used to derive the current chronic RfD_{oral}, and were specifically used by Region 8 to address the particular health concerns related to children; resulted in a subchronic RfD_{oral} for arsenic that is 50-times greater than the chronic RfD_{oral}. The chronic RfD_{oral} for arsenic is based on health effects that were observed primarily in adult men and women. Based on the review of several studies in the peer-reviewed literature, a special sensitivity of children to the toxicity of arsenic has not been clearly demonstrated.

4.5.2 Arsenic's Purported Role in Endocrine Disruption

The endocrine system is comprised of a group of glands and hormones that control a diverse array of bodily functions, from basic metabolism to reproduction. Some individuals have speculated that certain environmental chemicals (primarily organic chemicals) have the potential to disrupt normal endocrine function (Colborn *et al*., 1993). It is important to note that many naturally occurring chemicals

(particularly those which occur naturally in certain vegetables) display some endocrine activity. For example, both broccoli and cabbage contain the chemical indole-3-carbinole that has properties similar to the hormone estrogen (Liu *et al*., 1994). Studies of these alleged endocrine disruptors have typically shown that both naturally occurring and industrially-derived chemicals have activities that are several orders of magnitude weaker than the hormones they mimic (Shelby *et al*., 1996; Jobling *et al*., 1995; Safe, 2000). Thus, while endocrine disruption has attracted much public concern, scientific studies have not been able to convincingly show any likely adverse effect of low-level exposures to chemicals with endocrine activity (Safe, 2000).

Several studies (Lai *et al*., 1994; Rahman *et al*., 1998; Tseng *et al*., 2000) have observed statistically significant increases in rates of diabetes mellitus in residents of localities with elevated arsenic exposure. Diabetes mellitus (*i.e.*, diabetes) is a disorder of the system regulating blood glucose (*i.e.*, blood sugar), a system controlled in part by glucocorticoid hormones. Arsenite has been shown to inhibit binding to the glucocorticoid hormone receptor (Lopez *et al*., 1990; Kaltreider *et al*., 2001), although other hormone receptors (*e.g.*, the estrogen receptor) were unaffected (Lopez *et al*., 1990). The Lopez *et al*. study examined isolated receptor molecules in solution and the Kaltreider study employed rat cancer cells modified with a reporter protein construct, a piece of foreign DNA. Thus, the cells and isolated molecules employed in these studies bear little resemblance to cells inside a human liver. It is also unclear how the doses used in the Kaltreider and Lopez studies (single doses at micromolar concentration in culture media) would compare to actual exposure levels of cells in individuals (chronic doses distributed to cells *via* the GI tract and blood). Thus, additional work is required in order to demonstrate that arsenite affects the endocrine system *via* the glucocorticoid receptor. No studies could be located that discussed the effects of arsenate on the glucocorticoid receptor.

The potential effects of arsenic on the female reproductive system have recently been studied by one group of researchers in India. Chattopadhyay *et al*. (1999) examined the effects of arsenic (sodium arsenite) exposure on circulating hormone levels in rats. Female rats treated with 0.4 mg/L sodium arsenite in drinking water for four weeks had altered levels of luteinizing, follicle stimulating, and estrogen hormones. These animals also had decreased reproductive organ weights compared to controls. A subsequent study (Chattopadhyay *et al*., 2001) revealed that these effects could be prevented by simultaneous administration of ascorbate (vitamin C). The studies by Chattopadhyay *et al*. are apparently the only studies investigating the effects of arsenite exposure on female reproduction. The doses the animals received in this study, approximately 0.025 mg/kg-day, are over 400 times higher than the maximum dose of arsenic calculated for a child resident contacting CCA-treated wood.

Sarkar *et al*. (1991) studied the effects of sodium arsenite on the male reproductive system in rats. They observed that doses of 6 and 8 mg/kg-day for 7 or 13 days inhibited the activity of two key enzymes in testicular hormone metabolism (Δ^5-3β-hydroxysteroid dehydrogenase and 17β-hydroxysteroid dehydrogenase) and decreased weight of the accessory sex organs. However, a dose of 4 mg/kg did not have any effect on enzyme activity or sex organ weights. This dose is over 67,000 times greater than the maximum dose of arsenic calculated for a child resident contacting CCA-treated wood.

In summary, although a few studies have shown some endocrine-related effects of arsenic exposure, these effects occurred in an *in vitro* model of uncertain relevance, and at doses substantially higher than those that have been conservatively estimated for CCA-treated wood exposures. It should also be noted that the form or arsenic used in these reproductive studies, *i.e.*, arsenite, is a different and more toxic form of arsenic than the chromium arsenate in CCA-treated wood. Based on these conclusions, there is no convincing evidence that endocrine-related effects arising from exposure to arsenic in CCA-treated wood are likely.

4.5.3 Health Effects from Exposure to Arsenic in CCA-Treated Wood

Gradient reviewed the available animal toxicity and human occupational studies regarding exposure to arsenic associated with CCA-treated wood. Most of the available animal studies involved short-term oral or dermal exposures to sawdust from CCA-treated wood. The results of these animal studies are discussed below and summarized in Table D-1 in Appendix D. Overall, there was no evidence of any adverse health effects in the majority of the animal studies, despite arsenic exposure levels ranging from 0.4 to 130 mg/kg body weight, which is orders of magnitude greater than the maximum dose levels estimated for children (ages 2-6) exposed to dislodgeable arsenic on the surface of CCA-treated wood.

Six of the animal toxicity studies reviewed involved either oral or dermal exposures to CCA-treated sawdust in rats, dogs, mice, and rabbits. No adverse health effects were observed in these studies. For example, Hood (1979) fed pregnant mice (n=27) a diet containing 10% CCA-treated sawdust on gestation days 1-18, and found no adverse health effects in the developing mice or their mothers. Hood also found no evidence of teratogenicity or maternal toxicity in rabbits (n=17) with dermal exposures to CCA-treated sawdust on gestation days 7-20 (Hood, 1979). Graham (1979) found no signs of toxicity, illness, or chromosome damage in mice with exposures to CCA-treated sawdust (either 10% CCA-treated sawdust in food, or 10 mg/kg-day *via* gavage) for 21 days. In most of the animal toxicity studies

reviewed, the oral dose levels were greater than 1 mg/kg-day, which is nearly 17,000 times greater than the maximum estimated dose (*i.e.*, ~ 6 x 10^{-5} mg/kg-day) of arsenic for a child resident (ages 2-6) from ingestion and dermal exposure to dislodgeable arsenic on the surface of treated wood.

The only animal study reporting adverse health effects did not involve CCA or CCA-treated wood specifically. Mason and Edwards (1989) injected rats with high (up to 90 mg/kg) doses of arsenic, chromium, and copper; either alone or in combination, to study the possible combined effects of these three components of CCA. A dose of 90 mg/kg-day is approximately 1.5 million times greater than the maximum estimated dose of arsenic for a child resident exposed to dislodgeable arsenic on treated wood. Although severe acute toxicity, including increased mortality, was observed in some of the exposure groups in the injection study, the observed health effects are not relevant for typical human exposures because of the injection route of exposure and the high dose levels used.

The occupational studies reviewed involved workers at CCA wood treatment plants, carpenters, and other wood workers with daily exposure to CCA-treated wood. These studies are discussed below and are summarized in Table D-2 in Appendix D. Many of the wood treatment plant studies reported airborne arsenic concentrations at the facility (either from personal monitors or area samples), and/or urinary arsenic levels in the workers. Worker exposures were typically not estimated in terms of mg/kg body weight, but instead were evaluated based on arsenic air concentrations, urinary arsenic levels, and descriptions of the workers' daily contact with CCA formulation and CCA-treated wood.

The most important occupational studies (summarized below) involved comprehensive medical exams or reviews of medical records to assess acute and chronic health endpoints for fairly large groups of workers.

- Budy and Rashad (1977) reviewed the death records for two groups of Hawaiian carpenters, one group working before arsenical wood preservatives were introduced (n=232) and one after (n=293), and found no association between exposure to treated wood and health risks.

- Flickinger and Lawrence (1982) conducted an occupational health study of 109 workers from two wood preserving plants, and found no evidence of increased cancer or other diseases of the lung, liver, kidneys or skin. The mean airborne arsenic concentration, based on 70 personal air samples from these workers, was 1.07 $\mu g/m^3$ and ranged as high as 15 $\mu g/m^3$. (The lower of these concentrations is over 3000 times higher than the maximum concentration of respirable soil arsenic particulate estimated for the residential exposure scenario in the HHRA).

- Gilbert *et al*. (1990) studied 88 wood-treating workers in Hawaii, and reported no significant differences between the workers and the comparison group based on medical histories and physical examinations. The worker urine arsenic levels (mean = 126 ppb) were not significantly elevated compared to controls (mean = 148 ppb). These workers had been employed in the wood treating industry for 0.33 to 26.3 years with a median of 6.5 years.

- Rosenberg *et al*. (1980) found no significant differences in the medical records or physical exams for 44 workers at wood processing plants compared to controls, despite the fact that urine arsenic levels were increased for the exposed workers (mean = 63 µg/L). The average exposure duration for the workers was 6.6 years.

- Tabershaw Occupational Medicine Associates (1979) performed comprehensive medical exams on 63 workers at a CCA wood preserving plant and found no evidence of lung cancer, liver or kidney disease, blood abnormalities or skin disease. Over one third of the employees had worked at the facility for six years or more. Measured airborne arsenic concentrations in personal air samples ranged from 0.3 to 5.2 $\mu g/m^3$. (The lower end of this concentration range is approximately 850 times higher than the maximum concentration of respirable soil arsenic particulate estimated for the residential exposure scenario in the HHRA).

It should be noted that there are some limitations in the toxicological and epidemiological studies involving CCA-treated wood, such as the short exposure duration in the animal studies and the fact that the worker studies involved inhalation exposure rather than the oral and dermal pathways that are of primary concern for CCA-treated decks and play structures. Nonetheless, considering the consistency of the studies in multiple species, the available toxicity data are consistent with a reduced toxicity for arsenic as found in CCA-treated wood.

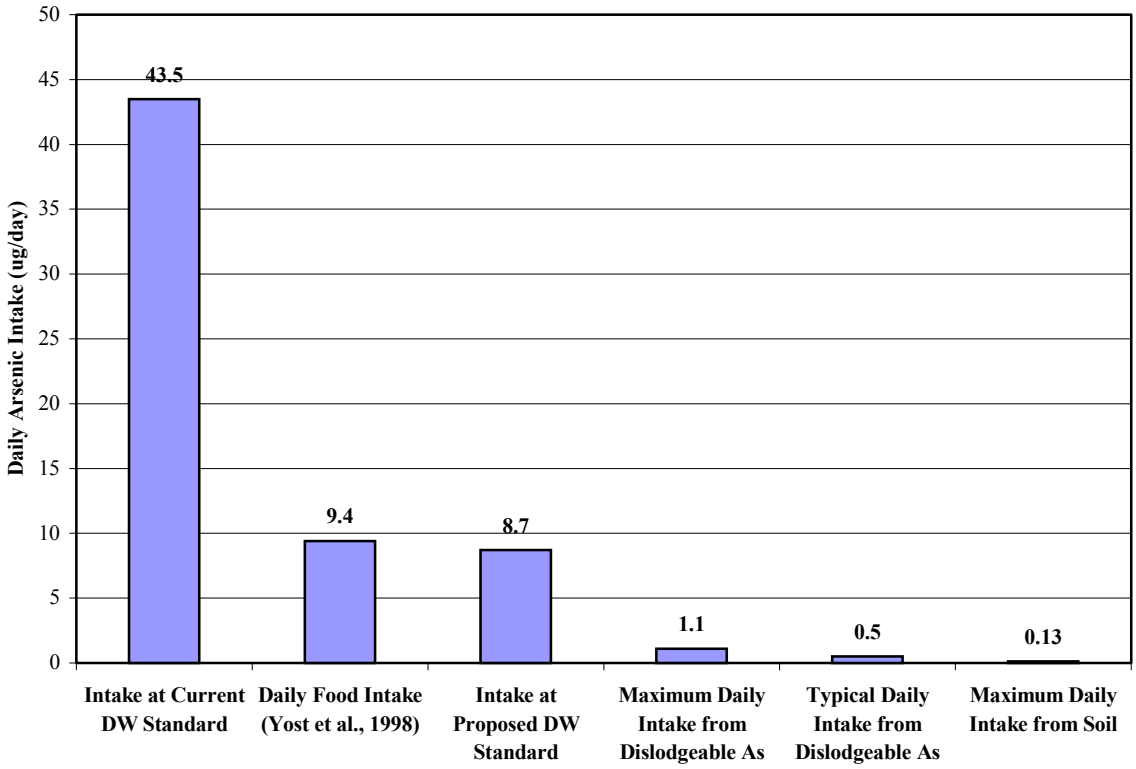
In addition to the animal and epidemiological studies discussed above, Gradient also reviewed three injury claims alleging arsenic poisoning from exposure to CCA-treated wood. The claims include a contractor asserting health problems due to CCA-treated wood splinters in his leg, a school teacher claiming neurological difficulties after building a deck made of CCA-treated wood, and a government employee who experienced internal bleeding after building picnic tables constructed with CCA-treated wood. Based on a review of each of these cases and the corresponding medical records, none of the alleged health problems experienced by these individuals appears to be attributable to arsenic poisoning. Refer to Appendix D for a more detailed description of each of these health claims and the corresponding medical diagnoses.

5 Exposure and Risk Characterization

5.1 Exposure Comparison

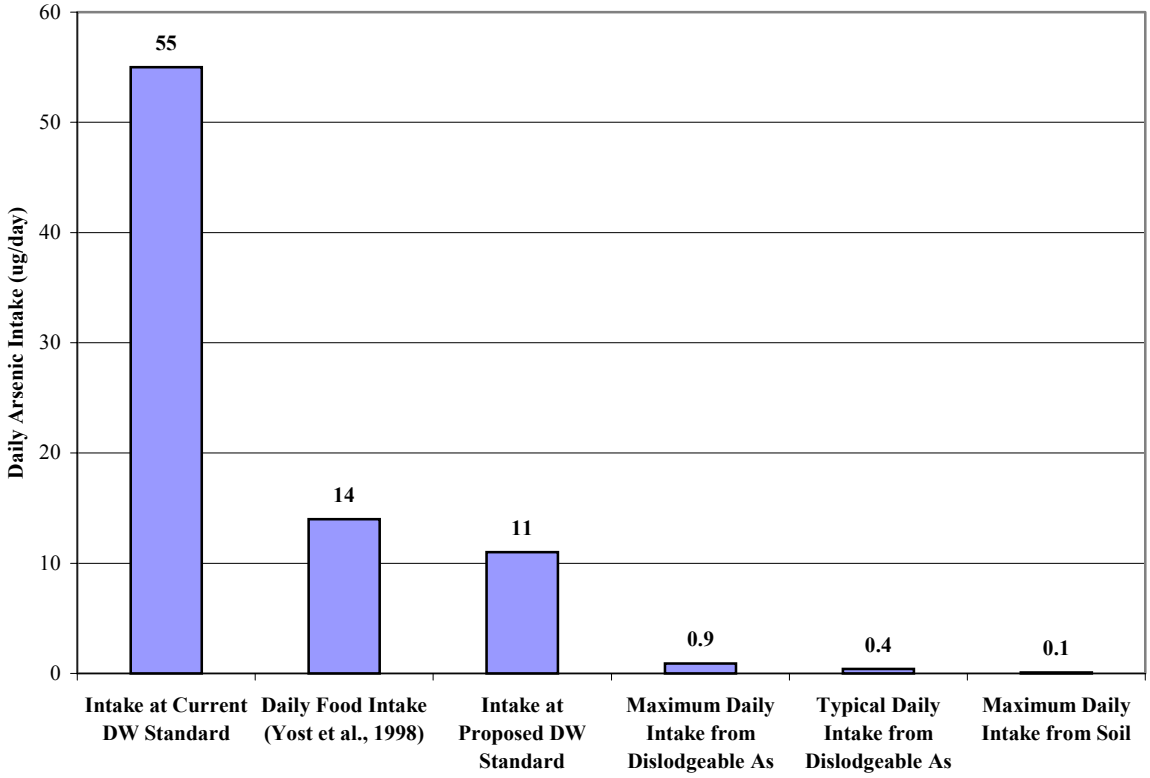
In order to put exposure to arsenic associated with CCA-treated wood into perspective, a comparison is made in Figure 5-1 between the daily intake of dislodgeable and soil arsenic for a child resident, ages 2-6, and the daily intake of inorganic arsenic based on the current and proposed federal drinking water standards for arsenic, and the typical U.S. diet. Figure 5-2 contains the same type of comparison for the child and adult resident ages 2-31.

Figure 5-1
Comparison of Inorganic Arsenic Doses for Child Resident Ages 2-6



Notes:
1) Arsenic intakes from drinking water are based on the current (50 µg/L) and proposed (10 µg/L) federal drinking water standards for arsenic, and were calculated using USEPA-recommended mean drinking water intake rates for children ages 3-5 (0.87 L/day); and a time-weighted average of drinking water intake rates for a child and adult ages 2-31 years (1.1 L/day) (USEPA, 1997a).
2) Dietary intake of inorganic arsenic for the child receptor ages 2-6 is based on the diet for a child ages 6 months – 2 years. This intake rate will likely underestimate the actual dietary intake of inorganic arsenic for the child receptor in the HHRA.
3) The maximum dislodgeable arsenic intake is based on the wood type (*i.e.*, CCA-Southern Pine with pressure-applied water repellent) resulting in the highest daily dose of arsenic from incidental ingestion and dermal contact.
4) The typical dislodgeable arsenic intake is based on the wood type (*i.e.*, CCA-treated Southern Pine) most commonly used in the U.S. (AWPA, 1998).
5) Soil arsenic intake is based on ingestion and dermal exposure to arsenic in soil.

Figure 5-2
Comparison of Inorganic Arsenic Doses for Child and Adult Resident Ages 2-31



Notes:
1) Arsenic intakes from drinking water are based on the current (50 µg/L) and proposed (10 µg/L) federal drinking water standards for arsenic, and were calculated using USEPA-recommended mean drinking water intake rates for children ages 3-5 (0.87 L/day); and a time-weighted average of drinking water intake rates for a child and adult ages 2-31 years (1.1 L/day) (USEPA, 1997a).
2) Dietary intake of inorganic arsenic for the child receptor ages 2-6 is based on the diet for a child ages 6 months – 2 years. This intake rate will likely underestimate the actual dietary intake of inorganic arsenic for the child receptor in the HHRA.
3) The maximum dislodgeable arsenic intake is based on the wood type (*i.e.*, CCA-Southern Pine with pressure-applied water repellent) resulting in the highest daily dose of arsenic from incidental ingestion and dermal contact.
4) The typical dislodgeable arsenic intake is based on the wood type (*i.e.*, CCA-treated Southern Pine) most commonly used in the U.S. (AWPA, 1998).
5) Soil arsenic intake is based on ingestion and dermal exposure to arsenic in soil.

These graphs indicate that even the maximum estimates of daily inorganic arsenic intake from CCA-treated wood and impacted soil, for both of the residential receptors evaluated in the HHRA, are significantly less than the daily intake of inorganic arsenic from the typical U.S. diet and from drinking tap water at the current and proposed federal drinking water standards for arsenic. The daily intake of inorganic arsenic for children at a playground will be even less than for residents because based on the USEPA's *Exposure Factors Handbook* (USEPA, 1997b), children spend more time outdoors at home than

they do at a playground. Therefore, children at a playground will have less exposure to dislodgeable and soil arsenic than a child resident.

5.2 Calculation of Cancer Risks

Cancer risks are characterized as the incremental probability that an individual will develop cancer during his or her lifetime due to chemical exposure under the specific exposure scenarios evaluated in the HHRA. The term "incremental" implies the risk above the background cancer risk experienced by all individuals in the course of daily life. Approximately one in four Americans die of cancer, so the background cancer risk is 0.25, or 250,000 in one million (ACS, 2000). The incremental risk is a measure of the additional estimated cancer risk due to a specific exposure. Cancer risks are expressed as a unitless probability (*e.g.*, one in a million, or 1 x 10^{-6}) of an individual developing cancer over a lifetime, above background risk, as a result of exposure to arsenic associated with CCA-treated wood.

Excess (incremental) cancer risks for the exposure pathways (oral ingestion, dermal absorption, and inhalation) evaluated in the HHRA are calculated using intake estimates (lifetime average daily doses), calculated in Section 3 as part of the Exposure Assessment, and cancer slope factors (CSFs) (*e.g.*, CSF_{oral}), which were described in Section 4 – Toxicity Assessment. Estimated intakes and CSFs are combined to calculate excess cancer risk according to the following equation (USEPA, 1989):

$$Cancer\ Risk = Intake\left(\frac{mg}{kg \cdot day}\right) \times CSF\left(\frac{mg}{kg \cdot day}\right)^{-1}$$

For the ingestion exposure route, the estimated oral intake (expressed as applied or administered dose) is multiplied by the CSF_{oral} (applicable to applied/administered dose). For inhalation exposure to arsenic, the estimated exposure point concentration ($\mu g/m^3$) is multiplied by the UR_{inhal} to quantify risk. To quantify risk from dermal exposure, the estimated dermal intake (expressed as an absorbed dose) is multiplied by the CSF_{oral}. Depending on the absorption of a chemical in toxicity studies, the CSF_{oral} may be adjusted so that it is applicable to an absorbed dose. However, because the absorption of arsenic in water in the gastrointestinal (GI) tract under experimental conditions is approximately 95%, no adjustment is made to the CSF_{oral} (USEPA, 1999a).

The estimated cancer risk for the resident receptor ages 2-31 is the sum of the cancer risks for the 2-6 year old and 7-31 year old resident receptors. Cancer risk estimates for the 2-31 year old receptor were calculated separately for each of the exposure routes evaluated in the HHRA.

5.2.1 Summary of Cancer Risks

Since the HHRA was prepared in accordance with current USEPA risk assessment guidelines, the estimated cancer risks are compared to the USEPA's acceptable cancer risk range of 1×10^{-6} to 1×10^{-4} (USEPA, 1991b). A cancer risk of 1×10^{-6} represents 1 case of cancer in every 1,000,000 population and a cancer risk of 1×10^{-4} represents 1 case of cancer in every 10,000 population (Section 5.4, below, contains a discussion of how the USEPA applies cancer risk limits to manage risk).

The child receptor with the greatest amount of exposure, and therefore risk, to both dislodgeable and soil arsenic is the child resident ages 2-6. The maximum estimated lifetime cancer risk for this receptor is 7.1×10^{-6} based on RME parameters, and 2.2×10^{-6} based on CTE parameters. These cancer risk estimates represent approximately 7 and 2 cases of cancer, respectively, in every 1,000,000 population and are within the USEPA's acceptable cancer risk range. These risk estimates include exposure to soil arsenic and the treated wood type (*i.e.*, Southern Pine with pressure-applied water repellent) that resulted in the greatest exposure to dislodgeable arsenic *via* ingestion and dermal contact. It should be noted that pressure-applied water repellent is a wood treatment process that is performed by the manufacturer and is not the same as the application of water repellent performed by consumers. Furthermore, Southern Pine with pressure-applied water repellent accounts for only about 6% of the treated lumber sold in the U.S. (RISI, 1990). CCA-treated Southern Pine without pressure-applied water repellent accounts for approximately 86% of the treated wood sold in the U.S. (AWPA, 1998) and the estimated cancer risk for the child resident for exposure to this wood type (including exposure to soil arsenic) is 3.8×10^{-6} based on RME parameters and 1.1×10^{-6} based on CTE parameters.

The receptor with the greatest amount of exposure and risk overall is the child and adult resident ages 2-31. The maximum estimated lifetime cancer risk for this receptor is 1.2×10^{-5} based on RME parameters, and 5.3×10^{-6} based on CTE parameters. The RME cancer risk estimate represents approximately 1 case of cancer in every 100,000 population, and the CTE cancer risk estimate represents approximately 5 cases of cancer in every 1,000,000 population. Both of these cancer risk estimates are within the USEPA's acceptable cancer risk range. These risk estimates include exposure to soil arsenic and Southern Pine with pressure-applied water repellent. The estimated cancer risk for this receptor is 6.6

x 10^{-6} based on RME parameters and 2.6 x 10^{-6} based on CTE parameters for Southern Pine without pressure-applied water repellent, and including exposure to soil arsenic.

The playground receptor with the greatest amount of exposure and risk is the child ages 2-6. The maximum estimated lifetime cancer risk for this receptor is 3.4 x 10^{-6} based on RME parameters, and 1.2 x 10^{-6} based on CTE parameters. These cancer risk estimates represent approximately 3 and 1 cases of cancer, respectively, in every 1,000,000 population and are both within the USEPA's acceptable cancer risk range. These risk estimates include exposure to soil arsenic and Southern Pine with pressure-applied water repellent. The estimated cancer risks for exposures to Southern Pine, including exposures to soil arsenic, are 1.6 x 10^{-6} based on RME parameters and 5.7 x 10^{-7} based on CTE parameters

Approximately 80-90% of the estimated cancer risks, based on either RME or CTE parameters, is attributable to dislodgeable arsenic exposures *via* incidental ingestion and dermal contact. Risks based on RME parameters represent upper-limit risks, and yet the estimated RME cancer risk for the receptor with the greatest amount of exposure and risk overall (*i.e.*, the child and adult resident ages 2-31) is 1.2 x 10^{-5}, which is within the USEPA's acceptable risk range. However, as discussed in more detail in the Uncertainty Assessment in Section 5.5, because there are considerable uncertainties associated with the estimated cancer risks, conservative assumptions have been made throughout the HHRA and actual cancer risks are probably lower.

The exposure parameters and equations used to quantify cancer risks are detailed in the risk calculation worksheets in Appendix E, which is split into two sections. The first section in Appendix E contains the worksheets used to calculate cancer and non-cancer risks from exposures to dislodgeable arsenic. The second section in Appendix E contains the worksheets used to calculate cancer and non-cancer risks from exposures to soil arsenic.

5.3 Calculation of Non-Cancer Risks

Non-cancer health risks are expressed as hazard quotients rather than as probabilities. A hazard quotient compares the estimated daily exposure or average daily dose of a chemical calculated as part of the Exposure Assessment in Section 3, to an acceptable Reference Dose (RfD) derived by the USEPA and described in Section 4 – Toxicity Assessment. The hazard quotient is calculated using an RfD according to the following equation (USEPA, 1989):

$$Hazard\,Quotient = \frac{Intake\left(\frac{mg}{kg \cdot day}\right)}{RfD\left(\frac{mg}{kg \cdot day}\right)}$$

For the ingestion exposure route, an estimated oral intake (expressed as applied or administered dose) is divided by an RfD_{oral} (applicable to applied/administered dose). To quantify risk from dermal exposure, the estimated dermal intake (expressed as an absorbed dose) is also divided by the RfD_{oral}. Depending on the absorption of a chemical in toxicity studies, the RfD_{oral} may be adjusted so that it is applicable to an absorbed dose. However, as discussed previously, because the absorption of arsenic in water in the GI tract under experimental conditions is approximately 95%, no adjustment is made to the RfD_{oral} (USEPA, 1999a). There is no USEPA-recommended RfD or Reference Concentration (RfC) available to quantify inhalation exposure to arsenic (USEPA, 2001b); therefore, this exposure route was not assessed for non-cancer risk in the HHRA.

In accordance with USEPA risk assessment guidance, hazard quotients are calculated for each receptor and exposure route, and then summed across the different exposure routes to calculate a hazard index (USEPA, 1989). Because a hazard quotient is simply a ratio of estimated exposures to reference exposure levels (*e.g.*, RfD_{oral}), hazard indices do not represent the probability that an adverse health effect may occur. Instead, a hazard index indicates whether estimated exposures for an individual present a potentially significant non-cancer health risk based on a comparison to a USEPA-recommended RfD.

Cumulative non-cancer health risks are the sum of the estimated risks for each exposure route (*e.g.*, incidental ingestion, dermal contact, *etc.*). Non-cancer risks for the resident receptor ages 2-31 are based on a combined time-weighted adjustment of the dose for the resident receptors ages 2-6 and 7-31. Refer to Worksheet E-3 in Appendix E for a description of the dose adjustment used to quantify non-cancer risks for the resident receptor ages 2-31.

5.3.1 Summary of Non-Cancer Risks

According to USEPA risk assessment guidance, if the cumulative hazard index is less than 1.0, then no further evaluation of non-cancer risks is necessary (USEPA, 1989). The maximum cumulative (*i.e.*, including all exposures to soil and dislodgeable arsenic) hazard index for the receptor with the greatest amount of exposure and risk overall (*i.e.*, the resident receptor ages 2-31) is 6.4×10^{-2} (0.064)

based on RME parameters and 2.7 x 10^{-2} (0.027) based on CTE parameters. These risk estimates include exposures to Southern Pine with pressure-applied water repellent. The cumulative non-cancer risks for Southern Pine without pressure-applied water repellent are even lower. Because the overall maximum risk estimate based on RME parameters is well below the USEPA's acceptable non-cancer risk limit of 1.0, no further discussion of the estimated non-cancer risks for the other exposure scenarios was deemed necessary.

Although these risks are negligible, approximately 80-90% of the estimated non-cancer risks, based on either RME or CTE parameters, are attributable to dislodgeable arsenic exposures *via* incidental ingestion and dermal contact. Risks based on RME parameters represent upper-limit risks, and yet the estimated RME non-cancer risk for the receptor with the greatest amount of exposure and risk overall (*i.e.*, the child and adult resident ages 2-31) is only 0.064, which is well below the USEPA's acceptable risk limit of 1.0. However, as discussed in more detail in the Uncertainty Assessment in Section 5.5, because there are considerable uncertainties associated with the estimated non-cancer risks, conservative assumptions have been made throughout the HHRA and actual risks are probably even lower.

The exposure parameters and equations used to quantify non-cancer risks are detailed in the risk calculation worksheets in Appendix E, which is split into two sections. The first section in Appendix E contains the worksheets used to calculate cancer and non-cancer risks from exposures to dislodgeable arsenic. The second section in Appendix E contains the worksheets used to calculate cancer and non-cancer risks from exposures to soil arsenic.

5.4 The Use of Cancer Risk Targets in Risk Management Decisions

This section of the report discusses the issue of significant risk in the regulation of chemical exposures. It is included to provide some perspective on the estimated risks from exposures to arsenic associated with CCA-treated wood.

The definition of an acceptable cancer risk in the federal government is not a single precise value, but rather a range of values that allows the selection of an acceptable risk within this range based on a number of considerations. The USEPA has established an "acceptable cancer risk range" of 1 x 10^{-6} to 1 x 10^{-4} and exposures to chemicals are regulated so that estimated risks are within this acceptable range. However, based on a review of the published literature and several federal regulatory decisions, it is clear

that cancer risks associated with USEPA-approved site remediations, and air and drinking water standards often exceed this range.

In 1973 the FDA initially adopted 1×10^{-8} or 1 case of cancer in every 100,000,000 population as a *de minimus* or negligible risk level to be used in the regulation of additives in the food supply (Kelly and Cardon, 1991). This risk limit was later changed to 1×10^{-6} when the final rule was issued in the Federal Register in 1977 and meant that the concentration of an additive in food that resulted in a lifetime cancer risk below 1×10^{-6} did not require additional regulation by the FDA because the Agency considered this level of risk "essentially zero" (Kelly and Cardon, 1991). Thus, 1×10^{-6} was established as the lifetime cancer risk below which no further regulatory action is warranted. This risk limit was subsequently adopted by other federal agencies and essentially used as an "acceptable" level of risk; however, this was not the intent of the FDA regulators when the legislation was drafted (Kelly and Cardon, 1991).

The 1990 National Contingency Plan (NCP), which is an environmental guidance document prepared by the USEPA, contains language indicating that that remediation of hazardous waste sites should be managed so that concentrations of chemicals remaining in soil are associated with cancer risks within a range of 1×10^{-6} to 1×10^{-4} (USEPA, 1990b). The NCP states that risks within this range are "generally acceptable" and that risks greater than 1×10^{-4} may be permitted depending on site-specific considerations. This risk range soon became policy for the Agency, as evidenced in an April 1991 memo from the Assistant Administrator to the Directors of the Waste Management, Emergency and Remedial Response, and Hazardous Waste Divisions in several regional offices (USEPA, 1991b). The memo states that cumulative cancer risks up to 1×10^{-4} can be used to develop remedial alternatives for Superfund sites and in risk management decisions, that remediation would not typically be required at a site if risks associated with reasonable maximum exposure (RME) parameters were 1×10^{-4} or less, and that in certain cases the Agency "may consider risk estimates slightly greater than 1×10^{-4} to be protective" (USEPA, 1991b).

Thus, 1×10^{-6} to 1×10^{-4} was established as an acceptable cancer risk range for Agency decisions regarding hazardous waste site cleanups and leaves open the possibility of even higher risks. For example, in the 1993 Record of Decision (ROD) for Commencement Bay in Tacoma, Washington (Operable Unit 04, Ruston/North Tacoma Study Area), a remediation action level for soil arsenic of 230 mg/kg was established based on a lifetime cancer risk of 5×10^{-4} (USEPA, Region 10, 1993). And a USEPA memorandum from the Office of Solid Waste and Emergency Response (OSWER), regarding

cleanup goals (CUGs) for dioxin and related compounds or dioxin toxicity equivalents (TEQs) in soil at Superfund and RCRA sites, states that the recommended residential CUG for dioxin/TEQs corresponds to a 2.5 x 10^{-4} lifetime cancer risk (USEPA, 1998).

Based on a review of 132 regulatory actions taken by the USEPA and other federal agencies over the years, Travis *et al*. (1987) concluded that the USEPA considers 1 x 10^{-4} as the *de minimis* risk level for small populations and that risks higher than this are acceptable if the affected population is small. For example, the authors provide several examples where the USEPA did not regulate estimated cancer risks as high as 6 x 10^{-4} because the total incidence of cancer, even on a national scale for the type of exposure in question (*e.g.*, chemical manufacturing), would be less than the incidence of cancer attributable to smoking and diet (Travis *et al*., 1987).

The USEPA has also applied an acceptable risk range to set emission standards for the Clean Air Act (CAA), and to establish Maximum Contaminant Levels (MCLs) for the Safe Drinking Water Act (SDWA). The emission standards for vinyl chloride and benzene from stationary sources, both known human carcinogens, are based on an upper-bound cancer risk limit of 1 x 10^{-4} for the maximally exposed individual (Sadowitz and Graham, 1995). The USEPA's policy for establishing MCLs, which are enforceable standards for chemicals in drinking water, is to base the standards on the smallest detectable quantity using available analytical methods. However, regardless of the technical obstacles with this policy, the Office of Drinking Water tries to ensure that the standards for carcinogens do not exceed the 1 x 10^{-6} to 1 x 10^{-4} range (Rosenthal *et al*., 1992). However, MCLs for arsenic (at 10 μg/L), vinyl chloride, and ethylene dibromide are associated with excess lifetime cancer risks between 1 x 10^{-4} and 1 x 10^{-3} (Rosenthal *et al*., 1992).

There is no bright-line that the USEPA uses to establish acceptable cancer risks. Based on the review of several regulatory decisions in different programs that the Agency is responsible to enforce, it appears that what constitutes an acceptable risk is determined on a case-by-case basis and several different factors are taken into consideration. However, regardless of the criteria the USEPA uses to manage risk, the maximum estimated cancer risk for the receptor with the highest overall exposures in the HHRA (*i.e.*, the child and adult resident ages 2-31) is 1.2 x 10^{-5}, which is within the USEPA's acceptable cancer risk range and below the risk level associated with Agency-regulated exposures to air, water, and several hazardous chemicals.

5.5 Uncertainty Assessment

The process of evaluating human health risks from exposure to environmental media involves multiple steps. Inherent in each step of the process are uncertainties that ultimately affect the final risk estimates. Uncertainties may exist in numerous areas, including the collection of samples used to identify contaminants, laboratory analysis of samples, estimation of potential exposures, and derivation of toxicity criteria. These uncertainties may result in either an over- or under-estimation of risks. However, for this HHRA, where uncertainties existed, a conservative was taken approach where appropriate so as to overestimate rather than underestimate potential exposures and risks.

Some of the uncertainties have been mentioned in the preceding report sections. Below is a discussion of the significant sources of uncertainty in the HHRA and the choices made in each of the four risk assessment steps (*i.e.*, Identification of Constituents of Concern, Exposure Assessment, Toxicity Assessment, and Risk Characterization). Other sources of uncertainty exist than those evaluated here; however, their impact on the estimated cumulative risks are comparatively insignificant.

5.5.1 Identification of Constituents of Concern

Gradient evaluated potential exposure and risk only to arsenic in the CCA complex of metals because most of the potential subchronic and chronic health risks associated with CCA-treated wood are from exposures to arsenic. Based on the toxicity of the other two metals in CCA, *i.e.*, copper and trivalent chromium, both of which are non-carcinogens according to the USEPA, their inclusion in the HHRA would not significantly affect the estimated non-cancer risks. As discussed in the beginning of Section 3.0 in the report, the chemical species of chromium in CCA-treated wood is based on the CCA treatment process where hexavalent chromium used in the CCA formulation is reduced to trivalent chromium when the complex of metals is "fixed" to the wood (Bull, 2000, 2001). Therefore, the species of chromium in dislodgeable material is most likely trivalent chromium. Furthermore, to the extent that minute quantities of non-reduced hexavalent chromium in dislodgeable material exists, hexavalent chromium has been shown to be non-carcinogenic *via* the oral route of exposure based on a report prepared by CAEPA (2001). Therefore, potential incidental ingestion of hexavalent chromium is unlikely to pose a cancer risk.

5.5.2 Exposure Assessment

Exposure Scenario. The playground scenario is included in the HHRA to evaluate exposures and risks for children exposed to dislodgeable and soil arsenic while playing on and around a CCA-treated play structure. The residential scenario also involves the use of a CCA-treated structure to evaluate exposures to dislodgeable and soil arsenic. However, the choice of structure in the residential scenario is not important, it could be a deck, a play structure, or dock. The assumptions and parameters used throughout the evaluation of exposure and risk for this scenario are applicable to either a treated deck or a play structure.

Exposure Point Concentrations

Dislodgeable Arsenic

It is conservatively assumed that the amount of dislodgeable arsenic on the surface of CCA-treated wood remains constant over time. Studies of dislodgeable arsenic from fresh and weathered treated wood indicate otherwise, however. Weathered treated wood yields less dislodgeable arsenic than freshly treated wood (SCS, 1998; Solomon and Warner, 1989). In addition, based on the results of leaching studies (described below), where samples of CCA-treated wood of various size were extracted with aqueous solutions at different pH's over time, the amount of releasable arsenic in the treated wood decreases over time. A conservative estimate of the amount of dislodgeable arsenic from aged wood compared to freshly treated wood is approximately 20%, based on the results of leaching studies. This reduction in the amount of dislodgeable arsenic would substantially decrease the EPCs used to quantify exposure and risk for each of the wood samples evaluated, except for the two wood samples that were already aged 5 years.

Evans observed that about 20% of the arsenic from the outer 5 millimeters (mm) of treated poles was lost in the first few months when kept under running water, after which no further depletion of arsenic in the wood was detected, even after 10 years under these conditions (Evans, 1978). In addition, studies that have examined the effect of weathering on the amount of dislodgeable arsenic have found that as the wood ages, the amount of dislodgeable arsenic decreases (SCS, 1998; Solomon and Warner, 1989). In the SCS study, 5-year old treated wood yielded approximately 20% the amount of arsenic compared to fresh wood, when sampled with Kimwipes. In the Solomon and Warner study, wood aged for one year yielded 5 to 43% of the amount of arsenic than when the wood was fresh. Stilwell (1998) found that in-

place wooden playscapes yielded 22% of the amount of dislodgeable arsenic than freshly purchased CCA-treated lumber subjected to only one month of weathering. Riedel *et al*. (1990) also note the decrease in dislodgeable arsenic with weathering. They discussed a value of 19% of the amount of dislodgeable arsenic in weathered samples compared to fresh samples, but did not state the duration of weathering. The results of these studies are consistent with those reported in studies conducted by the United States Consumer Product Safety Commission (CPSC) (CPSC, 1990) and the Canadian Wood Preservers Association (CWPA) (Cooper, 1990), in which the amount of leachable arsenic dropped quickly over a period of hours to days. Although they used different protocols, leaching studies conducted by Lebow *et al*. (2000b) and Townsend *et al*. (2001) observed a 3- to 10-fold reduction in the amount of arsenic leaching from 20-gram blocks exposed to acidic solutions (pH = 3.0) over a period of three to nine days. Most of the leaching occurred in the first three days.

The results of these studies indicate that under field conditions, the amount of arsenic released from CCA-treated wood will decrease with age and weathering. Therefore, the assumption used in the HHRA that the amount of dislodgeable arsenic on the surface of treated wood remains constant over time results in an overestimation of exposure to dislodgeable arsenic. This overestimate will impact the estimated risks for the resident receptor ages 2-31 the most because of the duration of exposure (*i.e.*, 30 years) assumed for this receptor.

Soil Arsenic

In order to provide some perspective on the EPCs used to assess exposure to arsenic in soil that has migrated from a CCA-treated structure, a discussion of soil arsenic background concentrations in the U.S., regulatory and site-specific soil arsenic cleanup levels, and other potential sources of soil arsenic is included in Appendix F. A summary of this discussion is included here.

The average background concentration of arsenic in U.S. soils is about 5 to 7 mg/kg (Shacklette and Boerngen, 1984; Dragun and Chiasson, 1991). These background levels are greater than the 95% UCLM of 4.1 mg/kg used as the EPC for the playground scenario. In fact, the 95% UCLM of the reported background concentrations from the study used to calculate the soil arsenic levels for the playground scenario is 4.4 mg/kg, which is consistent with the background concentration in U.S. soils and actually higher than the site-wide average used as the EPC.

Some state regulatory cleanup concentrations for soil arsenic are higher than the EPCs used for both the playground and residential scenarios in the HHRA. Residential cleanup criteria concentrations considered acceptable for residential exposures (including children) include values as high as 30, 20, 24, and 20 mg/kg for Massachusetts (MADEP, 1994), New Jersey (NJDEP, 1999), Texas (TNRCC, 2001), and Washington state (WA Ecology, 2001), respectively. Some of these regulatory cleanup levels approach, and even exceed in the case of Massachusetts, the 95% UCLM of 28.7 mg/kg, which is the EPC used for the residential exposure scenario in the HHRA.

In addition to state cleanup levels, several Superfund site-specific cleanup criteria are higher than the arsenic concentrations measured in soil under decks or on playgrounds. For example, the residential cleanup criterion for arsenic at Bartlesville, Oklahoma was 60 mg/kg (USEPA, 1996b), 230 mg/kg at Commencement Bay, Washington (USEPA, Region 10, 1993), and 250 mg/kg at Anaconda Smelter, Montana (USEPA, Region 8 and MDEQ, 1996). Superfund cleanup levels based on background have been as high as 48 mg/kg at the Jadco-Hughes site in North Carolina (USEPA, 1992d). Soil cleanup levels at Superfund sites have been as high as 1000 mg/kg (recreational use) in Montana (USEPA, Region 8, 1994; Valberg *et al.*, 1997).

Other sources of soil arsenic at a residence and/or a playground include fertilizer products, such as home-use fertilizers, micronutrients, and soil amendments (*e.g.*, bone meal, manure), which can contain arsenic at concentrations up to 75 mg/kg (WA Ecology, 1999). These materials have the potential to elevate soil arsenic concentrations above background levels.

Bioavailability

Bioavailability of Arsenic Based on the Results of Animal Studies

Soil Arsenic

As discussed in Section 3.4.1, the relative bioavailability of arsenic in soil in the vicinity of structures constructed of CCA-treated wood is assumed to be 16.3%, based on a primate study and other supporting evidence (*i.e.*, leaching and wipe studies, discussed below). The relative bioavailability of dislodgeable arsenic is estimated to be 47% based on studies in dogs, and supporting data from leaching studies. Uncertainty exists in results obtained from *in vivo* bioavailability studies because the anatomy and physiology of the animals used in these studies may differ from those of humans (Ruby *et al.*, 1999;

Valberg *et al*., 1997); however, the RBA estimates used in the HHRA were selected based on the best available scientific data and are not expected to underestimate the amount of arsenic absorbed into the body. In particular, the relative bioavailability absorption (RBA) selected for arsenic in soil in these risk analyses is based on a study of primates, which are the animal model which is most similar to humans for evaluating potential exposures and health effects. In selecting the RBA for arsenic in dislodgeable materials, the absolute bioavailability values reported in the studies are transformed into RBA estimates using a conservative estimate of the absorption of soluble arsenic in dogs.

The need for careful assessment of the bioavailability of arsenic from soil and other solid media is widely recognized. The standard quantitative toxicity factors for arsenic are based primarily on epidemiological studies in which arsenic exposures occurred through consumption of arsenic dissolved in drinking water (USEPA, 2001b). Results from human studies indicate that arsenic is readily absorbed *via* this exposure route, with absorption estimates ranging as high as 96% (Bettley and O'Shea, 1975; Buchet *et al*., 1981; Crecelius, 1977; Tam *et al*. 1979). By contrast, as discussed further in Appendix B**,** numerous studies using soil and other solid media indicate that arsenic absorption can be significantly lower when ingested arsenic is contained within these media.

Exposure Frequency. The number of day-equivalents used for the receptors evaluated in both the residential and playground scenarios is based on the number of hours spent outdoors for different age groups at specific locations, *i.e.*, outdoors in the yard or at a playground (USEPA, 1997b). For each receptor, all of the time outdoors at a given location is assumed to be spent on or around a CCA-treated structure. Because the time outdoors was not apportioned according to time exposed to a treated structure or nearby soils, the EF, as it is used in the HHRA, will likely result in an overestimate of exposure and risk since a receptor is unlikely to be exposed to both dislodgeable and soil arsenic simultaneously.

HTE. As discussed in Section 3.4.4, the hand transfer efficiency (HTE) for the child resident ages 2-6, was calculated to be 0.25 based on data regarding the adherence of soil to the hands, the skin surface area of the hands, and soil ingestion rates. Specifically, soil adherence values and median skin surface areas for both boys and girls were multiplied to estimate the mass of soil present on the hands. This mass of soil was then compared to a soil ingestion rate for boys and girls to derive the proportion of the total mass of soil on the hands that would correspond to the soil ingestion rate, *i.e.*, the number of "hand-loads" per day of soil that a child would need to ingest to equal the soil ingestion rate. This HTE factor was then applied to estimate the amount of dislodgeable material on a young child's hands that might be ingested. Because data for soil ingestion are less available for older children (*i.e.*, greater than

6 years of age) and adults, the HTE for the child and adult receptor (ages 7-31) was assumed to be half of the value estimated for the child resident. In both cases, the procedure used to derive the HTE factor reflects the underlying assumption that the adherence and transfer of dislodgeable arsenic from CCA-treated wood are similar to those for soil. This approach also inherently assumes that the primary source of incidentally ingested soil and dislodgeable arsenic is what has adhered to the hands.

In some cases (*e.g.*, Roberts and Ochoa, 2001), it has been suggested that an HTE factor should be calculated based only on the data for female children. This approach has been recommended due to the apparently greater hand transfer efficiency estimate based on the female data; however, this gender difference is likely to be an artifact of the method used in the study (*i.e.*, Roels *et al*., 1980) to calculate soil loading on the hands rather than a reliable observation of a gender difference in hand transfer efficiency. Specifically, the soil adherence rates measured in the Roels study for girls are less than those for boys; however, a single soil ingestion rate (based on both boys and girls) was used to estimate the HTE factor. When compared to the same soil ingestion rate, a lower soil adherence factor necessarily will yield a greater hand transfer efficiency to achieve the same intake rate. If gender-specific soil ingestion rates were available, it is likely that the soil ingestion rate for girls would be less than the average value for both genders and that the HTE factors for boys and girls might be more similar. Because a soil ingestion estimate based on both boys and girls was used in the hand transfer efficiency calculations, the average HTE factor calculated for both genders was derived and selected as the most representative value.

5.5.3 Toxicity Assessment

Toxicity Via Dermal Exposure. The approach used to evaluate risks from dermal exposure addresses systemic cancer and non-cancer effects by assuming that once a chemical is absorbed into the blood stream the health effects are similar regardless of whether the route of exposure was oral or dermal. However, there are uncertainties associated with this approach because dermally absorbed chemicals may have different patterns of distribution, metabolism, and excretion than orally absorbed chemicals (USEPA, 1999a). Use of oral toxicity criteria to evaluate dermal exposures may over- or under-estimate risks, depending on the chemical. Furthermore, this approach does not address potential dermal toxicity associated with direct contact (*i.e.*, "port of entry" effects), such as allergic contact dermatitis, chemical irritation, and skin cancer. Although the USEPA is currently in the process of developing chemical-specific dermal toxicity criteria for these types of health effects, such values are not currently available (USEPA, 1999a).

Oral Cancer Slope Factor. There is considerable debate among the scientific community regarding the CSF_{oral} for arsenic. A number of researchers believe that the current value of 1.5 $(mg/kg/day)^{-1}$ may overestimate cancer risks for U.S. populations (Chappell *et al*., 1997; Slayton *et al.*, 1996; Slayton and Beck, 2001). The key uncertainties are summarized below:

- **Exposure Assessment.** There are considerable scientific concerns about the exposure estimates in the Taiwanese study (Slayton *et al.*, 1996; Chappell *et al*., 1997; Brown *et al*., 2000). Individual exposures were not characterized; exposures were estimated based on average well arsenic concentrations in each village. The original data are not available. The analytical method used to measure arsenic concentrations may not have been accurate at low levels. Other possible sources of exposure (arsenic in rice and yams) were not controlled. Therefore, the Taiwanese data are inadequate for quantitative dose-response assessment for arsenic and skin cancers.

- **Dose-Response Modeling.** The USEPA calculated the current CSF_{oral} using a nearly linear dose-response relationship (USEPA, 2001b). However, a recent USEPA panel concluded that arsenic's dose-response relationship appears non-linear (USEPA, 1997c). Two reviews of the available epidemiology studies found that the current CSF_{oral} is likely to overpredict skin cancer cases at relatively low levels of exposure (Guo and Valberg, 1997; Valberg *et al*., 1998). In epidemiological studies of U.S. populations exposed to arsenic in drinking water, increased cancer rates have not been observed (see, for example, Lewis *et al*., 1999).

- **Human-to-human variations.** In general, dose levels, genetic factors, dietary patterns, or other lifestyle factors may alter arsenic metabolism and detoxification in different populations (Beck and Slayton, 1998; Del Razo *et al*., 1997). Protein deficiencies in the Taiwanese diets could have affected their ability to methylate, and therefore, detoxify arsenic (NRC, 1999). As a result, extrapolations from one population to another are highly uncertain.

Subchronic Oral Reference Dose. The level of confidence or certainty in a toxicity criterion such as an RfD is generally governed by four factors: (1) a preference for human rather than animal toxicity data (2) the existence of multiple studies showing similar results in different populations (3) the quality of the studies being considered, and (4) the availability of a NOAEL rather than a LOAEL (USEPA, 1993). Considering these factors, a high degree of confidence can be ascribed to the subchronic RfD_{oral} derived by USEPA, Region 8. This toxicity criterion is based on a large number of epidemiological studies that were conducted in different populations and under different exposure conditions. Some of the studies, such as those of Mizuta *et al*., and Borgono and Greiber involved a large number of subjects and appear to be well conducted (USEPA, Region 8, 2001). Furthermore, although the data define a LOAEL, Region 8 scientists were able to use the extensive data of Tseng (1977) and Tseng *et al*. (1968) to define a likely boundary range for a NOAEL. The lower boundary on this range (0.02 mg/kg-day) is essentially the

same as the subchronic RfD_{oral} (0.015 mg/kg-day). This suggests that the effect of any error in the subchronic RfD_{oral} would be quite small. And the fact that an independent group of ATSDR scientists derived a similar value of 0.005 mg/kg-day lends support to the Region 8 criterion (ATSDR, 2000).

Other Arsenic Toxicity Issues. Concerning the sensitivity of children to arsenic, relative to adults, the primary uncertainty lies in the limited amount of data available. Only a handful of studies have specifically examined the effects of arsenic exposure in childhood populations. Studies that have reported effects (*e.g.*, Bencko and Symon, 1977; Bencko *et al*., 1977) have not adequately addressed the role of possible confounding factors. In general, it would appear that any particular vulnerability experienced by children would be an important consideration only for higher exposures (*e.g.*, those resulting in urinary arsenic concentrations above 60 μg/L based on the Morse *et al*., 1979 study). Because the doses estimated for children exposed to arsenic associated with CCA-treated wood are well below this level, an increased susceptibility of young children, if it exists, is unlikely to be of concern with arsenic from this source.

With respect to the role of arsenic in endocrine disruption, the data are also quite limited. The few studies that have shown effects of arsenic on reproductive function in laboratory animals (Chattopadhyay *et al*., 1999, 2001; Sarkar *et al*., 1991) used doses that were orders of magnitude greater than the estimated doses from exposures to arsenic associated with CCA-treated wood. Studies suggesting a role for arsenic in diabetes have been limited to ecological epidemiology studies, in which it is difficult to demonstrate cause and effect (Lai *et al*., 1994; Rahman *et al*., 1998; Tseng *et al*., 1968). *In vitro* studies involving glucocorticoid receptor binding were conducted using isolated cells or molecules, and therefore, are of limited relevance to human health risks (Kaltreider *et al*., 2001; Lopez *et al*., 1990).

5.5.4 Risk Characterization

Because risk characterization serves as a bridge between risk assessment and risk management, it is important that major assumptions, scientific judgments, and estimates of uncertainties be described in the assessment. The certainty of risk estimates depends on the uncertainties inherent in each preceding step of the risk assessment process. Many uncertainties are generic to the risk assessment process, while others are specific to particular sites or categories of assessment.

A sensitivity analysis, described below, is conducted to put the estimated risks into context. Such a context is crucial for interpreting the risk assessment results and using them to make risk management

decisions. To accomplish this goal, factors that may tend to over- or underestimate risks are identified and the relative magnitude of uncertainty for each factor is evaluated so that the level of confidence associated with the risk estimates is clear. The approach taken in this report to discuss uncertainties in the risk assessment is consistent with USEPA guidance encouraging better characterization of the uncertainties inherent in risk calculations and the effects that these uncertainties, as well as variability in risk input parameters, have on the final risk assessment results (USEPA, 1992e; 1999b; USEPA, Region 3, 1994).

The uncertainty analysis presented in this report includes two basic components. First, in Section 5.5.4.1, the potential influence of uncertainties on the risk analyses described in this report is qualitatively evaluated in each of the four risk assessment steps. These discussions focus on those uncertainties that most significantly affect the risk assessment results. To supplement this qualitative evaluation, a quantitative uncertainty analysis is presented in Section 5.5.4.2. Focusing on key factors influencing the risk assessment results, the quantitative uncertainty analysis provides more detailed information regarding the magnitude of the uncertainty associated with specific elements of the risk assessment. Section 5.5.4.2 also describes the implications of these uncertainties for the risk assessment results.

5.5.4.1 Risk Characterization

General aspects of the risk characterization phase of the risk assessment that can introduce uncertainties into the risk assessment results include:

- The validity of adding carcinogenic risk estimates or non-cancer hazard quotients for multiple chemicals
- The validity of adding carcinogenic risk estimates or non-cancer hazard quotients across exposure pathways.

In some cases, chemicals may not affect similar target organs, may not act *via* similar mechanisms, or may interact in ways that are not additive. As a result, adding risk estimates or hazard quotients may not appropriately reflect the potential risks associated with multiple chemical exposures. Similarly, the risks posed by a chemical following exposure *via* different pathways may differ in ways that are not adequately reflected by simple addition of the risk estimates derived for each individual pathway. Moreover, when interpreting the results from combining risk estimates for different exposure pathways, the likelihood that such combinations of pathways or scenarios will occur must also be considered.

Because this assessment focused only on arsenic, it does not include uncertainties associated with adding risk estimates calculated for multiple chemicals. Some uncertainties exist, however, as to whether the two forms of arsenic examined in the risk assessment (*i.e.*, dislodgeable and soil arsenic) are completely comparable with regard to their mechanisms of exposure and toxicity. As a result, some uncertainty is added to the risk estimates when risk results for these two forms of arsenic are combined. Similarly, combining the risk estimates for the ingestion and dermal contact exposure pathways results is another source of uncertainty in the risk characterization step of this assessment.

Risk assessment methods are designed to be highly conservative to address the uncertainties associated with each step in the process. Thus, actual risks are not likely to be greater than (and may be significantly less than) risks estimated using standard risk assessment methods. In this risk assessment, key factors that are likely to overestimate rather than underestimate site risks include:

- Using 95% UCLM concentrations as the EPCs for soil and dislodgeable arsenic
- Assuming that the concentration of dislodgeable arsenic concentrations on the surface of treated wood remains constant throughout the entire period of exposure, despite data indicating that arsenic concentrations decrease over time with weathering
- Using exposure assumptions derived for a residential setting as a conservative screening evaluation for other scenarios where exposures are likely to be significantly less (*e.g.*, at a playground)
- Assuming that exposures for a particular individual would continue for up to 30 years
- Assuming that exposures to dislodgeable arsenic does not reduce exposures to soil arsenic or *visa versa*
- Using conservative intake assumptions to estimate reasonable maximum exposures
- Assuming that the dose-response relationship for arsenic is linear at low doses

Due to the factors listed above, actual site risks are unlikely to be underestimated, and are likely to be substantially overestimated, by the procedures applied in this risk assessment. Many of these factors were discussed in more detail in the preceding sections.

5.5.4.2 Quantitative Uncertainty Analysis

To supplement the qualitative review of risk assessment uncertainties that is presented above, a quantitative uncertainty analysis was conducted. As recommended in USEPA guidance (USEPA, 1989, 1999b), the goals of this analysis were as follows:

- To provide context for the exposure and risk estimates derived in the risk assessment
- To examine the quantitative implications for the risk assessment results of the choices made during the risk analyses (*e.g.*, to address parameter variability or sources of uncertainty)
- To identify those specific parameters or sources of uncertainty that most influence the risk assessment results
- To identify areas where additional data collection could improve the risk assessment results and risk management decision-making.

This analysis considered the influence of both variability and uncertainty on numerical risk estimates. Variability is the natural variation in potential values that exists for some exposure parameters. For example, measurements of body weight for a population will demonstrate inter-individual variability, *i.e.*, different individuals will have different body weights. Exposure parameters may also exhibit intra-individual variability, *i.e.*, the value of a parameter for a specific individual may vary at different times. For example, an individual child might have varying amounts of dermal contact with soil on different days, at different times of year, or at different ages. Uncertainty reflects limitations in existing data regarding the true value of a parameter or absence of knowledge about specific values. Both the potential sources of variability and uncertainty and the influence of these factors on the risk assessment results were considered in this analysis.

A variety of tools are available for conducting quantitative uncertainty analyses. These tools include methods that fully characterize the uncertainty and variability in risk assessment model results in a quantitative way and methods that provide semi-quantitative approaches to evaluating the impacts on specific aspects of the risk model results. Because available data are insufficient to fully characterize the potential distribution of values for each input parameter applied in the risk assessment, a comprehensive quantitative uncertainty analysis (*e.g.*, a Monte Carlo probabilistic assessment) was not conducted. Instead, a focused sensitivity analysis was performed to provide quantitative insights regarding the

relative contributions of various input parameters to variability and uncertainty in the risk assessment results.

The approach applied for this project included the following elements:

- Reviewing the results of preliminary risk calculations to identify those exposure pathways and risk assessment elements warranting more detailed analysis
- Reviewing available quantitative information regarding the potential values for the exposure input parameters applied in this assessment, including evaluating the relative magnitude of the variability and uncertainty associated with each parameter
- Summarizing the quantitative implications of parameter variability and uncertainty on the exposure and risk assessment results for specific individual parameters and for selected combinations of parameters
- Reviewing issues associated with parameter interactions and combinations that would affect the influence of the parameters on the risk assessment results (*e.g.*, correlations among parameters or the likelihood that multiple parameters would have high-end values simultaneously)

These quantitative uncertainty analyses focused on those parameters associated with incidental ingestion of dislodgeable arsenic, because this exposure pathway is the primary contributor to the total exposure and risk estimates calculated in this assessment. Specifically, this exposure pathway accounts for approximately 60 to 70% of the total cancer and non-cancer risk estimates for a given receptor in the HHRA. In addition, efforts in conducting this uncertainty analysis focused on providing a detailed review of those parameters that were derived specifically for these analyses (*e.g.*, the concentration of dislodgeable arsenic on the surface of treated wood, the material-specific RBA for dislodgeable arsenic, and the hand transfer efficiency factor). These factors were emphasized because of their important influence on estimates of exposure associated with dislodgeable arsenic. Because these values were developed specifically for this risk assessment, more limited documentation is available in the literature regarding the basis for these values and the potential variability and uncertainty in these values. As a result, more extensive evaluation of these parameters was deemed warranted in these evaluations.

To provide additional context for the quantitative evaluation, this uncertainty analysis briefly reviews quantitative issues associated with those parameters that were derived based on USEPA recommendations. Less emphasis was placed on these factors because they have less influence on the results of this risk assessment. In addition, they have been more extensively documented and used in

other risk assessment settings. As a result, more information is available from a variety of sources regarding the general influence of these factors on risk assessment results. In accordance with USEPA guidance (*e.g.*, USEPA, 1999b), this quantitative uncertainty analysis also focuses only on exposure parameters and excludes a detailed quantitative evaluation of the uncertainties inherent in toxicity assessment. The USEPA recommends this approach because the uncertainties associated with toxicity factors can span orders of magnitude, *e.g.*, depending on the model used to extrapolate from observed adverse effects in toxicity studies to the exposure levels estimated for environmental exposures. As a result, these uncertainties often are most usefully incorporated into risk management decisions by taking into account the types of qualitative issues described above rather than explicitly attempting to incorporate such wide potential ranges of values into quantitative risk estimates.

Exposure Parameter Review

This section discusses quantitative information regarding the variability and uncertainty associated with the key parameters for estimating exposures and risks associated with incidental ingestion of dislodgeable arsenic, *i.e.*, the concentration of dislodgeable arsenic on treated wood surfaces, the RBA for ingested dislodgeable arsenic, and the hand transfer efficiency factor. This section also briefly reviews information regarding the variability and uncertainty associated with those parameters that were developed based on USEPA recommendations. The results of the review presented in this section are summarized in Table 8 in the Tables section of the report.

Concentration of Dislodgeable Arsenic on Treated Wood Surfaces

As described above, when calculating potential exposures to dislodgeable arsenic, the concentration of dislodgeable arsenic on the hands (C_{DA}) is used as the EPC. The EPCs used in the risk calculations are best estimates of the 95% UCLM concentrations of dislodgeable arsenic reported for 8 treated wood samples (SCS, 1998). The EPCs were calculated in accordance with USEPA guidance (*e.g.*, USEPA, 1989; 1992b) based on data collected in controlled trials in which adult volunteers rubbed their on various wood types. The researchers then rinsed the volunteers' hands and analyzed the amount of arsenic in the rinsate. The 95% UCLMs for the 8 treated wood samples ranged between 0.030 and 0.130 $\mu g/cm^2$. The 95% UCLM for an untreated wood sample (negative control) was 0.005 $\mu g/cm^2$.

Based on several aspects of its design, this study was deemed the most relevant for deriving representative concentrations of dislodgeable arsenic on hands and evaluating the consequent potential for

incidental ingestion of this material. Specifically, the hand sampling data collected in this study were accompanied by measurements of the hand surface area for each study participant. In addition, this study included an extensive number of samples and wood types. By contrast, other hand sampling studies lacked accompanying hand surface area data or had too few samples to support statistically meaningful evaluations. Several other studies evaluated dislodgeable arsenic concentrations by wiping various wood types with substrates other than the hand. These studies generally yielded higher EPC values; however, the results obtained using these alternative materials are of questionable relevance for assessing the exposure scenario under consideration. Because of these factors, the 1998 SCS study was determined to be most relevant for these risk analyses.

The SCS data indicate that EPC estimates can vary depending on the properties of the CCA-treated wood type such as species, aging and use of sealants. In addition, EPC values for specific wood samples showed distinguishable concentration trends. For example, untreated Southern Pine has the lowest EPC (with a 95% UCLM of 0.005 $\mu g/cm^2$), while treated southern pine, the most commonly used type of treated wood, has a higher EPC (with a 95% UCLM of 0.061 $\mu g/cm^2$). The highest calculated EPC of all the tested wood samples was observed with treated Southern Pine with pressure-applied water repellant (with a 95% UCLM of 0.130 $\mu g/cm^2$). Because of these trends, risks are evaluated separately based on EPCs specific for each wood sample.

Inter-individual variability in the loadings of dislodgeable materials onto hands is also reflected in the SCS data. Intra-individual variability would also be expected in loadings rates associated with different activities or at different times; however, data regarding this aspect of hand loadings are limited and only reflect the potential influence of "handedness" on loadings. Specifically, the SCS study conducted two trials for each person on each substrate, *i.e.*, each person rubbed the wood sample using the left hand and then the right hand. The SCS study exhibits considerable systematic inter-individual variability in EPCs, which contributes to wide upper confidence intervals on the mean for certain wood samples. The maximum reported concentration for a given wood sample was 1.1 to 2.3 times larger than the 95% UCLM for that sample.

A number of sources of uncertainty exist in the estimates of the EPC parameter from the SCS study. First, the values are based on a relatively small study size, using five volunteers with one replication of each hand on each wood type. A study with a higher number of replications would have stronger statistical power to differentiate between the EPCs for specific wood samples. A second source

of uncertainty is whether the type of rubbing action employed by the volunteers in the study is representative of typical dermal exposures to treated wood, especially for a child. Third, uncertainty arises from the procedure used to calculate the surface area of the participants' hands, *i.e.*, by tracing the outline of the hand. This method differs from those used to calculate the surface area of hands or the whole body in other studies. In the HHRA, the hand tracing was assumed to approximate one-third of the total surface area of the hand. A fourth source of uncertainty is whether the wood types tested in the study sufficiently represent the wood types in the market place and used in structures that people may be exposed. To address this source of uncertainty, these risk calculations focused on both the most prevalent wood type used in the U.S. (*i.e.*, treated Southern Pine) and the wood type with the highest EPC (*i.e.*, treated Southern Pine with pressure-applied water repellant). The quantitative impact of these sources of uncertainty is not expected to be substantial. Moreover, they are expected to primarily affect the precision rather than the accuracy of the EPC estimate for each wood sample.

To provide some quantitative perspective on the influence of variability in the EPC estimates used to quantify risks in the HHRA, the 5^{th} and 95^{th} percentile EPC values were calculated based on the data for each wood type from the SCS study. The 5^{th} and 95^{th} percentile ranges are presented in Table 8, and include 0.002-0.009 $\mu g/cm^2$ for untreated Southern Pine, 0.004-0.101 $\mu g/cm^2$ for treated Southern Pine, and 0.055-0.155 $\mu g/cm^2$ for treated Southern Pine with pressure-applied water repellant. The values for only these wood types were used in the sensitivity analysis because they represent an untreated wood type, the most commonly used wood type, and the wood type resulting in greatest exposure.

Relative Bioavailability Absorption Factor for Dislodgeable Arsenic

The RBA factor for dislodgeable arsenic that was used in the risk calculations is a best estimate that was derived based on the most relevant animal study data currently available. Specifically, the value used (47%) is the mean value calculated based on results from two studies in which dogs were fed sawdust made from CCA-treated wood (Peoples, 1976; Peoples and Parker, 1979). These studies were deemed the most relevant for assessing the bioavailability of dislodgeable arsenic because the sawdust used in these studies is the most comparable of the materials evaluated in bioavailability studies to the material that would be dislodged from the surface of CCA-treated wood. Data from leaching studies of treated wood were also considered in developing the RBA factor used in the risk analyses.

The data from the dog studies indicate that inter-individual variability exists in this parameter. The estimates of absolute bioavailability reported for individual animals in these studies ranged from 26

to 40%. When adjusted to reflect relative bioavailability, the values derived for the individual animals ranged from 38 to 59%. Intra-individual variability is also likely to exist in the relative bioavailability of dislodgeable arsenic. For example, absorption of arsenic is likely to be influenced by whether the material is ingested with food or between meals; however, the magnitude of this source of variability is unknown.

A number of sources of uncertainty exist in the estimates for this parameter. First, these values are based on results from only three animals. Second, the sawdust used in the study may not be identical to the material that is typically dislodged from CCA-treated wood (*e.g.*, with regard to particle size or composition). A third source of uncertainty arises because the dog study protocol did not include an evaluation of the absolute bioavailability of soluble arsenic. As a result, the relative bioavailability of the arsenic in the sawdust was estimated using primate data for the absolute bioavailability of soluble arsenic and data indicating that absorption is similar in dogs and primates. Because the values obtained from these three animals are relatively consistent with each other and with the results of other studies of arsenic bioavailability in various solid matrices, the quantitative impact of these sources of uncertainty on the RBA factor estimates is not expected to be substantial. In particular, as described above, the results from other similar bioavailability studies of arsenic in various solid matrices have generally reported RBA factor estimates of less than 50%. These findings suggest that use of the current estimate for the relative bioavailability of dislodged arsenic in the risk assessment is unlikely to underestimate actual absorption and consequent risks. Moreover, the results from similar studies suggest that development of better data for this parameter would yield parameter estimates that are less than that derived based on the currently available data.

In theory, bioavailability estimates can potentially range from 0-100%. However, data from the most relevant materials, data from comparable studies of other solid matrices, and consideration of the chemistry of treated wood suggest that the likely range of values for the RBA factor for dislodgeable arsenic is likely to be within the middle to lower end of this range. Specifically, the available data from the most relevant studies indicate a range spanning from 40-60%. Results from other similar studies as well as information regarding the fixation process for CCA-treated wood suggest that the range of values for this parameter is unlikely to be significantly greater than the maximum value observed in the most relevant studies. To account for the various sources of uncertainty in the available data, a range that is slightly greater than that reflected in the available data from the most relevant studies was applied in the sensitivity calculations discussed in the next section. Specifically, a range of 30 to 70% was examined in these calculations.

Hand Transfer Efficiency Factor

The HTE factor for dislodgeable arsenic that was used in the risk calculations is a best estimate that was derived based on data regarding the adherence of soil to the hands, the skin surface area of the hands, and soil ingestion rates. Specifically, the value used for young children between the ages of 2 and 6 years old (0.25) is calculated based on mean estimates derived from studies of soil loading onto children's hands (Roels *et al*., 1980), national data regarding the skin surface area of the hands (USEPA, 1997a), and data from a study of soil ingestion in young children (Stanek and Calabrese, 1995a). Because soil ingestion data are not available for older age groups, the HTE value for older children and adults (0.13) was estimated based on the value calculated for younger children. The approach used to calculate this value is consistent with that taken in several of the risk assessments of CCA-treated wood that have been conducted to date (*e.g.*, CPSC, 1990; HS&WMR, 2001) and reflects some updates and revisions to the previously applied approaches.

Each of the elements included in calculating the HTE factor has components of variability and uncertainty. The primary data used to calculate the HTE factor is a set of measurements of lead loadings on the hands of children attending schools in the vicinity of a lead smelter (Roels *et al*., 1980). The published report of these data provides only mean estimates of the amounts of lead measured on the hands of various subgroups of the study population. As would be expected, these summary data indicate that inter-individual variability exists in this parameter. In particular, lead loadings onto hands varied between girls and boys. Variations were also seen for measurements collected at different distances from the smelter. Intra-individual variability would also be expected, *e.g.*, an individual child is likely to have different loadings at different times depending on such factors as soil moisture and the types of activities that the child engages in. The magnitude of these variations is not discussed in the published report of these data. As a result, these elements of variability also contribute uncertainty to this parameter.

Other sources of uncertainty in these data are associated with the rinsing technique used to measure the lead loadings on the children's hands and with the limited information available regarding the specific activities that the studied children engaged in prior to participating in the study. The applicability of these data to estimating hand loadings of soil for children at other locations or participating in other activities also provides a source of uncertainty in this parameter. In addition, uncertainty exists regarding the applicability of data regarding loadings of soil on hands to estimate hand loadings of dislodgeable arsenic from CCA-treated wood.

In the first step of the hand loading calculations, the lead mass on the children's hands was divided by the lead concentration measured in the school playground dust to estimate the soil loadings on the hands. Such environmental measurements would reflect variability in the concentrations present at specific locations and to which individual children would be exposed. In addition, such data would be subject to uncertainty arising from sampling and analytical techniques as well as uncertainty regarding whether the measured concentrations are representative of the relevant concentrations contributing to the studied children's exposures. In addition, because Roels *et al*. (1980) provides only mean concentrations for lead concentrations in playground dust for each of the four geographic areas examined, uncertainty exists regarding the magnitude of the variability observed in the sampling results.

To assess the potential variation in the measured lead concentrations in soil, data presented in an earlier paper by these authors (Roels *et al*., 1978) were used. This earlier report provides means and standard deviations for the school-yard dust concentrations in three of the four studied areas. To estimate the relative magnitude of these two parameters for each of these three areas, the standard deviation was divided by the mean concentration. Then, for each of these areas, the ratio between these two parameters for the concentration data used in the later report was assumed to be equal to that observed in the earlier study and was used to estimate the standard deviation of the data sets applied in the later report. For the fourth area, for which data were not presented in the earlier report (*i.e.*, the urban area), the standard deviation of the concentrations was estimated based on the average relative magnitude of the standard deviations reported for the other areas. To provide some quantitative perspective on the relative range of concentrations in these areas, these standard deviations were then used to estimate the 5^{th} and 95^{th} percentile values in the distribution of concentrations in each area. The results of these calculations are shown below in Table 5-1. As can be seen, the greatest range of potential concentrations is predicted for the area closest to the smelter, where the 95^{th} percentile concentration was approximately 4 times the 5^{th} percentile concentration. By contrast, for the data collected at 2.5 km from the smelter, the 5^{th} and 95^{th} percentile concentrations were within a factor of 1.5 of each other.

Table 5-1
Estimated Variability in School Yard Dust Concentrations Reported in Roels *et al.* (1980)[a]

Study Area	Dust Lead Concentrations (Roels *et al.*, 1978)			Dust Lead Concentrations (Roels *et al.*, 1980)				
	Reported Mean	Reported SD	Ratio of SD to Mean[b]	Reported Mean	Estimated SD[c]	Estimated 5th Percentile[d]	Estimated 95th Percentile[e]	Ratio of 95th to 5th Percentile
<1 km from smelter	3,541	1,310	0.37	2,560	947	1,002	4,118	4.1
2.5 km from smelter	397	33	0.083	466	39	402	530	1.3
urban	NA	NA	0.24[g]	112	27	68	156	2.3
rural	152	41	0.27	114	31	63	165	2.6

NA – Not available.
SD – Standard deviation

Notes:
[a] – All concentrations reported in mg/kg.
[b] – Calculated by dividing the reported SD by the reported SD, e.g., 1,310/3,541 = 0.37.
[c] – Estimated by multiplying reported mean concentration by the ratio between the SD and the mean observed in the data from Roels et al. (1978) for same study area, e.g., 2,560 × 0.37 = 947.
[d] – Estimated using standard formula for calculating percentiles in normal distributions of data (Zar, 1984), i.e., mean - (z-score for desired percentile × SD) = desired value. For example, 2,560 – (1.645 × 947) = 1,002.
[e] – Estimated using standard formula for calculating percentiles in normal distributions of data (Zar, 1984), i.e., mean + (z-score for desired percentile × SD) = desired value. For example, 2,560 + (1.645 × 947) = 4,118.
[f] – Calculated by dividing the 95th percentile by the 5th percentile, e.g., 4,118/1,002 = 4.1.
[g] – Estimated based on average value of ratios observed in other three areas.

In the second step of the HTE calculation, the soil density on the children's hands was estimated by dividing the mass of soil on the children's hands by the skin surface area of the portion of the hand that was washed in the study, *i.e.*, the palmar surface of one hand. Data corresponding to the age range included in the study, *i.e.*, 11 year old children, were used in this calculation. In the third step of the HTE calculation, skin surface area data for younger children, *i.e.*, 2-6 years old, were used to estimate the mass of material that might be present on the hands of children in this age range that were included in this risk assessment. The surface area estimates used in these calculations were derived from the compilation of data presented in the USEPA's *Exposure Factors Handbook* (1997a). These data are based on national studies measuring skin surface area in a variety of individuals as well as regression equations predicting areas associated with specific body parts or areas based on other parameters such as body weight. Inter-individual variability exists in the absolute skin surface area values for specific individuals as well as the amount of skin surface area that is exposed to soil or dislodgeable material during different exposure events. In addition, intra-individual variability is likely to exist in the amount of skin surface exposed during different events. Some uncertainties exist in these data (*e.g.*, their applicability to specific population subgroups); however, among the parameters commonly applied in risk assessments, this

parameter is considered relatively well-characterized and the data presented in the USEPA's *Exposure Factors Handbook* are widely accepted and used.

Data available for this parameter include gender-specific mean values, percentiles, and distributions for various age ranges. To provide some quantitative perspective on the magnitude of the variability in this parameter, the 5th and 95th percentile values for the skin surface area of the hands for the two ages applied in these calculations were examined. These values are summarized in Table 5-2, below.

Table 5-2
Summary of Skin Surface Area Values

Age Range	Surface Area Estimate (cm^2)			
	5th Percentile	**50th Percentile**	**95th Percentile**	**Ratio of 95th to 5th Percentile**
11 year olds[a]	91	112	142	1.6
2-6 year olds[b]	115	131	154	1.3

Notes:
[a] *– Surface area estimate for palmar surface of one hand (i.e., one-third of one hand), the area washed in the Roels et al. (1980) study.*
[b] *– Surface area estimate for the palmar surfaces of two hands (i.e., one-third of each of two hands), the area assumed in this risk assessment to have primary contact with dislodgeable arsenic and to have subsequent contact with the mouth.*

As can be seen, the differences between the 5th and 95th percentile values for this parameter are relatively small, with the 95th percentile values being a factor of approximately 1.5 greater than the 5th percentile values. It should be noted that the skin surface area of the hands is calculated by multiplying estimates of the total skin surface area by an estimate of the percentage of the total that is comprised of the area of the hands. Because the estimates of the hand area percentage are sometimes presented as a range, this element of the calculation reflects another source of variability in this parameter. Such ranges are not available for most of the age levels included in this calculation. Moreover, where available, such ranges are typically small (*e.g.*, for 3-4 year olds, the mean percentage is 6.07% and the range of percentages is 5.83-6.32%). For these reasons, this aspect of the calculation was not explicitly included in the quantitative sensitivity analyses.

In the final step in the HTE calculation, the HTE was derived by dividing a best estimate of the incidental soil ingestion rate in young children by the estimated mass of soil on young children's hands estimated based on the Roels *et al*. (1980) study. The estimate used in this calculation is based on the mean soil ingestion rate for the 50th percentile child observed in a study of soil ingestion in 64 young children between the ages of 1 and 4 years old residing in Massachusetts (Stanek and Calabrese, 1995a,

1995b). As described in Section 3.4.1, the Stanek and Calabrese (1995a, 1995b) data were used to estimate the soil ingestion rate for young children within the age range of interest for this risk assessment, *i.e.*, 2 to 6 years old. Available studies reflect inter-individual variability in incidental soil ingestion rates among different children. Intra-individual variability will also exist for children's soil ingestion on different days and under various exposure conditions. Uncertainty also exists in this parameter regarding the methods used to measure soil ingestion, the methods used to interpret the soil ingestion data, and the applicability of the available data to various population groups or exposure conditions.

The Massachusetts study used specific tracer elements and mass balance calculations to estimate soil ingestion rates in young children. These researchers have conducted a similar study of soil ingestion in young children residing in Anaconda, MT, and have examined the soil ingestion data collected by other researchers. In subsequent analyses of these data, these researchers have explored such issues as the relative reliability of the tracer elements included in the study, the inter- and intra-individual variability in daily soil ingestion rates observed in the study children, and the implications of using data from short-term studies to estimate long-term patterns of soil ingestion. In particular, recent analyses of the potential impacts of various factors on soil ingestion estimates suggest that the results from the currently available short-term studies (*i.e.*, conducted over a 4-7 day period) may overestimate typical soil ingestion rates applicable to long-term exposures (Stanek *et al*., 2001b). This result occurs because the short-term data may overestimate the variance in soil ingestion rates that would be observed over longer periods of time. In particular, these analyses indicate that high-end estimates of long-term soil ingestion rates (*e.g.*, 95th percentile estimates) may be overestimated by as much as 100% when based on short-term data.

Limited data also exist regarding significantly higher estimates of soil ingestion rates in children that exhibit pica behavior, *i.e.*, children that deliberately ingest soil and other nonfood items. Because the amount of material that is likely to be dislodged from treated wood structures by children's hands is unlikely to be sufficient to support such elevated ingestion rates, these data were not quantitatively considered in this sensitivity analysis.

To provide some quantitative perspective on the range of potential soil ingestion rates reflected in the available data, the estimate of the long-term average 95th percentile soil ingestion rate in young children (124 mg/day) calculated based on the Massachusetts study data was used (Stanek and Calabrese, 2000). This value was calculated to approximate average exposures over a one-year period. Because these data were collected only from 1 to 4 year old children, the same process described in Section 3.4.1 was used to adjust these data to reflect values for 2 to 6 year old children. Specifically, the 124 mg/day

value was assumed to apply for children between 1 and 4 years of age. Ingestion rates for other age ranges were calculated by assuming that the relative rates are the same as reflected in the USEPA's default soil ingestion rates identified for the IEUBK model based on outdated analyses of the Massachusetts study data. The average value for the 2 to 6 year old age range was then calculated based on these component values. As summarized below in Table 5-3, these calculations yield an estimate of the average 95th percentile long-term soil ingestion rate for 2 to 6 year old children of 100 mg/day.

Table 5-3
Estimated Age Range-Specific Soil Ingestion Rates (mg/day)

Age Range	IEUBK Default Values[a]	Estimated Average 95th Percentile Values[b,c]	Estimated Average 25th Percentile Values[c,d]
6-11 months	85	78	6
1-2 years	135	124	10
2-3 years	135	124	10
3-4 years	135	124	10
4-5 years	100	92	7
5-6 years	90	83	7
6-7 years	85	78	6
2-6 years	NA	100	8

IEUBK – Integrated Exposure Uptake/Biokinetic Model

Notes:
[a] *– Central tendency estimates presented in USEPA (1994), based on outdated analyses of Massachusetts study data.*
[b] *– Based on estimated long-term value presented in Stanek and Calabrese (2000).*
[c] *– Soil ingestion rate presented for 1-4 year olds is based on values measured in Stanek and Calabrese study; values for other age ranges estimated based on relative magnitude of age-specific soil ingestion rates included in U.S. EPA's IEUBK model.*
[d] *– Based on estimated long-term value presented in Stanek and Calabrese (2000).*

These researchers have not provided estimates of long-term soil ingestions rates for low percentiles (*e.g.*, the 5th percentile). As a result, a low-end alternative soil ingestion rate was estimated using the average 25th percentile soil ingestion rate (10 mg/day) calculated based on the Massachusetts data reflecting only a 7-day period (Stanek and Calabrese, 2000). Use of this value in these sensitivity analyses reflects a conservative approach as these researchers have reported that soil ingestion values based on short-term studies are likely to overestimate long-term ingestion rates (Stanek and Calabrese, 2000; Stanek *et al*., 2001b). Using the same procedures as above, an average 25th percentile soil ingestion rate for 2 to 6 year old children of 8 mg/day was calculated. These calculations are summarized in Table 5-3.

Each of the sources of variability and uncertainty identified above potentially influence the estimates of the HTE factor. To provide some quantitative perspective on the potential magnitude of the influence of each parameter on the HTE value, the HTE factor was recalculated using alternative values for the individual input parameters, *i.e.*, the low-end and high-end values identified above. The results of these calculations are shown below in Table 5-4.

Table 5-4
HTE Estimates Reflecting Variability and Uncertainty in Underlying Data

Parameter	HTE Value Based on:	
	Low-end Value[a]	High-end Value[b]
Soil Concentration	0.21	0.43
Skin Surface Area (11 year olds)	0.26	0.41
Skin Surface Area (2-6 year olds)	0.37	0.27
Soil Ingestion Rate	0.067	1.0
Combined Parameter Alternatives	0.021	1.7

Notes:
[a] – *5th Percentile estimates for soil concentration and skin surface areas; 25th percentile value for soil ingestion rate.*
[b] – *95th Percentile estimates for all parameters.*

As can be seen, the potential variation in the soil concentrations in the Roels *et al*. (1980) study and the skin surface area estimates have little influence on resulting estimates of the HTE factor. HTE estimates based on the low-end and high-end percentile values differ by less than a factor of two. By contrast, the estimates of the HTE value based on alternative soil ingestion assumptions span a broader range, with the estimates based on the low-end and high-end percentile soil ingestion rate estimates differing by approximately an order of magnitude. When the high- and low-end estimates for each of the component parameters are combined to yield the lowest and highest HTE estimates corresponding to these values (*i.e.*, 0.021 and 1.7, respectively), the resulting low- and high-end values differ by a factor of approximately 80 and are approximately a factor of 12 less than or a factor of 7 greater than the HTE value used in the risk analyses (0.25).

This range reflects a greater degree of variability than is likely to exist in actual HTE values. In particular, to generate the highest HTE estimate resulting from the low- and high-end alternative values, specific combinations of the component values are required. For example, the lowest HTE estimate is derived by combining the low-end estimates of the soil concentration, hand surface area for 11 year olds, and soil ingestion rate with the high-end estimate of the hand surface area for 2-6 year olds. It is unlikely that this combination of extreme parameter values would exist in an actual exposure situation. Instead,

some combination of higher and lower parameter values would more likely occur, with values that are higher than average for certain parameters balancing out values that are less than average for other parameters. As a result, actual HTE values are more likely to be closer to the middle of the estimated range than to equal the low- or high-end values.

To account for this factor, the sensitivity calculations discussed in the next section of this report were conducted using a range of potential HTE values that is slightly less than the range from the lowest to highest values resulting from the combinations of the alternative values for the component elements. Because the variation resulting from differences in soil ingestion rates was the primary driver of the HTE values, the range corresponding to the alternative values for this parameter was examined in these calculations (*i.e.*, 0.07 to 1.0 handloads per day for 2-6 year old children). As described in Section 3.4.4, the HTE value for individuals between the ages of 7 and 31 years was assumed to be one-half of that for younger children. Therefore, a range of values of 0.035 to 0.50 handloads per day was used for this age range in the sensitivity analyses described in the next section of this report.

Parameters Based on EPA Recommendations

Skin surface area – The skin surface area estimates used in the risk calculations are best estimates of the median hand surface area assumed to be available for contact with a treated wood surface for the age ranges of the residential receptors evaluated in the HHRA, *i.e.*, children between the ages of 2 and 6 years (132 cm^2/hand transfer) and older individuals between the ages of 7 and 31 years (267 cm^2/hand transfer). These skin surface area values are used to estimate the amount of dislodged arsenic that could adhere to the surface of both hands and subsequently be ingested *via* incidental hand-to-mouth contact. The available hand surface area was calculated to be equal to one-third of the total surface area of each hand based on SCS (1998); USEPA (1999c); and Rodes *et al*. (2001).

Skin surface area estimates have been developed based on a variety of measurement and estimation techniques, including techniques that estimate skin surface area based on body weight and height (USEPA, 1997a). Skin surface area estimates are based on extensive national data that are generally judged to be of high quality. Inter-individual variability exists in the skin surface area values for specific body parts. Both inter- and intra- individual variability and uncertainty exist in the amount of skin surface area that will actually come into contact with dislodgeable arsenic during each exposure event. Some uncertainties in the underlying data arise because of measurement error. Limitations also exist in the amount of data available for estimating the skin surface area of specific body parts (*e.g.*, the

hands) or specific age groups (*e.g.*, children between the ages of 2 and 6). Moreover, the currently available data may require updating to reflect more recently collected data. Because the skin surface area estimates reflect average values calculated across broad age ranges, they will tend to overestimate the median value of this parameter for some age ranges of interest and underestimate it for others. The impacts of these sources of uncertainty on exposure estimates are unlikely to be substantial.

To provide some quantitative perspective on the potential impacts of this parameter on the risk calculations, average values for the 5^{th} and 95^{th} percentile values of the hand surface areas were calculated for the two age ranges of interest based on the same data used to calculate the best estimate (USEPA, 1997a). For 2 to 6 year old children, the 5^{th} percentile estimate of the hand surface area is 115 cm^2, while the 95^{th} percentile is 154 cm^2. For 7 to 31 year old individuals, the 5^{th} percentile estimate of the hand surface area is 237 cm^2, while the 95^{th} percentile is 328 cm^2. These values were used in the sensitivity analysis calculations presented below.

Exposure Frequency – The RME exposure frequency values used in the risk calculations reflect upper-bound estimates of the number of days per year that an individual spends outdoors, *i.e.*, 150 day-equivalents/year for 2 to 6 year olds and 73 day-equivalents/year for individuals between the ages of 7 and 31. These exposure frequencies are based on an activity study by Tsang and Klepeis (1996), where over a 2-year period, researchers conducted a telephone survey of U.S. citizens and obtained information about the amount of time spent in the last 24 hours for a number of different activities, such as the number of minutes spent at a restaurant, at home in the kitchen or traveling in a car. Each respondent maintained a time-log of activities for the previous 24 hours and a total of 3,003 respondents were surveyed in the study. In the HHRA, all of the time spent outdoors was assumed to include contact with CCA-treated structures and impacted soil. As a result, this health-protective assumption is likely to overestimate the amount of exposure to treated wood that would be experienced by most individuals.

Inter-individual variability exists in the amount of time spent outdoors, while substantial uncertainty exists regarding the proportion of this time that would be spent in contact with treated wood structures. For some individuals, no contact with treated wood may occur, while the theoretical maximum exposure frequency would consist of daily exposure. As a conservative element, this sensitivity analysis examined exposure frequency values ranging from 0 days/year to 350 days/year (the USEPA's default RME assumption for a residential scenario).

Exposure Duration. The exposure duration values used in the risk calculations for both children and adults conservatively reflect the maximum amount of time any individual could spend in the specified age range. The overall age range examined in this risk assessment (30 years) reflects the 95th percentile duration of residence at a specific location. Both variability and uncertainty exist in the actual amount of time during which an individual might have contact with CCA-treated wood structures, with the range of exposure duration values including the possibility of no exposure (*i.e.*, an exposure duration of 0 years). Because the values used in the risk assessment reflect the upper-bound exposure durations, any site-specific or scenario-specific adjustments to this factor would tend to reduce the exposure duration and corresponding exposure and risk estimates. Thus, a range of exposure duration values extending from no exposure to the upper-bound estimates used in this risk assessment were considered in the sensitivity analyses presented below.

Body Weight. The body weight values for the age ranges of interest in this risk assessment (*i.e.*, 18 kg for 2 to 6 year olds and 59 kg for 7 to 31 year olds) are based on data from five national studies recommended by the USEPA (USEPA, 1997a). Specifically, these values were calculated by averaging the median body weight values for multiple age ranges within the age ranges of interest for this risk assessment. Inter-individual variability in this parameter is well-defined and the data are generally considered to be of high quality. Intra-individual variability and any uncertainties in this parameter would have an insignificant effect on any exposure and risk estimates calculated based on these data.

To provide some quantitative perspective on the potential impacts of this parameter on the risk calculations, average values for the 5th and 95th percentile values of body weights were calculated for the two age ranges of interest based on the same data used to calculate the best estimate (USEPA, 1997a). For 2 to 6 year old children, the 5th percentile estimate of body weight is 14 kg, while the 95th percentile is 23 kg. For 7 to 31 year old individuals, the 5th percentile estimate of body weight is 44 kg, while the 95th percentile is 81 kg. These values were used in the sensitivity analysis calculations presented below.

Averaging Time. The averaging time values for both children and adults are dependent on the exposure duration of interest and the type of health effect being examined. For carcinogenic effects (the primary focus of these sensitivity analyses), the exposure estimate of interest is the average dose received over a lifetime. As a result, the averaging time used in such risk calculations is set equal to a typical lifetime (*i.e.*, 70 years). This value also corresponds to the estimate of lifetime duration that is used in estimating most carcinogenic slope factors for quantifying carcinogenic risks. Thus, evaluations of a

range of averaging times is not appropriate and the averaging time was treated as a constant value in these sensitivity analyses.

Quantitative Implications of Variability and Uncertainty

In the next step of the sensitivity analysis, the implications of variability and uncertainty in the individual parameters used to calculate risks associated with incidental ingestion of dislodgeable arsenic were examined quantitatively. These evaluations began with the exposure calculations conducted using the best estimates described in the HHRA (the best estimates used in this analysis are the RME estimates used to quantify risk in the HHRA). Then, lifetime-averaged exposure estimates (in mg/kg-day) were calculated using the alternative parameter values described in the preceding section. Specifically, the alternative exposure estimates were calculated by first replacing the best estimate for an individual parameter with the low-end value described above, and then replacing the best estimate with the parameter's high-end value. In each case, the best estimate values were retained for all other parameters in the calculations. This approach was taken systematically for each parameter. The results of these calculations are summarized in Table 9.

To provide additional information regarding the implications of these results, two ratios were also derived for each set of calculations. First, the ratio between the exposure estimate derived using the high-end parameter value and that derived using the low-end parameter value was calculated. This ratio indicates the magnitude of the range of reasonable values for each parameter and the corresponding potential impact of parameter value choices on the exposure and risk assessment results. The second ratio examined is the ratio between the exposure estimate derived using the high-end parameter value and that derived using the best estimates of the parameter values. This ratio provides perspective regarding the degree to which use of high-end parameter values could yield exposure and risk estimates that are greater than those calculated using the best parameter assumptions.

As can be seen in Table 9, the use of high- and low-end alternative values for some parameters yields a broad range of corresponding exposure estimates. For example, the ratio between the high- and low-end exposure estimates for the range of parameter values examined for exposure frequency is 350. A ratio this large indicates that the underlying range of potential parameter values spans a large range and choices regarding the specific parameter values used in a risk assessment can significantly influence the exposure and risk assessment results. By contrast, this ratio was relatively small for other parameters. For example, the ratio between the high- and low-end exposure estimates for the range of parameter

values examined for skin surface area is approximately 1.4. Such a small ratio suggests that taking variability and uncertainty in the underlying parameter values into account in the calculations will have less impact on the exposure and risk assessment results.

The ratios between the exposure estimates based on the high-end parameter values and the best parameter estimates provide other insights. First, for many parameters, these ratios indicate that the best estimate used in the exposure and risk calculations is not substantially different from the high-end estimate. For example, for many parameters (*e.g.*, skin surface area, body weight, and RBA), this ratio is only slightly greater than 1. This finding suggests that the risk estimates derived using the best estimates would not be significantly altered by using alternative assumptions for these parameters. Second, for some parameters (*e.g.*, exposure frequency, exposure point concentration for treated Southern Pine, and HTE factor), the ratio between the high-end exposure estimate and the best estimate is substantially less than that between the high-end estimate and the low-end estimate. This result demonstrates the conservatism of the best estimate used in the exposure and risk calculations and suggests that actual exposures and risks are more likely to be less than the calculated best estimates than to exceed them.

Finally, with the exception of two parameters, all of the ratios between the high-end and best exposure estimates are less than a factor of two. For the remaining two parameters, the ratios between the high-end and best estimates are a factor of approximately 4 for the hand transfer efficiency factor and a factor of 2-5 for exposure frequency. These findings indicate that, even for these potentially most influential factors, the best estimates used in the risk estimates are not substantially less than alternative high-end values. In addition, these results provide quantitative support for identifying these two factors as key parameters that merit additional research to better characterize them for use in risk assessment. In addition, while waiting for additional information to be collected, special care should be taken in applying these parameter values in risk calculations and in interpreting results derived using currently available data.

The potential impacts of combinations of alternative parameter values were also considered in these sensitivity analyses. Some combinations of alternative parameter values could yield estimates of exposure and risk that exceed the best estimates by a factor that is greater than the maximum ratio between the high-end and best estimates calculated for any individual exposure parameter. Conversely, many combinations of alternative values could yield exposure estimates that are far less than those calculated using the conservative best parameter estimates. In actual exposure situations, many exposure parameter values are likely to be less than assumed in the best estimates, while some may be greater than

the assumed best values. The effects of such differences will tend to cancel each other out, making it highly unlikely that extreme worst-case combinations will occur.

5.5.5 Comparison of Risk Assessment Results with Other Studies

Additional context for the results of the risk assessment is provided by comparing them to the results of other assessments of exposures to CCA-treated wood. The assumptions and parameters used to assess exposures to dislodgeable arsenic, and the associated risks, in eight studies (including this HHRA) prepared by state and federal agencies, a non-profit environmental group, and private consultants are discussed below. This comparison is limited to dislodgeable arsenic because most (*i.e.*, ~80 to 90%) of the risks associated with CCA-treated wood are from exposures to dislodgeable arsenic.

Most of the risk estimates in the reviewed studies are based on CTE parameters. EPCs based on average or 95% UCLMs of dislodgeable arsenic concentrations are used in combination with CTE (*i.e.*, mean or median) parameters, including the HTE factor, relative bioavailability, and exposure frequency (EF). For most of the studies, estimated risks are based on playground exposures for young children (*i.e.*, 1-7 years old) exposed to dislodgeable arsenic on CCA-treated wood *via* incidental ingestion and dermal contact. The estimated risks in the MEDHS (1998), HS&WMR (2001), and Roberts and Ochoa (2001) studies are applicable for residential and/or playground exposures. Only the estimated risks for the child resident (ages 2-6) in this HHRA will be used in this discussion, and in comparisons with the other studies.

Based on the sensitivity analysis described above in Section 5.5.4.2 and the risks in the reviewed studies, it is apparent that differences in some key exposure assumptions can have a dramatic effect on estimated risks. For example, the EPCs (in μg As/cm^2 on the hand) for dislodgeable arsenic based on wipe study data are approximately 2-fold greater, on average, than EPCs based on hand loading studies. This difference is evident when EPCs from studies that relied on wipe data (*i.e.*, CPSC, 1990; Roberts and Ochoa, 2001; EWG, 2001; and Exponent, 2001) are compared to EPCs from studies that used hand loading data (*i.e.*, MEDHS, 1998; HS&WMR, 2001; and this HHRA). Data from the hand loading experiments conducted in the CADHS (1987) study were not used in this comparison because the average EPC calculated using these data is over 3-fold greater than the maximum EPC based on wipe sample data. This observation is not consistent with the results of studies (*i.e.*, SCS, 1998 and USEPA, 1999c) that have shown that a wipe sampling technique consistently removes more arsenic than a hand-loading method. It should be noted that very little detail is provided in the CADHS study, and therefore, it is

difficult to determine why the sampling procedure in this study yielded such elevated levels of arsenic on the hands.

Related to the use of wipe sample data, is the parameter that relates the amount of dislodgeable arsenic on the surface of treated wood to the amount actually transferred to the surface of the hands. Based on hand-loading and wipe sample results from a study conducted by SCS (1998), Exponent (2001) estimated that hand contact with treated wood removes approximately 3-fold less dislodgeable material than when a wipe is used. This is an important observation since several risk assessments, including CPSC (1990), Roberts and Ochoa, (2001) and EWG, (2001) do not employ a reduction factor to adjust the amount of dislodgeable arsenic removed by a wipe to what is transferred to the hands. This assumption can overestimate exposures to dislodgeable arsenic by approximately 3-fold.

Another key parameter is the HTE factor, which is used to estimate the percentage of the total amount of dislodgeable arsenic on the surface of the hands that is incidentally transferred to the mouth during hand-to-mouth contact. The HTE values do not differ by more than a factor of two in all of the studies, thus this parameter by itself will not make a significant difference in the estimated risks in most of the studies. It should be noted that an HTE factor was calculated based on information in the Exponent (2001) study; however, a unique method (compared to the methodology used in all of the other studies) of estimating incidental ingestion of dislodgeable arsenic was used in this study. Exponent used age-specific hand-to-mouth contact rate data, in combination with other exposure assumptions, to assess the amount of dislodgeable arsenic on the hands that is subsequently ingested.

The bioavailability of dislodgeable arsenic is another important parameter in the assessment of exposures and risks. While all of the risk assessments that evaluated dermal exposure to dislodgeable arsenic use a dermal absorption factor of between 1 and 2%, only the CADHS (1987) study and this HHRA used a reduced bioavailability for the ingestion route. The other studies assumed 100% bioavailability. CADHS (1987) used a range of 20 to 100% for the bioavailability estimate (basis for this range not provided), and this HHRA estimated a bioavailability of 47% based on animal data and the physical and chemical properties of arsenic in CCA-treated wood (see Section 3.4.4 for a review of the basis of the bioavailability estimate for dislodgeable arsenic). An assumption of 47% bioavailability reduces the internal dose, and the associated risk, by approximately 2-fold for the oral exposure route. Many of the risk assessments indicate that the bioavailability of dislodgeable arsenic is probably less than 100%; however, only the two studies indicated here use a reduced value to quantify exposures and risks.

Another important parameter is the EF estimate. While this parameter will vary depending on the receptor and exposure scenario evaluated, some of the studies used EF values that do not seem likely (*i.e.*, EF = 365 days/year), regardless of the receptor or exposure scenario being considered. As mentioned previously, most of the risk assessments evaluated exposures at a playground. The range of EF values in the studies where only playground exposures for a child (ages 1-7) are evaluated, is 1 to 365 days/year. The 1 day/year value is from the Exponent (2001) study in which daily risk estimates were calculated for playground exposures with the intention that risk management decisions would be made based on the average number of days children in a particular area visit public playgrounds. Excluding the Exponent value from the range of EF values for playground exposures, the average EF estimate is approximately 215 days/year. This value is nearly two and a half times greater than the RME EF estimate of 88 day-equivalents/year used in this HHRA for a playground child, ages 2-6. The EF used in this HHRA is based on the 90th percentile amount of time that young children spend outdoors at a playground and/or on school grounds, and is based on time activity pattern data in the USEPA's *Exposure Factors Handbook* (1997b). There is a substantial amount of time activity data available in USEPA (1997b), and in the published literature, for a multitude of different activities, geographic regions, and age ranges. The use of professional judgment to estimate this important parameter will contribute to the uncertainty in the assessment of exposures and can result in a substantial overestimate of risk.

By convention, arsenic toxicity criteria (*e.g.*, RfD_{oral} and CSF_{oral}) on the USEPA's IRIS website is used in the assessment of human health risks. However, USEPA, Region 8 (2001) has developed a subchronic RfD_{oral} that is applicable for exposure durations up to 7 years. Therefore, this toxicity criterion would be appropriate for most of the exposure scenarios involving children in the studies, and is the non-cancer arsenic toxicity criterion used in this HHRA to assess risks for children exposed to dislodgeable arsenic in both a residential and a playground scenario. However, because cancer risks are the primary driver in these studies, application of a subchronic RfD_{oral} would not have a noticeable effect on the conclusion of significant risk. The evolving scientific debate regarding the carcinogenicity of arsenic could ultimately have a significant impact on the estimated cancer risks from exposures to CCA-treated wood, depending on how the current CSF_{oral} for arsenic is adjusted.

While there are other exposure assumptions and parameters that affect estimated risks for exposures to dislodgeable arsenic, the most significant parameters are discussed here. Based on this brief review of the leading risk assessments of CCA-treated wood, it is apparent that the choice of exposure assumptions and parameters can have a dramatic effect on the estimated risks. The estimated cancer risks in these studies range from 1.0×10^{-7} to 9.0×10^{-3} with a mean of 1.5×10^{-3}; and the estimated non-cancer

risks range from 0.002 to 20.0 with a mean of 3.7. As noted previously, these risks are based on conservative assumptions and average exposure parameters, in most cases. The estimated cancer and non-cancer risks in this HHRA, using RME parameters for a 2-6 year old resident exposed to dislodgeable arsenic from treated Southern Pine, are 3.0×10^{-6} and 1.9×10^{-3}, respectively. Both of these values are near the bottom of the estimated risk ranges for all of the studies. As discussed above, there are a number of reasons why the risk estimates in this HHRA are lower than the risks in the other studies, including the use of EPCs based on hand-loading data, a HTE factor near the lower end of the range, a reduced bioavailability of 47% for dislodgeable arsenic, an EF based on an RME estimate of the amount of time spent outdoors, specifically at a residence, and the use of a subchronic RfD_{oral} for arsenic. By themselves, these exposure and toxicity assumptions do not have a substantial effect on the estimated risks; however, when used in combination, the effect can be significant. Gradient is confident that the exposure and toxicity assumptions used in this HHRA are supported by current scientific knowledge, and that these assumptions do not underestimate actual risks, and in fact, may overestimate actual risks.

6 Summary and Conclusions

6.1 Summary

Gradient prepared a HHRA for Arch Wood Protection, Inc., and Osmose, Inc., to quantify potential health risks from exposure to arsenic associated with CCA-treated wood. Using conservative assumptions and parameters to evaluate exposures, the results of the HHRA indicate that use of CCA-treated wood in both a residential and playground setting does not pose a significant health risk to children or adults.

The HHRA was conducted in accordance with current USEPA risk assessment guidance and recent scientific literature. Central tendency exposure (CTE) or average, and reasonable maximum exposure (RME) parameters were used to quantify exposures for a residential, and a playground exposure scenario. The residential scenario included a male/female child ages 2-6 years, and a male/female child and adult ages 2-31 years. The playground scenario included a male/female child ages 2-6 and 7-12 years. Both exposure scenarios evaluated incidental ingestion and dermal contact with dislodgeable arsenic; and incidental ingestion, dermal contact, and inhalation exposure to CCA-impacted soil.

6.2 Results and Conclusions

The estimated cancer and non-cancer health risks for both exposure scenarios are summarized below in Tables 6-1 through 6-4. Cancer risks for the residential and playground scenarios are summarized in Tables 6-1 and 6-2, respectively. Non-cancer risks for the residential and playground scenarios are summarized in Tables 6-3 and 6-4, respectively.

Tables 6-1 and 6-2
Summary of Cancer Risks

Table 6-1
Residential Scenario

Exposure Medium	Exposure Pathway	Estimated Lifetime Cancer Risk			
		Central Tendency Exposure		Reasonable Maximum Exposure	
		Ages 2-6	Ages 2-31	Ages 2-6	Ages 2-31
Soil Arsenic	Ingestion, dermal, and inhalation	1.7×10^{-7}	3.9×10^{-7}	8.2×10^{-7}	1.4×10^{-6}
Dislodgeable Arsenic from Southern Pine*	Ingestion and dermal	9.6×10^{-7}	2.3×10^{-6}	3.0×10^{-6}	5.2×10^{-6}
Dislodgeable Arsenic from Southern Pine w/ Pressure-Applied Water Repellent**	Ingestion and dermal	2.0×10^{-6}	5.0×10^{-6}	6.3×10^{-6}	1.1×10^{-5}

Table 6-2
Playground Scenario

Exposure Medium	Exposure Pathway	Estimated Lifetime Cancer Risk			
		Central Tendency Exposure		Reasonable Maximum Exposure	
		Ages 2-6	Ages 7-12	Ages 2-6	Ages 7-12
Soil Arsenic	Ingestion, dermal, and inhalation	2.5×10^{-8}	1.4×10^{-8}	1.3×10^{-7}	6.0×10^{-8}
Dislodgeable Arsenic from Southern Pine*	Ingestion and dermal	5.4×10^{-7}	4.8×10^{-7}	1.5×10^{-6}	1.3×10^{-6}
Dislodgeable Arsenic from Southern Pine w/ Pressure-Applied Water Repellent**	Ingestion and dermal	1.2×10^{-6}	1.0×10^{-6}	3.3×10^{-6}	2.7×10^{-6}

** Most commonly used type of treated wood in the U.S. (AWPA, 1998).*

*** Treated wood type resulting in greatest risk. This wood type accounts for only about 6% of the treated lumber sold in the U.S. (RISI, 1990).*

Tables 6-3 and 6-4
Summary of Non-Cancer Risks

Table 6-3
Residential Scenario

Exposure Medium	Exposure Pathway	Hazard Quotient			
		Central Tendency Exposure		Reasonable Maximum Exposure	
		Ages 2-6	Ages 2-31	Ages 2-6	Ages 2-31
Soil Arsenic	Ingestion and dermal	9.3×10^{-5}	1.6×10^{-3}	4.9×10^{-4}	6.2×10^{-3}
Dislodgeable Arsenic from Southern Pine*	Ingestion and dermal	6.0×10^{-4}	1.2×10^{-2}	1.9×10^{-3}	2.7×10^{-2}
Dislodgeable Arsenic from Southern Pine w/ Water Repellent**	Ingestion and dermal	1.3×10^{-3}	2.6×10^{-2}	3.9×10^{-3}	5.8×10^{-2}

Table 6-4
Playground Scenario

Exposure Medium	Exposure Pathway	Hazard Quotient			
		Central Tendency Exposure		Reasonable Maximum Exposure	
		Ages 2-6	Ages 7-12	Ages 2-6	Ages 7-12
Soil Arsenic	Ingestion and dermal	1.5×10^{-5}	6.9×10^{-6}	8.0×10^{-5}	3.0×10^{-5}
Dislodgeable Arsenic from Southern Pine*	Ingestion and dermal	3.4×10^{-4}	2.5×10^{-4}	9.6×10^{-4}	6.6×10^{-4}
Dislodgeable Arsenic from Southern Pine w/ Water Repellent**	Ingestion and dermal	7.2×10^{-4}	5.3×10^{-4}	2.0×10^{-3}	1.4×10^{-3}

** Most commonly used type of treated wood in the U.S. (AWPA, 1998).*
*** Treated wood type resulting in greatest risk. This wood type accounts for only about 6% of the treated lumber sold in the U.S. (RISI, 1990).*

The cancer risk estimates for both exposure scenarios, based on either central tendency or RME parameters, are within the USEPA's acceptable cancer risk range of 1×10^{-6} to 1×10^{-4}. The non-cancer risk estimates for both exposure scenarios are below the USEPA's acceptable non-cancer risk limit of 1.0. Based on the results of the HHRA, average and high-end exposures to arsenic associated with CCA-treated wood in both a residential and playground setting does not pose a significant health risk to children or adults.

7 References

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